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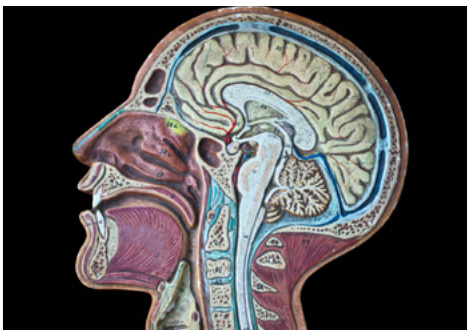

Head and neck

AJGP Australian Journal of General Practice

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We acknowledge the Traditional Custodians of the lands and seas on which we work and live, and pay our respects to Elders, past, present and future.

Contents

Approach to allergic rhinitis in the primary care setting

Deepika Gunda, Javaria Mustafa, Nicholas Agar, Peter Goss

This article is part of a longitudinal series on ear, nose and throat conditions.

Background
Allergic rhinitis (AR) is a common condition that affects 19% of Australians in the community, accounting for approximately 0.6% of all general practitioner presentations. Recent years have seen the emergence of new treatment options, many of which can be delivered in the primary care setting.

Objective
The aim of this paper is to provide a contemporary and accessible framework for the clinical assessment, investigation and management of AR in the primary care setting, and to establish appropriate referral criteria for ear, nose and throat and/or allergist/immunologist referral.

Discussion
AR is common, and can have a significant effect on both quality of life and function. The diagnosis can be made based on history, examination and appropriate investigations, including serum specific allergen IgE (immunoglobulin E) and/or skin prick testing. Radiological imaging is not part of the work-up for AR. Management consists of four main aspects: allergen avoidance, pharmacotherapy, immunotherapy and procedural interventions. Biological pharmacotherapies are on the horizon.

ALLERGIC RHINITIS (AR) is a Type 1 IgE-mediated hypersensitivity reaction to airborne allergens, most commonly dust mites, tree, weed or grass pollens, in addition to animal dander and mould.

The pathophysiology is well understood, with allergen exposure triggering an immediate early phase response with mast cell degranulation and a histamine response in the nasal mucosa[1] causing rhinorrhoea, postnasal drip, sneeze, itch and nasal congestion.[2] Eosinophils and other inflammatory cells then contribute to late phase symptoms of chronic nasal obstruction, hyposmia and nasal mucosal hyper-reactivity.

AR is a prevalent condition affecting people of all ages, with the prevalence in Australia during 2017–18 being almost one in five.[3] Worldwide, the prevalence of AR ranges from 5% to 50%;[4] however, it is often underdiagnosed and inadequately managed.[5] AR can have a significant effect on quality of life and affect school/work, productivity, sleep quality, mood and concentration in both adults and the paediatric age group.[4,6,7]

Although AR peaks in the 25–44 years age range,[3] it is important not to miss this diagnosis in children presenting with sleep disordered breathing, obstructive sleep apnoea, snoring, behavioural issues and frequent upper respiratory tract infections. Children are underdiagnosed because they are unable to describe their symptoms. AR accounts for 0.6% of general practitioner (GP) presentations,[3] likely an under-representation due to self-medicating from pharmacies. AR should be considered in all of those with asthma because the concurrence is 60–80%.

The risk factors for developing AR include genetic factors, atopy during childhood, eczema and higher socioeconomic status.[4] Interestingly, some factors previously thought to contribute to the risk of AR, such as tobacco smoking, pollution, and early exposure to dust mites, pollen, animal dander and fungal allergens, all have inconclusive evidence.[4] The prevalence of AR in large polluted Asian cities is on the rise compared with the rural areas of the same countries.[8] Although the connection between poor air quality and AR is not understood, it is clear that more people are developing AR in those cities with poorer air quality.[8]

There are certain factors that have been demonstrated to be protective against developing allergic rhinitis. This includes Level 2 evidence for breastfeeding, and some evidence for microbial diversity.[4,9]

In recent years, there have been several developments in the investigation of AR, including component resolved diagnostics,[10] e-diaries[11] and aeroallergen monitoring applications.[12] Management has broadened also, including the recent introduction of combination intranasal corticosteroid and antihistamine sprays, as well as the availability of self-administered sublingual immunotherapy wafers and dissolvable

tablets that can target grass pollen and house dust mites.[13] Antigen-specific immunotherapy is effective, available and the only form of disease-modifying therapy available currently.[4]

For comprehensive information on this topic, the authors highly recommend accessing the Australasian Society of Clinical Immunology and Allergy (ASCIA) *Allergic rhinitis clinical update*, published in 2022 and available online.[14] Alternatively the updated *International consensus statement on allergy and rhinology* is an extensive document that has pooled evidence-based reviews and recommendations for the investigation and management of allergic rhinitis.[4]

The objective of this article is to provide a contemporary and accessible framework for the clinical assessment, investigation and management of AR in the primary care setting and to establish appropriate referral criteria for ear, nose and throat and/or allergist/immunologist referral.

Assessment

The aim of assessment is to determine whether the condition is indeed AR, to gauge the severity and effect of disease and to exclude other differential diagnoses, including common conditions (ie non-allergic rhinitis, chronic rhinosinusitis), and to exclude red flag conditions, including malignancies and benign neoplasms. A detailed and targeted history is the first (key) element of the clinical assessment for AR. This includes establishing the hallmark symptoms of rhinorrhoea, sneeze, itch, nasal obstruction and concurrent allergic conjunctivitis, as well as associated symptoms such as postnasal drip and nocturnal cough. Establishing seasonal patterns and/or environmental triggers, including pets in the house and soft toys, is of value and can help guide subsequent specific allergen testing. A family or personal history of atopy might be corroborative. An early referral to otolaryngology should be considered if unilateral nasal obstruction, progressive blood-stained, malodorous nasal discharge or unilateral nasal polyps are present. Validated survey instruments can be used to screen for AR and might offer a more structured and standardised means of obtaining the clinical history, including the effect on quality of life; in addition, they can more objectively assess treatment response. For primary care, the Control of Allergic Rhinitis and Asthma Test (CARAT-10) is a good option, being relatively short and accessible.[15] These questionnaires can be used in children and adults and concurrently assess for conjunctivitis and asthma.

Physical examination with anterior rhinoscopy might show an increased volume of clear, sticky mucus and pale oedematous swelling of the inferior turbinates. Other causes of nasal obstruction, such as a significant septal deviation, polyps or a sinonasal mass, should be excluded. Fibreoptic nasendoscopy undertaken by an otorhinolaryngologist or an allergist can assess for allergic/oedematous changes of the middle turbinate, exclude a large adenoid in older children who can tolerate this examination and exclude alternative pathologies. Nasoendoscopy is not essential to the diagnosis of AR, but direct anterior rhinoscopy, with an effective light source, is highly informative and should be performed in all patients with nasal symptoms.

If the history and examination is suggestive of AR, the diagnosis can be further supported by either referring the patient to an allergist for skin prick testing (generally accepted as the gold standard) or requesting a specific panel of blood tests available from any pathology provider. The latter is more accessible to most GPs and might include a full blood count and as well as serum allergen-specific IgE levels (previously known as the radioallergosorbent test or RAST) to specific allergens that are suggestive from the history. Medicare will rebate four single allergens, two mixed allergen panels or two single and one mixed allergen panel per testing episode, with additional panels available usually at a minor cost to the patient. These tests should be targeted to the patient's specific environmental triggers: Dust and Mite Mix, Grass Mix, Pollen Mix and Animal Danders should be considered because they are common. Animal Danders should be specific if the patient has exposure to a certain animal because mixes are not specific. Other available tests include Weed Mix, Tree Mix, Mould Mix and Occupational Mixes (only to be requested if a person has a specific occupational exposure; eg bakers).[16]

For accurate skin prick testing, it is important to ensure that patients have withheld oral H_1 receptor antihistamines for at least two to seven days prior and topical antihistamines for at least two days. Tricyclic antidepressants might affect results; this will be taken into consideration by the allergist if skin prick testing is inconclusive. Systemic corticosteroids and topical corticosteroids (provided not used at the testing site) can be continued. Of note, an elevated serum IgE or an eosinophilia is non-specific, unless in eosinophilic airways disease,[17] and false positives are common. A positive test without corroborative clinical symptoms is not relevant and should not be acted on.

Radiological imaging is not indicated in the work-up for AR. Imaging should only be considered to rule out other causes of nasal obstruction/rhinorrhoea if the history and examination is unconvincing.[18] It is important to note that nasal obstruction might be a combination of allergy and anatomical obstruction from large adenoids and/or septal deviation.[19]

Management

The four main aspects to the management of AR are allergen avoidance, pharmacotherapy, immunotherapy and, occasionally, surgery.

Allergen avoidance

Avoidance measures are considered a low-risk option and can be effective. They do frequently entail significant inconvenience, cost, lifestyle modification and even disruption to one's occupation. Adherence can be challenging, even for the most diligent of patients. Allergen avoidance has been found to be specifically effective for the following allergens: house dust mites, cockroaches, pets, rodents and pollen.[4] Portable home air filter units have increasing evidence of their effectiveness against some aeroallergens, especially pollen.[20] House dust mite avoidance is nearly impossible in areas of high prevalence, such as temperate and humid coastal regions of Australia. Reduction measures might be helpful, but certain measures, such as sheet/pillow protectors, might be expensive.

Pharmacotherapy

Pharmacotherapy can be separated into first-line agents, second-line agents and other options.

First-line agents are all available over the counter and include the following:

- Intranasal saline sprays or rinses: these can be used to clear mucous and reduce local allergen load. These are a cost-effective management option with no side effects and can help alleviate symptoms.[21]
- Intranasal corticosteroids (eg mometasone, budesonide, fluticasone): used regularly for a minimum of four weeks these are highly effective and have a preventative role.[22] Correct administration technique is important.[23] Poor technique might increase the risk of bleeding from Little's area and can be combated by both improved technique as well as the use of emollients or combination antibiotic, antifungal and corticosteroid ointments.
- Intranasal antihistamines (eg azelastine, levocabastine): these have minimal side effects and can reduce symptom burden acutely, with a reduction in itch, sneeze, rhinorrhoea and eye symptoms soon after they are used.[24]
- Combinations of intranasal corticosteroid and antihistamine sprays (eg azelastine/fluticasone, olopatadine/mometasone): these are now increasingly recognised as an excellent first-line option. In Australia, azelastine/fluticasone has recently been scheduled S2 and is available over the counter, whereas olopatadine/mometasone is prescription only. These combinations have the benefit of the preventative action of the steroid and the symptomatic relief of the antihistamine when used for a minimum of one month. They have only been available in recent years and have been strongly recommended for the management of AR.[4] The senior author of this paper feels a strong argument can be made to use one of these combination sprays as the first-line treatment, particularly as more rapid results might improve compliance.
- Oral H_1 receptor antihistamines (eg cetirizine, loratadine, fexofenadine): these are less effective, have a slower onset and have more side effects than topical antihistamines. However, they have a role to play in those unwilling to comply with topical delivery.[24]

One of the few second-line agents is intranasal sodium cromoglycate, which can be a useful prophylactic treatment and has minimal adverse effects.[25,26]

Other pharmacotherapy options that should only be considered if first- and second-line agents are insufficient include the following:

- A 'pulse' or brief course of oral corticosteroids (eg prednisolone) if not contraindicated: this can be prescribed with the aim of improving congestion to allow the penetration of first-line topical sprays. Oral corticosteroids should not be prescribed routinely or frequently due to systemic adverse effects.[4]
- Leukotriene receptor antagonists (eg montelukast): these can alleviate symptoms.[27] Despite having a rare side effect of behavioural disturbance, leukotriene receptor antagonists are particularly useful in young children and those with asthma who cannot tolerate nasal sprays.[28]
- Oral and intranasal decongestants (eg phenylephrine, diphenhydramine, pseudoephedrine): these are effective in the short term at improving nasal patency and provide rapid temporary benefit. However, they cause significant rebound congestion with extended use and patients can very easily become dependent on these medications (rhinitis medicamentosa); therefore, their use should be short-lived only.[29]

Immunotherapy

Immunotherapy can be considered if the above pharmacotherapy agents do not sufficiently treat the symptoms or the patient is seeking an alternative to medical management. Immunotherapy involves the controlled exposure of allergens to a patient in order to reduce the hypersensitivity response. It can be delivered using several methods:

- subcutaneous immunotherapy (SCIT) with serial injections is one of the most common delivery methods and has the strongest evidence of efficacy, but also the highest risk of systemic adverse events[4]
- sublingual immunotherapy (SLIT) also has evidence of efficacy and the added benefit of a less-invasive self-administration route and less risk of adverse side effects compared with SCIT.[4] The duration of protection from immunotherapy is 10–20 years. This might be much more acceptable for the paediatric population, who might wish to avoid injections.[4,30,31]
- GPs might be asked to assist with the maintenance injections for aeroallergen immunotherapy prescribed by allergists, using a shared care model. GPs need to be aware of the risks of this therapy, including possible anaphylaxis, and have necessary resuscitation equipment and have completed ASCIA anaphylaxis training. GPs should also have clear guidelines from the prescriber about dose adjustment if injection intervals vary or side effects occur.[32]

The non-Pharmaceutical Benefits Scheme prescription of grass, pollen and dust mite sublingual tablet or wafer therapy is safe and within the remit of GPs for monosensitised patients who have a consistent clinical history and aligning investigations. For more complex patients, usually an allergist will need to be involved.[33]

New biological agents have shown benefits that might be relevant in the management of AR.[34] These monoclonal antibodies can target specific aspects of the allergy pathway. Omalizumab, which targets IgE, has a significant body of evidence supporting its use in AR, but it is currently not approved for use for this indication.[34]

Surgery

If medical measures are insufficient to treat the patient's symptoms, then referral to an otolaryngologist should be considered. Inferior turbinate surgery might be performed to reduce the soft tissue bulk of the nasal sidewall, thereby decreasing nasal congestion and rhinorrhoea. If a significant septal deviation exists, then a septoplasty can help address nasal obstruction. In children, adenoidectomy and/or ablative procedures on the inferior turbinate can be undertaken. Of note, however, the underlying allergic tendency will not be addressed by surgery and the patient will likely still require pharmacotherapy to address itch, sneeze and rhinorrhoea.[35] Vidian neurectomy and radiofrequency ablation are procedures that disrupt the posterior nasal nerves and are only considered infrequently in the management of AR.[4]

See Figure 1 for a flowchart summary of the approach to the management of AR.

Conclusion

AR causes debilitating symptoms for up to 19% of Australians, with significant effects on quality of life and function.[3] An assessment in the primary care setting, with a targeted history, examination and panel of investigations, should be able to identify those with a significant burden of allergic disease. Radiological imaging is not part of the work-up for AR unless there is uncertainty regarding the diagnosis. Allergen avoidance, as well as treatment with intranasal saline, corticosteroids and antihistamines, might provide relief for the majority of those with AR. Combination corticosteroid/antihistamine sprays are highly effective and convenient and are increasingly considered a first-line therapy. The increased availability of prepackaged self-administered sublingual immunotherapies targeted towards dust mite and pollen allergies has made immunotherapy much more feasible and convenient. Referral to an allergist should be considered if specific allergen tests show significant airborne allergen sensitivity. Surgical referral is required if red flags are present, if significant anatomical obstruction exists or if there is inadequate response to medical management. Biological agents have an emerging role and might become part of the treatment paradigm in the future.

Key points

- Rhinorrhoea, postnasal drip, sneeze, nasal congestion and irritation are common symptoms.
- Serum specific IgE and skin testing help diagnose allergic rhinitis.
- Imaging does not play a role in the diagnosis.
- Combination intranasal corticosteroids and antihistamine sprays are a new and effective treatment.
- Immunotherapy has a well-established role in therapy, with self-administered wafers now available on prescription for dust mite and grass pollens.

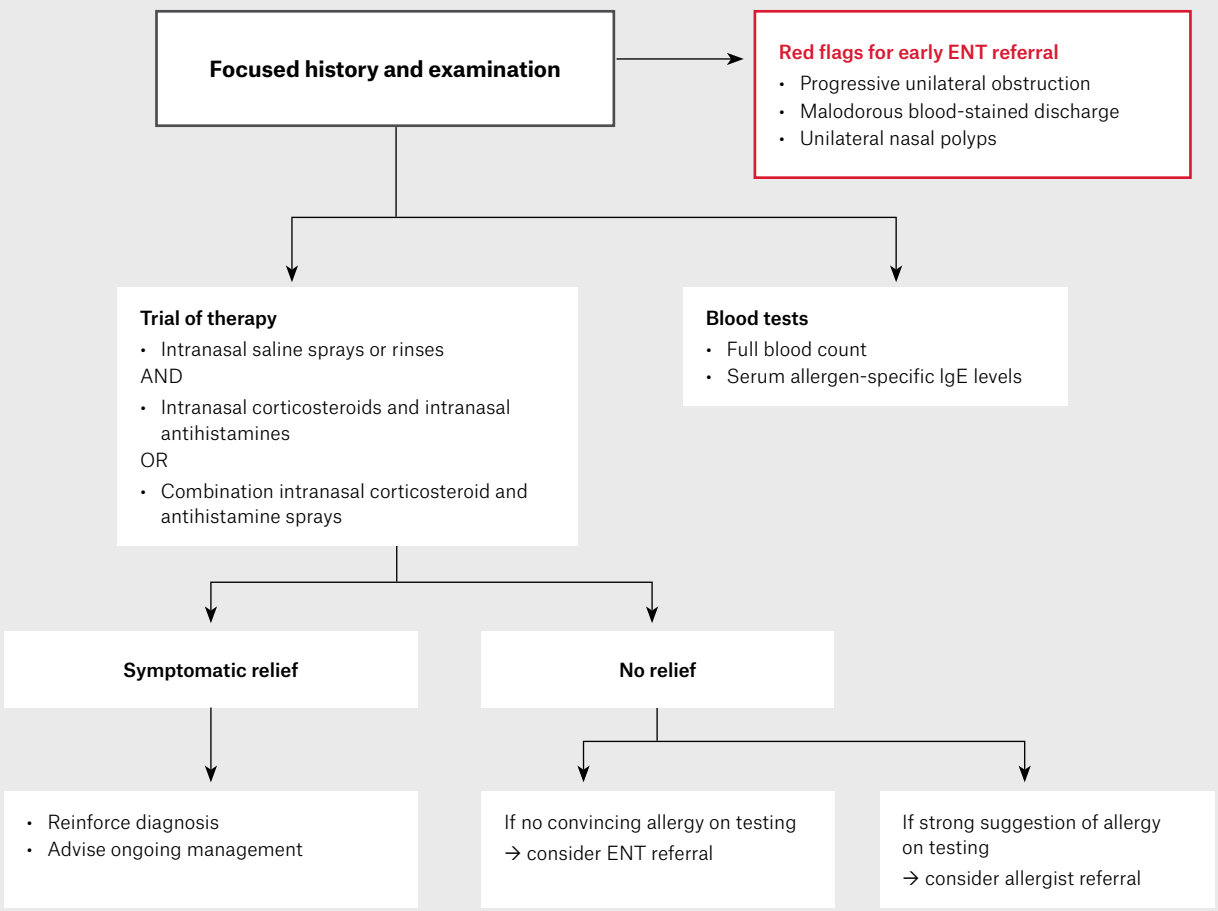

Figure 1. Flowchart of management for allergic rhinitis.
ENT, ear, nose and throat; IgE, immunoglobulin E.

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An approach to the postoperative parotidectomy patient in primary care

Michael Wong, Sarju Vasani

Background
Parotid gland masses form part of a heterogenous subset of head and neck pathology. Surgery for both benign and malignant disease is relatively common in Australia and is associated with a diversity of idiosyncratic postoperative phenomena that might represent a challenge to identify and navigate in the primary care setting.

Objective
The aim of this paper is to provide the primary care physician with a useful guide for the assessment, evaluation and initial management of common and not-to-be-missed clinical presentations post parotid surgery, and a framework for appropriate escalation and referral.

Discussion
Primary care can be a valuable setting for the identification and initial management of common complaints post parotid surgery and providing patient access to escalation and onward referral where necessary. Ambiguous, unresolving or worsening presentations should be referred to the treating (or local) surgeon or department.

PAROTID GLAND NEOPLASMS comprise a diverse subgroup of head and neck pathology. Up to four in five parotid neoplasms are benign, with pleomorphic adenoma and Warthin tumours being the most common.[1,2] Malignancy of the parotid is uncommon, comprising 20% of parotid tumours and 3–6% of all head and neck malignancies.[3] However, in Australia, high levels of solar ultraviolet light exposure lead to a concomitantly elevated rate of cutaneous malignancy, increasing the frequency of metastatic parotid lesions such as squamous cell carcinoma.[4]

The location of the parotid gland within the complex anatomical regions of the neck and face and its proximity to important neurovascular structures, including the facial nerve, warrant careful surgical technique to minimise complications.[1,5] Despite this complexity and risk, operative intervention for parotid disease – namely parotidectomy – has remained a mainstay of treatment for both benign and malignant disease.[6–9] It also continues to be of particular relevance in the treatment of benign disease secondary to the risk of malignant transformation of lesions, including pleomorphic adenoma and Warthin tumours.[7,10]

No procedure is without risk, and both common and rare presentations and complications after parotid surgery are well documented in the literature. Given the relative frequency of parotid surgery in Australia and the diversity of complaints patients might present with to their general practitioner (GP) thereafter, the aim of this article is to guide the primary care clinician in identification, triage, initial management and referral when encountering these complaints in general practice.

Anatomy and function of the parotid gland

Of the paired major salivary glands, the parotid glands are the largest.[11] They are exocrine glands responsible for the production and release of saliva – an acidic, mucoserous secretion – into the oral cavity.[11] Saliva is produced by acinar cells within the glands and has a key role in lubrication of the oral cavity, which aids taste, mechanical and enzymatic breakdown of food, and deglutition. It also has a role in pH buffering, humoral immunity and maintaining the health of dentition.[11] The parotid glands are responsible for 20% of unstimulated saliva flow and more than 50% of stimulated flow.[11]

The parotid is located within the parotid space, a deep compartment of the neck at the lateral neck and face.[12] It is bordered superiorly by the zygoma, posterosuperiorly by the external auditory meatus and posteroinferiorly by the styloid process.[13] Inferiorly, the tail of the gland overlies the

angle of the mandible.[14] The gland comprises two lobes separated by the facial nerve and its branches: a superficial and a deep lobe.[14] The superficial lobe overlies the masseter, with the deep lobe positioned deep to the ramus of the mandible.[14] Stensen's duct, the main excretory duct of the parotid, originates from the superficial lobe, coursing superficial to the masseter until piercing the buccinator to emerge in the oral cavity opposite the superior second molar.[14] Other structures within the gland include the retromandibular vein, external carotid artery and auriculotemporal nerve (part of V_3), which also receives secretomotor parasympathetic fibres from the otic ganglion.[13,15]

Typical postoperative course

After a parotidectomy, and in the absence of complications, patients will typically have an inpatient stay of at least two nights. For a minimum of 48 hours, the surgical drain is left in situ and then removed provided that the drain output is less than 20–30 mL in a 24-hour period. As an inpatient, the patient's facial nerve function will be assessed clinically, and any issues with the wound will be noted.

Upon discharge, patients will usually be reviewed in the outpatient setting 7–10 days postoperatively. If skin closure was performed with non-absorbable sutures, at this visit, the sutures will be removed. The wound is also reviewed at this time in addition to the patient's facial nerve function. The histology results will have a further role in determining the type of follow-up. In the case of benign masses completely excised with clear margins, non-urgent follow-up several months after the procedure is usually recommended. Depending on the type of benign mass, the patient might still undergo surveillance thereafter or be discharged to the referring GP. For malignant masses, patients will typically be discussed at a head and neck multidisciplinary team meeting to guide further treatment and follow-up, which will include regular review with at least a three-monthly frequency.

Evaluation and initial management of common presenting complaints post parotid surgery

Parotid surgery can be associated with several postoperative presentations relating to nerve function, the surgical wound, infection, operative site swelling and the function of eating (Table 1).[5,16] These phenomena might present at various time points after surgery (Table 2).

Facial muscle weakness

One of the most pertinent risks of parotid surgery is the risk of injury to the facial nerve, and its associated consequences. Patients can present with facial muscle weakness and asymmetry in the immediate postoperative period depending on the branches of the nerve affected, and facial nerve function should be regularly monitored clinically. It is not uncommon for traction on the facial nerve to occur intraoperatively, particularly when the tumour abuts the nerve,[7] leading to a neuropraxia that typically resolves within 4–6 weeks after surgery.[16] Nerve monitoring, which can provide an indication of facial nerve function intraoperatively, is commonly used, but injury – unless observed during surgery – might only be apparent after the procedure. At this stage, patients should be reassured that this is likely temporary, and the operator should be made aware of the patient's status and outcome.

Persistence of this weakness and asymmetry, and synkinesis, might be a sign of long-term or permanent injury to the facial nerve.[5] This can result in a cosmetic and functional problem for the patient. Any facial muscle weakness resulting in incomplete closure of the eye at any point following surgery should be managed with regular lubricating eye drops, accompanied by eye protection at night.[5] Persistence of poor eye closure warrants review by an ophthalmologist for consideration of tarsorrhaphy[5] and/or implantation of a gold weight in the eye lid. More generally, referral to a plastic surgeon might also be considered for facial reanimation, depending on the type of parotidectomy performed and nerve branches affected. It is recommended that the treating surgeon or unit be involved throughout this course.

Altered cutaneous sensation

Altered facial, neck and ear sensation is a common complaint after a parotidectomy. The great auricular nerve (GAN) provides cutaneous sensation to the earlobe and inferior to the pinna.[7] It is routinely sacrificed during parotidectomy to facilitate surgical access and aid dissection.[7] Patients in which this has occurred will be notified by their surgeon and should expect lifelong hypoesthesia in the sensory distribution of the nerve. Although this is often very noticeable in the immediate postoperative period, patients will usually become accustomed to this over time.

Disruption of cutaneous nerves at the incision site is also common, and an area of hypoesthesia that decreases in size and severity over time is expected. However, the surgical scar will remain insensate. Likewise, altered sensation in the distribution of the auriculotemporal nerve might be expected, given its anatomical relationship to the parotid.

Pain and trismus

Operative site and wound pain is to be expected in all patients after surgery and is usually adequately controlled with regular simple analgesia. This should subside by approximately 14 days after surgery. Any pain that is persistent or out of proportion to the surgical incision or wound status is rare and should be escalated to the treating surgeon for further management.

The presence of facial pain and spasm in the parotid region on the first bite of a meal that diminishes over subsequent bites might be indicative of first bite syndrome, which is associated with surgery to the deep lobe of the parotid.[17] Incidence can be as high as 10% and generally presents within the first three months after surgery.[17] Conservative management with simple analgesia and behaviour modification might be instituted at first instance, with speech pathology review and surgical follow-up recommended in the medium to long term. This is not to be confused with trismus, which might arise from masseteric inflammation and is usually mild and self-limiting with jaw exercises.[5] Trismus in the setting of infective signs and symptoms is more concerning for a deep neck space infection and care should be escalated immediately.

Gustatory sweating

Frey syndrome, or gustatory sweating, is a phenomenon that arises later from aberrancy of the parasympathetic secretomotor innervation of the parotid gland with autonomic cutaneous innervation of the face.[5,7,10] This is documented to arise in

as many as 25 to 60% of patients, with a median time to presentation of 11 months after surgery, and presents with flushing and sweating with meals.[9,10] Patients with Frey syndrome should be referred to their treating surgeon for consideration of treatment with botulinum toxin.[5,19]

Neck swelling

Neck swelling is most often observed in the immediate postoperative period. It is usual practice in Australia for a surgical drain to be kept in the neck for 48 hours postoperatively to safeguard against the formation of a haematoma and to aid in its early detection.

New, rapidly progressive neck swelling with or without the presence of bleeding is uncommon and should be managed with compression and timely referral to the local emergency department for ongoing management.[2] Infection, including the presence of a collection, is also uncommon and might present with neck swelling in addition to overlying erythema and induration at the site. The patient might also present with systemic features of infection. Minor superficial infection might be amenable to treatment with oral antibiotics but in general should be referred early to the local emergency department or treating team for consideration of cross-sectional imaging, intravenous antibiotics and surgical intervention.

Table 1. Summary of common presenting complaints, features and initial management

Presenting complaint (differentials)	Frequency	Features	Red flags	Investigations/treatment/ escalation
Facial muscle weakness (iatrogenic facial nerve injury, neuropraxia or sacrifice)	• Common – temporary (up to 42%)[9] • Rare – permanent, synkinesis (<1%)[9]	• Partial or complete weakness of one or more branches of the facial nerve on the operated side – 38% resolve within 1 month, 78% within 3 months[9] • Weakness of eye closure can increase the risk of corneal injury	• Progression of weakness	• Conservative management initially – reassurance and monitoring • Nocte eye protection, lubricating eye drops, ophthalmology referral if indicated (weakness of eye closure) • Monitoring and review with treating surgeon/unit • Speech pathology referral for synkinesis and long-term or permanent dysfunction • Ophthalmology or plastic and reconstructive surgery referral for consideration of tarsorrhaphy/gold weight and facial reanimation for long-term/severe dysfunction • Progression warrants urgent surgical referral
Altered sensation, particularly in GAN distribution (iatrogenic GAN sacrifice or injury)	• Common (34%)[18]	• Altered facial and/or ear sensation	• Increase in severity of sensory loss or paraesthesia	• Conservative management – reassurance and monitoring • Progression warrants urgent surgical referral
Scarring	• Common (affects all patients to an extent) • Uncommon – keloid or hypertrophic scarring (4%)[18]	• Scarring occurs to varying degrees in all patients	–	• Reassurance – should fade over time • Scar massage • Vitamin E cream • Surgical referral in severe instances for consideration of scar revision or steroid injection
Asymmetry of facial contour	• Common (affects all patients to an extent)	• Depression or hollowing of the face at the surgical site, which matures over time	–	• Reassurance
Facial gustatory sweating and flushing (Frey syndrome)	• Common (up to 60%)[10] • Uncommon – severe symptoms (up to 15%)[5]	• Facial flushing and sweating with meals	–	• Re-referral to treating surgeon for consideration of botulinum toxin injection
Pain on first bite that improves with subsequent bites (first bite syndrome)	• Common (10%)[17]	• Typically arises within the first 3 months after surgery[17]	–	• Conservative management with simple analgesia, (soft) diet and behaviour modification (chewing with contralateral side) might be instituted at first instance, with speech pathology review and surgical follow-up recommended in the medium to long term

Table continued on the next page

Table 1. Summary of common presenting complaints, features and initial management (cont'd)

Presenting complaint (differentials)	Frequency	Features	Red flags	Investigations/treatment/ escalation
Trismus	• Uncommon	• Usually associated with masseteric inflammation and typically mild and self-limiting	• Association with local or systemic features of infection • Association with neck swelling and reduced neck range of motion • Association with new neck mass/ lymphadenopathy	• Jaw-opening exercises • In the case of trismus associated with infection, US or cross-sectional imaging, commencement of oral antibiotics and urgent referral to the treating surgeon or local emergency department (depending on severity) is recommended • Trismus associated with a new neck mass/lymphadenopathy might be concerning for recurrence or new malignancy and should be urgently referred to the treating surgeon
Neck swelling excluding infection (haematoma, sialocoele, seroma)	• Uncommon – sialocoele (5%),[20] haematoma (3%)[18] • Rare – seroma[5]	• Slow-forming cystic collection of saliva at surgical site – sialocoele • Gradual to rapid collection of blood at surgical site with firmness to palpation and overlying discolouration – haematoma • Cystic collection of serous fluid at surgical site ± serous fluid leak – seroma	• Bleeding/haematoma, pus/abscess, rapid increase in size of neck swelling	• First aid measures (in active bleeding) • Full blood count, coagulation profile (in haematoma) • CRP (in infection) • US ± cross-sectional imaging • For haematoma or active bleeding, refer urgently to the local emergency department • The treating surgeon should be notified in all instances of postoperative neck swelling to expedite next surgical review
Wound dehiscence	• Uncommon (1–4%)[1,8]	• Separation of wound margins	• Association with infective signs and symptoms	• Occlusive dressing, urgent surgical referral
Flap necrosis	• Uncommon (2%)[18]	• Discolouration and devitalisation of the soft tissue flap used in wound closure	• High extent of necrosis • Association with infective signs and symptoms	• Occlusive dressing, urgent surgical referral
Wound site or systemic features of infection	• Uncommon (2%)[18]	• Local swelling, erythema, tenderness • Pus exudate • Palpable collection • Trismus, decreased neck range of motion • Fevers and other sequelae of systemic infection	• Presence of collection or systemic features of infection	• Send swab for microscopy/culture/ sensitivities (if applicable) • Commencement of empirical oral antibiotics • Consider cross-sectional imaging (CT) • For active infection beyond minor soft tissue infection, refer urgently to the local emergency department for consideration of admission for IV antibiotics and surgical input • Notify the treating surgeon
Cutaneous saliva leak (parotid fistula)	• Uncommon (5%)[18]	• Continuous saliva leak onto skin	• Association with infective signs and symptoms	• US, empirical oral antibiotic cover, surgical referral
Neck mass/ lymphadenopathy (recurrence, amputation neuroma)	• Uncommon (1–4%) for pleomorphic adenoma and typically occurs 7–10 years postoperatively[7] • Uncommon (up to 5%) when malignant disease included and typically from 5 months to 9 years (median 5 years)[9]	• Differs in benign vs malignant disease • New neck mass in operated field with or without associated lymphadenopathy • New neurological (motor or sensory) symptoms or signs consistent with nerve involvement • Constitutional symptoms such as fevers and night sweats	• Any concern for a new neck mass should be urgently investigated and followed up	• Investigation with US ± cross-sectional imaging • Tissue biopsy with fine needle aspiration or core biopsy where possible • Referral back to treating surgeon or nearest otolaryngology (ear, nose and throat) service

CRP, C-reactive protein; CT, computed tomography; GAN, great auricular nerve; IV, intravenous; US, ultrasound.

Table 2. Time to onset after surgery of common presenting complaints

Time to onset	Presenting complaint
<72 h	• Pain • Bleeding • Neck swelling • Facial muscle weakness/facial muscle asymmetry • Altered sensation • Trismus
Up to 2 weeks	• Pain • Bleeding • Neck swelling • Local or systemic features of infection • Wound dehiscence, flap necrosis • Altered sensation
>2 weeks	• Gustatory sweating (Frey syndrome) • Altered sensation • Pain on first bite (first bite syndrome) • Cutaneous saliva leak (parotid fistula) • Scarring • Asymmetry of facial contour • Neck mass/lymphadenopathy

The presentation of a serous fluid leak onto the skin that increases with meals might be a sign of a parotid fistula.[5] The added finding of a mobile cystic swelling that is slow growing in the absence of infective signs might be further consistent with sialocoele or seroma (less common) formation.[5] Evaluation with ultrasound (US) is initially useful. These complications are associated with wound healing issues and should be referred early to the treating surgeon for further investigation and management.[20]

Neck mass

The formation of a neuroma from the amputated free end of the GAN is a less frequent complication[5] and might first present as a painful, tender mass up to 10 years postoperatively.[21] The pain might also radiate to the face and neck, and treatment is usually by excision.[21,22] However, any new mass in the surgical field or neck postoperatively should be investigated as a potential recurrence of disease and referred appropriately until proven otherwise.[21,22]

Over the course of months, the development of a new mass in the surgical field with or without associated lymphadenopathy is concerning for recurrence and should be evaluated with US ± cross-sectional imaging and re-referred early to the treating surgeon for review.[9]

Wound complaints

A degree of scarring is to be expected in all patients postoperatively, with a smaller likelihood (4%) of hypertrophic and keloid scarring in predisposed individuals.[5,18]

Patients should be reassured at first instance that scarring usually fades over time, and this might be aided with conservative measures such as scar massage and vitamin E cream. Any requests for further intervention, such as steroid injection and/or scar revision,[5] should be referred back to the treating surgeon.

Flap necrosis characterised by discolouration and devitalisation of the soft tissue flap used in wound closure should be recognised early and referred to the local emergency department. Wound dehiscence should be covered with an occlusive dressing and referred to the treating team for early review.

Facial contour asymmetry on the operated side should be expected postoperatively due to loss of tissue mass.[18] This usually presents as a depression or hollowing of the face at the surgical site that matures over time, and reassurance should be given.[18]

Conclusion

The relative frequency of parotid surgery as a subset of head and neck surgery in Australia makes familiarity with its consequences and potential complications beneficial in general practice. As this article demonstrates, presentations post parotidectomy can be diverse and idiosyncratic. Common complaints relating to nerve function and the wound can often be conservatively managed and monitored in the primary care setting. Although less frequent, more acute presentations (eg those relating to haematoma, necrosis and infection) cannot be missed and warrant effective early management and escalation to the patient's surgeon or, in some instances, the emergency department. More generally, and especially where there is ambiguity or new concern regarding a patient's status, early involvement of the surgical treating team is always advisable.

Key points

- Benign and malignant parotid gland neoplasms often receive surgical intervention and represent a diverse group of pathology.
- Parotid surgery is associated with a variety of idiosyncratic postoperative presentations in the immediate and long-term postoperative periods, which might present a conundrum in primary care.
- Becoming familiar with the early recognition, differentiation and triage of common presenting complaints post parotid surgery will aid initial management and appropriate referral.
- Acute complications in the immediate postoperative period should be escalated early to the local emergency department with surgical cover.
- Unclear, persistent or progressive complaints should be referred to the treating (or local) surgeon or department.

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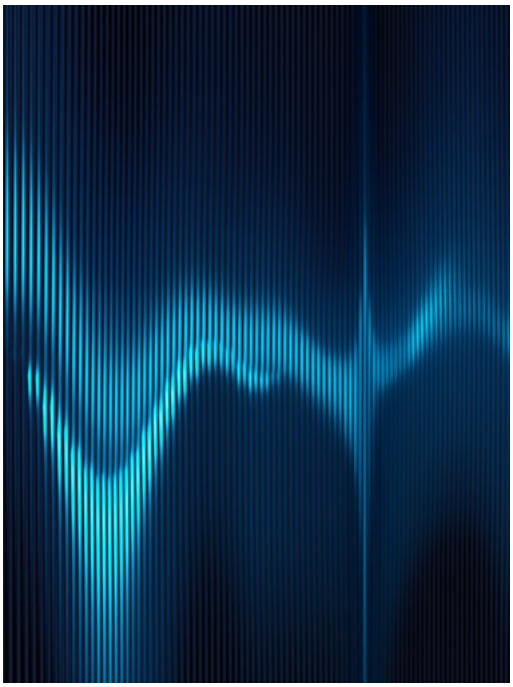
Management of sudden sensorineural hearing loss: A time-critical diagnosis



Jennifer Chen, Kristy Fraser-Kirk

Background
Sudden sensorineural hearing loss (SSNHL) is an otologic emergency requiring urgent medical attention and care.

Objective
This article, based on up-to-date evidence and clinical guidelines, aims to equip general practitioner (GP) specialists with a structured and practical approach to SSNHL management, emphasising the need for prompt evaluation and appropriate interventions. From initial evaluation to treatment strategies and follow-up, this guide offers a step-by-step framework to optimise patient care and improve outcomes in patients suffering SSNHL.

Discussion
Distinguishing SSNHL from conductive hearing loss (CHL), with early identification and prompt initiation of high-dose corticosteroid therapy, are key considerations for the management of SSNHL. Appropriate referrals when indicated for audiometric evaluation, emergency and/or otolaryngology services are also essential. Furthermore, this study aims to outline emerging therapies including intratympanic steroid administration, hyperbaric oxygen therapy and their potential roles in augmenting standard treatment approaches.

SUDDEN SENSORINEURAL HEARING LOSS (SSNHL) is an otologic emergency that demands prompt evaluation and intervention. This review article provides an extensive overview of the current state of SSNHL management, encompassing its epidemiology and aetiology, diagnosis and evolving treatment modalities.

Aim

This review aims to equip healthcare providers with a comprehensive understanding of SSNHL and its management strategies through collating up-to-date research and clinical guidelines. Furthermore, an evidence-based approach on the aetiology, diagnosis, investigations, treatment and prognosis is examined, along with Australian-specific healthcare considerations to guide the Australian general practitioner (GP) specialist in providing comprehensive care for the patient who presents with SSNHL.

Aetiology

SSNHL is a rapidly progressing hearing loss (defined as occurring within a 72-hour period), often unilateral in nature. The cause of SSNHL is idiopathic in over 70% cases.[1,2] Other causes can also be infectious or inflammatory (often viral), neoplastic, autoimmune, vascular, trauma or iatrogenic.[1]

Bilateral SSNHL is exceedingly rare; however, it can still occur. Examples include ototoxic medication (eg aminoglycosides or chemotherapy), syphilis or certain autoimmune or vascular conditions. SSNHL due to aminoglycoside ototoxicity might initially progress undetected as it typically affects the higher frequencies first before progressing to lower speech frequencies.[3,4] Patients with bilateral SSNHL are often older, have a positive antinuclear antibody titre and have a higher prevalence of vascular disease.[5]

Although the aetiology is important for prognosis and might change future management, the immediate management of SSNHL at the initial GP specialist appointment remains the same for all SSNHL cases.

The exact incidence of SSNHL is unknown, as many patients do not seek medical attention or the condition might spontaneously resolve. Within the literature, estimates of SSNL annual incidence ranges from five to 27 cases per 100,000.[6,7]

Since the COVID-19 pandemic, it was theorised that a viral pandemic might lead to an increased incidence of viral-induced SNHL; however, the literature on this relationship remains equivocal.[8]

Diagnosis

Diagnosis of SSNHL can be challenging and delay to diagnosis can result in poorer outcomes due to the time-critical relationship between onset of symptoms, initiating steroid therapy and prognosis.

SSNHL can often be masqueraded by other benign ear conditions, such as otitis media with effusion or cerumen impaction. Further, the prevalence of pre-existing hearing impairment in the general Australian population is high; 3.6 million people have some degree of hearing loss and more than one in three Australians have noise-related aural damage, which can often further confound the diagnosis.[9] In these cases, if the clinical history is not entirely clear, then comparison to previous recorded audiometry is of value to aid in diagnosis.

Table 1 outlines the key features in the history and physical examination in SSNHL. Box 1 details the key differentiating factors between conductive hearing loss (CHL) and SSNHL. Box 2 demonstrates how the authors conduct the tuning fork examination within clinic.

Audiogram is the gold standard and most definitive method in diagnosing SSNHL; however, if same day audiogram cannot be achieved, do not delay treatment while awaiting investigations, as delay to treatment results in poorer outcomes.[10] Figure 1 shows the audiogram of a patient with right SSNHL.

In cases where a tuning fork might not be available to the GP specialist, it has been shown that a smartphone application (The Real Razor™) might be a practical alternative in the primary care setting.[11] When placed on silent mode, the app causes the mobile to vibrate at a frequency of 163 Hz. The Weber test is then conducted with the corner of the vibrating smartphone; results from the study showed >97% agreement when compared to the standard tuning fork for the Weber test.[11]

Treatment

As per recently updated National Institute for Health and Care Excellence (NICE) Guidelines, an adult with sudden hearing loss within the last 30 days requires immediate referral (to be seen within 24 hours) to an ENT service or an emergency department.[12,13]

Oral high-dose systemic corticosteroids are the gold standard treatment for SSNHL, with initiation of therapy immediately at the time of suspected diagnosis essential. At our centre, a daily morning dose of prednisone 1 mg/kg (max 75 mg) for a minimum of five to seven days is the targeted treatment of choice. Further dosing and tapering of medication is generally clinician dependent. Early initiation of therapy improves prognosis, with the greatest advantages observed within the first two weeks of onset. Following this window, there is a significant decrease in the amplitude of hearing improvement.[14]

Box 1. Clinical pearl

- Often, otoscopic examination for sudden sensorineural hearing loss is normal, whereas acute hearing loss secondary to conductive causes more often has a 'positive' finding.
- Clinical examples of sudden CHL include: otitis externa (pain, swelling and purulent canal debris), otitis media (pain, red TM ± fevers), acute TM perforation from infection or barotrauma (perforated TM), otitis media with effusion (aural fullness or 'blocked ear', might have preceding coryzal symptoms).

CHL, conductive hearing loss; TM, tympanic membrane.

Contraindications to the corticosteroids, patient counselling and monitoring for side effects might be considered.

For SSNHL greater than 30 days ago, urgent referral to an ENT service is indicated to view candidacy for intra-tympanic

Box 2. Tuning fork examination

Tuning fork examination is key to differentiating CHL from SSNHL and should be performed in all patients presenting with hearing loss. The authors describe how this is typically conducted in an ENT specialist clinic:

1. Optimise environment
   a. Position/environment: ideally the patient should be sitting on a chair, with any head gear/glasses removed. Examination should be performed in a silent room.
   b. Equipment: a 512-Hz tuning fork.
2. Weber's test
   a. Strike the fork against knee/elbow or surface to create reverberation.
   b. Place the fork midline on the patient's forehead.
   c. Ask the patient to identify if the sound is louder in one ear or the same in both.
      i. In SNHL, the sound will be heard louder in the contralateral (unaffected) ear.
3. Rinne's test
   a. Introduce the test prior to performing it; explain to the patient that the fork will be held in two positions; behind the ear and in front of the ear. Ask the patient to identify in which position the sound is loudest.
   b. Strike the fork against the knee/elbow/surface to create reverberation.
   c. Place the fork directly on the mastoid process (tests bone conduction), then move the fork to approximately 1 cm from the ear (tests air conduction).
      i. In SNHL, this test will be 'normal' with air conduction and greater than bone conduction.

Clinical pearl: Take caution in interpreting Rinne's test in the patient with total SNHL (ie a 'dead' ear) as testing will yield a 'false-negative' result.

   a. Clinical example: in a patient with left total SNHL, on testing Rinne's on the left ear, bone conduction will be louder than air conduction, thereby producing a false-negative Rinne's result.

CHL, conductive hearing loss; ENT, ear, nose and throat; SNHL, sensorineural hearing loss; SSNHL, sudden sensorineural hearing loss.

Table 1. Key features in the history and examination for sudden sensorineural hearing loss

History	Examination
• Often unilateral hearing loss • Sudden onset • ± vertigo • ± preceding coryzal symptoms	• Overall benign or unremarkable otoscopic examination with absence of infection or pathology • Pinna/external ear; unremarkable • External auditory canal; clear or with benign cerumen • Tympanic membrane; clear A full cranial nerve examination should also be performed to exclude any concomitant or causative pathology.

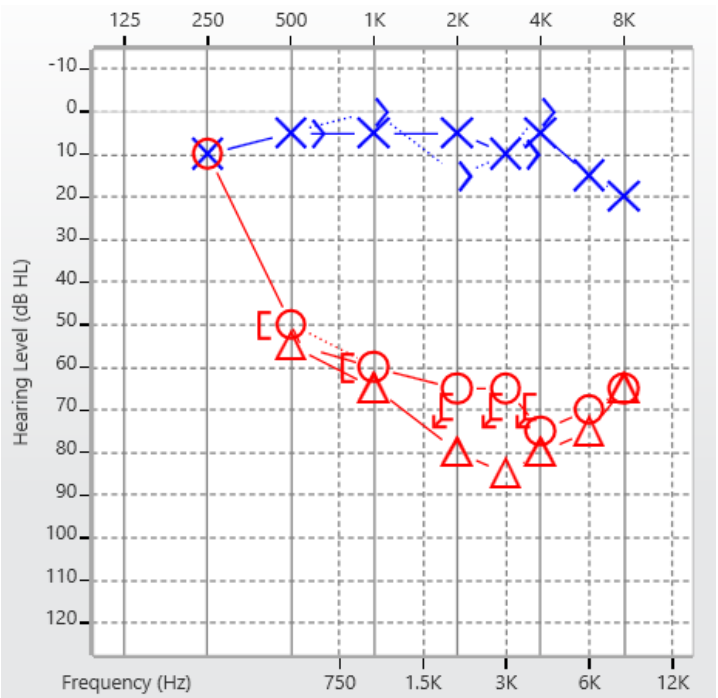
corticosteroid injection in which the steroid is directly infiltrated into the middle ear space with diffusion via the round window into the inner ear to reduce local inflammation.

A systematic review and meta-analysis demonstrated the role of intra-tympanic dexamethasone to be equivalent to oral steroid therapy as primary treatment but has greater improvement in hearing outcomes as salvage treatment, with a mean improvement of 13.3 decibels (dB).[15]

Overall, SSNHL greater than 30 days ago is a clinical 'grey window' within primary care. Apart from referral to an ENT service, there is no specific treatment algorithm to follow and cases would require a case-by-case analysis and discussion with each individual patient. Many clinicians continue to offer systemic steroid treatment for SSNHL lasting longer than 30 days but generally not past six weeks. We recommend a shared decision-making model, including discussions of risk, benefit and expected prognosis (which is poor).

Hyperbaric oxygen therapy (HBOT) is a useful adjunct for the treatment of SSNHL in adults and might have benefit when combined with steroid treatment in the acute setting or as salvage treatment within one month of onset.[16]

A shared decision-making model might be of benefit when considering HBOT due to social and financial considerations. Factors that might influence the patient's decision include accessibility to the service, travel and the time-intensive nature of treatment typically requiring 10–20 sessions within a few weeks. Further, if sought privately, HBOT can be costly if not covered by the patient's health fund. In Australia, there are nine publicly funded hyperbaric facilities with comprehensive cover; referral to these services to assess patient eligibility might be worthwhile.



Keys to Symbols	Right	Unspecified	Left
AC unmasked	O		X
AC masked	●		⧗
BC unmasked	<		>
BC masked	[		]
AC aided	H	△	V
AC sound field		□	

Figure 1. Audiogram in a patient presenting with sudden sensorineural hearing loss.

Investigations

Audiogram is the most definitive investigation to diagnose SSNHL. There are varying diagnostic criteria for SSNHL, with the most common being a sensorineural hearing loss (SNHL) of 30 dB or greater over three consecutive test frequencies over a 72-hour period.[2,16] SSNHL has also been described as a loss of 10–20 dB in two or three frequencies occurring within 12 hours.[17]

Magnetic resonance imaging (MRI) of the internal acoustic meatus (IAM) with contrast is the most sensitive and specific test in detecting retrocochlear pathology and can detect cerebellopontine angle tumours as small as 1 or 2 mm.[18] It is recommended that an MRI be performed within three months of onset in all patients with SSNHL.[16] Other pathologies that might be identified on MRI include intracranial lesions, such as multiple sclerosis or meningeal carcinomatosis.[19]

For patients in which MRI is contraindicated, computed tomography (CT) with contrast of the temporal bone in conjunction with an auditory brainstem response (ABR) on audiometry, can be performed.

The most recently updated guidelines and systematic review from the American Academy of Otolaryngology Head and Neck Surgery do *not* recommend obtaining routine laboratory tests in patients with idiopathic SSNHL.[16] However, a tailored clinical approach to the patient is still recommended, particularly if the clinician suspects an underlying aetiology such as vascular or autoimmune conditions. Ultimately, the management of SSNHL can be challenging, thus a treatment algorithm or 'roadmap' for the management of SSNHL (see Figure 2) has been created for use within the primary care setting.

Future considerations: Where to from here?

Overall, patients who have experienced only one episode of SSNHL, particularly when idiopathic, are unlikely to have another

episode. However, providing counselling on the management of future episodes and ensuring the patient understands the emergent nature of the condition can lead to better patient empowerment and improved health literacy. A repeat audiogram in 6–12 months to ensure hearing levels are stable might be useful.

If the patient experienced concomitant vertigo, this typically indicates a poorer prognosis in recovery of hearing loss. Meniere's disease might also be considered; a condition characterised by two episodes or more of vertigo with aural symptoms (hearing loss, aural fullness, tinnitus). The hearing loss is predominantly a low frequency SNHL, and tinnitus is characteristically 'roaring' in nature. The vertiginous episodes typically last from 20 minutes to 12 hours with the likelihood of diagnosis less likely if only lasting minutes or on the contrary for days.[20]

Indications for hearing rehabilitation devices are generally indicated, particularly if the hearing loss is moderate–severe or if it is affecting the patient's quality of life. It might also be useful to consider the eligibility criteria under the Australian Government Hearing Services Program for conventional hearing aids, as privately purchased hearing aids can be costly for the patient. If the patient is aged <26 years or >75 years, then they will be eligible for a government subsidy. Therefore, if patients are close to these age thresholds and remain unsure, appropriate counselling and education on this might encourage them to seek hearing aids while they are still eligible. The full eligibility criteria are available on the Australia Government Health website.[21] Referral to ENT services for discussion of hearing rehabilitation options might be indicated if the hearing loss is not managed by conventional hearing aids alone due to the severity and/or if the hearing loss is impacting the patient's quality of life.

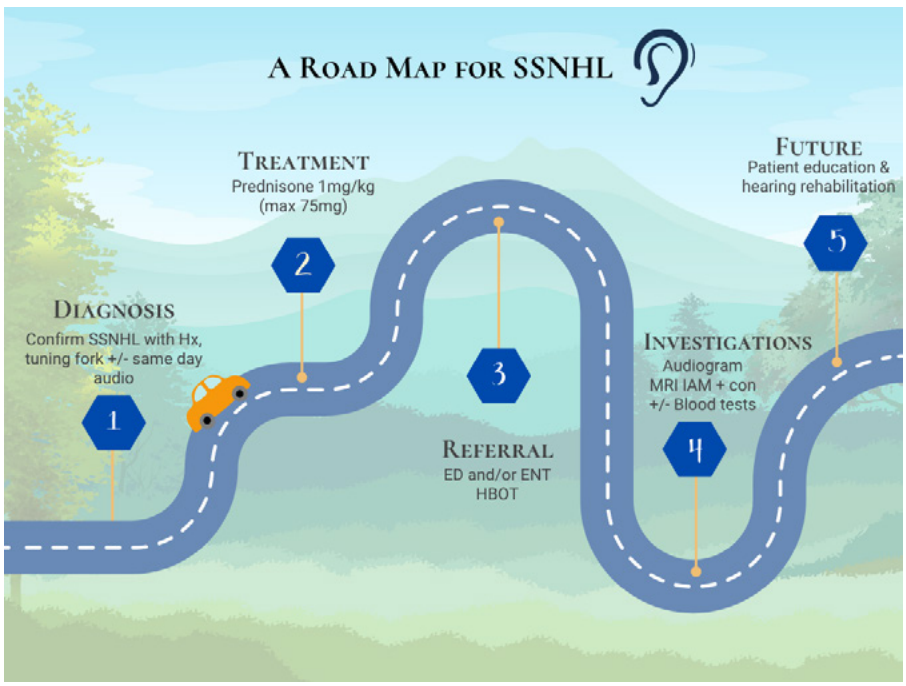


Figure 2. A road map for sudden sensorineural hearing loss (SSNHL) in the primary care setting. This figure demonstrates a practical algorithm to guide general practitioner specialists in the management of SSNHL.

ED, emergency department; ENT, ear, nose and throat; HBOT, hyperbaric oxygen therapy; Hx, history; IAM, internal acoustic meatus; MRI, magnetic resonance imaging.

Conclusion

In conclusion, effective SSNHL management in primary care necessitates a multidisciplinary approach, with GP specialists often serving as the initial point of contact. Improved awareness, timely diagnosis and evidence-based therapeutic interventions can significantly impact patient outcomes in this challenging clinical scenario. Ultimately, primary care providers play a pivotal role in optimising care for patients presenting with SSNHL, contributing to improved hearing preservation and quality of life.

Key points

- Sudden sensorineural hearing loss is an otologic emergency.
- Prompt diagnosis and initiation of treatment with high-dose corticosteroids improves patient hearing outcomes.
- Do not delay treatment while awaiting investigations (ie audiogram).
- Prompt referral through to an ENT service and/or an emergency department is recommended.
- Consider adjuncts to therapy including hyperbaric oxygen therapy, audiovestibular services and/or intra-tympanic dexamethasone.

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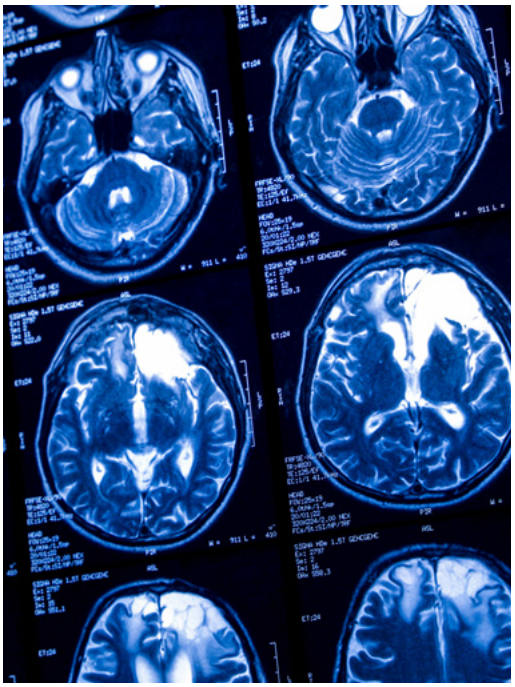

Anticoagulation in the context of post-intracerebral haemorrhage: A narrative review

Tuan Tran, Julia Tsolakis, Fintan O'Rourke

Background
Recommencement of oral anticoagulation (OAC) for patients post-intracerebral haemorrhage (ICH) remains a challenging decision for clinicians. High-quality evidence to assist with this decision is lacking and current guidelines primarily focus on balancing thromboembolic and bleeding risk.

Objective
This study evaluated the literature and current guidelines for recommencement of OAC in patients who have experienced an incident ICH.

Discussion
Patients with recurrent ICH while on anticoagulation therapy have associated poor outcomes. However, predicting which patients will experience recurrent ICH with OAC resumption remains challenging, and failure to resume OAC carries risks of thromboembolic events. Current data suggest that it is reasonable to resume OAC in many patients post-ICH, depending on careful consideration of individual risk factors for haemorrhagic and thromboembolic events. The application of existing risk stratification tools for thromboembolism and haemorrhage, and radiological biomarkers such as cerebral microbleeds, might also assist in decision making.

THE RISK OF STROKE associated with atrial fibrillation (AF) is 1.5% at age 50–59 years, increasing to 23.5% by age 80–89 years.[1] Oral anticoagulation (OAC), with warfarin or direct oral anticoagulants (DOACs), is highly effective at reducing risk of embolic stroke in the presence of AF by approximately two-thirds, and this is supported by multiple randomised controlled trials and Class I guidelines.[2,3]

The incidence of intracerebral haemorrhage (ICH) and other haemorrhages while on OAC is comparatively low (0.8% for warfarin, and <0.3% for DOACs),[4] but is important to consider when commencing anticoagulation medication for the first time.

Patients with ICH while on OAC have high morbidity and mortality, with adverse effects usually more severe compared to patients with spontaneous ICH who are not on OAC.[3] The decision when or if to resume OAC after ICH is challenging for clinicians and patients because of a lack of high-quality evidence. Carefully balancing risks of thromboembolism and bleeding, especially recurrent ICH, is the primary concern.[2]

Background

Warfarin has undoubted efficacy in stroke prevention in patients with AF, with evidence spanning >30 years.[5] Meta-analysis of six placebo-controlled studies (N=2900) demonstrated a significantly reduced risk of stroke for patients by 64% (95% CI: 49–74%) versus placebo in patients with AF.[6] However, the rate of ICH averaged 0.3% per year versus 0.1% for placebo. The relative risk for major extracranial haemorrhage was 2.4 (95% CI: 1.2–4.6; absolute risk reduction [ARR] 0.3% per year).

In recent years, DOACs have superseded use of warfarin in many patients, with a 2014 meta-analysis of trials involving four DOACs demonstrating superiority compared to warfarin, with reductions of 19% for stroke and systemic embolism (risk reduction [RR] 0.81, 95% CI: 0.73–0.91; P<0.0001), 10% for all-cause mortality (RR 0.90, 95% CI: 0.85–0.95; P=0.0003) and 52% for ICH (RR 0.48, 95% CI: 0.39–0.59; P<0.0001). Only gastrointestinal bleeds were higher in patients treated with DOACs compared with warfarin (RR 1.25, 95% CI: 1.01–1.55; P=0.04).[7]

Apixaban demonstrated superiority to warfarin in preventing stroke or systemic embolism, causing less bleeding, with lower mortality, in patients with AF.[4] The rates of primary outcome (ischaemic or haemorrhagic stroke or systemic embolism) were 1.27% per year and 1.60% per year in apixaban and warfarin groups, respectively (HR for apixaban: 0.79 [95% CI: 0.66–0.95]).

Rivaroxaban demonstrated non-inferiority to warfarin for prevention of stroke or systemic embolism,[8] with rates of primary outcome being 1.7% and 2.2% per year in the rivaroxaban and warfarin groups, respectively. Bleeding rate was 3.4% for the

warfarin group and 3.6% for the rivaroxaban group. The ICH rate was 0.7% and 0.5% for the warfarin and rivaroxaban groups, respectively.

In the randomised evaluation of long-term anticoagulation therapy (RE-LY) study, dabigatran (110 mg twice daily [BD] dose) reduced rates of stroke or systemic embolism (1.53% per year) similarly to warfarin (1.69% per year, RR with dabigatran: 0.91, 95% CI: 0.74–1.11], $P<0.001$ for non-inferiority), with lower rates of major haemorrhage.[9] A higher dose of dabigatran (150 mg BD) further lowered rates of stroke and systemic embolism (1.11% per year; RR 0.66, 95% CI: 0.53–0.82, $P<0.001$ for superiority), but there were similar rates of major haemorrhage.[9]

Current guidelines and evidence

Current guidelines for starting or restarting OAC in the context of recent ischaemic stroke, but not ICH, are largely based on expert consensus[10] and the '1–3–6–12' rule recommended by the European Society of Cardiology (ESC) in 2013, with similar variations used in American and Australian Stroke Foundation (SF) guidelines. These do not specifically mention infarct size, tending to use the National Institutes of Health Stroke Scale (NIHSS) as a proxy for severity and bleeding risk, along with subjective assessment by the physician.[10] SF guidelines recommend anticoagulation one day after transient ischaemic attack (TIA), three days after a small stroke, five to seven days after a moderate stroke and 10–14 days after a severe/large stroke.

New trial evidence in 2023 has supported this approach, or an even earlier re-commencement of anticoagulation medication after ischaemic stroke. The ELAN trial randomly assigned participants to receive early anticoagulation (within 48 hours of a minor or moderate stroke, day six or seven after a major stroke) or later anticoagulation (day three or four after a minor stroke, day six or seven after a moderate stroke and day 12–14 after a major stroke). Recurrent ischaemic stroke occurred in 1.4% of the early-treatment group and in 2.5% of the later-treatment group (OR 0.57; 95% CI: 0.29–1.07) by 30 days; and in 1.9% and 3.1%, respectively, by 90 days (OR 0.60; 95% CI: 0.33–1.06). Symptomatic ICH occurred in only two participants (0.2%) in both groups by 30 days.[11]

Guidelines and high-quality evidence for restarting OAC after ICH are more lacking, and again, mostly based on consensus. The optimal timing for resumption of OAC after ICH is uncertain without randomised trial data to guide the decision. Although DOACs have a lower associated risk of ICH than warfarin, their usefulness as alternatives after ICH is undetermined.[12]

Clinical features associated with recurrent ICH include Asian ethnicity, ICH history, cerebral microbleeds, amyloid angiopathy, arteriovenous malformation, cerebral aneurysm and lacunar infarcts.[3]

Observational studies of anticoagulant-related ICH have found low rates of cardioembolic events when patients are not receiving anticoagulation, and low rates of recurrent ICH when anticoagulation therapy resumed, but results are limited by small sample sizes and short durations of follow-up.[13] Among 141 patients who discontinued warfarin, only three suffered ischaemic events within 30 days compared to none who restarted. In the 35 patients who restarted OAC during hospitilisation, with a median of 10 days (range 0–30) off OAC, there was no recurrence of bleeding. This study concluded that brief (one- to two-week) discontinuation of OAC was relatively safe. It also demonstrated that ICH occurring with anticoagulation therapy resulted in a higher mortality rate of 43%.

A retrospective, multicentre study of 2869 patients with ICH, of which 234 were warfarin-related and with 59 resuming warfarin, found recurrent ICH risk was highest with early OAC resumption in the first 35 days, exceeding the risk of thromboembolism compared to when resumption of warfarin was delayed.[14] Recurrent ICH risk was 0.75% per day within the first 35 days if anticoagulation was restarted, compared to 0.18% if not (HR 4.13). The observed rate of ischaemic stroke was low in the first 77 days whether OAC was restarted (0%) or not (0.068% per day; HR 0). A time period of 10–30 weeks was recommended as optimal for OAC resumption, when the combined risk of recurrent ICH or ischaemic stroke approached a nadir.

A 2018 meta-analysis of 12 observational studies with 3431 patients showed that restarting anticoagulation after ICH significantly reduced thromboembolic events (RR 0.31, 95% CI: 0.23–0.42, $P<0.001$) with no increase in mortality or recurrent ICH.[15]

A 2023 Cochrane review concluded that the benefit or harms associated with antithrombotic treatment post ICH are uncertain.[16] Long-term OAC for AF post ICH was found to probably reduce the risk of major adverse cardiovascular events, but also likely to increase the risk of ICH, resulting in little or no difference in the death rate and minimal effect on independent function. It suggested further randomised controlled trials be conducted to resolve uncertainties, but made no specific recommendations for clinical practice.

Currently, Australian and New Zealand Clinical Guidelines for Stroke Management make no recommendation on commencement or recommencement of anticoagulation medication post ICH.[17] The American Heart Association and American Stroke Association guidelines recommend starting oral anticoagulation medication four days after ischaemic stroke and 14 days after ischaemic stroke with haemorrhagic transformation.[18]

European Society of Cardiology (ESC) 2020 guidelines on recommencement of OAC after ICH, stated as based on observational data with RCTs ongoing, offer more practical advice.[19] Consideration of non-modifiable risk factors such as age, male sex, Asian ethnicity, amyloid angiopathy and cerebral microbleeds; and optimising modifiable risk factors such as hypertension, smoking, alcohol consumption and concomitant anti-platelet medications is recommended to help weigh risks and benefits.[19] Although not specifying a treatment preference, the ESC guidelines offers three options: (i) recommencing anticoagulation two to four weeks after ICH; (ii) left atrial appendage closure; or (iii) no stroke prevention therapy.[19]

Although not the focus of this paper, re-commencing antiplatelet medication after ICH also appears to be safe, and perhaps even beneficial, with restart or stop anti-thrombotics randomized trial (RESTART) data demonstrating non-significant reductions in both ICH (8.2% versus 9.3%) and major vascular

events (26.8% vs 32.5%). The latter finding in particular is being further studied in the current anti-platelet secondary prevention international randomised study after intracerebral haemorrhage (ASPIRING) trial.[20]

Existing risk assessment tools for thromboembolism recurrence

Although several risk assessment tools have been developed to evaluate risk of thromboembolism, their application to post-ICH settings is more limited.

CHA_2DS_2-VASc score

The congestive heart failure, hypertension, age, diabetes mellitus, prior stroke or TIA or thromboembolism, vascular disease, age, sex category (CHA_2DS_2-VASc) thromboembolism risk stratification score is validated in patients with AF for stroke, transient ischaemic attack and systemic embolism.[21] The apixaban versus no anticoagulation after anticoagulation-associated intracerebral haemorrhage in patients with atrial fibrillation (APACHE-AF) trial evaluated the CHA_2DS_2-VASc score in 101 AF patients with history of ICH and CHA_2DS_2-VASc of at least two, who survived ICH while on OAC.[22] Patients were randomised to resume or avoid anticoagulation, and followed for a median of 1.9 years, with the primary outcome of non-fatal stroke or vascular death. Overall, four of 50 (8%) patients resuming anticoagulation medication, compared to one of 51 (2%) avoiding medication, sustained ICH (adjusted HR 4.08 (0.45–36.91); P=0.21). There was no difference in the incidence of ischaemic stroke (12% in each group) or major vascular events including death (26% resume vs 25% avoid) between the two groups.

A retrospective cohort study suggested that resumption of anticoagulation medication post-ICH, with a strong indication for anticoagulation based on the CHA_2DS_2-VASc score, reduces risk of ischaemic stroke without increasing recurrent ICH.[23] Most participants had a CHA_2DS_2-VASc score above four. Patients either recommenced or avoided anticoagulation for a median follow-up period of 0.7 and 0.5 years, respectively. Risk of ischaemic stroke was 3.5% for patients who resumed treatment, compared to 4.9% who avoided treatment (adjusted HR 0.61, [95% CI: 0.42–0.89]). Recurrent ICH was similar, with 1.4% of patients resuming treatment and 1.6% avoiding treatment (adjusted HR 1.15 [95% CI: 0.66–2.02]), with a similar risk of major bleeding and all-cause mortality.

Existing risk assessment tools for recurrent intracerebral haemorrhage

HAS-BLED

The hypertension, abnormal renal/liver function, stroke, bleeding history or predisposition, labile INR, elderly (age >65 years), drugs/alcohol (HAS-BLED) score is the only tool validated for predicting recurrent ICH following initial spontaneous ICH.[3] The National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand AF guidelines suggest that HAS-BLED might be useful in detecting patients at higher risk of bleeding.[21] Chan et al sought to evaluate HAS-BLED as a prognostic tool for recurrent ICH in a cohort of 434 patients who had initial spontaneous ICH and were not subsequently prescribed antiplatelet medication or OAC. Risk of ICH recurrence increased with HAS-BLED score; a score of one corresponded to a risk of recurrent ICH of 1.37 per 100 patient-years, and a score of three corresponded to a risk of 3.39 per 100 patient-years.[24]

Application of biomarkers to improve existing risk assessment tools

Cerebral microbleeds and intracranial haemorrhage risk in patients anticoagulated for AF after acute ischaemic stroke or transient ischaemic attack (CROMIS-2), an observational cohort study, sought to determine whether cerebral microbleeds (CMB), as a magnetic resonance imaging (MRI) neuroimaging biomarker, could improve predictive ability of clinical risk scores like HAS-BLED for ICH.[25] In total, 1490 participants with AF and recent acute ischaemic stroke or TIA commenced on either warfarin or DOAC, were followed for 24 months with a primary outcome of symptomatic ICH. CMB presence was an independent risk factor for ICH. Compared with HAS-BLED alone (C-index 0.41, 95% CI: 0.29–0.53), models including CMB and HAS-BLED (C-index 0.66, 95% CI: 0.63–0.80) and CMB, diabetes, anticoagulant type and HAS-BLED (C-index 0.74, 95%CI: 0.60–0.88) predicted symptomatic ICH significantly better. However, this clinical and neuroimaging combination has not yet been validated in patients who have survived a previous ICH.

The presence of cerebral amyloid angiopathy, a predictor of ICH and therefore conferring a higher risk of ICH if anticoagulation recommenced, might be diagnosed on MRI by the presence of CMB and cortical superficial siderosis (cSS). CT scan biomarkers are less useful as they might identify the presence of cerebral amyloid angiopathy, but not reliably exclude it.[26]

Conclusion

ICH occurring while a patient is taking anticoagulation medication can result in high morbidity and mortality if it occurs. Deciding if and when to restart OAC in patients post-ICH remains challenging. Most studies, largely observational, demonstrate low rates of rebleeding and ischaemic events, with OAC recommencement recommendations varying from two to four weeks or after 10 weeks. Awareness, and modification if possible, of existing risk factors might mitigate the risk of recurrent bleeding. And use of individualised risk scores such as CHA_2DS_2-VASc and HASBLED, in conjunction with neuro-imaging biomarkers such as cerebral microbleeds, can assist in advising patients of relative risks when making an informed treatment decision.

Key points

- Spontaneous intracerebral bleeding while patients are being treated with anticoagulants carries a high mortality.
- Warfarin and DOAC significantly reduce risk of ischaemic stroke by 64%.
- Incidence of intracerebral haemorrhage while on DOAC varies between 0.5% and 1.6%.
- Most guidelines recommend delayed restarting of anticoagulant treatment after ICH, but recommendations differ on timing.

- Informed decision making about anticoagulant commencement and recommencement requires awareness of potential risks and benefits.

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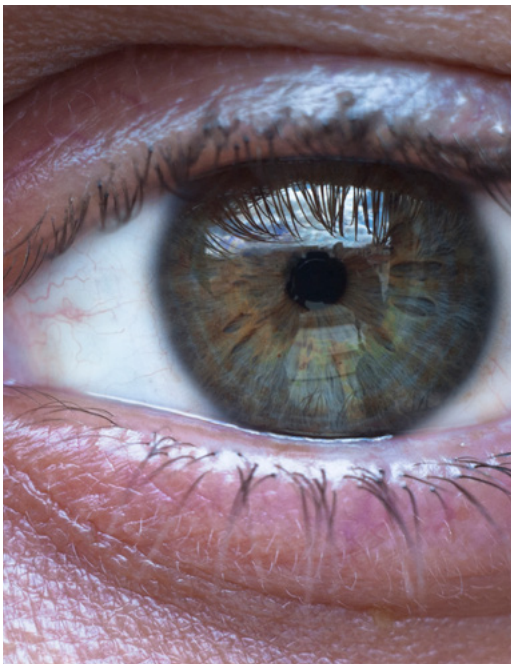
Approach to the watery eye



Raymond Li, Ye Li, Ben McArdle

Background
Epiphora, or the watery eye, is a common presenting complaint seen by general practitioners and can have significant quality-of-life implications.

Objective
This article aims to review epiphora, its many causes and a systematic approach to its assessment, management and escalation in the primary care setting.

Discussion
Many causes of epiphora can be managed in the primary care setting. A clear and structured approach to work-up is essential in narrowing down the broad range of differential diagnoses, and might help clinicians recognise when involvement of the appropriate specialist service is required.

A WATERY EYE is a common presentation that might perplex the primary care physician with a wide range of differential diagnoses and varying acuity. Epiphora refers to eye watering that involves a distinct overflowing of tears down the face, and typically points to lacrimal outflow obstruction.

A distinction is made between true epiphora and the watery eye that instead presents with tear welling; the latter being more suggestive of reflex tear production following ocular surface stimulation.[1] Excess tear production is usually reactive and, rarely, can occur as a result of primary hypersecretion. Common causes of reflexive lacrimation include dry eye, blepharitis, foreign bodies and eyelid malposition.

Impaired drainage can result in tear pooling, and might result from lid malposition, as seen in ectropion and entropion, or be due to blockage anywhere along the nasolacrimal duct from puncta to the intranasal opening.[2]

The watery eye has vast differential diagnoses, and it is important to consider the diversity of causes across different patient populations. Paediatric glaucoma, for example, is important to consider given its significant treatment implications for the patient if left unmanaged. Common causes of epiphora and the watery eye are outlined in Table 1.[3]

Aim

This article aims to review potential causes of the watery eye, discuss its work-up with helpful adjuncts, and provide a framework for its systematic assessment and management in the primary care setting.

Anatomy and drainage

An understanding of the lacrimal apparatus and tear drainage system might help clarify how epiphora arises (Figure 1). Tears are primarily produced by the lacrimal gland, which resides in the superolateral aspect of the globe.[4] Tears are produced as a combination of a baseline rate of secretion as well as a reflex following stimulation of the optic nerve (light reflex) or trigeminal nerve (touch reflex).[5] After coating the ocular surface, contraction of the orbicularis oculi muscle assists with fluid drainage through the upper and lower puncta of the eyelids and into the upper and lower canaliculi.[4] The upper and lower canaliculi then join to form a common canaliculus, which, in turn, drains into the lacrimal sac.[4] The lacrimal sac lies lateral to the nasal cavity and is connected by the nasolacrimal duct to the nose through Hasner's valve.[4] Obstruction at any point along this pathway can impair drainage and lead to a watery eye.

Assessment

History

A systematic history is important in the evaluation of the watery eye, and general questions regarding onset, duration, laterality of disease and pattern (constant or intermittent) should be noted.

Age at presentation is useful when considering likely causes. Congenital

nasolacrimal duct obstruction is the most common cause of eye watering in patients aged less than four years, whereas issues such as lid malposition and dry eye disease might be considered in an older cohort.

A distinction might be made between acute or chronic presentations. Eye watering that has persisted over three months is considered chronic and usually implies the presence of lacrimal outflow obstruction in an otherwise normal examination.

Ocular symptoms should be explored, and patients should be asked about any visual changes including diplopia and reduced visual acuity.[1] The presence of crusting or any discharge should be noted. Tear colour might be altered in some patients, in addition to the presence of any blood or purulent discharge. A history of ocular trauma or previous ocular/periocular surgery should also be enquired.[1,6]

A clear sinonasal history should also be taken for patients with undifferentiated epiphora, including rhinorrhoea, nasal obstruction or anosmia. Laterality is important when considered in conjunction with patients' ocular symptoms, and unilateral epistaxis should be explored to rule out a potential sinonasal mass.[7] A history of any previous sinonasal surgery and trauma is also essential, given the risk of nasolacrimal obstruction.[1]

Table 1. Causes of epiphora

General	Causes
Reflex tear production	• Eyelash malposition – trichiasis, distichiasis • Blepharitis/meibomian gland dysfunction • Conjunctivitis – allergic, viral, bacterial • Foreign body • Dry eye • Keratitis • Uveitis • Angle-closure glaucoma
Reduced tear outflow	Lid malposition • Entropion (in-turned eyelid) – Involutional – Cicatricial – Spastic – Congenital • Ectropion (out-turned eyelid) – Involutional – Cicatricial – Mechanical – Paralytic – Congenital (rare) • Floppy eyelid syndrome Punctal stenosis Nasolacrimal duct obstruction • Iatrogenic – rhinoplasty, sinus surgery • Traumatic – facial fractures, canalicular involving lid lacerations • Sinusitis • Systemic inflammatory conditions • Neoplastic
Paediatric specific	• Ophthalmia neonatorum (neonatal conjunctivitis) • Congenital glaucoma • Punctal atresia • Congenital nasolacrimal duct obstruction

Examination

A well-rounded nasolacrimal examination can be performed without a slit lamp and should commence with a thorough general inspection of the face. Obvious facial asymmetry or palsy should be noted, in addition to the presence of eyelid malposition (ectropion or entropion).[8] The presence of any ingrown lashes, eyelid masses, poor lid closure or any other periorbital anomaly should be considered.

An eye examination should then follow, starting with visual acuity and an assessment of extraocular movements. Lid position should be assessed for ectropion causing reduced tear drainage or entropion causing ocular surface disease and reflex tearing.[1] Access to slit-lamp examination can be limited in the general practice setting, but use of a direct ophthalmoscope with a blue filter can be useful in identifying obvious corneal pathology such as keratitis, foreign bodies and dry eye. Clinics equipped with a tonometer might consider measuring intraocular pressure, which, when elevated, might suggest the presence of intraocular inflammation or acute angle closure glaucoma as a cause of reflex tearing.

Fluorescein dye is reasonably accessible and can offer helpful information regarding tear drainage and corneal abnormalities. A dye disappearance test can assist with visualisation of tear drainage and the residual tear lake.[1,9] Fluorescein is instilled to the inner aspect of the lower lid, and the eye is reinspected after five minutes to assess for any excessive dye in the tear film. This may be assessed with a blue-filter light, although any bright light source should adequately highlight residual dye. The presence of residual fluorescein suggests failure of appropriate drainage due to nasolacrimal obstruction, and is thus considered a positive test. It should be noted that patients with blepharospasm or strong blinking might disrupt the tear film, and thus prevent accurate interpretation.[1,9]

A sinonasal examination should be considered in patients with history concerning

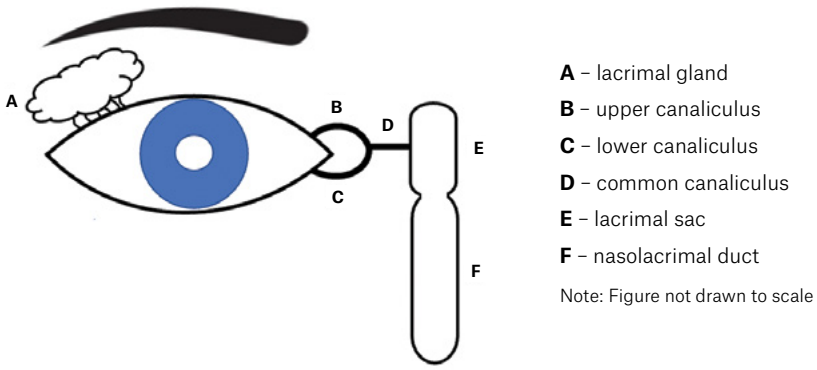


Figure 1. Anatomy and drainage pathway of the lacrimal system.

for nasal causes of epiphora.[10] Inspection of the nose should be performed to describe any obvious nasal bone or septal deformity and is helpful in the setting of recent trauma, fracture or surgery. Anterior rhinoscopy should follow and can be performed with an otoscope to further assess for septal deviation, turbinate hypertrophy, nasal polyps or any other obvious mass lesion within the nasal cavity.[1] Flexible nasoendoscopy is a helpful adjunct to examination, although the patient might require referral to an ear, nose and throat (ENT) specialist service.

Due to variable access to specialised equipment, particularly in rural and remote settings, it is understandable that some of the aforementioned ancillary investigations might not be possible to perform in a primary care setting. In such cases, liaison with the local optometry, ophthalmology or ENT service might be advised.

Investigations

Imaging is not routine in the primary care work-up of all epiphora and is preferably organised on the advice of a specialist ophthalmologist.[1] Computed tomography (CT) of the orbits might help assess suspected traumatic, mechanical, sinonasal or neoplastic causes of nasolacrimal obstruction.[11] Other adjuncts such as magnetic resonance (MR) imaging and dacrocystography have limited application in the primary care setting, and the patient should be referred to an ophthalmologist for consideration.[1,12,13]

Blood tests are not routinely organised and also have a limited role in the primary work-up of the watery eye. Primary autoimmune or inflammatory causes of epiphora are rare, and inflammatory markers should only be considered at the advice of an ophthalmologist, or if there is an otherwise strong suspicion of an underlying systemic inflammatory condition.[1,14]

Conjunctival swabs should be considered if there is suspicion of conjunctivitis; however, this typically presents with acute ocular discharge rather than true eye watering.[15] If taken, swabs should at least include herpes simplex, varicella–zoster virus and adenovirus, whereas swabs for *Chlamydia trachomatis* and *Neisseria gonorrhoeae* should also be performed if there is significant concern for a sexually transmitted infection, with urgent referral to an ophthalmology service.

Management

First-line management

Although definitive management for epiphora can only be instituted once its underlying aetiology is confirmed, some treatment can be initiated in the primary care setting in the interim.

If the suspected cause is reactive secondary to ocular surface disease, such as dry eye disease, treatment can be initiated in a general practice setting but should be referred to an optometrist for ongoing management due to complexity of disease.[16,17] Preservative-free lubricants four-times daily can be initiated with or without chloramphenicol ointment depending on severity.

Warm compress, gentle eyelid massage and lid hygiene should be suggested for patients with suspected blepharitis or meibomian gland dysfunction. A cotton bud or pad dipped in a dilute solution of baby shampoo might be utilised to gently cleanse the lid and lash line twice a day.

Patients with suspected rhinosinusitis or allergy should be commenced on intranasal topical corticosteroids, either in conjunction with an antihistamine orally or as a combined spray.[1]

Escalation to a specialist otolaryngologist should be considered in patients with obvious sinonasal symptoms, or by the involved ophthalmologist.

Paediatric epiphora

The management of paediatric epiphora differs and should always be conducted alongside consultation with the ophthalmology service. Due to sight and life-threatening implications of ophthalmia neonatorum (neonatal conjunctivitis) secondary to chlamydia or gonorrhoea,[15] neonates aged under one month should be discussed with an ophthalmology service to arrange urgent review. Similarly, children with suspected congenital glaucoma who present with signs of buphthalmos (a bulging eye) or photophobia should be urgently discussed with an ophthalmology service.[17] Children with nasolacrimal duct obstruction without red flags, such as those outlined above, can be managed with lacrimal massage and lid cares until non-urgent review with an optometrist or ophthalmologist can be arranged.[18] The vast majority of infants who present with congenital nasolacrimal duct obstruction will have spontaneous resolution of their symptoms.

Surgical management

Surgical management might be considered following discussion with a specialist ophthalmologist or ENT surgeon. Type of intervention varies depending on the underlying cause of epiphora. Examples of surgical procedures that might be considered include ectropion or entropion repair, and dacryocystorhinostomy (DCR).[1,19] Patients with eyelid malposition, punctal ectropion and eyelid laxity should be referred to the oculoplastics service for consideration of surgical correction. DCR involves the formation of a fistula from the lacrimal sac to the nasal mucosa and can either be performed

via an external approach or endoscopically in conjunction with an ENT surgeon.[1,19,20]

Common causes

Reflex lacrimation

Reflexive lacrimation can occur as a result of a multitude of stimuli, including but not limited to: dry eye disease, blepharitis, foreign bodies and eyelid malposition. Onset is typically seen in an older demographic of patients, with management typically involving a trial of lubricants and correction of the underlying irritative stimuli.

Nasolacrimal duct obstruction

Nasolacrimal duct obstruction (NLDO) should cause a true tearing epiphora that lasts over at least three months. Patients' symptoms can be either uni- or bilateral, and will usually present with an otherwise normal examination. Intervention for NLDO, on failure of first-line management with lubricants and warm compress, might require surgical management. Surgery would aim to correct the underlying cause of obstruction and maintain duct patency, to prevent secondary complications including mucocoeles and dacryocysitis. Patients should be referred if their symptoms significantly impact their day-to-day activities.

Facial palsy

Facial nerve palsy might cause both true epiphora and reflex lacrimation through respective lacrimal pump failure and subsequent drying of the ocular surface. Management normally involves at least lubricating the eye, while trying to identify and reverse any underlying cause of facial nerve dysfunction. Permanent paralysis might require more definitive surgical correction and protection of the ocular surface, which can be achieved through surgical means of tarsorrhaphy, gold weight insertion or other forms of surgical facial re-animation.[8]

Eyelid malposition

Eyelid malposition can lead to reflex lacrimation from both ocular surface drying and corneal injury. This can normally be addressed with lubrication in the first instance. Forms of malposition, including entropion, ectropion, excessive eye laxity or Floppy eyelid syndrome, do respond well to surgical correction and should be considered for referral to an oculoplastics service.

Conclusion

The watery eye is a common presenting complaint for primary care practitioners. A good understanding of the presenting symptoms and signs of epiphora is essential in differentiating between its various causes, and will help guide clinicians in its appropriate investigation, management and escalation in the general practice setting. Early involvement of an optometrist might assist in the triage and delivery of multidisciplinary care for cases with preserved vision. Significant loss of visual acuity warrants direct consultation with an ophthalmology service.

Key points

- The differential diagnoses for epiphora is broad, and a systematic history and examination is crucial in its assessment.
- Both ocular and sinonasal causes should be considered in the patient with undifferentiated epiphora.
- A fluorescein-assisted eye exam can be a simple but helpful ophthalmoscopic adjunct.
- Most causes of paediatric nasolacrimal duct obstruction can be managed with lacrimal massage and lid cares in the absence of any red flags signs or symptoms.
- A multidisciplinary team for the patient with epiphora should ideally involve the primary care clinician, optometrist, ophthalmologist and an otolaryngologist.

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Effects of seasonal, geographical and demographic factors on otitis externa microbiota in Queensland, Australia

Akila Wijesekera, Amanda Chiam Xu Wen, Abigail Walker, Cervin Anders

Background and objective
Otitis externa (OE) is a very common disease in Australia. It is associated with swimming and exposure to water. Typically, treatment consists of aural toileting and the use of topical antimicrobial drops. Antimicrobial treatment is empiric, and most Australian guidelines advise the use of dexamethasone/framycetin/gramicidin as first-line therapy. This study aimed to identify the most prevalent pathogens implicated in OE in Queensland, Australia, and determine if there was any variability with the season, proximity to a coastline, age, gender and First Nations status.

Methods
The primary pathogen cultured, the specimen type, the date of collection and the patient demographics were retrieved from microbiology swabs sent from hospitals to Pathology Queensland. Multivariate analysis was performed on the swabs.

Results
Pseudomonas aeruginosa was the most prevalent pathogen cultured in the external ear in Queensland, at 37.9%. In inland regions, *Staphylococcus aureus* was the most prevalent organism. Children were three-fold less likely to have OE resulting from a fungal pathogen.

Discussion
The use of targeted antimicrobials against *Pseudomonas aeruginosa* in coastal regions during summer is sensible. Due to the low burden of fungal disease in children, there should be a high threshold for the commencement of antifungal ear drops.

OTITIS EXTERNA (OE) is a very common presenting complaint to both the general practitioner (GP) and the ear, nose and throat (ENT) specialist, with a lifetime risk of approximately 10%.[1] OE is colloquially referred to as 'swimmer's ear' as it is five-fold more prevalent in swimmers.[1] Factors such as a temperate environment, humid climate, canal trauma, narrow external auditory canals (EACs), EAC obstruction, radio/chemotherapy and immunosuppression predispose an individual to OE.[2]

The year-round temperate and humid climate of Queensland, Australia, makes it perfectly suited for OE.[3,4] In a survey conducted with 34 Queensland GPs, over 94% reported seeing OE at least 10 times per year, with 47.2% reporting seeing >30 cases.[5] Treatment consists of mechanical lavage and topical antimicrobials.[5] In Australia, the first-line treatment is dexamethasone/framycetin/gramicidin (DFG) combination ear drops, with only severe cases warranting oral antibiotics.[6–8]

The rationale for DFG drops is based on previous international literature that implicates *Pseudomonas aeruginosa* (PA) *and Staphylococcus aureus* (SA) as common pathogens.[9] However, diversification might occur during seasonal variations; in summer, PA has a greater incidence due to higher average temperatures, increased water contact and greater rainfall.[10] Demographics such as age and sex might also add to the variation of OE microbiota across the population.[11]

There is a large gap in data regarding pathogens responsible for OE in Australia. Most microbiological data originates from North America and Europe. We sought to explore the common pathogens causing OE in Queensland and to investigate our hypothesis that a variation in pathogens would be seen in different geographic locations with identifiably different climates. In addition, we wished to explore the hypothesis that there would be a variation in pathogens based on season, patient age and First Nations status.

Study aim

The primary aim of this study is to identify the most common pathogens implicated in OE that warrant ear swab culture in Queensland, Australia.

The secondary aims are to assess the effects of the following variables on the pathogen cultured:

1. Season: summer (October to March) compared to winter (April to September)
2. Location: coastal (within 50 km of a geographical coastline) compared to inland (greater than 50 km from a geographical coastline)
3. Age: under age 18 years (child) compared to older than 18 years (adult)
4. First Nations status: First Nations individuals compared to non-First Nations individuals.

Methods

Retrospectively, 1979 microbiology swabs sent to Pathology Queensland from hospitals statewide were retrieved from 1 January 2022 to 31 December 2022. The primary pathogen cultured, the specimen type, the date of collection and the patient demographics were retrieved.

A total of 232 specimens were excluded based on the specimen site information (eg abscess, pustule and lesion). Twenty-seven specimens were excluded due to the postcode being 'null' and 32 specimens because the postcodes were outside Queensland.

The following definitions were established to allow the classification of the variables:

1. Summer and winter were classified according to a six-month split of the highest temperatures and lowest temperatures in Queensland. In summer (October to March), the average temperature ranged between 19.1 and 28.23°C, and the average humidity at 3 pm was 57%. In winter (April to September), the average temperature ranged between 12.3 and 22.3°C and the average 3 pm humidity was 49%.[3,4]
2. Patients were classified according to age, with those aged ≥18 years considered adults and those aged <18 years children.
3. Coastal and inland locations were determined based on the postcode listed on the individual specimen. With the use of online mapping, each postcode that was greater than 50 km from a coastline was classified as inland. Those that were within 50 km of a geographical coastline were classified as coastal.

Statistical analysis

Multivariate analysis was performed using Statistical Package for the Social Sciences (SPSS) Version 29 (IBM Corp., Armonk, NY, USA). To allow a meaningful comparison, only the eight most prevalent organisms were analysed, accounting for 1559 of 1688 (92.4%) specimens.

Ethics approval

Ethics approval was attained from the Royal Brisbane and Women's Hospital Human Research Ethics Committee in January 2023 (approval EX/2023/MNHA/92667). This approval encompasses the entirety of the Pathology Queensland network.

Results

Demographics

Of the 1688 specimens analysed, 43% (726) were from female and 57% (959) from male patients, with gender not disclosed for <0.2% (3); 60% (1014) were classified as adults and 40% as children (674). First Nations individuals comprised 22.75% (384) and non-First Nations individuals 78.25% (1304) of the specimens analysed. Of the specimens for First Nations People, 45% (172) were classified as adults and 55% (212) as children. In terms of location, 86.3% (1457) were from coastal and 13.7% (231) from inland locations. Finally, 53% (888) of specimens were cultured in summer and 47% (800) in winter.

Pathogen distribution

Addressing the primary aim, the most prevalent pathogen cultured in the external ear in Queensland was PA at 37.9% (640), followed by SA at 22.5% (380), *Candida spp.* (CS) at 13% (219), *Streptococcus pyogenes* (SPy) at 6.7% (112), *Haemophilus influenzae B* (HIB) at 4.1% (69), *Aspergillus spp.* (AS) at 3.4% (58), *Streptococcus pneumoniae* (SPn) at 2.8% (48) and *Fungus spp.* (FS) at 2% (33) (Figure 1). All other pathogens combined comprised 7.6% (129) of the pathogen cultured in the external ear in Queensland (Appendix 1; available online only).

Comparing summer and winter, there was a decrease in the percentage of PA cultured from 43% to 33% (Figure 2). The incidence of SA, CS, SPy, AS and FS remained largely equivocal. HIB increased from 3% to 6% and SPn increased from 1% to 5%.

Looking at the location data, there was a reduction in PA cultured from 40% down to 32% between coastal and inland (Figure 3). There was an increase in SA from 21% to 32%, with an absolute difference of 11%. This was reflected as the most common pathogen in coastal locations being PA and in inland locations SA. CS decreased from 13% in coastal regions to 10% in inland regions. SPy, AS, HIB, FS and SPn remained largely equivocal.

Adults and children tended to grow different pathogens, with PA again being the most prevalent at 43% in adults and 31% in children (Figure 4). SA was seen in 20% of

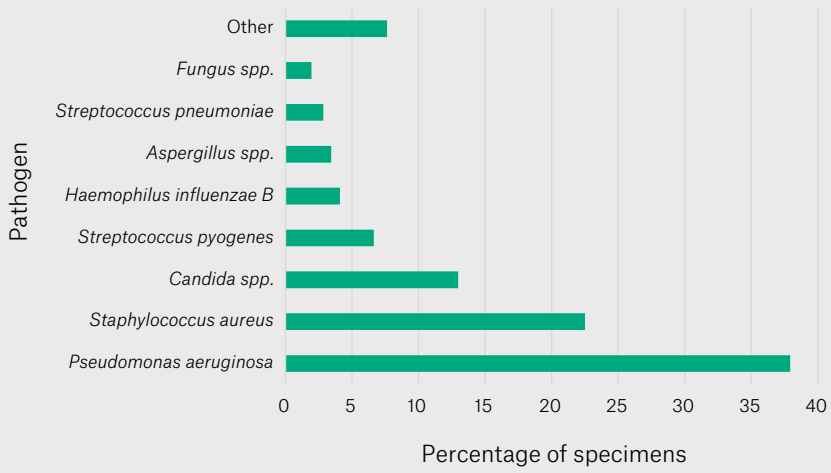

Figure 1. Pathogen prevalence.

adults and 26% of children. CS was cultured in 20% of adults and 6.2% of children. SPy was cultured in 0.7% of adults' specimens and 15.6% of children's. AS was cultured in 5.5% of adults' samples and 0.3% of children's. HIB was seen in 0.5% of adults and 9.5% of children. FS was seen in 2.3% of adults and 1.5% of children. SPn was seen in 2.1% of adults and 3.7% of children.

When examining the difference between First Nations people and non-First Nations individuals, PA was 35% in First Nations people and 39% in non-First Nations individuals (Figure 5). SA, AS and SPn were not significantly different. CS was 11% in First Nations people and 14% in non-First Nations individuals. SPy was 8% in First Nations people and 6% in non-First Nations individuals. HIB was 8% in First Nations and 3% in non-First Nations individuals. FS was 0.5% in First Nations people and 2.4% in non-First Nations individuals.

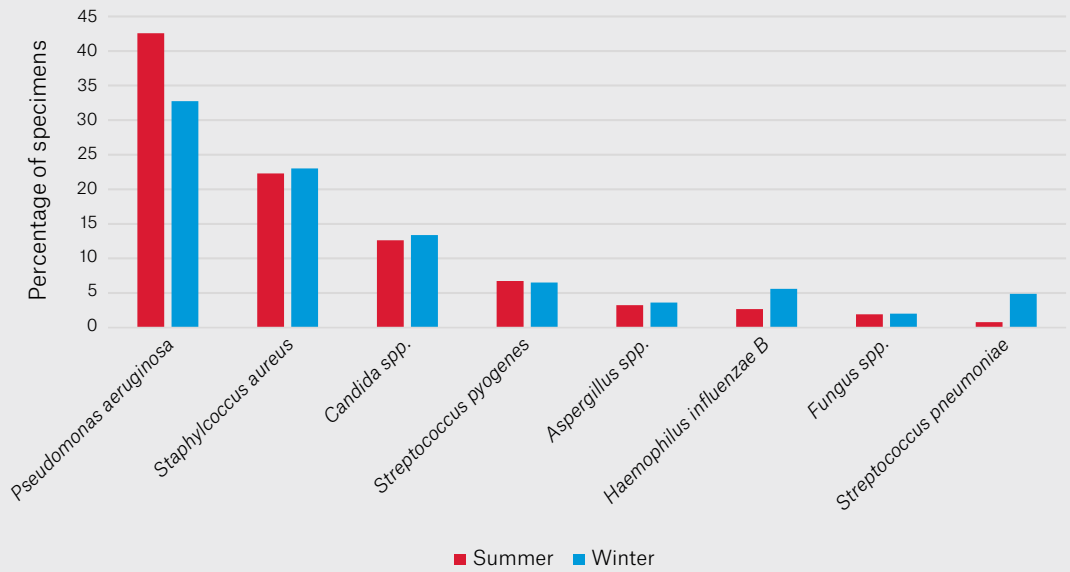

Figure 2. Seasonal variation of pathogens cultured.

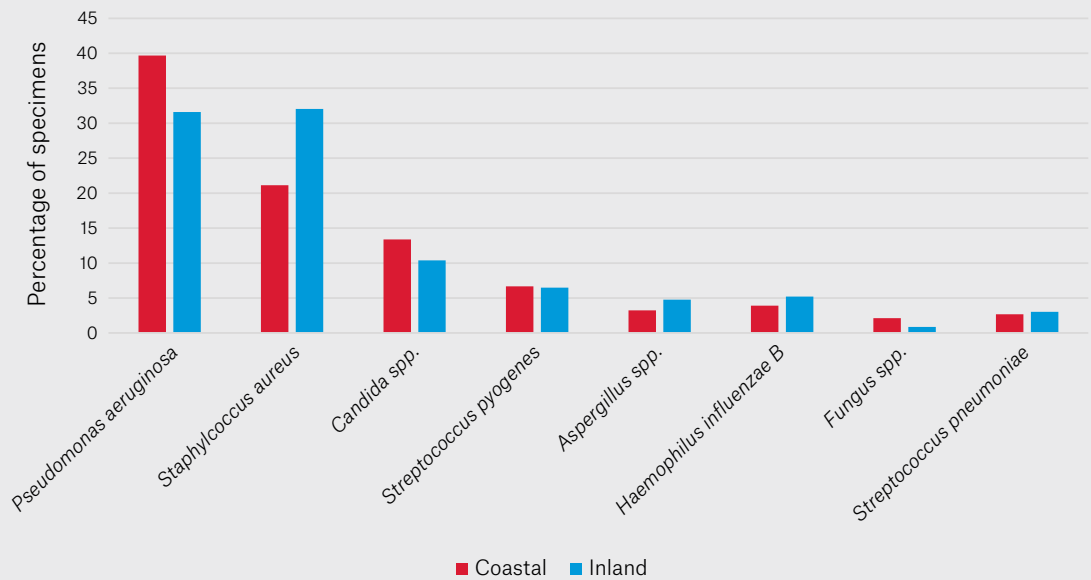

Figure 3. Locational variation of pathogens cultured.

Discussion

In 2022, PA was the most prevalent pathogen causing OE in Queensland, being present in 37.9% of all ear swab cultures.

Our results are consistent with those published earlier for Far North Queensland by Nofz et al (2019), who identified that out of 5580 total specimens, 39% cultured PA, 22% SA, 18% CS, 10% SPy, 10% HIB, 8% AS and 7% SPn.[12] The higher rates of respiratory pathogens (SA, SPy, HIB and SPn) are likely explained by the proportion of specimens from First Nations individuals, with Nofz et al having 60.3% specimens collected from First Nations people compared to only 22.7% in our dataset.

Interestingly, our results differ greatly from those published by Roland et al in 2002. In the North Americas, of 2048 individuals clinically diagnosed with OE, 98.3% cultured bacteria and only 1.7% cultured fungal species.[13] In contrast, our data showed a bacterial prevalence of 80.1% and a fungal prevalence of 19.9%. Yet the percentage

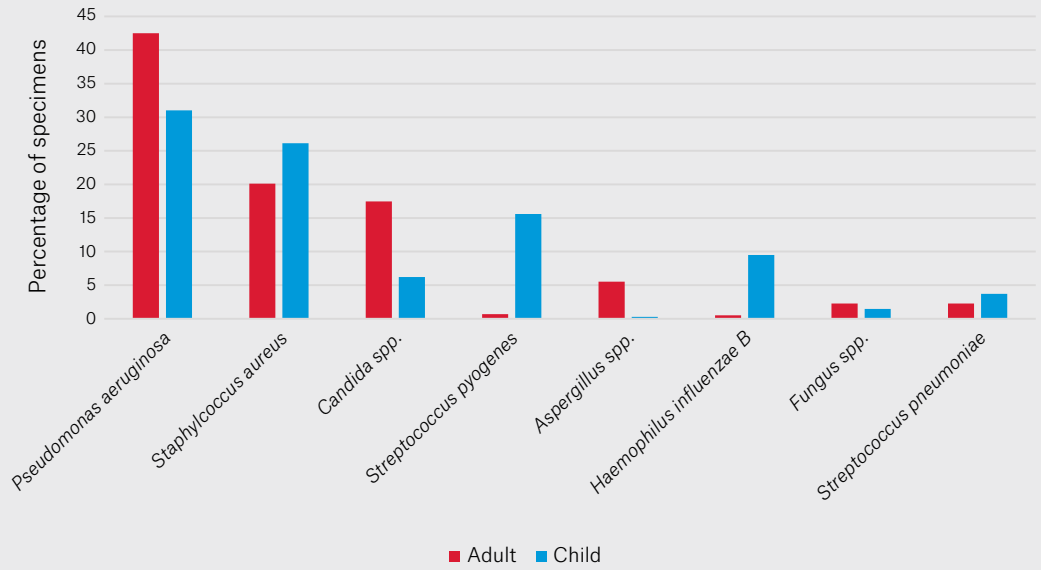

Figure 4. Variation of pathogens cultured with age.

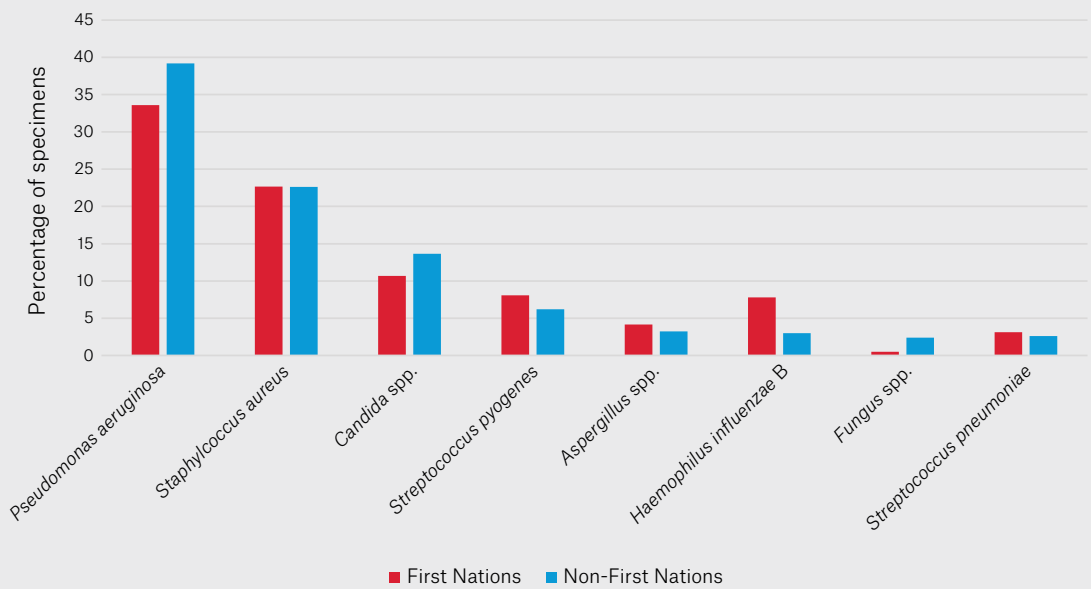

Figure 5. Variation of pathogens cultured based on First Nations status.

of PA cultured was near identical at 38%. Similarly, in the USA, OE clinical guidelines quote a prevalence rate of 20–60% for PA and 10–70% for SA, with fungal species being noted to be distinctly uncommon in acute OE.[14] Furthermore, Weigand et al (2019) had similar results, finding that >90% of OE are caused by bacteria, with the prevalence of PA being 22–62% and SA 11–34%. They came to a similar conclusion that fungal acute OE was rare, noting that long-term antibiotic use, immunosuppression and diabetes mellitus increased an individual's risk of OE.[1]

The higher rate of fungal pathogens cultured from specimens in our dataset is likely explained by the relatively higher humidity and average temperature compared to the Northern Hemisphere where much of the other available data are from. Higher humidity and temperature have been shown to lead to an increased incidence of fungal infections.[15]

Further analysis

To allow further analysis, 41 of 49 low-incidence (7.6%) pathogens were removed. A multivariate analysis revealed that the following were statistically significant: season (P<0.001), age (P<0.001), location (P<0.001) and First Nations status (P<0.001). Sex was not (P=0.362).

Three trends emerged, which remained consistent throughout the analysis. First, when compared to the overall results, there was a higher prevalence of SA (overall 23%) in both adults (33%) and children (35%) in inland regions, both of which were statistically significant (P<0.05). Similarly, for adults that lived within 50 km of a coastline, the rates of PA were higher: 48% compared to the overall 38%. A potential explanation for this might be related to access to swimming pools, rivers and beaches, as PA is a common pathogen in water.[16]

Most interestingly, there was a large discrepancy in the rates of fungal OE between adults and children. When the three analysed fungal species incidence was combined, this translated to 256 of 926 specimens (27.6%) for adults and 54 of 633 specimens (8.5%) for children: an over three-fold higher prevalence of fungal pathogens in adults than in children. The literature does not reveal a clear answer for this phenomenon. It might be attributable to fungal OE tending to occur in immunosuppressed, diabetic patients and those with a history of recurrent OE.[1]

Interpreting the results comparing First Nations people to non-First Nations individuals is challenging. The results will invariably be skewed from specimens collected for acute otitis media (AOM) and chronic suppurative otitis media (CSOM), rates of which are considerably higher in First Nations people.[17]

Confounding factors and limitations

The true incidence of OE is likely far higher than reflected here. Most GPs, emergency departments and ENT clinicians will not take a sample for microscopy, culture and sensitivity (MCS) unless there is reason to believe a patient will be non-responsive to first-line antimicrobial treatment. Conversely, the prevalence of pathogens in this dataset is demonstrated only in the same population of patients who are likely to present to medical services for treatment. Therefore, although this study might not fully represent patients with mild/subclinical OE, it is relevant to inform antibiotic guidance from the population illustrated here. Further, as these data are an aggregate of all specimens collected from the ear canal, as alluded to previously, there would invariably be specimens that were collected for AOM and CSOM. This makes the interpretation of respiratory pathogens, particularly in winter, more difficult. Similarly, the SA and SPy data need to be interpretated with caution, as both these pathogens are implicated in OE and otitis media. A further prospective exploratory study would be beneficial to ratify the trends identified in our study.

Clinical implications

At a population level, PA is common in Queensland, particularly in coastal regions during summer. Specific groups, especially in winter, might warrant MCS when PA is not the most common pathogen. Fungal OE in children is rare (<9%); as such, there should be a high level of clinical suspicion to commence antifungal ear drops. Conversely, SA is common, particularly in children living within inland communities, and this should be considered when selecting the appropriate antimicrobial ear drops.

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Palliative management of nausea and vomiting in advanced cancer

Taylan Gurgenci, Bridget Podbury, Cian O'Leary, Phillip Good

Background
Patients with cancer often experience nausea. In some cases, a specific cause such as chemotherapeutic adverse effects, raised intracranial pressure or malignant bowel obstruction is identified. In other cases, no specific cause is apparent.

Objective
This article summarises the evidence-based management of nausea in advanced cancer. It includes the nausea of select, specific contexts such as raised intracranial pressure and bowel obstruction.

Discussion
It is not commonly appreciated that low-dose oral haloperidol is the standard of care arm for many trials looking to reduce nausea in advanced cancer. It is available cheaply through the Pharmaceutical Benefits Scheme. The relative merits of other medications are discussed, as are the merits of an empiric versus a mechanistic approach to treatment.

NAUSEA has been reported in 6–68% of patients with cancer.[1] A subset of these patients will have a specific cause identified, leading to treatment according to a relevant guideline. The remainder will be explained as multifactorial nausea and vomiting, to which their opioids, decreased peristaltic function, hypercalcaemia, subacute renal function decline and other factors might contribute.[2] One recent prospective study of 821 Danish cancer patients estimated the prevalence of such multifactorial nausea to be 46%.[3]

This article will describe how best to manage this multifactorial nausea and vomiting, which is referred to in the palliative care literature and throughout this paper as 'the nausea and vomiting of advanced cancer'.[3] The management of select, specific conditions with specific management approaches is discussed.

Aim

This article aims to demystify the treatment of nausea and vomiting in advanced cancer and provide a practical approach using medication formulations that are available and affordable in Australia.

Nausea and vomiting in advanced cancer

This section considers the treatment of multifactorial nausea in cancer patients (ie the nausea and vomiting of advanced cancer).

The traditional approach was to try and discern the main mechanism of the nausea (eg delayed gastric emptying or vestibular impairment) and use this mechanism to guide management (eg metoclopramide or cyclizine). Whether this was superior to an empiric approach was the subject of a recent Australian randomised controlled trial (RCT).[4] This RCT was adequately powered for the primary outcome, which was response rate (not magnitude) at day 3, where response was defined as a two or more (out of 10) point drop in the nausea score (0–10) and an average nausea score <3 out of 10 for the preceding 24 hours, measured at 72 hours. There was no difference in response rate (53% for the empiric arm) nor was there a significant difference in the secondary outcome of response magnitude.

A subsequent RCT of haloperidol versus methotrimeprazine (levomepromazine; Nozinan, GL Pharma, Vienna, Austria) showed a response rate of 74% and a complete response rate of 55.9%, with no significant difference between the two arms.[5] In both trials, metoclopramide was used as the rescue antiemetic. No increase in bradykinesia or similar adverse effects was observed.

Based on the above, use of haloperidol for the nausea and vomiting of advanced cancer is accepted as evidence based.[6] Levomepromazine is non-inferior but caused more sedation. Both can be used in conjunction with metoclopramide for

breakthrough nausea. Table 1 shows our recommended doses, which differ slightly from the trial doses. Haloperidol does come in a 0.5-mg tablet, although this dose is probably too low to serve as a routine starting dose in the absence of extenuating circumstances such as marked patient concern regarding adverse effects.

There has been no rigorous trial to evaluate metoclopramide in this setting. Such trials are difficult to recruit for as metoclopramide is often used empirically by primary care or emergency clinicians prior to reaching a palliative care specialist conducting a trial. One such trial found no difference between ondansetron, metoclopramide and placebo for opioid-induced nausea and emesis, although the inference is limited by the small sample size.[7] Other small trials have shown a benefit. Some guidelines give a consensus recommendation for metoclopramide as the first-line medication.[8]

Some practitioners use ondansetron. Certainly, it has good evidence for chemotherapy-induced nausea and vomiting. The few trials that consider its use for advanced cancer are at significant risk of bias due to lack of blinding.[9–11] They are not powered for their primary outcome. The measurement of nausea is hours of nausea rather than intensity. This is atypical and limits comparison to most other trials, which measure magnitude at a fixed time point. There are no blinded placebo-controlled, adequately powered trials evaluating ondansetron of which we are aware.

It is also known to be quite constipating, a problem to which palliative patients are uniquely vulnerable. It is more expensive than haloperidol (Table 1). Like ondansetron, cyclizine can be an option for patients with Parkinson's disease.[6]

As a practical matter, it is worth noting that the primary outcome in these trials is response rate. Thus, if someone has responded to metoclopramide, then it would be an incorrect application of the available evidence to switch them to haloperidol. Rather, the evidence helps guide medication choice for a patient whose nausea is uncontrolled.

An important caveat is that there is no toxicity data for long-term, low-dose haloperidol use. This would be more relevant

Table 1. Dosing, adverse effects and cost of commonly used antiemetics in advanced cancer[A]

Drug	Recommended daily dosing	Adverse effects	Cost ($) per day
Metoclopramide	• 10 mg TDS PO PRN, to a maximum of 30 mg in 24 h • 5–10 mg TDS IM/SC PRN, to a maximum of 30 mg in 24 h	• Diarrhoea, dizziness, drowsiness, akathisia, increased serum prolactin, galactorrhoea, extrapyramidal side effects, neuroleptic malignant syndrome, arrythmias	• 0.40–1.20 • 3.23–9.69
Haloperidol	• 1.5–3 mg PO BD PRN or 1 mg SC BD PRN. The maximum dose we would use for the indication of nausea is 6 mg PO per day, or 5 mg SC per day • The dose in the RCT was 1.5 mg orally once daily, increased to 1.5 mg twice daily if nausea continued[5]	• Sedation, extrapyramidal side effects, hypotension, dystonia, neuroleptic malignant syndrome, parkinsonism, tardive dyskinesia, stroke, VTE, QT prolongation, arrythmias	• 0.07–0.22 • 1.56–4.70
Cyclizine	• 25 mg TDS PRN PO, to a maximum of 150 mg in 24 h	• Sedation, dizziness, constipation, urinary retention, dry eyes/mouth, dyskinesia, hallucinations, agranulocytosis, hepatic dysfunction	• 1.16–3.49
Ondansetron	• 4–8 mg TDS PO PRN, up to 24 mg in 24 h • Most trials used tropisetron.[9,10,11] One trial used ondansetron 24 mg per day[7]	• Constipation, extrapyramidal side effects, QT prolongation, arrythmias	• 1.2–5.10
Levomepromazine	• Not AMH/PBS listed • Start at 3.125–6.25 mg PO BD, to a maximum of 12.5 mg per day orally. Or 3.125 mg SC once daily, to a maximum of 3.125 mg SC twice daily • The trial dose was 6.25 mg PO OD, increased to 6.25 mg PO BD if nausea continued[5]	• Sedation, extrapyramidal side effects, dry mouth, postural hypotension	• Not readily available in community but is available through hospital pharmacies

[A]Prices are for Pharmaceutical Benefits Scheme (PBS)-recommended formulations listed by a prominent Australian pharmacy chain. Dosages and prices are listed for PO, IM and SC preparations where relevant. Maximum doses are specific to the indication of nausea in advanced cancer

AMH, Australian Medicines Handbook; BD, twice per day; IM, intramuscular; OD, once a day; PO, oral; PRN, as required; RCT, randomised controlled trial; SC, subcutaneous; TDS, three times a day; VTE, venous thromboembolism.

for patients with metastatic disease but who seem to have achieved remission on – say – immunotherapy, which can sometimes go on indefinitely. There is appropriate concern regarding the risk of cerebrovascular accident from long-term antipsychotic use in the elderly and non-elderly alike.[12,13]

Another relevant factor that might affect the prescription of these medications is cost. The palliative patient cohort is socioeconomically heterogeneous. While the Pharmaceutical Benefits Scheme (PBS) subsidises the cost of most of these medications in Australia, it does not remove cost as an issue. Certain medications have an additional 'safety net' reduced cost, available via Medicare through a Health Care Card. As such, this might be an important issue for patients of lower financial means. A summary of these medications, as well as their relevant dosing regimens, adverse effects and cost, is presented in Table 1. Some dosing recommendations include practical considerations such as the fact that haloperidol comes in a 1.5 mg tablet. We have included trial doses along with our recommendations so that any differences are clear, although in our opinion, they are quite minor.

Some common, specific causes of nausea

Cancer patients can have a myriad of causes for their nausea. To cover them all in one article would be difficult. We have somewhat arbitrarily decided to highlight two important causes of nausea: raised intracranial pressure (ICP) and malignant bowel obstruction (MBO). We discuss these causes before outlining an approach to managing the nausea and vomiting of advanced cancer. Chemotherapy-induced nausea and vomiting are usually obvious and managed by the medical oncologist prescribing the chemotherapy, so a detailed discussion is omitted for brevity.

Raised intracranial pressure

Practitioners will often consider raised ICP in a palliative cancer patient who complains of a new or different headache but might not consider it for the complaint of nausea alone. Similarly, nausea alone would not trigger an immediate referral for cerebral imaging by palliative specialists. So when should imaging be pursued?

In our view, if the nausea is paired with a new or different headache, then brain imaging is warranted. Computed tomography (CT) of the brain without contrast performs poorly and should only be considered in patients who cannot tolerate any form of contrast enhancement. Contrast-enhanced magnetic resonance imaging (MRI) is the gold standard, as it has the highest sensitivity for brain metastases and outperforms the alternatives in excluding intracranial leptomeningeal disease.[14] MRI might not be readily available in rural and remote areas. A strategy of contrast-enhanced CT and then, if negative, a trial of empiric therapy (see below) can be justified on practical grounds.

What if there is nausea but no headache? In the aforementioned Danish study, six of 378 (1.6%) patients with nausea were diagnosed with raised ICP.[3] There is no comment as to what other symptoms these patients had.

For palliative care patients, it seems prudent to restrict imaging for nausea without headache until antiemetic therapy has been trialled or some other sign of raised ICP has appeared.

The management of raised ICP will be guided by the aetiology. Symptomatic brain metastasis (nausea and headache) is managed initially with high-dose dexamethasone (8 mg orally twice a day) and a discussion with a radiation oncologist and/or a neurosurgeon, if so desired by the patient.

If radiotherapy and/or neurosurgery are not indicated or desired, the lowest dose of dexamethasone that relieves the nausea and other symptoms should be used. There is no hard evidence to guide dose reduction, but our practice is to reduce the starting dose of 16 mg by 4 mg orally every second day and use the lowest dose that maintains the clinical improvement. There is some evidence to suggest a lower dose is equally effective.[15]

Malignant bowel obstruction

MBO is a common occurrence in gynaecological and colorectal malignancies. Surgery is not always indicated nor beneficial in this condition (although a discussion with a surgeon is always valuable); conservative management of MBO is well established.

Conservative (medical) management involves dexamethasone (8–16 mg per day), antiemetics (typically haloperidol 2 mg per day subcutaneously), consideration of intravenous or subcutaneous rehydration and consideration of nasogastric tube (NGT) insertion. MBO in gynaecological cancers is frequently managed without the need for NGT insertion.

Octreotide is advocated in some guidelines, although we agree with the lead authors of one of the RCTs, which led to it being recommended that its routine use is not justified by the available evidence.[16]

Oral water-soluble contrast (eg Gastrografin, Bracco, Geneva, Switzerland) is thought by some to hasten MBO resolution. Trials to test this hypothesis have struggled with recruitment. One of the authors of the present study was involved in a pilot study that demonstrated a benefit, although it was open label and was not adequately powered.[17] The trial used 100 mL of oral Gastrografin per day for patients who did not improve with conservative management of their MBO. Conservative management was defined as ceasing oral intake, parenteral fluid replacement, parenteral dexamethasone (8 mg per day) and parenteral ranitidine (200 mg per day). Ranitidine is no longer available in Australia in this formulation.

In our clinical practice, if patients struggle with the taste, we often use 25–50 mL orally once or twice daily. An abdominal X-ray could be performed 24 hours following administration to see if it passes through to the rectum. If it does pass through to the rectum, the interpretation is not necessarily that the MBO has resolved. In fact, defining resolution for MBO trials is quite difficult. We would more cautiously interpret it as evidence that the patient might tolerate an upgrade to a small amount of clear fluid diet, if this is concordant with clinical findings (less or no nausea, passing flatus or stool, and less or no abdominal pain). Similarly, it is difficult to say whether recurrence of these symptoms after Gastrografin has been imaged in the rectum means a new MBO has occurred or reflects the fact that MBO is a dynamic process. We feel this is not a purely academic matter: appreciating how hazy the definition of MBO resolution is helps us set the expectations of our patients for the days and weeks following discharge.

The most common adverse effects in this trial were diarrhoea, nausea and

vomiting, and abdominal pain. Severe pneumonitis has been reported when oral water-soluble contrast has been aspirated, so it should not be given where the patient has difficulties swallowing or an altered level of consciousness.

Most clinicians avoid metoclopramide in MBO to avoid worsening pain, although a minority advocate using it to hasten resolution, particularly in partial MBO.[18] Neither recommendation is evidence based. Whatever antiemetic is chosen, it will need to be given subcutaneously, intravenously or sublingually. Therefore, ondansetron would seem appealing, although the high rate of associated constipation and lack of evidence-based benefit compared to haloperidol discourages its use in this context.

Conclusion

Nausea is a common problem in advanced cancer. When caused by raised ICP due to cerebral metastases, dexamethasone and consideration of radiotherapy or neurosurgical intervention are the mainstays of management. MBO can be managed without the need for surgery or an NGT. Where no cause can be identified, there is no evidence to suggest that trying to treat mechanistically is superior to treating with haloperidol with rescue metoclopramide. Levomepromazine is as effective as haloperidol but seems to be more sedating. Metoclopramide and ondansetron are often used, although rigorous evidence is lacking on their efficacy. The latter commonly causes constipation and is expensive.

Key points

- Nausea is common in advanced cancer.
- Some specific causes of nausea have specific treatments.
- Where no specific, treatable cause is found, the nausea can be treated empirically.
- Haloperidol is an effective, evidence-based first-line treatment for nausea and vomiting in advanced cancer.

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Penile dermatology for the general practitioner: A pragmatic approach to diagnosis and management

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Background
Genital skin conditions are rare and pose a diagnostic challenge due to their diverse pathology. Patient anxiety and referral decisions add complexity for primary caregivers. Demographics and overlapping symptoms complicate diagnosis, causing anxiety for both patients and clinicians. Social stigma and apprehension to seek healthcare might delay treatment. Accurate differentiation between benign and potentially serious conditions is crucial.

Objective
We aim to provide clinicians with a clear and concise framework to assist them in risk stratification, treatment decisions and referral pathways for common genital skin conditions.

Discussion
Differentiating normal variations is crucial to minimise unnecessary investigations and alleviate patient anxiety. Circumcision status, pigmentation and genetics influence disease presentation. We highlight benign conditions for reassurance. Inflammatory genital lesions might arise from various causes. Biopsies remain essential for accurately diagnosing uncertain cases. Sexually transmitted infections (STIs) should be promptly diagnosed and treated. Neoplastic conditions can evolve rapidly, requiring an urgent specialist referral.

MALE GENITAL DERMATOSES are nuanced and demanding presentations for general practitioners (GPs). Their diverse clinical presentations and cross-specialty referral pathways can be onerous to navigate. Recognising and initiating management in the primary care setting is critical, and determining when to seek subspecialist advice remains controversial. The interplay of diverse demographics, patient anxiety and overlapping signs and symptoms often complicate the diagnostic pathway. Social stigma and reluctance to seek medical attention is a common reason for delayed treatment. Accurately distinguishing between normal variations, benign presentations and potentially infectious or malignant conditions is paramount to reducing morbidity and improving public health.

Aim

We aim to provide an up-to-date and concise framework to guide clinicians in their risk stratification and decisions for treatment, and potential referral of these conditions.

History and examination

Establishing a safe and culturally sensitive rapport is paramount when assessing penile dermatological concerns.[1] A thorough examination, using accurate descriptors of the dermatosis, is essential for documentation and potential referrals. Take note of circumcision status and location of the abnormality, and compare findings with the skin condition elsewhere on the body.[2] Clinical photography with patient consent is a valuable diagnostic aid and is being increasingly used in artificial intelligence–augmented diagnostic tools.[3]

Non-malignant/non-infectious conditions

Normal penile dermatological variation

Normal genital skin variations are common and can produce anxiety for patients and physicians. It is crucial to distinguish normal variants from treatable pathology to minimise unnecessary investigations and alleviate patient concerns.

In circumcised males, the glans epithelium becomes keratinised, and might appear purple. Similarly, there are variations in scrotal colour and the hue of the midline raphe.[4]

Variations can be attributed to skin phototypes, ancestry/ethnicity and genetic factors. Although reassurance is adequate, pigmentation changes might also accompany inflammatory diseases, and normal pigmentation differences can impact a patient's sexuality and psychological wellbeing.

Common benign presentations

Pearly penile papules are common, presenting as tiny pale papules localised to the coronal sulcus, and might form multiple rows (Figure 1A). Fordyce spots are ectopic sebaceous glands appearing as pale micropapules (Figure 1B).

Median raphe cysts are benign, pale, fluid-filled sacs along the scrotal raphae (Figure 1C). Angiokeratoma of Fordyce are vascular malformations appearing as tiny red–purple scrotal spots that might occasionally bleed (Figure 1D). These conditions do not require intervention, and offering reassurance and observation is safe.

Red penile lesions

> *Genital disease is mostly non-infectious skin disease*

Isolated glans inflammation is termed balanitis, whereas balanoposthitis encompasses reactions involving the glans and foreskin. These terms describe red genital dermatoses but do not specify the underlying cause. The mnemonic 'RED-PENIS' is helpful for recalling these aetiologies (Table 1).

Reactive arthritis

Reactive arthritis is a multisystem autoimmune inflammatory condition with a genetic predisposition, characterised by urethritis, arthritis and conjunctivitis with cutaneous involvement (Table 1, section 1.1).[5] It most commonly affects young men aged 20–30 years, preceded by a triggering infection.[6]

Eczema/dermatitis

Irritant dermatitis is common and is often misdiagnosed, especially in uncircumcised men (Table 1, section 1.2), as the preputial recess is an intertriginous site where skin rests against. Scrotal skin is particularly susceptible, and identifying a single causative agent is challenging.[7] Uncontrolled skin inflammation over time can lead to lichenification, characterised by scaly, thickened skin with surface excoriation. Atopic dermatitis should be considered in association with previous atopy.

Allergic contact dermatitis of the male genital skin manifests as acute, severe dermatitis. A thorough history, patch testing and avoiding allergen exposure form the cornerstone of management.

Drug reaction

An immunological reaction to a range of drugs, causing a fixed reaction of variable cutaneous involvement (Table 1, section 1.3), occur on the genitalia in 20% of patients, and might be immediate or delayed up to one to two months.[8]

Psoriasis

Psoriasis affects approximately 3% of the population and 29–40% will experience genital manifestations (Table 1, section 1.4).[9,10] Over two-thirds of men with chronic plaque psoriasis will experience genital involvement at some point in their disease course, characterised by salmon-red, scaly plaques. Genital psoriasis might occur independently but is frequently associated with inverse (flexural) psoriasis, often impacting the groin.[11] Patient concern centres around appearance and negative impacts on sexual wellbeing. When diagnosis is uncertain, biopsy can rule out neoplasia, particularly for solitary glans lesions.[12]

Neoplasia

Penile intraepithelial neoplasia

Penile intraepithelial neoplasia (PIN) encompasses in situ squamous cell carcinoma (SCC) and erythroplasia of Queyrat. PIN is a non-invasive, pre-malignant proliferation of cells demonstrating variable levels of abnormality including keratinisation and nuclear atypia (Table 1, section 1.6.1).[13] This condition can be differentiated based on cell-turnover drivers, including lesions arising from human papillomavirus (HPV) infections and chronic inflammation.[14,15] Diagnosis is made by biopsy or excision. Regular surveillance is crucial as the recurrence rate is nearly 50%; however, with adequate treatment only 2% will progress to invasive carcinoma.[16]

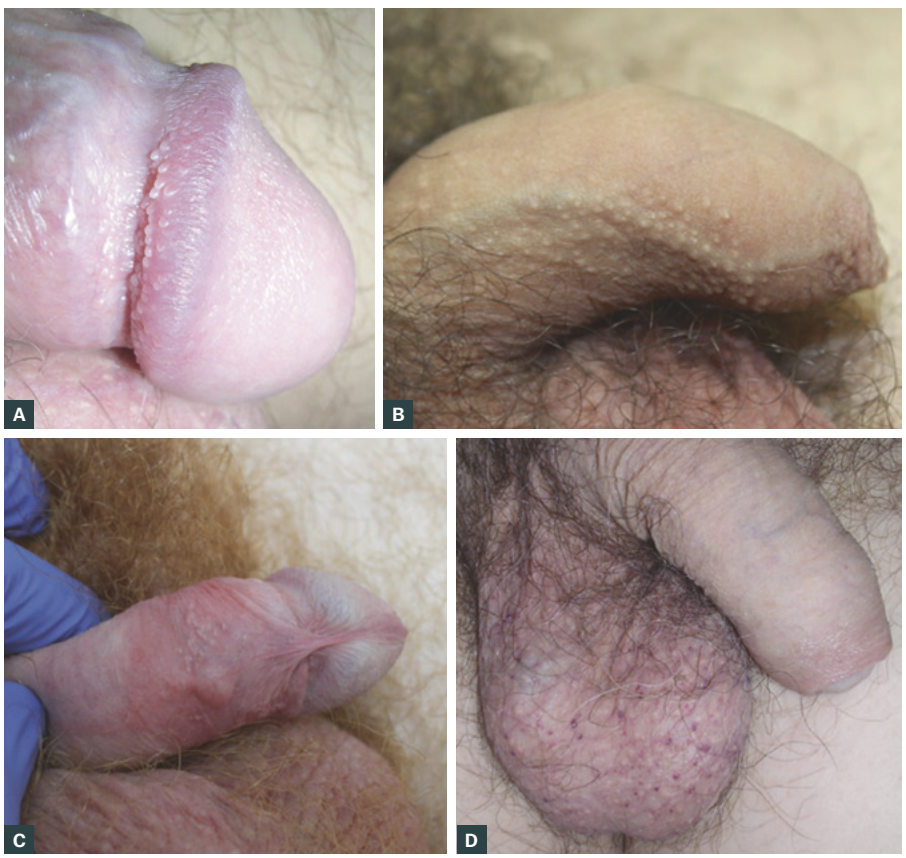


Figure 1. Photographic examples of common benign penile dermatological presentations. (**A**) Benign pearly penile papules; (**B**) Fordyce spots; (**C**) Median raphe cysts; and (**D**) Angiokeratoma of Fordyce.

Reproduced from Hall A. Atlas of male genital dermatology. Springer Nature Switzerland AG, 2019, doi: 10.1007/978-3-319-99750-6, with permission from Springer Nature Switzerland AG.[39]

Squamous cell carcinoma of the penis

Penile SCC is a rare but potentially aggressive malignancy that requires urgent management by a uro-oncologist (Table 1, section 1.6.2). The contemporary five-year survival for regionally advanced disease is 51%.[17] Australian penile cancer incidence is <0.1 per 100,000 or approximately 300 diagnoses per year.[18] Early treatment is paramount to penile-preserving intervention and survival. However, many men experience a treatment delay of more than six months due to social stigma, neglect and access to care, resulting in poorer outcomes.[19] Palpable inguinal lymphadenopathy will be present in 28–64% of men at presentation, with almost 50% of these representing regionally advanced disease.[19]

Diagnosis is by excisional biopsy and minimally invasive sentinel lymph node biopsy.[20] For low-risk penile SCC, early surgical intervention might be curative. There is a minimal role for chemoradiotherapy in physically fit patients with lymph node-positive disease.[21,22] Radical lymphadenectomy offers the best outcomes for high-risk disease.[23] Although more research is needed for this rare disease, recent data suggest that men experience better outcomes when managed by an experienced uro-oncology team at a high-volume tertiary hospital.[24]

Infections

Non-sexually transmitted

Opportunistic fungal infection is typically caused by the Candida species. Uncircumcised men with pre-existing immunocompromise are most at-risk (Table 1, section 1.7.1).[25] Diagnosis is confirmed through a skin swab. Effective treatment usually only requires a topical imidazole cream. Persistent candidal infection might require oral treatment agents.

Sexually transmitted

Approximately one in six Australians might contract an STI in their lifetime.[26] Partner contact tracing should be conducted whenever possible. Genital warts (*Condyloma acuminata*) are common in the anogenital region, caused by specific types of HPV (Table 1, section 1.7.2). Although primarily attributed to HPV types 6 and 11, co-infection with high-risk types 16 and 18 can lead to malignant transformation.[27] Treatments include cryotherapy, topical imiquimod and podophyllotoxin. There is no curative treatment as the virus embeds within host cell DNA.[28] All patients should undergo partner contact tracing, and female partners should undergo a swab for HPV DNA detection. Routine vaccination of all adolescents with a HPV vaccination is an important public health measure to reduce the likelihood of infection.

High-risk individuals and those aged <26 years should receive the Gardasil-9 vaccine.[29]

Genital herpes (herpes simplex virus-2) can be confirmed on dry swab PCR (Table 1, section 1.7.3). Patients often present with first reactivation of the virus and can be effectively treated with topical or oral antivirals.

Primary syphilis (*Treponema pallidum)* in males typically presents initially with a painless chancre[30] (Table 1, section 1.7.4), although these might be overlooked at examination if occurring in the oral mucosa or rectally. Untreated syphilis can ultimately affect many organ systems. High-risk groups include those engaging in unprotected sex with multiple partners and is more prevalent in men who have sex with men. Diagnosis requires a combination of serology and nucleic acid amplification tests from a lesion swab. Patients should be screened for co-infection with HIV.[30]

Sclerosis

Lichen planus

Lichen planus is a multifocal inflammatory skin condition that occasionally presents on genitalia in both males and females (Table 1, section 1.8.1). Chronic ulcerative lichen planus can lead to genomic instability, increasing malignant potential.[31] Careful monitoring and consideration of circumcision is critical for mitigating the risk of penile SCC.

Plasma cell balanitis (Zoon's)

Zoon's balanitis (plasma cell balanitis) occurs in uncircumcised older males (Table 1, section 1.8.2). It might develop gradually over months to years and is generally asymptomatic.[32] A biopsy is recommended to rule out inflammatory or premalignant conditions.

Genital dysaesthesia

Male genital dysaesthesia usually presents as a burning sensation of the genital skin, akin to vaginal vulvodynia (Table 1, section 1.8.3).[33] In more severe cases, male genital dysaesthesia might be painful, resulting in dyspareunia and avoidance of sexual activity. Specialist referral is often warranted.

White lesions

Lichen sclerosus

Lichen sclerosus is a chronic, sclerosing inflammatory condition of uncertain cause of the anogenital skin (Table 2, section 2.1).[34] Biopsy should be considered when the diagnosis is uncertain. Untreated cases with persistent phimosis and chronic inflammation can lead to malignant transformation, but there is debate on this issue. The lifetime risk of malignant transformation has not been established.[35,36]

Phimosis

In Australia, circumcision rates have declined, dropping from a peak of 85% in the 1950s to 19% in 2019.[37] Physiological phimosis is normal at birth and might variably persist in children. Phimosis is considered physiological if penile function or voiding are unaffected.[38]

Pathological phimosis exists when the foreskin remains non-retractile beyond puberty, resulting in problematic voiding or dyspareunia (Table 2, section 2.2). Retraction of a pathologically phimotic foreskin might lead to a tight 'waisting' of the band around the glans or penile shaft.[38]

Lichen sclerosus with phimosis can lead to pinhole phimosis, causing painful erections and dyspareunia, and rarely urinary obstruction or recurrent infections, requiring circumcision.[34]

Investigations

All investigations for genital skin lesions are specific to the suspected pathology and should be considered in the setting of detail history and examination (Table 3). Histopathological analysis is an invaluable diagnostic tool for penile skin dermatoses. It is crucial to rule out premalignant or malignant disease, but histopathology might have limitations in interpretation of inflammatory lesions (Figure 2). The commonest complications of genital skin biopsies are vasovagal reactions, bleeding and scarring. All biopsies should be performed after informed consent is obtained, under local anaesthetic, in a sterile fashion.

Table 1. Red penile dermatoses – organised by the mnemonic 'RED-PENIS'

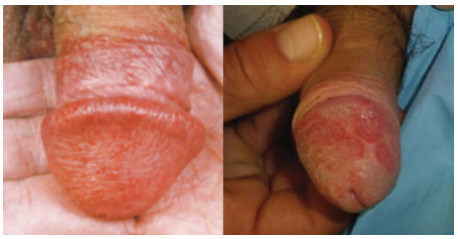
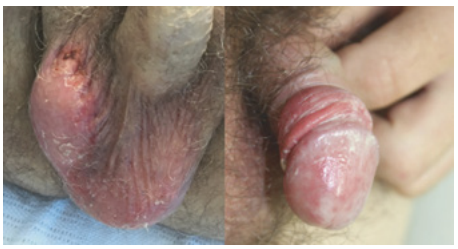
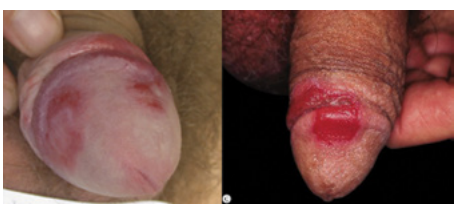
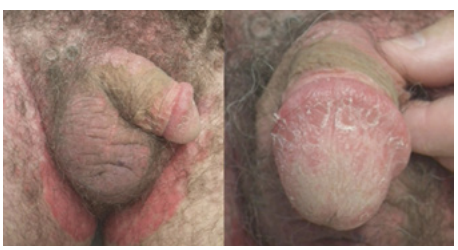
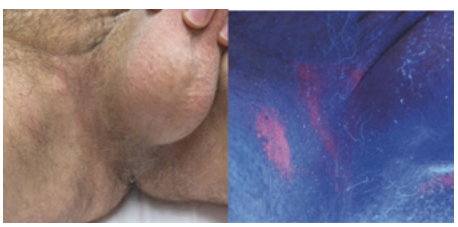
Condition	Examples[A]	Aetiology	Clinical features	Treatment
1.1 Reactive arthritis		• Likely autoimmune with genetic predisposition • Associated with HLA-B27 positivity[1]	• Urethritis, arthritis, conjunctivitis with cutaneous involvement (1/3 genital) • Scaly psoriasiform plaques • Might have ulcers/erosions • Might crust and be tender	• Usually self-limiting within 12 months • Might relapse • Analgesia/NSAIDs • Corticosteroids (topical and oral) • DMARDs
1.2 Eczema/dermatitis • Irritant/contact • Allergic contact • Atopic		• Often no clear allergen/irritant	• Asymptomatic • Balanoposthitis – red macules, plaques and patches with poorly defined erythema • Intractable itch • Chronic – skin thickening	• Minimising irritants • Emollients • Cool (sitz) baths • Low–moderate potency topical corticosteroids
1.3 Drug reaction • NSAIDs • Antibiotics • Sulfasalazine • Paracetamol		• Exact mechanism unclear – drug likely combines with protein to cause an immunological reaction	• Commonly on limbs, but on genitals in 20% of patients • Asymptomatic • Might have itching, swelling, tenderness and lower urinary tract symptoms (urethritis) • Solitary erythematous/hyperpigmented patch/plaque (+/– red halo)	• Recognition and avoidance of the causative drug (after skin biopsy to confirm diagnosis) • Moderate potency topical corticosteroid
1.4 Psoriasis • Chronic plaque • Inverse (flexural)		• Immunological basis with a genetic predisposition, influenced by environmental factors[14] • Risk factors: diabetes, immunocompromised, other skin disease, age, obesity, antibiotic use	• Red scaly plaques • Might be confined to genitals • Flat red macules in uncircumcised men • Affects other parts of body – skin/nails	• Minimising irritants, use of emollients • 6-week trial of high-potency topical corticosteroids • Systemic psoriasis treatment (biologics, immunosuppressants)[15]
1.5 Erythrasma • *Corynebacterium minutissimum* (gram-positive skin commensal) in flexures		• Benign intertriginous eruption associated with diabetes, hyperhidrosis, obesity and immunosuppression	• Symmetrical scaly red–brown plaques in flexures • Can be itchy • Fluoresces under Wood's lamp examination • Involvement of other intertriginous sites	• Avoidance of skin irritants • Reduction of sweating • Topical/systemic antibiotic treatments (clindamycin, fusidic acid, erythromycin, mupirocin)[16]

Table continued on the next page

Table 1. Red penile dermatoses – organised by the mnemonic 'RED-PENIS' (cont'd)

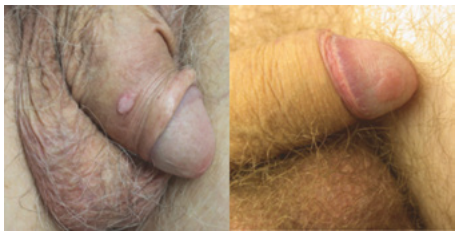
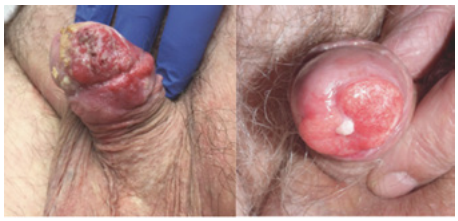
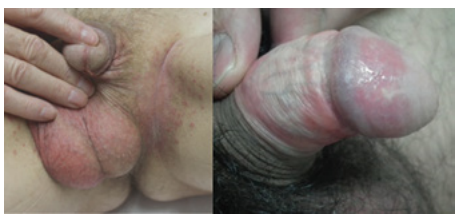
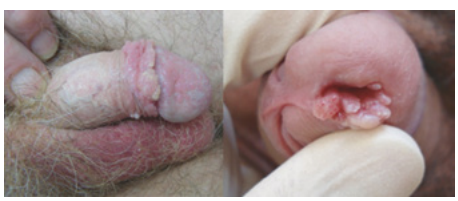
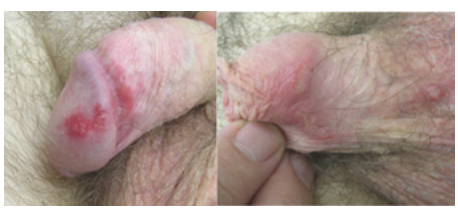
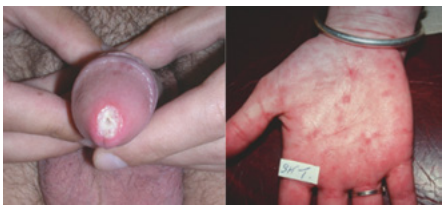
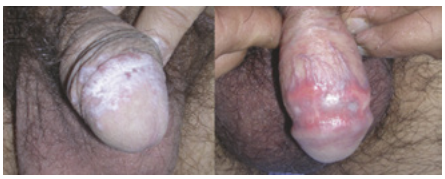
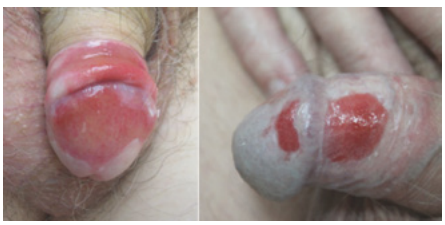
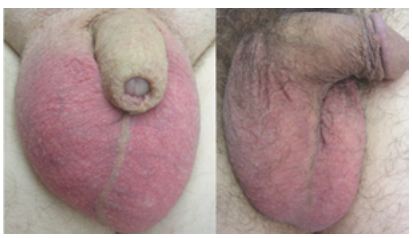
Condition	Examples[A]	Aetiology	Clinical features	Treatment
1.6 Neoplasia				
1.6.1 PIN (erythroplasia of Queyrat)		• Risk factors: uncircumcised men, HPV infection, lichen sclerosis, smoking, immunocompromise[17]	• Solitary, slow growing, discoloured plaque, and might have itch, crusting or tenderness	• Urgent referral to a urologist is required • Local treatments can include shave excision, cryogenic/laser therapy, topical treatments (5-fluorouracil, imiquimod), surgical excision • HPV vaccination
1.6.2 Penile SCC		• Risk factors: history of phimosis/chronic inflammatory conditions (eg lichen sclerosis), previous UVA treatments/ exposure, smoking and high-risk HPV infection (HPV subtypes 16 and 18)[17]	• Asymptomatic • Itchy, painful, bleeding, malodorous lesions • Progressive erythematous skin lesion/palpable nodule on glans, coronal sulcus or foreskin • Invasive: exophytic ulcerated/eroded nodules fungating locally destructive lesions • Can cause voiding dysfunction	• Early urgent referral to a urologist and histopathological diagnosis • Surgical excision +/– sentinel lymph node biopsy, radiotherapy
1.7 Infectious				
1.7.1 Candidiasis • Mostly *Candida albicans*		• Risk factors: diabetes, immunocompromised	• Balanoposthitis with itch, pain, weeping/ white discharge • Might spread to groin/thigh regions • Uncommon in circumcised men	• Cooling baths • Topical/oral azole treatment • Low-potency topical corticosteroids
1.7.2 Genital warts • Condyloma accimunata • Clinical +/– skin biopsy		• Mostly caused by HPV types 6 and 11 (low risk) • Note: infected individuals are ALSO at risk of co-infection with subclinical high-risk HPV types, mostly 16 and 18	• Warts in the genital/ inguinal and anal region • Might be recurrent	• Spot cryotherapy • + imiquimod cream/ podophyllotoxin • Regular review for resolution and follow-up for malignant change
1.7.3 Genital herpes • Dry swab for PCR		• Herpes simplex virus-2	• Primary infection – asymptomatic • Reactivation – severe pain/dysuria/ discharge with acute red papules +/– blisters/ulceration	• Oral antiviral therapy to reduce severity of flares (acyclovir/valaciclovir/ famciclovir) • Analgesia, topical local anaesthetic • Avoid sexual activity during symptomatic/ acute phase

Table continued on the next page

Table 1. Red penile dermatoses – organised by the mnemonic 'RED-PENIS' (cont'd)

Condition	Examples[A]	Aetiology	Clinical features	Treatment
1.7.4 Syphilis • Syphilis serology		• *Treponema pallidum*	• Primary: painless ulcer/chancre (urethral, anogenital, oral) • Secondary: skin/mucous membranes – macules/papules (palms/soles) • Tertiary (neurosyphilis)	• Intramuscular penicillin – benzathine benzylpenicillin 2.4 million units • Long-term monitoring of serology
1.8 Sclerosis				
1.8.1 Lichen planus • Hyperkeratotic • Annular • Lace pattern • Erosive		• Might be T-cell mediated[18]	• Asymptomatic • Multifocal itchy papules +/– erosions, weeping • Erosive lesions can be painful	• High-potency topical corticosteroids for control • Needs monitoring for malignant change
1.8.2 Plasma cell balanitis (Zoon's)		• Mild trauma/irritation of the subpreputial space	• Solitary moist orange–red heterogenous plaque of the glans and/or foreskin (+/– mirroring)	• Combination topical antibiotic + corticosteroids
1.8.3 Genital dysaethesia		• Unknown • Neuropathy, inflammation, neurovascular, iatrogenic (steroids), allergic	• Might not have any signs • Uniformly red scrotal +/– genital skin with sensation change	• Avoid irritants/remove triggers (alcohol/caffeine) • Use emollients • Low-dose amitriptyline/SSRIs

[A]All images have been reproduced from Hall A. Atlas of male genital dermatology. Springer Nature Switzerland AG, 2019, doi: 10.1007/978-3-319-99750-6, with permission from Springer Nature Switzerland AG.[39]

DMARDs, disease-modifying antirheumatic drugs; HLA-B27, human leukocyte antigen B27; HPV, human papillomavirus; NSAIDs, non-steroidal anti-inflammatory drugs; PCR, polymerase chain reaction; PIN, penile intraepithelial neoplasia; SCC, squamous cell carcinoma; SSRIs, selective serotonin re-uptake inhibitors; UVA, ultraviolet A.

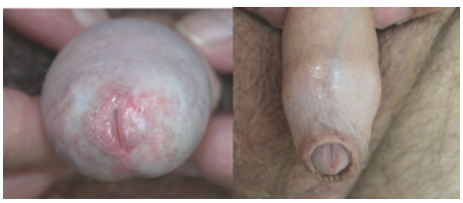
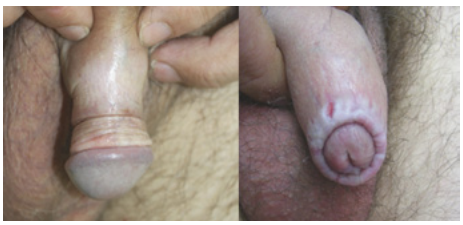
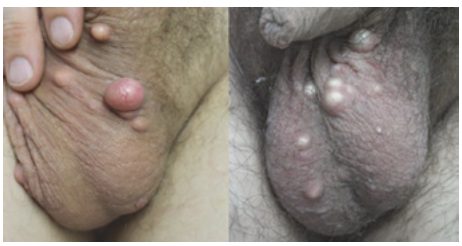
Conclusion

Urological dermatology can be a complex area for general practitioners and urologists, as it encompasses diverse pathology with overlapping clinical presentations. Fortunately, many can be effectively managed in a primary care setting. However, considering a biopsy or seeking multidisciplinary specialist guidance is crucial if there is uncertainty in diagnosis or if initial treatments do not lead to timely improvement. This proactive approach will alleviate patient anxiety and ensure optimal care.

Key points

- Normal variations and appearance in genital skin are common and might cause unnecessary anxiety for patients and physicians alike.
- Genital skin disease is mostly non-infectious in aetiology.
- Pathological phimosis is best treated with upfront topical corticosteroid therapy referred for consideration of circumcision if refractory.
- Opportunistic screening and prevention of STIs should be undertaken whenever appropriate, especially in the context of co-infection.
- Genital skin biopsy is essential to confirm or rule out penile neoplasia.

Table 2. White penile dermatoses

Condition	Examples[A]	Aetiology	Clinical features	Treatment
2.1 Lichen sclerosus		• Likely autoimmune in origin (but not associated with other conditions)	• Asymptomatic • Patchy pallor of foreskin/glans • Usually in uncircumcised males • Itching • +/– phimosis, skin atrophy • +/– purpura/ telangiectasia, erythema	• 4- to 6-week trial of moderate potency corticosteroid • Referral to specialist • Needs monitoring for malignant change
2.2 Phimosis (pathological)		• Lichen sclerosis • Trauma/injury	• Symptomatic non-retractile foreskin beyond puberty • Waisting on retraction • Spraying urinary stream • Painful erections/sex	• Primary: 4- to 12-week course of moderate potency topical corticosteroids • Secondary: circumcision can be considered
2.3 Epidermal cysts • Idiopathic scrotal calcinosis		• Cysts arise from pilosebaceous follicles and calcinosis is dystrophic calcification of these cysts	• Asymptomatic firm lesions varying in size and colour that are mobile over deeper structures	• Reassurance • Might be surgically excised for cosmetic reasons

[A]All images have been reproduced from Hall A. Atlas of male genital dermatology. Springer Nature Switzerland AG, 2019, doi: 10.1007/978-3-319-99750-6, with permission from Springer Nature Switzerland AG.[39]

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Table 3. Investigations and procedures useful for the diagnosis of penile dermatoses in the general practice setting

Investigation	Examples	Procedure
UVA (Wood lamp)	• Erythrasma • Pigmentation disorders • Fungal infection	Utilising UV light from different sources from mercury vapour to LEDs. A range of pathologies fluoresce variably under UV light
Bedside microscopy	• Scabies • Pubic lice	A microscopic examination of skin scraping might reveal skin mite/insect infestation
Skin swab/laboratory MCS	• HSV • *Neisseria gonorrhoeae* • *Chlamydia trachomatis*	Useful for bacterial/fungal infections. Might also increase detection of STIs through NAT PCR
Serological testing	• Syphilis, HBV, HCV, HIV	Screening for underlying STI, if indicated. Completion screening for other STIs is mandatory if one is found
Skin patch testing	• Allergic contact dermatitis	Patch testing against common allergens
Procedure	**Indication**	**Technique**
Punch biopsy (3–4 mm)	• Non-pigmented lesions of glans • Suspected non-malignant lesions	Rotate the disposable punch biopsy without pressure. Elevate the specimen with a needle and cut the specimen at proximally. An absorbable suture might be used for haemostasis
Deep incisional biopsy Excisional biopsy	• Suspicion of invasive cancer • Pigmented lesions	Using a small blade, excise the lesion with some normal tissue around it (skin margin), in an elliptical fashion. An absorbable suture(s) might be used for haemostasis
Shave biopsy	• Pigmented lesions • Lesions on the penile shaft	A flexible razor or scalpel might be used to shave the lesion. Chemical haemostasis might be utilised in this type of biopsy
Curette/snip biopsies	• Pedunculated lesions	A blade might be used to remove a lesion on a stalk

HBV, hepatitis B virus; HCV, hepatitis C virus; HIV, human immunodeficiency virus; HSV, herpes simplex virus; LEDs, light emitting diodes; MCS, microscopy culture and sensitivity; NAT PCR, nucleic acid testing by polymerase chain reaction; STI, sexually transmitted infection; UV, ultraviolet; UVA, ultraviolet A.

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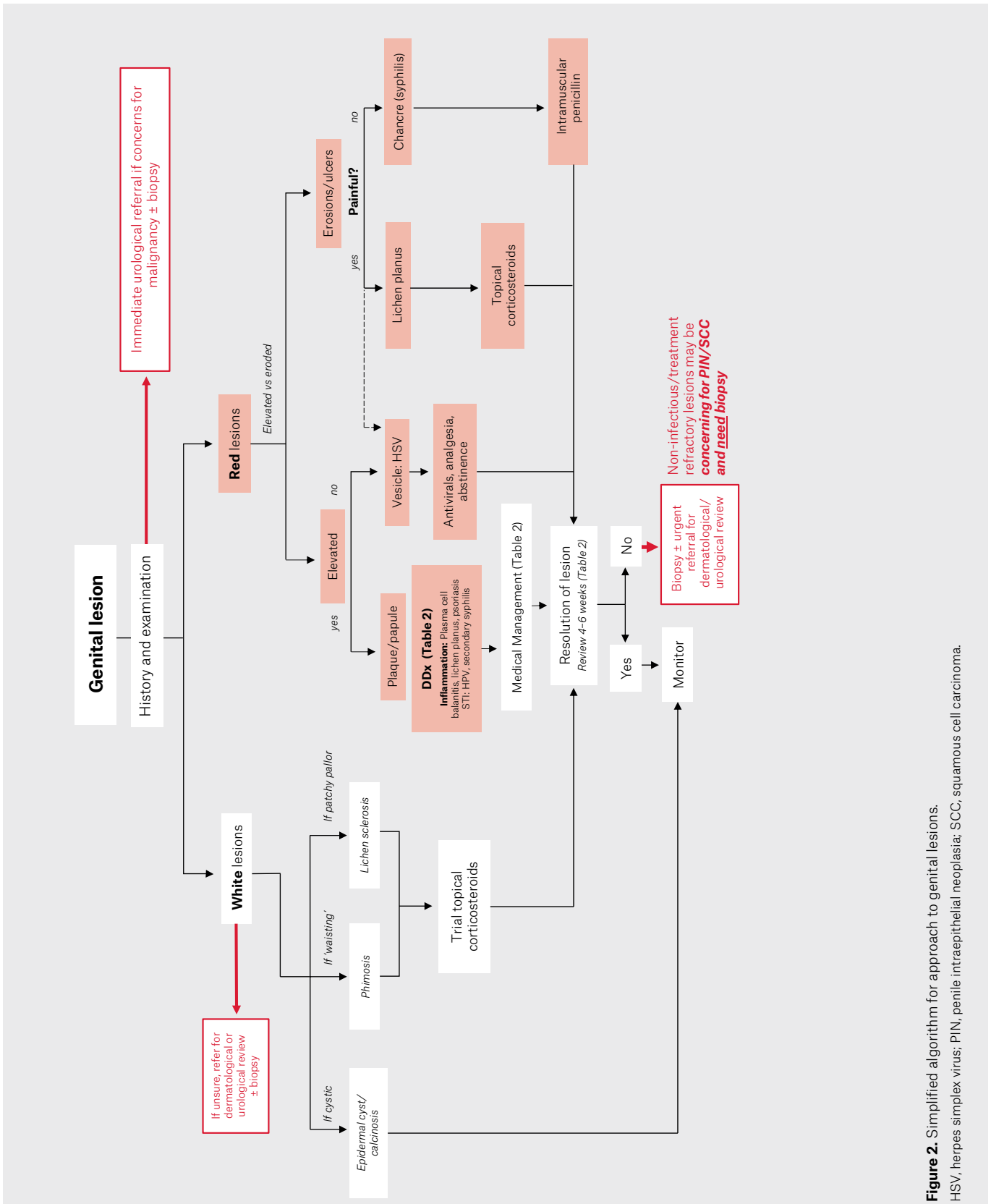

Figure 2. Simplified algorithm for approach to genital lesions.
HSV, herpes simplex virus; PIN, penile intraepithelial neoplasia; SCC, squamous cell carcinoma.

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Common incidental urological lesions on computed tomography images: What to do with renal and adrenal computed tomography incidentalomas in a primary care setting

Jianliang Liu, David C Homewood, Nieroshan Rajarubendra, Prem Rashid, Damien Bolton, Nathan Lawrentschuk

Background
The widespread use of cross-sectional imaging has led to the increased detection of urological incidentalomas. Incidental renal and adrenal masses are the most commonly detected urological incidentalomas and are often encountered by general practitioners.

Objective
This review aims to provide an evidence-based approach to managing renal and adrenal masses.

Discussion
Renal lesions occur in 14% of computed tomography (CT) scans. Differentials include cysts (benign or malignant), angiomyolipomas, oncocytomas and renal cell carcinomas (RCCs). The Bosniak classification should be used for cystic renal lesions. Active treatment should be considered for RCCs that are >4 cm, symptomatic or rapidly growing. Patients with adrenal lesions should undergo functional work-up. If clinically concerned, screening tests include 1 mg overnight dexamethasone suppression test and plasma or urinary metanephrines. In the presence of hypertension or hypokalaemia, screening for hyperaldosteronism with the plasma aldosterone-to-plasma renin ratio should be considered. Benign adrenal adenomas on CT are <4 cm, homogenous and hypodense (Hounsfield unit <10).

WIDESPREAD USE of cross-sectional imaging has resulted in increased detection of urological incidentaloma. Renal and adrenal masses are the most common, respectively accounting for 14% and 5% of incidentalomas in patients undergoing computerised tomography (CT).[1,2] Incidentalomas pose a diagnostic challenge, balancing between over-investigation and misdiagnosis.

Aim

This clinical review aims to provide an approach for the management of renal and adrenal incidentalomas.

Approach to renal incidentaloma

Renal cyst

The incidence of renal cystic disease increases with age, with most cysts being benign.[3] The Bosniak classification is used to characterise renal cysts, predict malignancy risk and guide treatment (Table 1).[4–7] Contrast-enhanced CT (CECT) is helpful when uncertainty exists regarding the classification of a non-contrast CT. Bosniak I (simple) and Bosniak II cysts are benign and do not require further follow-up.[7] Bosniak IIF cysts harbour a 5–10% risk of malignancy, and 12% of them progress to Bosniak III/IV cysts during follow-up.[7,8] Patients with Bosniak IIF cysts should undergo active surveillance (AS) with six-monthly scans initially, extending up to 12-monthly for a total of five years. The modality of scans is dependent on tumour location, body habitus, radiation exposure and renal function. Patients with stable Bosniak IIF cysts can undergo surveillance by general practitioners (GPs). Triggers for re-referral to a urologist are increase in size, symptoms or concerns of metastatic disease. Bosniak III and IV cysts harbour a higher risk of malignancy (50% and 90–100%, respectively) and should be referred to a urologist with a category 2 classification (seen within 90 days) if the lesion is <4cm. A category 1 (seen within 30 days) referral should be made if the lesion is >4 cm or if there are concerns of metastatic disease. Although AS of Bosniak III might be an option,

the decision should be made by a urologist in association with a multidisciplinary team.

Angiomyolipomas

Angiomyolipomas (AML) are benign mesenchymal neoplasms.[9] Oestrogen contributes to their development; therefore, their predilection is towards females. AMLs are often asymptomatic but could present with pain or haemorrhagic complications. Risk factors for haemorrhage include tumour size, vascularity and periods of elevated oestrogen (pregnancy or exogenous hormone). Asymptomatic AML <2 cm might not require follow-up due to the low risk of haemorrhage (Figure 1). AML between 2 and 4 cm can be considered for AS. Currently, there is no consensus on the ideal AS protocol. A common approach involves ultrasound every 6–12 months for up to two years and to consider discharge if stable.[10] AS over a two-year duration appears to provide the same benefit as follow-up over five years.[10] Frequency and duration of AS should be tailored to patients' comorbidities, age and risk of bleeding. Patients with a stable AML can undergo surveillance by GPs, and a non-urgent urology referral can be made to clarify follow-up. Treatment should be considered if patients experience pain, recurrent haemorrhage or are at risk of haemorrhage (AML >4 cm or progressive in size or the patient is planning for pregnancy).[6,11] Treatment options include selective arterial embolisation (SAE) or nephron-sparing surgery (NSS), such as partial nephrectomy. SAE is less invasive but associated with higher re-treatment rates compared to surgery (31% vs 0.85%).[11] AML in a woman of childbearing age or ≥4 cm can be referred to a urologist as category 2.

Oncocytoma

Oncocytomas are benign and present in the sixth to seventh decade of life.[12] Oncocytomas are radiologically challenging to differentiate from renal cell carcinomas (RCCs) as they present as solid masses within the renal cortex. CT and magnetic resonance imaging (MRI) have limited utility for diagnosis, and histopathological confirmation is preferred. Any solid renal mass should be referred to a urologist as category 2 if <4 cm or category 1 if >4 cm. Treatment options include surgery or AS in selected biopsy-proven oncocytoma because of the risk of harbouring chromophobe RCC.[6,13] AS consists of 6- to 12-monthly scans with either CT or ultrasound.[14] A clear AS plan should be obtained from a urologist as oncocytomas have a median growth of 1.5 mm/year.

Renal cell carcinoma

The incidence of RCC has been increasing. Risk factors include tobacco use, obesity and family history of RCC.[15,16] Most RCCs are asymptomatic and incidental; the triad of flank pain, macroscopic haematuria and palpable abdominal mass is now rare due to earlier detection.[17,18] Referral to a urologist is warranted: as category 2 if <4 cm or as

Table 1. Simplified summary of Bosniak classification, version 2019[4–6,8]

	Class	Estimated risk of malignancy (%)	Radiological features	Recommendations
Simple	I	0	Thin, smooth wall (≤2 mm), might enhance, no septa, no calcification, no solid component	No follow-up
Complex	II	0	Thin (≤2 mm) and few smooth septa (1–3) ± calcification or Hyperattenuating (HU>20), non-enhancing, small (≤3 cm) and homogeneous	No follow-up
	IIF	5–10	Many (≥4), thin (≤2 mm) septa that must enhance or Minimally thickened (3 mm) septa that must enhance or Cystic masses at MRI that are heterogeneously hyperintense on fat-saturated unenhanced T1-weighted imaging that do not meet criteria for Bosniak III or Bosniak IV	Surveillance (US or CT, initially 6-monthly, extending up to 12-monthly for a total of 5 years) Referral to urologist if symptomatic, unclear diagnosis, or concerning of metastasis
	III	50	≥1 thick (≥4 mm) or irregular (≤3 mm focal or diffuse protrusion with obtuse margin with wall or septa) enhancing walls or septa without nodular enhancement	Referral to urologist for consideration of surgery or active surveillance in selected cases[A]
	IV	90–100%	≥1 enhancing nodule (focal enhancing convex protrusion of any size that has acute margins with the wall or septa or a focal enhancing convex protrusion ≥4 mm)	Referral to urologist for consideration of surgery

Radiological features are based on contrast-enhanced CT unless specified otherwise.

[A]The decision for active surveillance should be made by a urologist and associated multidisciplinary team.

CT, computed tomography; HU, Hounsfield unit; US, ultrasound.

category 1 if >4 cm or there are concerns of metastatic disease. The decision for renal biopsy should be made by a urologist and is reserved for patients planned for ablative therapy or if diagnostic uncertainty remains. Renal biopsies are avoided for a cystic mass due to lower diagnostic yield.[6] Treatment options for localised RCC include AS, surgery, ablation or watchful waiting (WW). AS is reserved for patients with good life expectancy and renal mass <4 cm, as risk of metastasis is only 1.8% at this size.[19] CT is preferred and performed initially six-monthly extending to 12-monthly if stable. Surgery is performed minimally invasively (laparoscopic or robotic) and NSS (partial nephrectomy) whenever possible. Ablation is typically reserved for smaller lesions <3–4 cm. Ablation could be considered based on patient preference or in patients with good life expectancy but not suitable for surgery. Ablation has low complication rates, but data is lacking on long-term oncological outcomes compared to surgery.[20] WW is reserved for comorbid patients with limited life expectancy where the risks of active treatment outweigh the benefits. Patients are followed up symptomatically and follow-up imaging might not be required.

Approach to incidental adrenal incidentaloma

Adrenal incidentaloma can be categorised as benign nonfunctional, benign hyperfunctional or malignant (Figure 2). The initial diagnostic consideration is whether the adrenal lesion is unilateral or bilateral. Bilateral adrenal lesions account

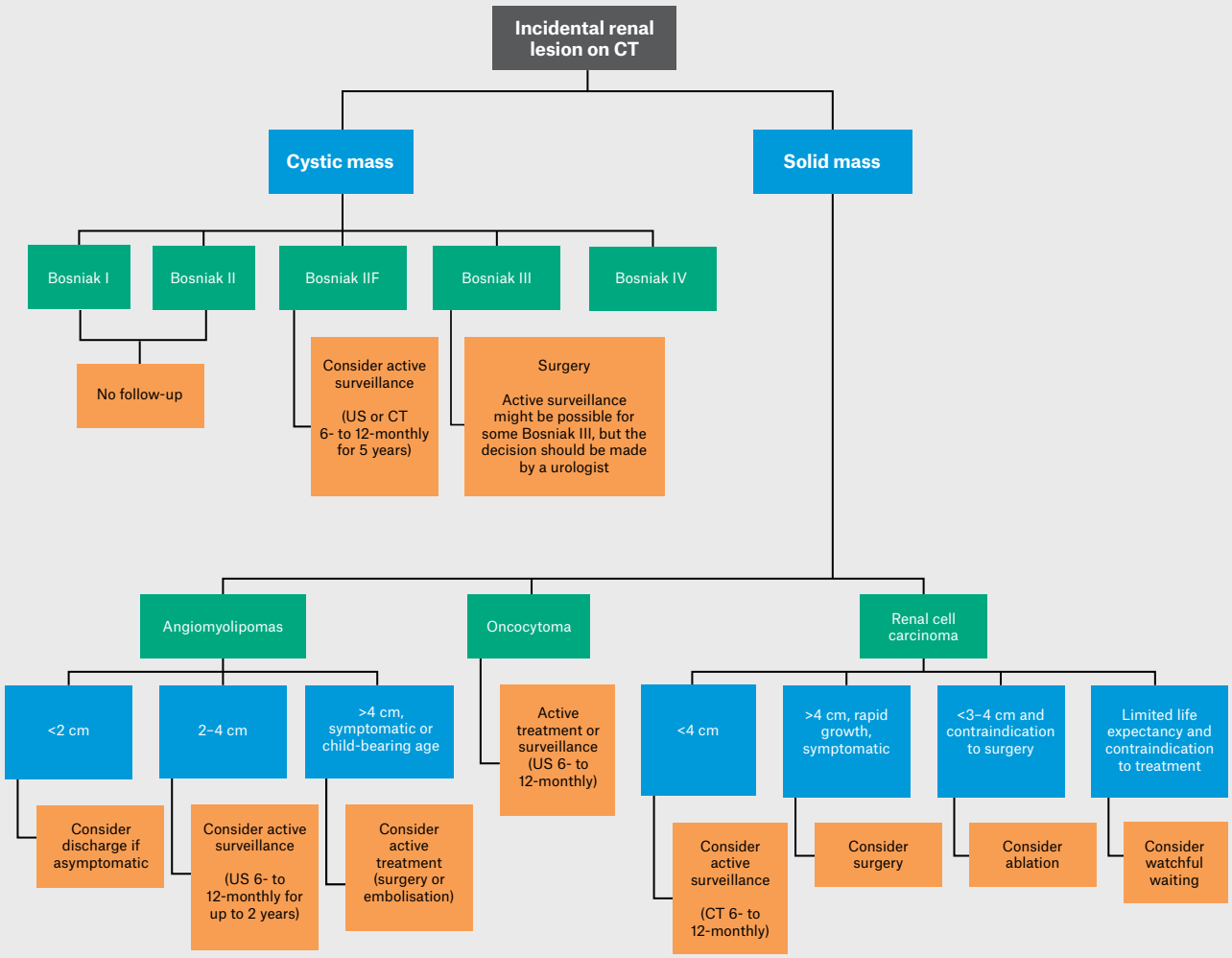

Figure 1. Simplified diagnostic algorithm for incidental renal lesion on CT.
CT, computed tomography; US, ultrasound.

for 10% of cases and can be associated with benign adenomas, infections (eg fungal or tuberculosis), congenital adrenal hyperplasia, lymphoma or metastatic disease.[21] Some primary malignancies (eg of lung, colorectal, breast, pancreas or renal origin) might metastasise to either unilateral or bilateral adrenal glands. However, the presence of non-adrenal primary malignancies does not rule out benign adrenal adenomas.

The second consideration is whether the lesion is functional. Benign nonfunctional adenomas account for 80% of adrenal incidentalomas. Functional adrenal lesions are mostly benign and are made up of 5% phaeochromocytoma (PCC), 5% cortisol secreting and 1% aldosterone secreting.[22] The remaining consist of primary malignancy and adrenal metastasis.

PCCs secrete adrenaline and noradrenaline. Patients with PCCs can present with palpitation, headaches, tremors, sweating, weight loss and hypertension. This could be screened with plasma or urinary metanephrines.[23] Cortisol-secreting adrenal mass results in Cushing's syndrome, characterised by moon face, buffalo hump, central obesity, hypertension and diabetes. Screening can be conducted with a 1 mg overnight dexamethasone suppression test and confirmed with an 8 mg dexamethasone suppression test or midnight salivary cortisol.[23] An aldosterone-secreting adrenal mass will manifest as Conn's syndrome, which is often asymptomatic but can present with refractory hypertension or hypokalaemia. This could be screened with plasma aldosterone to plasma renin ratio and confirmed with 24-hour urinary

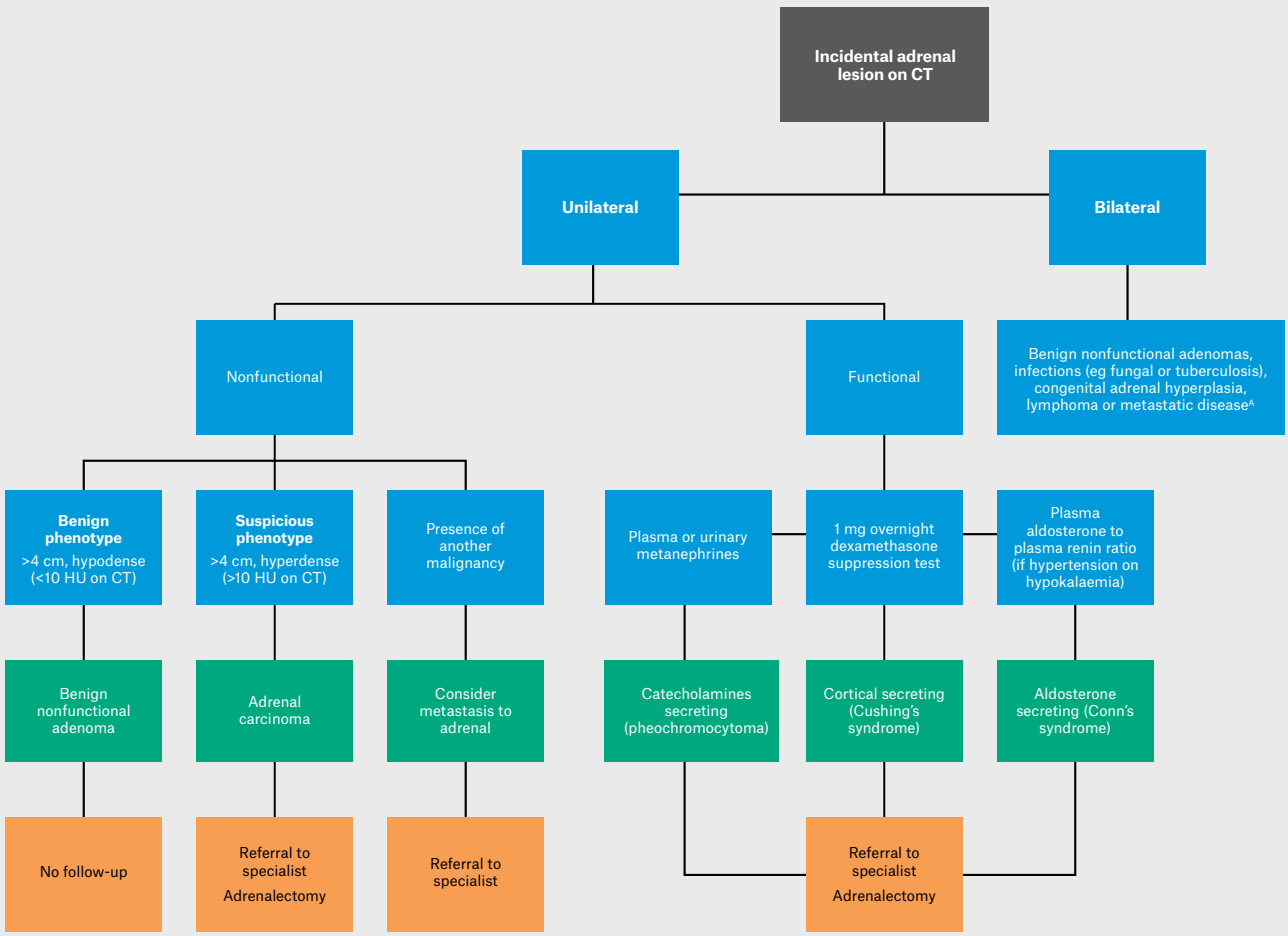

Figure 2. Simplified diagnostic algorithm for incidental adrenal lesions on CT.
[A]Metastatic disease can be either unilateral or bilateral.
CT, computed tomography; HU, Hounsfield unit; US, ultrasound.

aldosterone.[22,23] In the primary care setting, all patients with adrenal incidentaloma should undergo clinical assessment for signs and symptoms of hormone excess. If hyperfunctioning adrenal lesion is suspected, GPs can screen with the 1 mg overnight dexamethasone suppression test and plasma or urinary metanephrines. Screening with the plasma aldosterone-to-plasma renin ratio should be reserved for patients with hypertension or unexplained hypokalaemia. Confirmatory tests should be deferred to a specialist (eg 8 mg dexamethasone suppression test or midnight salivary cortisol). All functional adrenal incidentalomas should be referred for specialist review and classified as category 1.[24] Medical management is performed by an endocrinologist, and adrenalectomy is typically performed by an endocrine or urology surgeon.

Finally, benign adrenal masses exhibit three radiological features: they are <4 cm in size, homogeneous and hypodense (<10 Hounsfield unit [HU]). Risk of adrenal malignancy increases with size: the risk is 5% for masses <4 cm, 10% for those 4–6 cm and 25% for those >6 cm.[23] Benign adrenal adenomas contain intracytoplasmic fat and appear hypodense on non-contrast CT.[25] If non-contrast CT is consistent with a benign adrenal adenoma, no further imaging is required. However, up to 30% of adenomas are lipid poor and might not appear hypodense. In these situations, a CECT with adrenal protocol might be helpful. This involves a delayed-phase CECT to determine the rate of contrast washout.[26] Both benign adenomas and carcinomas enhance rapidly with contrast, but benign adenomas typically exhibit a faster contrast washout compared to carcinomas. MRI is comparable to non-contrast CT for identifying intracytoplasmic fat; however, it is comparable to washout CT scans.[22] Specialist referral is recommended if the diagnosis remains unclear or if there are concerns of adrenal malignancy. Benign adrenal adenomas do not require follow-up or specialist referrals if <4 cm.

Conclusion

If a renal incidentaloma is found on an ultrasound or non-contrast CT and the diagnosis is unclear, consider a CECT. Bosniak I and II renal cysts do not require further imaging. Non-contrast CT might be sufficient to exclude a malignant adrenal lesion. CECT with adrenal protocol can be helpful if the diagnosis is unclear.

Key points

- Renal cysts should be characterised using the Bosniak classification.
- Bosniak I (simple) and Bosniak II cysts do not require further work-up or treatment.
- Solid renal masses should be referred for urological opinion; urgency will depend on size and symptoms.
- Adrenal incidentalomas should undergo functional assessment, which includes clinical evaluation, and, if necessary, endocrinological screening tests.
- If non-contrast CT of adrenal incidentaloma have benign features (ie <4 cm, homogenous, and hypodense [HU<10]), no further imaging or follow-up is required.

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Conservative management of patients with end-stage chronic limb-threatening ischaemia in the community

Kay Hon, Madeleine Bain, Georgina Minns, Eliza Colley, Robert Fitridge

Background
Chronic limb-threatening ischaemia (CLTI) is an advanced and severe form of lower limb peripheral arterial disease (PAD) and can pose significant challenges in clinical management. Not all patients are able to undergo surgical intervention due to patient-related and disease-related factors.

Objective
This review article aims to provide general practitioners with an overview of conservative management of patients with end-stage CLTI in the community.

Discussion
The review aims to provide an overview of end-stage CLTI and approaches that are required to preserve patients' quality of life. It outlines symptom control, wound care, psychosocial support and end-of-life considerations to preserve the quality of life for patients facing this challenging condition.

PERIPHERAL ARTERIAL DISEASE (PAD) has been reported to affect up to 10% of patients in Australian primary healthcare settings.[1,2] Chronic limb-threatening ischaemia (CLTI) represents the advanced progression of PAD where patients experience ischaemic rest pain and/or tissue loss in the form of gangrene or ulceration.[3] The prognosis for patients with CLTI is poor, with a mortality risk of least 20% in one year, and over 60% in five years.[4–6] At one year, at least 30% of the patients would have undergone amputation.[7]

In order to avoid major amputation and manage ischaemic symptoms, patients with CLTI require revascularisation. Revascularisation surgery (open or endovascular) for patients with CLTI might be limited by patient-related factors such as significant comorbidities contributing to high surgical and anaesthetic risk, or disease-related factors such as multi-level atherosclerotic disease or extensive tissue loss making it technically not feasible for revascularisation. Despite surgical advances, some patients might have no revascularisation options or are unfit to undergo surgery. Even patients who have undergone revascularisation might not be pain free, or the tissue loss might be too extensive to recover. This subset of patients is known to have end-stage CLTI. The term 'no-option' CLTI is sometimes used, but it inaccurately suggests that treatment options are completely absent when in reality, there are still options to improve symptoms and quality of life.

For patients with end-stage CLTI, most interventions are aimed at symptom relief or to avoid major amputation. Patients remain at high risk of disease progression despite surgical intervention. Hence, end-stage CLTI should be considered a life-limiting albeit non-malignant condition requiring mixed models of care, where active treatment and palliative care is provided until either end of life or major amputation.[8]

The article offers a comprehensive review of strategies to address symptoms for patients experiencing end-stage CLTI who are not candidates for surgical revascularisation (open or endovascular) and are for conservative management only.

Symptoms of CLTI

Patients with end-stage CLTI present with progressive or longstanding severe rest pain or tissue loss in the form of gangrene or ulceration longer than two weeks. The primary goals of management are to maintain their quality of life by managing pain, providing good wound care to prevent further deterioration of ulceration and ideally aim to heal wounds, while also providing psychosocial support.

Management of end-stage CLTI

The management of end-stage CLTI requires a multidisciplinary team to create an individualised management approach for the patient. Each patient's symptoms and the impact on their quality of life will present in different ways and will evolve over time, so management should adapt to these changes. Their comorbidities, functional status, social and financial circumstances, and access to specialised healthcare and rurality should be considered when formulating their management.

General consideration

For all patients, simple measures should be maximised, and risk factors optimised to help control symptoms and curb progression. As part of their assessment, risk factors including smoking, hypertension, diabetes and hyperlipidaemia should be identified and addressed as part of patient management. Exercise can be limited by claudication, frailty or previous amputations, but should be encouraged to promote collateralisation and maintain mobility. Pressure area care, appropriate footwear and bedding are key to protecting wounds and preventing formation of new ulcers, particularly in those with reduced sensation. Limitations to a patient's mobility from pain or amputation might affect their need for additional support or aids at home and referral to community or allied health services.

Adequate nutritional intake is needed to support wound healing and improve overall health. There is a high prevalence of malnutrition and undernutrition being reported in patients with CLTI.[9] Apart from encouraging healthy diets, screening and replacement of deficient micronutrients known to improve wound healing such as vitamins A, B, C and D, and zinc should be considered.[10]

Wound care

Patients with end-stage CLTI often have tissue loss requiring ongoing wound care in the community, which can be managed via community nursing services or wound clinics. The patient should be counselled that without adequate perfusion, it is challenging for a wound to heal. However, there has been a study showing wound healing with conservative management despite the presence of severe CLTI (and very low perfusion).[11]

Wound care for patients with end-stage CLTI should be person- and symptom-specific, with the goal of reducing discomfort from the wound and addressing any negative aspects of the wound such as itch, exudate, odour, bleeding and pain that increase discomfort (Table 1).[12] Vascular wound nurse advice can be accessed by the general practitioner (GP) or directly from community nurses if there are any concerns with the dressing regimen or alternative recommendation (intolerance, cheaper alternative).

Adjunct therapies to wound healing

Appropriate wound care remains the priority for patients with ulceration due to CLTI; however, there are adjunct therapies with limited evidence of promotion of wound healing and amputation prevention that might be considered (Table 2).

Access to wound dressings and associated costs

In the community, wound care can be undertaken by community nurses or by attending general practice clinics. Unfortunately, there is limited Medicare funding for the management of chronic wounds, with no specific Medicare Benefit Schedule (MBS) item for consumables used in wound dressing. Historically, this cost would be borne by the patients and general practices. Silver-based dressings are particularly costly ($292 for five pieces of 10x10 cm dressing). For patients on home care packages, this extra cost affects the availability of funds for other essential services; hence, it is important to balance the clinical benefits with cost considerations to help mitigate the financial impact of wound care.

The incoming Chronic Wound Consumable Scheme (CWCS) is due to commence in Australia in mid-2025 and is designed to subsidise the expenses associated with wound consumables for eligible patients. This funding scheme will hopefully reduce the financial burden on eligible patients aged >65 years (or >50 years for Aboriginal or Torres Strait Islander Australians) as well as service providers.

Pain management

Pain is one of the most challenging symptoms for patients with end-stage CLTI and the impact on quality of life is comparable to that of advanced cancer patients.[13] Management of pain relating to CLTI is challenging due to the complex underlying pain pathophysiology and coexisting comorbidities limiting therapeutic options. Additionally, many patients also have other underlying pathologies such as arthritis or venous insufficiency, which also contribute to pain. There should be realistic expectations of treatment, and the most appropriate therapy should be a personalised approach balancing efficacy and side effects. For those with complex or difficult-to-manage pain, palliative care or chronic pain services can be involved to guide management as it might be a life-long issue.

Pain associated with CLTI is often of mixed nociceptive, inflammatory and neuropathic origin, with complex interacting mechanisms making management of pain in CLTI challenging (Figure 1).[14] Management generally requires multi-modal analgesia such as non-steroidal anti-inflammatory drugs (NSAIDs), anti-neuropathic agents and opioid therapy in conjunction with non-pharmacological pain management (Tables 3 and 4).

There has been increased interest in the use of cannabis in management of chronic pain; however, the role of cannabis in patients with end-stage CLTI is still to be determined, in particular noting that long-term cannabis users have an associated higher risk of PAD and incidence of acute limb ischaemia.[15,16]

Vasomodulating therapies

In addition to or when pharmacological analgesic regimens remain inadequate, pharmacological or interventional therapies aiming to promote vasodilation can be considered with input from specialists (Table 5). Evidence for these interventions is limited.

If patients' symptoms cannot be effectively managed in the community, it would be appropriate to refer them back to a vascular surgeon. This would allow for the evaluation

Table 1. Common wound-specific concerns for patients with end-stage chronic limb-threatening ischaemia

Concerns	Recommendation
Pruritus	• Wound itch can be due to significant unviable tissue burden leading to scratching, overgrowth of granulation tissue, maceration of wound edges, irritation from exudate, dermatitis or wound infection • Encourage good skin care with hypoallergenic wash and moisturiser and avoid using hot water for cleaning • Assess for contact dermatitis and eliminate allergens if identified. Consider a short course of topical corticosteroid (eg hydrocortisone) if evidence of ongoing dermatitis
Exudate and transudate	• Inflammatory response in chronic wound or infection can lead to significant exudate, which can damage peri-wound tissue and contribute to significant discomfort • Assess for evidence of infection and manage as required • The dressing should be absorbent and non-adherent to the wound. Depending on available resources, some dressing options are: – Foam dressing such as Allevyn™ (Smith and Nephew, Watford, England), Lyofoam™ (Mölnlycke Health Care, Gotherburg, Sweden) – Pad dressing such as Zetuvit® (Paul Hartmann Ltd, Heidenheim, Germany), combine dressing – Hydrofiber dressings such as Aquacel® (ConvaTec, London, England) – Hydroactive dressings such as Biatain™ (Coloplast, Humlebaek, Denmark) • Advanced wound dressings such as negative pressure wound therapy (such as V.A.C® [KCI Licensing, Inc., San Antonio, USA] can sometimes be used to assist with exudate management and can assist with wound closure (particularly after surgical debridement). However, this should not be used for wounds with necrotic tissue or eschar • Dressings for exudate management should aim at maintaining a moist wound environment while preventing excess exudate from coming into contact with surrounding skin as this leads to maceration and deterioration of the wound • Barrier creams to protect wound edges and frequent dressing changes can prevent oversaturation
Odour	• Malodorous wounds in CLTI can be due to presence of necrotic tissue, colonisation of bacteria that release compounds (cadaverine and putrescine), infection or large volume exudate • Assess for evidence of infection in the first instance • Maintain good wound hygiene • Consider debriding devitalised tissue to prevent further bacteria colonisation • Managing exudates with appropriate dressing and consider dressing that contains charcoal or carbon that might help to reduce odour
Gangrene, tissue loss and auto-amputation	• Necrotic, devitalised tissues provide an optimal environment for bacterial growth • Antiseptic products such as topical povidone-iodine; Inadine™ (Systagenix, Gargrave, UK) should be used to dry out gangrene and reduce bacterial burden • In some cases of expected auto-amputation, patients and carers must be aware to monitor for the occurrence
Pain	• Chronic ischaemic wounds are often painful, which can be exacerbated by dressing changes. Pain with dressing changes is commonly due to dressings drying out and adhering to the wound base causing pain on removal. The wound bed might be packed too tightly or the physical act of wound cleaning might also cause pain. Pain can be minimised by: – considering oral analgesia prior to dressing change. This will need to be prescribed with medication authority provided to community nurses – minimising the frequency of dressing changes where possible • For highly exudative wounds requiring frequent dressing changes, apply non-adherent dressing (eg Mepitel® [Mölnlycke Health Care, Gotherburg, Sweden]; Atrauman® [Paul Hartmann Ltd, Heidenheim, Germany], Jelonet [Smith and Nephew, Watford, England] as the primary dressing to the wound base, and only change the secondary absorbent dressing without the need to disturb the wound base • Consider lidocaine-soaked gauze or topical lidocaine jelly to assist with painful dressing removal. This will need to be prescribed with medication authority provided to community nurses

Table continued on the next page

Table 1. Common wound-specific concerns for patients with end-stage chronic limb-threatening ischaemia (cont'd)

Concerns	Recommendation
Fragile skin and pressure area, incidental injury	• Patients with end-stage CLTI often have limited mobility and are at risk of developing pressure ulcers. Poor perfusion puts these pressure ulcers at high risk of becoming non-healing wounds. For leg or foot pressure care, consider: – prevention of pressure area with a protective boot such as Z-flex heel boot at rest – referral to a podiatrist for regular foot care and footwear advice to prevent new wounds due to poorly fitting footwear. Consider the use of a general practitioner management plan to document the patient's individualised goals, coordinate the patient's multidisciplinary care with team care arrangement, and assist the patient with the cost of allied health appointments • Patients with restricted mobility who require a wheelchair or who are unable to leave bed will be at risk of developing sacral and heel pressure sores. They might also be at risk of incidental injury during transfer, or from dressing application or removal. A comprehensive wound management plan should ensure that appropriate equipment is in place to prevent injury, and that high-risk areas are monitored regularly. This might require a team including occupational therapists, physiotherapists, wound care nurses and carers

CLTI, chronic limb-threatening ischaemia.

Table 2. Adjunctive therapies to wound healing

Hyperbaric therapy	• Hyperbaric therapy involves the administration of a high concentration of pure oxygen to a patient in a sealed pressurised oxygen chamber (hyperbaric chamber) to increase the amount of oxygen dissolved in the bloodstream • This aims to promote wound healing by enhancing tissue oxygenation, reducing inflammation and swelling, promoting angiogenesis and inhibiting bacterial growth[3] • The role of hyperbaric therapy is adjunctive in wound healing and does not prevent major limb amputation[3] • Non-medical hyperbaric therapy services exist in Australia; however, given the comorbidities of the patients with CLTI, patients should only be referred to medically supervised hyperbaric therapy providers • Referrals can be made by the general practitioner to the hyperbaric medicine unit in each state. There is at least one public hyperbaric medicine unit within each state
Topical oxygen	• Topical oxygen can be administered directly to the wound using small pressurised medical devices purchased by the patient from a local distributor • It can be delivered via a specialised interface (dressings) or as a chamber over the extremity • The International Working Group for Diabetic Foot (IWGDF) guidelines recommend it to be used only if standard care has failed and resources are available to provide this intervention[22]
Biologic and regenerative medicine therapies	• Biologic and regenerative medicine therapy consists of gene therapy or stem cell therapy that aims to promote growth of new blood vessels (angiogenesis) in the extremities • Preliminary safety and efficacy trial findings are promising for angiogenic therapies; however, efficacy has not yet been demonstrated in phase 3 trials

CLTI, chronic limb-threatening ischaemia.

of their condition, adjustment of pain management strategies and wound care regimen as necessary, and consideration of major limb amputation for those who might consider it.

Major limb amputation

> *The need for amputation indicates the end of a limb and possibly the end of life.*[8]

For patients who have intractable pain or a life-threatening infection, or who find long-term dressings intolerable, or the risk of complex revacularisation outweighs the benefit, major limb amputation (MLA) can be considered. Minor amputations (toes or forefoot amputations) are often futile given that without adequate perfusion, patients will likely experience ongoing pain and leave behind a larger non-healing wound. Nevertheless, some patients unwilling to accept long-term wound care or major amputation might require staged amputation as a physical or psychological bridge to major amputation.

The goal of MLA is to preserve quality of life and relieve symptoms due to CLTI; however, this is not without the risk of negatively impacting patients' quantity and quality of life. It has been reported that more than 50% of patients who have undergone major limb amputation due to CLTI will die within five years.[4,17] Mortality rates of up to 85% within five years have been reported in

no-option CLTI elderly amputees.[18,19] This has also been observed in a local study in New South Wales (NSW), Australia, where 28.3% of patients died two years following discharge from their primary CLTI procedures.[20] Nevertheless, for patients who have good rehabilitation prospects, this might allow them to achieve an improved functional outcome. Multidisciplinary pre-amputation counselling is often helpful in these settings.

Patients will need to be counselled on the risks associated with surgery, including but not limited to perioperative cardiovascular events, reduced independence, phantom limb pain and risk of non-healing of amputation stumps. The psychological effect of MLA and its impact to their functional status needs to be considered by patients and care providers, and additional supports or aids might be required to assist them at home afterwards.

Psychosocial support

Living with severe CLTI can be mentally and emotionally challenging for patients and their carer(s). It is the key to caring for patients with end-stage CLTI to have a good therapeutic relationship with a regular GP who has a thorough understanding of their goals and is able to coordinate their multidisciplinary care. Psychosocial support via counselling and referral to support groups or therapists familiar with care of patients with end-stage CLTI should be considered. Encourage carers to make regular appointments to ensure that their own psychosocial wellbeing and physical health are attended to as well.

Palliative approach to end-stage CLTI

CLTI is a progressive disease and prognostication is difficult. Patients with untreatable, no-option end-stage CLTI will deteriorate slowly over a period of time, or sometimes acutely due to wound infections, or due to other underlying comorbidities. Despite being palliative from a CLTI perspective, many patients with end-stage CLTI wish to pursue active treatment for their other medical conditions as they still maintain reasonable quality of life. Ongoing advanced care planning should occur.

Some patients might wish to pursue long-term suppressive antibiotics for infected wounds, acknowledging that it would be a temporising measure, and might lead to antibiotic resistance. It is encouraged for patients and families to have discussions about goals of care and advanced care planning in the event that patients deteriorate acutely. When appropriate, patients with end-stage CLTI should be linked in with palliative care services to ensure that their needs are being addressed.

If pain remains unmanageable despite the discussed management options, referral to chronic pain or palliative care services would be warranted. Early referral to these services (if available) is wise as CLTI pain will usually never resolve.

Social and cultural factors

Access to specialised healthcare and allied health input will be affected by the patient's social circumstances and their location. Patients with CLTI typically have increased care needs in the community. For patients who are aged >65 years, comprehensive aged

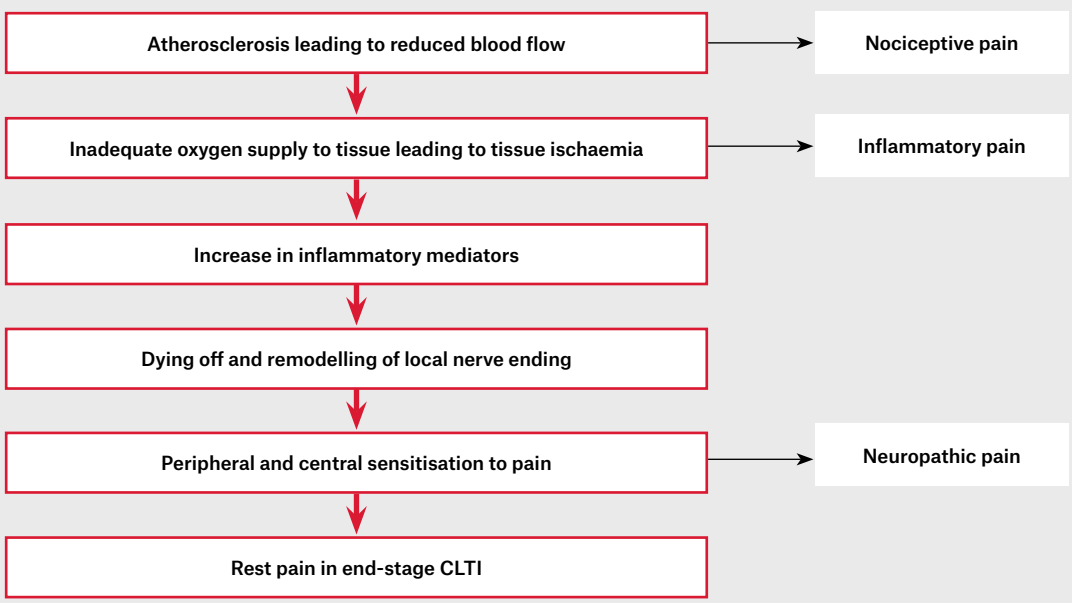

Figure 1. Physiology of pain in CLTI.[14]
CLTI, chronic limb-threatening ischaemia.

Table 3. Non-pharmacological pain management

General advice	• Some patients might find relief by positioning their legs in a dependent position (hanging their legs down over the end of the bed or allowing them to hang freely while seated) • This position helps improve blood flow by taking the advantage of gravity; however, this might not be suitable for patients who are at risk of developing dependent oedema, which will contribute to worsening pain • Patients might benefit from OT assessment for home care equipment such as recliner chairs or medical beds that can be adjusted to the position that can alleviate pain
Compression therapy	• For patients who are experiencing pain due to oedema, gentle low level compression therapy might be considered to improve venous return and reduce swelling in the affecting limb • Compression therapy in this patient cohort requires careful consideration due to the risks of further compromising arterial blood flow • Toe pressures and ABI should be performed prior to commencement of compression therapy and can be organised via an outpatient vascular clinic or radiology services with a vascular sonographer • Tubigrip (elastic bandage) can be considered for patients with toe pressures <40 mmHg and not suitable for more aggressive compression therapy • Intermittent pneumatic compression can also be considered (refer to vasomodulating therapy in Table 5) • Additionally, close monitoring is required to detect any complications promptly, especially at the initiation of compression therapy
CBT	• CBT has not been widely studied in the management of pain in CLTI; however, it has an established role in the management of chronic pain[23] • Integrating CBT into the treatment plan can be considered to minimise psychological factors that could further aggravate pain and improve adherence to medical treatment
Complementary therapies	• A variety of complementary therapies, such as meditation, yoga, music therapy, guided imagery and biofeedback, are frequently employed for chronic pain patients. Their efficacy in managing pain for individuals with CLTI lacks comprehensive research to advocate for their standard implementation • Nonetheless, patients might reasonably contemplate these modalities to enhance their psychological welfare

ABI, ankle-brachial index; CBT, cognitive behavioural therapy; CLTI, chronic limb-threatening ischaemia; OT, occupational therapist.

care assessment should commence early to ensure that patients receive adequate funding and the support they require to remain at home or should they require residential aged care. For younger patients, the National Disability Insurance Scheme (NDIS) can be considered to address these increased care needs. In rural or remote areas, there might be limited allied health, outpatient wound clinics or dressings available for patients, especially in community settings. Patients with limited mobility as a result of CLTI depend on family, friends or other services to attend appointments. This poses a challenge for those requiring regular dressing changes, as the frequency of appointments might be influenced by how often their transport is available. In all regions, the costs of expensive frequent dressings and poorly rebated regular appointments pose significant financial challenges for both the patient, the GP and the clinic/practice.

Healthcare practitioners need to consider a patient's cultural and spiritual beliefs when managing CLTI. An open and culturally sensitive discussion is key, and should consider involving a suitable cultural liaison if appropriate and available. In Australia, First Nations people are more likely to have symptomatic CLTI and have higher rates of limb loss and major adverse cardiovascular events.[21] Discussions regarding amputation should be sensitive to the spiritual connection between body and soul. Spiritual grief and loss can follow amputation as this connection is disrupted, and it is not uncommon for requests for amputated limbs to be preserved.

Conclusion

Management of end-stage CLTI is complex and GPs play a vital role in facilitating a patient-centred approach to their care, even in the absence of curative treatment. It involves a multidisciplinary approach and management should adapt to progressive symptoms over time.

Key points

- CLTI is a progressive disease where not all patients are suitable for revascularisation.
- Patients with end-stage CLTI have variable levels of symptom burden and impact on quality of life that requires individualised care in the community.
- Management requires a multidisciplinary team including community nurses, GPs and allied health services, with input from specialised vascular wound nurses (Figure 2).
- Pain management in patients with end-stage CLTI can be complex, and will require a multi-modal analgesic regimen and optimisation of non-pharmacological measures.
- Clear communication with patients and carers is important to manage expectations.

Table 4. Pharmacological analgesia regimen for patients with end-stage CLTI

Non-opioid agents • Paracetamol	• These should be first-line medication prescribed to all patients if there is no contraindication[3] • It is crucial to discuss with patients the importance of having regular paracetamol for its opiate-sparing activity that might reduce adverse events and risk associated with high doses of opioids
Non-opioid agents • NSAIDs	• Despite the inflammatory component of CLTI, prescribing NSAIDs must be undertaken with caution for this patient population • Prescribing of NSAIDS requires close monitoring and only for a short period of time as they are known to be associated with gastrointestinal, cardiovascular and renal adverse events • Patients with CLTI are often on ACE-I and aspirin for which co-prescription of NSAIDs could reduce their therapeutic effects while increasing risks of side effects • The PRECISION trial demonstrated that a moderate dose of a COX-2 inhibitor, Celecoxib 200 mg, has lower rates of adverse events when compared to non-selective agents such as ibuprofen.[24]
Adjuvant agents • Anti-neuropathic agents such as gabapentinoids (pregabalin, gabapentin), antidepressants (amitriptyline, duloxetine)	• Patients with CLTI might experience significant neuropathic pain due to the neural damage from chronic ischaemia, in addition to other co-existing neurological damage such as diabetic neuropathy[14] • Agents to reduce sensitisation due to nerve damage such as antidepressants (serotonin and noradrenaline reuptake inhibitors and tricyclic antidepressants, but not selective serotonin reuptake inhibitors) and gabapentinoids can be considered[25] • Both gabapentin and pregabalin have been studied and shown to have a role in pain management, reduce opioid use and side effects in patients with CLTI • Anti-neuropathic agents need to be prescribed with care for elderly patients, patients with renal failure or heart failure as it can contribute to peripheral oedema and worsening wound exudate[26]
Conventional opioids (immediate and extended release) • Oxycodone • Morphine • Fentanyl	• The use of opioids should be avoided where possible. When used in isolation, they are rarely effective for ischaemic pain • Studies have shown that opioids might diminish immune activation, reduce tissue oxygenation and angiogenesis and hence hinder wound healing[27] • Doses should be titrated to effects, and are available in short-acting and slow-release formulations • There is a risk of opioid-induced hyperalgesia, abuse and dependence[18] • Use is also limited by renal and hepatic comorbidities in patients with end-stage CLTI • Therefore, prescription of opioid in this patient population must involve thorough assessment of risks against the benefits
Atypical opioids • Buprenorphine • Tramadol • Tapentadol	• If opioid analgesia is deemed required, consider prescribing atypical opioids • Atypical opioids have demonstrated better efficacy in the treatment of ischaemic pain when compared with conventional opioids[28] • Buprenorphine and tapentadol are noted to have better side effect profiles and are better tolerated in older patients compared to tramadol and conventional opioids

ACE-I, angiotensin-converting enzyme inhibitors; CLTI, chronic limb-threatening ischaemia; COX-2, cyclooxygenase-2; NSAIDs, non-steroidal anti-inflammatory drugs; PRECISION, Prospective Randomised Evaluation of Celecoxib Integrated Safety Versus Ibuprofen or Naproxen.

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Table 5. Pharmacological and interventional vasomodulating therapy

CLS	• CLS decreases pain by denervating lumbar sympathetic ganglia, which interrupts the pain pathway and neurolyse the nociceptive fibre[3] • It also leads to vasodilation of the collateral circulation and shunting of blood through cutaneous arteriovenous anastomoses, which in turn assists with pain relief by improving tissue oxygenation • These effects might be short-lived; however, due to the minimally invasive nature, CLS can be repeated[29] • This is commonly performed by interventional radiologists
SCS	• Electrodes implanted into the lumbar epidural space are used to stimulate sensory fibres, which leads to vasodilation and improves microcirculation. It also suppresses sympathetic vasoconstriction and pain transmission[3] • Spinal cord stimulators are being trialled, but its future in the management of end-stage CLTI remains uncertain at this stage and is greatly limited by cost, accessibility and complications • Spinal cord stimulators are normally implanted by a neurosurgeon
IPC	• IPC involves the application of external pressure to the lower limbs using inflatable cuffs or sleeves, which intermittently inflate and deflate • It promotes blood flow by increasing the arteriovenous pressure gradient and reversing vasomotor paralysis. It also promotes the release of nitric oxide, a potent vasodilator • It is also effective in oedema management • IPC can be hired via a lymphoedema clinic, vascular clinic or a medical equipment hire company
Prostanoids such as prostaglandin E1 (Alprostadil), prostacyclin and iloprost	• Prostanoids promote vasodilation via anti-thrombotic and profibrinolytic activities • Prostanoids can be used selectively with input from vascular specialists for patients in whom revascularisation is not possible or has failed • Agents such as iloprost require a 5-day, 6 hours per day intravenous infusion with strict monitoring as it is known to be associated with common side effects and should be used with caution for patients with ischaemic heart disease or heart failure • There is limited evidence to support its routine use for rest pain relief[30]
Vasoactive medications such as naftidrofuryl, pentoxifylline or cilostazol	• There is very limited evidence to support the use of vasoactive medications in improving symptoms of end-stage CLTI
Topical vasodilators such as topical GTN or ISDN paste or patch	• Topical nitrates such as GTN or ISDN have local vasodilatory effects on vascular smooth muscles, which might lead to improvement in pain[31] • Topical nitrates can be applied to limited areas of discoloured digits, or skin adjacent to dry gangrene • Application of a large volume could lead to systemic absorption and cause unintended hypotension • There has been evidence for use in vasopressor-induced digital ischaemia or meningococcal-induced digital ischaemia, but there is a lack of evidence in the treatment of CLTI

CLTI, chronic limb-threatening ischaemia; CLS, chemical lumbar sympathectomy; GTN, glyceryl trinitrate; IPC, intermittent pneumatic compression; ISDN, isosorbide dinitrate; SCS, spinal cord stimulation.

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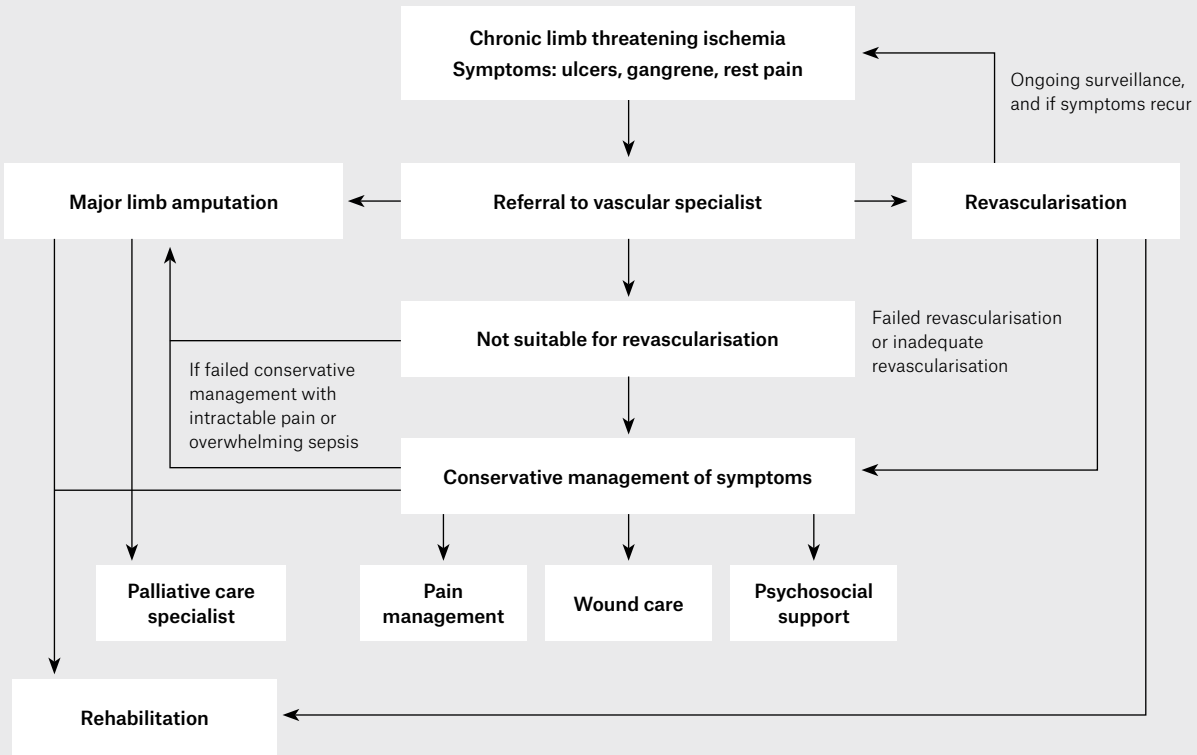

Figure 2. Flow diagram of management of end-stage chronic limb-threatening ischaemia in the community.

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The importance of comprehensive cancer survivorship care plans in general practice

Christina Green, Carolyn Ee, Kylie Vuong

Background
The number of people living with or beyond cancer are expected to rise. General practice-led cancer survivorship plans have been proposed as a way to address ongoing healthcare needs (including physical and psychosocial care) and care coordination, as well as the prevention and management of other chronic illnesses.

Objective
The aim of this paper is to discuss the role of general practice in the long-term care of cancer survivors and provide a summary of recommendations for comprehensive cancer survivorship care planning in general practice.

Discussion
General practice provides cancer survivors with ongoing support within their community from pre-diagnosis onwards. It is recommended that comprehensive cancer survivorship care plans include the cancer treatment summary and follow-up care planning; the management of other comorbid chronic conditions; health promotion and disease prevention with tailoring to shared goals; and the cancer survivor's unique situation.

CANCER SURVIVAL RATES are increasing, with Australia having some of the highest survival rates internationally.[1–3] The number of cancer survivors exceeds one million and is projected to increase.[4–6] General practice, which includes general practitioners, practice nurses and, in many instances, allied health practitioners, provides cancer survivors with ongoing support within their community from pre-diagnosis onwards.[7–11] Its focus on whole-person patient-centred care is essential.[7,9,10,12–14]

General practice-led cancer survivorship plans have been proposed as a way to address ongoing healthcare needs (including physical and psychosocial care) and care coordination, as well as the prevention and management of other chronic illnesses.[7–9,14–16] This paper discusses the role of general practice in the long-term care of people living with and beyond cancer, and provides a summary of recommendations for comprehensive cancer survivorship care.

The role of general practice and models of cancer survivorship care

The phase after primary cancer treatment is crucial to the care trajectory. General practice has important roles in the monitoring and early intervention of treatment-related effects, cancer surveillance, the prevention, screening and management of other chronic illnesses, and health promotion.[17] Increasing the role of general practice in cancer survivorship care offers multiple advantages. The long-term therapeutic partnership that general practice has with patients places it in a unique position to support ongoing survivorship care in the context of the patient's other care needs.[9,10,16,18] Patients experience a higher level of trust and confidence when their general practitioner (GP) is involved.[13] General practice facilitates a more collaborative team approach to respond to the patients' care needs in a timely and resourceful manner.[8,18]

Various models of cancer survivorship care have been described based on the disease, care setting, healthcare discipline leading the follow-up care and the purpose of the care provided (Table 1).[17,19] Long-term cancer survivorship care from the tertiary cancer services that provided the primary cancer treatment might not be sustainable, nor are they designed to provide comprehensive longitudinal care.[9,20]

Comprehensive cancer survivorship care planning

Survivorship care plans summarise the cancer history, goals and follow-up care. Patients find care plans helpful, especially when focused on shared areas of concern, such as the management of treatment-related

effects and healthy lifestyles, and provide a clear follow-up treatment summary.[21] Cancer organisations such as the Clinical Oncology Society of Australia (COSA) and the American Society of Clinical Oncology (ASCO) advocate for survivorship care plans to support communication and the transition to survivorship.[22–24] More recently, the Australian Cancer Survivorship Centre (ACSC) launched mycareplan.org.au, an online resource that empowers survivors to manage their long-term care.[25] Although it is not currently integrated with general practice information systems, it shows promise in improving patient engagement and self-management. However, as these care plans are disease-focused, they might not encompass the broader survivorship needs.

A comprehensive survivorship care plan in general practice should consider long-term care needs using a patient-centred approach, to encompass the patient's needs and diversity, as well as their cultural, family and social context.[26] This is especially important for under-served populations such as those living in rural or remote areas, who come from culturally or linguistically diverse or Aboriginal and Torres Strait Islander backgrounds, people with disability, and other priority populations as outlined in the Australian Cancer Plan.[27–31] The plan should consider incorporating the cancer survivor's relevant demographics, cancer treatment summary and follow-up care planning, including both physical and psychosocial effects, as well as care of other comorbid chronic conditions, health promotion and disease prevention that is tailored to shared goals, and the cancer survivor's unique situation (Table 2).[21,26] Additionally, to provide for a supportive patient and caregiver experience, the plan should promote effective care delivery, collaboration, care coordination and communication.[26] Engaging in shared decision making to align with the patient's goals, priorities and expectations will likely improve patient empowerment and satisfaction.[32] Patient education plays an important role in influencing knowledge, attitudes and behaviour towards more favourable health outcomes.[33]

The GP might consider accessing existing Medicare funding arrangements, such as chronic disease management plans (MBS item number 721), team care arrangements (MBS item number 723) and multidisciplinary care planning (MBS item number 729), to prepare, coordinate and review cancer survivorship plans.[34,35] Eligible cancer survivors could receive funding for up to five consultations with allied health practitioners each year as part of chronic care planning. For example, a cancer survivor with post-cancer fatigue might benefit from a consultation with an exercise physiologist.[36] However, there are limitations with the current Medicare funding arrangements, as the provision of only five allied health visits might not be adequate to meet the complex needs of cancer survivors. In addition, the failure of Medicare rebates to align with rising practice costs leaves cancer survivors vulnerable to increasing out-of-pocket health expenses, especially as there are often other comorbid chronic conditions to be managed.[37–39] Cancer survivorship care would benefit from additional financial support to assist with meeting holistic care needs, and policy change is required to adequately provide for survivorship care in general practice.

Table 1. Cancer survivorship care models

Model	Focus
Disease-specific	Tumour type (ie breast cancer, lung cancer)
Setting of care	Where the follow-up will be conducted (ie oncology practices, survivorship clinics, primary care settings)
Type of clinician	Who will lead the follow-up care (ie general practitioner-led, nurse-led, specialist-led care and shared care)
Purpose of care	Intention of care (ie transition of care from tertiary care to primary care, development of long-term cancer survivorship care plan)

Table 2. Summary of recommendations: Survivorship care plan

Dimensions	Sections
Demographics	Patient details, general practitioner details, medical history, current medication, allergies, immunisations
Cancer treatment summary	Oncologist details, diagnosis, treatment, familial cancer risk assessment
Cancer follow-up care plan	Additional cancer treatment, schedule of clinical visits, cancer surveillance and recommended tests, current treatment side effects, possible late- or long-term effects, referrals
Physical effects	Symptoms assessment, referrals, treatment/risk-reducing strategies
Psychosocial effects	Symptoms assessment (psychological, financial and/or employment, interpersonal), referrals, treatment/risk-reducing strategies
Other chronic conditions	Evaluation and treatment, referrals, treatment/risk-reducing strategies
Health promotion and disease prevention	Prevention-focused visits and testing, age- and gender-appropriate cancer screening, vaccination advice
Care goals	Patient's goals of care, practitioner's goals of care (for cancer and other chronic conditions)

Potential barriers to implementing comprehensive survivorship care plans

Several important factors might impact the successful implementation of care plans for

cancer survivors in general practice. Patients might not recognise the role that general practice plays in their ongoing care after completing cancer treatment, potentially leading to delayed or inadequate access to follow-up care.[17] Additionally, insufficient information and communication from tertiary services might limit the ability to produce a comprehensive plan with all the required diagnosis and treatment details.[9] This, combined with the current fee-for-service model in general practice, which does not reward longer consultations needed for comprehensive survivorship care, makes it difficult to create and implement effective care plans. Although GPs might perceive that they lack the confidence in, and knowledge of, how to care for cancer survivors and, in general, receive little training on cancer survivorship, it is important to note the importance of holistic whole-person care in survivorship care.[9,15,40] Identifying and addressing these challenges to ensure the successful implementation of survivorship care plans in general practice is critical to improving the quality of care and outcomes for cancer survivors.

Conclusion

GP-led cancer survivorship care, developed in collaboration with patients with contribution from cancer services, offers a patient-centred and sustainable approach to meet the long-term care needs of cancer survivors. We recommend that comprehensive cancer survivorship care plans include the cancer treatment summary and follow-up care planning, the management of other comorbid chronic conditions, health promotion and disease prevention, tailored to shared goals and the cancer survivor's unique situation. Policymakers should review funding arrangements to provide additional support for cancer survivorship care in general practice.

Key points

- Cancer survivorship is projected to increase in Australia.
- The increasing role of general practice in cancer survivorship care offers multiple advantages.
- Developing a comprehensive cancer survivorship care plan in collaboration with the patient would help to address long-term care needs.
- A comprehensive cancer survivorship care plan in general practice should consider cancer follow-up care planning as well as care of other comorbid chronic conditions, health promotion and disease prevention.
- The general practitioner might consider accessing existing Medicare funding arrangements to prepare, coordinate and review cancer survivorship plans.

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Adrenocortical tumour:
A case of precocious puberty

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CASE

A young girl, aged 20 months, presented with pubic hair growth and clitoromegaly. She was previously well with an uncomplicated perinatal history and normal neurodevelopment. There was no relevant past medical or family history.

QUESTION 1

What is precocious puberty? What is virilisation?

ANSWER 1

Precocious puberty refers to the appearance of secondary sexual characteristics (eg breast growth, testicular enlargement, pubic hair) before the ages of eight years in girls and nine years in boys.[1]

Virilisation occurs when there is excess production of androgens leading to the development of exaggerated masculine features such as hirsutism (excessive male-pattern hair growth), pubic/axillary hair growth, clitoral or penile enlargement, adult-type body odour, acne, acceleration of growth, increased musculature, voice changes and irregular menstruation in postmenarcheal women.[2]

CASE CONTINUED

This girl had features of virilisation with pubic hair (Tanner stage 2), mild facial acne and clitoromegaly. She did not have axillary hair, adult-type body odour, Cushingoid features or features of true (central) precocious puberty such as breast buds, acceleration in linear growth or vaginal discharge or bleeding. Gastrointestinal examination was within normal limits with no palpable abdominal masses. The rest of her clinical examination, including neurological (cranial nerve) examination, was unremarkable.

QUESTION 2

What is the diagnostic approach to precocious puberty?

QUESTION 3

What is the difference between central and peripheral precious puberty?

ANSWER 2

The diagnostic approach includes a clinical history, examination and investigations.

- Relevant history would include the following: details around the puberty changes, including breast development, vaginal bleeding, genital changes (testicular, penile, clitoral enlargement), pubic/axillary hair growth, adult-type body odour, acne and change in voice. For each of these changes, identify the age of onset and its progression. Growth acceleration or stalling of linear growth, weight gain. Exposure to exogenous sources of oestrogen or testosterone.
- A review of systems, including abdominal (abdominal mass, abdominal pain), neurological (visual changes, headaches) and symptoms of other hormone(s) deficiency/excess (eg symptoms of hypothyroidism). Family history, including onset of puberty, the heights of family members and endocrine, autoimmune and genetic conditions.
- Clinical examination would include the following (Table 1): a general examination, including growth parameters, syndromic features (eg Beckwith–Wiedemann, Li–Fraumeni), muscle/adipose tissue distribution and the skin (neurocutaneous stigmata, hyperpigmentation, acne, striae). A focused examination, including Tanner staging for puberty; signs of virilisation/excess androgen production (excess cortisol production: Cushingoid features; feminisation/excess oestrogen production [eg breast development]; other hormone(s) excess or deficiency); and abdominal and neurological examinations (eg abdominal mass, visual field deficit).[3]

In children who present with features of precocious puberty, differentiation between

gonadotropin-dependent precocious puberty, gonadotropin-independent precocious puberty and benign pubertal variants without underlying pathology is crucial.[1]

First-line investigations include testosterone, oestradiol, dehydroepiandrosterone sulfate (DHEAS), 17-hydroxyprogesterone (17OHP), and basal and stimulated luteinising hormone (LH):follicle-stimulating hormone (FSH) ratio. A stimulated LH:FSH ratio is used to determine non-progressive precocious puberty versus central precocious puberty.[4] A rise in testosterone and DHEAS can be due to normal pubertal development or suggestive of a secondary cause, whereas a rise in 17OHP can be suggestive of congenital adrenal hyperplasia (CAH) and requires further investigation by an adrenocorticotropin stimulation test.[4]

Imaging should include an ultrasound and computed tomography of the abdomen and pelvis to assess for possible tumours. In addition, a bone age assessment is performed to look for advances in height age correlating with precious puberty.[4]

ANSWER 3

Precocious puberty can be either central or peripheral in origin (Table 2). Central precocious puberty is gonadotropin-releasing hormone (GnRH) dependent and due to early activation of the hypothalamic–pituitary–gonadal axis. Peripheral precocious puberty is GnRH independent and due to the production of sex steroids from either endogenous or exogenous sources.

In women, activation of the hypothalamic–pituitary–gonadal axis leads to rising levels of oestrogen, which stimulate breast development (thelarche), the first clinical sign of true puberty. This is followed by a pubertal growth spurt and then onset of menarche around two years after the onset of thelarche. In men, the first clinical sign of true puberty is testicular enlargement. The pubertal growth spurt in men occurs later in the course of puberty than it does in women.[6]

QUESTION 4

How do adrenocortical tumours present?

ANSWER 4

Most adrenocortical tumours present in childhood with signs/symptoms of hormonal excess. The clinical features of these tumours will differ depending on the hormone(s) being produced in excess.[7]

CASE CONTINUED

Investigations showed elevated androgens (Table 3). Other hormone levels were within normal limits, with no signs of hypothalamic–pituitary–gonadal axis activation, hypercortisolism, hyperaldosteronism or hypothyroidism. Computed tomography imaging of the abdomen and pelvis showed a well-defined left adrenal mass measuring 57 mm × 45 mm × 46 mm with no features of invasion or distant metastatic spread (Figures 1 and 2). A left adrenalectomy was subsequently performed without any complications. Postoperatively, the elevated androgen levels rapidly normalised (Table 3) and there has been regression of the virilisation.

Table 1. Clinical presentations of hormonal excess[6]

Hormone secreted in excess	Clinical features	% Childhood adrenocortical tumours
Cortisol	Cushing syndrome	3–8
Aldosterone	Conn syndrome	<1
Androgens	Virilisation	40–55
Oestrogen	Feminisation	<1
Mixed	Mixed features	45–50

Table 2. Causes of central and peripheral precocious puberty[5]

Central precocious puberty (GnRH-dependent)	Peripheral precocious puberty (GnRH-independent)
• Idiopathic	• Congenital adrenal hyperplasia
• CNS trauma	• Gonadal tumours
• Tumours	• Adrenocortical tumours
• Infections (meningitis, encephalitis)	• McCune–Albright syndrome
	• Primary hypothyroidism
	• Exogenous exposure to sex steroids

CNS, central nervous system; GnRH, gonadotropin-releasing hormone.

Table 3. Hormone concentrations pre- and postoperatively in the clinic case

	Preoperatively	Postoperatively			Reference intervals
		3 days	2 months	5 months	
Adrenal: androgens					
Testosterone (nmol/L)	8.4	0.7	<0.5	<0.3	<0.5
DHEAS (µmol/L)	41	0.8	0.2	<0.5	<1.0
Androstenedione (nmol/L)	2.3	0.7	<0.4	0.2	0.1–0.7
Adrenal: glucocorticoid					
ACTH (ng/L)	10	38	26	21	10–50
Cortisol (nmol/L)	130 (at 4 pm)	303 (at 9.40 am)	197 (at 8.30 am)	284 (at 9.15 am)	60–570
Glucose (mmol/L)	4.4		4.8	4.1	3.0–7.8
Adrenal: mineralocorticoid					
Aldosterone (pmol/L)	92				100–1500
Aldosterone/renin ratio	<1				<55
Sodium (mmol/L)	136		139	138	133–144
Potassium (mmol/L)	4.2		4.8	4.6	3.9–5.6
Hypothalamic–pituitary–gonadal axis					
LH (U/L)	1				<6
FSH (U/L)	2.4				<10
Oestradiol (pmol/L)	15				<100
Thyroid axis					
TSH (mU/L)	2.6				0.7–5.9
FT4 (pmol/L)	14				8.7–16
Tumour markers/genetics					
β-hCG (IU/L)	0.1				0.1–0.6
TP53 gene				No pathogenic variants detected	
SNP array analysis				Normal female molecular karyotype	

ACTH, adrenocorticotropic hormone; β-hCG, beta human chorionic gonadotropin; DHEAS, dehydroepiandrosterone sulphate; FSH, follicle-stimulating hormone; FT4, free thyroxine; LH, luteinising hormone; SNP, single nucleotide polymorphism; TP53, tumour protein 53; TSH, thyroid-stimulating hormone.

Key points

- It is important to distinguish central versus peripheral causes in precocious puberty with meticulous history, examination and investigations.
- Any signs of virilisation in a young child need to be investigated further.
- Adrenal tumours should be considered in cases of peripheral precocious puberty.

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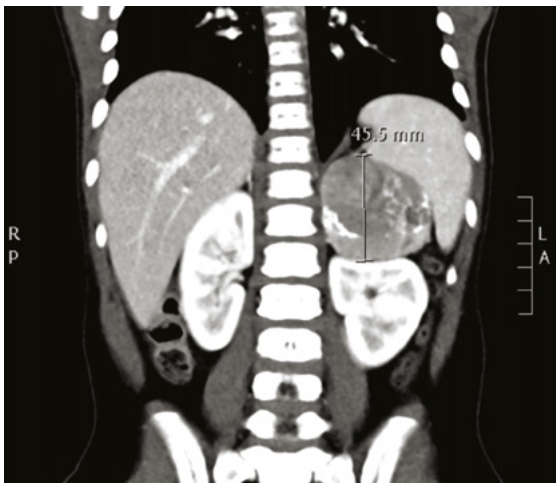

Figure 1. Computed tomography showing a well-defined left-sided lesion displacing the kidney inferiorly.

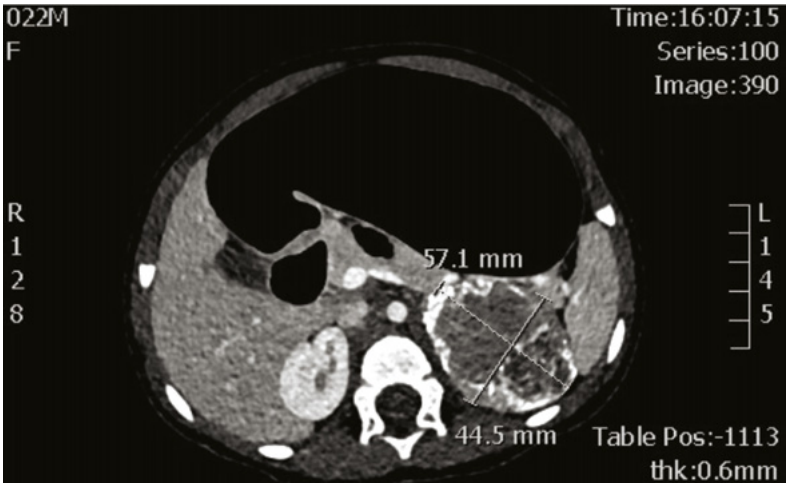

Figure 2. Computed tomography showing a well-defined left-sided lesion with soft tissue and calcific densities.

the clinical details of the case. The authors also acknowledge Dr Jacob Therakathu, radiologist, Mackay Base Hospital, for helping with the interpretation of the computed tomography images for this case.

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A hormonally active struma ovarii: A rare tumour mimicking ovarian cancer

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CASE

A woman, aged 72 years, approached our clinic with symptoms of abdominal discomfort and difficulty in micturition and defecation. She had a medical history of hypertension for 10 years and newly onset hyperthyroidism (for three months). She had undergone an ovary cystectomy 25 years ago and a subtotal thyroidectomy operation 15 years ago. She had been postmenopausal for 22 years. On pelvic examination, a mobile, palpable mass from the symphysis pubis to the umbilicus was noted. Transvaginal ultrasonography revealed a cystic mass larger than 15 cm in diameter and ascites. Ultrasound findings of the mass were well-vascularised solid components with smooth contours that resembled a malignant mass. The patient underwent thorax computed tomography and abdominal magnetic resonance imaging, which revealed pleural effusion and the heterogeneous complexity of the adnexal mass (Figure 1). CA-125 was 2289 U/mL (normal range <35 U/mL) and CA 19-9 was 138 U/mL (<37 U/mL), respectively. Further, because the patient had tachycardia, blood thyroid stimulating hormone (TSH), T3 and T4 levels were examined, and hyperthyroidism was determined as present. The blood TSH level was 0.05 uIU/mL (normal range 0.4–3.8 uIU/mL), and T3 and T4 levels were 3.1 ng/mL and 24.6 ng/mL, respectively (normal range for T3 is 0.9–1.95 ng/mL and for T4 is 4.4–12.5 ng/mL).

The patient underwent a laparotomy. At the exploration, there were 2000 cc ascites present in the pelvic area. At the right adnexa, there was a 20×25 cm multilobulated cystic mass (Figure 2). Intraoperative frozen section analyses suggested a mucinous cyst. Total hysterectomy, bilateral salpingo-oophorectomy and omentectomy were performed. The patient tolerated the surgery well. Following surgery, blood plasma thyroid levels regressed to normal levels and tumour markers became negative. Further, ascites and pleural effusion were regressed.

Postoperative histopathological evaluation showed a classic pattern of variable-sized follicles filled with colloid, mature thyroid tissue, ovarian stroma with fibrosis, oedema and corpus albicans (Figure 3). A diagnosis of benign, hormonally active struma ovarii was made. The diagnosis of struma ovarii in this case was based on histopathological criteria of entopic thyroid tissue. Before the surgery was performed,

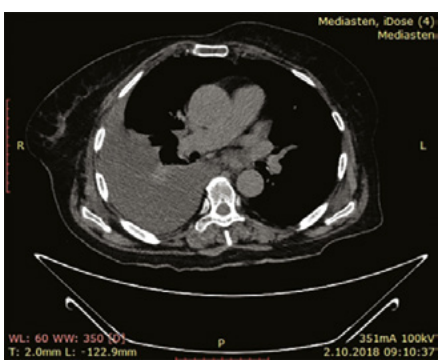

Figure 1. Magnetic resonance image revealing pleural effusion and heterogeneous complexity of adnexal mass.

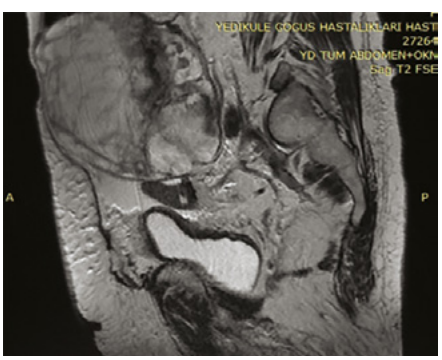

Figure 2. A 20×25 cm multilobulated cystic mass at the right adnexa.

due to hyperthyroidism that had started, the patient had to use methimazole 15 mg daily and propranolol 2×20 mg daily. Still, the blood TSH levels were lower than the normal range. After the operation was performed, there was no need for use of methimazole, and blood TSH, T3 and T4 levels were in the normal range. At six-month follow-up after the operation, the patient's thyroid hormones were in the normal range. Abdominal ultrasonography was also performed, and no problems were present.

QUESTION 1

Considering the possible mechanisms of thyrotoxicosis in this woman diagnosed with struma ovarii, what do the thyroid results mean?

QUESTION 2

What are the potential risks for malignancy?

QUESTION 3

What is the aetiology of ascites and pleural effusion?

ANSWER 1

Thyroid tissue is observed as a component of benign cystic teratomas in 5–7% of cases.[1]

Thyrotoxicosis occurs in approximately 8% of affected patients. In patients with thyrotoxicosis, the level of TSH is suppressed and the levels of T3 and T4 are elevated. Thyroglobulin is secreted by benign and malignant ovarian stroma, mostly from malignant stroma. Thyrotoxicosis with a benign struma ovarii is rarer, and these women should be treated with antithyroid drugs and beta blockers if needed before the surgery. If the struma ovarii is malignant, the patient must also undergo a thyroidectomy after iodine treatment.

In this report, a rare form of a benign adnexal mass with malignant features was discussed. These hormonally active benign struma ovarii cases are rare. Hyperthyroidism due to struma ovarii was present, which disappeared after the operation. Because of the rarity of these hormonally active tumours, the diagnosis is difficult to make and is mostly done by histopathological evaluation after the operation. Hormonal tumour activity disappears postoperatively and thyroid levels become normalised right after the operation.

ANSWER 2

Struma ovarii might be benign or malignant. The distinction between these lesions is challenging because there is a lack of firm criteria for diagnosis. Most studies show that the diagnosis could be made based on the presence of entopic thyroid carcinoma (overlapping nuclei, mitotic activity and vascular invasion).[1–5] In our case, there was no mitotic activity or vascular invasion. However, entopic thyroid tissue with colloid-filled variable-sized follicles was observed.

ANSWER 3

The aetiology of ascites and pleural effusion in patients with struma ovarii remains unclear. Meigs described Meigs syndrome in 1937 as cases with benign ovarian tumours with ascites and hydrothorax; the tumours are limited to fibroma, thecoma and granulosa cell tumours. Pseudo-Meigs syndrome consists of ascites and pleural effusion with serous and serosanguinous fluid and tumours such as benign ovarian cysts, uterine leiomyomas and teratomas, including struma ovarii.[6] The mechanism of the rise in CA 125 levels in pseudo-Meigs syndrome remains unclear, although Mui et al suggested that the increase in CA 125 levels could be due to the irritation from free fluid leading to the inflammation of pleural and peritoneal surfaces.[7] The rise of CA-125 levels and the presence of ascites and pleural effusion in our case are suggestive of a malignant mass. However, the levels of CA-125 normalised on the postoperative eighth day after the cause of the inflammation and irritation was removed. There was no need for any adjuvant therapy after the operation. The thyroid hormone profile of the patient was also normalised after the operation. That is an unusual and rare situation that is noteworthy and a new clinical insight.

Obeidat et al presented 26 struma ovarii cases that mimicked malignant adnexal masses with ascites and raised blood CA-125 levels.[8] Preoperatively, all cases

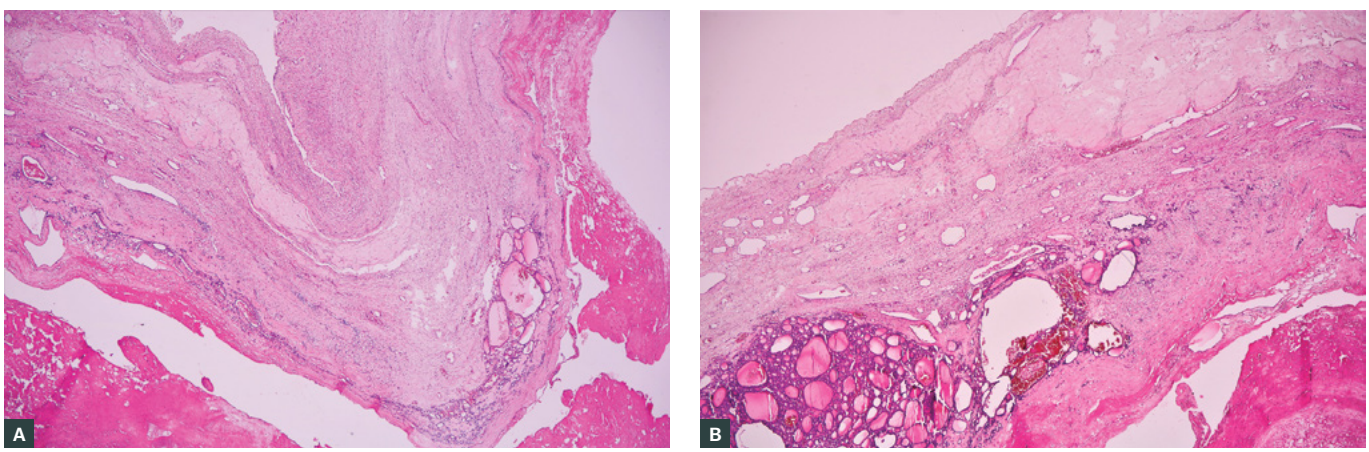

Figure 3. Classic pattern of variable-sized follicles filled with colloid, mature thyroid tissue, ovarian stroma with fibrosis, oedema and corpus albicans. (**A**) Variable-sized follicles filled with colloid. (**B**) Ovarian stroma with fibrosis, oedema and corpus albicans.

were thought to be malignant before the histopathological examination. Similarly, in the present case, the preoperative diagnosis was a malignant adnexal mass. In all cases, blood CA-125 levels normalised and ascites disappeared after the operation. Most of the cases occurred in the fifth decade of life or later. The tumours were between 4 cm and 23 cm in dimension. In the presenting case, the tumour was sized 20×15 cm. The most common presenting symptom was abdominal discomfort, which was the main symptom in the presenting case. In 15 of the cases, pleural effusion was also present. There was no relation between pleural effusion and ascites. Their levels were not affected by each other. Most of the cases were treated with total abdominal hysterectomy and bilateral salpingo-ophorectomy. Just three of the cases studied by Obeidat et al underwent conservative surgery.[8] Choosing conservative surgery is related to the fertility wishes of the patient.

Key points

- Thyrotoxicosis with a benign struma ovarii is rarer and should be treated with antithyroid drugs and beta blockers if needed before the surgery.
- If the struma ovarii is malignant, the patient must also undergo a thyroidectomy after iodine treatment.
- Studies show that the diagnosis of malign struma ovarii could be made based on the presence of entopic thyroid carcinoma.

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Feasibility, acceptability and efficacy of a pilot exercise physiology group service for older people with type 2 diabetes

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Background and objective
Type 2 diabetes affects over half a million older Australians. Australian Medicare group exercise and education interventions can support older adults' diabetes management. However, the feasibility and acceptability of accredited exercise physiologist (AEP)-delivered services are yet to be assessed. This study aimed to assess the feasibility, acceptability and preliminary efficacy of a Medicare type 2 diabetes group exercise and education intervention for older adults.

Methods
This study was a single-arm feasibility, acceptability and preliminary efficacy trial of an AEP-delivered type 2 diabetes group service for older adults with the condition. Participants attended the diabetes clinic once per week for eight weeks, through Medicare, for a group exercise and education session. Attendance, participation, enjoyment, suitability, usefulness and pre–post clinical health outcomes were assessed.

Results
The intervention was feasible and acceptable, with 40 participants (mean [±standard deviation] age 71.8±4.5 years [range 65–81 years]; 45% female) attending 87% of sessions. Almost all participants (97%) strongly agreed that the program was enjoyable. Participants also improved fitness and cardiometabolic health outcomes.

Discussion
More Australians should be referred to and attend Medicare-subsidised exercise physiologist-delivered group sessions. The potential for additional sessions to achieve greater physical activity engagement and diabetes self-management should be further investigated.

TYPE 2 DIABETES (T2D) affects 15–19% of older Australians.[1] Exercise and self-management are recommended for T2D due to their ability to improve health outcomes.[2] Exercise interventions for T2D can reduce healthcare burden and are cost-effective (up to $50,000 per quality-adjusted life year gained or disability-adjusted life year averted).[3] Group exercise and education interventions, including the combination of recommended aerobic training and progressive resistance training tailored to the individual, have been demonstrated to be capable of improving the health outcomes of older adults with T2D in community-based settings.[4,5] Such interventions directly benefit glycaemic control, cardiorespiratory and muscular fitness, cardiovascular risk, body composition, quality of life and physical function.[6,7]

Despite growth in the number of accredited exercise physiologists (AEP) able to deliver rebateable group services under Medicare in Australia, older adults with T2D remain less likely to attend or meet the physical activity guidelines than people without diabetes, with less than 0.01% of older adults with T2D accessing such services.[8,9] There is no evidence evaluating AEP-delivered T2D Medicare group programs, which includes a subsidised initial assessment and eight group sessions annually. Therefore, the aims of the present study were to assess: (1) the feasibility and acceptability of an evidence-based, consumer-driven T2D group exercise and education intervention using AEP Medicare services for older adults with T2D (diabetes clinic); and (2) the effect of the diabetes clinic program on cardiometabolic health and fitness outcomes. It was hypothesised that the diabetes clinic would be feasible and acceptable among older adults with T2D.

Methods

This study used a single-group quasi-experimental design to investigate the feasibility, acceptability and preliminary efficacy of an AEP-delivered T2D group Medicare service for older adults. This study was approved by the Human Research Ethics Committee at the University of New South Wales (UNSW) (HC200973), and the trial was registered with the Australia New Zealand Clinical Trial Registry on 30 April 2021 (ACTRN12621000505808). All participants provided written informed consent. The study is reported in

accordance with the CONSORT statement (feasibility trials)[10] and the intervention reported in line with the Template for Intervention Description and Replication (TIDieR) checklist[11] and the Consensus on Exercise Reporting Template (CERT).[12]

All assessments and group sessions were conducted at the UNSW Medicine and Health Lifestyle Clinic (UNSW Sydney, Australia) by a single AEP with over 12 years clinical experience. Completion of assessments and delivery of the intervention were in accordance with Medicare requirements for Item numbers 81110 and 81115, respectively, with each group having a minimum of two and maximum of 12 participants. All participants were bulk-billed under Medicare and were not charged any out-of-pocket fees for the exercise physiology services.

Permission was sought from Medicare Australia on 19 February 2021 for this trial to be completed; trial recruitment, enrolment and data collection occurred between May 2021 and October 2022. Participants were included if they were aged ≥65 years and had been diagnosed with T2D. Exclusion criteria included diabetes diagnosis other than T2D, abnormal cardiovascular response to exercise, unable to speak English and without a translator, or other health conditions that prevented exercise participation.

Initial assessment (90–120 minutes) included a health interview, physical assessment and questionnaires. Participants then received eight group sessions of 90 minutes once per week. Using baseline exercise assessment results, the AEP individualised and monitored exercise intensity and facilitated education (Table 1; Appendix 1, available online only). Where participants were unable to perform the listed exercises due to health or movement limitations, exercises were modified to a body weight or dumbbell alternative. The intervention did not provide a structured home exercise program, although it did include education on physical activity away from the program. A final assessment occurred in the week after the final group session.

The primary outcomes were feasibility and acceptability. Feasibility was examined in terms of group session attendance, quantified as total sessions attended out of a maximum possible of eight and reported as a percentage. The criterion for feasibility was attendance at five or more of the eight (62.5%) group sessions, in line with the current AEP Medicare attendance rates of 5.27 of eight group sessions.[8,13] Feasibility was further quantified as session compliance with criteria for success requiring performance of at least 10 minutes of aerobic training and five progressive resistance training exercises per session. Participant attendance, exercise programming and adverse events were recorded each session on pre-prepared hard copy sheets by the AEP during the sessions. Participants' perspectives on the acceptability of the interventions were based on the final diabetes clinic assessment participant evaluation and feedback questionnaires. Acceptability was analysed through individual scores from five questions using a five-point Likert scale encompassing participants' self-reported agreement with enjoyment, exercise appropriateness, improved understanding of diabetes self-management, improved health and willingness to attend again (Appendix 2, available online only). Participants also completed the global perceived effect scale.

Table 1. Diabetes clinic exercise session programming principles

Principle	Aerobic exercise	Resistance exercise
Frequency	• 1×week	• 1×week
Intensity	• Light to vigorous (light 40–55% HR_{max}/RPE 8–10; moderate 55–70% HR_{max}/RPE 11–13; vigorous 70–90% HR_{max}/RPE 14–16)	• Light to vigorous (light 30–49% 1RM/RPE 9–11; moderate 50–69% 1RM/RPE 12–13; vigorous 70–84% 1RM/RPE 14–17)
Time	• 20–30 min	• 20–30 min • Sets: 3 • Repetitions: 8–12 (aim for 10)
Type	• Cycling, rowing, stepping	• Pin-loaded weight plate machines, body weight, dumbbell exercises
Pattern	• One continuous bout (eg 15–20 min on one type) or two bouts (1×10 min cycling and 1×10 min rowing)	• 2-s concentric, 3-s eccentric with 1-min rest between sets
Progression	• Commence first sessions Week 1 at low–moderate intensity depending on training experience and gradually progress towards moderate–vigorous intensity as tolerated by the individual when HR and RPE consistently (two consecutive sessions) at lower end of intensity range • Progression implemented by increased speed or workload	• Commence first sessions Week 1 at low–moderate intensity depending on training experience and gradually progress towards moderate–vigorous intensity as tolerated by the individual when RPE consistently (two consecutive sessions) at lower end of intensity range and technique/form stable • Progression implemented by increased load

1RM, one-repetition maximum; HR, heart rate; HR_{max}, heart rate maximum; RPE, rate of perceived exertion.

Secondary outcomes were changes in health from the initial to final assessment and included resting heart rate, resting blood pressure, body weight, height, body mass index (BMI), waist circumference (WC), six-minute walk test (6MWT) distance, short physical performance battery including times to perform the five times sit-to-stand (5×STS) test, side-by-side stance, semi-tandem stance, full tandem stance and eight-foot walk, duration of single leg balance, handgrip strength, whole body strength (composite outcome calculated by the addition of all upper body and lower body one-repetition maximum [1RM] results: supported row, leg press, chest press, leg extension, lat pulldown and leg curl) and glycaemic and lipid profiles. These outcome measures were identified a priori due to their clinical meaningfulness across multiple aspects of health and fitness with protocols for participant assessment presented in Appendix 3 (available online only).

There were limited data to support a precise a priori sample size calculation to power this feasibility study. However, 30 participants is considered an acceptable number for pilot feasibility studies.[14] Medicare data demonstrate that the 65- to 75-years age group was largest group accessing group T2D AEP services in 2019, with close to 3000 and 4000 group services delivered in New South Wales to men and women, respectively,[8] indicating a single-site sample of 40 would be realistic to achieve over a 12-month period given the resource constraints of the intervention. Assuming at least a 10% drop out for exercise interventions,[15,16] and based on Kirwan et al,[17] who reported a sample size of 43 for a community-led diabetes exercise and education program, we considered a sample size of 40 would be sufficient to meet our primary aim of determining the feasibility and acceptability of the diabetes clinic.

Data were analysed using IBM SPSS (version 28.0.1.0). Demographic, health condition, attendance and acceptability data are reported as percentages and as the mean and standard deviation (SD) unless stated otherwise. The preliminary efficacy of the diabetes clinic in terms of secondary outcomes was analysed using linear mixed models on the pre- and post-test data, with pre–post change entered in the fixed-effects model and participant ID entered in the random-effects model, with the linearity assumption visually inspected.[18] Statistical significance was accepted at $P \leq 0.05$. The clinical significance of changes in applicable outcome measures is also presented.

Results

Overall, 59 people were screened for eligibility, with 80% (n=40; mean age 71.8±4.5 years; 18 [45%] female) of those eligible participating in the diabetes clinic initial assessment (Figure 1). Of these 40 participants, 93% (n=37) participated in the group sessions (87% overall session attendance) and 90% (n=36) completed the final assessment. Table 2 shows the results of participants' baseline assessment. Participants' years since diagnosis with T2D ranged from one to 40 years (mean 13.6±1.6 years). Because pathology was not collected as part of the trial protocol but rather requested from the participants' latest results with their medical doctor, not all participants had glycaemic (only 80% available) and lipid (72.5% available) profiles available for analysis. This adds an interesting aspect to the study in that 35% of participants did not have regular three-monthly glycated haemoglobin measurement as per Australian guidelines for patients with T2D undergoing therapeutic changes such as participating in the intervention.[2] Participants' demographic and lifestyle behaviour characteristics are presented in Appendix 4 (available online only).

The criterion for successful feasibility was achieved with participants attending 7.0±2.2 of the maximum eight sessions, equating to 87% overall attendance, with no significant difference (P=0.8) in attendance between men (6.9±1.8; 86%) and women (7.1±2.6; 88%). A group size of five participants was selected due to clinic space availability, with attendance ranging between two and six participants per session. Overall, participants were compliant with the aerobic training (86%), progressive resistance training (86%)

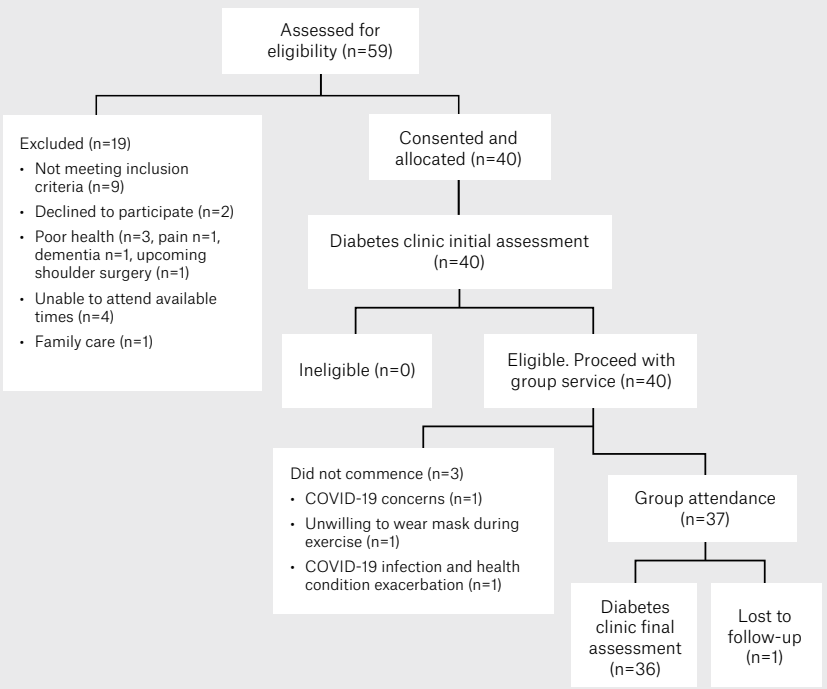

Figure 1. Participant flow diagram for the diabetes clinic program.

Table 2. Diabetes clinic participant baseline outcome measures

Characteristic	Total		Men		Women	
	n	Mean±SD	n	Mean±SD	n	Mean±SD
Age (years)	40	71.8±4.5	22	72.0±4.6	18	71.6±4.6
Years since T2D diagnosis	40	13.6±10.3	22	15.0±10.8	18	11.8±9.6
Vital signs and body composition						
Resting heart rate (bpm)	40	72±10	22	71±10	18	73±11
SBP (mmHg)						
Right	39	141±19	22	143±17	17	140±22
Left	40	140±17	22	140±14	18	141±21
DBP (mmHg)						
Right	39	78±9	22	80±8	17	76±10
Left	40	78±10	22	80±10	18	77±10
Weight (kg)	40	82.4±16.2	22	89.0±16.4	18	74.3±11.9
Height (m)	40	1.66±0.08	22	1.72±0.05	18	1.60±0.07
BMI (kg/m^2)	40	29.7±4.6	22	30.2±5.2	18	29.1±3.9
WC (cm)	40	106.3±14.0	22	111.1±15.1	18	100.5±10.2
Functional capacity and balance						
6MWT distance (m)	39	455±89	22	457±88	17	452±92
5×STS (s)	40	9.1±2.2	22	9.2±2.0	18	9.1±2.4
Side-by-side stance (s)	40	10±0	22	10±0	18	10±0
Semi-tandem stance (s)	40	10±0	22	10±0	18	10±0
Full tandem stance (s)	40	9.5±1.9	22	3.3±2.1	18	9.6±1.6
8-foot walk (s)	40	1.9±0.4	22	1.8±0.3	18	2.0±0.4
SPPB summary ordinal scale	40	12±0.7	22	12±0.6	18	12±0.8
SLB eyes open (s)						
Right	39	33.8±52.6	21	31.8±36.7	18	36.0±67.6
Left	39	30.2±50.1	22	26.1±28.3	17	35.6±69.7
SLB eyes closed (s)						
Right	36	2.9±2.0	19	2.5±1.4	17	3.3±2.5
Left	36	2.7±1.8	20	2.4±1.3	16	3.3±2.3

Table continued on the next page

Table 2. Diabetes clinic participant baseline outcome measures (cont'd)

Characteristic	Total		Men		Women	
	n	Mean±SD	n	Mean±SD	n	Mean±SD
Muscular strength						
Handgrip (kg)						
Right	40	28.3±8.2	22	32.9±6.9	18	22.7±6.0
Left	40	27.5±7.8	22	32.4±5.7	18	21.5±5.3
Total	40	55.8±15.6	22	65.3±12.1	18	44.2±10.8
1RM supported row (kg)	40	43.1±14.0	22	52.6±10.4	18	31.4±7.2
1RM chest press (kg)	39	31.9±11.0	21	39.5±8.8	18	22.9±4.8
1RM lat pulldown (kg)	31	42.1±10.7	18	48.4±9.3	13	33.5±5.0
1RM leg press (kg)	38	102.0±37.0	21	123.6±28.8	17	75.3±27.5
1RM leg extension (kg)	39	34.8±13.2	22	39.9±13.7	17	28.2±9.3
1RM leg curl (kg)	39	17.8±9.5	22	22.3±7.2	17	12.1±9.1
Upper limb strength (kg)	40	106.8±40.3	22	129.9±35.9	18	78.5±24.5
Lower limb strength (kg)	39	151.9±56.6	22	179.9±52.2	17	115.6±39.6
Whole body strength (kg)	40	255.0±93.7	22	310.0±79.4	18	187.6±60.1
Pathology						
BGL (mmol/L)	28	6.3±1.0	16	6.3±1.2	12	6.3±0.8
HbA1c (%)	32	6.6±0.7	17	6.7±0.7	15	6.4±0.7
TC (mmol/L)	29	3.9±0.7	15	3.7±0.8	14	4.0±0.6
LDL-C (mmol/L)	27	1.9±0.6	14	1.9±0.7	13	1.9±0.6
HDL-C (mmol/L)	27	1.4±0.4	14	1.3±0.4	13	1.5±0.1
TG (mmol/L)	28	1.2±0.6	15	1.2±0.5	13	1.3±0.7
Total PA time (min)	40	308±197	22	316±210	18	299±185

1RM, one-repetition maximum; 5×STS, five times sit to stand; 6MWT, 6-min walk test; BGL, blood glucose levels; BMI, body mass index; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; PA, physical activity; SBP, systolic blood pressure; SD, standard deviation; SLB, single leg balance; SPPB, short physical performance battery; TC, total cholesterol; TG, triglycerides; WC, waist circumference.

and educational components (87%) of the sessions. Reasons for non-compliance with aerobic training and progressive resistance training included session non-attendance (41/320 sessions), late attendance (aerobic training only, 3/320 sessions) and exercises requiring modification due to health/movement limitations (1/320 sessions of aerobic training; 2/320 sessions of progressive resistance training).

Participants reported high levels of acceptability (Figure 2). Of the 36 participants (90%; 15 females) who completed the final assessment and feedback questionnaires, 97% strongly agreed that they enjoyed attending the diabetes clinic. Overall, most participants somewhat or strongly agreed the exercises were appropriate to their individual level (97%), felt they had a better understanding of how to manage their diabetes (97%), felt their health was better (94%) and indicated they would attend the diabetes clinic again in the future (94%). When participants were asked how they would describe the change (if any)

in activity limitations, symptoms, emotions and overall quality of life related to their T2D, most indicated an improvement (Appendix 5, available online only). When asked to rate the degree of change on a 0–10 scale (with 0 being much better, 5 no change and 10 much worse), the mean score was 3±2 (range 0–8).

Changes in participants' health outcome measures are summarised in Table 3. There were statistically significant and clinically relevant reductions in systolic blood pressure on the right side (mean difference [MD] −7 mmHg; 95% confidence interval [CI]: −12, −2 mmHg; *P*=0.007) and left side (MD −6 mmHg; 95% CI: −11, −1 mmHg; *P*=0.013) and diastolic blood pressure on the left side (MD −3 mmHg; 95% CI: −5.0, 0 mmHg; *P*=0.021), but not on the right side (MD −1.8 mmHg; 95% CI: −4, 0.0 mmHg; *P*=0.84). There were significant improvements in weight (MD −0.7 kg; 95% CI: −1.3, −0.0 kg; *P*=0.039), WC (MD −1.3 cm; 95% CI: −2.2, −0.4 cm; *P*=0.006), 6MWT distance (MD 22.8 m; 95% CI: 9.1, 36.5 m; *P*=0.002), 5×STS (MD −0.8 s; 95% CI: −1.2, −0.5 s; *P*<0.001) and short physical performance battery ordinal scale (MD 0.3; 95% CI: 0.1, 0.5; *P*=0.009); however, none of these changes was clinically significant.

Significant improvements in all upper and lower limb 1RM assessments were achieved, except for leg curl; whole body strength improved by 15.6% (MD 32.2 kg; 95% CI: 22.3, 42.1 kg; *P*<0.001). No adverse events were reported during the exercise testing or training sessions. One participant's resting blood pressure was elevated prior to commencing one of the exercise sessions. They did not participate in the exercise component of that session and continued the following week after cardiologist review of their hypertensive medication.

Discussion

This study is the first to investigate the feasibility and acceptability of an AEP-led group T2D program for older adults under the Australian Medicare group T2D item. The intervention was feasible and acceptable, with an 87% attendance rate and 97% of participants strongly agreeing the intervention was enjoyable. Participation was also associated with clinically improved blood pressure, body weight, WC, functional exercise capacity, 5×STS, balance and muscular strength.

Community-delivered group services offer a means to provide exercise and education in a cost-effective manner through Medicare. Despite its introduction to the Australian Medicare system over a decade ago, the diabetes group service remains underutilised, with less than 1% of older Australians accessing the individual AEP assessment to

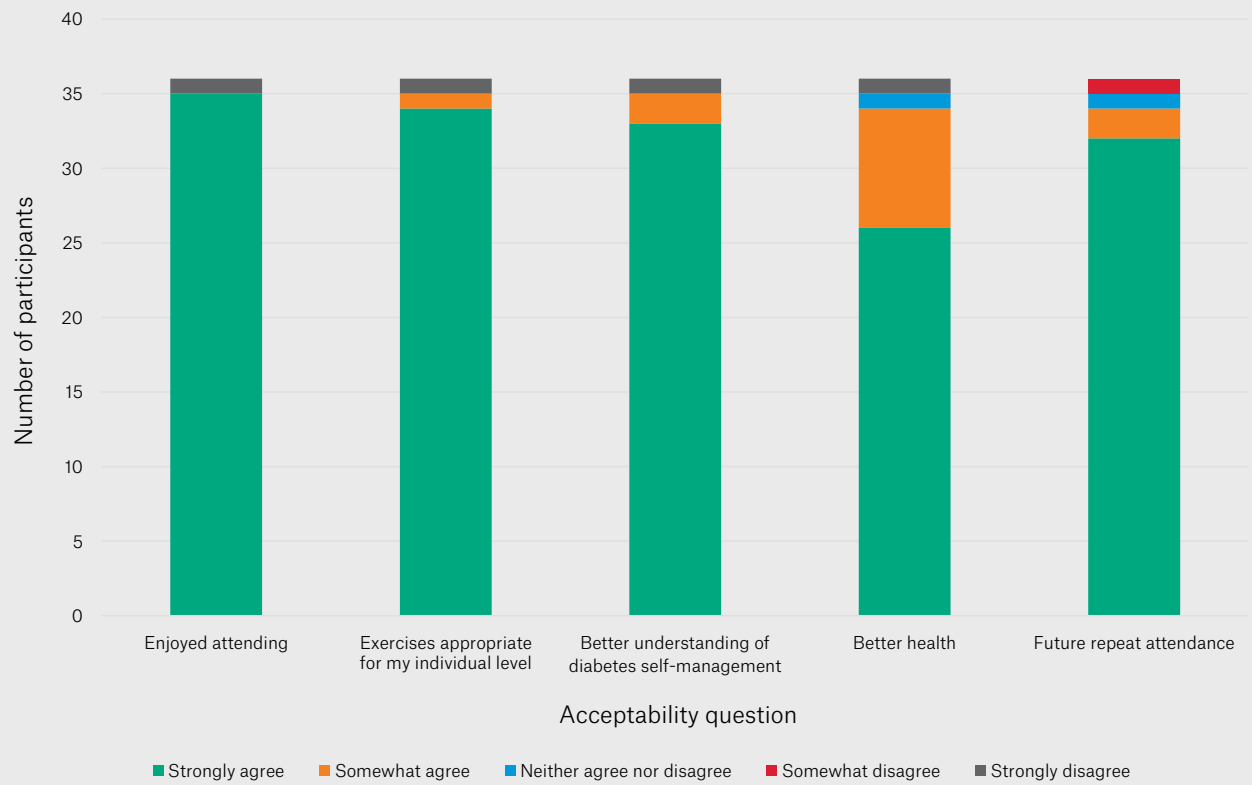

Figure 2. Participant acceptability of the diabetes clinic.

enter group T2D services under Medicare in 2022.[8] With over 678,300 older Australians estimated to have diabetes, evaluation of these services is valuable in understanding the nuances of older adults' engagement in healthcare and providing insights as to why such services are not being accessed.[1] Our diabetes clinic included a final assessment that is currently not included in the Medicare group service subsidised sessions. In line with the 2018 Allied Health Professions Australia recommendations, the call for the addition of a final assessment is strongly supported.[13] Having information on feasibility and acceptability, as well as potential intervention effects on health outcomes, can contribute to a more thorough program evaluation, which can better inform program design and public health policy to ultimately enhance healthcare for older adults with T2D.

Participation in the diabetes clinic was not associated with a significant improvement in glycaemic control through reductions in either glycated haemoglobin or fasting blood glucose levels, potentially due to the short duration and session frequency of the intervention and lack of dietary prescription,[19] as well as pathology results not being collected as part of the trial but rather as part of routine care with the participants' general practitioner or endocrinologist. Future interventions should look to include the routine collection of bloods and longer-term (greater than six months) follow-up to further understand the effects of the intervention on cardiometabolic health.

In Australia, referrals to AEPs continue to be underutilised, especially for older adults and those from non-English-speaking backgrounds.[20] Despite online programs being shown to have similar effectiveness to in-person programs for older adults with T2D,[21] the existing Medicare scheme does not accommodate telehealth for group services, which might pose accessibility challenges for many Australians. Lifestyle interventions have been shown to be cost-saving in Australia, with exercise interventions capable of reducing total healthcare costs by up to 50% per day in the intervention group compared with controls.[3] The T2D consumer benefits–cost ratio sees an expected health return of $8.50 for every $1 spent on AEP interventions.[22] It is important to note that while participants in other studies experienced an increase in costs for diabetes care associated with engagement in T2D programs, other health service costs decreased or remained unchanged,[23] highlighting the potential value of such services. Therefore, further evaluating the delivery of group services in different contexts, including rural and remote areas, can accommodate the wide spectrum of individual needs.

The addition of education to these group sessions, as delivered in the 'Beat It' intervention facilitated by Diabetes NSW and ACT (https://digital.diabetesaustralia.com.au/beat-it), demonstrates how services including a holistic range of lifestyle

Table 3. Change in pre–post assessment outcome measures after diabetes clinic intervention

Outcome measure	Mean difference	95% CI	*P*-value	Clinical significance
Vital signs and body composition				
RHR (bpm)	0	−2, 3	0.711	N
SBP (mmHg)				
Right	−7	−12, −2	0.007	Y
Left	−6	−11, −1	0.013	Y
DBP (mmHg)				
Right	−2	−7, 0	0.084	Y
Left	−3	−5, −0.0	0.021	Y
Weight (kg)	−0.7	−1.3, −0.0	0.039	N
Height (m)	−0.00	−0.00 – 0.00	0.472	N
BMI (kg/m^2)	−0.2	−0.4, 0.0	0.093	N
WC (cm)	−1.3	−2.2, −0.4	0.006	N
Functional capacity and balance				
6MWT (m)	22.8	9.1, 36.5	0.002	N
5×STS (s)	−0.8	−1.2, −0.5	<0.001	N
Side-by-side stance (s)	0.0	0, 0	–	–
Semi-tandem stance (s)	0.0	0, 0	–	–
Full tandem stance (s)	0.5	−0.1, 1.2	0.088	N
8-foot walk (s)	−0.1	−0.2, 0.1	0.375	N
SPPB summary ordinal scale	0.3	0.1, 0.5	0.009	N
SLB eyes open (s)				
Right	2.0	−3.7, 7.6	0.486	N
Left	1.9	−6.1, 10	0.627	N
SLB eyes closed (s)				
Right	0.3	−0.5, 1.1	0.477	N
Left	0.2	−0.5, 0.8	0.611	N

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Table 3. Change in pre–post assessment outcome measures after diabetes clinic intervention (cont'd)

Outcome measure	Mean difference	95% CI	*P*-value	Clinical significance
Muscular strength				
Handgrip (kg)				
Right	0.6	0.4, 1.6	0.232	N
Left	0.7	−0.2, 1.6	0.109	N
Total	1.3	−0.4, 3.1	0.129	N
1RM supported row (kg)	3.5	2.0, 4.9	<0.001	N/A
1RM chest press (kg)	3.4	2.1, 4.6	<0.001	N/A
1RM lat pulldown (kg)	2.88	1.0, 4.7	0.004	N/A
1RM leg press (kg)	13.1	8.0, 18.1	<0.001	N/A
1RM leg extension (kg)	5.8	3.4, 8.3	<0.001	N/A
1RM leg curl (kg)	1.4	−0.1, 2.8	0.065	N/A
Upper limb strength (kg)	8.9	6.1, 11.7	<0.001	N/A
Lower limb strength (kg)	19.9	13.7, 26.1	<0.001	N/A
Whole body strength (kg)	32.2	22.3, 42.1	<0.001	N/A
Pathology				
BGL (mmol/L)	0.1	−0.4, 0.5	0.783	N
HbA1c (%)	−0.1	−0.4, 0.1	0.224	N
TC (mmol/L)	−0.0	−0.2, 0.2	0.899	N
LDL-C (mmol/L)	−0.1	−0.4, 0.1	−1.3	N
HDL-C (mmol/L)	0.1	−0.0, 0.1	0.075	N
TG (mmol/L)	0.0	−0.1, 0.2	0.595	N

1RM, one-repetition maximum; 5×STS,five times sit to stand; 6MWT, 6-min walk test; BGL, blood glucose levels; BMI, body mass index; CI, confidence interval; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; RHR, resting heart rate; SBP, systolic blood pressure; SLB, single leg balance; SPPB, short physical performance battery; TC, total cholesterol; TG, triglycerides; WC, waist circumference.

intervention options have the potential to improve on fitness measures and quality of life.[5] Such programs often include longer-duration and more session designs, and on occasion out-of-pocket expense, compared with the eight sessions available under Medicare. Because the exercise guidelines for people with T2D call for exercise to be performed most days of the week, with these behaviours ideally continuing for more than eight weeks,[6] it is understandable that developed T2D programs adopt a higher session frequency or encourage a structured home-based component in an attempt to have a greater effect on health outcomes. Because AEPs, accredited practising dieticians and diabetes educators are eligible allied health professional providers of the group T2D services under Medicare, future interventions should consider multidisciplinary delivery. Investigating feasibility from a clinician and business perspective would also be advised to improve the understanding as to why this valuable Medicare service is underutilised across Australia and could encompass general practitioner referral.

Conclusion

The diabetes clinic, an AEP-led group T2D service delivered under Medicare, is both feasible and acceptable to older Australians who attend, while also improving participants' cardiometabolic health and fitness. Such programs have the potential to improve diabetes management in a diverse range of older adults through increased participation in healthy lifestyle behaviours, including exercise. Healthcare policy should focus on improving general practitioner and allied health professional awareness of the efficacy of such programs to enhance referrals. Additional Medicare-subsidised group sessions have the potential for greater engagement with physical activity guidelines, health outcome improvement and reduced health expenditure.

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Australian general practitioners' perception of modifiable risk factors in reducing infective complications following hip and knee joint replacement

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Background and objective
Smoking, poor diabetic control and excessive body mass index (BMI) increase the risk of infection following joint replacement. This study investigated Australian general practitioners' (GPs) perception of these modifiable risk factors in patients with end-stage osteoarthritis.

Methods
A structured online survey tool was developed and widely distributed to Australian GPs.

Results
Responses were received from 131 GPs. Most attempted to address current smoking (90%), poor diabetic control (94%) and excessive BMI (89%) prior to referral. The majority felt that joint replacement should be delayed until these risk factors had been modified (57%, 84% and 74%, respectively). However, many respondents did not believe that these risk factors were contraindications to joint replacement (76%, 46% and 43%, respectively).

Discussion
This study suggests that Australian GPs are mindful of modifiable risk factors in patients with hip and knee osteoarthritis; however, many do not support restricting access to joint replacement.

AUSTRALIA IS EXPERIENCING a growing burden of hip and knee osteoarthritis and subsequent joint replacement.[1,2] In 2021, over 41,000 total hip replacement (THR) and over 59,000 total knee replacement (TKR) procedures were performed nationally.[2] By 2030, the incidence of THR and TKR in Australia could exceed 79,000 and 161,000, respectively.[3]

Although THR and TKR are highly successful procedures for patients with end-stage osteoarthritis, they are not without risk.[4,5] Prosthetic joint infection (PJI) is a devastating complication, carrying significant morbidity for the patient and substantial economic cost to the healthcare system.[6] Infection is the most common reason for revision of primary THR and TKR in Australia, representing 22.7% and 26.6% of all revision procedures performed, respectively.[2] The Australian incidence of PJI is estimated to be 3900 cases annually, with yearly costs exceeding $50 million.[7]

With increasing demand being placed on finite health budgets, improving efficiency in healthcare, minimising complications and reducing unnecessary costs are vital.[8] Modifiable risk factors, such as smoking, poorly controlled diabetes and excessive body mass index (BMI) increase the risk of PJI following THR and TKR.[6,9] Although restricting access to joint replacement in the setting of modifiable risk factors is controversial,[10] interventions to address them preoperatively have been shown to improve outcomes and reduce costs.[11,12]

A 2021 survey of the Arthroplasty Society of Australia (ASA) found that 91% of orthopaedic surgeons who responded restrict access to THR and TKR in the setting of modifiable risk factors.[13] This study surveyed Australian general practitioners (GPs) regarding their perceptions and management of smoking, poor diabetic control and excessive BMI in patients with end-stage hip and knee osteoarthritis prior to joint replacement.

Methods

The survey tool used in the ASA study was modified for use with a GP audience and was piloted by six GPs with experience in managing hip and knee osteoarthritis.[13] The final tool included two demographic questions,

three clinical questions and an optional, free text question (Appendix 1, available online only). No identifiable data were collected. The survey was administered via Qualtrics XM (Seattle, Washington), an online survey platform. Invitations to complete the survey were widely distributed to Australian GPs via Primary Health Networks, University GP departments and GP Facebook groups. The survey remained open from 1 February–31 March 2023.

Statistical analysis of results was performed using Microsoft Excel (Redmond, Washington). Descriptive statistics (counts and proportions) were used to present the main findings. Ethics approval for the study was granted by the Darling Downs Health Human Research Ethics Committee (LNR/2021/QTDD/82223) and ratified by The University of Queensland Research Ethics and Integrity Department (2022/HE000211).

Results

A total of 131 survey responses were received. Respondents had a range of clinical experience: 34% (44/128) had been GPs for less than 10 years; 25% (32/128) had been practising for 10 to 20 years; and 38% (48/128) had over 20 years' experience. Four responses (3%) were received from GP registrars. There were 56 responses (44%) from metropolitan GPs and 72 responses (56%) from rural GPs.

Most respondents attempted to modify risk factors prior to referring a patient with symptomatic hip or knee osteoarthritis to see an orthopaedic surgeon. Over 90% (111/123) attempted to address current smoking; 94% (116/123) attempted to improve diabetic control; and 89% (109/123) tried to reduce excessive BMI prior to referral. Specific HbA1c (glycated haemoglobin) and BMI targets are presented in Figure 1.

Regarding timing of surgery, most respondents felt that it was appropriate for patients to have their hip or knee replacement delayed until risk factors had been modified. For current smokers, 57% of GPs (68/120) thought that surgery should be delayed until cessation; 84% (101/120) felt that diabetic control should be optimised preoperatively; and 74% (89/120) responded that surgery should be delayed until patients had reduced their BMI. Specific HbA1c and BMI targets are presented in Figure 2.

However, many GPs did not believe that modifiable risk factors were absolute contraindications to hip or knee replacement. Over 76% of respondents (90/118) did not feel that current smoking was a contraindication; 46% (54/117) did not think that poor diabetic control precluded surgery; and 43% (51/118) did not believe that excessive BMI was a contraindication to joint replacement. For respondents who did believe that these risk factors were contraindications, specific HbA1c and BMI thresholds varied (Figure 3).

Overall, 47 GPs provided comments in the optional, free text question. A common theme was the challenge primary healthcare providers faced trying to address modifiable risk factors, in particular excessive BMI. A lack of referral pathways for morbidly obese patients, particularly in the public sector, was identified. Several GPs commented that using strict 'cut-offs' for surgery was inappropriate;

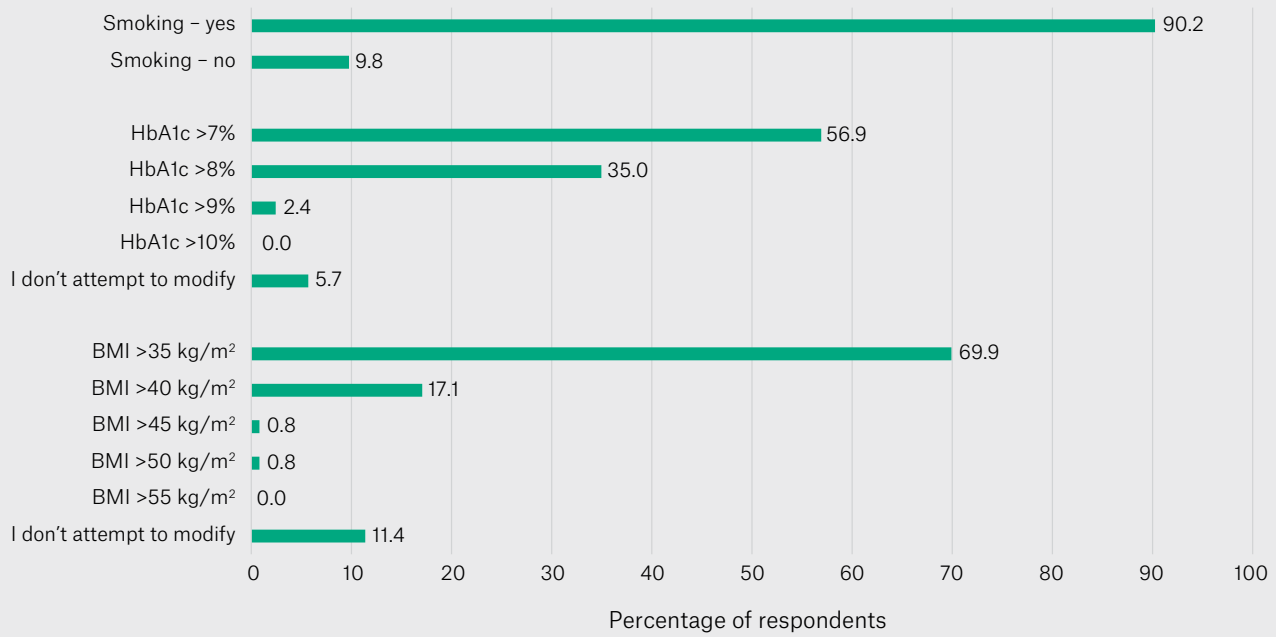

Figure 1. Responses to the question, 'Prior to referring a patient with symptomatic hip or knee osteoarthritis to see an orthopaedic surgeon, do you attempt to modify the following risk factors?'

BMI, body mass index; HbA1c, glycated haemoglobin.

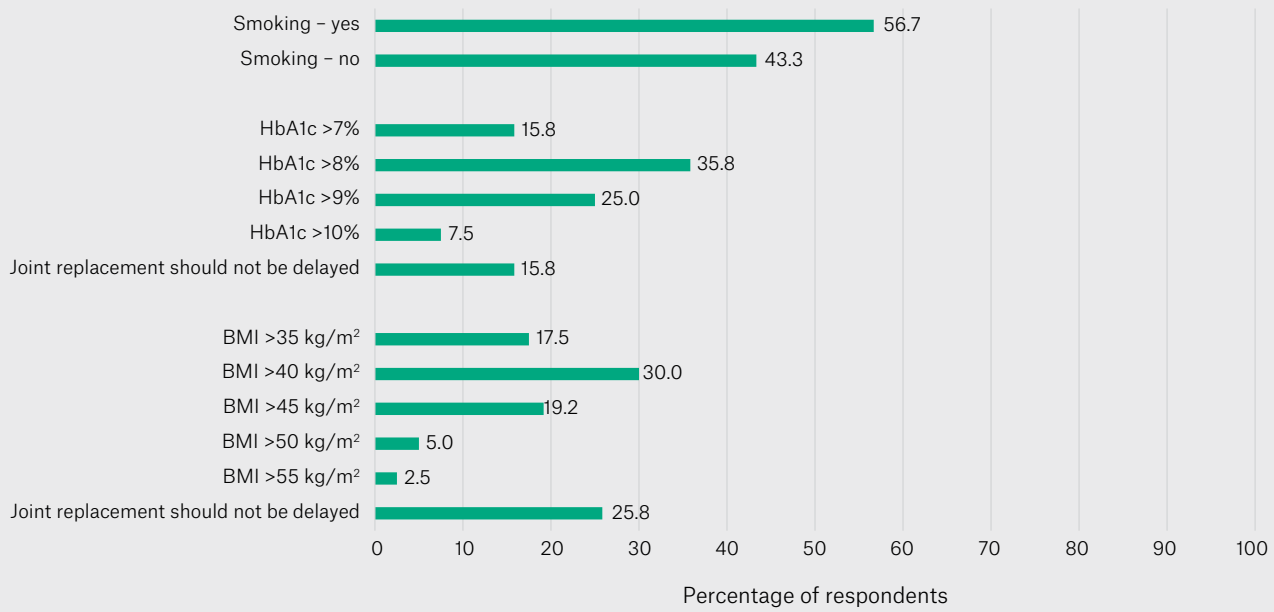

Figure 2. Responses to the question, 'Do you think that patients should have their hip or knee replacement delayed until the following risk factors have been modified?'

BMI, body mass index; HbA1c, glycated haemoglobin.

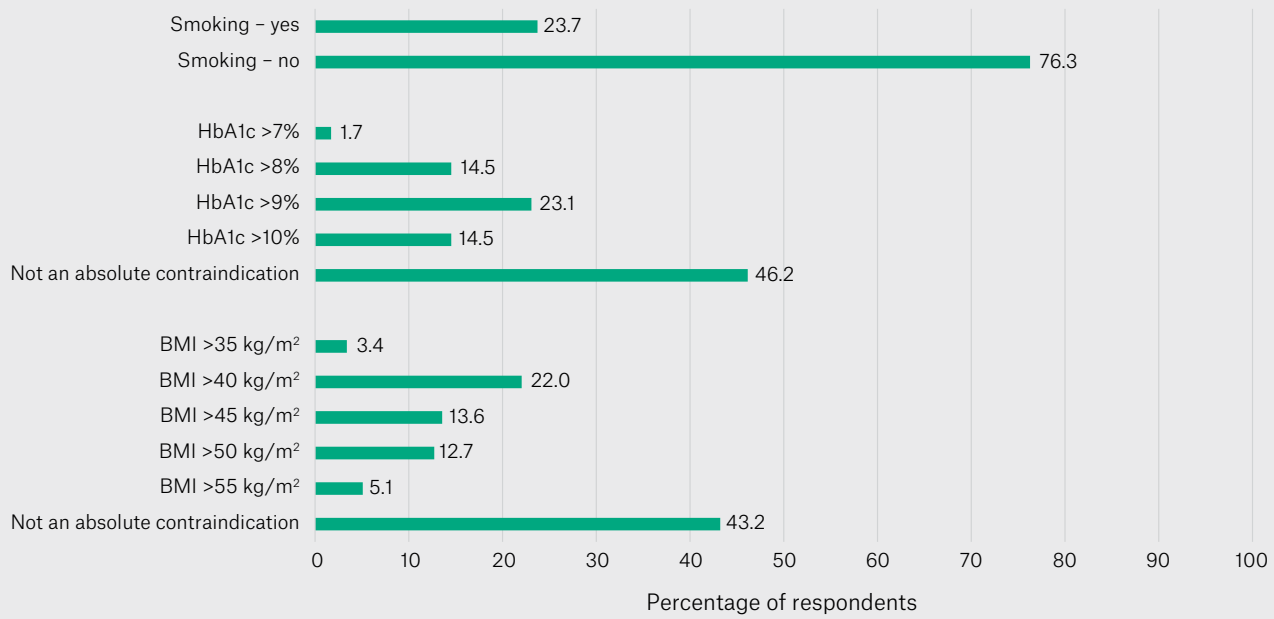

Figure 3. Responses to the question, 'Do you think that the following risk factors are absolute contraindications to hip or knee replacement?'

BMI, body mass index; HbA1c, glycated haemoglobin.

others suggested that if patients had made a significant effort to reduce their BMI but were still morbidly obese, they deserved to undergo joint replacement. In contrast, three respondents stated that health service-imposed restrictions were beneficial in their efforts to assist patients to modify risk factors.

Discussion

The results of this study suggest that Australian GPs are aware of the significance of modifiable risk factors in patients with end-stage hip and knee osteoarthritis requiring joint replacement. Most respondents attempted to address current smoking, poor diabetic control and excessive BMI prior to referral to an orthopaedic surgeon. Similarly, most GPs surveyed felt that joint replacement should be delayed until modifiable risk factors had been addressed. However, many did not believe that current smoking, poor diabetic control or excessive BMI represented absolute contraindications to THR or TKR.

In 2021, a similar survey was distributed to the membership of the ASA, a subspecialty group of the Australian Orthopaedic Association (AOA).[13] Responses were received from 77 of the 121 members, representing a response rate of 64%. Most respondents were experienced, high-volume joint replacement surgeons. Overall, 91% of surgeons restricted access to THR or TKR in patients with modifiable risk factors. The majority (72%) employed a BMI threshold in their practice, although specific cut-offs varied. BMI thresholds of 40, 45 and 50 kg/m^2 were used by 34%, 15%, and 21% of respondents, respectively. Most respondents (85%) did not offer joint replacement to patients with poorly controlled diabetes, and nearly half (46%) restricted access to current smokers. A comparison of Australian surgeons' and GPs' responses is presented in Table 1. An earlier survey of the American Association of Hip and Knee Surgeons (AAHKS) found that American surgeons were more restrictive than Australian surgeons in all domains.[14]

When comparing the results of the present study and the ASA study, it is interesting to note that Australian orthopaedic surgeons are more conservative in offering joint replacement to patients with modifiable risk factors than referring Australian GPs believe is appropriate. The discrepancy in these findings might be explained by a lack of universally accepted guidelines for the management of modifiable risk factors.

As far as we are aware, only one previous study has investigated GPs' perceptions of modifiable risk factors in patients undergoing joint replacement. In 2011, Wright et al reported the results of a survey of orthopaedic surgeons and referring physicians (family physicians and rheumatologists) in Ontario, Canada, regarding indications for TKR.[15] In total, 135 surgeons, 165 family physicians and 111 rheumatologists responded to the survey, representing response rates of 67%, 36% and 79%, respectively. Respondents were asked how 34 different patient characteristics, one of which was obesity, affected their decision to either perform a TKR or refer a patient for TKR. Overall, 42% of surgeons stated that they were less likely to perform a TKR in an obese patient, and 2% felt that obesity was a contraindication to TKR. Similarly, 48% of referring physicians stated that they were less likely to refer an obese patient for a TKR and none felt that obesity was a contraindication to TKR. Interestingly, obesity was one of only two patient characteristics (out of 34 in total) where surgeons and referring physicians agreed on the influence it would have on their decision making.

There has been a dramatic increase in the incidence of global obesity since 1975.[16] Nearly one-third of Australian adults are obese.[17] Obesity is a known risk factor for the development of hip and knee OA and the subsequent need for joint replacement.[18,19] Obesity, and in particular morbid obesity (BMI ≥40 kg/m^2), increases the risk of PJI and other postoperative complications for both THR[20] and TKR.[21,22]

Obesity and type 2 diabetes mellitus (T2DM) often co-exist.[23] Approximately one million Australians (4.1%) have T2DM, and this figure has increased from 3.3% in 2001.[17] Patients with T2DM are at higher risk of PJI and other postoperative complications following joint replacement.[24,25] However, good diabetic control reduces these risks.[26] The Royal Australian College of General Practitioners' guidelines recommend a glycated haemoglobin (HbA1c) of ≤7% (≤53 mmol/mol) in patients with T2DM.[27]

Despite ongoing public health messaging about the detrimental effects of smoking, 2.6 million adult Australians (13.8%) continue to smoke tobacco daily.[17] Smoking increases the risk of PJI and other postoperative complications following THR and TKR.[28,29]

Restricting access to joint replacement in the setting of modifiable risk factors is controversial.[10] Taking morbid obesity as an example, restricting joint replacement to patients with a BMI <40 kg/m^2 would prevent one major complication but would deny 14 patients from having a successful, complication-free procedure.[30] Obese patients have similar improvements in patient-reported outcome measures following THR and TKR compared to non-obese patients, albeit from lower baselines.[2]

However, structured interventions to address modifiable risk factors preoperatively have been shown to reduce complications and costs associated with joint replacement. In implementing a preoperative optimisation protocol, screening for 19 specific risk factors, Bernstein et al demonstrated reduced hospital length of stay (LOS) and reduced costs for patients undergoing THR and TKR.[11] Similarly, by instituting a preoperative

Table 1. Proportion of surveyed Australian arthroplasty surgeons who restricted access to joint replacement in the setting of modifiable risk factors compared to the proportion of surveyed Australian GPs who felt that these risk factors were absolute contraindications to joint replacement[13]

Modifiable risk factor	Surgeons (%)	GPs (%)
Excessive body mass index	71.8	56.8
Poor diabetic control	84.5	53.8
Current smoking	46.5	23.7

risk factor optimisation protocol, focusing on 16 different risk categories, Dlott et al found a reduction in LOS, improved rate of home discharge, and reduced 30- and 90-day emergency department presentations following THR and TKR.[12] The risk factors screened for in these two preoperative optimisation protocols are presented in Table 2. Recommended targets for the modifiable risk factors in this study were included in Wall and de Steiger (Table 3).[9]

Our study has several strengths. As far as we are aware, this is the first study to investigate Australian GPs' perceptions of modifiable risk factors for patients with end-stage hip and knee osteoarthritis requiring joint replacement. The survey tool was designed to facilitate comparison with results from the recent ASA survey.[13] Improving joint replacement outcomes, minimising complications and reducing costs are critical, and it is hoped that the results of this study will stimulate ongoing discussion around this topic.

We also acknowledge several limitations of our study. Unlike the ASA study, we did not distribute the survey to all GPs in Australia, and we are unable to calculate the response rate.[13] Although we attempted to distribute the survey widely, it is possible that the respondents were not representative of the entire Australian GP population. Metropolitan GPs were under-represented, and hence the results might not be generalisable. We did not define metropolitan or rural practice locations, and we acknowledge that there might be differences in the interpretation of these terms. In addition to the three modifiable risk factors included in the current study, the ASA study investigated other medical and behavioural risk factors known to influence the risk of complications following THR and TKR.[13] We elected not to include these other risk factors to shorten the survey and improve the response rate.

Table 2. Previously published preoperative optimisation protocols for patients undergoing total hip and knee replacement[11,12]

Risk factors screened for by Bernstein et al[11]	Risk factors screened for by Dlott et al[12]
Complete blood count	Allergies
Haemoglobin	Medical history
Comprehensive metabolic panel (Chem-12)	Infection risk
Prothrombin time/internationalised normalised ratio	Smoking
Haemoglobin A1c	Obesity
Methicillin-resistant *Staphylococcus aureus* screen	Malnutrition
Electrocardiogram	Cardiovascular disease
Body mass index	Venous thromboembolism
Modified risk assessment and prediction tool	Neurocognitive compromise
Deep vein thrombosis prophylaxis evaluation	Substance dependence
Depression screen	(Suitability for) telerehabilitation
Anxiety screen	Diabetes
Dental screen	Anaemia
Obstructive sleep apnoea screen	Systemic steroid use
Tobacco use	Obstructive sleep apnoea
Lower extremity skin check	Social support
Narcotic use	
Cardiac records/results	
Alcohol use	

Table 3. Recommended preoperative targets for modifiable risk factors[9]

Modifiable risk factor	Recommendation
Obesity	Aim for a BMI <40 kg/m^2 prior to joint replacement
Diabetes mellitus	Aim for a HbA1c of ≤53 mmol/mol (≤7%) prior to joint replacement
Tobacco use	Aim for smoking cessation at least 4 weeks prior to joint replacement

BMI, body mass index; HbA1c, glycated haemoglobin.

Conclusion

The results of this study suggest that Australian GPs are mindful of modifiable risk factors in patients with hip and knee osteoarthritis. Most respondents attempted to address current smoking, poorly controlled diabetes and excessive BMI prior to referral to an orthopaedic surgeon, and they supported delaying surgery until these risk factors had been modified. However, many GPs did not support restricting access to joint replacement.

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Competing interests: CJW, RNdS and CJV are current or former Clinical Directors for the Australian Orthopaedic Association National Joint Replacement Registry. CJW has given paid educational presentations for Stryker, unrelated to this project. CJV is a Director for Knee Research Australia and a former Director and Treasurer for the Australian Orthopaedic Association. TAJ and SK-C have no competing interests to declare.

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Acceptability and feasibility of a chronic breathlessness diagnostic clinical algorithm in Australian primary care

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Background and objective
Chronic breathlessness is a frequent diagnostic challenge in primary care. Our aim is to evaluate the feasibility of a stepwise breathlessness diagnostic algorithm for primary care.

Methods
This mixed-methods study included: (1) a general practitioner (GP) nominal group technique study; (2) focus groups on GPs' views on the algorithm; and (3) analysis of algorithm alignment against patterns of diagnostic referrals and diagnoses of breathlessness presentations (2014–19) from the MedicineInsight primary care electronic health record (EHR) dataset of 1,961,264 patients (405 general practice sites).

Results
All the tests in our algorithm, except for echocardiography, were ranked in the top 10 tests used by most GPs for patients presenting with chronic breathlessness. Themes from the focus group include similarity with current practice and test accessibility. Analysis of EHR diagnostic referrals revealed that all tests in the algorithm are regularly utilised and covered the major tests needed for breathlessness diagnoses recorded.

Discussion
The results of the three studies support the acceptability and feasibility of the clinical algorithm in primary care.

PATIENTS PRESENT WITH SYMPTOMS rather than diseases in clinical practice. However, in practice, most pathways and decision support tools focus on diseases as entry points. Breathlessness is a clinically prevalent symptom reported by approximately 10% of adults in the community[1] and is associated with a broad range of common conditions such as asthma, chronic obstructive pulmonary disease (COPD), heart failure, ischaemic heart disease, deconditioning, anxiety and obesity. It is also associated with less common but treatable conditions such as pulmonary thromboembolic disease, pulmonary hypertension, interstitial lung disease and valvular heart disease.

The multidimensional aspect and myriad possible causes of chronic breathlessness, ranging from respiratory to cardiac to metabolic diseases, mental health and deconditioning, create a major diagnostic challenge for this very common problem, especially in primary care where most patients first present.[2]

In the primary care setting, a study of patients presenting with breathlessness reported that less than 30% had a final diagnosis that was fully concordant with their referral diagnosis.[3] This finding was supported in another study, where less than 40% of breathlessness patients referred to secondary care with heart failure were confirmed to suffer from heart failure.[4] Higher accuracy was reported in a study by Pratter et al in the USA, where 55% of physicians' predictions following history and physical examination were concordant with the final diagnosis.[5] A qualitative study that we conducted revealed that patients and carers experienced misdiagnosis and knowledge gaps of health professionals.[6] Those findings aligned with the results of another study we conducted eliciting the perspectives of general practitioners (GPs), multidisciplinary non-GP specialists and allied health professionals.[7] That study also identified knowledge gaps in diagnostic testing and constraints on access to these tests as a barrier to optimal care for patients presenting with breathlessness.[7]

Previously, we published a proposed stepwise approach for the assessment of chronic breathlessness based on a review of diagnostic capacity of priorly evaluated diagnostic pathways that can ascertain the cause in up to 55% of patients with spirometry, electrocardiography and pulse oximetry as initial diagnostic tests.[8] Furthermore, 65–90% of presentations were diagnosed

when the above approach was combined with a chest X-ray, pathology tests (full blood count, thyroid function and brain natriuretic peptide [BNP]), chest computed tomography (CT) and echocardiography based on the results of prior studies.[8] However, none of the algorithms utilised in the proposed diagnostic clinical algorithm were validated or tested in Australia.

The present study aimed to understand the current general practice diagnostic patterns for patients presenting with chronic breathlessness to evaluate the acceptability and feasibility of implementing our proposed diagnostic clinical algorithm (ie a specific series of tests and their sequence) for chronic breathlessness.

Methods

The study consisted of three parts: (1) a nominal group technique (NGT) consensus GP focus group; (2) a focus group using thematic analysis; and (3) analysis of breathlessness referrals from a de-identified primary care breathlessness database cohort. Ethics approval was obtained from the University of New South Wales (UNSW) Human Research Ethics Committee (ID HC200534 and HC200847). Approval was also obtained from the NPS Data Governance Committee and the Australian Department of Health as custodians of the MedicineInsight dataset (2020-032).

Nominal group technique consensus GP focus group

The NGT was conducted by adapting methods from prior studies to fit a virtual design.[9] Nine GPs across two meetings participated in this process virtually from among 65 GPs (13.8%) who were invited to participate. They were recruited from a list of GPs who had prior engagements with The George Institute for Global Health, UNSW's Department of General Practice and the Western Sydney Primary Health Network (WentWest).

Prior to the focus group, practising GPs were provided with a list of 40 diagnostic tests and possible specialty referrals (the list is included in Appendix 1, available online only) relating to chronic breathlessness for their consideration based on our prior literature review.[8] The focus group was conducted virtually, and participants were asked, in a round robin style, to provide feedback on the list and whether any additional items should be added, based on their experience. New items from the prior focus groups were added to the list of proceeding items. Once no new items were suggested, private voting was completed via an online platform (Qualtrics XM) by ranking the diagnostic tests in response to the prompt 'What diagnostic tests would GPs be confident in ordering and interpreting to aid in their assessment of breathlessness?', with a maximum of 35 tests from the whole set. The diagnostic tests were presented in random order to reduce bias. The online platform auto-tallied the responses to generate the median ranks. These were then discussed, and if concerns were raised about the rank, a final vote was conducted to gain consensus. The results of the ranking process were presented descriptively with measures of inter-rater agreement (kappa, Gwet's AC and percentage agreement). Data analysis was conducted in Stata version 18 (StataCorp).

Qualitative focus group

A convenience sample of practising GPs were recruited from The George Institute, UNSW and health professional public databases using an email invitation. Participants were also asked to suggest peers who might be suitable for the study (snowball recruitment). A semi-structured guide was used for the virtual focus group, where participants were shown the proposed diagnostic clinical algorithm and asked to provide feedback. Focus groups were moderated by AS (male medical graduate, a PhD candidate with prior experience in conducting qualitative studies). Participants were prompted with the question, 'What do you think are the strengths and weaknesses of the pathway proposed?' Beyond our proposed pathway, which was developed based on a prior review of past diagnostic pathways,[8] participants were also presented with two other pathways commonly cited in the literature (Karnani et al [USA][10] and Berliner et al [Germany][11]) developed outside Australia for comparison and contrast. The focus group discussions were recorded and transcribed verbatim. Data were then analysed using thematic analysis using the software NVivo (QSR International) following an approach described by Terry et al.[12] Early coding was performed by AS based on the transcript and field notes. Themes developed were then discussed with the other investigators (CJ, AM, CA and GM) to obtain consensus. Further details regarding the qualitative study can be found in our prior study,[7] which focused on the participants' current experience in assessing and managing chronic breathlessness and the support needed.

Primary care routinely collected data analysis

This study utilised NPS MedicineWise's MedicineInsight primary care dataset of longitudinally collected de-identified electronic health records (EHRs) of 1,961,264 patients from 405 general practice sites in Australia between January 2014 and December 2019.[13] The geographic distribution of practice sites was 53.4% from major cities, 23.7% inner regional and 19.9% outer regional. Approximately one-third of practices were from NSW (34.6%), 22.0% were from Victoria, 20.4% from Queensland and 10.2% from Western Australia. Practices were also based in a range of socioeconomic strata, with 14.5% in Socio-Economic Indexes for Areas (SEIFA) Quintile 1 (most disadvantaged) and 21.9% in SEIFA Quintile 5 (least disadvantaged).[14] In this dataset, a patient is determined to have presented with breathlessness if the term 'breathlessness' or synonyms (eg exertional dyspnoea, shortness of breath and breathing difficulty) were reported as a coded condition or free-text entry in one or more of the Diagnosis, Reason for visit or Reason for prescription fields. Referrals for diagnostic tests recorded in the EHR at any stage of a breathlessness patient's journey (including those recorded one or two days after the breathlessness presentation) were analysed from free text (further details are provided in Appendix 2, available online only). Furthermore, we aimed to identify the main diagnoses in patients presenting with breathlessness and assess whether the tests proposed were feasible and appropriate to include or exclude the diagnoses. Data analysis was conducted in SAS version 9.4 (SAS Institute Inc) and Stata version 18.

Results

Nominal group technique consensus GP focus group

Nine GPs across two focus groups took part in the NGT study. The GPs had overall moderate

agreement in their rankings (kappa 0.56 [95% confidence interval [CI] 0.42–0.70], Gwet's AC 0.60 [95% CI 0.48–0.72], percentage agreement 85.2%).

All the tests in our proposed algorithm except for echocardiography were ranked in the top 10 diagnostic tests employed by most GPs for patients presenting with chronic breathlessness (Table 1).[8] GPs preferred starting with pathology tests then proceeding with imaging, before more functional tests such as spirometry. Among the list of diagnostic tests provided, we included an option relating to 'Response to disease specific therapy' and referral to a variety of specialties (cardiology, respiratory, psychiatry, geriatrics and exercise physiology). For response to therapy, the median rank was 18 (interquartile range [IQR] 15, 35, percentage agreement 86.1%). Among the specialty referrals, the highest median rank was for a cardiology referral, followed by respiratory, although with a wide IQR (Table 1).

Qualitative focus group

Eighteen GPs (nine from the NGT) participated across three focus groups (one from South Australia and the remainder from New South Wales, with medical experience ranging from one to 45 years). Qualitative

Table 1. GPs' diagnostic tests and specialty referral ranking

	n (N=9)	Level in proposed algorithm (1–4)[A]	Median rank in the GP NGT study (IQR)	Percentage agreement (ranked in the same tertile)[B]
Diagnostic tests				
Serum haemoglobin	8	2	2 (2, 5.25)	93.8
Full blood count	9	2	3 (1, 4)	94.4
Basic chemistries	9	NA	3 (2, 4)	100
Thyroid function test	9	2	4 (2, 6)	94.4
Chest X-ray	9	2	6 (5, 7)	100
Chest CT scan	9	3	6 (5, 8)	94.4
Electrocardiogram	9	1	8 (5, 9)	75.0
Oxygen saturation	9	1	8 (5, 10)	94.4
Spirometry	9	1	9 (7, 12)	87.5
BNP	9	2	13 (9, 17)	87.5
Ventilation/perfusion scan	9	NA	13 (12, 20)	75.0
Lung volumes	9	NA	13 (13, 28)	77.8
Arterial blood gas	8	NA	17 (7.5, 20.75)	65.2
Flow volume loop	9	NA	18 (11, 27)	77.8
Echocardiography[C]	9	3	19 (11, 21)	69.4
Specialty referral (assuming they are a diagnostic option)				
Cardiology	8	NA	15 (14, 28)	75.0
Respiratory[D]	3	NA	24 (19.5, 29)	83.3
Geriatrician[D]	3	NA	27 (26.5, 31)	100
Exercise physiology[D]	3	NA	29 (22.5, 31.5)	83.3
Psychiatry	8	NA	33.5 (25, 35)	89.3

[A]Level 4 meant a referral to secondary/tertiary care.

[B]Ranks were divided into three groups for this analysis: 1 to 10, 11 to 20 and 20+ (calculated from among those who rank the test/referral).

[C]Only in the top 15 for one-third of participating GPs.

[D]Only available as an option to 4 GPs in the second focus group.

BNP, brain natriuretic peptide; CT, computed tomography; GP, general practitioner; IQR, interquartile range; NA, not applicable; NGT, nominal group technique.

analysis of the focus group discussions elucidated several themes: similarity to current practice; expansion to include clinical observations; and breaking down silos and access to diagnostic tests.

Similarity with current practice

Compared with the two other algorithms presented (Karnani et al [USA][10] and Berliner et al [Germany][11]), GPs in the focus group found our proposed algorithm (Figure 1) 'more aligned with GP practice' and a 'more helpful pathway for GPs' that 'includes a more holistic review of patients'.[8] The GPs felt the other algorithms had less emphasis on 'psychosomatic causes and were not relevant for GP practice'. The GPs indicated a strong dislike for a prescriptive diagnostic clinical algorithm, especially regarding at which visits to do the test but were more flexible about the test order:

> *The algorithm should not direct which tests should be done in which visit.* (GP4)

Expansion to include clinical observations

Participants identified the need for initial careful history-taking, physical examination and mental health screening being incorporated into the algorithm and had mixed views regarding the utility and access to BNP, lung function testing and echocardiography. GPs wanted 'greater weight' assigned to thorough history-taking and physical examination in the algorithm. These add to the knowledge of the patient from the GPs' long association to provide a holistic assessment of the patient:

> *BNP is not funded* [in practice]. (GP11)

> *We know the patients well* [from a long association], *hence can provide a multifactorial assessment for their breathlessness.* (GP14)

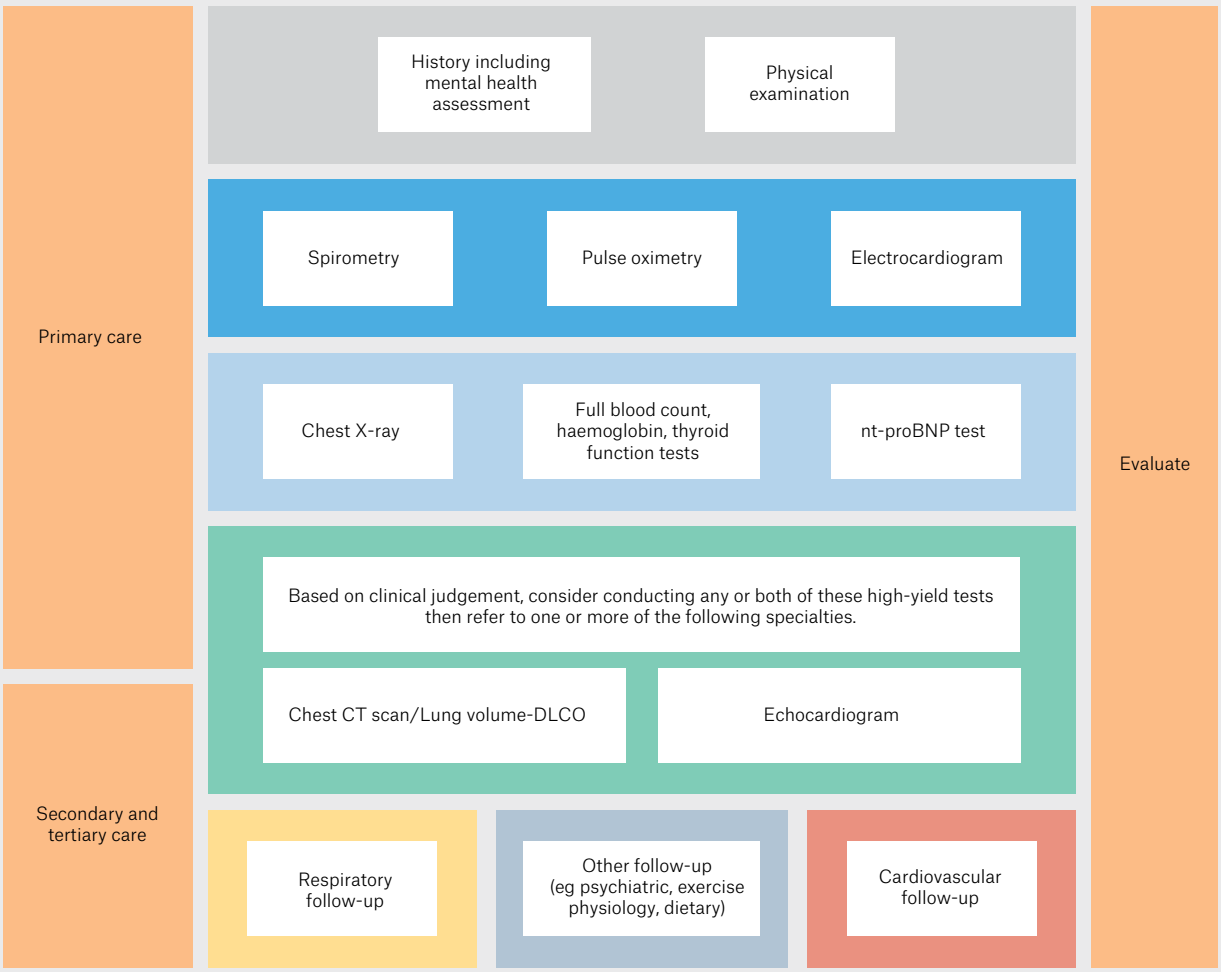

Figure 1. Proposed diagnostic clinical algorithm for chronic breathlessness.

CT, computed tomography; DLCO, diffusing capacity of the lungs for carbon monoxide; nt-proBNP, N-terminal-prohormone brain natriuretic peptide.

Breaking down siloes and access to diagnostic tests in the algorithm

GPs felt that it was important to ensure that chronic breathlessness was not assessed in a 'siloed manner' as either cardiac or respiratory but was evaluated holistically. Relating to referrals, GPs raised the usefulness of 'co-located specialty clinics', such as a 'complex breathlessness clinic' to which they could refer patients who are undiagnosed despite going through the whole diagnostic clinical algorithm rather than to individual specialties, as well as access to secondary care specialists without the need for a referral. They advocated for funding reform to support GP access to diagnostic tests in this breathlessness diagnostic clinical algorithm as some, such as BNP, are currently not reimbursed:

> *A key thing is to try to have one place to go for those with chronic breathlessness.* (GP10)

> *Something like a metabolic or obesity clinic that's issue specific rather than needing to refer to gastroenterology, cardiology etc in siloes.* (GP12)

> *How can we evaluate the heart and lungs together?* (GP16)

> *Phoning a consultant* [in secondary care] *as decision support.* (GP2)

> *Need to consider context, underlying medical conditions and psychosocial setting at the beginning. Chest high-resolution CT scan and echocardiography are easier to arrange than formal lung function testing or DLCO* [diffusing capacity of the lungs for carbon monoxide]. (GP14)

Primary care routinely collected data analysis

During the MedicineInsight data collection period (2014–19), 78,912 patients (4.02%) had at least one presentation with breathlessness for a total of 120,218 unique consultations for breathlessness.

All the diagnostic tests in the proposed algorithm are in common use. In this dataset, approximately 45% of patients had a referral for a full blood count followed by approximately 40% for chest X-rays. Considering the prevalence of cardiovascular and airway disease, there was very infrequent use of simple tests such as BNP even when compared to more advanced and expensive tests such as chest CT scans and echocardiography (Table 2).

Twelve diagnoses accounted for over 80% of patient presentations (Table 3). These were diagnoses obtained not only from the consult for breathlessness but included those recorded in follow-up visits up to November 2021. For the most common causes of breathlessness and concurrent diagnoses, the diagnostic tests covered in the pathway would allow GPs to either include or exclude a diagnosis in most cases.

Discussion

Findings from the three studies support the acceptability and feasibility of our proposed diagnostic clinical algorithm in primary care, although modifications might be needed in its implementation to align with existing practice realities and enhance its uptake.

Previous studies have reported that over 70% of individuals with breathlessness had multimorbidity (two or more comorbid conditions), and 90% of those with COPD aged ≥45 years[15] had at least one other chronic condition. Regardless, our analysis of the primary care dataset found that a relatively limited number of diseases were diagnosed in patients presenting with breathlessness, all of which could be further assessed using diagnostic tests in the proposed algorithm. The conditions identified were aligned with the major diseases identified in the National Breathlessness Survey of over 10,000 Australian adults, of which over 4000 had breathlessness, where they were asked to self-report the presence of a prior medical diagnosis of major conditions relating to breathlessness.[1]

The present study identified that, in general, GPs' diagnostic tests referral align in practice with their theoretical ranking (ie pathology tests being most common). An Australian primary care study of 7255 breathlessness consultations between 2000 and 2009 reported that 26.3% ordered a radiological test and 62.4% a pathology test, which is a similar pattern to our findings.[16] There was moderate agreement across the diagnostic tests between the GPs, but a wide IQR for the ranking of many diagnostic tests.

However, the present study highlights a significant gap between the tests GPs order and the diagnoses made. For example,

Table 2. Comparison of diagnostic tests in the proposed pathway as a proportion of patients with breathlessness in the primary care dataset

Diagnostic test	Level in proposed algorithm (1–4)[A]	Proportion of patients n (%) (N=78,912)
Serum haemoglobin	2	34,975 (44.3)
Full blood count	2	
Chest X-ray	2	29,657 (37.6)
Thyroid function test	2	22,473 (28.5)
Electrocardiogram	1	16,370 (20.7)
Spirometry	1	9260 (11.7)
Echocardiography	3	5055 (6.4)
Troponin	NA	4467 (5.7)
Chest CT scan	3	4523 (5.7)
BNP	2	2739 (3.5)
Oxygen saturation	1	No data

[A]Level 4 meant a referral to secondary/tertiary care.

BNP, brain natriuretic peptide; CT, computed tomography; NA, not applicable.

although 30.5% and 22.7% of patients had an asthma and COPD diagnosis, respectively, across the six-year period, only 12% of diagnostic test referrals were for spirometry testing. This finding contrasts with previous studies in Australian general practice, which have reported over-testing as a significant problem but is in keeping with observations that 25–75% of tests are not supported by evidence or expert opinion.[17] Overall, these results strongly support a need to develop pathways and services that would allow a more focused, efficient approach to assessing individuals presenting with breathlessness. They suggest that despite reflecting the prevalence of conditions associated with breathlessness in Australian practice, the proposed algorithm would have a greatly reduced efficacy if current diagnostic practices remain unchanged.

Spirometry is an essential tool in primary care for the assessment and management of breathlessness-associated conditions such as asthma and COPD. Our findings suggest that in patients presenting with breathlessness, spirometry was neither top of mind nor appropriately utilised, possibly leading to poorer patient outcomes. A Canadian population-based study showed that the use of spirometry testing to diagnose COPD patients in the ambulatory setting was associated with a 20% reduction in risk of death and admission to hospital.[18] Although we note that the use of routinely collected data from primary care meant that there would be cases where diagnostic tests such as spirometry were conducted but not recorded as they were not digitised,[19] previous studies in Australia and other settings have found similarly low rates of spirometry utilisation in primary care.[20,21] As suggested in other studies,[22,23] practical barriers such as knowledge gaps in technique and interpretation, along with poor access for referral services, must be addressed to solve this underutilisation of a vital diagnostic test.[7,24] Solutions identified in prior studies include changes to current workflows; funding reform (as highlighted by GPs in our focus group to appropriately reimburse GPs for the cost of spirometry); and support for technical training, quality control and interpretation, including through decision support systems.[25] Beyond this, the use of alternative tools that provide similar clinical utility to spirometry, such as forced oscillation technique (FOT) and breathomics, should be explored, especially in the current post-COVID-19 spirometry restart period.[26,27]

Similarly, there is low utilisation of BNP testing to screen for heart failure; a test recommended in clinical guidelines.[28] Similar healthcare environments such as Europe and the UK provide public funding for this test to triage patients requiring cardiac imaging and cardiology assessment, as well as facilitate early diagnosis of heart failure in practice.[29] Prior studies have shown that low N-terminal-prohormone brain natriuretic peptide (NT-proBNP) levels (≤125 pg/mL according to the European Society of Cardiology and ≤400 pg/mL according to the National Institute for Health and Care Excellence, UK) were both highly specific in excluding patients who did not have heart failure.[30] However, a population-level study in the UK found that most patients (76.7% in 2017) with heart failure had no BNP test done prior to a heart failure diagnosis, suggesting that ongoing education and use of evidence in clinical pathways is vital.[29] Our previous GP focus group found reimbursement and access to be major barriers to uptake in Australia.[7] There is a need to support greater access to this high-yield test in primary care for which health economic studies have demonstrated cost savings when implemented widely.[31,32]

Independent of our study, another group in the UK also recently proposed a possible diagnostic clinical algorithm for chronic breathlessness,[33] which included similar diagnostic tests and is being tested in 10 practices across Leicestershire. A major difference in their implementation is a focus on having all these tests and examinations completed within one month of a patient's presentation for breathlessness rather than following a stepwise approach prioritising certain tests before others as with our proposed algorithm. If such an approach is implemented at scale, it could significantly raise the costs of assessment and increase pressure on the health system considering the

Table 3. Prevalence of conditions (concurrent diagnoses) among adults presenting with breathlessness (in order of prevalence)[A]

Disease	N (%)[B]	Related diagnostic
Lower respiratory tract infection	54,322 (68.8)	Chest X-ray, full blood count
Depression	27,821 (35.3)	NA
Anxiety	23,410 (29.7)	NA
Asthma	24,062 (30.5)	Spirometry
Coronary heart disease	18,366 (23.3)	ECG, echocardiogram, troponin
COPD	17,914 (22.7)	Spirometry, chest X-ray, thoracic CT scan
Heart failure	14,517 (18.4)	ECG, BNP, echocardiogram
Atrial fibrillation	14,051 (17.8)	ECG
Pulmonary embolism	4005 (5.1)	Thoracic CT scan
Hyperthyroidism	2636 (3.3)	Thyroid function test
Lung cancer (including suspect cases)	1218 (1.6)	Chest X-ray, thoracic CT scan
Rheumatic heart disease	631 (0.8)	ECG, echocardiogram, chest X-ray

[A]An individual can have more than one condition. These diagnoses were those noted by general practitioners in the dataset as of November 2021.

[B]Percentages calculated after excluding cases with incomplete details (n=2756).

BNP, brain natriuretic peptide; COPD, chronic obstructive pulmonary disease; CT, computed tomography; ECG, electrocardiogram; NA, not applicable.

prevalent nature of breathlessness in the community.

Beyond diagnostics, our previous study with GPs, non-GP specialists and allied health professionals identified a detailed medical history and physical examination as an essential part of a clinical algorithm to ascertain the cause of chronic breathlessness.[7] This is especially so given that multimorbidity is present in over 70% of individuals with clinically relevant breathlessness. Further work could build upon this current study to develop a practical clinical algorithm that supports an efficient, evidence-based prioritisation of these diagnostic tests and leverages technologies such as machine learning. This would facilitate combinations of history, physical examination and diagnostic results from tests that are feasible in primary care to improve the accurate diagnosis in patients presenting with chronic breathlessness.

A limitation of the qualitative components of this study was that most GP participants were based in NSW and their responses might not be representative of the realities of practice in states and countries with different health system structures. However, we utilised results from a national primary care EHR dataset to complement the findings from the qualitative study, which includes practices from throughout Australia in its network. We do note, however, a limitation of the EHR study is that we cannot ascertain chronicity of the breathlessness presentation, and it was not possible to distinguish a new diagnosis from worsening of a pre-existing diagnosis. We reported the diagnoses for individuals at the end of the follow-up period, which includes the possibility that multiple visits, sometimes over years, can be required before a definitive diagnosis is achieved in an individual presenting with breathlessness.

Conclusion

The results of the three studies support the acceptability and feasibility of the proposed clinical algorithm in primary care, although modifications through testing it in practice will be needed to align with existing practice realities and enhance its uptake.

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Alcohol consumption in early middle-aged Australian women and access to primary healthcare services: A cross-sectional study

Suzannah Bownes, Alexa Seal, Catherine Harding

Background and objective
This study describes the prevalence of risky alcohol consumption in Australian women aged 40–45 years. It explores the relationship between demographic factors and access to and usage of primary healthcare services.

Methods
Data were obtained from the Australian Longitudinal Study on Women's Health, Survey 8 (1973–78 cohort). Descriptive statistics and univariate logistic regression were used to assess associations of specific factors with risky alcohol consumption.

Results
Eleven per cent of respondents reported drinking >10 standard drinks per week. These 'risky alcohol drinkers' attend general practice as frequently as low-risk drinkers despite perceived poorer health. They reported 'rarely or never' seeing the same general practitioner (GP) and described themselves as having 'poor' access to a GP that bulk bills.

Discussion
This study provides unique insight into the primary healthcare attendance patterns and health status of early middle-aged Australian women who are 'risky alcohol drinkers'. They do not consistently see the same GP, which might present challenges in identifying them in primary care.

ALCOHOL CONSUMPTION is associated with morbidity and mortality and is a public health priority.[1] It is the fifth highest risk factor contributing to the disease burden in Australia.[2] Alcohol has a complex role in Australian society, and the normalisation of alcohol consumption contributes to its potential for serious harm.[3,4] In 2019, 16.8% of Australians exceeded the lifetime risk recommendations and 25% were binge drinking at least monthly.[5] This drinking culture makes screening for and addressing alcohol misuse difficult, as many people do not identify themselves as someone with a potentially harmful relationship with alcohol.

There is increasing awareness regarding the levels of alcohol consumption among middle-aged Australian women;[5–7] however, there is limited research on the drinking levels of middle-aged Australian women and related factors. The reasons for risky alcohol consumption among middle-aged Australian women are complex and include social and biological explanations. It has become increasingly socially acceptable for women to drink alcohol to the same degree as that considered acceptable for men.[8] This has, to some extent, been driven by improved gender equality in environments such as the workplace and an imitation effect whereby some women have adopted male patterns of behaviour.[9] A study in the USA identified that binge drinking has increased in middle-aged women, especially in women of high socioeconomic status.[10] Women have been found to be more likely than men to drink when they are stressed and to regulate emotion.[11] However, there is a recognised phenomenon whereby having multiple roles, including work and home life, is protective against risky alcohol consumption in women.[9] Therefore, the role in stress-mediated risky alcohol consumption needs further exploration.

Alcohol misuse is associated with a range of short-term and long-term health consequences. Women who drink are more likely to develop medical problems than men,[12] whereas women with moderate-to-high levels of alcohol consumption are at increased risk of obesity and liver cirrhosis,[13] cardiovascular diseases, haemorrhagic stroke and multiple cancers.[8] This difference has been attributed to physiological gender differences,[9,14] and the health risks are exacerbated by ageing.[6]

Identifying risky alcohol drinking in middle-aged Australian women is a challenge for both patients and primary healthcare providers. Many middle-aged Australian women do not recognise their drinking as harmful.[15] Dare et al[16] reported that women place importance on the context in which alcohol is consumed rather than the quantity, thus creating opportunities for overconsumption. Further, healthcare providers might also fail to identify women who are drinking above the recommended amount. It has been suggested that because older women stereotypically drink less than older men, healthcare providers might be less

likely to recognise at-risk drinking and alcohol problems in this population.[6] In addition, older women who abuse alcohol are less likely to seek help in specialised addiction treatment settings.[6]

Middle-aged women have historically been under-recognised as risky drinkers in alcohol research, which is biased towards all-male samples; thus, this group is under-screened for risky alcohol consumption.[17] The primary care setting provides an opportunity to identify and support women, as general practitioners (GPs) are usually the first port of call in the Australian healthcare system. However, it is not known to what extent middle-aged risky drinkers in Australia present to general practice. The Bettering the Evaluation and Care of Health (BEACH) data suggested that alcohol intake for women who have presented to Australian generals practice has decreased from 23.5% in 2016–17 to 20.3% in 2015–16.[18] However, this is likely due to decreasing alcohol consumption among younger women,[4] and the report does not consider middle-aged women as a group. This study explores the relationship between early middle-aged Australian women who drink alcohol above the recommended amount, demographic factors, and their access to and usage of primary healthcare services.

Methods

Data were obtained from the Australian Longitudinal Study on Women's Health (ALSWH), a longitudinal population-based study of 57,000 women (four cohorts) funded by the Australian Federal Government Department of Health since 1996. Permission was obtained from the ALSWH Data Access Committee (EOI A853) for access to Survey 8 from the 1973–78 birth cohort (with participants aged 40–45 years when they completed Survey 8 in 2018). This group is respectfully referred to as 'early middle-aged' throughout this paper.

Risky alcohol drinking was the primary outcome variable. A composite variable titled 'risky alcohol drinking' was based on 'How often do you usually drink alcohol?' (Y8Q59) and 'When you drink alcohol, how many standard drinks do you usually have?' (Y8Q60). Both questions represent frequency and quantity of alcohol consumption. In this study, 'risky alcohol drinking' was based on the updated National Health and Medical Research Council 2020 guidelines[19] and defined as consuming more than 10 standard drinks per week. Our composite variable was calculated accordingly (Table 1).

Demographic variables of interest included area of residence (Accessibility/Remoteness Index of Australia [ARIA+] score), number of children living at home, ability to manage on available income (difficult some or all of the time and impossible responses were categorised as 'struggling to manage on available income') and highest qualification achieved. Health-related variables of interest included self-rated health status, diagnosis of depression or anxiety, current illicit drug use and smoking status. Additional factors included general practice and hospital attendance in the past 12 months, whether they usually see the same doctor and access to a GP who bulk bills.

Statistical Package for the Social Sciences (SPSS) Version 29 (IBM Corp., Armonk, NY, USA) was used for data analysis with an alpha of <0.05. Descriptive statistics were used to describe the groups, and Pearson's chi-square was used to compare categorical variables between groups. Binary logistic regression was used to obtain odds ratios (ORs) and 95% confidence intervals (95% CI). Participants with missing data for variables of interest were removed from the analysis.

Ethics approval was obtained by The University of Notre Dame Australia Human Research Ethics Committee (2021-030S).

Results

After removing women with incomplete data, 720 (n=720/6352, 11.3%) women who responded to Survey 8 were identified as 'risky alcohol drinkers' (consuming more than 10 standard drinks per week). The proportion of 'risky alcohol drinkers' with diagnosed depression (20.3%, P<0.001) and anxiety (16.7%, P=0.010) was higher than in 'low-risk drinkers' (15.1% and 13.2%, respectively). Whereas almost two-thirds of 'risky alcohol drinkers' reported smoking at least 100 cigarettes in their lifetime, only around one-third of low-risk drinkers reported having

Table 1. Risk of harm in the longer term associated with alcohol consumption based on the National Health and Medical Research Council guidelines[19]

		When you drink, how many standard drinks do you usually have?				
		I don't drink alcohol	1 or 2 drinks	3 or 4 drinks	5 to 8 drinks	9 or more drinks
How often do you usually drink alcohol?	Never	N	N	N	N	N
	Less than once a month	N	L	L	H	H
	Less than once a week	N	L	L	H	H
	1 or 2 days	N	L	L	H	H
	3 or 4 days	N	L	L	H	H
	5 or 6 days	N	L	H	H	H
	Every day	N	L	H	H	H

H, high risk (>10 drinks/week); L, low risk (≤10 drinks/week); N, non-drinker.

done so (*P*<0.001, Table 2). Less than 30% of 'risky alcohol drinkers' were of normal weight versus 42% of 'low-risk drinkers' (*P*<0.001).

Women who were 'risky alcohol drinkers' had increased odds of having no children living at home, living outside a major city, struggling to manage on their current income and not having obtained higher education (Table 3). In addition, 'risky alcohol drinkers' had increased odds of being a current smoker, a current illicit drug user, having self-reported 'fair or poor' health and having diagnosed depression and anxiety. 'Risky alcohol drinkers' were 1.71-fold more likely to be overweight or obese than 'low-risk drinkers' (95% CI 1.44–2.03).

Attendance at a GP was similar between 'risky alcohol drinkers' and 'low-risk drinkers' (0–9 standard drinks per week, n=5632) (Table 2). Approximately 60% of both risky and low-risk alcohol drinkers reported consulting a GP at least three times in the past 12 months; however, 'risky alcohol drinkers' were more likely than 'low-risk drinkers' to report that they 'rarely or never' see the same GP (OR 1.42, 95% CI 1.04–1.94) and have 'poor' access to a GP who bulk bills (OR 1.24, 95% CI 1.03–1.49; Table 3).

Table 2. Characteristics, self-reported health status and general practice attendance of Australian women aged 40–45 years by drinking risk status

Characteristic	High risk n (%)	Low risk n (%)	*P*-value
Relationship status			
Single	178 (24.7)	1208 (21.4)	0.045
Coupled	542 (75.3)	4424 (78.6)	
Children living with them	513 (71.3)	4455 (79.1)	<0.001
Lives outside major city	332 (46.1)	2335 (41.5)	0.017
Weight status			
Normal weight	208 (29.4)	2306 (41.7)	<0.001
Overweight	232 (32.8)	1588 (28.7)	
Obese	267 (37.8)	1640 (29.6)	
General health			
Excellent	64 (8.9)	799 (14.2)	<0.001
Very good	264 (36.7)	2354 (41.8)	
Good	293 (40.7)	1858 (33.0)	
Fair	88 (12.2)	508 (9.0)	
Poor	11 (1.5)	113 (2.0)	
Depression	146 (20.3)	853 (15.1)	<0.001
Anxiety	120 (16.7)	742 (13.2)	0.010
Smoked >100 cigarettes ever	468 (65.0)	1987 (35.3)	<0.001
Ever used illicit drugs	570 (79.2)	3030 (53.8)	<0.001
Has been pushed, grabbed, shoved, kicked or hit	155 (16.3)	565 (10.5)	<0.001
Consultations with a GP in the past 12 months			
None	48 (6.7)	353 (6.3)	NS
1–2 times	238 (33.1)	2017 (35.8)	
3–4 times	215 (29.9)	1606 (28.5)	
5–6 times	120 (16.7)	832 (14.8)	
7–9 times	48 (6.7)	384 (6.8)	
10–12 times	21 (2.9)	198 (3.5)	
>12 times	30 (4.2)	242 (4.3)	

GP, general practitioner; NS, non-significant.

Discussion

Results from this study are consistent with the literature that suggests middle-aged women are a high-risk group for alcohol consumption. According to the Australian Institute of Health and Welfare, 11% of Australian women aged 35–44 years were drinking alcohol above the recommended amount,[4] which is similar to the proportion found (11.3%) in Australian women aged 40–45 years who responded to Survey 8. This is in contrast to only 6.1% of women aged 18–24 years.[5]

This study also found that early middle-aged women who drink alcohol above the recommended level attend general practice at similar rates to women classified as low-risk drinkers. This presents an opportunity to target these women with brief interventions if effectively screened. Current recommendations suggest that all people presenting to general practice should be annually or opportunistically screened for alcohol consumption.[20,21] However, in 2021, Mauro et al[22] reported that 'over a quarter of older adults who used alcohol were not asked about their drinking, and older women were less likely than men to discuss alcohol use with providers'. Screening might be less effective at targeting middle-aged women who are 'risky alcohol drinkers' as this group 'rarely or never' sees the same GP and reported having poor access to a GP who bulk bills. This might partially explain why a higher proportion of women who were 'risky alcohol drinkers' self-reported their health status as 'fair' or 'poor', as research shows that continuity of care in general practice benefits patient satisfaction and reduces mortality rates.[23]

Several characteristics were associated

Table 3. Odds ratio for risky alcohol drinking (>10 standard drinks per week) in Australian women aged 40–45 years

	Risky alcohol drinking	
	Odds ratio	95% confidence interval
Demographics/experience		
Lives outside a major city	1.21	1.03–1.41
No higher education	1.63	1.39–1.90
No children living at home	1.53	1.29–1.82
Struggling to manage on current income	1.34	1.15–1.57
Has been pushed, grabbed, shoved, kicked or hit	1.67	1.38–2.02
Health status		
Self-reported 'fair or poor' health	1.29	1.02–1.62
Overweight or obese	1.71	1.44–2.03
Current smoker	4.08	3.37–4.94
Illicit drug use in the past 12 months	3.26	2.71–3.94
Depression	1.42	1.17–1.73
Anxiety	1.32	1.07–1.63
Health behaviours		
Rarely or never sees the same GP	1.42	1.04–1.94
Poor access to bulk-billing GP	1.24	1.03–1.49

GP, general practitioner.

Box 1. Considerations for general practitioners (GPs) when raising alcohol consumption in consultations

- Ensure that you screen annually for alcohol use disorder (in addition to exploring illicit drug use and smoking)
- Screening is particularly important in patients with a diagnosis of depression or anxiety
- Endeavour to ensure that complex patients see the same GP, if possible
- Address limiting alcohol to 2 standard drinks or less per day in opportunistic discussion

with risky alcohol drinking, which might be useful for identification of at-risk women in the primary care setting. In this study, a diagnosis of depression or anxiety was associated with risky alcohol drinking. These findings are in accordance with alcohol research that has previously described comorbid diagnosis of anxiety and mood disorders. It is known that one-third (36%) of people with alcohol use disorders also have a diagnosis of either anxiety or depression, and people who drink alcohol above the recommended amount are four-fold as likely to have a mental illness.[3] In the current study, women aged 40–45 years living outside a major city were 1.2-fold more likely to drink alcohol above the recommended amount, which is similar to national data reporting that women living in outer regional and remote settings are 1.5-fold more likely to exceed lifetime risk compared to women living in major cities.[24] This disparity reflects deeply ingrained cultural attitudes in rural areas, which equate excessive alcohol consumption with mateship and perceive the harmful consequences of drinking to be a better alternative to social isolation.[25] Box 1 summarises some considerations for GPs when raising alcohol consumption during patient consultations.

The limitations of this study are those common to self-reported, longitudinal surveys, including selection bias and attrition risk. The ALSWH participant group has been benchmarked to Australian Bureau of Statistics Census data and the cohorts have been shown to over-sample Australian-born women and women with university degrees.[26,27] Further, it is difficult to accurately establish the true level of alcohol consumption as middle-aged women are known to under-report alcohol consumption in survey responses.[28]

Future research into alcohol consumption in middle-aged Australian women should examine the effects of the COVID-19 pandemic. A preliminary study demonstrated that alcohol consumption by Australians throughout the COVID-19 pandemic increased most in women aged 35–44 years.[29] The reported reasons for this included spending more time at home and increased stress. The study also found that this increase in alcohol consumption correlated to the increased carer responsibilities felt by women throughout the pandemic. Further, the social isolation that has been mandated in Australia might place vulnerable individuals at risk of excessive alcohol consumption, and the consequences of this need to be anticipated.[30] It might also be useful to determine which combination of factors are the best at demographically profiling at-risk women to allow more targeted screening by healthcare providers.

In conclusion, this study demonstrated that the prevalence of risky alcohol drinking among a group of surveyed middle-aged Australian women is comparable to known drinking levels in national data. This is one of the first studies to examine the primary healthcare behaviours of middle-aged Australian women who exhibit risky drinking behaviours. As they do not consistently see the same GP, there might be challenges in identifying these at-risk patients in primary care.

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The impact of preparation time on accreditation performance within Australian general practices

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Background and objective
Australian general practices are highly involved with accreditation programs; however, there is evidence to suggest variability in their levels of performance. The aim of the current study was to determine the association with between several metrics of preparation with accreditation performance outcomes.

Methods
Several metrics were synthesised that measured preparation time to general practice accreditation. Performance outcomes were: (1) conformity to 124 indicators of the standards; (2) time to remediate indicator non-conformities; and (3) level of assistance required.

Results
A greater number of months between registration with the accrediting agency and practice accreditation expiry date was associated with higher indicator conformity at the site visit (OR=1.04, *P*=0.001), as well as less time (ß=−0.02, *P*=0.002) and less assistance (ß=−0.66, *P*=0.02) to remediate non-conformant indicators post site visit.

Discussion
Adequate preparation time for several components within the accreditation framework for general practices were associated with small-to-moderate improvements in key performance outcomes.

ACCREDITATION PROCESSES have been developed to enhance quality and safety outcomes across several healthcare sectors.[1] Accreditation programs originated in hospital sectors; however, their implementation in general practice is now prevalent.[2,3] Australian general practices have shown a high rate of engagement with accreditation (~84% were accredited in 2020), which might be partly due to economic incentives.[4] However, concerns regarding levels of active implementation of quality assurance processes reflective of contemporary standards have been raised.[5]

Australian general practice accreditation is a multi-step process involving registration with an accrediting agency, completion of a self-assessment, an onsite surveyor audit according to the fifth edition standards,[6] and remediation of any non-conformant indicators, within a three-year cycle.[7] Registration with the accrediting agency might occur up to 18 months prior to a practice's accreditation expiry date, allowing for an appropriate period for facilitated learning and communication between the practice and the accreditation agency.[8] An ample period between assignation of an accrediting agency and the re-accreditation expiry provides sufficient opportunity to implement quality assurance processes in preparation for the site visit. The impact of preparation time between general practices and accrediting agencies with site-visit performance has not, until now, been empirically evaluated.

A measure of performance within the general practice accreditation process is the rate of indicator conformity at the site visit. Survey visits are completed by a two-person team that includes at least one general practitioner (GP),[6] and determines conformance to 124 indicators set out by The Royal Australian College of General Practitioners (RACGP).[6] Practices are then presented with a report reflecting their compliance with all indicators and provided with 65 businesses days to remediate any indicator non-conformity, prior to an accreditation certificate being issued. Performance at the site visit varies between practices,[9-11] and it is of particular interest to investigate whether time to prepare for the site visit between practices and accrediting agencies partially explains this variability in performance.

Two further outcomes within the accreditation process reflective of performance after the site visit are available. These include: (1) time to remediate non-conformant indicators; and (2) the number of transactions between practices and the accreditation agency required to remediate non-conformant indicators. These metrics are critical to the operational capacity of accrediting agencies and extend accreditation timelines for general practices.[5] It is important to identify predictors for practices that require a longer time and greater support to remediate non-conformant indicators, so resources (eg increased support from the agency) can by suitably distributed.

It is the aim of the current study to evaluate the association between several metrics reflective of the time to prepare for the site visit with: (1) indicator conformity at the site visit; (2) the time to remediate non-conformant indicators after the site visit; and (3) the level of assistance required to remediate non-conformant indicators after the site visit.

Methods

Data sources and study population

The data encompass consecutive Australian general practice accreditation cycles made between December 2020 and July 2022. Data were recorded from the practice prior to, during and soon after the accreditation site visit using a proprietary web-based application commissioned by the accrediting agency. As part of the National General Practice Accreditation Scheme, data are routinely reported to the Australian Commission on Safety and Quality in Health Care for performance monitoring.[7] Surveillance visits, non-standard medical practices and after-hours practices were excluded from the analyses.

The Macquarie University Human Research Ethics Committee confirms that our project is exempt from ethical review.[12]

Study variables

Time variables

Three variables were synthesised as measures of the time to prepare for general practice accreditation. These variables represent key periods throughout the accreditation process, with less time potentially representing reduced ability to prepare and implement quality assurance processes important for obtaining positive outcomes from the accreditation processes. Registration reflects the initial process in assigning an accrediting agency by general practices. The specific time periods available include:

- the period between date of registration with the accreditation agency and current expiry date of the practice accreditation cycle (months). Practices who were new to the accreditation process (had no current expiry date) were removed from analyses utilising this variable
- the period between date of registration with the accreditation agency and the planned site visit (months)
- the period between submission of the self-assessment and the planned site visit (months).

Indicator conformity

Met and *not met* compliance (binary) scores for each indicator from the site visit were provided. Site visits were completed by a two-person team and includes at least one GP. The GP surveyor must have at least five years' full-time equivalent experience as a vocationally registered GP and be working at least two sessions a week in face-to-face patient contact in an accredited practice.[6]

Time to remediate indicator non-conformity

Practices are provided with 65 business days to remediate any non-conformant indicators, with assistance from the accrediting agency. The actual number of business days required by practices to remediate all non-conformant indicators was counted. A longer time to remediate non-conformant indicators might be related to a higher number of non-conformant indicators or reduced compliance with the accreditation process.

Transactions required to remediate indicator non-conformity

Following the site visit, the accreditation agency will submit a report detailing the outcome of the assessment and provide recommendations to the practice on how to remediate indicators identified as non-conformant. The total number of transactions between the accreditation agency and practice were recorded. A higher number of transactions with the accreditation agency during this period represents greater assistance required by the practice to remediate non-conformant indicators.

Confounders

Available confounding variables were included in statistical models described below. These were the size of the practice (GP headcount), number of previous accreditation cycles made with the accreditation agency, and an urban or rural location.

Statistical analysis approach

Practice characteristics were displayed as counts (numbers) and percentages for categorical variables and means and standard deviations for quantitative variables. All analyses were conducted in STATA v17 (StataCorp).[13]

We evaluated the association between time variables prior to the site visits with indicator conformity assessed at the site visit using separate mixed-effect, multilevel logistic regression models. These were conducted with indicator conformity as the dependant variable and each of four time variables (registration expiry; registration COVID expiry; registration site visit; or self-assessment submission site visit) as the independent variables. In all statistical models, the primary sampling unit was the practice and included the three confounding variables. Multilevel models have been used as the data are viewed as arising from a multilevel sampling design in which repeated measures are taken on each practice, and a sample of practices has been recruited.

We evaluated the association between time variables prior to the site visit with compliance after the site visit. Separate regression models were conducted with the two measures recorded after the site visit (time to remediate non-conformant indicators and number of transactions required to remediate non-conformant indicators) as the dependant variables and the time variables described above as the independent variables. Both independent and dependant variables were standardised into the same time unit (months) to aid interpretation of model coefficients. Regression models were used to calculate practice performance post site visit (time to remediate and number of transactions) of all non-conformant indicators, as this is often viewed at a practice level.

Results

Of the 757 practices with data, 15 were removed from the analyses due to these practices transitioning over from another accreditation agency after their expiry date. Within this sample, 122 practices were new to the accreditation process, and therefore did not have an accreditation expiry date, thus were not included in the relevant statistical models. In addition, there were 88 (12%) practices that were conformant to all indicators at the site visit and therefore had their post site visit variables counted as zero. Table 1 shows available practice

characteristics and a summary of all study variables.

Table 2 shows the association between time variables prior to the site visit with the rate of indicator conformity. A small, statistically significant and positive association was identified regarding the registration–accreditation expiry period, indicating practices with a larger period between registration with the accrediting agency and their current accreditation expiry experienced a higher number of non-conformant indicators.

Table 3 shows the association between the time variables prior to the site visit with measures recorded after the site visit. A small, negative and statistically significant relationship was identified between the registration–accreditation expiry period and the time to remediate non-conformant indicators (Appendix 1; available online only). This indicates practices with a larger period between registration with the accrediting agency and their current accreditation expiry were associated with less time to remediate non-conformant indicators. A small, negative and statistically significant relationship was identified between the self-assessment submission–site visit period with the time to remediate non-conformant indicators (Appendix 2; available online only). This indicates practices who submitted the self-assessment early relative to their site visit exhibited a shorter time to remediate non-conformant indicators. A moderate, negative and statistically significant relationship was identified between the registration–accreditation expiry period and the number of transactions required to remediate non-conformant indicators, indicating practices who register early with the accrediting agency relative to their accreditation expiry require less assistance to remediate non-conformant indicators (Appendix 3; available online only).

Table 1. Practice characteristics and study variables

Sample size (n)	Variables	Metrics
742	Urban location, n / %	476 / 64
742	GP head count, m (SD)	5.92 (4.21)
742	Number of previous GP accreditation cycles, m (SD)	3.14 (1.79)
742	Corporate-owned practices, n / %	296 / 39.9
620	Registration – Expiry period, m (SD)	11.43 (4.14)
620	Registration – COVID-19 extension expiry period, m (SD)	23.42 (4.14)
742	Registration – Survey visit period, m (SD)	17.22 (5.31)
742	Submission of self-assessment – Survey visit period, m (SD)	4.68 (3.38)
742	Indicators non-conformant at survey visit, m (SD)	7.00 (7.27)
742	Days to remediate indicator non-conformity, m (SD)	36.9 (25.13)
742	Total transactions with accreditation agency to remediate indicator non-conformity, m (SD)	21.93 (28.72)

Date range calculated as months unless otherwise specified. Transactions represent communication within the proprietary system after the site visit related specifically to indicator non-conformities. Bracket numbers indicate standard deviation (SD) of the mean (m) practice characteristics and study variables.

Table 2. Association between time variables prior to the site visit with the rate of indicator conformity at the site visit

	Odds ratio (95% CI), z-score, *P* value
Registration – accreditation expiry	**1.04 (1.02–1.06), 3.26, 0.001**
Registration – site visit	1.008 (0.99–1.03), 0.93, 0.35
Self-assessment submission – site visit	1.009 (0.99–1.03), 0.65, 0.52

Time interval unit of measurement is months. Confounders included in the model include general practitioner headcount, number of previous accreditation cycles with accrediting agency and urban or rural location. Bold indicates statistically significant models.

Discussion

This study sought to evaluate the relationship between several metrics reflective of preparation time with accreditation performance at, and after, the mandatory site visit. Considering the widely adopted uptake of accreditation practices in healthcare, there continues to be a lack of empirically driven research in this field.[14] We identified that a greater period between registration with the accrediting agency and the practice accreditation expiry date was associated with higher indicator conformity at the site visit, as well as less time and less assistance required to remediate non-conformant indicators. Further, late submission of self-assessment prior to the site visit was associated with a longer period to remediate non-conformant indicators.

There were consistent results with respect to the period of registration with the accrediting agency and current general practice accreditation expiry date. Registration reflected the date at which general practices assigned an accrediting agency, which is the first point of contact in the (re)accreditation process.[7] General practice staff perceptions and attitudes towards accreditation might impact the positive outcomes of, and engagement to, accreditation.[8] Specifically, attitudes related to the accreditation process have consistently reported time constraints by support staff and practitioners in addition to their day-to-day roles.[8,15,16] Accreditation-specific tasks such as registering for accreditation, completion of the self-assessment, implementation of quality assurances processes and logistical

components related to organising site visits can take a considerable amount of time for general practices to complete. Although these tasks are required by all practices undergoing accreditation, our data highlight significant variation in the time general practices register for (re)accreditation with the accrediting agencies, as well as how long it takes to complete tasks, such as submission of self-assessment. Importantly, this variation in time to prepare for the accreditation site visit partially explains performance at the site visit, the time to remediate non-conformant indicators and the assistance required to remediate non-conformant indicators post site visit. Our results emphasise the importance of a larger time period to prepare for accreditation-specific tasks within the current three-year accreditation cycle for general practices, as well as potentially disincentivise late registration for re-accreditation or submission of the self-assessment.

There are several limitations to the current study. First, the study period overlaps with the COVID-19 pandemic and the associated impacts, and practices were provided with an extension to their accreditation expiry as a result. It is important to replicate these analyses in future periods without a global pandemic significantly impacting general practices. Second, the results presented reflect data from a single accreditation agency, and comparison with other providers is encouraged. Third, as this is a secondary analysis and we are limited in our investigation by the information currently measured, alterative and more specific metrics, such as self-reported questionnaires, might yield additional useful insights. Finally, our analyses assumed equal weighting to all non-conformant indicators, where in practice, non-conformant indicators are risk stratified as low, moderate, high or critical and this might be something that future research should consider.

Limited research exists investigating the relationship between general practice time to prepare for accreditation and performance outcomes. We identified that greater time to prepare within the accreditation process was associated with small-to-moderate improvements in performance at and after the mandatory site visit. These results provide an empirical examination of several components within the current Australian general practice accreditation framework and might guide the future implementation of the accreditation program.

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Table 3. Association between time variables prior to the site visit and post-site visit measures

	β (Standard error), z-score, *P* value	
	Time to remediate non-conformities	**Number of transactions**
Registration – accreditation expiry	**–0.02 (0.008), –3.07, 0.002**	**–0.66 (0.28), –2.40, 0.02**
Registration – site visit	–0.006 (0.006), –1.06, 0.29	–0.19 (0.21), –0.90, 0.37
Self-assessment submission – site visit	**–0.02 (0.009), –2.73, 0.006**	–0.40 (0.31), –1.29, 0.20

All time variables are standardised to months. Bold indicates statistically significant models. Confounders include general practitioner headcount, number of previous accreditation cycles with accrediting agency and urban or rural location.

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Direct-to-consumer telemedicine in primary care settings: A scoping review of contemporary empirical literature

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Background and objective
This study comprehensively reviews the contemporary empirical literature on direct-to-consumer (DTC) telemedicine services within primary care.

Methods
MEDLINE, Embase and SCOPUS were strategically searched and screened. Data on the modality of consultations, population of focus, condition of focus and treatment of focus were extracted, narratively synthesised and tabulated.

Results
Forty-four articles were included in this review. Most used quantitative methods, with predominantly cross-sectional or retrospective cohort designs. DTC telemedicine user characteristics and perspectives were most researched, followed by quality and safety. Most services used video or text messaging. Articles typically examined a specific health condition (eg acute respiratory infections) and its treatment, and several focused on a specific population (eg men).

Discussion
In light of the poor evidence base and lack of rigorous studies, there is a critical need for more robust research on DTC telemedicine within primary care. Quality assessment tool development and health economics analyses are necessary to support the integratation of DTC telemedicine services with traditional primary care systems and improve primary healthcare quality and efficiency.

THE COVID-19 PANDEMIC accelerated the adoption of telehealth in primary care, significantly increasing services and market growth. Projections suggest that the global telehealth market might reach US$500 billion by 2030.[1] This surge is particularly evident in commercial telemedicine services – defined here as direct-to-consumer (DTC) telemedicine. These services are characterised by predominantly virtual consultations through phone, video or messaging with medical practitioners that are marketed to, initiated by and mostly paid for by the consumer. There is usually no established doctor–patient relationship associated with these services, and they often feature strong marketing campaigns and cover a range of medical issues, from general consultations to specific treatments like weight loss or erectile dysfunction. Despite their rapid proliferation and significant market valuations, exemplified by US companies like Hims & Hers Health and Ro with valuations in the billions of dollars,[2,3] research in this area has not kept pace. A similar pattern is observed outside the USA,[4] with companies like Eucalyptus[5] and InstantScripts[6] in Australia and the rise and fall of Babylon Health in the UK.[7]

Existing research, mostly pre-pandemic, has looked at antibiotic use in acute respiratory infections (ARIs),[8,9] contraception,[10] hair loss[11] and erectile dysfunction.[12] Only a handful of studies have examined issues of accessibility,[13] healthcare utilisation,[14] clinician efficiency, and patient empowerment, satisfaction and preferences.[15,16] There has not been a comprehensive review of the contemporary empirical literature on DTC telemedicine services within primary care. Furthermore, recent systematic reviews on the quality of telehealth consultations have either excluded this service group[17] or focused on secondary or tertiary care settings.[18]

This review addresses this gap by identifying and assessing the nature and extent of the body of contemporary empirical research involving DTC telemedicine within primary care. We aimed to uncover knowledge deficits and propose areas for future research.

Methods

Protocol and registration

The reporting of this scoping review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews

(PRISMA-ScR) checklist.[19] The study protocol was registered with the Open Science Framework (osf.io/aw74p).

Search strategy

Three academic databases (MEDLINE, Embase and SCOPUS) were searched on 1 July 2023, for studies published from 1 January 2019 onwards. This five-year timeframe adequately captures the contemporary research in this field and the transition from pre- and post-COVID-19 periods. The search strategy utilised a combination of Medical Subject Headings (MeSH) terms and keywords to cover: (1) telehealth or telemedicine; (2) primary care; and (3) the concept of DTC health services. The search strategy was devised in consultation with a university clinical librarian and in conjunction with published search strategies for telehealth.[20] Table 1 shows a search strategy for MEDLINE.

Study selection criteria

Inclusion criteria

The inclusion criteria for this review were studies that:

- reported on DTC telemedicine services, being defined as a service providing predominantly virtual consultations with a medical practitioner for which the service was initiated by the consumer, and the main fee structure did not involve public funding or rebates
- reported research in the context of primary care settings
- reported on primary or secondary data
- were published from 2019 onwards
- were full text and published in English.

Exclusion criteria

The exclusion criteria for this review were studies that:

- reported on DTC telemedicine services provided by health providers other than medical practitioners (eg nurse practitioners, psychologists or physiotherapists)
- reported on DTC telemedicine services exclusively in secondary or)tertiary care settings
- reported on telehealth services provided by a traditional primary care health service, general practice clinic or hospital-based primary care service
- were non-empirical journal articles (eg reviews, protocols, editorials, conference proceedings, commentaries and letters)
- had no full text available.

Screening and data extraction

Reference details were downloaded into Endnote 20 and duplicates were excluded. References were then exported to Microsoft Excel and divided among the research team for title, abstract and full-text screening processes. To ensure consistency in screening, three reviewers (DF, SS and GD) independently reviewed 10% of the titles and abstracts and 10% of full-text articles during subsequent full-text screening.

Interrater reliability between the three reviewers was assessed to be sufficiently high during each stage with Cohen $\kappa \geq 0.80$. Iterative meetings were held to discuss any discrepancies in screening decisions that arose.

Data extraction was conducted using a customised form (Appendix 1; available online only). The form was piloted by each of the three reviewers with a subset of papers (n=5). Issues in consistency of data entry and usability of the template were discussed and modifications made accordingly. Key information extracted included article characteristics (authors, year and country), study design and method as defined by the Mixed Methods Appraisal Tool (version 18),[21] modality of consultations, stakeholder group of focus, and specific population, condition and treatment of focus.

Data synthesis

The overall characteristics for each data extraction field were summarised as categorical variables. A narrative synthesis of the included articles was performed to identify primary focuses or themes of the research. The five themes identified were: (1) user characteristics and perspectives; (2) quality and safety; (3) healthcare utilisation; (4) health economics; and (5) validation of tools. There were some overlaps between articles that had more than one research domain of focus. The country of the corresponding author was coded by income classification based on the World Bank's definitions of gross national income per capita per year.[22]

Table 1. Example search strategy for MEDLINE

Set	Search statement
1	exp Telemedicine/ or exp Remote Consultation/ or (cyberthera* or telecare or telecollaborat* or teleconsult* or teleconference* or teleeducat* or telediagnos* or telehealth or teleguide* or telediagnos* or telelearn* or telemed* or telementor* or telemonitor* or teleneurol* or teleopth* or telepediatric* or telepresence* or telerehab* or telerobotic* or telescreen* or teletherap* or teletransmi* or mhealth or "m heath" or ehealth* or "e health" or virtual or ((cyber or digital or remote* or distance* or tele) adj2 (care or collaborat* or consult* or conference* or educat* or diagnos* or health or guide* or diagnos* or learn* or med* or mentor* or monitor* or presence* or screen* or therap* or transmi*)) or ((cyber or digital or distance* or tele or remote* or sms or phone* or internet or "web based" or telephone* or texting or "mobile app*" or Instagram or Snapchat or Facetime or GMeet* or hangout* or Skype or Zoom or Web-ex or WebEx or Bluejeans or Facebook or e-mail* or email* or "e chat" or echat or "social media" or "text message*" or "answering machine*" or "voice mail*" or "video conferenc*" or "video link*" or "video chat*") adj3 (consult* or support* or diagnos* or "follow-up*" or "health" or doctor* or "primary care" or clinic or clinics or clinician* or nurs* or psycholog* or therap* or intervention* or delivery))).mp.
2	"direct to consumer*".mp.
3	DTC.ti,ab.
4	or/2-3
5	and/1,4
6	limit 5 to English language
7	limit 6 to year="2019 -Current"

Results

Search results

Our initial searches retrieved a total of 689 records. After title, abstract and full-text screening, 44 (6.3%) papers met the inclusion criteria. Figure 1 demonstrates the inclusion and exclusion decision for papers at each stage of the screening process.

Summary characteristics of included publications

A summary of the key characteristics of the 44 included articles is presented in Table 2. Almost all studies were conducted in high-income countries (n=41, 93%), with most from the USA (n=29/44, 66%). The remainder of the articles were from Australia (n=4/44, 9%), Germany (n=3/44, 7%), Sweden (n=2/44, 4.5%), China (n=2/44, 4.5%), Canada (n=2/44, 4.5%), Spain (n=1/44, 2.3%) and Brazil (n=1/44, 2.3%). There were no publications from low-income countries. Figure 2 illustrates the geographical distribution of the included articles.

Most articles used quantitative methods (n=41/44, 93%), with the small remainder comprising two mixed methods studies (5%) and one qualitative study (2%). A large proportion of included articles were cross-sectional (n=26/44, 59%) and retrospective cohort studies (n=12/44, 27%), and the remaining were quasi-experimental studies (n=6, 14%). Cross-sectional studies were further classified into analytical (n=4/26, 15%) or descriptive studies (n=22/26, 85%). Overall, the cross-sectional studies focused on user characteristics and perspectives of a particular DTC telemedicine service,[12,23–47] whereas the retrospective cohort and quasi-experimental studies focused on prescribing patterns, access to care and healthcare utilisation.[8,11,48–63]

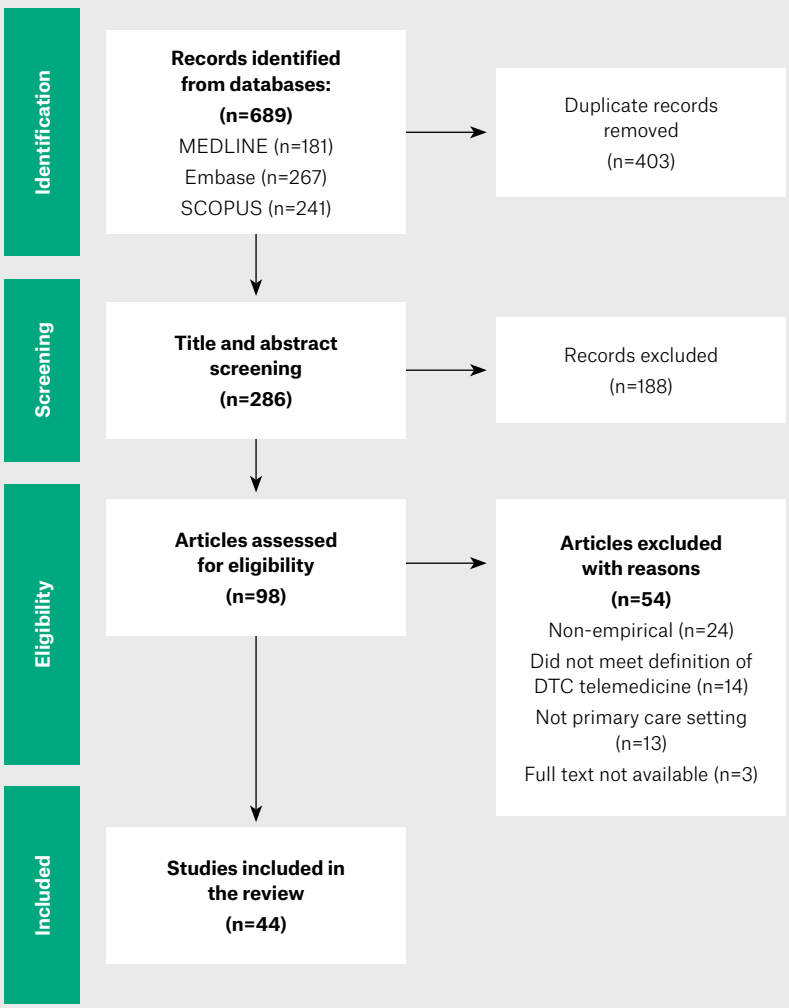

Figure 1. Inclusion and exclusion of papers at each stage of the screening process.

Research domain of focus

Most articles (n=32/44, 72%) focused on user characteristics and perspectives. These articles tended to describe the patient, clinician or encounter characteristics for a particular DTC telemedicine service and might have included analysis of data regarding patient preferences, attitudes or perceptions. Some articles had more than one research domain of focus, resulting in some overlap. Twenty-one articles (47%) discussed some form of care quality and patient safety. Within this group, most of these articles analysed prescribing patterns (n=12/21, 57%),[8,11,12,24,25,27,28,45,53,58,59,63] and the remainder assessed consult quality or patient satisfaction (n=9/21, 43%).[26,29,30,39,42,48,49,57,61] Three articles (7%)[37,52,56] considered healthcare utilisation and one article (2%)[60] was a health economics approach describing a cost analysis comparison of DTC telemedicine versus traditional prescriptions of phosphodiesterase 5 (PDE5) inhibitors for erectile dysfunction. One article (2%)[35] validated a checklist tool that was developed to assess the quality of skin lesion images submitted by consumers.

Modality of consultations

A large proportion of articles described services delivered using video (n=23/44, 52%), followed by messaging-based platforms (n=17/44, 39%) and telephone (n=10/44, 23%). There were some overlaps as some articles described services where multiple modalities were used. Any form of direct communication between the patient and the medical practitioner via a text-based message was categorised as using a messaging-based platform. This included messages via web-based applications, mobile phone applications or secure messaging platforms.

Table 2. Summary of key characteristics of included publications

Classification	Papers (N=44), n (%)[A]
Country of corresponding author	
USA	29 (65.9)
Australia	4 (9.1)
Germany	3 (6.8)
Sweden	2 (4.5)
China	2 (4.5)
Canada	2 (4.5)
Spain	1 (2.3)
Brazil	1 (2.3)
Country income classification of corresponding author	
High income	41 (93.2)
Middle income	3 (6.8)
Low income	0 (0.0)
Study methods	
Quantitative methods	41 (93.2)
Mixed methods	2 (4.5)
Qualitative methods	1 (2.3)
Study design	
Descriptive cross-sectional study	22 (50.0)
Retrospective cohort study	12 (27.3)
Quasi-experimental study	6 (13.6)
Analytical cross-sectional study	4 (9.1)
Modality: Synchronicity	
Synchronous	20 (45.4)
Asynchronous	12 (27.3)
Both	5 (11.4)
Not specified	7 (15.9)
Modality: Technology	
Video only	15 (34.1)
Messaging based only	12 (27.3)
Video and telephone and messaging based	5 (11.4)
Video and telephone	3 (6.8)
Telephone only	2 (4.5)
Not specified	7 (15.9)
Specific population of focus	
All adults	17 (38.6)
All patient groups	11 (25)
Adult men	6 (13.6)
Children or adolescents	5 (11.4)
Specific population of focus (cont'd)	
Adult women	2 (4.5)
Clinicians	1 (2.3)
Veterans	1 (2.3)
Not specified	1 (2.3)
Specific condition of focus	
ARIs	11 (24.4)
No specific focus	9 (20.0)
Dermatological conditions	8 (17.8)
Erectile dysfunction	3 (6.7)
Contraception	2 (4.4)
General paediatrics	2 (4.4)
Hair loss	2 (4.4)
UTIs	2 (4.4)
ADHD	1 (2.2)
Low acuity infectious diseases	1 (2.2)
Men's health	1 (2.2)
Mental health	1 (2.2)
Obesity	1 (2.2)
Urgent care conditions	1 (2.2)
Specific treatment of focus	
No specific focus	24 (54.5)
Antibiotics	9 (20.5)
PDE5 inhibitors	3 (6.8)
Finasteride or minoxidil	2 (4.5)
Oral contraceptives	2 (4.5)
Corticosteroids	1 (2.3)
Non-stimulant ADHD therapies	1 (2.3)
Oseltamivir	1 (2.3)
OSH (Plenity)	1 (2.3)
Research domain of focus	
User characteristics and/or perspectives	32 (55.0)
Quality and safety	21 (36.0)
Healthcare utilisation	3 (5.2)
Health economics	1 (1.7)
Validation of tools	1 (1.7)

[A]Columns might not equal 44 due to overlap in some categories.

ADHD, attention deficit hyperactivity disorder; ARI, acute respiratory infection; n, number of articles included in descriptive analysis; OSH, oral superabsorbent hydrogel; PDE5, phosphodiesterase 5; UTI, urinary tract infection.

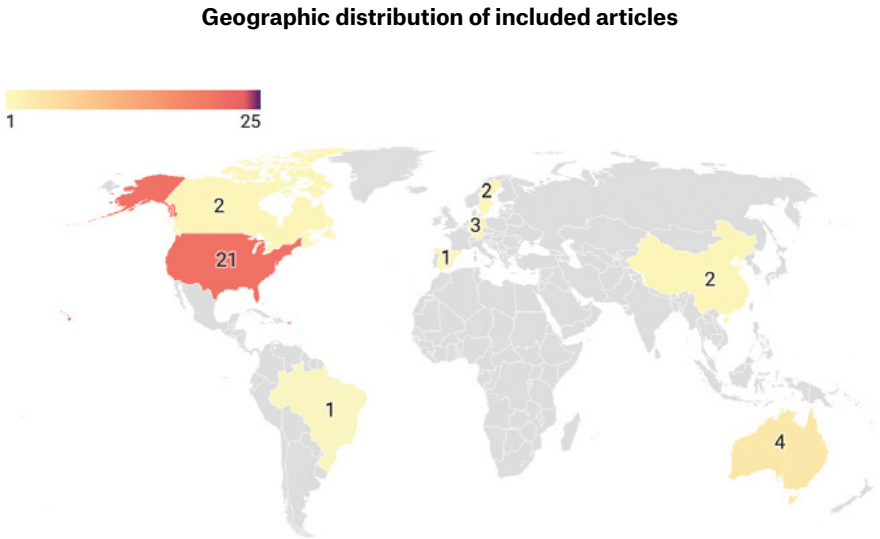

Figure 2. Geographic distribution of included articles. Numbers represent the number of articles.

Specific populations of focus

One-third of articles involved specific populations (n=15/44, 34%), such as men (n=6/44, 14%)[11,12,23,31,47,60] or women (n=2/44, 5%),[8,34,40,43,44,54,62] children and/or adolescents (n=5/44, 11%)[8,43,44,54,62] or veterans (n=1/44, 2%).[61] A single study (2%) examined clinicians rather than a patient group, investigating the relationship between consultation length and quality using standardised patients.[26] The remainder were articles that generalised to an adult population (n=17/44, 39%) or did not specify who the population was (n=1/44, 2%).

Specific conditions and treatments of focus

There were 13 specific conditions or condition groups of focus identified across 35 articles (80%), whereas the remaining nine (20%) had no specific disease or condition focus. There was a small amount of overlap, with one article investigating both ARIs and urinary tract infections (UTIs).[25] The predominant condition group of focus was ARIs (n=11/44, 25%),[8,24,25,27,28,45,53,54,56,62,63] followed by dermatological conditions (n=8/44, 18%),[26,33,35,36,46,48,49,57] general paediatrics (n=2/44, 5%),[43,44] low acuity infectious diseases in addition to ARIs (n=1/44, 2%),[58] men's health (n=1/44, 2%),[31] mental health (n=1/44, 2%)[30] and urgent care conditions (n=1/44, 2%).[41] The predominant condition was erectile dysfunction (n=3/44, 7%),[12,47,60] followed by contraception (n=2/44, 4%),[34,40] hair loss (n=2/44, 4%),[11,23] UTIs (n=2/44, 4%),[25,59] attention deficit hyperactivity disorder (ADHD) (n=1/44, 2%)[29] and obesity (n=1/44, 2%).[55]

Articles dealing with a specific condition or condition group generally had a corresponding treatment of focus. For example, articles describing services for the management of ARIs often had a focus on antibiotic prescribing (n=9/44, 21%).[8,25,27,28,53,54,58,59,63] Those that examined conditions such as erectile dysfunction, ADHD or obesity also focused on treatments for each corresponding condition, such as PDE5 inhibitors (n=3/44, 7%),[12,47,60] non-stimulant ADHD therapies (n=1/44, 2%)[29] or an oral superabsorbent hydrogel (n=1/44, 2%).[55]

Discussion

Summary of main findings

Our findings reveal a mismatch between the burgeoning demand and market growth of DTC telemedicine services and the pace of research conducted. The reviewed articles were largely cross-sectional analyses of user characteristics for various services. General user characteristics of DTC telemedicine services were considerably mixed and were largely dependent on the type of service being offered and if it was restricted to specific conditions or populations. Most articles examined acute or subacute conditions that were treated in isolation with a clear gap in the literature on the use of DTC telemedicine for chronic conditions or multimorbidity. Publications focusing on aspects of quality and safety mainly looked at prescribing patterns and particularly antibiotic prescribing. Even articles that specifically reported on the overall quality of a particular service[12,27,42] mainly analysed prescribing patterns and rates of side effects without assessing other aspects of quality, such as efficiency, effectiveness, safety, patient-centredness, timeliness and equity, as defined by the Institute of Medicine's theoretical framework.[64] There was also heavy reliance on quantitative research methods using retrospective study designs, leaving a substantial gap in qualitative insights and prospective research. Notably, there was also a lack of contemporary studies exploring clinicians' perspectives on DTC telemedicine.

Equity and access

There is a notable lack of understanding of equity in and access to DTC telemedicine services. Most studies were conducted in high-income countries, raising questions about the generalisability of these findings to global populations. This skew towards high-income nations might reflect a tendency of neoliberalist economies to rapidly adopt and integrate new technologies, particularly those with commercial potential.[65] Alternatively, it could be indicative of a broader reflection of global wealth disparities, where only regions with substantial financial resources can afford to invest in and research these emerging healthcare modalities. Both factors could be relevant. Regardless of which, this disparity highlights a significant concern about the equitable distribution of telemedicine services and the potential exclusion of low- and middle-income countries from both research and the benefits of such innovations.[66]

Health economics and healthcare utilisation

From a health economics perspective, the literature is notably lacking in cost–benefit analyses at the health system level. Although one study addressed the consumer cost of obtaining prescriptions for PDE5 inhibitors,[60] there is a paucity of work seeking to understand the overall economic effects of DTC telemedicine services on healthcare systems. Filling this gap is particularly critical for countries with predominantly publicly funded health systems, where it is essential to ascertain whether commercial telemedicine entities contribute to cost savings or result in increased healthcare expenditures for governments.[67,68]

Only three articles analysed the effects of DTC telemedicine on healthcare utilisation. All three studies explored downstream utilisation, whereas only one study looked at upstream utilisation. Lapointe-Shaw et al found that compared to patients who had consults with their regular physician, DTC telemedicine patients had lower previous healthcare use, were less likely to have a subsequent in-person physician visit and were more likely to visit the emergency department within 30 days.[37] Li et al and Dahlgren et al also found similar associations that DTC telemedicine use was predominantly associated with downstream care encounters.[52,56] Given the modest number of studies in this area, there is an ongoing need for data-linkage studies and thorough health economics analyses to understand the effects of these services on healthcare systems and guide policymaking for the optimal integration of DTC telemedicine into primary care.

Qualitative insights

The existing literature is heavily reliant on quantitative research methods, leading to a shortfall in qualitative insights. Only three studies used some form of qualitative method, one of which was a usability assessment of a services website interface;[50] the other two evaluated healthcare practitioners' views and consumer preferences on using mobile teledermoscopy for skin cancer diagnosis.[33,36] This lack of qualitative research leaves unanswered questions about the reasons patients choose to use DTC telemedicine services and their perceptions of these services. Qualitative studies looking at other conditions and more general services are essential to further explore the patient experience, understand the motivations behind the use of these services and assess patient satisfaction.

Clinicians' perspectives

Another notable omission in the current body of research is the lack of studies exploring clinicians' perspectives on DTC telemedicine. Only one study focused on healthcare practitioners' views.[33] Understanding the views and experiences of healthcare providers can shed light on the challenges and opportunities presented by this mode of service delivery, including aspects of care quality, the efficacy of communication methods and the effects on their professional practice.[69] Integrating the clinicians' viewpoint is essential to ensure that DTC telemedicine services are not only patient-centric but also sustainable and effective from a provider's standpoint.

Strengths and limitations

To the best of our knowledge, this review is the first to assess the contemporary empirical literature on DTC telemedicine within primary care. The findings highlight the type, breadth and range of DTC telemedicine services, as well as main health conditions serviced and treatments offered. This review has enabled the identification of critical gaps in the research on DTC telemedicine in primary care that can be leveraged in future research efforts.

This review has several limitations worth noting. Studies included in the review were not assessed for risk of bias, and the quality of evidence for each study was not appraised. The predominance of studies from high-income countries will limit generalisability to low-income regions with differing health systems. Eligible studies were restricted to those published within the past five years, potentially omitting relevant papers published in earlier years. However, this time frame was purposively selected as appropriate for capturing the evolving healthcare landscape since the onset of the COVID-19 pandemic. A grey literature search was not included, which might offer other insights and could be considered in the future.

Conclusion

Our review highlights a critical need for multifaceted research on DTC telemedicine within primary care. Future research should focus on understanding the reasons behind patients' preference for DTC services compared with traditional primary care, assessing the effects of these services on the primary care workforce, developing quality assessment tools and examining the health economic implications of DTC telemedicine services. Additionally, exploring these services from a commercial determinants of health perspective is essential, particularly considering growing investments from major corporations. Comprehensive research is vital to inform policy and regulatory decisions, ensuring that commercial services are integrated effectively with traditional primary care systems, ultimately enhancing the overall quality and efficiency of primary healthcare.

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Skin health of urban-living Aboriginal children attending a primary care Aboriginal Community Controlled Health Organisation clinic

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Background and objective
Despite increasing urbanisation, little is known about skin health for urban-living Aboriginal children and young people (CYP, aged <18 years). This study aimed to investigate the primary care burden and clinical characteristics of skin conditions in this cohort.

Methods
A one-year retrospective cohort study of urban-living Aboriginal CYP presenting for general practitioner (GP) consultation at an Aboriginal Community Controlled Health Organisation (ACCHO) was conducted.

Results
At least one dermatological diagnosis was made in 27% (253/939) of GP face-to-face consultations for the 585 urban-living Aboriginal CYP included. Infections and dermatitis accounted for 54% (152/284) and 18% (50/284) of all dermatological diagnoses, respectively. Bacterial skin infection (BSI) cumulative incidence was 13% (74/585; 95% CI 10–16%), with recurrent BSI affecting <1% (5/585; 95% CI 0.3–2%) and hospitalisation required in 1% (1/82; 95% CI 0.06–7%) of incident BSI cases.

Discussion
We present a culturally secure, multidisciplinary skin health assessment model within an urban ACCHO, where dermatological conditions account for a significant proportion of GP workload.

HEALTHY SKIN is essential for overall health. Understanding the skin conditions that impact different populations provides a framework to develop appropriate resources and services.[1] For Aboriginal families, this includes prioritising a holistic approach, integrating traditional health practices and acknowledging the connection of health to ancestry, country, culture, community and family.

To improve health outcomes for Australian Aboriginal people, and overcome structural racism embedded within mainstream healthcare, Aboriginal Community Controlled Health Organisations (ACCHO) were established during Australia's Black Power movement.[2] Through >140 member ACCHOs nation wide, this sector now delivers almost 3.1 million episodes of care per year and in 2020, collectively serviced >410,000 people.[3] The lifetime health impact of ACCHO-delivered interventions is 50% greater than that of mainstream healthcare services, emphasising the strength of this sector in achieving meaningful outcomes.[3]

Although a heavy burden of skin infections affecting remote-living Aboriginal children has been documented, little is known about skin health of urban-living Aboriginal children.[4] In Western Australia (WA), this is despite nearly 60% of all Aboriginal children and young people (CYP, aged <18 years) residing in urban settings (major cities and inner regional areas).[5] Hospitalisation rates for skin infections are 10-fold higher in urban-living Aboriginal CYP compared with non-Aboriginal CYP; this is associated with an increased risk of serious complications including invasive infection, acute post-streptococcal glomerulonephritis (APSGN), acute rheumatic fever (ARF) and rheumatic heart disease (RHD).[4,6] Yet, hospitalisation is likely an underestimate of skin infection burden, as most are managed in primary care.

Globally, atopic dermatitis (AD) or eczema, is the most common chronic inflammatory skin condition in CYP, with rising prevalence.[7] AD is a risk factor for skin infections and adversely impacts general health, school performance and quality of life.[4,8] AD and skin infections are more prevalent and severe among urban-living Indigenous CYP in high-income countries globally, as compared with non-Indigenous CYP.[9] AD is the leading cause of skin disease burden (59%) in Aboriginal children aged <5 years and the second most common (33%) for children aged 5–24 years.[10]

Across all ages, skin disorders represent 16% of primary care consultations for urban-living Aboriginal patients, and 22% for remote-living Aboriginal patients.[11,12] We aimed to investigate the burden and clinical characteristics of skin disorders affecting urban-living Aboriginal CYP in primary care.

Methods

Study design and setting

A retrospective cohort study was conducted at Derbarl Yerrigan Health Service (Derbarl), an urban ACCHO located on *Whadjuk Noongar* (Perth, WA) *boodjar* (land/place). The *Noongar Nation* are the traditional custodians of south-west WA and includes 14 clans/language dialects: *Whadjuk* refers to the Perth clan. The Noongar calendar includes six seasons indicated by flowering of local plants and insect/animal behaviours, following a Mediterranean climate (Figure 1).

Derbarl was established in 1973 and is the largest ACCHO in WA. It provides integrated primary health services across four sites to >15,000 Aboriginal patients annually, 30% of whom are CYP (Derbarl Yerrigan Health Service Business Information Unit, unpubl data). Derbarl utilises an Aboriginal health practitioner (AHP)-led model of care with patients receiving an age-appropriate assessment prior to a general practitioner (GP) consultation, excluding patients requiring urgent care or those presenting with an acute respiratory illness during the COVID-19 pandemic. For children aged <15 years, the AHP assessment includes skin and head lice examinations.

Eligible study participants were identified through an electronic medical record (EMR) search as those aged <18 years who attended a GP appointment at the central (East Perth) clinic over a 12-month period (01 October 2020 – 30 September 2021). Patients were excluded if they did not have a documented GP clinical interaction or if they attended for influenza/COVID-19 vaccination only (Figure 2). The 12-month audit period coincided with the COVID-19 pandemic, including three short COVID-19 lockdowns (February, April and July 2021). The Derbarl COVID-19 response included holistic COVID-19 care services for patients in isolation, with increased GP telehealth consultations.[13] A monthly paediatric dermatology clinic commenced at the central Derbarl site in May 2021.

Data extraction, study measures and statistical analysis

All clinic presentations for eligible participants were retrospectively reviewed and data were collected by the lead author and dermatologist (BMR). Baseline demographic data were collected at first presentation: birth date, sex, Indigenous status, residential address and past history of AD, APSGN, ARF and RHD. The 2019 Modified Monash (MM) category was used

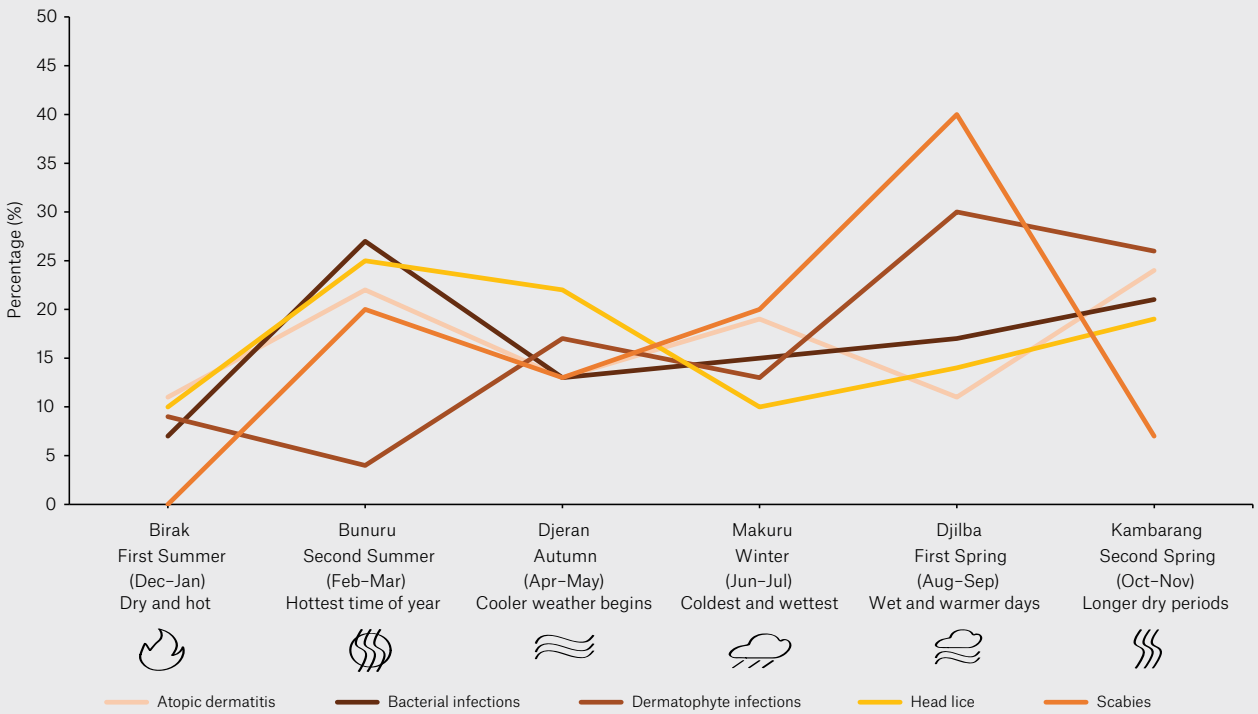

Figure 1. Noongar seasonal variation of dermatological disorders addressed in primary care.

Adapted from Barrow J, Nannup N (Uncle). Nyoongar seasonal calendar. Commonwealth of Australia, Bureau of Meteorology, 2016, www.bom.gov.au/iwk/calendars/nyoongar.shtml, with research and content by Barrow J and Uncle Noel Nannup, and with permission from Uncle Noel Nannup.

to classify geographical remoteness, with urban-living defined as MM1 (metropolitan areas) or MM2 (regional centres).[14]

GP consultations were recorded as separate episodes of care and data extracted regarding service delivery (date, mode of consultation, AHP skin and head lice assessments) and clinical presentation, including details of all dermatological disorder(s) addressed (primary or secondary diagnoses). Dermatological disorders were classified into broad diagnostic categories (Appendix 1; available online only). For skin infections (bacterial, dermatophyte and viral), scabies and AD, data on clinical features, investigations and management were extracted. *New bacterial skin infection (BSI)* was defined as a clinical presentation for BSI occurring >30 days from index diagnosis (to exclude the possibility of counting scheduled reviews, relapses or treatment failure). *Recurrent BSI* was defined as more than one new BSI episode per 12 months.

Primary outcome measures related to skin disease burden among urban-living Aboriginal CYP were: (1) *proportion* (proportion of GP consults addressing a dermatological disorder, AHP skin assessments documenting 'abnormal skin', AHP head lice assessments documenting head lice); (2) *incidence* (cumulative incidence and incidence rate for new BSI); and (3) *prevalence* (contact prevalence for head lice, dermatophyte infections, scabies, viral skin infections and AD; lifetime prevalence for AD, APSGN, ARF and RHD). Secondary outcome measures included clinical features, seasonal variation, investigations and treatment of common skin disorders.

All data were entered into a REDCap database and analysed using R version 4.1.2 (R Foundation for Statistical Computing, Vienna, Austria). Descriptive statistics summarised patient demographics, episodes of care, dermatological disorders and clinical features. Proportion, incidence and prevalence calculations were performed (Table 1). GP consult data were used for analysis of all disorders except head lice, where AHP assessment data were used. The frequencies of categorical variables (age, sex, MM category) were compared between outcome categories (infections, infestations, AD) using Fisher's exact test, and reported with odds ratios and 95% confidence intervals.

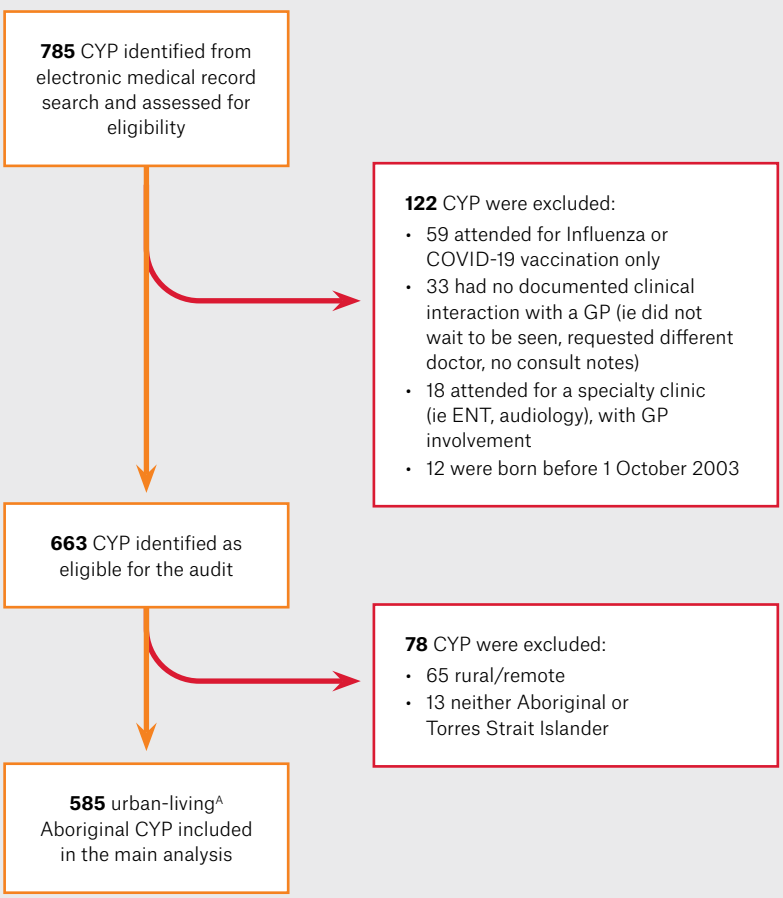

Figure 2. Participant selection.

[A]Urban-living defined as Modified Monash 1 (metropolitan areas) or Modified Monash 2 (regional centres).

CYP, children and young people (aged 0–18 years); ENT, ear, nose and throat; GP, general practitioner.

Ethics approval

Ethics approval was granted by the Western Australian Aboriginal Health Ethics Committee (HREC Ref No. 1059) and the University of Western Australia (File Reference – 2021/ET000536).

Results

Study population

A total of 785 patient EMRs were screened and 585 urban-living Aboriginal CYP were included in the analysis (Figure 2). The median age was seven years (IQR 3–12), 50% were female and 99% resided in MM1. There were 989 episodes of care recorded for 585 CYP, equating to 1.69 presentations (range: 1–10) per CYP over 12 months (Table 2).

Burden of skin disorders

Proportion results

At least one dermatological disorder was addressed in 26% (259/989) of GP consultations, and at least one skin infection in 14% (139/989). Face-to-face consultations accounted for 95% (939/989) of episodes of care and were associated with a three-fold (OR 2.7, 95% CI 1.13–7.86) likelihood of addressing a dermatological disorder (253/939, 27%) compared with telephone

Table 1. Definitions of numerators and denominators used in analysis

	Numerator	Denominator
Proportion		
Proportion of GP consultations or AHP assessments	Number of episodes of care for specified disorder	Total number of episodes of care
Incidence		
Cumulative incidence	Number of patients with a new episode of BSI over 12 months	Population at risk (total number of patients)
Incidence rate	Sum of all new episodes of BSI over 12 months	Child-years at risk[A]
Prevalence		
Contact prevalence	Number of patients with ≥1 contact for a specified skin disorder over 12 months	Population at risk (total number of patients)
Lifetime prevalence	Number of patients with an ever-recorded specified disorder in the EMR	Population at risk (total number of patients)

[A]The at-risk period is the period that a patient was not recorded having a BSI. We utilised a 30-day washout period for a new BSI and patients were able to contribute multiple events during the study period (recurrent BSI).

AHP, Aboriginal health practitioner; BSI, bacterial skin infection; EMR, electronic medical record; GP, general practitioner.

consultations (6/50, 12%). In total, 284 dermatological disorders were documented in 989 episodes of care, with BSI, dermatitis and dermatophyte infections accounting for 30% (84/284), 18% (50/284) and 8% (23/284), respectively (Figure 3).

AHP skin assessments occurred before 72% (673/939) of face-to-face GP consultations, with 'abnormal skin' documented in 21% (196/939). 'Rashes', 'infected sores', 'dry areas', 'eczema' and 'healing sores' accounted for 27% (53/196), 25% (49/196), 15% (30/196), 14% (27/196) and 12% (24/196), respectively. AHP head lice assessments occurred before 65% (606/939) of face-to-face GP consultations, with head lice detected and treated in 9% (88/939).

Incidence results

There were 82 incident cases of BSI affecting 74/585 urban-living Aboriginal CYP over 12 months (13% cumulative incidence; 95% CI 10–16%), which was highest in children aged 1–12 years (OR 2.44; 95% CI 1.28–4.98) affecting 15% in this age group (61/397). The incidence rate of all BSI in the 12-month follow-up was 142/1000 child-years-at-risk. Recurrent BSI affected <1% (5/585; 95% CI 0.3–2%).

Prevalence results

The 12-month contact prevalence of head lice, AD, dermatophyte infections, viral skin infections and scabies were 18% (74/419; 95% CI 14–22%), 6% (33/585; 95% CI 4–8%), 4% (22/585; 95% CI 2–6%), 3% (15/585; 95% CI 1–4%) and 2% (13/585; 95% CI 0.06–1%), respectively. Head lice was most prevalent in children aged 5–12 years (OR 2.49; 95% CI 1.44–4.35) affecting 25% in this age group (46/183), AD in children aged 0–4 years (OR 2.28; 95% CI 1.06–4.98) affecting 9% (18/208), and dermatophyte infection in children aged 1–4 years (OR 3.21; 95% CI 1.24–8.48) affecting 7% (12/165).

The lifetime prevalence of AD was 16% (94/585; 95% CI 13–19%), whereas ARF, RHD and APSGN were 0.9% (5/585; 95% CI 0.03–2%), 0.2% (1/585; 95% CI 0.008–1%) and 0.2% (1/585; 95% CI 0.008–1%), respectively.

Clinical characteristics of skin disorders

Seasonal variation

Of face-to-face consultations, BSI, AD, dermatophyte infections, viral skin infections and scabies were addressed in 9% (84/939), 4% (37/939), 2% (23/939), 2% (16/939) and 2% (15/939), respectively. The Noongar seasonal variation of skin disorders is shown in Figure 1; with BSI (25%) and head lice (27%) peaking in Bunuru (February/March), AD (24%) in Kambarang (October/November) and dermatophyte infections (30%) and scabies (40%) in Djilba (August/September).

Clinical features

Impetigo was the most frequent BSI subtype occurring in 63% (52/82) of affected children, followed by carbuncles/furuncles in 15% (12/82). Lower limbs (32%), upper limbs (23%) and face (20%) were most commonly affected. No precipitating event was documented in 60% (49/82), whereas skin injury/trauma (17%), AD (5%), scabies (5%) and head lice (4%) most frequently precipitated BSI.

Of 'all dermatitis' cases, AD accounted for 74% (37/50) and irritant contact dermatitis 26% (13/50). Exposed skin was most commonly affected (43%) in dermatophyte infections, followed by covered skin (35%) and the scalp (13%). Molluscum contagiosum accounted for most viral skin infection presentations (38%), followed by hand-foot-and-mouth disease (25%) and non-specific viral exanthems (19%).

Investigations

Wound swabs were collected in 11% (9/82) of

Table 2. Episodes of care by age group

		Age group (years)[A]			
	Total[A]	0 to <1	1 to <5	5 to <13	13 to <19
n (%)[B]	585	43 (7)	165 (28)	232 (40)	145 (25)
Episodes of care per participant					
1–3	540 (92)	42 (98)	151 (92)	211 (91)	136 (94)
4–6	36 (6)	0 (0)	12 (7)	16 (7)	8 (5)
7–10	9 (2)	1 (2)	2 (1)	5 (2)	1 (1)
Total episodes of care[B]	989	74 (7)	292 (30)	385 (39)	238 (24)

[A]n (%), percentages are column percentages.

[B]Row-wise percentages provided for this row.

incident BSI cases, with *Staphylococcus aureus* cultured in 100% (9/9) and *Streptococcus pyogenes* in 22% (2/9). Nail clippings were collected from one of two patients with onychomycosis. Hair plucks were not collected in the three patients with tinea capitis.

Treatment

Systemic antibiotics were commenced in 83% (68/82) of incident BSI cases; most frequently Flucloxacillin (34%, 23/68), Cephalexin (32%, 22/68) and Trimethoprim/Sulfamethoxazole (18%, 12/68). Topical mupirocin ointment and antiseptic washes were both prescribed in 11% (9/82). One patient (1.2%, 1/82; 95% CI 0.06–7%) was referred to the tertiary children's hospital for inpatient management of severe BSI.

Regarding AD consultations, 59% (22/37) were prescribed topical corticosteroids (TCS), 41% (15/37) emollient, 24% (9/37) soap-free wash and 5% (2/37) wet wraps. Referral to the ACCHO-embedded paediatric dermatology clinic was made for 27% (9/33; 95% CI 14–46%) of CYP with AD.

For dermatophyte infection, topical azole therapy (cream, shampoo, powder) was prescribed in 61% (14/23), combination topical steroid-antifungal in 22% (5/23) and terbinafine cream in 9% (2/23). No systemic antifungal therapy was prescribed for nail or scalp dermatophyte infection, but one patient with tinea capitis was referred to the ACCHO-embedded paediatric dermatology clinic.

All 15 patients with scabies were treated with Permethrin 5% cream.

Discussion

In the first study to investigate the burden and clinical characteristics of skin disease in urban-living Aboriginal CYP in a primary care ACCHO clinic, we found:

- dermatological disorders were addressed in 27% of face-to-face GP consultations, with a high burden of skin infections and dermatitis
- AHP skin screening aided GP assessment by identifying 'abnormal skin' in 21% of face-to-face GP consults, and facilitated head lice treatment in 9%
- BSI affected one-in-eight CYP, yet recurrent BSI affected <1% and only 1% of BSI episodes required hospitalisation
- ACCHO-embedded specialist dermatology care was sought for 27% of CYP with AD.

At least one dermatological disorder was addressed in 27% of face-to-face GP consultations in our study; these were most frequently infections and dermatitis. Data for comparison for non-Aboriginal urban-living CYP is limited; however, two studies provide data for all urban-living Australian CYP. In the first, 20% of consultations for CYP (aged 0–19 years) in two general practices concerned a dermatological problem; most commonly eczema/dermatitis (31%) and infections (15%).[15] In the second study that analysed adolescents (aged 10–19 years), skin problems were managed in 24% of consultations; with infections, acne and dermatitis most frequent.[16] Although the proportion of dermatological disorders addressed in our study is similar to that described for all urban-living Australian CYP, the burden of infections is higher, with 14% of all GP consultations addressing at least one skin infection, compared with 3–6% for all urban-living Australian CYP.[15,16] Our results are similar to those reported for remote-living Aboriginal children (aged <7 years), where skin infections accounted for 16–18% of primary care presentations.[17,18]

Our finding that more than one-quarter of GP assessments addressed a dermatological disorder is supported by the AHP-led model of care provided, where AHP skin assessments identified 'abnormal skin' in 21% for GP assessment. AHP head lice assessments occurred prior to 65% of face-to-face GP consultations, providing opportunistic treatment in 9% to potentially reduce BSI; our study also indicated head lice-precipitated BSI in 4%. Our study suggests an 18% prevalence of head lice, consistent with previous reports about urban-living Aboriginal CYP.[19] It is important to note that the background prevalence of head lice in non-Aboriginal urban-living CYP is unknown as the AHP-delivered assessment is unique to the ACCHO setting, hence head lice might go undiagnosed in mainstream primary care.

We found one new BSI for every eight CYP attending the urban ACCHO over 12 months, peaking in Bunuru (February/March), which is consistent with descriptions of increased BSI in late summer in hot, dry environments.[6] Our BSI incidence rate of 142/1000 child-years-at-risk in primary care is much higher than linked hospitalisation data (1996–2012) for urban-living Aboriginal

CYP in WA (20.8/1000 child-years-at-risk).[6] Primary care ACCHO consultations provide an opportunity to prevent hospitalisation through early detection and treatment of skin infections by the AHP and GP. Only 1/82 (1.2%) BSI episodes required hospitalisation, which compares favourably with a New Zealand study of Indigenous children that estimated there to be 14 primary care cases of BSI for every one case requiring hospitalisation.[20] Recurrent BSI affected <1% (5/585) of CYP, which is less than that reported in a 2000–01 carer-completed survey of urban-living Aboriginal children where 7.1% (720/10,200) described 'recurring skin infections such as school sores or scabies'.[21]

We report a 16% lifetime prevalence of AD, similar to the 19% lifetime prevalence of parent-reported 'eczema ever' described in a community cohort of urban-living Aboriginal CYP.[19] These results add to the growing knowledge of AD burden among urban-living Aboriginal CYP, where 'eczema ever' has been reported in 13–25% (for children aged <6 years).[22,23] In our study, the 12-month contact prevalence of AD was significantly less (6%) than the lifetime prevalence (16%). This reflects the chronic nature of AD and that once diagnosed and treatment has been initiated, patients might not need to visit their GP as often due to good control, improvement in disease severity (as can occur later in childhood) or if they are receiving specialist care. Alternatively, reduced GP visits might not correlate with better control, but perhaps reflect a lack of understanding of the chronic nature of AD, which benefits from regular physician review to ensure optimal control. In our study, AD presentations peaked in Kambarang (October/November), which is consistent with data from Korea, the Netherlands and US that show increased AD symptoms and/or healthcare utilisation in Spring, reflecting increasing ambient temperatures and/or the pollen season.[24–26] In the Netherlands, children with flaring AD in the pollen season more often had hay fever at a younger age and a dark skin type.[25]

The 12-month contact prevalence for dermatophyte infections was 4%, significantly less than the 19% point-prevalence of dermatologist-diagnosed dermatophyte infections (including 10% point-prevalence of tinea capitis) in urban-living Aboriginal CYP at a WA community skin screening event; this suggests possible underdiagnosis in primary care.[19] We advise a high index of suspicion for tinea capitis in CYP noted to have both scalp scaling and hair thinning (82.1% sensitivity) or scalp scaling alone (60% sensitivity).[27] Likewise, tinea affecting any skin or nail site should prompt thorough scalp examination.[28] In our study, dermatophyte infections peaked in Djilba (August/September), alongside the scabies peak. Although scabies has been reported to predominate in winter due to enhanced mite survival and closer living encouraged in cooler temperatures, the same

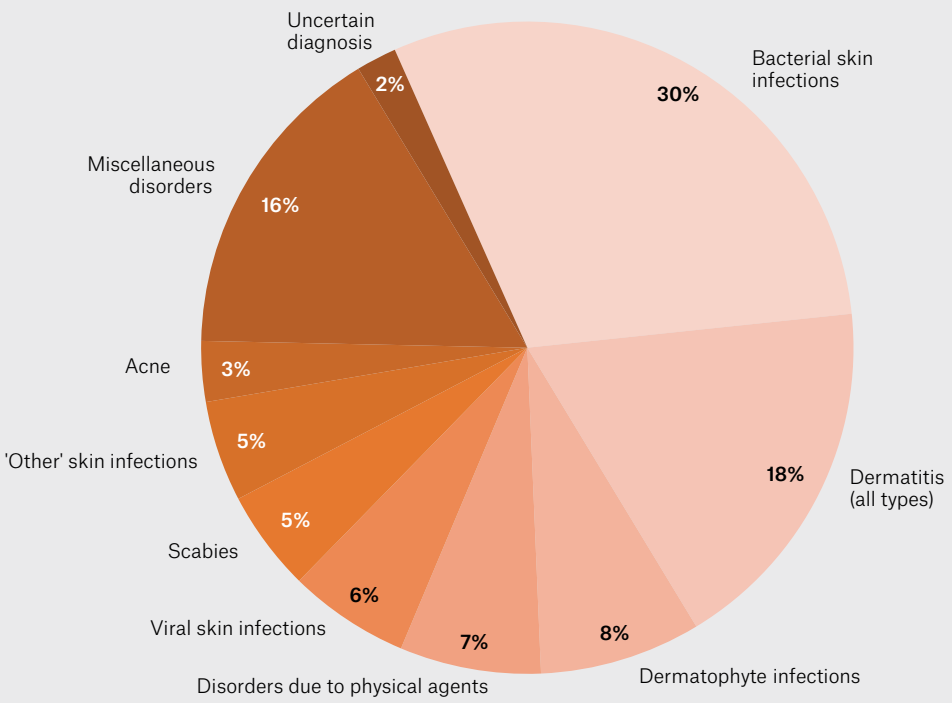

Figure 3. Proportion of dermatological disorders addressed in primary care.

predominance of dermatophyte infection has not been described; however, the element of closer living might be relevant.[29]

Evidence-based guidelines, such as the *Antibiotic and Dermatology Therapeutic Guidelines* (www.tg.org.au) and the *National Healthy Skin Guidelines*, are helpful resources for Australian primary care providers; the latter focussing on Aboriginal children and communities.[30] Our assessment of GP documented treatments found that, overall, these guidelines were well adhered to. We did note frequent (22%) prescription of topical steroid–antifungal combinations for tinea corporis, a practice we discourage due to the potential of the steroid component to worsen tinea, cause skin atrophy and striae, and contribute to emerging public health concern of antimicrobial-resistant tinea infections.[31] We also noted an absence of fungal culture and systemic antifungal prescription for tinea capitis, where species identification and prescription of oral terbinafine is recommended first-line treatment; however, referral was made to the ACCHO-embedded paediatric dermatology clinic.[32] Referral to this clinic was also made for over one-quarter of CYP with AD, supporting the value of this service in providing prompt and accessible care.

Limitations include retrospective design and potential for information bias as data were obtained from patient EMRs. The data collection period coincided with the COVID-19 pandemic. During this time, health-seeking behaviours were impacted, telehealth consultations increased and the clinician's approach to face-to-face consultations was adjusted to minimise patient contact time, resulting in reduced opportunistic skin examinations and potential underdiagnosis of skin disease. In addition, it is a single site study, which limits generalisability and external validity, and might have had a reduced number of infant assessments as young families often access the peripheral Derbarl clinics. Beyond Derbarl, urban-living Aboriginal CYP with a health complaint in metropolitan Perth might attend a non-ACCHO GP practice or one-of-four emergency departments that assess children. Univariate analysis was used to compare frequencies of categorical variables between outcome variables, with the inherent risk of confounding in these relationships. The findings of this study must be considered in the context of these sources of potential bias.

This is the first study to investigate skin health in urban-living Aboriginal CYP presenting to primary care. The results indicate that dermatological disorders account for a significant proportion of the ACCHO GP workload, with a high burden of skin infections and dermatitis identified. BSI affected one-in-eight urban-living Aboriginal CYP presenting to this ACCHO, yet recurrent BSI affected <1% and only 1% of BSI episodes required hospitalisation; this suggests early and appropriate treatment minimised recurrence and hospitalisation. We present a culturally secure, multidisciplinary skin health assessment model involving AHPs, GPs and dermatologists within an urban ACCHO, which can be emulated throughout the nation to help achieve optimal outcomes for patients and their families.

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Medical certificates:
More than just paperwork

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Background
Medical certificates communicate the needs and conditions of a person to (often) non-medical entities or other stakeholders. Medical certificates can have a profound effect on patients' access to social and financial support, and therefore wellbeing. However, general practitioners (GPs) are not formally trained in effective completion of medical certificates, leaving opportunity for workforce development.

Objective
This article discusses the significant role of medical certificates in patient care and the challenges GPs face in completing medical certificates.

Discussion
Medical certificates are often the only mode of communication between GPs and non-medical entities. The tone, comprehensiveness and content of medical certificates can influence the document's utility. There are limited guidelines and formal education for GPs on how to write an effective medical certificate. Designing and implementing guidelines along with appropriate training for GPs will likely result in better patient outcomes in line with their support needs.

MEDICAL CERTIFICATES are produced in general practice for a variety of reasons. Their purpose is usually to inform a receiver of an individual's medical condition(s) and their (in)ability to work or participate in occupational duties. The more familiar type for patients is a concise 'sick note', which certifies a temporary non-serious illness that has caused a short-term incapacity to work. However, there are more complex medical certificates produced by general practitioners (GPs) to communicate the circumstances of an individual to a third party. They range from certifying letters to specially designed forms, such as a Work Capacity Certificate (workers' compensation form [132m])[1] or a Centrelink Medical Certificate form (SU415).[2] In this article, we focus on the latter type. The receiver of these certificates is often a non-medical person, such as a social security officer, employer or insurance agent. Writing a medical certificate is widely acknowledged as a challenging task, especially for international medical graduates (IMGs) with limited knowledge and experience of the health and social systems where they are practising.[3,4] Our first author (PD) vividly recalls her first day working as a GP in Australia; needing to produce a worker's compensation certificate for an injured worker while feeling anxious and frustrated from lack of experience and training in writing medical certificates and also the need to communicate clearly and professionally in a second language.

This article aims to review the scant literature available on the topic of writing medical certificates and, in doing so, discuss some of the challenges GPs face in the process by putting two possible responses in the spotlight. We have divided the challenges of GPs producing medical certificates into two categories: (i) the double roles GPs play as assessor and advocate; and (ii) the importance of words used for the 'success' of the case. Subsequently, we will address potential measures aimed at improving the current situation.

GPs in double roles: Assessor and advocate

Completing a medical certificate can be perceived by GPs as a complicated and bothersome task.[3,4] This is because GPs often have a close relationship with their patient and might find it difficult to advocate for positive outcomes while also fulfilling the requirements of an objective assessor in certifying sickness/disability. In the nationwide cross-sectional study by Engblom et al of primary care physicians in Sweden, it was reported that sickness certification was challenging for half of the respondents.[3] Many GPs report that the certification process can

also be a source of conflict and tension in the therapeutic relationship, frequently resulting in dissatisfaction about the process.[4,5]

Words matter: Effective completion of certificates

Effective completion of a medical certificate begins with accessing the right form and selecting the appropriate option, which can be a challenge considering the wide variety of forms available for different stakeholders. Then comes the important job of completing the free-text sections (this can get more streamlined by the potential introduction and adoption of online certificates). Accessing social benefits or insurance claims heavily relies on the language used to communicate the patient's condition. This means that words that are used to complete a medical certificate can have a significant effect on the action taken by stakeholders.

Enhancing the coherence of the words in the medical certificate has been suggested as a way to improve the pace of recovery and return to work for the patient.[6] The approach to communication, whether it is emotionally persuasive or strictly factual, can also impact the success of the claim.[7] However, certificates worded based on emotions that appeal to the reader's goodwill are often overlooked by the authorities.[7] Similarly, how the certificate is worded can have a serious impact on a worker's mental health, especially considering the associated stigma and negative stereotypes often directed at claimants, portraying them as opportunistic in pursuing financial support.[8] This stigma can have a negative effect on a range of issues, including the desire to access worker's compensation by an injured worker, recovery and early return to work.

Navigating forward: The call for greater guidance and support

According to the Australian Institute of Health and Welfare, work-related injuries and illnesses cost $28.6 billion annually.[9] The majority of the injured workers are initially assessed by a GP to determine their capacity to return to work. More than 70% are deemed to be unfit-for-work on their initial certificate.[10] Similarly, GPs are less likely to write a fit-for-work certificate for mental health claims.[11] This trend is noteworthy considering the potential benefits of early return to work, which have been linked to increased employment participation and reduced societal costs.[12] The discrepancy in fitness assessments by GPs might be improved by further training.

Although several countries including Australia introduce some courses in communications for medical practitioners and assess a doctor's communication skills via objective structured clinical examinations (OSCEs),[13] little formal training exists for writing medical certificates. One positive initiative was the *Australian Family Physician* Journal that published a 'Paperwork' series in 2011 to provide guidance for GPs on how to best complete various medical certificates. Some of these articles focused on describing the legal aspect of sickness certification[14,15] and the legality of producing medical reports,[16] whereas others provided guides on filling out commonly used forms such as those for Centrelink,[17] Worker's compensation,[18] Department of Veterans' Affairs,[19] a death certificate,[20] motor accident insurance,[21] pre-employment medical[22] and fitness to drive forms.[23] Although these guides are very helpful in understanding the legal issues involved with producing a report, such as the structure and the relative code of conduct, they do not underscore the importance of the language used to achieve the desired outcome.

Another example is Sweden, which introduced nationwide guidelines for sickness certification in 2007.[24] Most Swedish GPs found the guidelines beneficial in ensuring accurate sickness certification and more effective communication with stakeholders.[25] However, almost half of those using the guidelines found it challenging to adhere to, highlighting the need for training to enhance competence. Similarly in Australia, when WorkSafe Victoria (WSV) and the Transport Accident Commission (TAC), Victoria's two statutory injury compensation authorities, redesigned their sickness certificates in 2013 to focus more on capacity rather than sickness, it was reported by four stakeholder groups (GPs, injured workers, compensation agents and employers) that more training for GPs is needed to improve the quality and outcomes of these certificates.[26]

Notably, simply developing guidelines for medical certificates might result in arbitrary decisions without considering the context in which the patient presents. A GP's assessment of a client's capacity is less of a technical matter and more of a normative one, meaning that doctors should be able to articulate their considerations and arguments in an 'open manner'.[27] Developing guidelines on medical certification, along with proper training for GPs on how to apply them in a manner that is open and flexible, can help GPs to improve the outcome for patients and health and social systems.

Discussion

Here, two examples are presented, which come from two forms that are regularly completed by GPs as part of their medical certification. Both are constructed by authors based on similar responses reviewed for this article. The first one is the Work Capacity Certificate – Workers' Compensation Form (132m),[1] where we focus on two possible responses that could be produced only for Part D. This is where GPs are asked to certify that there is no functional capacity for any type of work and why (Figure 1). Table 1 compares a common response written for Part D with what the authors recommend.

The common response does not indicate an actual medical diagnosis and fails to consider the worker's duties in assessing the functional capacity. This might confuse the Workcover agent who must make decisions based on such medical advice. In the recommended response, the words are chosen factually with detailed information necessary for an objective decision. It is, at the same time, not too detailed considering the time constraints GPs face.

Figure 2 is taken from the Centrelink Medical Certificate form (SU415)[2] where GPs are asked to write about the functional impact of all conditions related to the patient.

Similarly here, as shown in Table 2, the common response fails to address the symptoms of anxiety/depression and a medical diagnosis that might delegitimise one's entitlement to sickness benefits. Nor does it explain how major depression affects the ability to perform work duties. Objective wording helps GPs balance their double role of assessor and advocate for their patients. When words sound objective and are based on a clear diagnosis, it makes a better impact on the reader (the other stakeholder) assessing the form. The fact that medical certificates

are a *written* genre of communication by GPs makes it necessary to point out that, as shown in studies in linguistics, writing is permanent and as such, it is associated with authority and credibility.[24] In addition, as it is written, this piece will accompany the patient through their passage in the healthcare system.[28]

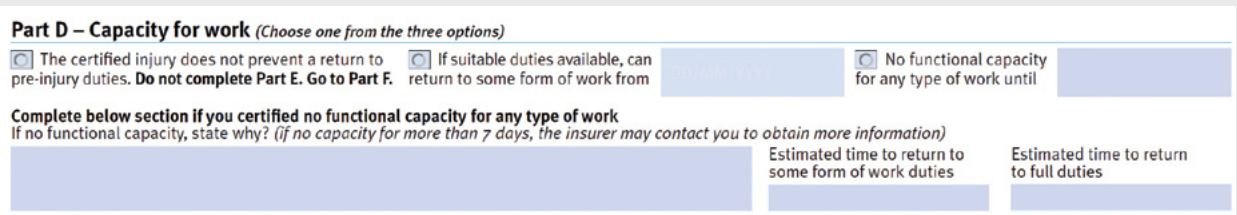
Therefore, it is of utmost importance to consider the impact this communication makes on various parties in this process. For patients, participating in the process of medical certification and accessing social benefits can have a major impact on their livelihood and mental wellbeing.[8] Fragmented interactions can impede recovery as patients feel that their entire situation is not being considered.[6] This is echoed by injured workers in Australia who report that they deal with disengaged case managers, negative stereotyping, insufficient related information, suspicious reactions from all the stakeholders and a lack of professionalism in the communication from service providers.[29] These challenges can hamper the recovery process and exacerbate the psychological toll on patients trying to navigate the complexities of medical certification.

From a GP's point of view, being the gatekeeper for sickness certification can adversely affect the doctor–patient relationship. GPs often find it impossible to reconcile the demands of the other stakeholders with the complexity of the patient's needs.[30] In contrast, the implementation of clinical criteria and standardisation might risk oversimplifying the evaluation process that overlooks the contextual reasoning and normative dimension of individual cases,[23] resulting in an unwanted conflict for the doctor.[31] Finally, the GP's dual role as both advocate for the patient and arbiter of clinical judgment introduces an additional layer of complexity, potentially influencing the doctor's objectivity in the certification process.[32]

Employers often believe that GPs have a poor understanding of the complexity of the Worker's Compensation system. This belief is argued to be due to the common occurrence of incorrect, incomplete or inadequately detailed certificates received by employers.[33] The deficiency in information extends to remuneration details and a lack of awareness about specific workplace conditions. Moreover, employers reported feeling excluded from the process while harbouring suspicions about injured workers exploiting the system.[33] This disconnect between GPs and employers underscores the urgent need for improved communication channels.

Conclusion

The challenges faced by GPs in writing medical certificates are multifaceted and have significant implications for patients, the healthcare system, doctors and employers. The wording in certificates, the double role of GPs, and the need for further training all reduce the likelihood of a certificate accurately and fairly supporting a patient. Professional training programs for GPs are needed to provide the necessary knowledge and skills to improve communication and navigate the complexity of medical certification effectively. These must include clear guidelines, protocols and standardised procedures that provide clarity on certification criteria, appropriate wording and decision making. They need to be flexible for GPs to word the certificates in an open


Part D – Capacity for work *(Choose one from the three options)*

The certified injury does not prevent a return to pre-injury duties. **Do not complete Part E. Go to Part F.**

If suitable duties available, can return to some form of work from

No functional capacity for any type of work until

Complete below section if you certified no functional capacity for any type of work
If no functional capacity, state why? *(if no capacity for more than 7 days, the insurer may contact you to obtain more information)*

Estimated time to return to some form of work duties

Estimated time to return to full duties

Figure 1. Workers' compensation form (132m).

Reproduced from the Queensland Government, WorkSafe. Work capacity certificate - medical providers. Queensland Government, 2020. Available at www.worksafe.qld.gov.au/service-providers/medical-providers/work-capacity-certificate, with permission from the Queensland Government.

Table 1. Responses to Part D: Work capacity certificate – Workers' compensation form (132m)

Question	A common response	Recommended response
'Complete below section if you certified no functional capacity for any type of work. If no functional capacity, state why'	After lower back injury, the worker is unable to perform their regular work duties	The diagnosis of lumbar strain with associated muscle spasms prevents (this worker) from fulfilling the requirements of their role, which include frequent lifting of objects weighing up to 20 kg, prolonged periods of standing, and frequent bending and twisting motions

manner. It is important to highlight that the introduction of technology in producing medical certificates can facilitate the process. This can be a relevant topic for further studies and practice-focused special issues. The art of communication in terms of ongoing education in communication skills[34] must extend to the written communications GPs make on a daily basis with clear consequences for the healthcare system and patients.

Key points

- The wording of medical certificates influences outcomes significantly, affecting patients' mental health, financial wellbeing and recovery.
- GPs struggle with dual roles as advocates and assessors, which leads to conflicts and dissatisfaction in therapeutic relationships.
- GPs' assessments of work-related issues require a nuanced approach to consider broader societal implications for better outcomes, hence highlighting the need for guidelines and training programs for effective communication.
- GPs are advised to choose factual words and expressions to describe the diagnosis, with detailed information necessary for an objective decision, considering the time constraints GPs face.
- Objective written communication that is based on a clear diagnosis makes a better impact on the reader (the other stakeholder) assessing the medical certificate.

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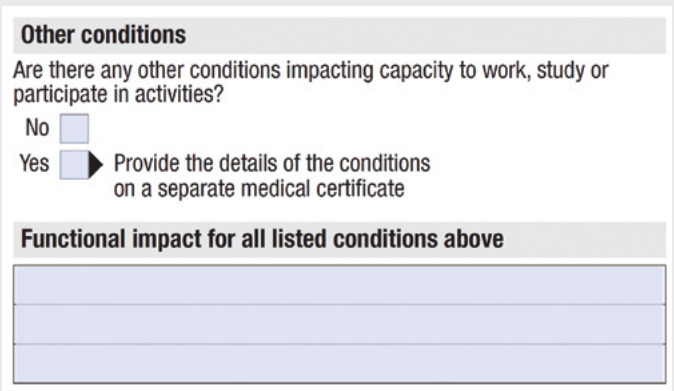

Figure 2. Centrelink Medical Certificate form (SU415).

Reproduced from Australian Government, Services Australia. Centrelink Medical Certificate form (SU415). Australian Government, Services Australia, 2023. Available at www.servicesaustralia.gov.au/su415, with permission from the Australian Government, Services Australia.

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Table 2. Responses to the Centrelink Medical Certificate form (SU415)

Question	A common response	Recommended response
'Functional impact for all listed conditions above'	(Patient name) cannot work due to anxiety/depression	(Patient name) is diagnosed with major depressive disorder causing feelings of sadness, hopelessness and worthlessness with increased irritability, difficulty concentrating and disrupted sleep patterns. These symptoms significantly impact her functioning and ability to perform her duties as an accountant effectively

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General practitioner professional identity formation: Much needed, (still) oft forgotten

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For the purposes of this article, we will be using the term 'general practitioner' (GP) to refer to providers of primary care in the community setting. In other countries, the terms 'family physician' or 'primary care practitioners' are also used. Similarly, the terms 'trainee' and 'training program' used in this article are synonymous with the terms 'resident' or 'registrar' and 'residency program', respectively.

Background
Professional identity formation (PIF) as a general practitioner (GP) enhances the experience of meaning at work, reduces burnout, promotes patient-centred decision making and strengthens advocacy for the unique role of family medicine within the healthcare system. Unfortunately, there is insufficient emphasis on PIF in GP residency programs; it often remains a hidden curriculum. External factors limiting GP trainees' exposure to their own general practice community of practice further impede PIF. Thus, fostering general practice PIF requires deliberate, focused efforts; however, there is little empiric evidence on the GP professional identity or how to foster it.

Objective
The aim of this paper is to discuss strategies for effective GP PIF in family medicine residency.

Discussion
The authors recommend explicit attention to PIF in three key areas: curriculum; teaching and learning; and faculty development. Additionally, the authors encourage GPs to unite as a community to provide continuing and coordinated support for GP residents on their PIF journey.

PROFESSIONAL IDENTITY is the unique set of values, beliefs, attitudes and behaviours that define and distinguish a professional group.[1] What constitutes the general practitioner (GP) professional identity is well described – GPs value and pursue the provision of personal, primary, preventive, comprehensive, continuing and coordinated healthcare of the individual in relation to their family, community and environment.[2,3] However, there is a lack of focus on GP professional identity *formation* (PIF),[4,5] described as the dynamic process of personal development and social construction in which these core values, beliefs, attitudes and behavioural norms of general practice are internalised, such that one thinks, acts and feels like a GP.[6,7]

PIF increases one's experience of meaning at work, through strengthening one's sense of coherence, purpose and significance.[8] It is through this that strong PIF is associated with positive outcomes, such as an improved sense of wellbeing,[8] increased job satisfaction and reduced burnout.[9] Strong PIF is also linked to lower turnover intention (the intention to quit one's profession).[10–12] PIF could be an important ingredient for communities struggling with retention of talent, such as the rural generalist community.[13,14] PIF is also postulated to enhance patient-centred care by enabling nuance in decision making within the complexities of social interactions and patients' relationships with their families.[15,16] Finally, GPs with a well-developed professional identity can also better collaborate with other healthcare providers and advocate for the unique role and value of primary care within the healthcare system, thereby promoting the advance of the entire speciality.[17]

PIF and its significance is not new to the GP community, with calls to strengthen PIF as early as 2014.[18] But why has there been so little change almost 10 years on?[4] One reason is that PIF often remains a hidden curriculum.[19] Although many GP training programs worldwide emphasise professionalism and ethical behaviour in their curricula,[20–23] GP PIF is alluded to at best.[4] The extent to which GP trainees experience PIF can vary depending on the favourability of existing program structures, the degree of opportunistic role modelling by clinical supervisors, and personal reflection on their professional practice.

Additionally, several external factors act as barriers to a GP trainee's PIF. Time constraints arising from high workload and long hours[4] can limit opportunities for reflective practice and self-reflection. Limited exposure to the GP community of practice, especially in the early or pre-vocational years of training, might impede the socialisation process that is the key driver of PIF.[6] Fragmented patient encounters that result from frequent rotations might also limit trainees' experience of long-term patient relationships and continuity of care, which is one of the cornerstones of general practice. During required hospital-based rotations, GP trainees can also experience cognitive dissonance between the working priorities of the hosting hospital department and what is relevant to learn for general practice. The culture within many healthcare institutions that prioritises the depth of specialist knowledge and skills training and research, over the personal, primary, preventive, comprehensive, continuing and coordinated healthcare values of general practice[24] further impedes GP PIF.

Thus, GP PIF is challenging and unlikely to develop without deliberate efforts to foster it. Curricular enhancements aimed at fostering PIF have been informed by social learning theories.[6] Such enhancements include reflective practice,[25,26] narrative medicine,[27,28] peer-assisted learning,[29,30] simulation,[31,32] role modelling,[33–35] involvement in professional communities of practice,[36–38] longitudinal integrated clerkships[39] and professional development programs.[40–42]

Much of the literature on PIF has been focused on medical students and young doctors forming the professional identity of being a compassionate and competent physician.[43–47] Little has been published on GP professional identity[5,48] and how to foster its formation.[18,34] Role modelling has traditionally been used to imbue GP trainees with beliefs, values and principles, through mentoring, supervision, coaching, tutoring and advising.[34] Innovative efforts include Nothnagle's 'Forum' intervention,[18] in the form of scheduled individual and group discussions, as opportunities for guided reflection on PIF.

Recognising that developing the professional identity of a GP is a process of socialisation, we use the framework described by Creuss et al[6] and propose being explicit about PIF in three key areas.

Curriculum: Creating space and time for PIF in curriculum

Prevocational training

To fully reflect the health needs of a population, prevocational training should occur in a range of settings, including hospitals in metropolitan, regional and rural communities, general practices and other community-based health services.

For example, in Australia and New Zealand's prevocational training program,[49] postgraduate year 2 doctors complete a longer 24-week 'blended' term in a rural setting that combines general practice, ward-based and emergency department experience. Different days of the week are spent in different settings with different supervisors, or some weeks are spent in one setting before switching to another. Incorporating opportunities for exposure to general practices and the GP community of practice allows potential GP trainees to begin developing GP PIF even before they enter GP training.

GP training

GP training programs should be structured to allow GP trainees to remain connected to the GP community of practice and the work of general practice, as well as adequate opportunity to reflect on and discuss GP PIF.

For example:

- Weekly general practice clinics in a 'home' clinic throughout the entire GP training period, where trainees see patients under the tutelage of dedicated GP supervisors; akin to a longitudinal integrated clerkship. Doing the work of a GP socialises the GP trainee into the GP community, and helps trainees shift from *doing* as a GP to *thinking, acting and feeling* like a GP (ie *becoming* a GP).[5] Starting this process early is especially helpful for residents in GP training programs that front-load hospital-based postings in the first one to two years of GP training, such as in Singapore and the United States.[20,23] Having a 'home' clinic throughout the entire GP training period allows GP trainees to build relationships with patients and provide continuing care. Having a dedicated GP supervisor creates room for mentorship and role modelling to take place.
- Designated learning activities focused on PIF; this can take the form of guided reflection opportunities by GP supervisors for their GP trainees to make meaning of challenging or conflicting experiences during training.[43]

Teaching and learning: Developing a language for professional identity

Much like naming and acknowledging emotions in difficult conversations, there is power in inviting the GP trainee to mentally place themselves in the role of a GP and explicitly commit to what they would do for their patients. There is also power in verbal affirmation[50] when GP trainees have demonstrated the values, beliefs, attitudes or behaviours of a GP. For example:

1. When GP supervisors discuss patients that GP trainees encountered in the hospital, we might ask 'If this patient were to have *presented to you in primary care*, how would you have managed him/her?'
2. When discussing patients that GP trainees see in primary care, we might ask 'What do you think *your role as a GP* is in this consultation, at this point in the patient's care?'
3. When a GP trainee has compassionately and effectively addressed a patient's specific concerns, we might say 'That was a therapeutic encounter for the patient, and you have done well to provide personalised care and advice.'

Faculty development: Emphasising on intentionality

Although programs or interventions are helpful for facilitating the development of GP PIF, they will lack effectiveness if GP supervisors are not intentional about or not equipped with the knowledge and skills to guide conversations around GP PIF. Rather than hyper-focus or over-rely on programmatic interventions, GP training programs should consider prioritising faculty development for PIF.[7,37] Using the cognitive apprenticeship model, faculty members should intentionally seize learning moments to role model, coach and explicitly articulate GP professional identity values, beliefs, attitudes and behaviours during structured teaching and observed clinical encounters.[51] GP supervisors should also be adept in facilitating GP trainees'

reflections and explorations of their GP professional identities.

Amidst rising concerns of physician burnout and waning job satisfaction, GP PIF is an important factor in sustaining physician wellbeing, upholding patient-centred care and in the growth of the GP community of practice – all of which are in line with the focus areas of the Australia and New Zealand Medical Deans Strategic Plan.[52] More can to be done to intentionally and explicitly develop GP values, beliefs, attitudes and behaviours through programmatic interventions, reducing barriers, as well as faculty development. Tools for measuring GP PIF need to be developed,[53,54] in order to evaluate outcomes of interventions and direct future efforts. The transformative journey of PIF does not end with medical schools, but should continue into GP training programs and beyond. Let GPs unite as a community to provide continuing and coordinated support for GP trainees on their GP PIF journey.

Key points

- GP professional identity formation (PIF) is important for the GP community of practice to grow. It protects against burnout, enhances patient-centred care and enables better advocacy for the value of general practice within the healthcare system.
- GP PIF is challenging in GP training and is less likely to develop without deliberate efforts to foster it.
- Strategies for overcoming the barriers to GP PIF include:
  - creating space and time for GP PIF within the training curriculum
  - developing a language for professional identity
  - developing faculty to be intentional and equipped to facilitate reflective conversations around GP PIF.
- Tools for measuring GP PIF need to be developed in order to evaluate outcomes of interventions and direct future efforts.
- Let GPs unite as a community to provide continuing and coordinated support for GP trainees on their GP PIF journey.

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How to use community HealthPathways: Practical tips to support decision making in the consulting room

Edwin Kruys, Jon Harper

Background
Community HealthPathways are clinical decision support tools, combining evidence-based guidelines with local service and referral information, collaboratively developed and collated by primary care and hospital clinicians. HealthPathways is being implemented throughout Australia, New Zealand and the UK, and often plays a role in supporting local service redesign and integrated care.

Objective
This article summarises the background and benefits of community HealthPathways and provides tips to support decision making in the consulting room. The article highlights the influence a community of clinicians can have on service redesign through developing and publishing HealthPathways.

Discussion
Clinical information-seeking is often done during consultations, and having access to evidence-based, decision support tools like HealthPathways can facilitate consistency of best practice clinical care across jurisdictions and streamline referrals to local health services. Through the process of collaboration, HealthPathways supports integration of health services. Knowledge about the structure of HealthPathways and how to find information is useful for busy clinicians to aid decision support at the point of care.

HEALTHPATHWAYS started in Canterbury, New Zealand, in 2008, and is being implemented throughout Australia, New Zealand and the UK. The HealthPathways team describes the initiative as 'an online manual used by clinicians to help make assessment, management and specialist request decisions'.[1]

There are many other descriptions of HealthPathways, published in various research articles, government websites and promotional materials. A concise one is 'a web-based tool designed to promote healthcare integration and patient management in primary care'.[2]

HealthPathways has two main functions: as a clinical decision-making tool based on existing guidelines; and as a healthcare services directory containing referral information. These functions exist separately in several other web-based platforms, but HealthPathways is unique in combining the two functions through a collaborative process.[3] HealthPathways can be used for continuing professional development (CPD), and reflective activity templates for The Royal Australian College of General Practitioners (RACGP) and The Australian College of Rural and Remote Medicine (ACRRM) have been developed and are available on HealthPathways.

The website is password-protected, and clinicians are given access at no cost. There is a strong focus on collaboration between local primary care and hospital services, which sets HealthPathways apart from traditional guidelines.[4,5] The information in HealthPathways is developed and collated by local HealthPathways clinical editorial teams, usually including general practitioners (GPs), in collaboration with specialist clinicians (Figure 1).

In Australia, the work is often coordinated between a State-funded health service and a Commonwealth-funded Primary Health Network (PHN).[6] Fifteen years ago, the development of HealthPathways content was both a catalyst for, and a product of, health service redesign.[7,8] Over the years, HealthPathways remains an important tool in many areas to assist local service redesign and integrated care implementation.[9–11]

During the COVID-19 pandemic, efficient and effective processes were developed to respond to rapid changes in the management of COVID-19-related problems and make new information available on HealthPathways to clinicians in a timely fashion.[12] Researchers describe several other benefits that can be attributed to local HealthPathways programs, with various levels of confidence.[3,13–17]

The reported benefits of HealthPathways fall into two main themes: care provision; and use of local resources and services (Box 1). HealthPathways content has been created to not only improve clinical practice, but also improve the patient journey through the local health system.

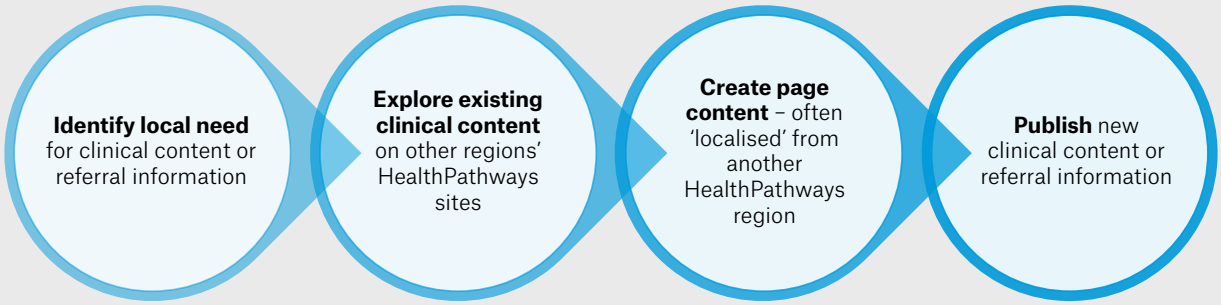

Figure 1. The process for HealthPathways content creation and regional localisation.

There is variation in utilisation of HealthPathways,[18] with long-established HealthPathways regions having greater clinician engagement,[2,9] as do regions that actively promote the HealthPathways role in education and service redesign.[19]

One observational economic analysis found a reduction in hospital outpatient visits and associated cost savings after the introduction of HealthPathways-containing comprehensive referral information.[16] Although clinical pathways are generally associated with improved patient outcomes and positive economic outcomes,[20] ongoing evaluations of the outcomes of HealthPathways remains a priority.[21,22]

In this article, we describe practical tips for the use of community HealthPathways during general practice patient consultations, addressing some of the feedback and questions from clinicians regarding use and navigation of the site as mentioned in the literature.[4,13,18]

Box 1. Community HealthPathways benefits

Care provision

- Adoption of best practice clinical care
- Consistency of care across jurisdictions
- Single source of truth during pandemics, natural disasters

Use of local resources and services

- Improved patient journey through the health sector
- Improved awareness of local services
- Appropriate use of resources and services
- Improved referral quality
- Equitable referral triage
- Reduction in cost/increase in value

Tips for using HealthPathways in the consulting room

Tip 1: Keep the HealthPathways website open

It appears that clinical information-seeking by clinicians is often done during consultations.[23] It can be useful to bookmark HealthPathways or create a desktop shortcut on a computer (or mobile device) used during clinic.

To save time, the website can be one of the resources that remain open within the desktop internet browser during patient clinic consultations for quick reference. Having the website open can also be a prompt to use and explore the resource, as HealthPathways might not always be front of mind for busy clinicians.[18]

The local version can be easily found by searching for 'HealthPathways' and the name of a region or district in online search engines. When accessing the website for the first time, registration is required to request login details. Depending on the clinic browser and security settings, login information can be saved and automatically filled for returning site visitors to facilitate ease of access.

Tip 2: Use HealthPathways for day-to-day clinical decision support

As HealthPathways summarises available evidence and puts it in a local context, it is a valuable source of information to support decision making at the point of care.[12,24]

After logging in, the homepage provides general information such as relevant public health alerts, regional health news, updates to HealthPathways, local service information and links to clinical and non-clinical resources.

From the homepage, it is easy to search for pathways about a specific problem ('condition pages') or find referral information ('request pages').

Tip 3: Search HealthPathways via the top search bar

To quickly find page results or suggestions, start typing keywords in the top search bar (Figure 2). The search function accommodates minor differences in spelling and common synonyms of conditions.

Alternatively, the left-hand table of contents can be used by clicking on the down arrows (⌄) to expand sections. This can also be useful to view all the available pathways for a certain specialty or discipline.

To return to the homepage at any time, click the HealthPathways logo icon at the top left of the header bar (next to the region name) or click the HealthPathways logo at the top of the table of contents. Alternatively, click the home icon in the page 'breadcrumbs' at the top of a pathway, or on the word 'Home' at the top of the table of contents.

Pages with a yellow background have not yet been localised in the user's region but might still contain useful content.

Tip 4: Know how the condition pages are organised

All condition pages have the following sections:

- Background
- Assessment
- Management
- Request (referral)
- Information.

Some pages also have a 'Red Flags' section at the top of the pathway; the content of these boxes is designed to remind the user of important clinical features not to be missed.

The 'Assessment' section is useful for looking up diagnostic considerations and prompts, whereas the 'Management' section provides treatment options. The 'Request (referral)' section gives information about when to refer, including links to 'Request pages' (see Tip 5). The 'Information' section contains further reading for health professionals and health consumer information, including links to handouts.

To expand specific information in a pathway, click on the blue text or the down arrows (⌄). Use the plus (+) sign in the right top corner to expand all information in a pathway and access other options including printing, sharing and copying of information contained in HealthPathways.

Tip 5: Know where to find referral information

Request or referral pages can be accessed directly from the top search bar or via the links in the 'Request (referral)' section towards the bottom of a condition page. These pages provide public and private referral options where available, as well as referral criteria including required information.

Checking a 'Request (referral)' page can provide guidance about the required steps to take prior to referring a patient to another service. Health services, particularly State-run services, update their triage and referral criteria from time to time; by viewing the 'Request (referral)' page information, referrers can ensure their referral letters are complete and meet the criteria to facilitate acceptance by the service. In some cases, HealthPathways is seen as the defacto directory for public services.[11] By housing cross-sector service information, HealthPathways facilitates an improved patient journey. In many areas, referral information has also been incorporated in electronic referral software.

Tip 6: Contribute to the HealthPathways community

There are several ways to participate in the development and improvement of HealthPathways: providing specific feedback; working as a clinical editor (CE); providing subject matter expert (SME) review; and contributing to working groups looking at service redesign.

To help improve HealthPathways content, use the 'Send Feedback' button at the bottom right of the page to provide feedback directly to the local HealthPathways team. Feedback might include: suggesting new condition pages; providing clinical resources; reporting

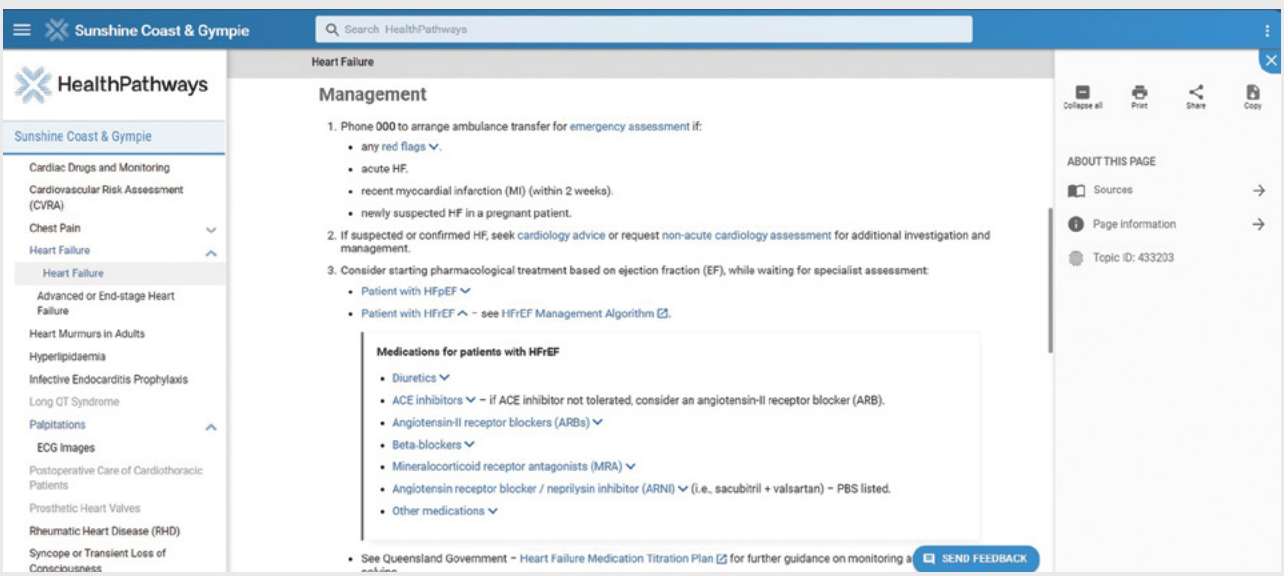

Figure 2. Screenshot of (part of) a HealthPathways management section (Heart Failure). As this is a pathway from the Sunshine Coast in Queensland, it contains the Queensland Government Heart Failure Titration Plan (bottom of screen). Localised pathways also contain region-specific referral information. Use the top search bar to quickly find pages, or alternatively refer to the left-hand table of contents. To expand specific information, click on the blue text or the down arrows (⌄). Access other options in the right top corner, such as expanding or collapsing all information, printing, sharing and copying of information.

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inaccuracies or broken links; and updating service information or referral criteria. Subscription to HealthPathways email updates is available by clicking on the three vertical dots in the top right corner.

Opportunities to write and edit clinical content, as a CE or SME, regularly come up. Contact the local HealthPathways team for more information.

The process of reviewing and publishing hospital specialist service information on HealthPathways is often a catalyst for service redesign.[6,22] In these instances, a formal or informal working group is created with, for example, representation from local GPs, hospital specialists and specialist outpatient administrators. Clinicians involved in these working groups have found the experience valuable, improving relationships between primary and secondary care.[4] Indeed, the successful implementation of HealthPathways requires the engagement of local clinicians.[14]

The authors (EK, JH), both GPs, have been involved in local service redesign, initiated by the need to develop and publish clinical service criteria in HealthPathways. They found developing service criteria led to increased communication between administrators and hospital specialists, as well as between hospital specialist departments. This allowed improvements in outpatient triage and clinic allocation, innovative models of care and clinical phone support for GPs. The authors consider that the involvement of GPs, with their unique overview of local health systems, is key to the success of local service redesign.

Conclusion

Community HealthPathways is a key resource for GPs and can be used to support decision making during consultations. It combines evidence-based guidelines with local service and referral information, collaboratively developed and collated by local primary care and specialist clinicians. Knowledge about the structure of HealthPathways and how to find information is useful for busy clinicians to aid rapid decision support at the point of care. GPs have a key role in local service redesign through involvement in working groups and developing HealthPathways content.

Key points

1. As HealthPathways summarises available evidence and puts it in a local context, it is a useful source of information to support decision making in the consulting room.
2. Keep the HealthPathways website open during consultations for quick reference.
3. Start typing keywords in the top search bar to quickly find page results or suggestions.
4. Condition pages have the following sections: Background, Assessment, Management, Request (referral) and Information, whereas Request pages provide referral options and required referral information.
5. Through the process of collaboration, HealthPathways supports integration of health services and an improved patient journey through the local health system.

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The anatomy of coercive practices in healthcare settings

Robert D Schweitzer, Cherie Sonnenburg, Rohani Mitchell

Background
In healthcare settings, there can be a fine distinction between genuine performance management and vexatious complaints occurring in the context of bullying. The most common manifestation of such behaviour involves repetitive interpersonal abusive behaviours within the context of a power hierarchy. These interactions might well be experienced as bullying behaviour; however, the interpersonal dynamics underpinning such behaviours remains largely unexplored.

Objective
This paper offers a psychological perspective on bullying and harassment and adopts a psychodynamic case study approach, utilising a case vignette involving a senior and junior doctor within a general practice context. Conflict can be mitigated by understanding the intra- and interpersonal dynamics that interfere with rational performance management.

Discussion
Psychological processes such as projection, displacement and projective identification are useful in understanding the genesis of bullying and harassment within demanding workplaces. Reflecting upon the psychological processes underpinning such conflict might help mitigate coercive workplace behaviour.

THE BODY OF LITERATURE addressing bullying and harassment within healthcare services is growing. Issues of prevalence, trends and impacts upon healthcare practitioners are well known.[1] For instance, one in four doctors in the Australian workforce has reported experiencing persistent behaviours that undermine their professional confidence or self-esteem,[2] with 21% of general practitioners (GPs) affected.[3] Bullying includes repeated, unreasonable and systematic behaviours that adversely affect an individual, including harassment, intimidation, degradation and humiliation.[4] Jamieson et al observed a 'tacit tolerance of intimidating and destructive behaviours' affecting some 60% of medical trainees.[1] The detrimental impacts of bullying and harassment on healthcare professionals are significant, leading to shame, anxiety, depression and even a move away from medicine.[5] Furthermore, a lack of psychological safety for healthcare professionals might hinder quality improvement efforts and jeopardise patient safety.[6]

The characteristics inherent to healthcare settings might amplify interpersonal dynamics and intrapsychic processes that predict conflict, bullying and harassment. These settings are characterised by high-stakes decision making, alongside an entrenched culture emphasising hierarchy, self-sacrifice, resilience and deference.[6,7] Within this context, medical doctors identify 'fear of making mistakes' and 'making the right decisions' as significant sources of work-related stress.[8] These stressors might be both initiated and amplified by difficult relations with senior colleagues.[8] Senior medical staff have been consistently identified as the most common source of bullying, harassment, discrimination and/or racism targeting doctors.[1,2,9,10] In healthcare settings, the responsibility and strong desire to 'protect the public' from harm might serve as justification for dominating, hierarchical behaviours, which might or might not be deemed appropriate.

This paper aims to better understand harassment and bullying through a case study (Box 1), drawing upon contemporary psychodynamic explanatory constructs within a healthcare setting.

Methods

Design

The paper draws upon familiar behaviours within healthcare settings to present a vignette. The vignette describes the dynamics of common occurrences of conflict within such settings and the opportunity for analysis of behaviour. The paper refers to key psychodynamic concepts such as power hierarchy, projection and blaming behaviour, displacement and projective identification. These concepts serve as fundamental explanatory constructs for understanding the psychological mechanisms underpinning bullying and harassment behaviours,

particularly in high-pressure contexts where irrational forces might be at play.

The power hierarchy refers to the unequal distribution of authority, decision making and influence within a setting. Relationship dynamics are fundamentally affected by power differentials and social positionality, and an individual's level of power might impact the way they unconsciously manage anxieties in different situations. Projection is commonly understood as an ego defence mechanism in which individuals unconsciously attribute their own undesirable thoughts, feelings or traits onto others. It allows them to avoid acknowledging or dealing with these aspects of themselves, often leading to misinterpretations and conflicts in interpersonal relationships.

In the workplace, attributing blame or projecting blame onto others might serve to shield one's ego, solidify positions within a power hierarchy and defend against anxiety. In contrast to constructive performance management, which aims to foster growth and development in employees, being subjected to blame can trigger defences in the recipient, potentially paving the way for interpersonal conflict and undermining morale and performance. Displacement refers to a process that involves redirecting emotions or impulses from their original target to a less threatening alternative. Similar to projection, displacement serves to manage uncomfortable feelings, but can lead to misunderstandings in relationships if not addressed.

Projective identification is more complex and involves a person unconsciously projecting their own undesirable qualities onto others and then influencing them to adopt and express these qualities. It is a complex defence mechanism that can influence interpersonal dynamics and might serve as a way to manage internal conflicts or exert control over the person's environment.

Box 1. Vignette[A]

A senior general practitioner (GP) in a group practice had taken to closely monitoring the practice of a junior registrar, offering frequent, unsolicited commentary. Besides overseeing the registrar's client numbers, cancellations, appointment lengths and interventions, the senior GP had begun intermittently reviewing incoming correspondence and test results. Additionally, they sought regular performance reports from practice nurses and other administrative staff regarding the registrar's work. In this instance, the senior GP was towards the end of their career and dealing with a number of issues relating to their giving up a role that had been important in their sense of esteem. Despite meeting the minimum required professional development, they found themselves exposed to frequent revisions of theories of disease aetiology and rapid developments in practice and technology. They were also observing the retirement of many colleagues from their generation and found it challenging to connect with emerging practitioners.

A recent case handled by the junior registrar was selected for review during a general practice peer review meeting. The senior GP observed substantial deviations from their usual practice in a procedure carried out by the registrar. Despite the registrar citing recent research and practice guidelines, the senior GP questioned their approach, critiquing multiple aspects of the procedure in a dismissive, condescending and somewhat denigrating manner. When the registrar expressed dissatisfaction with the delivery of the senior GP's comments and sought constructive dialogue with the other GPs present, the senior GP became enraged and adopted an intimidatory stance, threatening to escalate the matter. Following a heated exchange, the senior GP indicated their intention to escalate the incident for investigation, alleging incompetence.

Subsequently, the senior GP initiated a private meeting with the practice manager responsible for handling complaints. The senior GP was a partner in the practice and the practice manager was therefore the senior GP's employee, with whom they had enjoyed a long-standing working relationship. The senior GP reportedly referred to the registrar in disparaging terms and complained about multiple aspects of the junior registrar's performance. The senior GP suggested termination of the registrar's employment and hinted at reporting them to the Australian Health Practitioner Regulation Agency (AHPRA). Without fully understanding the professional and interpersonal dynamics at play, or seeking information from the registrar, the practice manager promptly ordered a review of the registrar. The registrar was subsequently instructed to immediately cease performing the routine procedure without explanation. A formal investigation found no deficits in the registrar's practice and the senior GP's claims of incompetence to be unsubstantiated. However, no apology was offered, and the practice manager continued to oversee a 12-month performance management process. During this time, the registrar reported being threatened, humiliated and undermined by both the senior GP and the practice manager. Subsequently, the registrar left the practice and was denied a professional reference.

[A]Although this vignette is an adaptation of a real case example, details have been omitted and changed for anonymity and speculative details added to demonstrate the common psychological concepts that might underpin cases of bullying and harassment in the medical setting. It is noted that the dynamics described might be applicable to multiple medical disciplines and contexts.

Reflexive comment

Each author comes from a background in psychology and shares an interest in healthcare settings. The first author, a cisgender man, academic and clinical psychologist, has taught clinical psychology students for over two decades and has extensive experience as a psychodynamic teacher and practitioner. Additionally, he has chaired Australian Health Practitioner Regulation Agency (AHPRA) State Boards and dealt with complaints arising within healthcare settings for almost a decade.

The second author is a cisgender woman and general psychologist with a Master of Clinical Psychology, who is currently undertaking further training in psychodynamic therapy as part of the Clinical Registrar program. Her previous work included almost a decade spent in Human Resources, addressing incidents of workplace conflict and bullying.

The third author is a postgraduate trainee and Provisional Psychologist practising from a psychodynamic and experiential orientation. She is a cisgender woman of Anglo-Celtic and New Zealand Māori (Ngāi Tahu) ancestry, born and raised in Australia. In her previous work in the social services sector, she has engaged with issues of power, conflict and collaboration, including involvement in family violence support, mental health advocacy and community engagement.

A specific vignette

Although the dynamics of complaints and workplace conflicts in the workplace vary depending on the situation and the

organisational culture, the following example sheds light on the underlying dynamics involving a senior general practitioner (GP) (Box 1). The example is used to gain a better understanding of conflict, excessive criticism and undermining approaches to resolution.

Results and discussion

Interpretation

Implicit intrapersonal factors

There are several psychological theories that might provide insight into coercive and bullying behaviours that occur within healthcare settings. Inquiry into these underlying processes can be particularly useful where a person's actions seem disproportionate or entirely irrational to the situation at hand. In considering the actions of the supervising senior GP in the case example, it is useful to explore the role of the psychological processes referred to as *projection* and *displacement*.

Projection might have involved the senior GP's unwitting ejection of their own unacknowledged sense of incompetence, and projection of this incompetence onto the registrar. This sense of incompetence was likely activated in the senior GP during the debate about latest practice recommendations. This is likely to have left the senior GP with unconscious, unprocessed anxieties about their current practices, and an increasing sense of professional obsolescence, compounded in the context of a range of rapid developments in the field. Through the process of *projection*, the senior GP disavows and ejects any anxiety from their conscious awareness. The senior GP therefore failed to reflect on their internal processes that might be influencing their responses to the interpersonal process, genuinely believing that the incompetence belonged to the registrar. Similarly, the senior GP might have redirected (*or displaced*) their negative emotions influenced by factors arising within their personal life and impending retirement, which are experienced as unsafe, and instead directed them towards a 'safer' receiver, the less-threatening younger registrar. The level of hostility, which was not commensurate with the situation, might reflect an array of complex feelings and conflicts associated with their potential transition away from a professional career, which had been a core part of their identity.

Ageing and identity

Writing from the stance of psychotherapists supervising junior colleagues, Yerushalmi suggests older supervisors' self-esteem can be threatened by encounters with young supervisees, facilitated in part by the losses experienced with ageing.[11] He describes the psychological difficulties in terms of a need to be idealised by younger colleagues, with problems integrating the consequential polarities in their self-image when this does not occur. This process might be particularly salient where traits of narcissism are involved, a commonly identified element in organisational bullying.[12]

Applying this observation to the current case, the senior GP might have experienced feelings of humiliation arising from the perceived challenge to their authority and dismissal of their advice by a younger colleague during the case review meeting. This humiliation might have been amplified by the senior GP's awareness of their professional limitations as they confront their retirement and begin to experience rivalry – a perceived obstruction to their acceptance and esteem – from upcoming younger practitioners. Their initial reaction to the registrar, who was the target of their complaint, might constitute an expression of 'narcissistic rage', which might involve feelings of envy in response to the perceived reputation and acceptance of the colleague.[13]

In narcissism, rivalry might be experienced as a severe threat or injury to one's self-esteem. The sense of threat might elicit intense anger towards others who fail to comply with their need to be idealised. The threat might also elicit a desire to devalue non-complying others, to reduce the threat through both internal processes and external actions. The target individual is subsequently used to embody the narcissistic individual's emergent unwanted feelings of incompetence through the process of *projection*, as described above.[14]

Implicit organisational and interpersonal factors

Those in positions of leadership who are involved in overseeing performance management might equally be unknowingly engaged in implicit processes that emerge in the workplace. Scholarship suggests the health milieu engenders anxiety.[15] Anxiety might be understood in terms of the German expression, *angst*, referring to unfocussed feelings of profound anxiety or dread. Medical environments might engender such feelings due to chaos, under-resourcing, the pressures of general practice, and exposure to human pain and sometimes misery, in a context where outcomes can be critical. The ways in which we defend against anxiety includes the use of psychological processes including *denial*, *projection* and *collusion*. These phenomena emerge within both general practice and institutions such as hospitals as a way of defending against the anxieties permeating the workplace.[16] As such, health environments can cultivate 'tribal' and collusive organisational dynamics, where members of the same rank or discipline engage in tacit agreements regarding decision-making processes or perceptions of others to meet or avoid shared needs or anxieties respectively.

How do we understand the practice manager taking actions without first seeking any input from the registrar being complained about? Similarly, how do we understand a decision to implement performance management processes and not attend to bullying behaviours following an investigation where no deficits had been identified? Through an understanding of dynamics involving the organisational culture, it might be suggested that the practice manager unknowingly 'colluded' with the senior GP's perceptions and emotions in order to meet their shared needs of self-esteem preservation and connection, and to prevent shared anxieties around patient safety, exclusion or loss of power within the hierarchy.

On an interpersonal dynamic level, one way to understand this process is by drawing upon a concept well known in the psychotherapy literature, called *projective identification*. *Projective identification* refers to a dynamic phenomenon whereby one person places their unwanted feelings, perceptions or parts of the self upon another person (*projection*), with the receiver of those projections unwittingly taking them on board and behaving accordingly (*identification*). For example, the practice manager might have automatically identified with the feelings of the senior GP (complainant), the depth of emotion associated with their experience, and their projected expectation that the

practice manager act, without conscious regard or consideration for the registrar who was the target of the senior GP's anger. In this way, the practice manager identifies with the *projection*; that is, they become what the senior GP projects onto them and expects of them, albeit unconsciously. *Projective identification* likely resulted in the bypassing of the mental processes necessary for procedural fairness, including consideration and reflection upon multiple sources of information, and unbiased decision making independent of the senior GP influence. In contrast, a performance management approach would focus upon a goal-directed process in which risk would be identified from the available evidence, and actual deficits and areas for remediation would form the basis for any action.

Conclusions

Implications for practice

Doctors in leadership positions have a duty to promote patient safety, supervise constructively and oversee complaints processes with sensitivity. Medical settings are improving responses to unhelpful behaviours to ensure client safety (see for example,[17–19]); however, further work is needed to ensure worker safety, particularly the safety of less senior workers. Healthcare settings impose significant demands upon professionals involved in the provision of care. Where senior colleagues perpetrate bullying and harassment, it can be difficult to delineate problematic behaviours from the legitimate processes of direct line management and performance management. Understanding the intrapsychic dynamics in the context of power hierarchies that influence workplace bullying, conflicts and complaints might help avert harmful outcomes.

Intrapsychic dynamics, by nature, frequently operate at the unconscious or semi-conscious level, and therefore are not always immediately apparent to parties involved, or amenable to intervention. To prevent and respond to coercive management practices, it is therefore imperative that self-reflection is embedded in management processes, and that remedial action is taken at a systemic, interpersonal and individual level.

Although existing workplace bullying interventions exist (for reviews, see[10,20–22]), these have frequently focussed on broader organisational activities, such as policy change and awareness raising; interventions targeting bystanders or victims, such as assertiveness intervention training, or victim support; or instruction on appropriate or civil behaviour and communication. Few interventions have directly targeted perpetrators' potential internal motivations or mental processes (see for example,[23]). We assert the need to make intra- and interpersonal processes overt, to mitigate conflict and facilitate procedural fairness. Interventions at the individual level might include minimum training for healthcare leaders and managers in reflective practices. Adopting a psychodynamic understanding, such reflection could focus on the dynamics of power and coercion in hierarchical management structures; how this intersects with the operation of power in the broader social context of race, gender, class and more, particularly around who bullies compared to who is bullied, and how those in powerful positions might be unintentionally influenced to manage their unconscious anxiety through power over and projection onto others less powerful than themselves. Such training could develop managers' capacity to reflect on and identify current and future mental processes that might put them at risk of unintentionally adopting extreme or coercive management practices. Such risk factors might theoretically include feelings of loss of control, threats to status or feelings of inadequacy at work, precipitated by circumstances such as prolonged workplace pressure, personal stress or professional or personal life changes.

It is further imperative that others adjacent to managers are not unconsciously or consciously co-opted into coercive, bullying dynamics. Cultural interventions to prevent such behaviours might include manager-level peer supervision meetings that promote non-judgemental reflection on leadership pressures, management practices and debriefing. Other structural changes might include mandatory review of all newly initiated performance management or internal complaints processes, by a manager of equivalent status and adequate distance and impartiality. Such a review might include independent assessment of the objective evidence for poor performance of junior staff, and consideration for any interpersonal dynamics and the intrapsychic reflective capacity of the manager – before more formal performance management processes are imposed.

Where coercive, bullying behaviours have occurred, there remains an imperative to address this injustice through disciplinary action, as well as restorative justice processes to (at least partially) repair the intrapsychic wounds inflicted on victims. Such processes should further promote reflective practice in perpetrators to prevent reoffending. In certain circumstances, those culpable of frequent re-perpetration, and resistant to self-reflection, might need to be removed from the workplace, for the safety of both staff and patients, and the overall health of the workplace culture.

To return to the challenge posed by Jamieson et al,[1] will the practice of medicine remain complicit in bullying culture or champion a new era of workplace civility within healthcare settings?

Key points

The following key principles provide a basis for understanding the psychological mechanisms that might underpin coercive and bullying behaviour in healthcare settings.

- Healthcare settings involve high-stakes decision making, which can foster a culture of blame and lead to coercive and controlling behaviours.
- This paper highlights how blaming others, especially those in subordinate positions, can function as a defence mechanism against stress and uncertainty in chaotic healthcare environments.
- Key mechanisms include blame, displacement and projection, whereby junior staff members might be subjected to coercive behaviours irrespective of their role.
- Psychological theories underpinning coercive and bullying behaviours emphasise how individuals, such as the senior GP in the presented vignette, might unconsciously project anxieties onto others, thereby fuelling workplace conflicts.
- Recognising the dynamics involved in conflicts within healthcare settings might contribute to a more rational appraisal of key issues.

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