check

Independent learning program for GPs

Unit 574
August 2020

Skin conditions

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Skin conditions

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The five domains of general practice

- Communication skills and the patient–doctor relationship
- Applied professional knowledge and skills
- Population health and the context of general practice
- Professional and ethical role
- Organisational and legal dimensions

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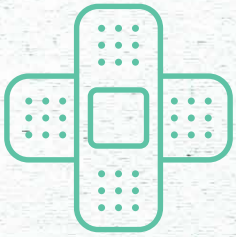

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About this activity

Skin conditions, including pathology affecting the nails and hair, account for 15.3 of every 100 patient encounters in general practice in Australia, and 11.3% of the total reasons for encounters.[1]

Contact dermatitis was the most common skin-related presentation to general practice in 2015–16, accounting for 1.1% of total reasons for encounters.[1]

Tinea is common[2] but, in the case of tinea incognita, may present with an 'atypical' appearance.[3] It is therefore important that general practitioners (GPs) are alert to cases of tinea that have been previously incorrectly diagnosed and treated with a topical corticosteroid[3] so that this condition can be treated correctly and potential spread limited.

The lifetime risk of developing alopecia areata is approximately 2%, and onset generally occurs before the age of 40 years.[4] The prevalence is the same in men and women.[4]

The incidence of herpes zoster (shingles) increases with age. Approximately 630 individuals per 100,000 in the 50–59-year age range are affected, compared with 1531 per 100,000 people aged 70–79 years.[5]

Correct identification of lichen sclerosus is crucial as, although the condition is uncommon, progressive scarring can occur without treatment; in a small number of cases, untreated disease has also progressed to malignancy.[6]

This edition of *check* considers the investigation and management of skin conditions in general practice.

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Learning outcomes

At the end of this activity, participants will be able to:

- discuss the process of differentiating between irritant and allergic contact dermatitis
- outline the Australian Immunisation Handbook recommendations for vaccination for herpes zoster
- describe the diagnostic criteria used to determine the cause of hair loss
- identify the differential diagnosis for a poorly demarcated erythematous rash
- outline the optimal management of penile lichen sclerosus.

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Abbreviations

ACD	allergic contact dermatitis
GLS	genital lichen sclerosus
GP	general practitioner
HSV	herpes simplex virus
HZ	herpes zoster
HZ/su	herpes zoster subunit
HZO	herpes zoster ophthalmicus
ICD	irritant contact dermatitis
Ig	immunoglobulin
PCR	polymerase chain reaction
PHN	post-herpetic neuralgia
PLS	penile lichen sclerosus
RCT	randomised controlled trials
SCC	squamous cell carcinoma
STI	sexually transmissible infection
TCS	topical corticosteroid
VZV	varicella zoster virus

CASE

1 Jessica has itchy hands

Jessica, aged 28 years, presents with a red, dry, scaly rash on both hands associated with itch and a burning sensation (Figure 1). The rash has been present for a few months and is worsening despite Jessica's use of emollients. She does not have any active medical conditions and has been working full time as a hairdresser for the past six months.

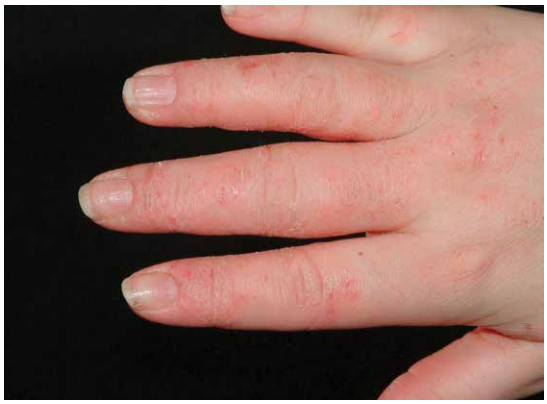

Figure 1. Dorsum of Jessica's right hand, which shows redness, scaling, cracking and dryness

Question 1

What potential exposures or other relevant history would you seek from Jessica?

Question 2

What conditions would you include in your differential diagnosis for Jessica?

Further information

Jessica tells you that she frequently washes her hands at work; more than a dozen times per day. She also comes in contact with hair dyes, bleaches, perming chemicals and shampoos every day. She usually wears rubber gloves when she handles dyes and bleach. She uses cosmetics occasionally, but has never developed a rash on her face or other parts of the body. She does not have any significant contact with animals or plants. She reports a history of childhood eczema only and occasional bouts of hay fever in spring.

Question 3

What is the most likely diagnosis based on Jessica's history?

Question 4

What investigations would you consider for Jessica?

Question 5

What are some common occupations at risk of significant work-related dermatitis?

Further information

You take skin scrapings from Jessica and send the sample for microscopy and fungal testing. After weeks of culture, no fungus has grown. You refer Jessica to a patch testing clinic. The results of the pertinent patch test are shown in Table 1.

Table 1. Results of Jessica's patch test

Allergen	Presence of reaction after five days
Nickel sulfate	No reaction
Thiuram mix	No reaction
Methylisothiazolinone	Minimal erythema
Formaldehyde	No reaction
Balsam of Peru	No reaction
Paraphenylenediamine base	Minimal erythema
Toluene diamine sulphate	No reaction
Ammonium persulfate	Minimal erythema
Glyceryl monothioglycolate	No reaction

Question 6

Based on the results of the patch testing, what is your diagnosis for Jessica?

Question 7

What would you suggest as a management plan for Jessica's hand dermatitis?

Question 8

Would you refer Jessica to a skin specialist? If so, when?

CASE 1 Answers

Answer 1

A basic screen of questions should include the below, but not be limited to:

- occupational details (current and previous, duration of employment, routine tasks, exposures [eg hair dye, hairdressing bleach, shampoos])
- exposure to water and frequency of wet work
- skin care (eg liquid soaps, fragrances, moisturisers, cosmetics)
- glove use (work and home), as well as type of glove (eg latex/rubber, polyvinyl chloride or nitrile)
- hobbies – use of any glues or chemicals, plants
- pets
- history of atopic eczema, asthma and hay fever, including family history
- treatment used and effectiveness (eg over-the-counter, prescribed, from friends)
- any improvement when not at work (eg on holiday or annual leave).

Answer 2

Conditions to include in the differential diagnosis are:

- irritant contact dermatitis
- allergic contact dermatitis
- atopic dermatitis
- vesicular hand dermatitis
- fungal infection
- contact urticaria.

Answer 3

The most likely diagnosis is contact dermatitis (irritant or allergic).

Contact dermatitis is classified as irritant or allergic depending on the underlying precipitant and mechanism of injury. Irritant contact dermatitis (ICD) is a direct cutaneous response to the physical or toxic effects of external agents in the environment or workplace. ICD accounts for approximately 80% of cases of contact dermatitis.[1]

Allergic contact dermatitis (ACD) results from an activation of antigen-specific acquired immunity leading to T cell–mediated skin inflammation.[2] Antigens are usually non-protein chemicals, termed haptens, and sensitisation occurs via topical application and usually takes between 10 days and three weeks. However, sensitisation may not occur following the first exposure and sometimes does not occur for years. Clinical symptoms after sensitisation are often delayed, appearing 48–72 hours after exposure to an allergen. Symptoms may arise earlier with each subsequent exposure, sometimes within hours.[2]

In some instances, the distribution of skin inflammation can also assist with the diagnosis. For example, the most common sites for nickel dermatitis are the wrists (from watch straps), lower abdomen (from jean studs) and ear lobes (from earrings).[3]

Clinically, it is often difficult to distinguish between ACD and ICD. Furthermore, most people with work-related contact dermatitis are exposed to a variety of agents that can cause both irritant and/or allergic reactions. Existing ICD or breaches of the skin barrier also increase the likelihood of complicating ACD. As a result, hand dermatitis is often multifactorial.

Fungal skin infections are an important differential, as they may mimic hand dermatitis. Clinical features that favour fungal hand infection (tinea manuum) over contact dermatitis include asymmetrical involvement (one hand is usually affected; if both hands are affected, involvement is asymmetrical), presence of tinea pedis ('one hand, two foot' syndrome), involvement of both dorsum and palm, and elevated borders of the rash.[4] Nevertheless, as a result of similar predisposing factors (eg gloves, wet work), tinea manuum may coexist with or complicate hand dermatitis.

Answer 4

Skin scrapings

Scrapings from the rash can be sent for microscopy and fungal culture. Microscopy may immediately identify hyphae, but a result for the culture may take weeks.

Referral for patch testing

Patch testing is indicated if ACD is suspected, and referral to a patch testing dermatology clinic is required. The basis of the test involves eliciting an immune response by challenging an already-sensitised individual with standardised amounts of allergens prepared on adhesive patches.[5] The tests usually include a baseline series of allergens that frequently cause ACD (this can vary between countries and patch testing centres), as well as additional allergens on the basis of the individual's daily and occupational exposures. The site of patch application (usually the back) is marked to guide interpretation later. After application, the patches are removed and interpreted after 48 hours and then again after 4–5 days. Positive reactions are usually erythematous and infiltrated, often with vesicles or bullae. Difficulty arises in interpreting weakly positive reactions, as these can be confused with irritant reactions. Fading from the initial to the second reading may be suggestive of an irritant reaction (ie the 'crescendo–decrescendo' phenomenon).[5]

Other (less relevant) tests

Allergen-specific IgE testing

Laboratory testing of serum immunoglobulin (Ig) E levels against certain allergens depends on the testing panel, and may include foods, insects, plants, latex, mould, dust mites, etc. IgE testing is generally of limited utility and is not a diagnostic test by itself; however, it may support a clinical diagnosis if there is a suggestive clinical history (eg suspected contact urticaria to latex).

Skin prick testing

Skin prick testing involves depositing allergens into the skin of the forearm with a sterile lancet. Skin reactions are observed after 15–30 minutes and compared against positive and negative controls. Skin prick testing's main utility lies in the assessment of immediate hypersensitivity reactions, and a referral to an immunologist or allergist is usually required. Immediate reactions may occur to ammonium persulphate (hairdressing bleach), and, not uncommonly, hairdressers may present with immediate symptoms including rhinitis or asthma.

Answer 5

People working in the following occupations may be at risk of significant work-related dermatitis:[6]

- healthcare workers
- metal workers
- concreters/bricklayers
- food handlers
- machine operators
- hairdressers
- mechanics
- printers
- florists.

Answer 6

Jessica's symptoms and patch testing results support a diagnosis of ICD. As a result of her occupation, Jessica is exposed to several irritants at work including wet work, hot water, shampoos and conditioners, and sweating from occlusive gloves.

A diagnosis of ICD requires exclusion of other cutaneous disorders, especially ACD. Although several chemicals in Jessica's patch testing series elicited very mild reactions, these were not consistent with true hypersensitivity

reactions. Experience is required to interpret patch testing results. Based on Jessica's history of frequent hand washing, negative skin scraping results and negative patch testing results, ICD is the most likely diagnosis. There is no test available for ICD, and it is often a default diagnosis.

Notably, there is a much higher prevalence of ACD among hairdressers when compared with other professions.[7] This is probably a result of the large number of allergens found in hairdressing chemicals, especially those found in hair dyes (toluene diamine sulphate, paraphenylenediamine), bleach (ammonium persulfate), perming solutions (glyceryl monothioglycolate) and rubber chemicals in gloves.[7,8] Causes of ICD in hairdressers include frequent hand washing with hot water, friction from handling damp hair, use of occlusive gloves, shampoos and conditioners, and contact with irritating hair chemicals.

Skin conditions are among the most common occupational diseases and an important reason for workers' compensation claims.[8] Contact dermatitis accounts for 90% of all occupational dermatoses.[1] Non-occupational contact dermatitis is important to consider during the work-up, especially if a work-related precipitant is not apparent. Furthermore, if a non–work related exposure is responsible, this may have a significant implication for a worker's compensation claim.

Answer 7

The management of contact dermatitis is based on the principles of avoidance, protection, substitution and treatment of dermatitis.[9,10]

Avoidance

The cornerstone of managing contact dermatitis is based on identification of the irritant or allergens followed by avoidance. Complete avoidance of water during work may be impractical as a hairdresser. A change in occupation may result in a more favourable long-term prognosis and should be considered if Jessica's condition is refractory to treatment.

Protection

If precipitant avoidance is not possible, reduction of contact is advised (eg through use of personal protective equipment). In Jessica's case, wearing gloves for all water work and chemical handling would be advised. Glove type should be selected on the basis of chemical exposure according to the workplace's materials safety data sheet. For basic household tasks, rubber or polyvinyl chloride gloves (ideally with a cotton liner or over cotton gloves) should still be worn.

Barrier creams may also be an effective option in preventing occupational dermatitis by reinforcing the skin barrier and reducing transepidermal water loss.[9]

Substitution

Replacing other potential allergens or irritants is advised, even if these are not the cause of the contact dermatitis (eg using fragrance-free skin care products).

Treatment

Regular application of emollients, especially after finishing work, is the key to preventing dermatitis, alleviating symptoms of dermatitis and promoting skin barrier recovery.[9]

Treatment of contact dermatitis often necessitates prescription of topical corticosteroids or calcineurin inhibitors. Treatment regimens are generally similar to that of atopic dermatitis.[9]

If contact dermatitis is severe or acute, a short course of oral corticosteroids (25–50 mg daily for up to one week, then tapered over two weeks) may be useful.[10]

Answer 8

Referral to a patch testing dermatologist or clinic should be considered if ACD is likely (especially occupational). ACD is often suspected when an eczematous disorder persists or fails to respond to standard therapies and there is a possible trigger for the rash. Topical or oral corticosteroids should be weaned prior to appointments to avoid interference with patch testing results. Furthermore, patients should be encouraged to bring in all cosmetics, fragrances, jewellery and chemicals with which they are in regular contact.

Referral to a dermatologist should also be considered for severe or recalcitrant contact dermatitis despite precipitant avoidance/protection and basic management with emollients and topical steroids or tacrolimus. Patients may require second-line treatment options such as systemic immunosuppression or ultraviolet therapy.[9]

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CASE

2 Apinya thinks she is going bald

Apinya, aged 27 years, has come to see you concerned about patches of hair loss on her scalp. She first noticed the patches three weeks ago and thinks she had a similar episode with just one small patch eight months earlier, which resolved on its own. She is self-conscious about the hair loss and worried that she might lose all her hair and have to wear a wig like her aunt.

Question 1

What would you ask Apinya to narrow your differential diagnosis for this presentation?

Question 2

What physical assessment would you undertake?

Further information

Apinya tells you she is otherwise well and has regular periods. Her only regular medication is a salbutamol inhaler, which she uses as needed for well-controlled asthma. She recalls having mild eczema as a child. Apinya admits to being more stressed lately because she has started a new job. She tells you her father has been bald for many years, while her maternal aunt lost all her body hair many years ago.

On examination, you see three well-defined round/oval patches of hair loss on the scalp. Using a dermatoscope, you see small exclamation mark hairs towards the periphery of the area of hair loss. A hair pull test is negative. Apinya's eyebrows and eyelashes appear normal, and she reports no change in other body hair. Three of her fingernails reveal small pits. The remaining physical examination is unremarkable.

Question 3

What diagnosis do you suspect, given the results of the history and examination?

Question 4

What, if any, investigations would you request for Apinya? What would you expect to find?

Further information

From the history and examination findings, you diagnose alopecia areata. Apinya asks you what, and how common, alopecia areata is.

Question 5

How would you answer Apinya?

Question 6

What treatment options can you offer Apinya?

Further information

After a discussion of the treatment options, you administer intralesional triamcinolone 5% with lignocaine 1% to Apinya's scalp patches. She tolerates the procedure well.

Apinya comes back to see you and asks if she is likely to have further episodes of hair loss in the future.

Question 7

How would you respond?

Further information

You explain to Apinya her unpredictable long-term prognosis and arrange to see her again every 4–6 weeks to repeat the intralesional steroid injections. If she does not show improvement after six months, you plan to commence systemic treatment and refer her to a dermatologist.

Question 8

What else is important for Apinya's management and for any patient presenting with hair loss?

CASE 2 Answers

Answer 1

Key aspects to consider on history-taking include the duration, onset and pattern (diffuse or patchy) of hair loss as well as experiences of hair thinning (ask about changes in ponytail thickness) and hair shedding (ask about hair on the brush or that comes out when washing). This last symptom can be quantified using the Sinclair hair shedding scale, which allows comparison between visits and assessment of treatment response over time.[1] Additionally, it is important to ask Apinya about details of previous episodes of hair loss and regrowth, loss of other body hair and any associated pain, itchiness or burning of the scalp.[2] Some patients may identify a trigger for the hair loss, such as recent stress, changes in medications, illness or travel.[3,4] Finally, history-taking should include questions about haircare practices such as tight ponytails, use of hair pieces, hair products and curlers.[2] A general past medical history and family history is also essential, with special attention to autoimmune diseases as well as symptoms of thyroid dysfunction or anaemia. Women should be screened for symptoms of androgen excess (eg irregular menses, acne and hirsutism).

Table 1 outlines how to group conditions considered in the differential diagnosis and potential clues on history.

Answer 2

Good lighting and positioning are important for examination of the hair and scalp. It is necessary to determine the pattern of hair loss (diffuse thinning or localised loss) and the extent of scalp involvement. A dermatoscope should be used to examine the area(s) of hair loss and the underlying scalp skin; in particular, it is important to look for perifollicular scale or erythema and loss of hair follicles (suggestive of scarring alopecia), exclamation mark hairs (indicating active alopecia areata), areas of hair regrowth (seen in telogen effluvium), broken hairs (seen in tinea capitis and trichotillomania), comma hairs (seen in tinea capitis) and hairs of different lengths (seen in trichotillomania).[2,5]

A positive hair pull test, in which at least 5–6 hairs are removed when 50–60 hairs are pinched and pulled firmly but gently away from the scalp, is consistent with telogen effluvium, active alopecia areata and thinning areas of androgenic alopecia.[2,6] However, this test is difficult to standardise, and its sensitivity is poor; as a result, negative tests cannot be used to reliably exclude diagnoses.[6]

For many people with skin and hair disorders, including more than one-third of patients with alopecia areata, involvement of the nails can occur. Therefore, examination of fingernails and toenails may reveal additional clues to the diagnosis.[7]

A routine physical examination with attention to growth and distribution of other body hair is also necessary.

Table 1. Classic signs and symptoms from a patient's history that are consistent with different aetiologies of hair loss

Aetiology	Signs and symptoms on history
Non-scarring hair loss	
Male or female pattern hair loss (androgenic alopecia)	• Diffuse hair thinning • Gradual onset • May have family history of same
Alopecia areata	• Typically aged <40 years • Abrupt onset • May have personal or family history of autoimmune disease or atopy
Telogen effluvium	• Abrupt onset • Diffuse hair thinning • May be secondary to iron deficiency, thyroid dysfunction or postpartum
Tinea capitis	• Usually occurs in children • Gradual or abrupt onset • Localised hair loss • History of contact with animals or travel
Trichotillomania	• Typically occurs in children and adolescents • Gradual or abrupt onset • Patient may report that hair pulling relieves an inner tension • May have associated psychiatric disorders
Traction alopecia	• History of wearing hair in tight braids or ponytails
Scarring hair loss (various causes)	• Typically gradual onset • Associated with pruritic, burning and/or painful scalp • No hair regrowth

Answer 3

Patchy hair loss in a young female with atopy is suggestive of alopecia areata. This can be confirmed by the characteristic finding of exclamation mark hairs on dermoscopy.[8] Apinya's history and physical examination are consistent with alopecia areata.

Answer 4

Alopecia areata is a clinical diagnosis. Further investigations are not indicated at this stage. If there is uncertainty, or a scarring alopecia is suspected, then a scalp biopsy may be indicated. In these cases, it is important to biopsy an area of active disease (with persistent hair fibres) that is ideally also cosmetically inconspicuous.[9] Histologically, alopecia areata is characterised by a lymphocytic (T cell) infiltrate in and around the anagen hair bulb or the lower part of the hair follicle.[10]

If tinea capitis is considered as a differential diagnosis, scalp scrapings for microscopy and culture are required.

Alopecia areata is known to be associated with other organ-specific autoimmune disorders such as Grave's disease, vitiligo and type 1 diabetes.[11,12] Despite these associations, there is insufficient evidence to recommend routine screening for autoimmune disease at the time of alopecia areata diagnosis.

Routine full blood examination and screening for infectious diseases would be necessary prior to the initiation of systemic immunosuppressive therapy.

Answer 5

Alopecia areata is an immune-mediated disorder that classically presents with one or more discreet patches of non-scarring scalp hair loss.[13] These patches are asymptomatic but may progress to involve the whole scalp (alopecia totalis) or all body hair (alopecia universalis). The lifetime incidence of alopecia areata is approximately 2%.[14] It typically affects people aged <40 years, although there are exceptions, and there is no predilection for any specific ethnicity or sex.[12,14]

Answer 6

There is no cure for alopecia areata and no known method for preventing future relapses; however, treatment options are available that aim to arrest disease progression and reverse hair loss. In 2019, an Australian expert consensus statement included an easy-to-use alopecia areata treatment algorithm. Treatment options include conservative management, topical therapy (with corticosteroids, minoxidil or immunotherapy), intralesional corticosteroids and systemic therapies, including corticosteroids and steroid-sparing agents.[15] However, none of the systemic therapies included in the expert consensus statement are approved for alopecia areata by the Therapeutic Goods Administration. The consensus statement recommends initiation of such medication only by experienced dermatologists.[15]

For hair loss that is limited and has a recent onset, the Therapeutic Guidelines recommend 3–4 months of topical corticosteroids as first-line therapy.[16] However, for more severe cases of alopecia areata, an initial trial of topical corticosteroids has been shown to lack efficacy and delay patient referral.[17] In cases such as Apinya's, in which multiple alopecia areata patches are present, the consensus algorithm recommends the use of intralesional corticosteroids administered every 4–6 weeks as first-line therapy, with the potential for topical or systemic immunotherapy if there is no significant response within six months.[16] Intralesional injections can be administered in the general practice setting if the practitioner feels comfortable doing so; otherwise, patients can be referred to a dermatologist for treatment.

While waiting for hair to regrow or as part of a conservative management strategy, there are various cosmetic solutions that can be offered to Apinya including colour-matched wool fibres to conceal the scalp, hair pieces, wigs or hair extensions.[16]

Answer 7

The natural course and treatment response of alopecia areata are unpredictable. Approximately 40% of patients experience full regrowth of a solitary patch of alopecia areata within six

months, while 27% develop additional patches.[18] Many people who develop additional patches still achieve persistent remission at 12 months.[18] Of the patients whose alopecia areata follows a chronic relapsing–remitting course that persists beyond 12 months, 30% ultimately progress to alopecia totalis and 15% to alopecia universalis.[18] Poor prognostic factors include extensive hair loss (>50%), ophiasis pattern, associated nail changes, early age of onset (before six years of age), a positive family history and concomitant atopy or autoimmune disease.[12,19]

In Apinya's case, her initial history indicates that she has had a prior episode of alopecia areata in the past 12 months. Her associated nail changes, positive family history (aunt wears a wig) and history of atopic disease (asthma, eczema) are all poor prognostic factors. She may go on to have a chronic disease course and develop additional areas of hair loss that are persistent, and she may never achieve complete remission.[15]

Answer 8

Alopecia areata has a significant, often underappreciated, psychological impact on patients and their families. Patients experience an increased lifetime prevalence of psychiatric disorders, especially mood and anxiety disorders.[20,21] Similar to patients with other chronic relapsing skin disorders such as psoriasis, patients with alopecia areata consistently report poor health-related quality of life.[22] Therefore, it is recommended to screen Apinya for symptoms of anxiety and depression and provide early referral to support services as required. You can also direct patients to local support groups or the Australian Alopecia Areata Foundation.

Resources for health professionals

- DermNet New Zealand – Alopecia areata, https://dermnetnz.org/topics/alopecia-areata

Resources for patients

- Australian Alopecia Areata Foundation, https://aaaf.org.au

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CASE

3 Holden has a rash on his buttocks

Holden, a male accountant aged 58 years, presents with a rash affecting his buttocks. It has been present for several weeks, causing a persistent itch that he finds embarrassing. His medical history is significant for type 2 diabetes and obesity.

Question 1

What further history would be helpful in determining the aetiology of Holden's rash? What specifically would you look for on examination?

Further information

Holden has not noticed the rash anywhere else on his body and he has never had this problem before. He lives with his wife, who does not have a rash or any symptoms; they have no pets. Holden has never been diagnosed with skin disease previously.

He reports using hydrocortisone 1% cream for two weeks that he purchased over the counter at the recommendation of his pharmacist. The itch lessens when he uses the topical corticosteroid, but the rash has continued to increase in size, and the itch returns on cessation of therapy.

Examination reveals an annular erythematous patch that is poorly demarcated from the normal skin with no scale (Figure 1). There are no pustules, vesicles or bullous lesions. You find no evidence of psoriasis or other skin disease elsewhere.

Question 2

What is your working diagnosis and the differential diagnosis for this rash?

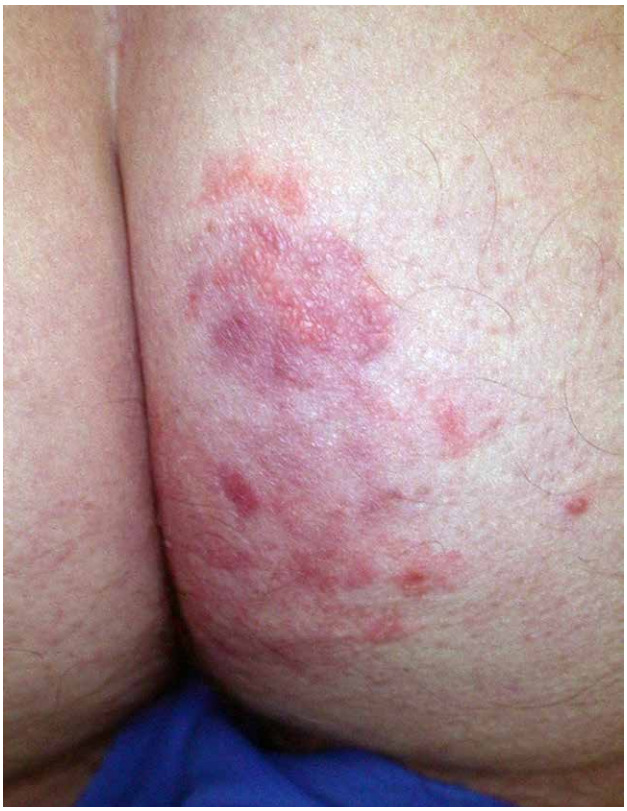

Figure 1. Erythematous rash on the buttocks, which is poorly demarcated with no evidence of scale

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Question 3

What further investigations would you consider?

Further information

Skin scrapings are obtained, and a potassium hydroxide preparation reveals segmented hyphae consistent with dermatophyte infection. Fungal cultures are positive for *Trichophyton rubrum.*

Question 4

What treatment would you recommend?

Question 5

What advice would you give Holden and his wife to minimise the risk of transmission?

Further information

Holden's rash completely resolves after two weeks of treatment, but he re-presents a month later with the same problem, this time affecting his groin.

Question 6

Why has Holden's rash recurred? Aside from the skin surface, what would you also examine?

Further information

Examination of Holden's feet reveals multiple nails with chalky yellowish-brown discolouration, subungual hyperkeratosis and onycholysis (Figure 2).

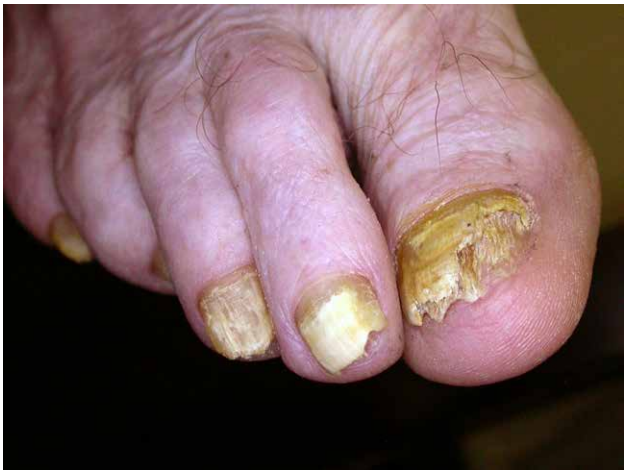

Figure 2. Multiple dystrophic nails exhibiting yellow discolouration suggestive of onychomycosis

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Question 7

How would you confirm whether Holden has onychomycosis?

Further information

Nail clippings are acquired for fungal microscopy and culture, which confirms onychomycosis with *T. rubrum*. The nails have hence been a reservoir for Holden's recurrent tinea corporis.

Question 8

What is the appropriate treatment for Holden's onychomycosis?

CASE 3 Answers

Answer 1

A detailed history should be obtained as this will often yield important information about the likely aetiology. The duration and distribution of the rash, as well as any previous episodes or treatment, should be determined.

Enquiring about the patient's close contacts who exhibit similar symptoms may assist in determining either an infectious or familial aetiology. Similarly, exposure to animals including domestic pets may indicate zoonotic transmission of tinea infection. It would also be useful to know whether Holden has a personal or family history of skin disease, particularly psoriasis, which commonly affects the natal cleft.

Examination of his skin from head to toe should be undertaken, paying attention to areas commonly affected by psoriasis including the extensor surfaces of the limbs, nails, scalp and hairline.

Answer 2

Several conditions should be considered as part of the differential diagnosis.

Tinea incognita

Tinea incognita refers to tinea that has been treated with a topical immunosuppressive agent, most commonly a corticosteroid, resulting in an atypical appearance of the rash.[1] There can be reduced scale, erythema and loss of the well-demarcated leading edge. Note: Ive and Marks published a case series in 1968 coining the term 'Tinea incognito', which is widely accepted and understood but is grammatically incorrect. Tinea incognita is the correct term.

Psoriasis

While psoriasis commonly affects the extensor surfaces, there is a variant that affects the skin folds called flexural (or inverse) psoriasis. Flexural psoriasis lacks the characteristic thick white scale of the other types of psoriasis, often exhibiting a smooth, shiny surface but retaining the prominent erythema and well-demarcated raised border.[2]

Eczema/dermatitis

Eczema can give rise to an itchy patch of skin that partially responds to topical corticosteroids. However, eczema tends to be less annular in appearance and typically does not have central clearing.

Candidal intertrigo

Candida albicans infection of the skin folds could produce this appearance and, unlike tinea cruris, will be uniformly red without central clearing. It occurs more commonly in patients with diabetes and those who are obese.[3]

Erythrasma

Erythrasma is an infection caused by *Corynebacterium minutissiumum*, a Gram-positive bacillus. It typically affects interdigital and intertriginous areas and presents as well-defined erythematous patches or thin plaques. Erythrasma may have fine scale and wrinkling described as 'cigarette paper' appearance. It typically lacks an active scaling border.

The short duration of Holden's symptoms and lack of response to topical corticosteroids favour an infectious aetiology, and the clinical appearance is more in keeping with tinea incognita.

Answer 3

Appropriate investigations would include obtaining skin scrapings and swabs for fungal and bacterial microscopy and culture. Tinea and candidal intertrigo are readily diagnosed on microscopy and fungal culture. Skin scrapings can be obtained from the leading edge of the lesion with the blunt side of a No. 15 blade.[4] Topical treatments can diminish the amount of scale, making it difficult to obtain skin scrapings; cessation of topical treatments allows the scale to return after a few days. A skin biopsy for histology and periodic acid–Schiff staining for fungal elements is an alternative investigation, but it is more invasive.

Answer 4

Dermatophytes is the collective name for fungal pathogens capable of invading keratinised tissue (skin, hair and nails) and resulting in an infection called tinea.[3] Tinea is further defined by appending the body site affected in Latin (Table 1).

Table 1. Classification of tinea affecting different areas of the body[3]

Classification of tinea	Location
Tinea pedis	Feet
Tinea manuum	Hands
Tinea barbae	Beard
Tinea cruris	Groin
Tinea capitis	Scalp
Tinea corporis	Body, excluding the sites above
Tinea unguium (onychomycosis)	Nails

Holden should commence topical terbinafine, which is the first-line treatment for tinea corporis, tinea cruris and tinea pedis for both adults and children.[5,6] He should continue treating the area once or twice daily for up to two weeks.

Answer 5

General advice should be provided to avoid the infection propagating to other areas, prevent transmission and mitigate risk of recurrence:

- Avoid walking barefoot in communal bathing areas.
- Wash socks daily and avoid moist footwear.
- Do not share footwear, clothing or sports equipment.
- Avoid touching the affected area.
- Wash hands after touching the affected area.
- Regularly wash clothing that comes into contact with the affected area.

Answer 6

Tinea may spread to other areas of the body via autoinoculation, thus examination of other sites that may serve as a reservoir for infection is necessary. The recurrence of tinea must always prompt examination of the feet and toenails as this area is a common source of fungal pathogens.[7,8] Treatment of these areas is usually needed to cure the condition.

Resistance to terbinafine outside of India is not common and is unlikely to be the reason treatment has failed in this case.[9]

Answer 7

The diagnosis of onychomycosis should be confirmed by sending a nail clipping for fungal microscopy and culture. It is important to note that the false-negative culture rate is approximately 30%, and repeated testing is sometimes required.[10] It is also important to remember that not all dystrophic, discoloured nails are due to onychomycosis; psoriasis, for example, is a common mimic.[11]

Answer 8

The recommended first-line treatment for onychomycosis of toenails is oral terbinafine 250 mg daily for 12 weeks.[12] Terbinafine is well tolerated, with mild, transient gastrointestinal adverse effects most commonly observed.[13] It is contraindicated in patients with acute or chronic liver disease because of rare cases of hepatic failure occurring in this subgroup.[14] Blood tests prior to initiation or during treatment for monitoring are not required for patients without significant comorbidity.[15] Studies have shown that, overall, 99.9% of monitoring tests resulted in no clinical action.[15]

More than 80% of patients treated with systemic antifungal therapy achieve mycological cure; however, complete cure (normal nail appearance and negative mycology) is only achieved in 25–50% of patients.[16,17] Patients concerned about ongoing abnormal nail appearance may be referred to a dermatologist for consideration of alternative causes of dystrophic nails that could have predisposed the nail to fungal infection. Topical treatments for onychomycosis achieve low rates of cure and are generally not recommended.[5,6]

Recurrence of onychomycosis is not uncommon, and it often occurs many years after the cessation of systemic therapy.[17]

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CASE

4 Paulo has uncomfortable genital itching

Paulo, aged 55 years, is a married man who presents with worsening of an itchy and painful penile foreskin that is difficult to retract, a condition he has had for two years. A year ago, he was treated with hydrocortisone 1%/clotrimazole 1% cream at another practice for assumed candidiasis with no satisfactory effect.

Question 1

How would you approach this presentation?

Further information

On examination, you note whitish discolouration of the previously pink-red mucosa of the prepuce. The prepuce is also characterised by white papules and plaques with hyperkeratosis, fissures and atrophy, resulting in partial phimosis. Paulo tells you that initially he experienced only pruritus, but symptoms have worsened recently, leading to pain and difficulty in retraction. Paulo discloses that he has painful sexual function, and anxiously says he has had no sexual partners other than his wife during his 25-year marriage.

Question 2

What conditions would you include in your differential diagnosis, on the basis of this presentation?

Question 3

From those conditions listed in the differential diagnosis, which is the most likely?

Question 4

How would you proceed to diagnose this condition?

Question 5

What is lichen sclerosus and what are the clinical features of penile lichen sclerosus (PLS) or genital lichen sclerosus (GLS)?

Question 6

What is the aetiology and epidemiology of GLS?

Further information

You explain to Paulo the typical appearance of GLS (Figure 1) and explain its aetiology and epidemiology. Paulo is worried about the long-term effects of his condition.

Question 7

What are the potential complications of GLS?

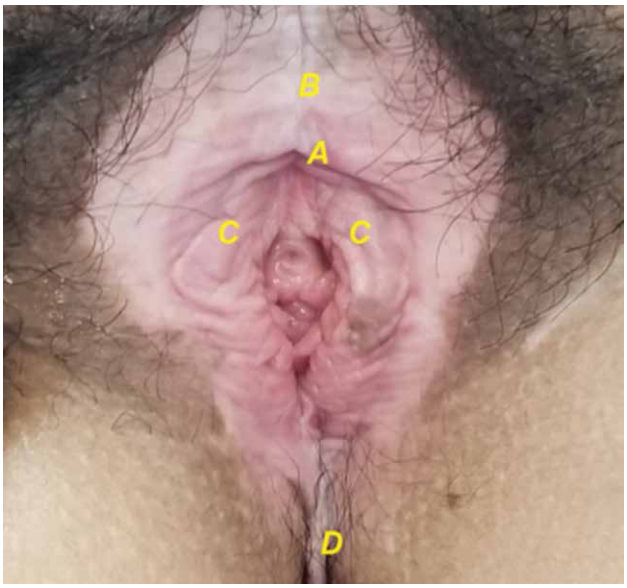
Further information

You reassure Paulo that PLS is not a sexually transmissible infection (STI) and refer him to a urologist for structural treatment for phimosis. Paulo wants to know if there are any other treatments available.

Question 8

What are the management options for GLS?

Penile lichen sclerosus
White papules and plaques, and fissures with phimosis. The loose white material is wet scales of lichen sclerosus in the mucosa; it is not induced by *Candida* sp. infection.



Vulva lichen sclerosus
A. Buried clitoris; B. Involvement of clitoris hood; C. Distorted labia minora; D. Extending to perineum

Figure 1. Genital lichen sclerosus in a man (upper) and woman (lower)

CASE 4 Answers

Answer 1

More information should be sought regarding the progression of the symptoms and whether an STI should be considered as part of the differential diagnosis. With Paulo's permission, it is important to conduct a physical examination of his foreskin.

Answer 2

Conditions to consider in the differential diagnosis include:[1,2]

- lichen sclerosus (itchy white sclerotic lesions; almost always found in the anogenital area; fissures; phimosis)
- lichen simplex chronicus (itchy patches that are temporarily relieved with scratching; usually crusted)
- psoriasis (characterised by prominent erythema with scales)
- vitiligo (no symptom of itch; generally homogenous white patch)
- post-inflammatory hypopigmentation (no itch; important to enquire about past history of any genital lesion)

- lichen planus (more pain than itch)
- *Candida* sp. infection (Candida balanitis; rare in men; excluded with swab)
- Zoon balanitis (shiny red mucosa)
- morphoea (no itch, with hard and thick skin; rare in genital areas)
- penile neoplasm (slow-growing tumour; important to enquire about any bleeding from the lesion).

Answer 3

Given the pale-white prepuce with white sclerotic papules and plaques (hyperkeratosis), fissures and anatomical distortion, the most likely diagnosis is PLS.

Answer 4

Diagnosis of PLS can be made clinically without a mandatory biopsy. If uncertainty exists, a punch biopsy from the whitest area is warranted to confirm the diagnosis and exclude alternative diagnoses including development of squamous cell carcinoma (SCC). The histopathology usually shows an atrophic or hyperkeratotic epidermis with lichenoid infiltrate in the dermal–epidermal junction, and homogenisation of collagen in the upper dermis.[2–4]

Answer 5

Lichen sclerosus is a chronic inflammatory dermatosis commonly affecting the anogenital region. It is characterised by white sclerotic papules, plaques and patches that subsequently coalesce, becoming a shiny porcelain-white or ivory-white colour. When it affects the penis, lichen sclerosus is called PLS; when it affects the vulva, it is called vulvar lichen sclerosus. The condition was previously known as balanitis xerotica obliterans (in men) and lichen sclerosus et atrophicus, leukoplakic vulvitis and lichen albus (in women).[1,5] The clinical manifestations include:

- Intractable pruritus, and pain and bleeding from fissuring and erosion.
- Sexual dysfunction.
- Phimosis and stricture of urethral meatus from atrophy and scarring. Anatomical distortions in females include burying of the clitoris, fusion or loss of labia minora, stenosis of the introitus, and distortion of urethral orifice (Figure 1).
- Morphological features include white sclerotic papules, plaques and patches on the prepuce and/or glans penis, or vulva. Areas of purpura, fissures and erosion can occasionally be seen. Extragenital lichen sclerosus is seen in 15–20% of patients with lichen sclerosus[2] and usually presents as hypopigmented and atrophic patches, commonly affecting the shoulder, arm, neck, thigh, buttock and breast.
- Dermoscopic features include patchy white structureless areas, ice slivers, comedo-like openings (hair-bearing area), purpuric globules, scales and dotted or sparse thin linear vessels.[3]

Lichen sclerosus often can be associated with autoimmune-related diseases such as thyroid disease, vitiligo, alopecia areata and pernicious anaemia.[2,6]

Answer 6

The exact aetiology of lichen sclerosus remains unknown. Several theories have been proposed such as autoimmune (approximately 20% association), genetics (12% positive family history), hormonal factors, chronic trauma and irritation.[1,6,7] Lichen sclerosus commonly affects individuals aged in the fifth decade and onwards but can be seen in patients of any age including prepuberty. The precise incidence and prevalence of GLS is difficult to ascertain. This is due to lack of awareness of the condition, embarrassment resulting in reluctant disclosure of symptoms, and presentation at and referral to different practitioners such as general practice, sexual health, gynaecology, urology and dermatology.[1,3,6] However, GLS is 10 times more common in women than men. Although early literature reported that lichen sclerosus affects a greater proportion of people of Caucasian ethnicity, it can occur in people of all ethnicities.[1]

As a result of the association with autoimmune diseases, a blood test for autoantibodies may be ordered if the patient has any suggestive symptoms.

Answer 7

The complications of GLS are:

- anatomical distortion (as described in clinical manifestations) – resulting in sexual dysfunction and urinary problems
- psychological effects – psychological distress and low self-esteem affecting sexual function and quality of life
- cancer – the increased risk of SCC is approximately 5%.[1,2]

Answer 8

Goals of treatment for GLS are to alleviate symptoms such as pruritus, fissuring and pain; to improve sexual function and quality of life; and to reduce scarring (structural distortion) and the risk of cancer.[8]

An ultra-potent or potent topical corticosteroid (TCS) is the first-line treatment for lichen sclerosus. It provides symptom relief as well as clinical improvement, reducing complications of scarring and malignant change.[3,4,9] A TCS can be applied twice daily until symptoms (itch, sore) are relieved (approximately one week), then applied daily until the texture of the skin has returned to normal (usually one month) and then on alternating days after this period. The total treatment time is approximately three months. Frequency of TCS use and duration should be individualised depending on the extent of hyperkeratosis. Following initial treatment, maintenance treatment using twice-weekly application of a lower-potency (mid-strength) TCS such as betamethasone valerate (0.02%), triamcinolone acetonide (0.02%) or methylprednisolone aceponate (0.1%) is recommended (Table 1).[3,4,6] A TCS with an ointment base is preferred to a cream in the genital area as it is better absorbed and has a barrier function.[6,9] It is advisable to schedule a review in 4–6 weeks from the start of TCS use and again in three months' time. A 6–12

month follow-up is recommended during maintenance.[3,6] TCS therapy is safe, effective and inexpensive when compared with other treatment modalities such as topical calcineurin inhibitors, systemic oral therapy and phototherapy. Treatment failure may indicate an incorrect diagnosis, noncompliance issue, development of SCC or superimposed factors such as allergy to specific medication, infection (*Candida* sp., herpes, bacteria) and irritation from sweat and urinary and faecal materials.

General management options include:

- Counselling for the nature of disease, course, treatment and regular follow-ups. Some individuals may need reassurance that the condition is not related to STIs.
- Avoidance of scratching and irritation of the genital area through use of soap-free emollients and a protective barrier (eg paraffin or emollient) to minimise contact with urine and faeces. Tight underwear and any activities that can aggravate the sensitive mucosa (such as riding a bicycle or horse) should be avoided.
- Referral to a dermatologist for review of difficult and recalcitrant cases and alternative treatments such as topical calcineurin inhibitors, intralesional injection of steroids, systemic oral therapy (pulsed prednisone, methotrexate, acitretin, cyclosporine), phototherapy and fractionated CO_2 laser treatment.[4,5,8]
- Surgery for correction of anatomical distortion or carcinoma. Referral to a relevant specialist (eg urologist, gynaecologist or urogynaecologist) is recommended.

Conclusion

While waiting to see the urologist, Paulo is treated with a potent TCS (mometasone furoate 0.1% ointment), which results in some improvement in symptoms and texture of the lesion. He has responded well to your counselling regarding the condition.

Table 1. Classification of topical corticosteroid potency in Australasia[9–11]

Potency	Corticosteroid
Mild [Class I]	Hydrocortisone 0.5–1% Hydrocortisone acetate 0.5–1%
Moderate (mid-strength) [Class II]	Clobetasone butyrate 0.05% Hydrocortisone butyrate 0.1% Betamethasone valerate 0.02–0.05% Triamcinolone acetonide 0.02–0.05% Methylprednisolone aceponate 0.1%*
Potent [Class III]	Mometasone furoate 0.1% Betamethasone dipropionate 0.05% Betamethasone valerate 0.5%–0.1%
Ultra/super/very potent† [Class IV]	Clobetasol propionate 0.05% Betamethasone dipropionate 0.05% in optimised vehicle

*Some countries classify methylprednisolone aceponate as potent.[10]
†Topical cortiocosteroids in the very-potent group are reserved for dermatologists' prescription in Australasia.
Note: Some of topical corticosteroids unavailable in Australia are not listed here.

Summary

Early detection and treatment with timely referral for genital skin disorders such as GLS will reduce patient morbidity, physically and emotionally. The prognosis of GLS is usually favourable if it is diagnosed and treated in the early non-scarring stages, and the patient is compliant with treatment.

Resources for health professionals

- DermNet New Zealand, www.dermnetnz.org

Resources for patients

- Australian and New Zealand Vulvovaginal Society, www.anzvs.org/patient-information
- The Association for Lichen Sclerosus & Vulval Health, www.lichensclerosus.org

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CASE

5 Zivko has a painful rash

Zivko, aged 69 years, presents to your general practice and tells you he has a painful rash.

Question 1

What further history would you take from Zivko?

Question 2

What would you look for on examination?

Further information

Zivko states that the rash, which started the previous day, is on the right side of his back. He has never had a similar rash before and has no idea what might have triggered it. He has not been in contact with anyone with a similar rash. He also cannot clearly recall whether he had chickenpox as a child. The rash is only slightly itchy but intensely painful. He describes a constant throbbing pain that started as a tingling sensation two days before he noticed the rash. He grades his pain as 6/10 on a verbal numerical rating scale of zero (no pain) to 10 (worst pain imaginable). He took paracetamol tablets but they did not provide adequate pain relief, and he is hoping you will prescribe more effective pain relief medication. Other than the bothersome pain, Zivko is otherwise well. He takes amlodipine 5 mg daily for hypertension and has not recently started any new medication.

Clinically, Zivko appears systemically well with normal vital signs. Examination of his skin shows a right-sided unilateral dermatomal vesicular rash on his low back area. There is no visible excoriation and no sign of superimposed bacterial infection (Figure 1).

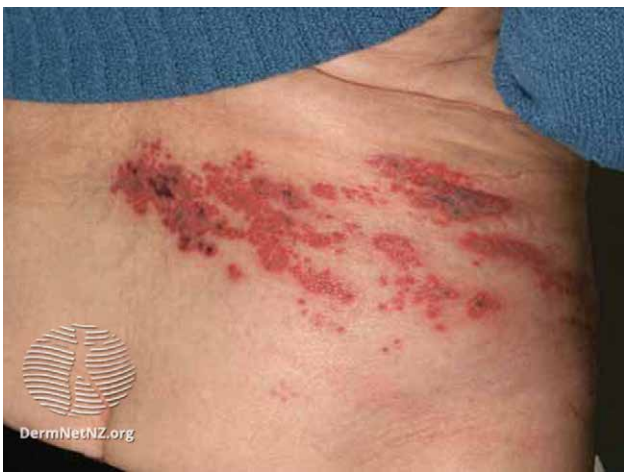

Figure 1. Zivko's rash

Question 3

What is your working diagnosis?

Question 4

What diagnostic tests, if any, would you consider? What other conditions would you consider in your differential diagnosis?

Further information

Zivko wonders why he got shingles. He is anxious about the diagnosis and his risk of infecting others.

Question 5

How would you address these concerns?

Question 6

How would you manage Zivko's presentation?

Further information

Zivko wants to know how to care for the rash.

Question 7

What would you recommend?

Question 8

What are the potential complications of shingles?

Further information

Zivko is worried about the recurrence of shingles and asks what he can do to prevent this.

Question 9

What would you advise Zivko?

Further information

As with most cases of shingles, you are able to successfully manage Zivko within the general practice setting. However, as part of your routine reflective practice, you consider situations in which patients with shingles should be referred/discussed with a non–general practitioner (GP) specialist.

Question 10

What are some of the scenarios in which you might refer a patient with shingles to a non-GP specialist?

CASE 5 Answers

Answer 1

A targeted history should explore the following aspects of the presentation.

History of the rash

Important questions to ask Zivko about his rash include:[1]

- Where is it?
- When did it start?
- Has he had previous episodes of a similar rash?
- Does he have any idea of what could have caused/triggered the rash?
- Has he had any contact with someone with a similar eruption?
- Are there other associated symptoms (eg pruritus, fever)?
- Has he used any treatment for the rash?

Pain history

An acute pain history exploring the location, duration, nature, severity, periodicity and any associated functional impairment (in relation to mood, work, sleep and activities of daily living) is important. Furthermore, Zivko should be asked about circumstances surrounding the pain onset and the effect of any treatment that he may have taken.[2]

Medication and medical history

It is important to ask Zivko whether he has started any new medication or non-prescribed products that could have triggered the rash, and whether he has a medical condition that may have dermatological associations.[1]

Systemic and constitutional symptoms

Fever in the setting of a rash is often due to an infective process,[1] which may be benign (eg viral exanthem) or potentially life-threatening.[1,3] Without systemic sepsis, most localised rashes will only cause minimal systemic symptoms.[3] Therefore, constitutional symptoms such as fever, unexplained significant weight loss, fatigue and athralgia in the setting of a rash should prompt evaluation for serious systemic illnesses such as malignancy, inflammatory conditions and infection.

Answer 2

A focused examination should include an assessment of the patient's general wellbeing and vital signs in addition to the nature and distribution of the rash. Patients with acute signs of severe systemic illness will require urgent transfer to the emergency department.[3]

The morphological appearance[3] (macules, papules, vesicles, pustules, etc) of the rash and distribution (symmetrical/asymmetrical, localised/generalised or dermatomal) will inform the differential diagnosis. It is important to check for excoriations (seen with pruritic rash) and signs of superimposed bacterial infection.[3]

The remainder of the clinical examination will be guided by the information gathered during history-taking. This may involve checking for signs of suspected systemic diseases such as lymphadenopathy or hepatosplenomegaly.

Answer 3

Zivko's presentation is consistent with a diagnosis of herpes zoster (HZ), also known as shingles. The painful unilateral dermatomal vesicular rash typically seen in shingles is due to reactivation of latent varicella zoster virus (VZV) in a dorsal root or cranial nerve ganglion.[4] Sometimes, natural variation in innervation may cause few lesions beyond the midline or the affected primary dermatome.[5]

For 70–80% of patients, the onset of HZ is heralded by prodromal acute neuralgia often described as a constant or intermittent burning, tingling, shooting, throbbing or stabbing pain.[5] Typically, after 2–3 days of prodromal pain, the patient develops a crop of erythematous macules and papules that evolve rapidly to vesicles.[5,6] Pustulation, ulceration, crusting and healing occur over the following 2–4 weeks.[5,6] Shingles can affect any part of the body but often affects the thoracic, lumbar and cervical dermatomes. Rarely, HZ manifests solely as dermatomal pain and it is called zoster sine herpete.[5]

Approximately 20% of patients with HZ have systemic symptoms such as headache, fever and fatigue.[5] Patients who are immunocompromised are at risk of severe shingles with potentially life-threatening complications. They may present with disseminated shingles, atypical skin lesions, multidermatomal disease, systemic illness and visceral HZ with multi-organ involvement.[4,5]

Answer 4

Typical shingles, such as Zivko's presentation, can be diagnosed clinically.[4] Diagnostic laboratory tests are reserved for atypical presentations such as zoster sine herpete, patients presenting with disseminated skin infections or visceral HZ.[5] Differentiating between VZV and herpes simplex virus (HSV) infection as a cause of painful oral or genital lesions may be difficult.[5] Zosteriform HSV infection may be misdiagnosed as 'recurrent HZ'.[4] Other diagnoses to consider include impetigo and contact dermatitis.[5,7]

Also, depending on the location, pre-eruptive acute neuralgia of HZ or zoster sine herpete may mimic pain related to coronary artery disease, renal colic, appendicitis and cholecystitis.[8]

Laboratory tests to confirm a diagnosis of HZ include VZV polymerase chain reaction (PCR), direct immunofluorescence antigen testing and viral cultures using viral swabs of the base of deroofed vesicles.[5,7,8] A comparison of these methods concluded that VZV PCR testing is the method of choice for rapid laboratory diagnosis of HZ as it has the highest sensitivity (95%) and specificity (100%).[9] VZV PCR testing is widely available in Australia. Serological testing has limited diagnostic value.[8]

Answer 5

Appropriate education about the diagnosis of shingles and expected course will address Zivko's concerns, dispel any myths and provide appropriate reassurance. Zivko should understand that anybody who has had chickenpox (more than 90% of adults in Australia) is at risk of developing HZ.[6] Patients may not clearly recall the episode of chickenpox.[8] Those without vaccination against chickenpox or shingles have a one-in-three lifetime risk of developing HZ.[10] Increasing age is a risk factor for shingles and for severe disease.[4] In Australia, most cases of shingles occur in adults who are immunocompetent; however, occasionally a person's immunocompromised state because of illness (eg from human immunodeficiency virus infection or malignancy) or immunosuppressive therapy is the trigger.[11]

It is also recommended to advise Zivko that shingles is contagious – through direct or indirect contact with fluid from vesicles – to people who have never had chickenpox. As such, Zivko should exercise contact precautions[4] by covering his rash[8] and avoiding contact with susceptible individuals (eg children, pregnant women and immunosuppressed individuals) until all the lesions have crusted. Additional airborne precautions are required for patients with HZ who are immunocompromised or those with disseminated lesions.[4]

Answer 6

Antiviral therapy

Multiple randomised controlled trials (RCTs) have shown that commencement of either oral aciclovir, famciclovir or valaciclovir within 72 hours of rash onset reduces the severity and duration of both acute pain and rash in HZ.[5] These antiviral medications are safe and generally well tolerated but require dosage adjustment for patients with renal failure.[5] Famciclovir and valaciclovir are the recommended first-line treatments[12] because of more convenient dosing, greater bioavailability[5] and better analgesic effect than acyclovir (Table 1).[5,12]

Antiviral therapy is indicated for immunocompetent adults and adolescents who present within 72 hours of the appearance of the HZ rash.[12] Generally, shingles in children is less painful and requires no antiviral treatment.[12] However, all patients who are immunocompromised, those with severe or fulminant shingles, or those with HZ ophthalmicus (HZO) should receive antiviral therapy irrespective of the duration of the rash.[12] Referral for intravenous aciclovir should be arranged for patients with severe, fulminant or non-responding HZO and patients who are immunocompromised with disseminated disease or HZO.[12]

Pain management

Severe acute pain may be a risk factor for post-herpetic neuralgia (PHN),[5] hence the importance of accurate assessment and prompt treatment of acute neuralgia. Paracetamol[12] and non-steroidal anti-inflammatory drugs[5] (if not contraindicated) are the recommended first-line treatment for mild-to-moderate HZ pain. Combination treatments with oral opiates (oxycodone), corticosteroids (prednis[ol]one) and analgesic adjuvants (eg amitriptyline, pregabalin) are recommended options for patients with moderate-to-severe pain and no applicable contraindications.[5,12]

Table 1. Antiviral therapy recommendations for shingles[12]

Antiviral therapy	Dosage	Notes
Valaciclovir	1 g (child >2 years: 20 mg/kg up to 1 g) orally, eight-hourly for seven days	While not licensed in Australia for use in children aged <12 years, it is licensed internationally for use in children aged >2 years. Emerging evidence from safety data and clinical experience suggest valaciclovir is safe to use in pregnancy.
Famciclovir	500 mg orally, eight-hourly for seven days	Treatment duration for patients who are immunocompromised is 10 days. Famciclovir is not recommended for use in children.
Aciclovir	800 mg (child: 20 mg/kg up to 800 mg) orally, five times daily for seven days	Aciclovir has the most evidence on safety data to support use in pregnancy.
	10 mg/kg (child ≤12 years: 500 mg/m^2) intravenously, eight-hourly	In disseminated disease, after significant clinical improvement, change to an oral antiviral therapy to complete a total of 10–14 days.

Answer 7

Zivko should cover his rash with a non-adherent dressing following removal of crusts and exudate with a regular saline bath[8,11] and application of protective ointment such as petroleum jelly.[6] Until the results of sensitivity testing are available, empirical oral antibiotics should be initiated for any superimposed bacterial infection with *Streptococcus pyogenes* and *Staphylococcus aureus*.[12] Topical antiviral therapy lacks efficacy,[12] while topical antimicrobials or adhesive dressings may delay healing and worsen irritation.[7]

Answer 8

In most cases, shingles is a self-limiting illness.[8] Patients who are elderly and/or immunocompromised are particularly at risk of complications.

PHN refers to persistent (neuropathic) pain that occurs after resolution of shingles rash. It is the most common complication of shingles, and risk increases with age.[13] The reported estimated risk varies from 5% to more than 30%.[13] PHN is usually severe and difficult to treat, and it may cause significant functional impairment.[12] Simple analgesics are the recommended first-line treatment for PHN.[12] Treatments

options (either individually or in combination) for those who fail to respond to first-line management include adjuvant analgesics (tricyclic antidepressants, pregabalin or gabapentin), transcutaneous electrical nerve stimulation, topical anaesthetic or capsaicin.[12] Psychological interventions also have a role in the management of PHN.[7,12]

Post-herpetic itch is a poorly understood complication of HZ characterised by persistent pruritus over dermatomes previously affected by HZ.[5,14] It may exist with or without PHN[5] and has no established treatment.[14] Patients with HZO are at risk of periocular (cicatricial ectropion, paralytic ptosis and trichiasis) and ocular (keratitis, uveitis and glaucoma) complications.[5,15]

Neurological complications of HZ are uncommon.[13] They include Ramsay Hunt syndrome, myelitis and meningoencephalitis, with risk factors being an immunocompromised state, cranial nerve HZ and cutaneous dissemination.[5]

Answer 9

With an estimated recurrence risk of 5% in patients who are immunocompetent,[5] Zivko should be reassured that recurrence of shingles is uncommon. However, he should still be offered a single-dose live attenuated varicella-zoster vaccine (Zostavax) if there are no contraindications. In Australia, Zostavax is publicly funded for people aged 70 years. There is a catch-up program available for those aged 71–79 years until October 2021.[16] As the humoral immunity boost from an episode of shingles lasts at least one year, Zivko should be advised to wait at least one year before receiving Zostavax.[10] The efficacy of Zostavax in reducing the incidence of HZ and PHN has been shown in RCTs and post-marketing studies.[17]

Recently, a non-live recombinant adjuvanted HZ subunit vaccine (HZ/su; Shingrix) was registered in Australia.[16] It is given in two doses and can potentially be used in patients who are immunocompromised.[7,17] HZ/su offers more protection against HZ and PHN than Zostavax,[16,17] but supply is a challenge.[16]

Answer 10

GPs should consider referring or consulting an appropriate non-GP specialist for advice in the following situations:

- patients who are immunocompromised[7]
- severe shingles, for example, multidermatomal disease, suspected central nervous system involvement (encephalitis, meningitis, altered sensorium), disseminated disease or severe systemic infection[5]
- HZO – those with suspected/confirmed ocular involvement require acute ophthalmological assessment[4,5,8,12,15]
- failure to respond to therapy – it is necessary to exclude rare aciclovir-resistant VZV, which has been reported in patients who are immunocompromised[15]
- refractory PHN, which should be managed in consultation with a specialised pain clinic[4,12]
- Ramsay Hunt syndrome – management in consultation with an ear, nose and throat surgeon[5] or neurologist should be considered.

Resources for patients

- Better Health Channel – Shingles, www.betterhealth.vic.gov.au/health/conditionsandtreatments/shingles

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ACTIVITY ID **207936**

Skin conditions

This unit of *check* is approved for six CPD Activity points in the RACGP CPD Program. The expected time to complete this activity is three hours and consists of:

- reading and completing the questions for each case study
  - you can do this on hard copy or by logging on to the RACGP website (www.racgp.org.au), clicking on the My Account button and selecting the *gplearning 2020* link from the drop-down
- answering the following multiple choice questions (MCQs) by logging on to the RACGP website (www.racgp.org.au), clicking on the My Account button and selecting the *gplearning 2020* link from the drop-down
  - you must score ≥80% before you can mark the activity as 'Complete'
- completing the online evaluation form.

You can only qualify for CPD points by completing the MCQs online; we cannot process hard copy answers.

If you have any technical issues accessing this activity online, please contact the *gplearning* helpdesk on 1800 284 789.

If you are not an RACGP member and would like to access the *check* program, please contact the *gplearning* helpdesk on 1800 284 789 to purchase access to the program.

Case 1 – Tanvir

Tanvir, aged 26 years, is a professional football player who comes to see you regarding a recurrence of tinea that is causing itchiness between the toes of both feet. He has previously managed this condition with over-the-counter preparations but is now concerned his toenails are affected. Your physical examination confirms yellow nail discolouration on three of Tanvir's toes, suggesting onychomycosis. You recall your knowledge of the diagnosis and management of tinea and onychomycosis.

Question 1

Which one of the following statements is true regarding tinea?

A. It is caused by the organism *Corynebacterium minutissiumum.*

B. The appearance of the rash may be altered by topical corticosteroids.

C. Resistance to systemic antifungal agents in Australia is common.

D. Topical griseofulvin is first-line therapy for tinea corporis.

Question 2

Which one of the following statements is true regarding onychomycosis?

A. Recurrence is uncommon after successful mycological cure.

B. Topical treatments are usually sufficient to effect cure.

C. Autoinoculation to other body sites can occur.

D. Fortnightly liver function tests are recommended during systemic antifungal therapy.

Case 2 – Wallace

Wallace, aged 69 years, is a man who presents to you with a four-day history of a painful rash on his right forehead. The eruption was preceded by a two-day history of burning pain in the area of the eruption. He is otherwise well and has no ocular symptoms. Clinical examination shows a unilateral, dermatomal vesicular rash on an erythematous base. He has a normal eye examination.

Question 3

Which one of the following best indicates how you would manage Wallace's presentation?

A. Commence oral famciclovir

B. Immediate referral to hospital for intravenous acyclovir

C. Prescribe topical aciclovir 5% cream

D. Supportive treatment only

Further information

Wallace is worried about the possibility of shingles recurring and asks what he can do to prevent this.

Question 4

Which one of the following best represents the advice you would give Wallace?

A. Appropriately reassure Wallace that recurrence of shingles is uncommon

B. Advise Wallace to wait at least one year before receiving varicella-zoster vaccine (Zostavax)

C. Appropriately reassure Wallace that a single episode of shingles confers lifelong immunity

D. Offer Wallace a single-dose of live attenuated varicella-zoster vaccine (Zostavax) as soon as the lesions have crusted

Case 3 – Abbas

Abbas, aged 28 years, presents with a new rash around his right wrist. The rash started one week ago, and it is itchy and

red with blisters. Abbas works as a printer and uses inks, solvents and wash-up solutions regularly; however, he reports minimal contact with these chemicals as he wears gloves. Other than wearing a new watch, he has not made any changes to his skin care products or fragrances. You examine the rest of his skin; his hands and feet are unaffected, but you notice a small coin-sized area of faint erythema below the umbilicus.

Question 5

Which one of the following is the most likely diagnosis?

A. Irritant contact dermatitis from handwashing and chemical exposure

B. Allergic contact dermatitis secondary to nickel

C. Pompholyx

D. Dermatophyte infection

Question 6

Which one of the following is the most appropriate test to confirm the diagnosis of Abbas's rash?

A. Skin prick testing

B. Allergen-specific immunoglobulin E testing

C. Skin scrapings for fungal microscopy and culture

D. Patch testing

Case 4 – Rhea

Rhea, aged 32 years, is a nurse who comes to see you as she is concerned about a small patch of hair loss on the back of her scalp, which was noticed by her hairdresser at a recent appointment. The hairdresser had not noticed any hair loss at Rhea's appointment six weeks earlier, and Rhea does not report any previous hair loss. In your differential diagnosis, you consider alopecia areata as a possible cause for Rhea's symptoms.

Question 7

Which one of the following best indicates signs or symptoms that may suggest alopecia areata?

A. Diffuse hair thinning, irregular menses, acne and hirsutism

B. Young age, family history of autoimmune disease, abrupt onset

C. Pain, itchiness or burning of the scalp

D. Abrupt postpartum hair thinning

Question 8

Which one of the following is a dermoscopy feature characteristic of alopecia areata?

A. Perifollicular scaling and erythema

B. Broken hairs and hairs of different lengths

C. Comma hairs

D. Exclamation mark hairs

Case 5 – Manisha

Manisha, aged 54 years, comes to see you reporting itchiness of the vulva; she is concerned she has thrush. She also reports that she is finding sex painful. You undertake a thorough history and a physical examination, which shows white patches of skin in the vulval area. A skin biopsy confirms Manisha has lichen sclerosus and you consider how to manage her condition.

Question 9

Which one of the following topical corticosteroids is the most appropriate for initial treatment of lichen sclerosus?

A. Clobetasone butyrate 0.05%

B. Betamethasone dipropionate 0.05%

C. Triamcinolone acetonide 0.02%

D. Hydrocortisone 1%

Question 10

Which one of the following is a structural complication of vulvar lichen sclerosus?

A. Burying of the clitoris

B. Fusion or loss of labia minora

C. Stenosis of the introitus and urethral orifice

D. All of the above

check

Independent learning program for GPs