# **Red in the face:** Approach to diagnosis of red rashes on the face

### Ankit Gupta, Sara Lee de Menezes

### **Background**

A red rash on the face in an adult patient is a common presentation to general practice in Australia. Rashes on the face significantly affect quality of life because this is a cosmetically sensitive site. Ascertaining the correct diagnosis is therefore of utmost importance so that appropriate treatment can be initiated.

### Objective

This article discusses the assessment of red rashes on the face in an adult patient.

### Discussion

Diagnosing a red rash on the face requires assessment of symptomology, age of onset, rash morphology and 'clinical clues' that help delineate between differentials. Although the list of differential diagnoses is wide, many of the common diagnoses can be made clinically without the need for investigations. Investigations such as skin biopsy are useful if the diagnosis is unclear, if the rash is not responding to initial treatment and/or a referral to a dermatologist is being considered.

A RED RASH ON THE FACE in an adult patient is a common presentation to primary care. A thorough clinical assessment is often able to delineate between common causes. In undifferentiated rashes, investigations such as skin biopsy, skin scrapings and swabs might be helpful.

### Aim

This article discusses the assessment of red rashes on the face in adults.

### Assessment of red facial rashes

Red rashes on the face have a wide differential diagnosis. Assessment of these patients requires a thorough history and examination. Many of the common diagnoses can be made clinically without the need for investigations.

### Differential diagnosis

A red rash on the face can be due to an underlying dermatological condition or infection, or it could be triggered by exogenous factors (Table 1). These causes can be differentiated by assessing the typical age of onset, distribution and morphology (Table 2).

# Approach to red rash on the face in adults

An efficient method of assessing a red rash on the face in adults is to take a history of the rash, followed by a clinical examination, and to then ask targeted questions based on examination findings (ie 'clinical clues') to help differentiate between remaining possible diagnoses (Figure 1). An assessment of rash morphology allows the clinician to narrow the differential diagnoses (Figure 2).

### History of presenting issue

The history would ideally begin with open-ended questions that help clarify the rash onset, symptomology and effects on quality of life. The differential diagnosis can be narrowed depending on the typical age of onset and the pattern of involvement (Tables 1, 2). For example:

- seborrhoeic dermatitis might begin in adolescence and follow a relapsing and remitting course, with men being more commonly affected than women<sup>6</sup>
- young female patients are prone to irritant and allergic contact dermatitis due to exposure to cosmetic products<sup>5</sup>
- atopic dermatitis might initially present in childhood and either persist or recur in adulthood to include facial involvement<sup>19</sup>
- acne vulgaris shows peak incidence in the late teens, with a progressive decrease in prevalence with age.<sup>7</sup>

The distribution of the rash provides valuable information about the possible cause (Table 1):

- involvement of the frontal hairline, occipital scalp and ears can suggest psoriasis
- seborrhoeic dermatitis is common in skin with high sebum production, such as the scalp, eyebrows, glabella, nasolabial folds and beard

Diagnosis	Morphology	Distribution	Clinical clues	Diagnostic tests
Atopic dermatitis (eczema)	Erythema, scale, excoriations, xerosis     Lichenification     Ill-defined plaques     Honey-coloured crusts if secondarily infected	Anywhere on face, neck, scalp     Variants:	<ul> <li>History of atopy (eg asthma, allergic rhinitis, eczema [including in family members])</li> <li>Keratosis pilaris on upper arms/ thighs</li> <li>Lichenification suggestive of chronicity</li> <li>Itch can be significant</li> <li>Predominance of face/neck eczema might suggest aeroallergens (eg dust mites, pollen, grasses, animal dander)</li> </ul>	Clinical diagnosis Bacterial MCS if secondarily infected Allergen-specific IgE testing for common aeroallergens if indicated Skin biopsy: histopathology if diagnosis unclear
Irritant contact dermatitis	Xerosis     Erythema and scale might be present	Worse in skin creases (eg corners of mouth, ala creases)     Sparing of eyelids, ears, frontal hairline or neck if irritant not in contact with those areas	Timeline with use of one or more new products Over-cleansing of face Might or might not be itchy Patients with atopic dermatitis more prone to developing irritant contact dermatitis Might have single exposure to strong irritant or chronic exposure to weak irritants	Clinical diagnosis     Cessation of new or unnecessary products and gentle skin care (eg bland unscented moisturiser, sunscreen)
Psoriasis	Well-defined, scaly, salmon-pink plaques     Scale can be significant especially in scalp	Favours ears and scalp (especially frontal hairline and occipital scalp)     Might have psoriasis elsewhere (eg extensors, umbilicus, natal cleft, palmoplantar)	Mostly asymptomatic but can be itchy     Concurrent seborrhoeic dermatitis (sebopsoriasis)     Personal or family history of psoriasis	Clinical diagnosis     Skin biopsy:     histopathology if     diagnosis unclear
Seborrhoeic dermatitis	• III-defined erythema, greasy scale	Favours seborrhoeic areas: scalp, forehead, eyebrows, nasolabial fold, conchal bowl, post-auricular	<ul> <li>History of dandruff or flaking scalp</li> <li>Other sites might be involved including chest, axillae</li> <li>Can be itchy if inflamed, otherwise relatively asymptomatic</li> <li>Winter flares with improvement in summer</li> </ul>	Clinical diagnosis     Skin biopsy:     histopathology if     diagnosis unclear
Actinic keratoses	Ill-defined erythema and scale     Might be discrete or confluent to involve large areas     Hyperkeratotic papules or nodules in some areas	Photo-exposed sites (eg nose, cheeks, temples, ear helices); scalp involvement if bald or thin hair  Might have actinic keratoses elsewhere (eg dorsal hands, forearms, lower legs  Sparing of photoprotected sites	<ul> <li>Asymptomatic</li> <li>Gradual onset over time</li> <li>History of chronic sun exposure/ limited photoprotection</li> <li>No history or other features of primary dermatosis</li> </ul>	Skin biopsy:     histopathology     (particularly for     hyperkeratotic, tender     or indurated lesions to     exclude invasive SCC)
Acne	Polymorphic erythematous papules, pustules and nodules Cysts might be present in severe cases Closed or open comedones Postinflammatory pigmentation Excoriations (if picking) Acne scars (eg box car, rolling, atrophic, ice pick scars)	Favours face, back and chest	<ul> <li>Family history of acne</li> <li>Papules, pustules and nodules are in different stages of evolution</li> <li>Teens: onset around puberty</li> <li>Adults: persistence since teens or onset early 20s. In women, might flare with menses</li> <li>Might be associated with testosterone or anabolic steroid usage</li> </ul>	Clinical diagnosis

Diagnosis	Morphology	Distribution	Clinical clues	Diagnostic tests
Rosacea	<ul> <li>Monomorphic erythematous papules and pustules (papulopustular subtype)</li> <li>Absence of comedones (unless there is coexisting acne)</li> <li>Telangiectasia and erythema (in erythematotelangiectatic subtype)</li> </ul>	Favours dorsum of nose, cheeks, chin, forehead	<ul> <li>Favours fair-skinned patients</li> <li>Might be associated with flushing</li> <li>History of sensitive skin to various products</li> <li>Flares with sun exposure</li> <li>Family history of rosacea</li> </ul>	Clinical diagnosis
Malassezia folliculitis	Monomorphic erythematous papules and pustules based around hair follicles     Absence of comedones	<ul> <li>Favours forehead, chin, lateral cheeks</li> <li>Central face usually spared</li> <li>Chest and back might be involved</li> </ul>	Might have other forms of cutaneous Malassezia spp. overgrowth (eg seborrhoeic dermatitis, pityriasis versicolour)     Oily skin     Flares with heat, sweat, occlusion or in hot, humid conditions     Might be itchy	Clinical diagnosis Yellow-orange fluorescence on Woods lamp Skin scrapings of a pustule, KOH test (culture not routinely performed because isolation and culture of Malassezia spp. is difficult)
Pseudofolliculitis parbae	<ul> <li>Erythematous papules and sterile pustules based around hair follicles</li> <li>Associated ingrown hairs</li> <li>Postinflammatory pigmentation</li> </ul>	'Beard' area of the face (ie cheeks, upper lip, chin, neck)	<ul> <li>Flares with hair removal (eg shaving, waxing, plucking)</li> <li>Favours patients with curly hair</li> <li>Most common in men with African ancestry</li> <li>Might be itchy</li> </ul>	Clinical diagnosis Bacterial MCS to exclude bacterial folliculitis Fungal skin scrapings to exclude tinea barbae
Gunburn	<ul> <li>Acute phase: erythema and oedema; blistering in severe cases</li> <li>Resolution phase: peeling and flaking of affected skin</li> </ul>	Photo-exposed sites (eg face, trunk, limbs)  Sharp cut-off and sparing of areas covered by clothing  Associated ephelides or lentigines (if chronic) in sun-exposed sites	<ul> <li>Associated with total duration of sun exposure and UV index</li> <li>Favours patients with lighter skin phototype</li> <li>Might be more likely to occur if taking photosensitising medications</li> </ul>	Clinical diagnosis
Periorificial dermatitis/ steroid-induced osacea	<ul> <li>Classically numerous papules and pustules; might be more confluent than rosacea</li> <li>Absence of comedones</li> <li>Might be erythematous, oedematous and scaly in some cases</li> </ul>	<ul> <li>Clustered around orifices (eg skin inferior to nostrils, chin, lateral cheeks/temples around eyelids)</li> <li>Sparing vermillion border of lip</li> </ul>	Might be associated with steroid exposure (eg topical mid- to highpotency steroids). Other triggers include intranasal/inhaled or oral steroids     Paradoxical flare with cessation of steroid     Associated stinging or burning sensation	Clinical diagnosis
Tinea faciei	<ul> <li>Annular eruption with a leading edge, central clearing and peripheral scale</li> <li>Might become non- scaly/non-specific if treatment modified with topical steroids (tinea incognito)</li> </ul>	Anywhere on face, neck, scalp	<ul> <li>Paradoxical worsening with use of topical steroids</li> <li>Asymmetrical</li> <li>History of recent visit to barber</li> <li>Pets with scaly patches of hair loss (eg cats, dogs, guinea pigs)</li> <li>Might have associated onychomycosis</li> </ul>	Skin scrapings for fungal microscopy and culture     Skin biopsy (if required): histopathology

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Diagnosis	Morphology	Distribution	Clinical clues	Diagnostic tests
Allergic contact dermatitis	Acute phase: oedema, erythema, vesicles or bullae     Chronic phase: mimics atopic dermatitis (erythema, scale, excoriations, lichenification)	Common sites: eyelids, lips/perioral skin, neck     Other sites also exposed to allergen might be affected	Can occur to products/chemicals that have previously been tolerated for many months or years, not just newly introduced products  Associated itch  In acute phase might be triggered 24-48 h following exposure to allergen  Ongoing exposure can lead to chronic phase	<ul> <li>Clinical diagnosis</li> <li>Referral for allergy skin patch testing</li> <li>Skin biopsy: histopathology if diagnosis unclear</li> </ul>
Shingles	<ul> <li>Prevesicular phase: crops of erythematous papules and macules</li> <li>Vesicular phase: pustules, vesicles followed by crusting</li> </ul>	<ul> <li>Favours dermatomal distribution or is unilateral</li> <li>Might be multidermatomal or disseminated, especially in immunosuppressed patients</li> </ul>	Associated burning, pain or tingling sensation precedes skin manifestations     Might be associated with systemic symptoms (eg malaise)     Red flags: ocular involvement, facial weakness, ear canal involvement	VZV swab for PCR
Cutaneous lupus	ACLE: smooth, erythematous, oedematous plaques     DLE: coin-shaped, scaly hyperpigmented plaques, follicular plugging. Might have scarring     Note: subacute cutaneous lupus erythematosus might less commonly present on the face as annular scaly plaques	ACLE: 'Butterfly' distribution, namely bilateral cheeks and across the dorsum of the nose, sparing nasolabial folds. Can affect other photoexposed sites     DLE: face, scalp, conchal bowl and ears. In diffuse form, might occur on trunk	Might have associated systemic symptoms of lupus (eg synovitis, chest pain, shortness of breath)	Skin biopsy: histopathology, direct immunofluorescence Blood tests: ANA, ENA, dsDNA, complement Urine microscopy: proteinuria, dysmorphic red blood cells, red cell casts
Dermatomyositis	<ul> <li>Pink to violaceous erythema of eyelids; might have associated oedema (heliotrope rash)</li> <li>Erythema, scale and diffuse alopecia affecting the scalp</li> </ul>	Eyelids, forehead, scalp     Might have other clinical signs of dermatomyositis (eg shawl sign, V-neck sign, Holster sign, Gottron's papules, Gottron's sign, nail fold changes)	Might have associated muscle weakness (except in amyopathic forms)     Associated photosensitivity	<ul> <li>Skin biopsy: histopathology</li> <li>Special blood tests: CK, myositis-specific antibodies</li> <li>Consider malignancy screen</li> </ul>
Facial erythema secondary to a non- dermatological systemic process (eg pulmonary hypertension, liver failure, SVC obstruction, carcinoid syndrome)	Plethora, fixed erythema, oedema, facial flushing Absence of more specific skin changes such as pustules, papules, scale, vesicles or erosions	<ul> <li>Variable distribution</li> <li>Might present as facial flushing, fixed erythema and telangiectasia involving the cheeks, forehead, periorbital region and/or neck</li> <li>Might also involve other body sites (eg palmar erythema, spider naevi on chest)</li> </ul>	Known respiratory, gastrointestinal or cardiovascular comorbidities     Systemic symptoms such as dyspnoea, hoarse voice, chest pain, weight loss, fatigue, peripheral oedema, diarrhoea and/or jaundice     Abnormal cardiorespiratory and/or gastrointestinal examination	Depending on suspected cause:

5-HIAA, 5-hydroxyindoleacetic acid; ACLE, acute cutaneous lupus erythematosus; ANA, antinuclear antibodies; CK, creatine kinase; DLE, discoid lupus erythematosus; dsDNA, double-stranded DNA; ENA, extractable nuclear antibodies; IgE, immunoglobulin E; KOH, potassium hydroxide; MCS, microscopy, culture, sensitivity; PCR, polymerase chain reaction; SCC, squamous cell carcinoma; SVC, superior vena cava; UV, ultraviolet; VZV, varicella zoster virus.

- involvement of the evelid alone can be a clue to irritant or allergic contact dermatitis from cosmetics, because this is a thin area of skin sensitive to exogenous compounds
- rosacea favours the central face, such as the cheeks and nose
- periorificial dermatitis is classically distributed around the mouth, but can also present around the nose and eyes
- actinic keratoses tend to present on photo-exposed areas such as the scalp, forehead, nose, ear helices and cheeks.

Associated symptoms can help provide clues to the underlying aetiology:

- Pruritus: atopic dermatitis is itchy and can disrupt sleep. Tinea, irritant and allergic contact dermatitis might also present with itch. In contrast, actinic keratoses are not itchy.
- Pain: shingles can present with neuropathic pain prior to rash onset. Severe acne is often described as painful.
- Burning or stinging: patients with rosacea might describe sensitive skin with a burning or stinging sensation.

Given that the face is a cosmetically sensitive area, patients might experience reductions in self-confidence and/or discrimination from their condition. Assessing the effect on a patient's quality of life will provide useful insights when assessing the motivation to seek treatment and developing a management plan.

A review of the medical and family history for evidence of prior atopy, psoriasis or connective tissue disease is helpful.

### Atopic dermatitis

· Seborrhoeic dermatitis

Age >60 years

- Psoriasis · Actinic keratosis
- Shingles · Cutaneous lupus erythematosus
- · Dermatomyositis

### Table 2. Causes of red rashes on the face categorised by typical age of onset<sup>2,5-18</sup>

### Age 20-40 years

- Common:
- · Acne vulgaris
- · Atopic dermatitis
- Rosacea
- · Seborrhoeic dermatitis
- · Periorificial dermatitis/steroidinduced rosacea
- · Irritant contact dermatitis
- Sunburn

### Less common:

- Psoriasis
- · Allergic contact dermatitis
- · Malassezia folliculitis
- · Pseudofolliculitis barbae
- · Tinea faciei
- · Cutaneous lupus erythematosus

## · Age at onset **Presenting** Symptoms Affected areas issue Quality of life impact Distribution **Examination** Morphology · Differentiating features · Targeted questions to Clinical clues narrow differentials Working diagnosis Figure 1. Suggested approach to red rashes on the face.

Age 40-60 years

· Actinic keratosis

· Cutaneous lupus

ervthematosus

Dermatomyositis

Rosacea

Shingles

· Seborrhoeic dermatitis

· Irritant contact dermatitis

· Allergic contact dermatitis

### Examination

A thorough examination of the face helps characterise the distribution of the rash and ascertain the predominant morphology. Rashes on the face can be broadly classified under scaly, papulopustular and mixed morphology (Figure 2).

### Papulopustular rashes

Papules can be identified as raised areas of skin that are <1 cm in diameter. Pustules are papules that contain white/yellow pus. Acne vulgaris can be differentiated from other papulopustular conditions by the presence of comedones, although this might not be prominent in patients with primarily inflammatory acne.

Rosacea presents with papules and pustules with background erythema and telangiectasia. Some patients with rosacea might lean more heavily towards the papulopustular spectrum, whereas others will have more erythema, flushing and telangiectasia.20 Rosacea and acne might co-exist, in which case comedones might be seen due to underlying acne. Malassezia folliculitis characteristically presents with monomorphic papules and pustules affecting the forehead, chin, neck, upper back and chest. Periorificial dermatitis presents with

clusters of papules and pustules that can be more confluent than in rosacea with sparing of the vermillion border of the lip. Pseudofolliculitis barbae occurs on areas where the hair is shaved, especially in those with curlier, course hair.

### Scaly rashes

Scale can accompany many dermatological conditions and is a sign of excess keratin.21 Seborrhoeic dermatitis presents with ill-defined erythema and greasy scale predominantly affecting the scalp, eyebrows, glabella, nasolabial folds and beard. Atopic dermatitis can present anywhere on the face but is more common around the eyes, eyelids, neck and lips. It is often associated with dry skin and troublesome itch. The presence of keratosis pilaris is a useful clinical clue to atopic dermatitis.2 Allergic contact dermatitis can be triggered by a range of products used in daily life, such as shampoos, washes, serums, toners and make-up. It might also be caused by intermittent exposure to products applied on other parts of the body that make contact with the face (eg nails and aerosolised fragrances). Irritant contact dermatitis is usually confined to sites of contact with the irritant (eg eyelids, peri-alar and angles of mouth).

Psoriasis presents with well-defined scaly plaques commonly affecting the frontal hairline, occipital scalp and the ears. It might be accompanied by plaques elsewhere on the body (eg elbows, knees, umbilicus and natal cleft). Actinic keratoses are asymptomatic scaly plaques on photo-exposed areas that develop gradually. Background sun damage is a common feature. Tinea faciei classically presents with an annular eruption with a leading edge, central clearing and peripheral scale. It is usually asymmetrically located. Topical steroid use on tinea faciei might lead to initial improvement, but it might be followed by rash with a less raised margin, less scale, more pustules and enlarged area of involvement (ie tinea incognito; Figure 3).

### Mixed rashes

Some conditions might have a combination of features that make them difficult to classify under one morphology. Sunburns vary in their morphology from erythema and peeling to blistering rashes following periods of extended ultraviolet (UV) exposure despite sunscreen use. Shingles might start

off with macular erythema in a dermatome followed by vesicles and/or crusting. On the face, shingles might occur in one or two contiguous dermatomes but do not cross the midline.<sup>22</sup> In the immunosuppressed patient, the rest of the body would need to be examined to exclude disseminated shingles. Allergic contact dermatitis occurs in areas of allergen exposure 24–72 hours following initial sensitisation.<sup>8</sup> It might present with erythema, oedema and possible blistering.

On the face, cutaneous lupus might manifest as either a 'malar' rash with a smooth, indurated erythematous plaque over the nose and cheeks (ie acute cutaneous lupus) or as discrete red and scaly plaques with follicular plugging (ie discoid lupus). Discoid lupus more commonly affects the scalp, ears and conchal bowl. On the face, dermatomyositis presents as a pink to violaceous eruption along with swelling of the eyelids and periocular skin.

Examination for other dermatological signs of dermatomyositis is warranted if it is suspected. Other systemic illnesses (eg pulmonary hypertension, liver failure) might present with facial erythema and should be suspected if there are systemic symptoms or abnormalities in non-dermatological examination (Table 1).

### **Clinical clues**

Once the differential diagnosis has been narrowed based on initial history and clinical morphology, further questions can be targeted at risk factors and associated symptoms (see 'clinical clues' in Table 1). Salient examples include the following:

- Associated flushing that is triggered by heat, alcohol or spicy food can be a clue to underlying rosacea.
- Prior topical or inhaled steroid use is a risk factor for periorificial dermatitis and steroid-induced rosacea.

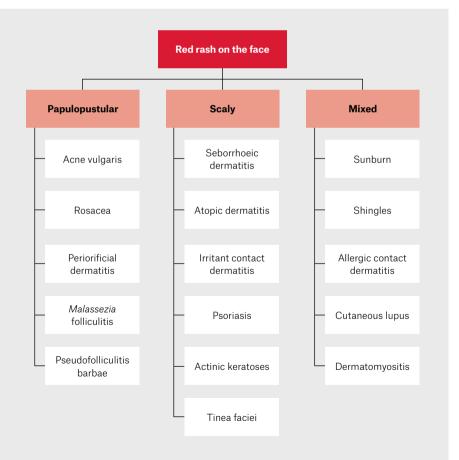


Figure 2. Causes of red facial rashes categorised by predominant morphology.



**Figure 3.** Tinea incognito on the face with more pustules, less scale and indistinct clinical borders.

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- The use of multiple cosmetic products can increase the risk of irritant and/or allergic contact dermatitis.
- Contact with animals and soils can be a risk factor for tinea faciei. Dermatophytes can also be transmitted via barbers.
- Rheumatological symptoms (eg joint aches, photosensitivity, chilblains, shortness of breath or chest pain) can be a clue for systemic lupus erythematosus. Muscle weakness might suggest dermatomyositis.

### Investigations

If the diagnosis is unclear after initial assessment, or if the eruption is not responding to initial treatment, investigations to help achieve a diagnosis can be considered, such as:

- swabs for bacterial microscopy and culture to look for infection
- skin scrapings for fungal microscopy and culture to look for evidence of tinea
- a swab for varicella zoster virus (VZV) polymerase chain reaction, which is recommended if shingles is suspected
- a skin biopsy, which is a useful tool, especially in cases where the diagnosis is not clear or referral to a dermatologist is being considered.

### Conclusion

A diagnosis for a red rash on the face can be reached with a thorough clinical assessment and the use of appropriate investigations when required.

### **Key points**

- A red rash on the face in adult patients is a common presentation to general practice.
- Assessment of symptoms, age of onset, rash morphology and 'clinical clues' can help achieve a clinical diagnosis in many cases.
- Investigations such as a skin biopsy are useful if the diagnosis is unclear, the rash is not responding to initial treatment and/or a referral to a dermatologist is being considered.

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Competing interests: None

Fundina: None

Provenance and peer review: Not commissioned, externally peer reviewed.

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#### **Acknowledgements**

The authors thank DermNet New Zealand (www.dermnetnz.org) for granting permission to use a clinical image in this article.

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