check

Independent learning program for GPs

Unit 593
May 2022

Dermatology

RACGP

www.racgp.org.au/check

Disclaimer

The information set out in this publication is current at the date of first publication and is intended for use as a guide of a general nature only and may or may not be relevant to particular patients or circumstances. Nor is this publication exhaustive of the subject matter. Persons implementing any recommendations contained in this publication must exercise their own independent skill or judgement or seek appropriate professional advice relevant to their own particular circumstances when so doing. Compliance with any recommendations cannot of itself guarantee discharge of the duty of care owed to patients and others coming into contact with the health professional and the premises from which the health professional operates.

Whilst the text is directed to health professionals possessing appropriate qualifications and skills in ascertaining and discharging their professional (including legal) duties, it is not to be regarded as clinical advice and, in particular, is no substitute for a full examination and consideration of medical history in reaching a diagnosis and treatment based on accepted clinical practices.

Accordingly, The Royal Australian College of General Practitioners Ltd (RACGP) and its employees and agents shall have no liability (including without limitation liability by reason of negligence) to any users of the information contained in this publication for any loss or damage (consequential or otherwise), cost or expense incurred or arising by reason of any person using or relying on the information contained in this publication and whether caused by reason of any error, negligent act, omission or misrepresentation in the information.

Subscriptions

For subscriptions and enquiries please call 1800 331 626 or email check@racgp.org.au

Published by

The Royal Australian College of General Practitioners Ltd
100 Wellington Parade
East Melbourne, Victoria 3002, Australia

Telephone 03 8699 0414
Facsimile 03 8699 0400
www.racgp.org.au

ABN 34 000 223 807
ISSN 0812-9630

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

check
Independent learning program for GPs

Dermatology

Unit 593 May 2022

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

RACGP

Topical Cream

EFUDIX®
fluorouracil

Refine your management of solar keratoses

Efudix removes 8 out of 10 SK/AK lesions[*1,2]*
while improving the signs of photoageing.[3,4]

*Treatment with 5% 5-fluorouracil reduced mean number of SK/AK lesions by 79.5% (range 59.2%-100%), systematic review of 13 RCTs (3 trials with histological, 6 trials with clinical and 4 trials not stating method of diagnosis) with variable treatment strength and length of therapy.[1,2]

The most frequently encountered reactions include pain, pruritus, hyperpigmentation, burning, crusting, allergic contact dermatitis, erosions, erythema, hyperpigmentation, irritation, photosensitivity, scarring, rash, soreness and ulceration at the site of application. Leukocytosis is the most frequent haematological adverse effect. Advise patients of the temporary unsightly appearance and local discomfort to be expected during treatment.[5]

SK/AK: solar/actinic keratosis RCT: randomised controlled trial

Download your healthcare professional resources at **treatphotodamage.com.au**

Please scan the QR code to review the Full Product Information (PI) before prescribing.
To have a copy of the PI sent to you, telephone 1800 630 056.

PBS Information: Efudix® is not listed on the PBS.
RPBS Information: Efudix® is listed on the RPBS. Refer to the PBS Schedule for full information.

References: 1. Askew DA, *et al. Int J Dermatol* 2009;48(5):453-63. **2.** Wu Y, *et al. Dermatol Ther* 2019;32(3):e12822. **3.** Lanoue J, *et al. Cutis* 2015;96(3):165-72,93. **4.** Sachs DL, *et al. Arch Dermatol* 2009;145(6):659-66. **5.** Efudix Product Information.

Level 10, 12 Help Street, Chatswood NSW 2067 Australia. Toll-free 1800 630 056. www.inovapharma.com. AU-2022-03-0060. Prepared April 2022.

inova pharmaceuticals

About this activity

Skin conditions are one of the most common presentations in Australian general practice, accounting for 17 out of every 100 patient encounters.[1]

In Australia, acne is seen by general practitioners (GPs) at a frequency of 0.4 of every 100 consultations.[1] Acne affects approximately 93.3% of teenagers aged 16–18 years, but it can also persist into adulthood, with data showing 64% of those aged 20–29 years and 43% of those aged 30–39 years affected.[2]

Vitiligo affects approximately 1–2% of the population. It can begin at any age, but 50% of cases commence prior to the age of 20 years.[3]

Psoriasis is thought to affect at least 2% of Australians; GPs are well placed to diagnose both psoriasis and common comorbidities such as psoriatic arthritis, metabolic syndrome and cardiovascular disease.[4]

Nail problems are common presentations to general practice; onychomycosis has a prevalence of up to 50% in people aged over 70 years,[5] while nail psoriasis presents as an isolated symptom in 5–10% of patients but affects half of patients with psoriasis.[6]

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

References

1. Britt H, Miller GC, Henderson J, et al. General practice activity in Australia 2015–16. General practice series no. 40. Sydney, NSW: Sydney University Press, 2016.
2. Brown H, Tapley A, van Driel ML, et al. Acne in primary care: A cross-sectional analysis. Aust J Gen Pract 2019;48(11):781–88. doi: 10.31128/AJGP-02-19-4864.
3. Vitiligo Association of Australia. The facts. Vitiligo Association of Australia, [date unknown]. Available at http://vitiligo.org.au/the-facts [Accessed 30 March 2022].
4. Daniel BS. The multiple comorbidities of psoriasis: The importance of a holistic approach. Aust J Gen Pract 2020;49(7):433–37. doi: 10.31128/AJGP-08-19-5035.
5. Kovitwanichkanont T, Chong AH. Superficial fungal infections. Aust J Gen Pract 2019;48(10):706–11. doi: 10.31128/AJGP-05-19-4930.
6. Tendais-Almeida J, Fátima Aguiar F, Torres T. Nail pitting and onycholysis. Aust Fam Physician 2016;45(3):120–21.

Learning outcomes

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

- outline the various treatment options available for acne, taking into account the stage of acne
- summarise the comorbidities that are associated with psoriasis
- describe the management of a patient presenting with signs of onychomycosis
- discuss the indicators of prognosis in vitiligo.

Authors

Case 1

Joseph Joseph BPharm (Hons I), MD, SCHP is a General Practice Registrar in Central and Western Sydney region, currently employed at Minto Family Medical Centre. Dr Joseph has a strong interest in skin diseases.

Saxon D Smith MBChB, MHL, PhD, GAICD, FAMA, IFAAD, FACD is a Specialist Dermatologist affiliated with The Dermatology and Skin Cancer Centre, St Leonards, NSW. He is also Clinical Professor at Australian National University College of Health and Medicine, ACT.

Case 2

Sapna Walles BMed, MD, MIPH (Hons), FRACGP is a General Practitioner based in Melbourne. She has special interests in dermatology and public health.

Case 3

Hilary Brown BMed, FRACGP is a Dermatology Registrar at Cairns Base Hospital.

Lisa Byrom BPhty, MBBS, MPhil, FACD is a Consultant Dermatologist who works in private practice in Brisbane and Rockhampton. She also is a Visiting Medical Officer at the Mater Hospital, Brisbane, and Townsville University Hospital. She has a special interest in adult-onset acne.

Case 4

Leila Asfour BSc, MBChB, MRCP(Derm) is a UK-trained Consultant Dermatologist, currently undertaking a hair fellowship at Sinclair Dermatology in Melbourne.

Rodney Sinclair MBBS, MD, FACD is a Professor of Dermatology at the University of Melbourne and Director of Sinclair Dermatology.

Peer reviewers

Joel Chan MBBS, BMedSci, MPH, DCH, FRACGP works as a General Practitioner in Prahran, Melbourne, and in the emergency department at the Royal Children's Hospital Melbourne. He has a role in Key Feature Problem (KFP) development for The Royal Australian College of General Practitioners' fellowship examinations and has special interests in paediatrics, mental health and dermatology.

Jeremy Hudson is Chair of The Royal Australian College of General Practitioners Dermatology Specific Interests, a Senior Lecturer in Dermatology and Skin Cancer Medicine and Surgery at James Cook University, and Clinical Director of the North Queensland Skin Centre, a research-based clinic in Townsville. He is a member of various advisory groups for clinical practice standards.

John Rowlands MBBS/BA, FRACGP is a General Practitioner in the Albany Skin Clinic at Pioneer Health Albany, Western Australia. He has a special interest in dermatology and skin cancer medicine.

Abbreviations

BMI	body mass index
BPO	benzoyl peroxide
CO_2	carbon dioxide
COCP	combined oral contraceptive pill
DHEA-S	dehydroepiandrosterone sulfate
DLQI	Dermatology Life Quality Index
GP	general practitioner
ICI	immune checkpoint inhibitor
IGA	Investigator Global Assessment

IgG1λ	immunoglobulin G1 lambda
JAK	Janus Kinase
MBS	Medicare Benefits Schedule
PBS	Pharmaceutical Benefits Scheme
PCOS	polycystic ovary syndrome
SAPHO	synovitis, acne, pustulosis, hyperostosis, osteitis
SCC	squamous cell carcinoma
UVB	ultraviolet type B

CASE

1 Sabina has a bothersome rash

Sabina, aged 54 years, presents with a generalised skin eruption that started two weeks ago. She has pruritic scaly lesions on her elbow, beneath her breasts, on her back and around her vagina. She works in a nursing home and is quite worried about having contracted an infectious skin disease. She has trouble sleeping because of the pruritus. Sabina is anxious about the cause of her rash.

Question 1

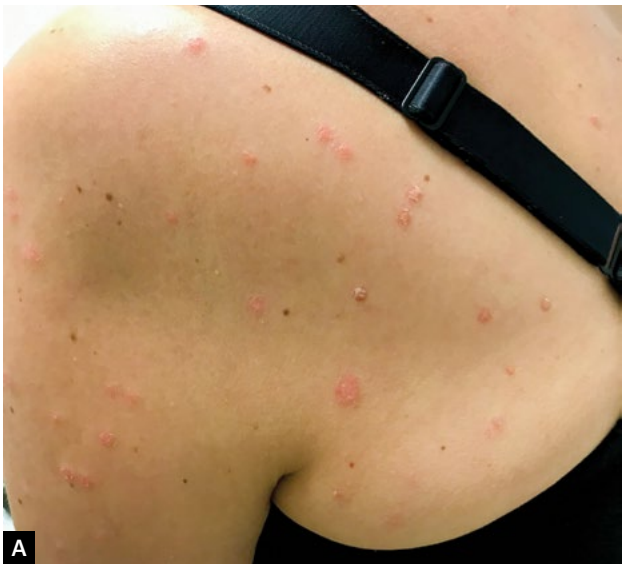
What further history and examination would be helpful when assessing Sabina?

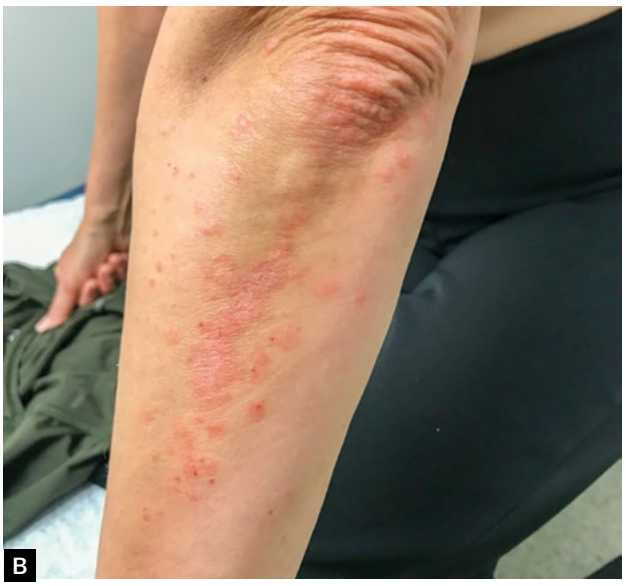
Further information

On further history-taking, Sabina says the eruption started quite suddenly with no obvious trigger. She has not had any recent illnesses. She thought she had scabies and tried two courses of topical permethrin on her whole body; this had no appreciable effect on the skin or pruritus.

Sabina has a history of rosacea and Crohn's disease, which is stable on azathioprine and mesalazine. There have been no new medications commenced recently. There is no family history of skin issues. She works as an administrator at the nursing home and does not have significant patient contact. She drinks four glasses of wine each night and does not smoke. She lives alone and has been quite stressed as a result of increasing workloads and the death of her husband last year.

On examination Sabina weighs 73 kg and is 156 cm tall (body mass index [BMI] 30 kg/m^2). She is afebrile, and there are scaly pink papules and plaques on her shoulder and back (Figure 1A) and extensor surface of the elbows (Figure 1B). She also has pink macules beneath her breasts (Figure 1C) and in the groin folds. There are no lesions between the fingers. There is subungual hyperkeratosis of her right toenail and a slightly chalky-yellow discolouration.





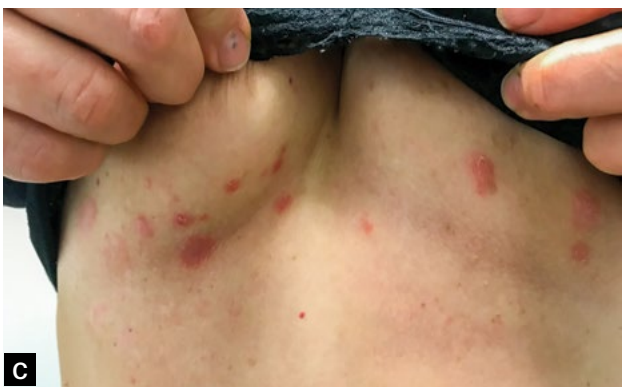


Figure 1. Photographs of Sabina's rash

A. Scaly pink macules on the left posterior shoulder and back.
B. Scaly pink plaque with macules and excoriations on the left elbow.
C. Erythematous macules beneath breasts.

Question 2

What are your differential diagnoses at this stage?

Question 3

What investigations can help differentiate these conditions?

Question 4

What is the likely diagnosis in this case?

Question 5

How is this condition managed?

Question 6

Is there any link between Sabina's stress and her condition?

Further information

Sabina returns for review in two months' time. Most of her psoriasis has cleared, and she only occasionally needs to treat the stubborn areas on her elbows. She would like to know what she can do to maintain and improve her skin health.

Question 7

What changes can Sabina make to improve her skin condition and prevent recurrence?

Question 8

Are there any other comorbidities to consider in this situation?

Further information

Sabina returns a few months later with a flare of her condition. She would like another prescription for the previous treatment, which worked well. Sabina is teary on review and remarks that she is worried about losing her job. She was asked by her supervisor to cover her skin when interacting with the families of residents. She has been feeling low, finds herself avoiding social situations and is becoming more reclusive. As a result, she has stopped exercising and now has a BMI of 31 kg/m^2.

Question 9

What else can be done for Sabina to help with her issues?

CASE 1 Answers

Answer 1

Sabina presents with a sudden-onset eruption of scaly skin lesions. The history should focus on determining potential causes of such an eruption, as well as her general medical and social history. This should include:

- symptoms – pruritus, pain, scalp involvement, nail issues, dyspareunia
- potential triggers – recent illnesses, new medications, new cosmetic products, new detergents, stress, pets and hobbies
- treatments already trialled
- medical history – current medications, allergies, history of atopy, history of skin issues
- social history – occupation and exposures, alcohol intake, smoking history, housing situation, contacts with similar eruptions
- family history – skin issues such as psoriasis, atopy.

The examination should include vital signs, weight, height and a general skin inspection including the nails and scalp.

Answer 2

The differentials for papulosquamous lesions are broad, and it is important to consider a variety of causes (Table 1).

Table 1. Differential diagnosis for papulosquamous lesions

Inflammatory	• Guttate psoriasis • Chronic plaque psoriasis • Pityriasis rosea • Discoid eczema • Atopic dermatitis • Lichen planus • Contact dermatitis
Infective	• Tinea corporis • Scabies
Malignant	• Mycosis fungoides
Iatrogenic	• Lichenoid drug eruptions

In Sabina's case, the most likely differentials would be:

- guttate/plaque psoriasis
- pityriasis rosea
- discoid eczema.

Answer 3

Skin scrapings can be taken for fungal microscopy, culture and sensitivity if a fungal aetiology is suspected. This is important because the treatment of fungal infections with topical corticosteroids can lead to progression of tinea incognita. Skin scrapings are taken from the edge of a rash using a scalpel blade at 90° to the skin and catching the sample in a dry urine specimen jar; a nail sample for fungal investigations could also be considered. Even when a positive result suggests fungal infection, other conditions that cause nail hyperkeratosis such as trauma, malignancy (squamous cell carcinoma of the nail) and psoriasis should be considered where improvement is not seen with treatment.

Histopathology can also be useful in some cases and can help differentiate inflammatory conditions such as dermatitis and psoriasis. In rare cases, dermatological malignancies such as mycosis fungoides can also be identified this way.

Answer 4

The most likely diagnosis in this case is chronic plaque psoriasis. This is an immune-mediated inflammatory disorder that affects 2% of the population and is characterised by pink scaly plaques that affect the extensor surfaces of the knees, elbows, sacrum and scalp.[1]

Answer 5

Treatment of psoriasis in primary care is based on *Therapeutic guidelines* recommendations, and options include topical tar, corticosteroids and calcipotriol.[2] General skincare advice should always be given, including use of soap-free washes and generous daily emollient application. Table 2 lists suggestions for product choice for psoriasis, treatment timeframes and length of treatments for specific areas. The amount of product used should be rationalised using fingertip units. A product squeezed across the distal phalanx of the finger should cover the area of an adult palm and roughly correlates to 0.5 g of product.

Tars

Compounded creams with coal tar solution and salicylic acid are commonly used for treatment; excoriated areas and inflamed skin should be avoided because of discomfort. Percentages recommended by practitioners vary, and *Therapeutic guidelines* suggest 6% coal tar solution/3% salicylic acid in an aqueous cream.[2]

Topical corticosteroids

Topical corticosteroids are potent anti-inflammatories with good effect in psoriasis. Higher-potency treatment is used for thicker plaques (betamethasone dipropionate 0.05%), and medium-potency treatment can be used for sensitive areas such as the face or skin folds (methylprednisolone aceponate 0.1%).

Calcipotriol

Calcipotriol is a vitamin D analogue that regulates proliferation and differentiation of keratinocytes. It is currently only available as a combination with betamethasone and can be implemented for long-term use (>2 months).

An appropriate regimen to commence for Sabina could be:

- betamethasone dipropionate 0.05% ointment plus calcipotriol 0.005% foam spray once daily to elbows and back until skin is clear (2–6 weeks)
- methylprednisolone aceponate 0.1% fatty ointment to the inframammary and groin areas until skin is clear (2–4 weeks).

Where treatment is ineffective or the patient has significant areas of involvement, they should be referred to a dermatologist or a general practitioner (GP) with a special interest in dermatology for further management. Dermatologist treatments include narrow-band ultraviolet B therapy, acitretin, apremilast, methotrexate, ciclosporin and biologic agents. To qualify for Medicare Benefits Schedule (MBS)–funded biologic therapy, the patient must have trialled and failed at least two of the aforementioned treatments.

Table 2. Initial treatment of psoriasis depending on location (adapted from *Therapeutic guidelines*[2])

Location of psoriasis	Recommended product	Length of use	Other notes
Scalp	Methylprednisolone aceponate 0.1% lotion Mometasone furoate 0.1% lotion once daily	2–6 weeks	Add coal tar–based shampoo after symptoms are controlled
Trunk and limbs	LPC 6% + salicylic acid 3% in aqueous cream twice daily	4 weeks	If the response is inadequate, change to betamethasone dipropionate 0.05% + calcipotriol 0.005% foam spray
	Methylprednisolone 0.1% fatty ointment once daily	2–6 weeks	
Palms and soles	LPC 6% + salicylic acid 3% in aqueous cream twice daily	4 weeks	Psoriasis affecting the palms or soles is hyperkeratotic and more resistant to topical therapy
	Calcipotriol + betamethasone 50 + 500 µg/g ointment daily	6 weeks	
Nails	Calcipotriol + betamethasone 50 + 500 µg/g ointment in proximal nail fold and under nail once daily at night	12 weeks	Topical therapy of the nails is not effective; systemic treatment is usually required
Flexural	Methylprednisolone 0.1% fatty ointment once daily LPC 2% in emulsifying ointment once daily	2–6 weeks	Introduce tar after initial flare settles due to potential irritation
Face	Methylprednisolone 0.1% fatty ointment once daily	2–6 weeks	Avoid calcipotriol use on the face

LPC, liquor picis carbonis, also known as coal tar solution

Answer 6

Psoriasis can be connected with psychosocial issues; stress can act both as an exacerbating factor for pre-existing disease and have a role in disease development. It is interesting to note that the brain and the skin both originate from the ectoderm during embryonic development, and this provides a possible explanation for this link.[3]

Answer 7

Lifestyle factors play a part in psoriasis, and counselling Sabina about these issues is an important part of the treatment of her condition.[4] Important advice includes:

- stress management techniques – give advice regarding deep breathing exercises when stressed and links to mindfulness techniques
- exercise – aim for 30 minutes of moderate-intensity exercise on most days of the week
- weight loss – aim for a modest 5% weight loss over three months using dietary changes and increased physical activity
- smoking cessation – for patients who smoke, a brief intervention involving opportunistic advice, discussion and encouragement to stop smoking would be beneficial
- reduced alcohol intake – limiting alcohol intake to two standard drinks each night with at least two alcohol-free days per week.

Although these therapies reduce flares, psoriasis is a chronic disease that relapses and remits, and the patient must be counselled to return for review if flares occur.

Answer 8

Psoriasis is considered a systemic inflammatory disease, and several potential comorbidities should be discussed.[5] Psoriatic arthritis is a potentially debilitating spondyloarthropathy that can predate the appearance of skin lesions. Patients with psoriasis should be assessed for joint pain, swollen digits (dactylitis), nail dystrophy and tendon inflammation (enthesitis).[6] Patients with suspected psoriatic arthritis should be promptly referred to a rheumatologist. Cardiovascular disease and metabolic syndrome are also closely linked with psoriasis. There are increased rates of myocardial infarction and stroke in patients with psoriasis, as well as increased cardiovascular risk factors such as hypertension, dyslipidaemia and obesity.[7] Assessment and optimisation of risk factors is essential to prevent the significant morbidity and mortality associated with cardiovascular disease. Inflammatory bowel diseases are more common in patients with psoriasis when compared with the general population.[8]

Depression and anxiety are common in patients with psoriasis, which is associated with several factors including reduced self-esteem, perceived or actual stigmatisation due to the skin lesions, social isolation and avoidance of intimacy. These factors play a significant part in reducing the patient's quality of life, and it is recommended that practitioners actively enquire about them.[9]

Answer 9

There are several things the GP can do to assist Sabina in this situation. She can be provided with a letter that clarifies her skin condition is not infectious. She should be screened for depression with a validated tool, such as the Depression, Anxiety and Stress Scale, and given psychoeducation regarding depression, anxiety and stress. A mental health treatment plan can be formulated with Sabina. Goals may include trying to deal with potentially unresolved grief from the loss of her husband, incorporating stress management techniques, improving mood and increasing physical activity. Treatments may include cognitive behavioural therapy with a psychologist, structured problem solving and exercise planning.

Psoriasis is a chronic disease expected to last longer than six months; therefore, the GP can also formulate a chronic disease management plan (GP Management Plan) and Team Care Arrangements. Through these, Sabina can access MBS-subsidised allied health visits including a dietitian and exercise physiologist, who can help her develop a comprehensive plan for weight loss. These can be done over several visits, and through such endeavours, the chances of her weight, mood and psoriasis improving are vastly increased.

References

1. Langley RG, Krueger GG, Griffiths CE. Psoriasis: Epidemiology, clinical features, and quality of life. Ann Rheum Dis 2005;64(Suppl 2):ii18–23;discussion ii24-25. doi: 10.1136/ard.2004.033217.
2. Expert Group for Dermatology. Dermatology: Psoriasis. In: eTG complete [Internet]. West Melbourne, Vic: Therapeutic Guidelines Limited, 2021.
3. Sathyanarayana Rao TS, Basavaraj KH, Das K. Psychosomatic paradigms in psoriasis: Psoriasis, stress and mental health. Indian J Psychiatry 2013;55(4):313–15. doi: 10.4103/0019-5545.120531.
4. Ko SH, Chi CC, Yeh ML, Wang SH, Tsai YS, Hsu MY. Lifestyle changes for treating psoriasis. Cochrane Database Syst Rev 2019;7(7):CD011972. doi: 10.1002/14651858.CD011972.pub2.
5. Kovitwanichkanont T, Chong AH, Foley P. Beyond skin deep: Addressing comorbidities in psoriasis. Med J Aust 2020;212(11):528–34. doi: 10.5694/mja2.50591.
6. Lenman M, Abraham S. Diagnosis and management of psoriatic arthropathy in primary care. Br J Gen Pract 2014;64(625):424–25. doi: 10.3399/bjgp14X681181.
7. Hu SC, Lan CE. Psoriasis and cardiovascular comorbidities: Focusing on severe vascular events, cardiovascular risk factors and implications for treatment. Int J Mol Sci 2017;18(10):2211. doi: 10.3390/ijms18102211.
8. Hohenberger M, Cardwell LA, Oussedik E, Feldman SR. Interleukin-17 inhibition: Role in psoriasis and inflammatory bowel disease. J Dermatolog Treat 2018;29(1):13–18. doi: 10.1080/09546634.2017.1329511.
9. Daniel BS. The multiple comorbidities of psoriasis: The importance of a holistic approach. Aust J Gen Pract 2020;49(7):433–37. doi: 10.31128/AJGP-08-19-5035.

CASE

2 Chad has ugly toenails

Chad, a Caucasian man aged 65 years, presents to you on insistence from his wife with concerns about his 'ugly toenails'. He has noticed some discolouration and distortion affecting the two great toenails for at least the past 12 months, and it seems to be getting worse. Chad has hypertension and takes ramipril 2.5 mg to treat this condition. He has no other known medical conditions and does not take any other medications. He rarely drinks alcohol, and he smokes occasionally at parties.

Question 1

What further history would you seek from Chad?

Further information

Chad tells you that the nail changes are restricted to the bilateral great toenails, with no involvement of fingernails. He has never had surgery to the toenails and does not recall any specific trauma. He has not noticed any specific skin rashes, although he has an uncle with psoriasis. He reports having tried some over-the-counter topical nail therapies a few months ago, but these have not provided any benefit.

Question 2

What differential diagnoses would you consider?

Question 3

What would you look for on examination?

Further information

Chad's blood pressure is 135/80 mmHg. Other vital signs are normal. On general examination, you do not find any features of systemic disease, including any skin rashes. Nail changes are restricted to the two great toenails, affecting less than 20% of each nail. You notice pitting, onycholysis and subungual hyperkeratosis bilaterally (Figure 1).

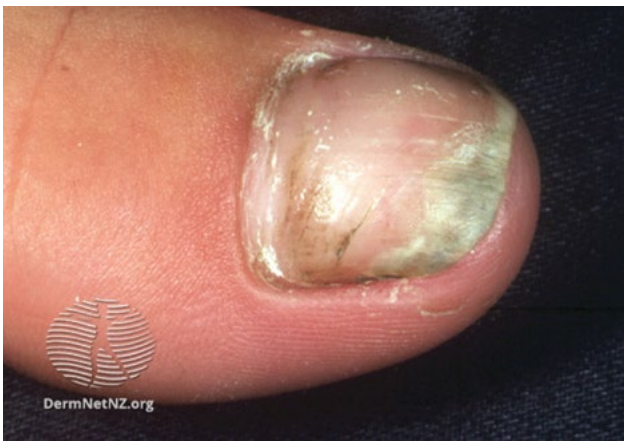


Figure 1. Chad's toenail
Image courtesy of DermNetNZ[9]

Question 4

What would be your provisional diagnosis for Chad's presentation?

Further information

Chad has recently completed routine blood tests including liver function tests, all of which were within reference ranges.

Question 5

What additional tests would you do now for further work-up of Chad's toenail problem?

Further information

Chad's fungal microscopy reveals evidence of *Trichophyton rubrum*. The results for culture samples are pending.

Question 6

How will you manage Chad now?

Further information

You follow up with Chad after 12 weeks and note that the onychomycosis has resolved nicely. He has since moved interstate.

The following year, Chad returns to you seeking a second opinion regarding some new nail problems. He reports that similar nail changes have occurred, but this time only the index finger on his right hand is affected. He has already tried another 12 weeks of treatment with terbinafine prescribed by another general practitioner, with no benefit.

A thorough history and examination reveal isolated nail changes on the right index fingernail. There is no history of trauma. This time, the nail appears to have a distinct yellow discolouration in the centre, with associated pitting, onycholysis and small black streaks visible across the nail surface (Figure 2). You also notice some white flakes across his shirt. Scalp examination reveals a small erythematous plaque with silvery scales at the periphery. The remainder of the examination is unremarkable.

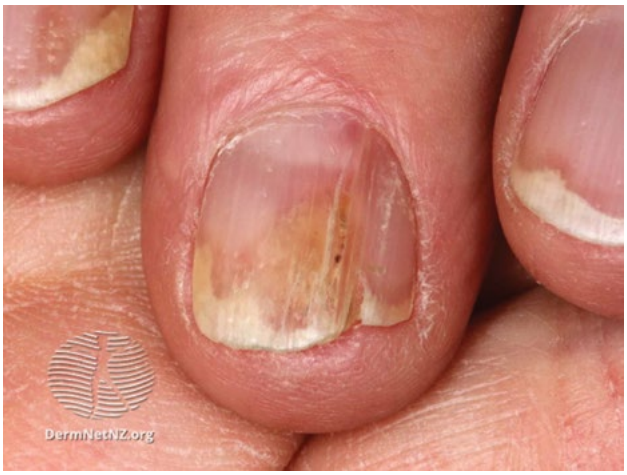


Figure 2. Chad's fingernail
Image courtesy of DermNetNZ[10]

Question 7

What is your provisional diagnosis?

Question 8

What is your approach to managing Chad's fingernail dystrophy?

CASE 2 Answers

Answer 1

A discoloured toenail is a vague description that may encompass a wide range of differential diagnoses, both local and systemic.[1]

Further history should include:

- distribution of nail changes, including any fingernails
- associated skin rashes on the body and scalp (consider psoriasis)
- history of nail trauma
- previous surgery on the affected nails
- any treatments tried to date, and when (recent antifungal use may affect fungal microscopy and culture results)
- family history of nail problems or other dermatological diagnoses
- previous history of nail issues
- occupation
- presence of pain – this is predictive of malignancy.

Answer 2

Possible differentials include onychomycosis, psoriatic nail changes, nail bed tumours, alopecia areata, lichen planus, connective tissue diseases, twenty-nail dystrophy, Darier disease, traumatic and iatrogenic (secondary to local nail surgery) nail changes.[1]

Answer 3

In addition to closely examining the affected toenails, it is important to assess for features of systemic conditions that may have caused Chad's nail changes.

Examine the skin (including scalp) and joints for rashes or inflammatory changes.

Determine the distribution of nail changes by examining all toenails and fingernails.

Carefully examine each nail plate and surrounding tissue, including nail folds and under the nail plate. The use of dermoscopy may help to better characterise the deformity as well as identify features of malignancy.[2]

Some common deformities and their associated conditions are listed in Table 1.[3]

Given Chad's family history of psoriasis, it would be recommended to look specifically for:

- typical psoriatic skin rashes (raised silvery-scaly plaques)
- joint inflammation, which may suggest psoriatic arthritis
- psoriatic nail changes (though these are not all specific to psoriasis): pitting, horizontal ridges, onycholysis, oil spots, subungual hyperkeratosis.[3]

Answer 4

The classic features of onycholysis, subungual hyperkeratosis and pitting, preferentially affecting the toenails, points to onychomycosis as the likely diagnosis in this case.[4]

Answer 5

It is essential to confirm the diagnosis of onychomycosis with positive microscopy and culture prior to commencing treatment.[3] Appropriate sampling is crucial, bearing in mind that infection may affect any part of the nail. Sampling may include scrapings of the nail plate, collection of subungual debris and punch biopsy.

False negatives may occur, with approximately 80% and 50% of positive cases being correctly detected by microscopy and

Table 1. Common nail signs and associated conditions[3]

Deformity	Description	Associated conditions
Pitting	Small nail plate depressions	Psoriasis, alopecia areata, reactive arthritis, eczema, twenty-nail dystrophy
Ridging	Horizontal or longitudinal lines	Horizontal (Beau) lines – severe systemic illnesses Longitudinal lines – lichen planus, rheumatoid arthritis, physiological, Darier disease
Leukonychia	White nail plate	Alopecia areata, Darier disease, trauma, psoriasis, fungal infections, malnutrition, cirrhosis, hyperthyroidism, diabetes
Oily spot	Yellow patch of discolouration	Psoriasis
Subungual hyperkeratosis	Thickening underneath nail plate	Onychomycosis, psoriasis, eczema, Darier disease, pityriasis rubra pilaris
Onycholysis	Separation of nail plate from nail bed	Onychomycosis, psoriasis, connective tissue disorders, reactive arthritis, trauma, hypothyroidism
Melanonychia	Linear brown-black discolouration of the nails	Physiological, especially in darker-skinned patients; melanoma

culture, respectively.[5] False negatives are more likely if antifungal medications have been used in the preceding month.[3] If a negative result occurs when onychomycosis is strongly suspected, it is recommended to take three samples a few weeks apart to confirm the diagnosis.[6]

It is important to note that the clinical features of onychomycosis overlap with other serious conditions, including nail bed malignancy. A positive fungal culture does not rule out a malignancy, as the two can co-exist.[2] Clinicians must be alert for features suspicious for squamous cell carcinoma (SCC), regardless of the presence of fungal growth.

Answer 6

Chad's toenail appearance is due to the presence of dermatophyte infection, and topical therapies are often ineffective.

If Chad chooses to treat the condition, he requires a systemic antifungal agent.[3] Oral terbinafine is the treatment of choice, at a dose of 250 mg daily for 12 weeks for toenails (six-week duration if only the fingernails are affected).[5] Other alternatives include oral itraconazole and fluconazole, although evidence for these agents is more limited.[5]

There is no requirement to routinely monitor liver function in healthy individuals without underlying hepatic or haematological conditions.[7]

It is important that Chad understands that recurrence rates may be as high as 50%.[6] Extensive infection may require multiple courses of treatment, with the nail appearance sometimes not returning to normal until the affected segment has completely grown out.[5]

The patient should be reviewed after one full course of treatment. If there is healthy nail emerging at the proximal nail fold, a second course of treatment should be given. If there is no evidence of new healthy nail growth, a referral to a dermatologist is recommended for consideration of alternatives including laser treatment.[5]

Answer 7

While onychomycosis is still a possibility, it does not typically affect fingernails in isolation (particularly without a history of preceding trauma). The lack of improvement with oral terbinafine for 12 weeks should also prompt the consideration of alternative diagnoses.

The yellow discolouration and black streaks are likely to be 'oil spots' and splinter haemorrhages, respectively. Both of these features are typical of nail psoriasis.[4] Though pitting and onycholysis may both be found in many other causes of nail dystrophy, the presence of the distinctive psoriatic skin rash on Chad's scalp points to psoriasis as the key provisional diagnosis in this case.

It is crucial to be alert for nail SCC as a potential diagnosis in isolated fingernail pathologies such as this. Nail SCCs tend to affect one fingernail (often the thumb) and are more common in males in the fifth decade of life.[2] A high index of suspicion, with careful history, dermoscopic examination and appropriate biopsy, if needed, will help to identify this frequently misdiagnosed condition.[2]

Answer 8

Nail psoriasis is very difficult to treat.[8] Although topical treatments with potent steroids and calcipotriol are available, they must be tried for at least three months and are often not successful.[8] Nail psoriasis may progress to involve further nails, as well as predisposing patients to concomitant fungal nail infections.[4] The psychological and functional impact of this cannot be underestimated. As such, the appropriate course of action would be to refer Chad to a specialist dermatologist for further assessment and consideration of systemic therapy.[4] Systemic therapies are associated with potentially serious side effects and are therefore not routinely used for patients with nail psoriasis in the absence of skin or joint involvement.[4] In this case, Chad has evidence of skin and nail psoriasis and therefore would be a good candidate for systemic therapy. Treatment options include cyclosporine, methotrexate and newer biologic agents.[4]

Conclusion

Chad receives a formal diagnosis from his dermatologist of psoriasis with skin and nail involvement. Chad's skin manifestations worsen over the year and eventually require biologic therapy with adalimumab. Both skin and nail psoriasis improve significantly following commencement of biologic therapy.

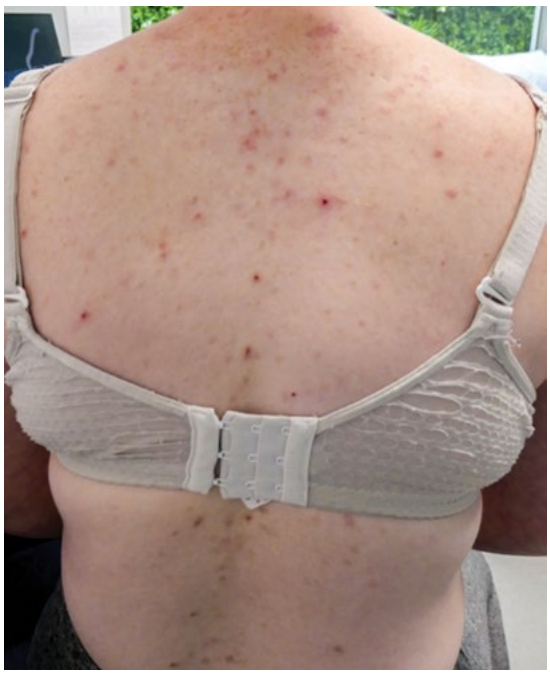
References

1. Peralta L, Morais P. Great toenail deformity – Case studies. Aust Fam Physician 2012;41(6):408–09.
2. Starace M, Alessandrini A, Dika E, Piraccini BM. Squamous cell carcinoma of the nail unit. Dermatol Pract Concept 2018;8(3):238–44. doi:10.5826/dpc.0803a17.
3. Grinzi P. Hair and nails. Aust Fam Physician 2011;40(7):476–84.
4. Tendais-Almeida J, Fátima Aguiar F, Torres T. Nail pitting and onycholysis. Aust Fam Physician 2016;45(3):120–21.
5. Expert Group for Dermatology. Nail disorders. In: eTG complete [internet]. West Melbourne, Vic: Therapeutic Guidelines Limited, 2019.
6. Pitney T, Muir J, Sun C. Trouble afoot: A review of common skin conditions of the feet and nails. Aust J Gen Pract 2020;49(5):257–60. doi: 10.31128/AJGP-11-19-5163.
7. Kovitwanichkanont T, Chong A. Superficial fungal infections. Aust J Gen Pract 2019;48(10):706–11. doi: 10.31128/AJGP-05-19-4930.
8. Expert Group for Dermatology. Dermatology: Psoriasis. In: eTG complete [Internet]. West Melbourne, Vic: Therapeutic Guidelines Limited, 2021.
9. Oakley A. Fungal nail infections. Hamilton, NZ: DermNet NZ, 2003. Available at https://dermnetnz.org/topics/fungal-nail-infections [Accessed 30 March 2022].
10. Ngan V, Oakley A. Nail psoriasis. Hamilton, NZ: DermNet NZ, 2003. Available at https://dermnetnz.org/topics/nail-psoriasis-images [Accessed 30 March 2022].

CASE

3 Xanthe has concerns about her acne

Xanthe, aged 30 years, presents with concerns about her acne (Figure 1). She reports mild acne as a teen, which became more severe in her early twenties when she ceased her contraceptive pill. Xanthe currently uses a 2% salicylic acid cleanser and gentle moisturiser. She took doxycycline for six months last year with only mild improvement.



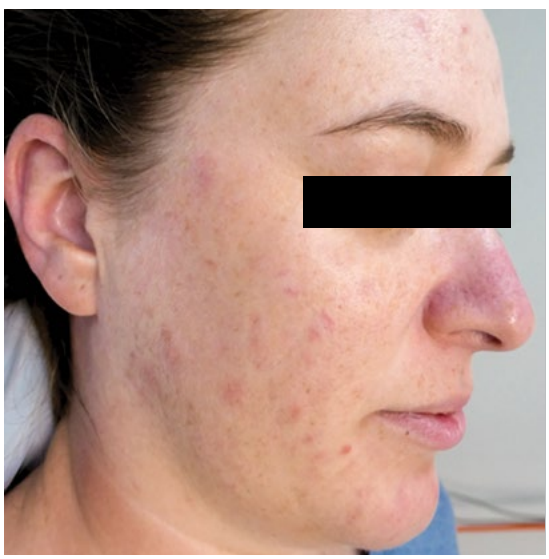


Figure 1. Xanthe's acne at the first consultation

Question 1

What else would you like to know about Xanthe's history?

Question 2

How would you assess acne severity?

Further information

Xanthe does not have any other medical problems. She does not take any other medications, supplements or protein shakes.

She recalls menarche at age 12 years with a regular cycle until starting the combined oral contraceptive pill (COCP) for contraception aged approximately 16 years. Xanthe stopped the COCP at age 23 years, and her cycle has since remained regular. She reports recent hair thinning on the scalp. There is no history of hirsutism. She reports recent intentional weight loss of 10 kilograms over three months, and her current body mass index (BMI) is 29 kg/m^2.

Xanthe works indoors and spends a lot of time outdoors horse-riding in her spare time. She is not in a sexual relationship and does not have any short-term plans to become pregnant.

She feels embarrassed by her acne, in particular when meeting new people.

On examination, Xanthe has open and closed comedones, papules and pustules. There are no cystic lesions. The acne is widespread and involves her face, chest and upper back.

Using the Investigator Global Assessment (IGA) scale, you determine that she has moderate acne (grade 3). There is some mild scarring and post-inflammatory hyperpigmentation.

Question 3

What investigations would you consider?

Question 4

What are the diagnostic criteria for polycystic ovary syndrome (PCOS)? What are the other causes of hyperandrogenism?

Question 5

What are the differential diagnoses for acne in adults?

Further information

Xanthe's blood test results are all within normal ranges. There are no features of PCOS on the ultrasound.

Question 6

What are the treatment options?

Question 7

What are the dietary considerations for acne?

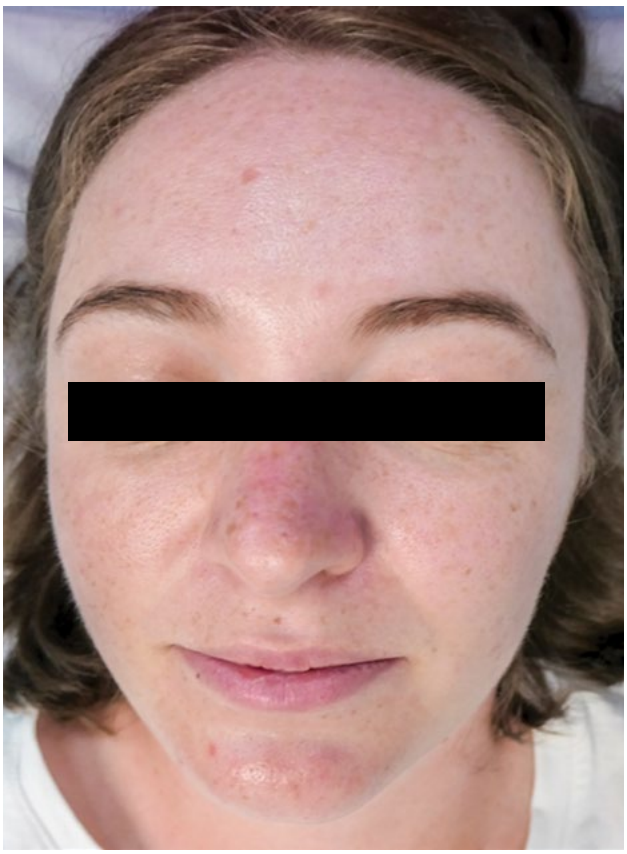
Further information

Xanthe commences oral erythromycin ethyl succinate 800 mg twice daily and topical trifarotene 0.005% lotion. Erythromycin is chosen in preference to a tetracycline antibiotic because of her outdoor lifestyle and previous poor response to doxycycline**.** Erythromycin does not interact with oral isotretinoin, which will allow for the two to be used concurrently if needed. A wash-out period is required between doxycycline and isotretinoin (five half-lives, which is approximately 10 days). Topical trifarotene is available in a pump-pack lotion and is approved for truncal acne.

You refer Xanthe to a dietitian to discuss her weight-loss goals and acne-specific dietary modification.

She returns for review after eight weeks and has considerable improvement of her acne. There is complete clearance on her chest and back with mild acne remaining on her face (Figure 2). However, Xanthe is keen to improve her skin further and you refer her to a dermatologist for additional treatment.

The dermatologist recommends isotretinoin, and this is commenced after Xanthe's fasting lipid profile is assessed, pregnancy is excluded and two forms of contraception are started (COCP and condoms).



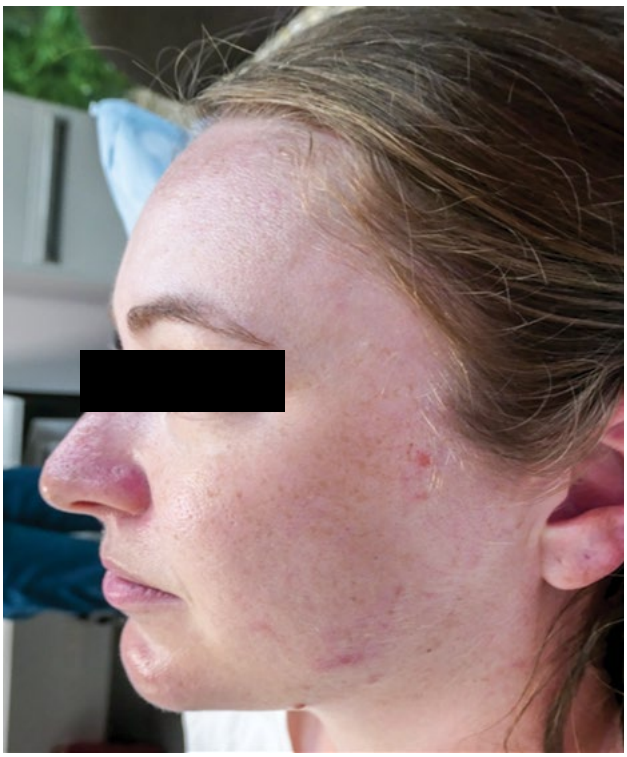


Figure 2. Xanthe's skin after eight weeks of treatment with erythromycin and topical trifarotene

Question 8

What are the common side effects of isotretinoin?

Question 9

What is the typical course of treatment for isotretinoin?

CASE 3 Answers

Answer 1

Information to ask about in the patient's history includes:

- How much does the acne bother her? Does it limit her activities? Does it affect her mood?
- What past and present acne treatments does she use? This includes over-the-counter, beautician-recommended and prescription treatments.
- Does she ever drink protein shakes or take anabolic steroids or other supplements?
- Does she have any signs and symptoms of PCOS or hyperandrogenism including menstrual irregularities, hair thinning, weight loss or gain, adult-onset acne, hirsuitism, history of miscarriage or difficulty falling pregnant?
- What other medical problems (including dermatological) does she have?
- Is there a family history of acne?
- What work and hobbies does she do? What is her social history? Include any features that may exacerbate acne or

may affect treatment options (eg working in hot environments, sun exposure, alcohol intake).

- Is she on any contraception? Is she sexually active? Does she have plans for pregnancy in the near future?
- What are her goals for treatment? For example, does she have an important social event for which she would like clear skin?
- Is she a smoker?
- Does she have any fevers, arthralgia or bone pain? This may suggest acne fulminans or synovitis, acne, pustulosis, hyperostosis, osteitis (SAPHO) syndrome requiring urgent dermatology referral.

Answer 2

There is no universally recognised method for categorising acne severity. However, grading acne as mild, moderate, severe or very severe (acne conglobata) is important as it guides treatment choice.

The IGA scale is recommended by the US Food and Drug Administration and considers lesion type and quantity (Table 1).

Table 1. Investigator Global Assessment scale

Grade	Clinical description
0	Clear skin with no inflammatory or noninflammatory lesions
1	Almost clear; rare noninflammatory lesions with more than one small inflammatory lesion
2	Mild severity; greater than grade 1; some noninflammatory lesions with no more than a few inflammatory lesions (papules/pustules only, no nodular lesions)
3	Moderate severity; greater than grade 2; up to many noninflammatory lesions and may have some inflammatory lesions, but no more than one small nodular lesion
4	Severe; greater than grade 3; up to many noninflammatory and inflammatory lesions, but no more than a few nodular lesions

Involvement of the chest and back and the presence of scarring and post-inflammatory hyperpigmentation should also be recorded.

The psychological impact of acne can be severe and is not always in proportion to the clinical appearance. Therefore, asking about and recording the impact acne has on the patient is important and affects treatment selection. You can ask the patient to complete a Dermatology Life Quality Index (DLQI).[1]

Answer 3

Further investigations are not always indicated.

Xanthe reports some hair loss and weight loss, which may be associated with hyperandrogenism; therefore, endocrinological assessment is warranted.

A typical hormone screen includes free and total testosterone, dehydroepiandrosterone sulfate (DHEA-S), androstenedione, luteinising hormone and follicle stimulating hormone. Thyroid stimulating hormone, fasting insulin, sex hormone–binding globulin, free 17β-hydroxysteroids, free androgen index, prolactin, oestrogen and progesterone may also be considered.[2] The hormone screen is best performed between days 3–5 of the menstrual cycle between 8.00 am and 10.00 am.

Pelvic ultrasonography to assess the ovaries for signs of PCOS may also be indicated.

A swab for microscopy and bacterial culture may be warranted if there are atypical features or the diagnosis is uncertain.

Answer 4

The diagnosis of PCOS requires two of the following three criteria to be met:

- androgen excess (clinical or biochemical)
- ovulatory dysfunction (oligo or anovulation)
- polycystic ovaries on ultrasonographic evaluation.

Other causes of hyperandrogenism can be classified as:

- ovarian
  - benign or malignant ovarian tumours
- adrenal gland disease
  - congenital adrenal hyperplasia
  - benign or malignant adrenal tumours
- pituitary gland disease
  - Cushing's syndrome
  - acromegaly
  - prolactinoma
- other
  - obesity
  - metabolic syndrome
  - medications.

Answer 5

Xanthe's presentation is very typical of adult acne and the diagnosis is clear.

Differential diagnoses include rosacea, periorificial dermatitis, folliculitis, pityrosporum folliculitis and gram-negative folliculitis.

Answer 6

Xanthe's acne is too severe and widespread to be effectively treated with topical agents alone.

Treatment options include:[3,4]

- medium-term (<6 months) antibiotic + topical treatments

- hormonal treatment +/– topical treatments
- isotretinoin (in Australia, this may only by prescribed by a dermatologist in all states and territories with the exception of Western Australia).[5]

Antibiotic treatment should be limited to 3–6 months' duration and be combined with topical benzoyl peroxide (BPO) and/or a retinoid to reduce the risk of antibiotic resistance. Medium-term oral antibiotic options include macrolides (erythromycin) and tetracyclines (doxycycline, minocycline). These antibiotics have both anti-inflammatory and antibiotic effects on acne. Trimethoprim/sulfamethoxazole, cephalosporins and penicillins may be used but are less effective for acne.

Adverse effects of antibiotics occasionally limit their use. Vaginal candidiasis and drug eruptions may occur with any antibiotic. Erythromycin is associated with gastrointestinal disturbance and rarely causes cardiac conduction abnormality and hepatotoxicity. Tetracyclines may cause photosensitivity and liver derangement. Doxycycline can cause gastrointestinal disturbance, in particular reflux. Minocycline is associated with tinnitus, dizziness and pigment deposition in the skin, particularly with longer courses.

There are many options for topical treatments, and the choice of treatment depends on vehicle, cost, side-effect profile and patient preference (Table 2).

Hormonal treatments include the COCP and spironolactone.

COCPs containing cyproterone acetate or drospirenone are most effective. Cyproterone acetate may also be prescribed without oestrogen. These medications improve acne by their anti-androgenic properties but can also be helpful for women without signs of hyperandrogenism. Improvements are slow and usually take three months to become apparent (if at all). COCP risks include venous thromboembolic events, cardiovascular disease and breast cancer. When a COCP is used for acne alone (rather than for contraception or menstrual irregularities), the risks of COCP use must be balanced against the risks of acne.

Table 2. Topical treatments for acne

Treatment	Considerations
Skin care	All skin care, make-up and sunscreen should be oil free and non-occlusive formulations.
Benzoyl peroxide (BPO)	BPO kills *Cutibacterium acnes* and is also mildly comedolytic. There is no resistance to BPO, and the addition of BPO to antibiotic regimens (oral and topical) improves results and reduces the risk of antibiotic resistance. BPO can cause skin irritation and bleaches linen. BPO is available over the counter in 2.5%, 5% and 10% creams, lotions and washes.
Topical antibiotics	Topical antibiotics (clindamycin, erythromycin) are thought to work by both antibacterial and anti-inflammatory mechanisms. Clindamycin is the preferred topical antibiotic for acne and is commonly prescribed as a clindamycin 1% lotion.
Topical retinoids (adapalene, tretinoin, trifarotene)	Retinoids are comedolytic and anti-inflammatory. Tretinoin may not be photostable so it should be applied at night. It can also be degraded by BPO, so the two agents should not be applied together. Topical retinoids should be avoided in pregnant women. Retinoids can cause skin irritation, and patients may need to commence treatment with alternate-day dosing. Retinoids are also photosensitising. Commonly prescribed topical retinoids include: • facial acne: adapalene 0.1%; tretinoin 0.025%, 0.05% or 0.1% • truncal acne: trifarotene 0.005%.
Azelaic acid	Azelaic acid is mildly comedolytic, antibacterial and anti-inflammatory. It also has a lightening effect so is particularly helpful in patients prone to post-inflammatory hyperpigmentation. Azelaic acid is commercially available as a 15% gel or 20% lotion.
Salicylic acid	Salicylic acid is comedolytic and may be helpful for acne, although research is lacking. Salicylic acid is available over the counter in 0.5–2% strengths as creams, lotions or washes.
Niacinamide	Topical niacinamide (nicotinamide, vitamin B3) may be helpful for acne, although research is lacking. Niacinamide is available over the counter in variable strengths and compositions.
Fixed-combination agents	Fixed-combination treatments of topical antibiotics and retinoids or BPO are available, which can make application easier. Commonly prescribed fixed combinations available in Australia include adapalene 0.1% + BPO 2.5%, clindamycin 1% + BPO 5% and clindamycin 1% + tretinoin 0.025%.
Blue LED light	The blue wavelength range of 405–20 nm is absorbed by the porphyrins produced by *C. acnes*. This kills *C. acnes*, thus reducing the bacteria causing acne.

Spironolactone is an aldosterone-receptor antagonist with anti-androgen activity. Common side effects include diuresis, menstrual irregularities, breast tenderness, breast enlargement, fatigue and headache. It can cause gynaecomastia in men and is classified as Category C for pregnancy. Spironolactone is particularly helpful for patients with hirsuitism.

Answer 7

Many patients are interested in the role of diet for acne management. There is emerging evidence that adherence to a low glycaemic-index diet is beneficial for acne.[6] There is some evidence that reducing dairy products (in particular skim milk) may be helpful.[7] Protein shakes (especially whey protein) may exacerbate acne.[8,9]

Answer 8

Isotretinoin is a potent teratogen and classified as Category X for pregnancy. Patients must be counselled about the risks of taking this medication before/during pregnancy, and most dermatologists recommend that patients adhere to two forms of contraception for the duration of treatment. Contraception should be continued for at least one ovulatory cycle after stopping isotretinoin.

GPs are best placed and trained to prescribe appropriate contraception for patients taking isotretinoin.

Dry lips, dry skin, dry eyes and dry nose leading to epistaxis are common and to be expected. These are managed with regular lip balm, moisturisers, lubricating eye drops and lubricating nose oils. Isotretinoin is photosensitising, and patients must use sun protection.

Other side effects include myalgia, headache, elevated cholesterol and triglycerides and hepatotoxicity. There is a rare risk of night-blindness. Patients should limit their alcohol intake.

Isotretinoin does not cause depression or suicidality and has been shown to improve mood overall.[10,11] However, given the high rate of depression in patients with skin disease, mood should be assessed throughout treatment.

Isotretinoin was thought to be associated with inflammatory bowel disease, but recent studies do not show any relationship.

Answer 9

Isotretinoin is usually prescribed at a low dose for 9–12 months. Current guidelines recommend isotretinoin is continued for a further 1–2 months after the skin is completely clear.

Isotretinoin is a very effective treatment for acne, with 70–80% of patients achieving long-term remission after a single course.[12] Risk factors for relapse include a macrocomedonal pattern of acne, smoking, younger age (<14 years) and older age (>25 years).[5]

Conclusion

After six months of treatment with isotretinoin, Xanthe's acne has improved significantly. Treatment will continue until 1–2 months after her skin is completely clear.

Resources for patients

- DermNet NZ – Acne vulgaris, https://dermnetnz.org/topics/acne-vulgaris
- The Australasian College of Dermatologists – Acne vulgaris, www.dermcoll.edu.au/atoz/acne-vulgaris

References

1. Finlay AY, Khan GK. Dermatology Life Quality Index (DLQI) – A simple practical measure for routine clinical use. Clin Exp Dermatol 1994;19(3):210–16. doi: 10.1111/j.1365-2230.1994.tb01167.x
2. Thiboutot DM, Dréno B, Abanmi A, et al. Practical management of acne for clinicians: An international consensus from the Global Alliance to Improve Outcomes in Acne. J Am Acad Dermatol 2018;78(2 Suppl 1):S1–S23.e1. doi: 10.1016/j.jaad.2017.09.078
3. Zaenglein AL, Pathy AL, Schlosser BJ, et al. Guidelines of care for the management of acne vulgaris. J Am Acad Dermatol 2016;74(5):945–73.e33. doi: 10.1016/j.jaad.2015.12.037
4. Expert Group for Dermatology. Acne. In: eTG complete [Internet]. West Melbourne, Vic: Therapeutic Guidelines Limited, 2015.
5. The Australasian College of Dermatologists. The Australasian College of Dermatologists Position Statement: Isotretinoin for treatment of acne. St Leonards, NSW: The Australasian College of Dermatologists, 2018. Available at www.dermcoll.edu.au/wp-content/uploads/ACD-Position-Statement-isotretinoin-updated-Oct-2018.pdf [Accessed 18 March 2022].
6. Burris J, Shikany JM, Rietkerk W, Woolf K. A low glycemic index and glycemic load diet decreases insulin-like growth factor-1 among adults with moderate and severe acne: A short-duration, 2-week randomized controlled trial. J Acad Nutr Diet 2018;118(10):1874–85. doi: 10.1016/j.jand.2018.02.009.
7. Aghasi M, Golzarand M, Shab-Bidar S, Aminianfar A, Omidian M, Taheri F. Dairy intake and acne development: A meta-analysis of observational studies. Clin Nutr 2019;38(3):1067–75. doi: 10.1016/j.clnu.2018.04.015.
8. Maarouf M, Platto JF, Shi VY. The role of nutrition in inflammatory pilosebaceous disorders: Implication of the skin-gut axis. Australas J Dermatol 2019;60(2):e90–e98. doi: 10.1111/ajd.12909.
9. Baldwin H, Tan J. Effects of diet on acne and its response to treatment. Am J Clin Dermatol 2021;22(1):55–65. doi: 10.1007/s40257-020-00542-y.
10. Oliveira JM, Sobreira G, Velosa J, Telles Correia D, Filipe P. Association of isotretinoin with depression and suicide: A review of current literature. J Cutan Med Surg 2018;22(1):58–64. doi: 10.1177/1203475417719052.
11. Rowe C, Spelman L, Oziemski M, et al. Isotretinoin and mental health in adolescents: Australian consensus. Australas J Dermatol 2014;55(2):162–67. doi: 10.1111/ajd.12117.
12. Rademaker M. Isotretinoin: Dose, duration and relapse. What does 30 years of usage tell us? Australas J Dermatol 2013;54(3):157–62. doi: 10.1111/j.1440-0960.2012.00947.x.

CASE

4 Tobias is worried about his skin changes

Tobias, a man aged 40 years, attends your general practice reporting whitening of the skin on his hands (Figure 1) and forearms that has become more prominent over the summer months. He works as a salesman. He is Fitzpatrick skin type 3.

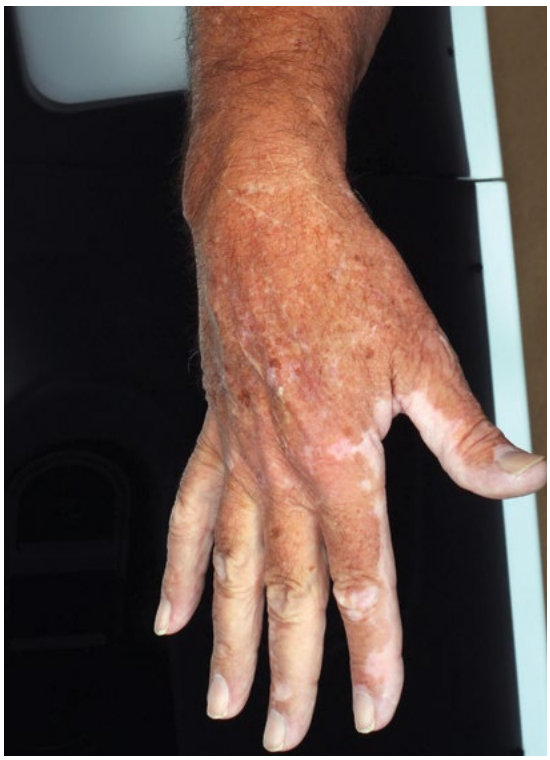


Figure 1. Appearance of Tobias's hand

Question 1

What key points from his history, examination and relevant symptoms would you specifically ask about?

Further information

Tobias recalls having an injury on his hand prior to the discolouration developing. He is distressed by his skin's appearance and wants to know if this will be permanent. He does not have any itch or pain from his skin. He does not recall any rashes.

Tobias has a recent diagnosis of coeliac disease and childhood atopic dermatitis. He has also been diagnosed with metastatic melanoma, which is responding to combination immunotherapy of nivolumab and pembrolizumab. He is not taking any other medication.

His mother has Hashimoto's thyroiditis, and his sister has a recent history of alopecia areata.

On examination, Tobias has distinct white patches bilaterally, extending distally from the metacarpal joints (Figure 1).

You notice very subtle areas of depigmentation around his mouth and eyelids. On closer inspection, his eyebrow appears depigmented, also known as leukotrichia (Figure 2). There is no mucosal involvement.

Figure 2. Leukotrichia on Tobias's eyebrow

Question 2

What key points in the history would aid the formulation of your primary diagnosis and exclusion of potential differentials?

Question 3

What tests would you need to perform?

Further information

When you examine Tobias, you ensure there is no evidence of scaling, which may require skin scraping for mycology to exclude tinea infection. You determine a biopsy is not necessary in this case as this is primarily a clinical diagnosis.

Question 4

What would you expect when using a Wood's lamp to evaluate Tobias's skin?

Further information

When using the Wood's lamp, you notice fluorescent blue-white colour on the areas affected on the skin. Tobias is distressed about his lesions as they affect visible parts of his body, and he feels this is contributing to his low mood. He has lost his confidence at work in view of his appearance.

Question 5

What is the final diagnosis for Tobias?

Question 6

What steps would you take in primary care to manage his case?

Further information

To assess the impact vitiligo is having on Tobias's emotional wellbeing, you ask him to fill in a Patient Health Questionnaire-9. Tobias's score is 19, indicating moderate-to-severe depression. Therefore, you refer him to a dermatologist and, in the interim, offer him topical tacrolimus 0.1% ointment twice daily for the facial involvement for three months. You recommend calcipotriol/betamethasone dipropionate ointment for his hand lesions applied once daily for three months. You also tell him about camouflage techniques and inform him of the available psychological therapies.

Question 7

What other medical or surgical treatments are available from a non-general practitioner specialist?

Question 8

What features in Tobias's history suggest poor prognostic outcomes with regards to his vitiligo, and what other comorbidities can be associated with vitiligo?

CASE 4 Answers

Answer 1

Tobias's clinical presentation and history are suggestive of vitiligo. Vitiligo can occur as a primary organ-specific autoimmune disease or be secondary to drugs, melanoma or a generalised autoimmunity (eg Vogt–Koyanagi–Harada syndrome).

It is important to explore if the skin whitening is likely to be hypopigmentation versus depigmentation. The following parameters in the history can help elucidate the diagnosis, exclude differentials and indicate severity of his condition:

- duration and progression of skin changes and history of relapse
- personal or family history of autoimmunity
- family history of vitiligo specifically
- medication that could contribute to pigmentary changes, including over-the-counter topical agents that contain corticosteroids
- occupation – how his skin could affect his work or any exposures to chemicals that can cause scarring/pigmentary changes
- travel history – any suggestion of infectious causes
- history of melanoma
- emotional stress, both as a causative factor and a consequence
- specific history screening for autoimmune conditions such as coeliac disease, thyroid disease, alopecia areata.

Answer 2

The key points from Tobias's history are that the lesions have developed following trauma, which indicates Koebner phenomenon, and his personal and family history of autoimmunity, which together point to a diagnosis of vitiligo. He does have a history of atopic dermatitis; therefore, there is a need to rule out any initial inflammation with pink/scaly skin and the development of subsequent post-inflammatory hypopigmentation. Infection is one of the differential diagnoses, with tinea versicolor being the most common mimicker. Finally, vitiligo can occur as an immune-related adverse effect in patients treated with immunotherapy (immune checkpoint inhibitor [ICI]) and is mainly described in patients with melanoma. Depigmentation can occur in up to 2–25% of patients with melanoma receiving ICI therapy, which is higher than the normal prevalence of vitiligo.[1]

Table 1 lists several differentials to consider in this clinical presentation.

Table 1. Differential diagnoses of vitiligo and hypopigmentation

Category	Conditions
Inherited/genetic-induced hypomelanoses	• Piebaldism • Tuberous sclerosis • Hypomelanosis of Ito • Waardenburg syndrome • Hermansky–Pudlak syndrome • Griscelli syndrome • Menkes syndrome • Nevus depigmentosus
Inflammatory/post-inflammatory hypomelanoses	• Atopic eczema • Psoriasis • Lichen planus • Pityriasis alba • Genital or extragenital lichen sclerosus
Neoplastic and paraneoplastic hypomelanoses	• Mycosis fungoides • Melanoma-associated depigmentation
Occupational or iatrogenic hypomelanoses	• Potent topical steroids • Imiquimod • Cryotherapy • Phenolic derivatives • Systemic medications (chloroquine, physostigmine, imatinib)
Post-traumatic leukoderma	• Deep burns • Scars
Infectious hypopigmentation	• Pityriasis versicolor • Leprosy • Leishmaniasis
Nevus depigmentosus	• Congenital

Answer 3

If the diagnosis is uncertain, a skin biopsy can help distinguish between vitiligo and other hypomelanoses. Melanocytes and epidermal pigment are absent in established vitiligo patches. In early or inflammatory vitiligo, a lymphocytic infiltration can be seen.

Vitiligo is associated with several autoimmune conditions. The incidence of thyroid disease is up to 52%, and 3–90% of patients with vitiligo have antithyroid antibodies. When compared with the general population, people with vitiligo are at increased risk of Graves' disease, Hashimoto's thyroiditis and thyroid cancer.[2] It is therefore recommended to consider testing for thyroid antibodies in patients with vitiligo.[2] No other blood tests are routinely indicated.

Given the associations with Addison's disease and type 1 diabetes, there should be a low threshold for testing for these conditions if the patient is unwell.

Answer 4

Wood's lamp examination is a diagnostic test that can help differentiate depigmented skin. A Wood's lamp emits black light, which can help identify the extent of pigmented or depigmented patches and to detect fluorescence on the examined skin or hair. While normal, healthy skin appears slightly blue, thickened skin appears as white spots. A Wood's lamp can be used to show areas of depigmentation (eg vitiligo) or reduced pigmentation (eg ash-leaf macules in tuberous sclerosis and hypomelanosis of Ito), where there is enhanced contrast between affected and unaffected skin. Hypopigmented skin has sharper borders under black light and fluoresces bright blue-white.[3]

Wood's lamp examination is commonly used in cases where it is unclear whether patches are hypopigmented versus depigmented, or in cases of inflammatory vitiligo where erythematous/pink border is noted.

Answer 5

In Tobias's case, there are several points in his history to suggest a diagnosis of vitiligo in the context of his familial autoimmunity. He may have developed melanoma-associated vitiligo in view of his age of onset, or he may have developed autoimmunity as a result of his checkpoint-inhibitor melanoma chemotherapy. Unfortunately, there is no test that can be done to attribute his vitiligo to a specific aetiology. The underlying mechanisms have been found to be the same in all types of vitiligo.[4]

Vitiligo has global prevalence of approximately 1%; however, estimates of prevalence range from <0.1% to >8%.[5] It can occur at any age. In 50% of people with vitiligo, onset is before the age of 20 years, and in approximately 80% of individuals it begins before the age of 30 years. In 20% of cases, other family members also have vitiligo. Males and females are equally affected. It occurs in all ethnicities and skin types. However, reporting may be higher in people with darker skin tones in view of the condition being more pronounced and having an associated stigma.[5]

Answer 6

There is currently no cure for vitiligo. There are a variety of treatments to help halt the disease or aim to improve repigmentation, especially when initiated early (and on the face and trunk). Options to disguise the discolouration include cosmetic dyes, stains and make-up. Sunscreen and adequate ultraviolet protection are vital as there is higher risk of sunburn in depigmented areas.

First-line treatment involves topical approaches such as potent and very potent topical corticosteroids or tacrolimus 0.1% ointment as an alternative. Table 2 outlines topical treatment regimens for vitiligo.[6] There is no topical therapy that is universally successful. Topical corticosteroids are more readily available on the Pharmaceutical Benefits Scheme (PBS). Pimecrolimus 1% cream is available on the PBS but more expensive than topical steroids. Tacrolimus ointment requires a compounding pharmacy and therefore would incur a higher cost.

Table 2. Topical therapies for vitiligo[6]

Treatment	Recommended regimen
Potent or very potent topical corticosteroid	**First-line treatment** for the **body and face**, **avoiding periocular area**, to be applied once daily.
Potent or very potent topical corticosteroid (+/– calcineurin inhibitor [tacrolimus 0.1%/ pimecrolimus 1% ointment])	An **intermittent regimen** of once-daily application of potent or very potent topical corticosteroids with or without topical calcineurin inhibitors (more evidence for tacrolimus), factoring in risks and benefits, especially in areas with thinner skin (eg the **periocular region, genital area and skin flexures**). Consideration of intermittent regimens: • one week of potent or very potent corticosteroids and at least one week off, or • one week of potent or very potent topical corticosteroids alternating with ≥1 week of topical calcineurin inhibitor.
Topical tacrolimus 0.1% ointment	Twice daily in people with facial vitiligo, especially periocular involvement, as an alternative to potent or very potent topical corticosteroids.
Topical tacrolimus 0.1% ointment	Twice daily under occlusion on photoexposed areas only in people with nonfacial vitiligo as an alternative to potent or very potent topical corticosteroids.
Topical vitamin D analogues	Insufficient evidence

Vitiligo can be linked to high levels of social anxiety.[7] Perceived stigma is significantly related to the extent to which vitiligo has an impact on social activities and distress.[8] The high levels of depression and anxiety associated with vitiligo can be successfully addressed with cognitive behavioural

therapy.[9] Patient Health Questionnaire-9 is a useful tool for screening for depression in the primary care setting.[10]

A referral to a dermatologist would be recommended for patients who have a poor response to topical treatments (unsatisfactory response after 3–6 months), progressive or extensive disease or associated severe psychosocial burden.

Answer 7

There is variable evidence for all current vitiligo therapies. Table 3 gives an overview of treatments. Table 4 gives an overview of surgical/physical therapies available.

Vitiligo therapeutics are currently rapidly expanding, with ongoing clinical trials on Janus Kinase (JAK) inhibitors as understanding increases about the associated pathways. Although JAK inhibitors are not yet part of standard practice, GPs are well placed to increase patient awareness of the promising landscape of therapeutics for vitiligo, as well as the potential option of trials.

There is a theoretical risk of skin cancer with ultraviolet type B phototherapy, especially when combined with topical tacrolimus.[11] In Tobias's case, he already has a history of melanoma. However, it is not an absolute contraindication, and phototherapy can be an effective treatment for vitiligo, so there should be a discussion of the risks and benefits of treatment.

Table 3. Overview of vitiligo treatment options

Conservative management	Cosmetic camouflage Supportive psychotherapy
Medical management	Topicals (as outlined in Table 2) Intralesional corticosteroids Systemic corticosteroids Oral steroid-sparing agents (insufficient evidence as monotherapy but can be effective in combination)
Physical therapies	Phototherapy Laser
Surgical interventions	Skin grafting Melanocyte suspension transplantation
Emerging therapies	Oral Janus Kinase inhibitors Topical Janus Kinase inhibitors Biologics: Tildrakizumab – human monoclonal immunoglobulin G1 lambda [IgG1λ] antibody (Figure 3)
Depigmentation in extensive and recalcitrant disease	Hydroquinone Monobenzyl ether of hydroquinone

Table 4. Vitiligo surgical/physical interventions in secondary care

Phototherapy[17]	Ultraviolet type B (UVB) phototherapy, for which the patient attends 2–3 times/week for an average of six months. Phototherapy works as an immunosuppressant and leads to direct stimulation of melanocyte proliferation. Risks: burns, melanoma and non-melanoma skin cancer Repigmentation rate: between 41.6% and 100% (Figure 4) Side effects: sunburn, blistering, dryness and photoageing
Laser	Monochromatic excimer laser (308 nm)[18] is a non-ablative laser. It is delivered 1–3 times/week for approximately 12 weeks. It is approved by the Food and Drug Administration and Therapeutic Goods Administration for vitiligo treatment. Risks: theoretical risk of melanoma and non-melanoma skin cancer Repigmentation rate: works well for localised vitiligo; superior efficacy when combined with topical tacrolimus Alternative: carbon dioxide (CO_2) laser[19] CO_2 laser in combination with 5-fluorouracil in adults with nonsegmental vitiligo on the hands and feet if other treatments have been ineffective[20]
Skin grafting Blister grafting[21] Split-thickness skin graft[22]	Surgery is suitable only for stable recalcitrant segmental and non-segmental vitiligo, and larger areas can be treated. Grafts are taken from sites that are cosmetically less important. Repigmentation rate: up to 90% when combined with adjuvant UVB
Melanocyte suspension transplantation[23]	Melanocyte suspension transplantation is an alternative technique. Cells are separated into a suspension of individual cells. The suspension is then transplanted onto the de-epithelialised recipient site. Melanocytes within the suspension repopulate the epidermis during wound healing and restore pigmentation to the areas of vitiligo. Repigmentation rate: 90–100% repigmentation in patients with stable localised vitiligo and 54% in those with generalised type

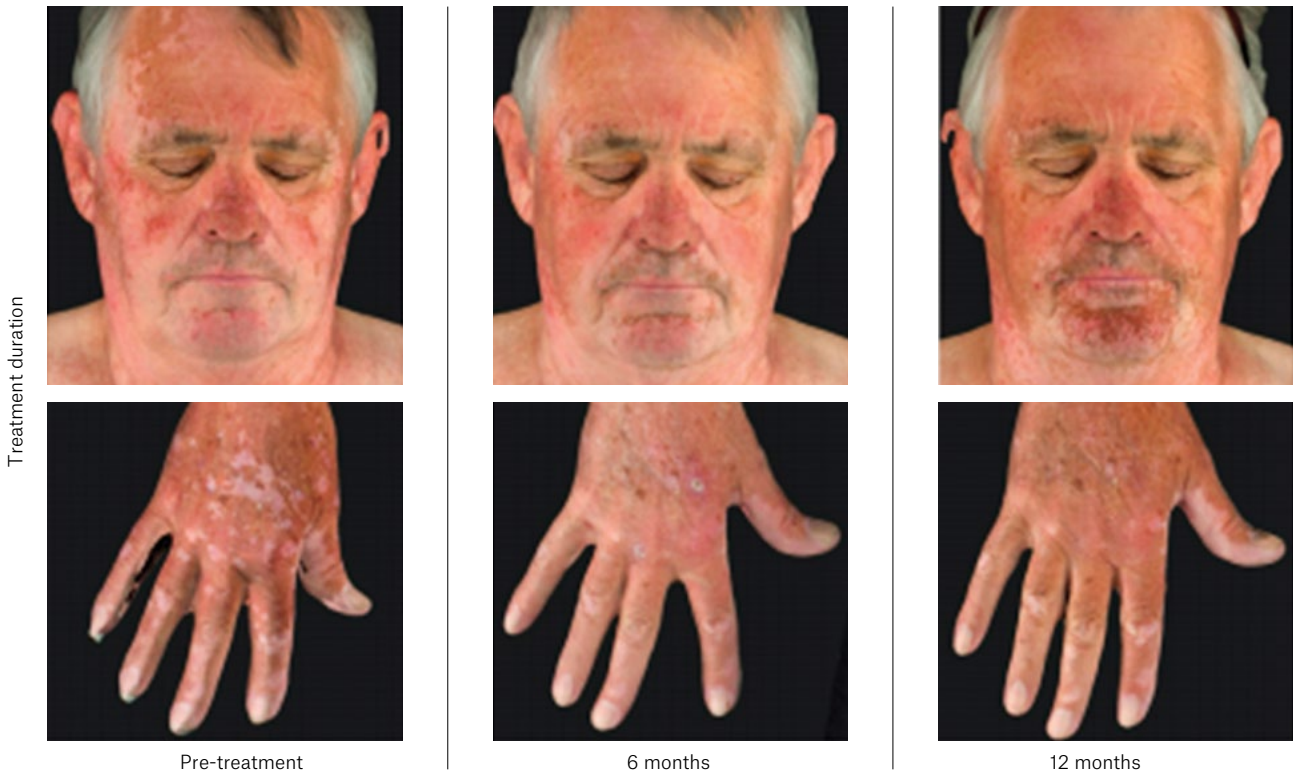


Figure 3. Treatment with tildrakizumab

Reproduced with permission of John Wiley & Sons, Inc from Jerjen R, Moodley A, Sinclair R, Repigmentation of acrofacial vitiligo with subcutaneous tildrakizumab, Australas J Dermatol 2020;61(4):e446–48.

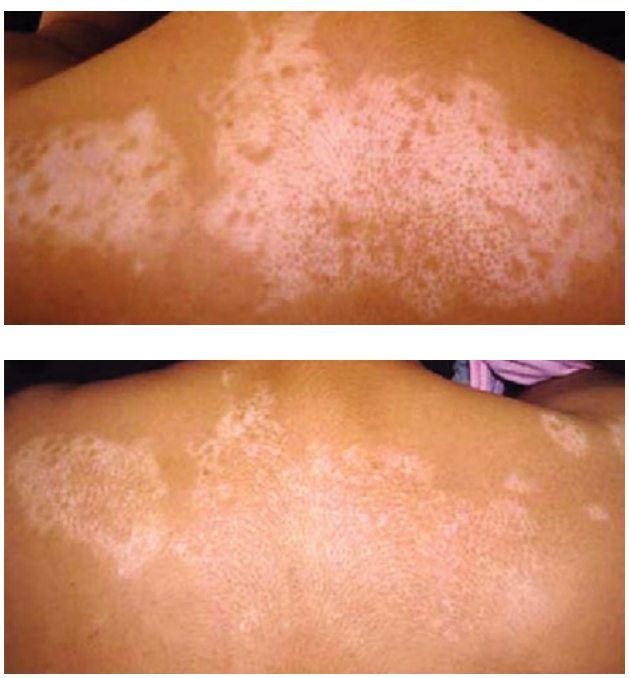


Figure 4. Examples showing before (upper) and after (lower) 60 sessions of ultraviolet type B phototherapy on the back

Answer 8

Tobias has depigmentation of his eyelashes (leukotrichia). Leukotrichia can affect between 10% and >60% of patients with vitiligo.[12] The presence of leukotrichia decreases the chance of repigmentation. This is thought to be secondary to the loss of the melanocyte stem cell reservoir in the epithelium of the folliculosebaceous unit.[12] Below are negative and positive prognostic factors related to vitiligo.[6]

The following are considered poor prognostic factors associated with vitiligo:

- mucosal involvement
- a family history of vitiligo
- koebnerisation – the appearance of new skin lesions on previously unaffected skin secondary to trauma
- nonsegmental vitiligo
- leukotrichia.

The following parameters are associated with better treatment outcomes:

- shorter duration of disease
- darker skin types

- lesions on the face, neck and trunk; these respond better than those on the distal extremities and those involving the mucosa.

It has been suggested that patients with vitiligo have a lower risk of developing melanoma and nonmelanoma skin cancers than the general population.[13,14] Vitiligo may be protective against skin cancer, potentially because of the genetic and autoimmune profile of patients with vitiligo or the fact that patients with vitiligo are vigilant with sun protection. There is a suggestion that patients with vitiligo may have a higher risk of thyroid and prostate cancer than the general population.[15,16]

Conclusion

Vitiligo is a chronic autoimmune condition with significant psychosocial morbidity that affects people of all ethnicities and ages. General practitioners have an important part to play in supporting patients with their diagnosis and reducing any associated stigma. They can offer first-line therapies and advise on conservative measures such as camouflage/psychological interventions. In recent years, there has been greater understanding of the underlying immune pathways related to the development of vitiligo, which has enabled the creation of new targeted therapies and an expansion of clinical trials in this field.

Resources for doctors

- DermNet NZ – Vitiligo, https://dermnetnz.org/topics/vitiligo
- British Association of Dermatologists – Vitiligo (patient information leaflet), www.skinhealthinfo.org.uk/condition/vitiligo

Resources for patients

- Vitiligo Association of Australia, https://vitiligo.org.au
- The Australasian College of Dermatologists, www.dermcoll.edu.au/atoz/vitiligo
- The Global Vitiligo Foundation, https://globalvitiligofoundation.org
- Vitiligo Friends, www.vitfriends.org
- Vitiligo Support International, https://vitiligosupport.org

References

1. Teulings HE, Limpens J, Jansen SN, et al. Vitiligo-like depigmentation in patients with stage III-IV melanoma receiving immunotherapy and its association with survival: A systematic review and meta-analysis. J Clin Oncol 2015;33(7):773–81. doi: 10.1200/JCO.2014.57.4756.
2. Yuan J, Sun C, Jiang S, et al. The prevalence of thyroid disorders in patients with vitiligo: A systematic review and meta-analysis. Front Endocrinol (Lausanne) 2019;9:803. doi: 10.3389/fendo.2018.00803.
3. Gupta LK, Singhi MK. Wood's lamp. Indian J Dermatol Venereol Leprol 2004;70(2):131–35.
4. Cohen BE, Manga P, Lin K, Elbuluk N. Vitiligo and melanoma-associated vitiligo: Understanding their similarities and differences. Am J Clin Dermatol 2020;21(5):669–80. doi: 10.1007/s40257-020-00524-0.
5. Alkhateeb A, Fain PR, Thody A, Bennett DC, Spritz RA. Epidemiology of vitiligo and associated autoimmune diseases in Caucasian probands and their families. Pigment Cell Res 2003;16(3):208–14. doi: 10.1034/j.1600-0749.2003.00032.x.
6. Eleftheriadou V, Atkar R, Batchelor J, et al; British Association of Dermatologists' Clinical Standards Unit. British Association of Dermatologists guidelines for the management of people with vitiligo 2021. Br J Dermatol 2022;186(1):18–29. doi: 10.1111/bjd.20596.
7. Shah R, Hunt J, Webb TL, Thompson AR. Starting to develop self-help for social anxiety associated with vitiligo: Using clinical significance to measure the potential effectiveness of enhanced psychological self-help. Br J Dermatol 2014;171(2):332–37. doi: 10.1111/bjd.12990.
8. Kent G. Correlates of perceived stigma in vitiligo. Psychol Health 1999;14:241–51.
9. Kussainova A, Kassym L, Akhmetova A, et al. Vitiligo and anxiety: A systematic review and meta-analysis. PLoS One 2020;15(11):e0241445. doi: 10.1371/journal.pone.0241445.
10. Kroenke K, Spitzer RL, Williams JB. The PHQ-9: Validity of a brief depression severity measure. J Gen Intern Med 2001;16(9):606–13. doi: 10.1046/j.1525-1497.2001.016009606.x.
11. Bae JM, Jung HM, Hong BY, et al. Phototherapy for vitiligo: A systematic review and meta-analysis. JAMA Dermatol 2017;153(7):666–74. doi: 10.1001/jamadermatol.2017.0002.
12. Gan EY, Cario-André M, Pain C, et al. Follicular vitiligo: A report of 8 cases. J Am Acad Dermatol 2016;74(6):1178–84.
13. Spritz RA. The genetics of generalized vitiligo: Autoimmune pathways and an inverse relationship with malignant melanoma. Genome Med 2010;2(10):78. doi: 10.1186/gm199.
14. Ban L, Labbouz S, Grindlay D, Batchelor JM, Ratib S. Risk of skin cancer in people with vitiligo: A systematic review and meta-analysis. Br J Dermatol 2018;179(4):971–72. doi: 10.1111/bjd.16703.
15. Vrijman C, Kroon MW, Limpens J, et al. The prevalence of thyroid disease in patients with vitiligo: a systematic review. Br J Dermatol 2012;167(6):1224–35.
16. Li CY, Dai YX, Chen YJ, et al. Cancer risks in vitiligo patients: A nationwide population-based study in Taiwan. Int J Environ Res Public Health 2018;15(9):1847. doi: 10.3390/ijerph15091847.
17. Hallaji Z, Ghiasi M, Eisazadeh A, Damavandi MR. Evaluation of the effect of disease duration in generalized vitiligo on its clinical response to narrowband ultraviolet B phototherapy. Photodermatol Photoimmunol Photomed 2012;28(3):115–19.
18. Bae JM, Hong BY, Lee JH, Lee JH, Kim GM. The efficacy of 308-nm excimer laser/light (EL) and topical agent combination therapy versus EL monotherapy for vitiligo: A systematic review and meta-analysis of randomized controlled trials (RCTs). J Am Acad Dermatol 2016;74(5):907–15. doi: 10.1016/j.jaad.2015.11.044.
19. Tawfik YM, Abd Elazim NE, Abdel-Motaleb AA, Mohammed RAA, Tohamy AMA. The effect of NB-UVB on noncultured melanocyte and keratinocyte transplantation in treatment of generalized vitiligo using two different donor-to-recipient ratios. J Cosmet Dermatol 2019;18(2):638–46. doi: 10.1111/jocd.12759.
20. Mohamed HA, Mohammed GF, Gomaa AH, Eyada MM. Carbon dioxide laser plus topical 5-fluorouracil: a new combination therapeutic modality for acral vitiligo. J Cosmet Laser Ther 2015;17(4):216–23. doi: 10.3109/14764172.2014.1003241.
21. Ashique KT, Kaliyadan F. When half-slide can deliver double efficiency for blister grafting in vitiligo. J Am Acad Dermatol 2020;83(3):e181–82. doi: 10.1016/j.jaad.2019.04.056.
22. Razmi T M, Afra TP, Parsad D. Vitiligo surgery: A journey from tissues via cells to the stems! Exp Dermatol 2019;28(6):690–94. doi: 10.1111/exd.13807.
23. Huggins RH, Henderson MD, Mulekar SV, et al. Melanocyte-keratinocyte transplantation procedure in the treatment of vitiligo: The experience of an academic medical center in the United States. J Am Acad Dermatol 2012;66(5):785–93.

ACTIVITY ID **392238**

Dermatology

This unit of *check* is approved for two CPD hours per case study in the RACGP CPD Program; this comprises one hour of educational activity and one hour of reviewing performance. The expected time to complete this activity is two hours per case study 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* 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* link from the drop-down
- completing the online evaluation form.

You can only qualify for CPD hours 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 – Keegan

Keegan, aged 85 years, is a patient who was seen by a colleague at your practice a few weeks ago for dystrophic toenail changes. The fungal microscopy and culture were both negative. Keegan has had onychomycosis several times before and has no red flag features pointing to another systemic diagnosis. His nail changes seem consistent with another episode of onychomycosis. Keegan tells you he has tried some topical nail therapies with no benefit.

Question 1

Which one of the following would be your next step in management?

A. Refer Keegan to a dermatologist.

B. Advise Keegan that the negative microscopy and culture results mean he does not need treatment this time.

C. Start methotrexate as Keegan may have psoriasis.

D. Take further nail samples a few weeks apart before commencing treatment.

Case 2 – Keri

Keri, aged 35 years, is a ballet dancer who presents with bilateral great toenail deformities. She has no significant family or personal medical history. She has never been treated for onychomycosis previously. Microscopy of nail clippings is positive for *Trichophyton rubrum*.

Question 2

Which one of the following would be your first line of treatment?

A. Over-the-counter topical nail paint

B. Oral terbinafine 250 mg daily for six weeks

C. Oral terbinafine 250 mg daily for 12 weeks

D. Oral fluconazole

Case 3 – Huseyin

Huseyin, aged 45 years, is an accountant who presents with a 'funny spot' on his left index finger. You see a distinct yellow discolouration in the centre of his fingernail and recognise this to be an oily spot, also known as a salmon patch.

Question 3

Which one of the following conditions would you be concerned about?

A. Psoriasis

B. Onychomycosis

C. Lichen planus

D. Darier disease

Case 4 – Uzair

Uzair, aged 40 years, attends your surgery reporting discolouration of the skin on his hands and forearms that has become more prominent during the summer months. He recalls having an injury on his hand prior to the discolouration developing. He is distressed by the appearance of his hands.

Question 4

Which one of the following autoimmune conditions is most commonly associated with vitiligo?

A. Alopecia areata

B. Pernicious anaemia

C. Thyroid disease

D. Atopic dermatitis

Question 5

Which one of the following is considered an adverse prognostic indicator in cases of vitiligo?

A. Depigmentation of the hair shaft (leukotrichia)

B. Younger age

C. Facial lesions

D. Darker skin types

Question 6

Which one of the following statements is **true** regarding the epidemiology of vitiligo?

A. Vitiligo is more common in children aged under 12 years.

B. Vitiligo is more common in men.

C. The majority of cases present before the age of 30 years.

D. Vitiligo only happens in darker skin types.

Case 5 – Aline

Aline, aged 35 years, comes to see you with concerns about acne. She has had acne since her teen years, but it has become worse since her last pregnancy at age 32 years. She has mild-to-moderate comedonal and pustular acne over her face. There is no acne on her chest or back.

Question 7

Which one of the following is a treatment option for mild-to-moderate acne?

A. Cephalexin 500 mg four times daily for 10 days

B. Triclosan wash, regular moisturiser and sun protection

C. Oral doxycycline 100 mg daily as a sole agent

D. Fixed combination treatment with clindamycin 1% + tretinoin 0.025%

Question 8

Which one of the following is a hormonal treatment for acne?

A. Finasteride 1 mg daily

B. Combined oral contraceptive pill (COCP) containing cyproterone acetate or drosperinone

C. Depot medroxyprogesterone acetate every 12 weeks

D. Erythromycin ethyl succinate 800 mg twice daily

Case 6 – Alasdair

Alasdair, aged 25 years, is a man with chronic plaque psoriasis who presents with an exacerbation of his condition. He has been in considerable distress due to personal issues with his partner. He has lesions around the groin folds that are consistent with his usual psoriasis plaques.

Question 9

Which one of the following would be an appropriate treatment option for Alasdair?

A. Methylprednisolone aceponate 0.1% fatty ointment to the area for 2–4 weeks

B. Betamethasone dipropionate 0.05% in optimised vehicle to the area for six weeks

C. Hydrocortisone 1% ointment to the area for four weeks

D. Coal tar solution 6% with salicylic acid 3% to the area for four weeks

Further information

Alasdair comments that he has been having problems with his joints. He has pain and stiffness in the mornings, which slowly improves throughout the day. You are concerned he has comorbid psoriatic arthritis.

Question 10

Which one of the following physical examination findings would most support this diagnosis?

A. Single joint involvement

B. Erythema nodosum

C. Keratoderma blennorrhagica

D. Dactylitis of the second digit

Refine your management of sBCCs

Cream, 5%
Aldara®
(IMIQUIMOD)

Aldara removes 8 out of 10 sBCC lesions[1] with good to excellent cosmetic outcomes.[2,3]*

*Treatment with imiquimod cream 5 times per week for 6 weeks resulted in a histological clearance rate of 82% (95% CI: 76-87%) and complete clearance rate of 75% (95% CI: 68-81%) for patients (n=185) with histologically confirmed sBCC.[1]

In trials with 5x per week dosing, 58% of patients experienced at least one adverse event. The most frequently reported adverse events are application site disorders (28.1%). Some systemic adverse reactions, including back pain (1.1%) and influenza-like symptoms (0.5%) were reported.[4]

sBCC: superficial basal cell carcinoma CI: confidence interval

Download your healthcare professional resources at **treatphotodamage.com.au**

Please scan the QR code to review the Full Product Information (PI) before prescribing.
To have a copy of the PI sent to you, telephone 1800 630 056.

PBS Information: Aldara® sachets and pumps. Authority required for the treatment of superficial basal cell carcinoma. Aldara® sachets and pumps are not listed on the PBS for solar keratosis or external genital warts. **RPBS Information:** Aldara® sachets and pumps. Authority required for the treatment of solar keratosis. Aldara® sachets. Authority required for the treatment of superficial basal cell carcinoma. Aldara® sachets are not listed on the RPBS for external genital warts. Aldara® pumps are not listed on the RPBS for external genital warts and superficial basal cell carcinoma. Refer to PBS & RPBS Schedules for full authority information.

References: 1. Geisse J, *et al. J Am Acad Dermatol* 2004;50(5):722-33. 2. Bath-Hextall F, *et al. Lancet Oncol* 2014;15(1):96-105. 3. Jansen MHE, *et al. J Am Acad Dermatol* 2018;79(5):961-64. 4. Aldara Product Information.

inova pharmaceuticals

check

Independent learning program for GPs