DECEMBER 2021 VOL 5 NO 2

A MEDICINE TODAY PUBLICATION

Dermatology Collection

PEER REVIEWED UPDATES FOR MEDICAL PRACTITIONERS

Reprints in Dermatology

Keratinocyte skin cancers: diagnosis and current management

Discoid eczema – more than just dermatitis

Common skin problems in children. Infectious rashes and infestations

Common skin problems in children. Birthmarks

Folliculitis: diagnosis and management of subtypes

An acute generalised blistering eruption

Persistent pruritic purple papules

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A MEDICINE TODAY PUBLICATION

Dermatology Collection

PEER REVIEWED UPDATES FOR MEDICAL PRACTITIONERS

FOREWORD FROM THE EDITOR-IN-CHIEF, DERMATOLOGY COLLECTION

Our latest issue of *Dermatology Collection* comprises a selection of dermatology articles we consider among the most important published in *Medicine Today* since 2020.

Australia has one of the highest rates of skin cancer in the world, with keratinocyte cancers being the most common type. Our lead article highlights key changes in Cancer Council Australia's 2019 updated guidelines on diagnosing and managing these cancers.

Discoid, or nummular, eczema is a chronic form of dermatitis characterised by well-defined circular plaques. Read about its clinical variants, differential diagnoses and management.

Two articles focus on paediatric dermatological conditions. The most common types of birthmarks are reviewed. These can range from the small and harmless to those that are large, causing cosmetic or functional impairment, or those indicating an underlying abnormality or malignancy. And read about childhood infectious rashes and infestations and the key to their diagnosis and treatment-decision making.

Folliculitis is another condition that can be infectious, with numerous subtypes that vary in aetiology, presentation and management. Besides infection, other triggers of folliculitis include trauma, medications and underlying disease.

Finally, test your diagnostic skills in a case of an acute generalised blistering eruption and a case of persistent pruritic purple papules first appearing on a woman's back and hands. What are the diagnoses?

Gayle Fischer OAM, MB BS, MD, FACD
Professor of Dermatology at Sydney Medical School – Northern, The University of Sydney, Royal North Shore Hospital, Sydney, NSW.

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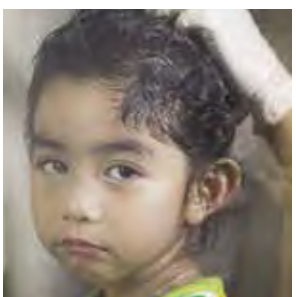

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Keratinocyte skin cancers

Diagnosis and current management

KEVIN PHAN MD
STEPHEN SHUMACK OAM, MB BS(Hons), FACD

Keratinocyte cancers, formerly known as nonmelanoma skin cancers, are an important cause of morbidity and mortality in Australia. In 2019, Cancer Council Australia published updated guidelines on diagnosis and management of keratinocyte cancers. This summary for GPs incorporates the latest update.

KEY POINTS

- Australia has one of the highest rates of skin cancer in the world, with keratinocyte cancer (formerly termed 'nonmelanoma skin cancer') the most common type.
- GPs play a crucial role in the detection and early management of keratinocyte cancers.
- Cutaneous squamous cell carcinoma (SCC) can metastasise; for patients with SCC unsuitable for surgery, treatment options include radiation therapy, chemoradiation, epidermal growth factor receptor (EGFR) inhibitors and immunotherapy.
- Basal cell carcinoma (BCC) is the most common keratinocyte cancer in Australia; hedgehog pathway inhibitors are a recent treatment option for patients with advanced metastatic BCC.
- Patients with keratinocyte cancers with poor prognostic features or locoregional spread should be referred to a specialist multidisciplinary team for planning and management.

Australia has one of the highest rates of skin cancer in the world, with keratinocyte cancers being the most common type. Keratinocyte cancers, formerly known as nonmelanoma skin cancers, comprise basal cell carcinoma (BCC) and cutaneous squamous cell carcinoma (SCC). Actinic keratosis is a precancerous lesion that can develop into SCC. Keratinocyte cancers cause about 560 deaths annually in Australia and accounted for over 939,000 treatments in 2015 alone.[1,2] Furthermore, keratinocyte cancers are responsible for a disproportionately high financial cost to the healthcare system, representing 8% of healthcare spending on cancer in 2008-09 and over $700 million in reimbursements for diagnosis, investigations and treatment annually.[1,2]

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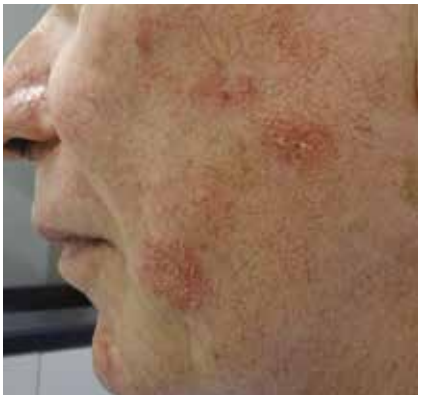
1. CHANGES IN THE 2019 UPDATED CANCER COUNCIL AUSTRALIA GUIDELINES ON MANAGEMENT OF KERATINOCYTE CANCERS[4]

- The term 'keratinocyte cancer (KC)' is now preferred to 'nonmelanoma skin cancer (NMSC)'
  - 'NMSC' can be ambiguous and includes several other malignant skin conditions, such as Merkel cell carcinoma and dermatofibrosarcoma protuberans
- Advances in radiation therapy mean the following patients should be referred to a radiation oncologist for an opinion:
  - patients not suitable for surgery (because of risk, preference, cosmesis, function)
  - primary treatment of stage T3 or T4 KC lesions and recurrent or persistent lesions
  - postsurgical treatment of high-risk KC lesions, including T3 or T4 stage lesions, invasion of more than 6 mm depth, inadequate margins on excision where further surgery is problematic, regional involvement, high-risk features
- Patients with locoregional advanced KCs should be managed by a multidisciplinary team (surgeons, dermatologists, radiation and medical oncologists):
  - platinum-based chemoradiation therapy is an option for unresectable cutaneous SCC
  - cetuximab (EGFR inhibitor) is approved for treatment of cutaneous SCC of the head and neck and can be offered in the context of radiation therapy for locally advanced disease or platinum-based chemotherapy for recurrent or metastatic disease
  - cemiplimab (PD1 inhibitor) treatment is TGA approved and is an option for treating cutaneous SCC if it is not amenable to surgery or chemoradiation therapy
  - hedgehog pathway inhibitors (sonidegib and vismodegib) are an option for treating BCC not amenable to surgery or radiation therapy

Abbreviations: SCC = squamous cell carcinoma; EGFR = epidermal growth factor receptor.



Figures 1a and b. Actinic keratoses. a (left). On the left cheek. b (above). On the forearm.

GPs have an important role in the prevention, early detection and management of keratinocyte cancers in Australia. Skin consultations account for 14.8% of GP consultations, and skin cancers are the second most common reason for specialist referral.[3] GPs are also well positioned to educate patients on the importance of sun-safe behaviours and to detect skin cancers at the earliest opportunity through full skin checks and opportunistic skin screening.

In 2019, Cancer Council Australia published updated guidelines on the diagnosis and management of keratinocyte cancers in the form of a 'living guideline'.[4] This update was a response to significant changes in treatment over the past decade, particularly radiotherapy and immunotherapy of advanced keratinocyte cancers. These changes led the Australian Government Department of Health to commission the Keratinocyte Cancer Guidelines Working Party to revise the 2008 guidelines on diagnosis and management of BCC and SCC.[5]

Here, we summarise the presentation, diagnosis and latest updated treatment and referral recommendations for keratinocyte cancers as per the updated 2019 guidelines.[4] Key changes in the updated guidelines are summarised in Box 1. The important role of GPs in educating patients about sun-safe behaviour to help prevent keratinocyte cancers is discussed elsewhere.[6]

Actinic keratosis

Actinic keratoses, also known as solar keratoses, are precancerous lesions commonly found on sun-exposed areas such as the face, scalp, ears and back of the hands (Figures 1a and b). They progress to SCC at the rate of 0.075% to 0.1% per lesion per year, sometimes extrapolated to up to 10% over 10 years.[7]

Associated factors

Actinic keratoses are clinically significant not only because they may progress to SCC but also because the presence of multiple actinic keratoses indicates significant ultraviolet (UV) light exposure. It is recommended that patients with multiple actinic keratoses have full skin checks on a regular basis with their GP, who may arrange referral to a specialist dermatologist if appropriate.

Presentation and diagnosis

Actinic keratoses have varied clinical presentations. They are classically described as gritty macules, papules or plaques on an erythematous base, often with rough yellow or white scale. They can also present as hyperkeratotic, pigmented or atrophic lesions. The lesions are typically asymptomatic but may sting or itch.

The diagnosis of actinic keratosis is predominantly clinical. Biopsy should be considered if there is concern that the lesion may be an early SCC. Hallmark signs raising concern include tenderness, bleeding, inflammation and growth in height or thickness.

On biopsy, the distinction between an actinic keratosis and SCC is the extent of keratinocyte atypia:

- in actinic keratosis, keratinocyte

TABLE. FIELD THERAPIES FOR ACTINIC KERATOSIS

Medication	Action	Treatment regimen	Comments
Fluorouracil cream	Antimetabolite	4% cream: applied by patient once daily for four weeks* 5% cream: applied by patient once or twice daily for four weeks	5% formulation has been one of the most common field therapies because of its cost (about $60/tube), ease of use and efficacy
Imiquimod cream	Enhances the local immune system	Applied by patient three nights a week (washed off the following morning) for three to four weeks	Review should be undertaken four weeks after treatment; treatment cycle can be repeated once if required As this immune alteration is achieved through toll-like receptor 7 activation, inflammatory reactions can vary in severity because of varying levels of expression in the population
Photodynamic therapy with a photosensitising agent (e.g. methyl aminolevulinate)	Apoptosis and necrosis of target cells	The photosensitising agent is applied to the treatment area, then occluded for three hours before being exposed to a dedicated light source	Usually only one treatment required, and the treatment area is reassessed at three months
Diclofenac sodium gel	Nonsteroidal anti-inflammatory agent	Applied by patient twice daily for 60 to 90 days	Used in practice but not included in the *Therapeutic Guidelines* recommendations

* Available from late 2021.
Adapted from Dermatology Expert Group. Solar damage and skin cancer. In: eTG complete [Internet]. Melbourne: Therapeutic Guidelines Limited; 2017.[8]

atypia is confined to the lower portion of the epidermis
- in SCC, keratinocyte atypia occupies the entire epidermis and may infiltrate deeper into the dermis.

Treatment of actinic keratosis

Localised treatment

The first-line modality for localised treatment of actinic keratosis is liquid nitrogen cryotherapy. This usually causes a blister that heals over seven to 10 days. The duration of therapy varies depending on lesion size and location, but a freeze of about three to five seconds is recommended.

Curettage, electrodessication and shave treatments can also be considered as treatment for actinic keratosis. However, these treatments are often reserved for larger, thicker lesions.

Field therapy

Field therapies should be considered for areas that contain multiple actinic keratoses. Field therapies are listed in the Table and include:[8]

- fluorouracil 4% or 5% cream
- imiquimod cream
- photodynamic therapy, involving application of a photosensitising chemical to the lesion, followed by irradiation with an appropriate light source
- diclofenac sodium gel.

Patient education about these therapies is paramount, as they act by inducing an inflammatory reaction, including redness, soreness and crusting. Patients are best counselled about this before therapy, including being shown pictures of expected reactions.

Squamous cell carcinoma

The second most common skin cancer in Australia is cutaneous SCC (Figures 2a and b). The age-standardised incidence rate of cutaneous SCC is about 387 per 100,000 in people aged 14 years and over.[9] The incidence increases steeply with age from mid-adulthood and appears higher in men than in women across all age groups.

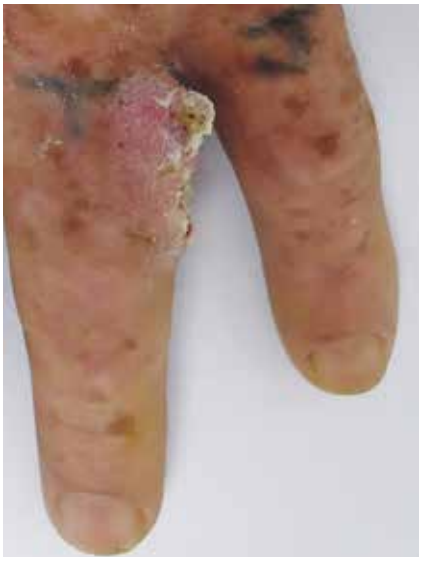
The most common sites of cutaneous SCC are the head and neck areas in men, and the upper limbs followed by the head and neck areas in women. After accounting for body surface area, the highest incidence of cutaneous SCC in both men and women is on the face, particularly the lips, ears, nose, cheek and eyelids.

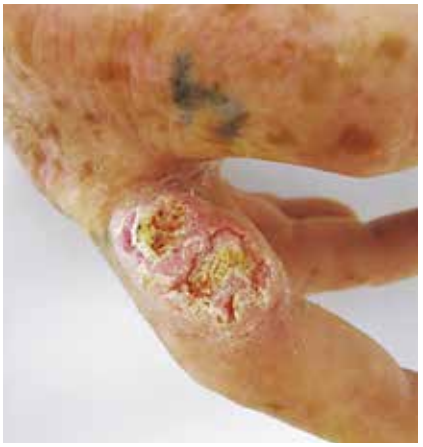
Associated factors

Cutaneous SCC can arise de novo with no risk factors or triggers. However, cutaneous SCC may also develop from actinic keratosis or be associated with the use of immunosuppressive medications, infection (e.g. by oncogenic subtypes of human papillomavirus), chronic inflammation or previous trauma (Figure 3). The two main risk factors for SCC are:

- cumulative UV exposure, which damages the DNA of keratinocytes and impairs the immune system
- lighter Fitzpatrick skin type, which increases susceptibility to the effects of UV exposure.

The primary concern with SCC is its ability to metastasise, with metastatic SCC accounting for about 20% of skin cancer deaths. A large nationwide study in England found the incidence of metastatic SCC in people with primary cutaneous SCC to be 1.1% in women and 2.5% in men over 36 months.[10]





Figures 2a and b. Squamous cell carcinoma on the right second finger. a (left). Dorsal view. b (above). Radial view.

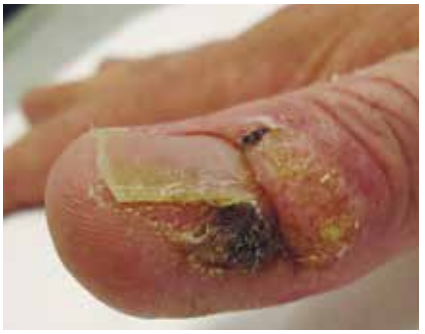


Figure 3. Persistent verruca vulgaris on the right thumb with histological changes overlapping with squamous cell carcinoma.

Presentation and diagnosis

Cutaneous SCC has a spectrum of presentations. In-situ SCC is termed Bowen's disease and presents typically as an erythematous patch or plaque with scale and, in rare cases, pigmentation. On histopathological examination, Bowen's disease shows keratinocyte atypia involving the full thickness of the epidermis. Without treatment, 2 to 5% of cases of Bowen's disease progress to involve the dermis, defined as invasive SCC.[7] SCC presents as an erythematous keratotic papule or nodule, which may be tender on palpation.

Diagnosis of Bowen's disease and SCC is via biopsy. If invasive cutaneous SCC is suspected, the biopsy should be deep enough to determine the extent of dermal involvement. Regional lymph nodes should be examined, and suspected metastases should be confirmed with fine needle aspiration. Key factors that indicate a poor prognosis for SCC include regional spread, perineural invasion, poorly differentiated histological appearance, higher-risk sites and comorbidities such as immunosuppression (Box 2).[4]

Treatment of SCCs

Surgery

Surgical treatment options for cutaneous SCC include local excision and Mohs micrographic surgery. Low-risk lesions can be treated with surgical excision, curettage and electrodesiccation, or punch excision. Larger lesions up to 20 mm can be removed using a scalpel, ensuring adequate lateral and deep margins. Adequate deep margins are particularly important, as inadequate deep margins significantly increase the risk of recurrence. Fear of damaging underlying anatomy and inexperience may lead to incomplete deep margins in otherwise easily resectable tumours. The orientation of the excision is important; tumours should be excised along relaxed skin tension lines, but in a line that avoids distortion.

For patients with lesions with poor prognostic features, such as size 20 mm or more, or poor differentiation (Box 2), referral to a multidisciplinary team or to a specialist for assessment and treatment should be considered.[4] Mohs micrographic surgery is a surgical option for cases where tissue preservation and cosmesis is of paramount importance. The technique involves histopathological examination of frozen sections of almost the entire peripheral and deep margins of the excised tissue, in contrast with standard sectioning, where 0.1 to 1% of the surgical margin is examined.

Surgical treatment of advanced cutaneous SCC with nodal involvement involves lymphadenectomy for disease in the axilla or groin, or selective neck dissection for cervical lymph node involvement. Dermal lymphatic spread (in-transit metastasis) should be managed by wide surgical excision followed by adjuvant radiotherapy.

Radiation therapy

Radiation therapy for SCC has improved significantly in the past few decades. Modified fractionation schedules and more precise fractionation techniques allow an improved balance between killing tumour cells and minimising effects on normal tissue.

There are several indications for radiotherapy of cutaneous SCC. First, it is a therapeutic option when surgery is not feasible because of patient frailty, comorbidities or high surgical or bleeding risk, or when surgery will have a significant effect on cosmesis or functional outcome. Secondly, referral to a radiation oncologist for multidisciplinary care should be considered for patients with T3 or T4 stage primary tumours or persistent or recurrent cutaneous SCC.

Referral for a radiation oncologist opinion about postoperative radiation therapy should also be considered for patients who have had complete excision of high-risk cutaneous SCC, such as T3 or T4 stage tumours, and those with more than 6 mm depth of invasion, inadequate margins on excision when further surgery is problematic, regional involvement or other poor prognostic features (Box 2).

2. FEATURES ASSOCIATED WITH POORER PROGNOSIS OF CUTANEOUS SQUAMOUS CELL CARCINOMA[4]

- Stage
  - regional spread
  - perineural invasion
- Size 20 mm or more, tumour depth over 4 mm
- Poorly differentiated subtypes or fibrosing on histological examination
- Clinical signs of rapid growth or greater spread (e.g. palpable thickness, diffuse infiltration and induration with poor demarcation of tumour edges, tenderness and inflammation)
- Higher-risk sites (e.g. ear, lips) and multiple skin cancers
- Previous recurrence or skin cancers
- Inadequately treated lesions
- Non-UV-induced lesions (e.g. oncogenic HPV subtypes, arsenic exposure)
- Comorbidities, including
  - immunosuppression
  - skin-related disorders (e.g. scleroderma, xeroderma pigmentosa)

Abbreviation: HPV = human papillomavirus.

Systemic treatment

Locoregional advanced cutaneous SCC represents an advanced stage of disease that may present de novo or after previous surgery and radiation therapy. The goal of treatment is to clear local disease and prevent further recurrence or regional metastasis. However, options may be limited by the location of the primary tumour and patient comorbidities and whether they are amenable to surgery or radiation therapy. Patients should be assessed on a case-by-case basis in a multidisciplinary setting.

For patients with resectable disease, surgery followed by adjuvant radiation therapy is the preferred treatment. For patients with unresectable disease, platinum-based chemoradiation may be considered.

Cetuximab, an epidermal growth factor receptor (EGFR) inhibitor, is approved for treatment of SCC of the head and neck. It can be used in combination with radiation therapy for locally advanced disease, or in combination with platinum-based chemotherapy for recurrent or metastatic disease.

For patients unsuitable for surgery or radiation therapy, treatment with cemiplimab (a PD-1 inhibitor) may be an option. Cemiplimab has TGA approval in Australia for adults with metastatic or locally advanced cutaneous SCC who are not candidates for curative surgery or radiation therapy.

3. FEATURES ASSOCIATED WITH POORER PROGNOSIS OF BASAL CELL CARCINOMA[4]

- Subtype
  - infiltrating
  - sclerosing (morphoeic)
  - micronodular
  - basosquamous
- Recurring tumours
- Perineural invasion

Basal cell carcinoma

BCC has the highest incidence of all cancers in Australia. The estimated age-standardised annual incidence rate of BCC is 884 per 100,000 overall, and is higher in men than in women (1041 versus 745 per 100,000). The incidence of BCC increases with age, although not linearly. It is higher in men compared with women up to age 50 years but similar at older ages. In both sexes, 50% of BCCs are found on the head and neck, 25% on the trunk and about 10% on each of the upper and lower limbs.[9]

Associated factors

As with SCC, UV radiation is the greatest risk factor for BCC, but unlike SCC, risk is associated with intense episodes of burning rather than cumulative exposure. BCCs rarely metastasise, and the main clinical concern is local destruction. BCC features associated with a poorer prognosis are listed in Box 3.

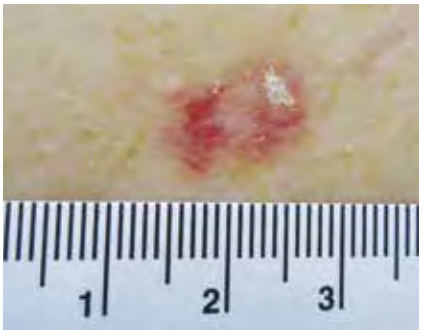


Figure 4. Superficial basal cell carcinoma on the mid-back.

Presentation and diagnosis

The most common BCC presentation is the nodular form, appearing as pearly papules or nodules with evident telangiectasia or umbilication. BCCs may ulcerate and are often found in the head and neck regions, even in areas of little sun exposure such as the inner canthus. Superficial BCCs present as erythematous patches or plaques, often on the trunk or extremities (Figure 4).

BCCs can also be pigmented, leading to a clinical differential diagnosis of melanoma, but an evident pearly edge and vascularity seen macroscopically or through a dermatoscope can aid in the distinction (Figure 5). Less common BCC variants include sclerosing (morphoeic or scar-like), micronodular and basosquamous BCCs.

BCCs are often diagnosed by biopsy. The BCC site and histological subtype dictate treatment.

Treatment of BCCs

Topical therapies

Superficial BCCs may be treated with topical therapies, including curettage and electrodesiccation, cryotherapy, imiquimod cream and photodynamic therapy. Cryotherapy is performed with a double freeze-thaw cycle of 20 to 30 seconds. This causes a significant blister that heals in about three to four weeks. It can lead to hypopigmentation, so it is important to counsel patients about this.

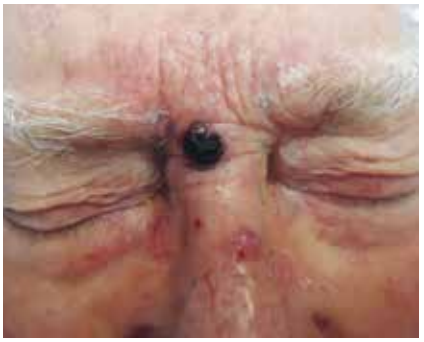


Figure 5. Pigmented basal cell carcinoma of the nasal bridge, along with a nodular basal cell carcinoma of the nose.

Similarly, imiquimod treatment of BCCs is more intensive than for actinic keratoses, with therapy applied by the patient for five nights per week for six weeks. Imiquimod stimulates the immune system and causes local skin inflammation, destroying the lesion. The degree of inflammation is variable and depends partly on the type of skin lesion and genetic factors. If severe inflammation occurs, patients may need to take a break from therapy for a week or two to allow time for the reaction to settle before continuing treatment. There is usually a good cosmetic outcome with little scarring after imiquimod treatment.

Photodynamic therapy can be used for superficial and thin nodular BCCs. It usually requires two treatment sessions, one week apart, with lesions first descaled or debulked, and the sensitising agent applied at a thickness of 1 mm and a margin of 5 mm.

Surgical treatment options

Surgery is usually the first-line therapy for nonsuperficial BCCs such as nodular or nodulocystic BCCs. It often involves excision and direct closure. Options include electrodessication and curettage in anatomically favourable areas.

Micronodular, infiltrating and fibrosing (morphoeic) BCCs are less well defined and need surgical excision with wider margins. For patients with these types of BCC or lesions with poor prognostic features (Box 3), referral to a specialist is appropriate for consideration of options such as Mohs micrographic surgery, postsurgical adjuvant treatments in certain cases and close follow up. The rate of recurrence of this group of tumours is higher if histological margins are close or the BCC is incompletely excised.

Radiation therapy

Radiation therapy using curative doses is an alternative treatment for BCCs if the patient declines surgery or surgery is inappropriate because of patient factors such as frailty, tumour-related factors (e.g. where tissue conservation or cosmesis is a high priority, such as in BCC of the eyelid) or treatment-related factors (e.g. concurrent anticoagulant therapy).

For patients with a T3 or T4 stage primary BCC or persistent or recurrent BCC, referral to a radiation oncologist for an opinion regarding radiation therapy should be considered. Patients with postoperative BCCs with high-risk features may also be referred for an opinion from a radiation oncologist.

Persistent BCCs after a curative dose of radiation therapy should be treated in consultation with a radiation oncologist. Biopsy and salvage excision surgery are often required.

Systemic therapy

Metastatic BCC is rare. If suspected, confirmation is required via biopsy. Patients with complex locally advanced disease are best treated by a multidisciplinary team that includes surgeons, dermatologists, radiation oncologists and medical oncologists.

Locoregional metastases should be treated if possible with surgical excision or radiation therapy. When treatment with curative intent (surgery, radiation

4. WHEN TO CONSIDER SPECIALIST REFERRAL FOR KERATINOCYTE SKIN CANCERS

- Uncertain diagnosis or doubts about appropriate treatment
- Lesions >1 cm (and certainly those >2 cm)
- Multiple lesions
- Technically difficult sites, such as the ear, tip of nose or eyelid
- Recurrent lesions despite treatment
- Incompletely excised lesions
- Recommended treatment is beyond the skills of the practitioner, or difficulty with technique or anatomy is expected
- SCC on the lips and ears
- Infiltrating or scar-like morphoeic BCC
- Cosmetic concerns
- Areas where palpable regional lymph nodes suggest metastatic spread of SCC
- When the GP will be unavailable for regular follow up, especially for an SCC

Abbreviations: BCC = basal cell carcinoma; SCC = squamous cell carcinoma.

therapy or both) is not possible, oral therapy with a hedgehog pathway inhibitor such as sonidegib or vismodegib should be considered. Sonidegib and vismodegib appear to have similar efficacy and have similar side effects, including muscle spasms, hair loss, taste loss and cramps. In some cases, if side effects are not tolerated, intermittent dosing with a hedgehog inhibitor can be used.

When should a GP refer?

GPs are well positioned to educate patients on the importance of sun-safe behaviours, to detect skin cancers at the earliest opportunity and to provide initial management. Recommendations on when to consider referring patients for specialist care are shown in Box 4.

Patients with suspicious lesions and those requiring surveillance or surgical management, including Mohs micrographic surgery, may be referred to a dermatologist. Referrals to plastic surgery and surgical oncology specialists are appropriate for lesions that require complex surgical management or have higher risk features. For patients who are unable to undergo surgery because of lesion features or comorbidities, referral for a radiation oncology opinion is appropriate. For complex tumours and metastatic tumours, referral to a multidisciplinary team that includes dermatologists, surgeons and medical and radiation oncologists is recommended for planning and management.

Conclusion

Keratinocyte cancers are the most common skin cancer encountered in Australia, and GPs play a crucial role in their detection and early management. Full-skin examination is recommended for patients at risk of skin cancers, as many areas of the skin cannot be adequately monitored by patients themselves. Prevention through sun-safe education, early detection and appropriate treatment can help reduce the impact these cancers have on patients' lives and the healthcare system. MT

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COMPETING INTERESTS: Dr Phan: None. Associate Professor Shumack was an advisory board member for Aspen Pharmacare this year.

Discoid eczema

More than just dermatitis

JOSHUA FARRELL MB BS; **STEPHEN SHUMACK** OAM, MB BS(Hons), FACD

Discoid, or nummular, eczema is a chronic form of dermatitis characterised by well-defined circular plaques. Associations include allergic contact dermatitis, low environmental humidity and excessive alcohol intake. It is managed by avoidance of triggers and use of emollients and topical corticosteroids.

The word eczema (also called dermatitis) derives from the Greek word 'ekzema', meaning 'to boil'. It is a clinical and histological pattern of inflammation of the skin characterised by pruritus and discomfort. It can be classified in several ways, one of which is morphologically.[1] Discoid eczema, or nummular eczema, is one form of dermatitis that can be classified this way. Discoid eczema is defined by circular or oval plaques of eczema with a clearly demarcated edge.[2,3] Many patients with eczema have at least one or two circular lesions, and few patients with discoid eczema have solely discoid lesions.[4]

Discoid eczema has an incidence of 2 per 1000 people and is particularly common in men aged 35 to 70 years.[2,5] The exact aetiology is unknown, although a significant percentage of patients may have an underlying allergic or irritant contact dermatitis.[3-7] Other associations include low environmental humidity, excessive chronic alcohol intake, staphylococcal colonisation, lifestyle factors (such as irritating soaps and fabrics and frequent exposure to hot water), trauma (the Koebner phenomenon) and certain medications including antivirals, interferon, isotretinoin, ribavirin and gold.[2,3,5,8-10] Chronic venous stasis has also been implicated, especially when the discoid eczema involves the lower legs.[3]

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KEY POINTS

- Discoid eczema is a subtype of nonatopic eczema defined by circular or oval plaques of eczema with a well-demarcated edge.
- It tends to follow a chronic course.
- The four clinical variants of discoid eczema are exudative nummular eczema, dry type nummular eczema, discoid eczema of the hands and exudative discoid with lichenoid chronic eczema.
- Differential diagnoses for eczema include tinea corporis, psoriasis and pityriasis rosea and are important to rule out for optimal management.
- It is managed through conservative skin care measures, as well as use of emollients and topical corticosteroids.
- Secondary infection with *Staphyloccocus* species is common and requires treatment with systemic or topical antibiotics.

Presentation

The diagnostic lesion is a coin-shaped plaque of closely-set thin-walled scaling and vesicles on a scaly erythematous base, classically on the extensor surfaces of lower limbs.[2,3,11] The lesions are sharply defined and range in size from 1 to 10 cm in diameter.[3] They arise rapidly from a confluence of tiny papules and papulovesicles.[2,3] In the acute phase, these lesions are dull red, very exudative or crusted and highly pruritic.[2] They progress towards a less vesicular and more scaly stage, with central clearing and peripheral extension. There is sometimes a drier scale and lichenification.[3] The lesions

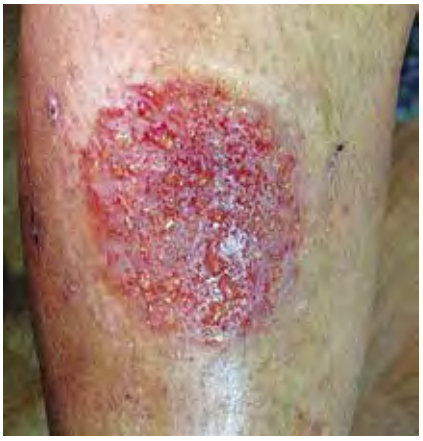

Figure 1. Exudative nummular eczema with fluid and crust formation from the lesion.

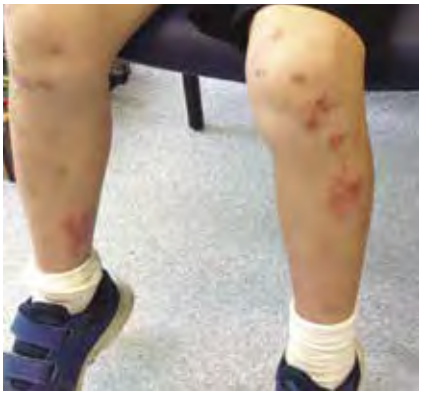

Figure 2. Dry type nummular eczema.
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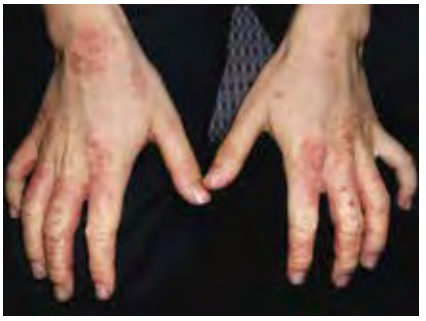

Figure 3. Discoid eczema of the hands.
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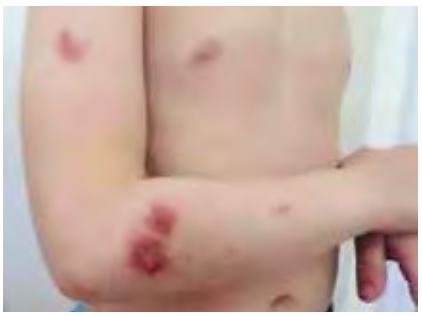
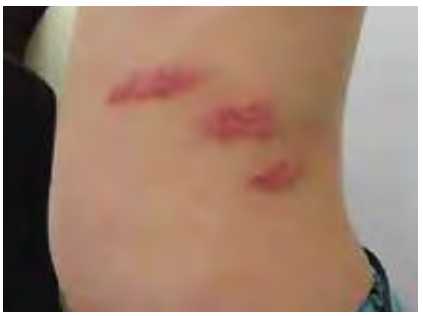
subsequently fade as dry, scaly patches.[2] The eruption can be precipitated by localised injury (such as from scratching, insect bites or burns), infections, contact dermatitis, dry skin or varicose eczema.[4,7] Secondary lesions occur between 10 days and several months after the primary eruption in a mirror-image configuration on the opposite side of the body. It is characteristic that dormant patches become active again especially if treatment is discontinued.[1]

There are four clinical variants of discoid eczema.

- Exudative nummular eczema involves leakage of serous fluid and crust formation from lesions (Figure 1).[1]
- Dry type nummular eczema is less common and consists of multiple dry scaly, round or oval discs on the arms or legs, with scattered microvesicles on an erythematous base on the palms and soles (Figure 2). Itching is minimal in this subtype. It is notably resistant to treatment and persists for years.[1]
- Discoid eczema of the hands affects the dorsa of the hands or backs and sides of the fingers (Figure 3). It often develops as a single plaque at the site of the original irritant. Secondary lesions may occur locally, but generalised spread is uncommon.
- Exudative discoid with lichenoid chronic eczema is a widespread, extremely pruritic eruption characterised by discoid lesions with lichenoid and exudative phases that coexist or may alternate rapidly over several days (Figures 4a and b). After a chronic course of months or years, it may end in spontaneous cure. It occurs predominantly in Jewish adult men aged 40 to 60 years.[12] There is some conjecture as to whether this is a distinct clinical entitity.[13]





Figures 4a and b. Exudative discoid and lichenoid chronic dermatosis. Unusual occurrence in a 6-year-old boy.

Differential diagnoses

Differentiating discoid eczema from other forms of dermatitis is important for optimal management, particularly in avoiding triggers. Differential diagnoses for discoid eczema are summarised in the Table and include the following.

- Discoid eczema may appear to be similar to tinea corporis with central clearing. Tinea corporis is an infection often found after close contact with an infected animal, and presents as annular, scaly erythematous lesions that spread centrifugally (Figure 5).[11,14]
- Psoriatic lesions differ in their dryness and more prominent silvery scaling with milder irritation (Figure 6).[11]
- Pityriasis rosea presents with a herald patch and salmon-coloured oval patches with long axes parallel to the ribs, although it may transiently resemble discoid eczema (Figure 7).[11]
- Pityriasis alba more commonly affects the face and proximal limbs in depigmented lesions.
- Chronic superficial dermatitis tends to affect the limbs more, is oval or round with no infiltration, and is chronic with few fluctuations.

TABLE. DIFFERENTIAL DIAGNOSES FOR DISCOID LESIONS

Differential diagnosis	Differentiating features	Treatment
Tinea corporis	• Pruritic oval lesions distributed over the limbs or trunk • Scrapings for microscopy and culture and periodic acid–Schiff stains often demonstrate fungus • Continues until treated	• Topical or oral antifungal medications
Psoriasis	• Lesions are dry with more prominent scaling and milder irritation • More commonly involves the scalp, elbows and knees	• Treatment is dependent on disease impact and severity, but includes avoidance of triggers and treatment with topical corticosteroids, phototherapy and systemic agents including biologics
Pityriasis rosea	• Presents with a history of a herald patch and oval patches with long axes parallel to the ribs • Self-limiting	• Is self-limiting and resolves after six to 12 weeks • Topical corticosteroids can help relieve itch
Pityriasis alba	• Involves depigmentation that affects the face and proximal limbs with spontaneous remission after >1 year	• Moisturisers • Short courses of topical corticosteroids
Chronic superficial dermatitis	• Oval or round lesions that tend to affect the limbs more than trunk, with a chronic benign course with no fluctuations	• Emollients • Topical corticosteroids • Phototherapy
Prelymphomatous eruption	• Angular pruritic lesions affecting the proximal limbs, flank or trunk that are persistent and may change to lymphoma • Biopsy shows a perivascular infiltrate in the dermis, predominantly of lymphocytes	• Systemic corticosteroids • Phototherapy

- Prelymphomatous eruption more commonly affects the flank, trunk and proximal limbs with angular lesions.
- Allergic contact eczema may present with nummular lesions, although can be differentiated on history and through patch testing.[3]

Discoid eczema is also more likely to occur in a generalised distribution and is less likely to occur on the face, neck, lips, ears and anogenital areas.[5] 30% of patients may have both discoid eczema as well as an irritant (or allergic) contact dermatitis.[5]

Pathophysiology

Triggers including bacterial colonisation, contact allergens, certain medications and chronic venous stasis may compromise the cutaneous lipid barrier. This results in the subsequent release of cytokines, including interferon gamma and interleukin 17, and recruitment of T cells, dendritic cells and Langerhans cells. This causes epidermal hyperplasia and the formation of the lesions characteristic of discoid eczema.[3]

Association of discoid eczema with surgical sites has been reported, with occurrence of the isomorphic Koebner phenomenon between three months and 10 years after surgery.[10] Alcohol misuse has also been associated with discoid eczema. This relationship might be due to alcohol impairing the immune response, thus predisposing the skin to a bacterial infection, which in turn triggers the eczema.[8,9] However, there is no reported association between alcohol misuse and other forms of eczema.[8,9]

Investigations

Discoid eczema is a clinical diagnosis.[3] However, exogenous contact dermatitis should be suspected if the condition is unusually severe and persistent, or if the lesions are few, asymmetrical or of unusual configuration. In these cases, patch testing can be performed.[6,7] Often, scrapings are taken for mycology to exclude tinea corporis.

Some studies have highlighted the role of infection in triggering and prolonging discoid eczema, and heavy colonisation of lesions by *Staphylococcus* species may increase its severity.[15] Allergic sensitivity to *Staphylococcus* may be partly responsible for secondary dissemination.[16] Thus, bacterial swabs may be taken, especially if the rash is not responsive to therapy.

Although discoid eczema does not generally require a biopsy, histological examination demonstrates a subacute dermatitis, with spongiotic vesicles and lymphohistiocytic infiltrate.[2,3] Electron microscopy has demonstrated that intercellular oedema leads to a reduction in the number of desmosomes between the cells of the basal layer, whereas those in the stratum spinosum are preserved.

Management

The management of discoid eczema is aimed at restoring the natural skin barrier and minimising exposure to triggering factors. This can be achieved through short, cool showers using gentle soaps or soap substitutes. Tight clothing and

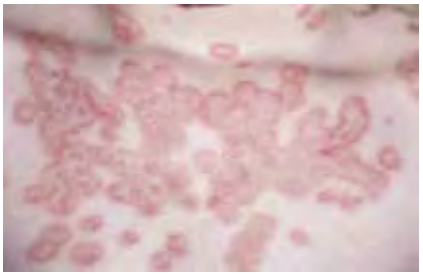


Figure 5. Tinea corporis.
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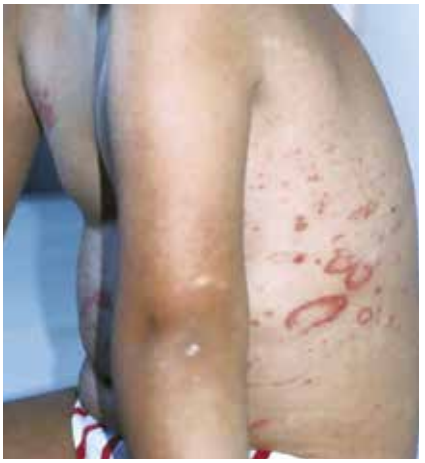

Figure 6. Psoriasis.
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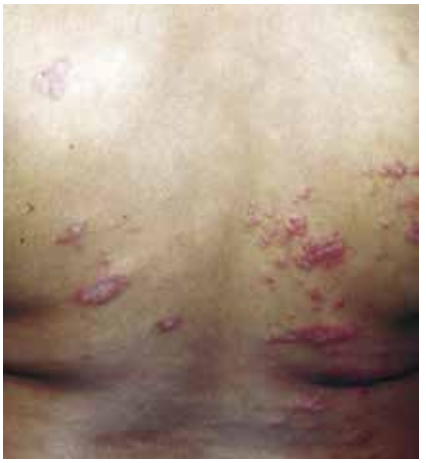

Figure 7. Pityriasis rosea.
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irritating fabrics such as wool and nylon should be avoided.[2,3]

Patients should frequently moisturise, including immediately after showering. Intermittent topical corticosteroids are used to dampen eczema flares.[2,3] Corticosteroid-sparing agents including topical calcineurin inhibitors (such as tacrolimus and pimecrolimus) may also be used in combination or alternately with topical corticosteroids.

Classically, coal tar pastes or ointments are used in the less acute stages of discoid eczema.[1] As with other types of eczema, there has traditionally been a role for rest and minimisation of stress. Sedating antihistamines also have a role in helping patients with severe pruritus to sleep at night.[3]

For widespread disease, alternative treatment therapies include narrowband UVB light therapy two to three times weekly, with duration titrated to treatment response.[3] Systemic immunosuppressants and immunomodulators have been used as more last-line options for extensive disease.[3] Dupilumab is an emerging treatment option for recalcitrant discoid eczema.[17] Other last-line therapies include immunosuppressants such as ciclosporin, methotrexate, mycophenolate and azathioprine.[18] Systemic corticosteroids should be avoided as they are associated with high rates of rebound disease.

Patients with the exudative subtype of discoid eczema may benefit from an oral antibiotic such as cefalexin, clarithromycin or a tetracycline. Secondary bacterial infections should be treated with topical or oral antistaphylococcal antibiotics depending on the size and distribution of the lesions, with further treatment based on bacterial swab results and local antibiotic resistance.[3] Discoid eczema associated with alcohol misuse may be particularly predisposed to becoming secondarily infected, and such patients should be examined carefully to ensure infection has been properly treated.[9]

Referral to a dermatologist is appropriate if the disease does not respond to conservative skin care measures, bland emollients, topical corticosteroids and antibiotics. Referral can also be considered should the diagnosis be in question.

Prognosis

Discoid eczema is chronic, with partial remission during which plaques tend to clear in their centres. Relapse occurs at variable intervals, and is often worse in winter. With appropriate therapy and avoidance of skin irritants, discoid eczema may either clear within a year, or if not, will often persist for many years.[2] As with other forms of eczema, some patients will experience postinflammatory dyspigmentation of the skin (hypo- or hyperpigmentation).[3]

Conclusion

Discoid eczema is a skin rash consisting of annular disc-shaped plaques typically affecting the trunk and lower limbs of adult male patients. It is sometimes associated with irritant (or allergic) contact eczema, chronic alcohol misuse, bacterial colonisation and certain medications. In most patients, discoid eczema can be controlled through general skin care measures of avoiding irritants and using regular emollients, intermittent topical corticosteroids and antibiotics as needed. MT

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A list of references is included in the online version of this article (www.medicinetoday.com.au).

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Discoid eczema

More than just dermatitis

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Common skin problems in children

Infectious rashes and infestations

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Cutaneous infections and infestations are common in children; the key to their diagnosis is a positive culture or scraping to confirm infection and/or response to anti-infective agents.

KEY POINTS

- Although impetigo is usually due to *Staphylococcus aureus*, certain groups (including Aboriginal and Torres Strait Islander people) are particularly susceptible to group A *Streptococcus* infections.
- Children with recurrent *S. aureus* infections are usually carriers of this bacterium.
- Not all cases of folliculitis are due to *S. aureus*; *Pseudomonas aeruginosa* acquired from contaminated water or bath toys may be responsible.
- Most acute genital infections in children are due to group A *Streptococcus*.
- Tinea capitis is a childhood condition, which may at times present with an inflammatory mass.
- Genital warts in children should always be a cause for concern.
- Scabies is often a very difficult diagnosis to make; it is often mistaken for dermatitis.

Few children escape having a *Staphylococcus aureus* skin infection of some sort, a viral exanthem, warts, molluscum contagiosum, head lice or scabies at some time in their childhood. Although most children with a rash are assumed to have an infection or a bite, this is not always the case, and many noninfectious rashes also affect children.[1,2] Positive cultures or scrapings are needed to confirm the diagnosis of an infectious rash or infestation.

Bacterial infections

Impetigo

Impetigo presents most often in children, although it may be seen at any age. It is caused most often by *S. aureus* and less often by *Streptococcus pyogenes*, and both organisms may occur together. Certain groups, including Aboriginal and Torres Strait Islander people, appear to be more susceptible to *S. pyogenes* infections. Impetigo is contagious, and if it is due to *S. pyogenes*, glomerulonephritis may follow within eight weeks. It is always helpful to confirm the diagnosis by a skin swab to define the infective organism and establish antibiotic susceptibility.

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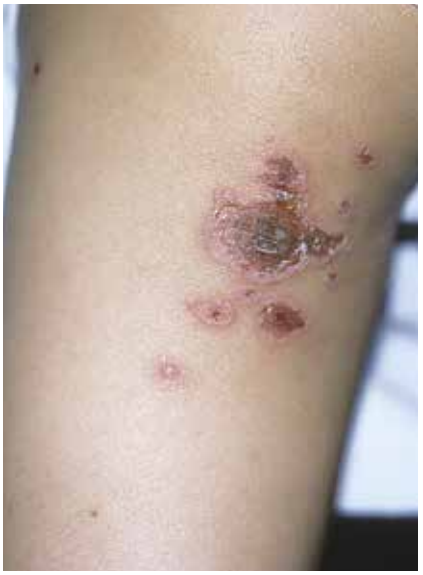

Figure 1. Brown crusts of bullous impetigo.

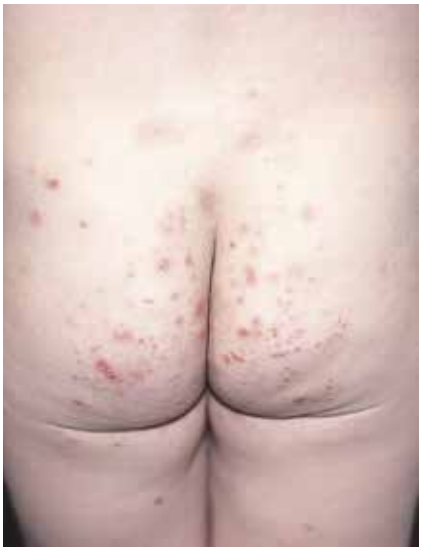

Figure 2. Folliculitis on a typical site. Note the pustules on an erythematous base.

Impetigo presents in three ways:

- crusted or nonbullous impetigo, presenting as yellow crusts and erosions that are itchy or irritating but not painful; this is the most common presentation and is often subacute
- bullous impetigo (Figure 1), which is always caused by *S. aureus*, and presents as mildly irritating blisters that erode rapidly leaving a brown crust
- ulcerative lesions, which are always caused by *S. pyogenes*.

Until culture results are available, it is reasonable to suspect *S. aureus* as the pathogen and to treat accordingly. Minor lesions may be successfully treated with topical antibiotics.

Community acquired methicillin-resistant (but not multiresistant) *S. aureus* (MRSA) is common. Treatment should be guided by susceptibility testing. These strains are generally susceptible to clindamycin or trimethoprim plus sulfamethoxazole as well as the drugs normally chosen for the multiresistant strains, such as vancomycin. It is always helpful to ask for expert advice from the laboratory or an infectious diseases physician before treating patients infected with any form of MRSA.

It is not uncommon for impetigo to become recurrent or resistant to treatment. Sometimes this is caused by an underlying dermatosis, but usually it is due to chronic carriage of *S. aureus*. This can be confirmed by taking a nasal and/or perineal swab for culture, depending on the location of the impetigo lesions. If swabs are positive, the whole family and close contacts need decolonisation treatment. Mupirocin 2% nasal ointment should be used intranasally twice daily for seven days and half a cup of bleach should be added to bathwater or, if showering, an antiseptic wash containing triclosan or chlorhexidine should be used for four weeks. During this time, clothes, towels and sheets should be washed in hot water. If lesions continue to recur despite these measures, treatment with a combination of rifampicin and flucloxacillin or clindamycin usually ends the problem.

Folliculitis and boils

Folliculitis is a common condition, particularly in hot weather. It presents with mildly itchy pustules on an erythematous base (Figure 2). It may affect any part of the hair-bearing skin, and it often occurs in macerated areas, including under dressings or nappies.

Although folliculitis is most often due to *S. aureus* infection (patients are often

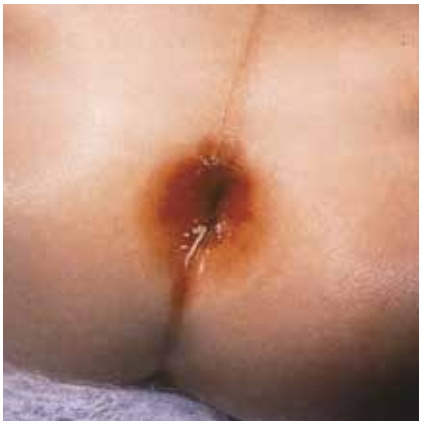

Figure 3. Perianal streptococcal dermatitis.

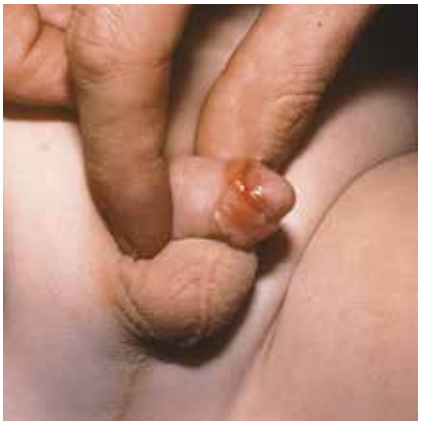

Figure 4. Acute streptococcal balanitis.

Figure 5. Vulvovaginitis due to *Streptococcus pyogenes*.

chronic carriers of *S. aureus*), this should be confirmed by culture. Folliculitis may also be caused by *Pseudomonas aeruginosa* (usually acquired from contaminated water supplies or mouldy bath toys), pityrosporum yeasts, dermatophytes and herpes simplex virus.

Folliculitis caused by *S. aureus* is treated in the same way as impetigo with a staphylococcal carrier state. For pseudomonal folliculitis, identify the source (by culturing water in the hot water tank, for example) and cease contact with it until it has been treated. If a child is still wearing nappies at night and the folliculitis involves the buttocks, there is usually marked improvement when nappies are no longer worn.

Boils are a deep form of folliculitis and are usually tender and painful. The causative organism is generally *S. aureus*, occasionally in association with *S. pyogenes*. It is common for boils to be recurrent, but this does not usually indicate any form of immunological abnormality. Small lesions may be treated with drainage alone, but it is usual to treat them in the same way as folliculitis and recurrent impetigo.

Streptococcal genital and perianal infections

Healthy prepubertal children do not suffer from candidal infections of the genital area, unlike adults. In children, most cases of infection of the penis, vulva and perianal area are due to the group A streptococcus *S. pyogenes*, which may possibly be acquired by haematogenous spread from the throat.

Streptococcal perianal dermatitis in children presents with a persistent perianal eruption (Figure 3). The rash is itchy and tender and may be complicated by painful fissuring. There is usually well-defined erythema, with scaling or weeping, which may extend to several centimetres from the anal verge. Bleeding and discharge may occur.

Acute balanitis (Figure 4), which is seen most often in uncircumcised boys, and vulvovaginitis (Figure 5) in girls are most often due to *S. pyogenes*. They may occur in conjunction with perianal lesions or in isolation.

Confirmation of the infection should be made by culturing a skin swab, and a low vaginal swab in cases of vulvovaginitis. The infection must be treated with oral antibiotics (penicillin, cefalexin or roxithromycin), and unless a full 10-day course is adhered to, recurrence is common. It is helpful to use topical mupirocin ointment twice daily at the same time to prevent recurrence.

Some children with this infection do not immediately lose their symptoms after adequate antibiotic treatment. This does not necessarily mean that the infection is persistent, and a second swab should be done before repeating antibiotics. Many simply have residual dermatitis or psoriasis that has been flared by the infection, and treatment with topical 1% hydrocortisone is all that is required.

Common fungal infections

Tinea

In children, tinea (dermatophyte infection) most often involves the scalp, face and body. Tinea pedis is less common in children than adults. In children, tinea is often acquired from dogs, cats, pet mice and guinea pigs, but human pathogens may also be responsible, especially in the case of tinea pedis. Animal dermatophytes tend to produce a more inflammatory and acute form of tinea.

Tinea capitis

Tinea of the scalp is very uncommon in adults and occurs almost exclusively in children. Usually there is loss of hair, which occurs most often in round patches, but occasionally is diffuse. This is associated with itching and scaling. A kerion is a very acute form of tinea capitis, usually caused by an animal dermatophyte, in which a large boggy, pustular mass appears on the scalp. Despite the alarming appearance, the child is well systemically and the lesion is not severely tender, which differentiates it from a bacterial infection where there would be fever and pain.

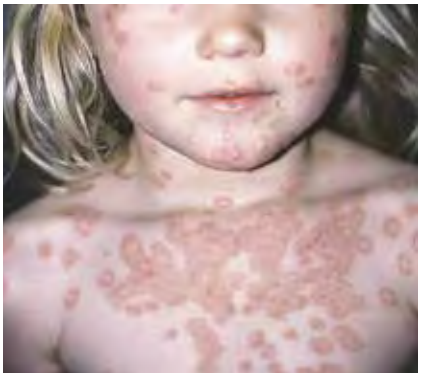

Figure 6. Florid tinea corporis acquired from contact with a kitten.

If tinea capitis is suspected, fungal culture of skin scrapings and plucked hairs should be done before starting therapy. It is important to confirm the diagnosis for future monitoring and to justify what is usually a prolonged course of oral treatment, but treatment may start before the results are available. Wood's light examination is unreliable for diagnosis.

Topical therapy is ineffective in treating tinea capitis and oral treatment must be used. Although tinea capitis is not a PBS indication for terbinafine, this antifungal agent has the advantage of being effective in about six weeks with once daily dosing. Overseas trials have confirmed its safety and usefulness in children. Other oral antifungals including fluconazole and itraconazole are also effective.

At the end of the treatment period, the culture should be repeated. If the culture is negative, the hair has regrown and there is no scalp inflammation, treatment can be ceased. If the culture is positive, continue therapy, repeating the culture every four to six weeks. Therapy may be stopped when a negative culture is obtained and hair has regrown. Occasionally this may take several months. Permanent alopecia is a very unusual sequel of tinea capitis.

Ketoconazole shampoo and selenium sulfide shampoo reduce shedding of spores and are a useful adjunct to therapy; however, if used alone they are ineffective.

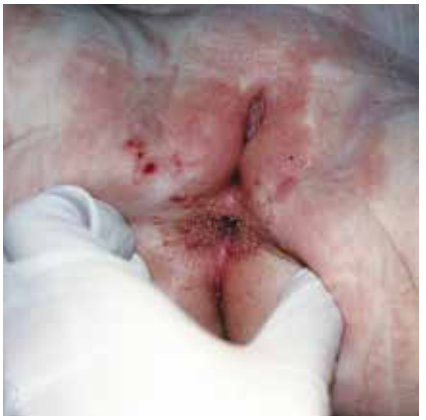

Figure 7. Genital warts in a baby.

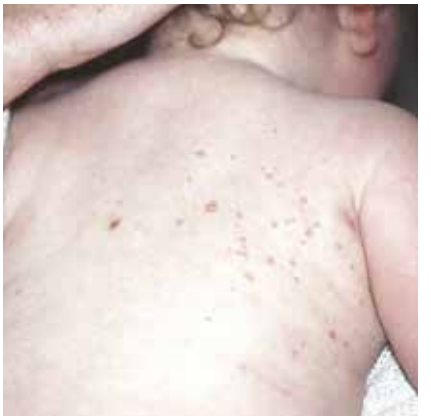

Figure 8. Molluscum contagiosum.

The use of antibiotics, oral corticosteroids and surgical debridement does not add to the management of a kerion. This is an important point as children with a kerion are sometimes taken to theatre in an attempt to drain the mass. This is unnecessary as the lesion does not contain pus.

Tinea corporis

Tinea corporis usually presents with annular, scaly lesions; there may be one or many (Figure 6). Usually they are itchy. Many rashes that are common in children, such as dermatitis and psoriasis, may mimic tinea, but a fungal scraping for culture makes it possible to differentiate tinea.

Localised tinea corporis in children is easily treated with topical antifungal creams, but if the infection is widespread, longstanding or has been treated with topical corticosteroids, oral treatment with griseofulvin is indicated, using the same antifungals as for tinea capitis. Griseofulvin may be used but is slow to work and hampered by the side effects of diarrhoea and photosensitivity. Clinical improvement usually occurs promptly, but ceasing treatment prematurely results in relapse. Treatment should be continued for a minimum of three weeks after resolution of all signs and symptoms.

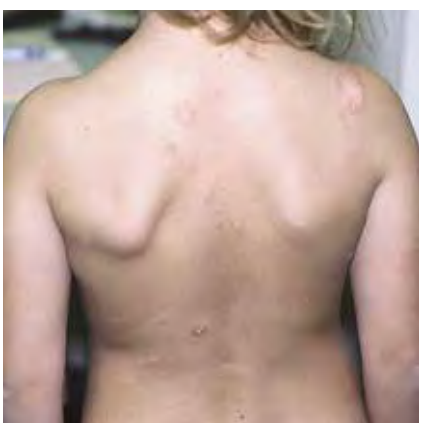

Figure 9. Pityriasis rosea.

Common viral infections

Warts

Warts are benign tumours that are seen at any age but most often in children. Common warts usually occur on the hands, feet and extensor surfaces. Facial warts often take the form of multiple tiny brown papular lesions. Warts adjacent to mucosal surfaces are often filiform. There is no specific or reliably effective treatment for warts, but in children they often resolve spontaneously within two years, making aggressive or painful therapy inappropriate.

The presence of warts in the genital area in children of either sex should raise the question of child sexual abuse (Figure 7). In children under 2 years of age there is the possibility of vertical transmission from a mother with genital or cervical warts. In theory, warts may be acquired from a parent with warts on the hands or by autoinoculation if the child has warts elsewhere on the skin. Although many published studies have stated that genital warts in children are not always sexually acquired, the fact is that children rarely disclose sexual abuse. Given that genital warts are usually sexually acquired in adults, in my opinion this finding is always cause for concern. The origin of genital warts in children is still controversial.

There are many over-the-counter treatments for warts. Plane warts may respond to topical tretinoin 0.05% cream, and genital warts to topical podophyllotoxin or imiquimod. Painful procedures such as cryotherapy, cautery or laser are often impractical in children.

Immunotherapy with the topical sensitiser diphencyprone is effective and well tolerated by children but can be hazardous because of the risk of severe allergic contact dermatitis and the possibility of systemic reactions. This treatment requires referral to a dermatologist.

It is often best to wait for warts to resolve spontaneously, but when they are causing distress or embarrassment and conservative treatment has failed, specialist referral of the patient is recommended.

Molluscum contagiosum

Molluscum contagiosum is a common contagious poxvirus infection (Figure 8). In young children, the lesions occur anywhere on the body and the infection is usually acquired from family members or others with whom they swim or bathe as the virus is spread in water.

The typical lesion is a pearly papule with central umbilication and a core that may be expressed by applying firm pressure to either side of the lesion. With dermoscopy, it is usually easy to see the central core. This differentiates mollusca from HPV induced lesions.

Molluscum contagiosum usually resolves spontaneously in immunocompetent patients, but this may take up

to two years. Atrophic scarring may occur, regardless of whether the lesions are treated. The infection may be complicated by dermatitis, particularly in atopic patients, and bacterial superinfection. Sometimes just before resolution there is an inflammatory flare of all lesions.

Showering rather than bathing may reduce the spread of lesions because of the transmissibility in water. It is not practicable or necessary to isolate children with molluscum contagiosum. Most chemical therapies are ineffective, although topical imiquimod works in some cases. In children, conservative management – i.e. waiting for spontaneous resolution – is usually best, unless lesions are widespread and interfering with lifestyle and function. If treatment is needed and the lesions are small, they may be curetted or expressed. When lesions are numerous, patients may require sedation, nitrous oxide analgesia or even a general anaesthetic. Secondary dermatitis requires treatment with topical therapy, and it is helpful for the patient to have a tube of mupirocin ointment at home in case of superinfection.

Viral exanthemata

Now that the classic exanthemata such as measles and rubella are uncommon because of immunisation, most of the viral rashes seen in children are due to enteroviridae such as coxsackie viruses and echovirus. Despite vaccine being available, varicella is still encountered from time to time.

Generally, viral exanthemata have two main presentations in children:

- generalised, bilaterally symmetrical maculopapular rashes, often with confluence on the face and involvement of the helix of the ears
- peripheral papular eruptions, mainly on the arms and legs.

In most cases, the child is well and afebrile but the rash may be quite itchy. Children may have had mild fever and diarrhoea at the onset of the rash. It is not uncommon for the lesions to take several weeks to resolve, and only symptomatic treatment is possible.

A few viral infections have specific presentations. These include:

- parvovirus infection (slapped cheek disease), which presents with confluent erythema of the cheeks associated with a reticulate rash on the arms and legs
- hand, foot and mouth disease, which presents with small vesicles on the hands and feet associated with mouth ulcers
- roseola, due to herpes virus 6, which is a transient maculopapular rash associated with fever and lymphadenopathy and seen in children under the age of 3 years
- pityriasis rosea (Figure 9), which presents with a single scaly herald patch, followed by multiple ovoid, erythematous macules with a peripheral scale mainly on the trunk; this is probably viral but is yet to be confirmed
- herpes simplex infection, which most often causes a stomatitis in children and only generalises in those with eczema, causing a widespread vesicular rash
- varicella, which presents with fever and a vesicular eruption that rapidly forms umbilicated pustules and crusts; all mucosal surfaces, including the conjunctivae, may be involved.

Common infestations

Scabies

Scabies is caused by infestation with a tiny six-legged insect that is a specific human pathogen. It is not visible to the naked eye. It is spread by close physical contact between infected persons and is not acquired from animals. Scabies is common in children and, if untreated, can spread to all members of a patient's family. Indigenous children living in crowded communities are particularly susceptible and scabies is a major cause of morbidity in these communities because of secondary bacterial infection with *S. pyogenes* to which this group is susceptible (see above).

The diagnosis of scabies is often difficult, especially if it is not suspected. The clinical picture is variable because it is due to an allergic reaction to the mite and this is very individual. Some patients have very few signs, and occasionally the only complaint is itch without an obvious rash. The itch exacerbates at night and after hot showers. Typically, there is an itchy, excoriated but nonspecific rash on the

PRACTICE POINTS: HOW INFECTIONS CAN MIMIC OTHER RASHES

- Streptococcal impetigo may ulcerate and be confused with trauma.
- Bullous impetigo may look annular and simulate tinea.
- Tinea corporis may be confused with discoid eczema and psoriasis.
- Rare immunobullous diseases are usually thought initially to be impetigo or varicella. Rashes in the genital area that do not resolve with antibiotics are often due to psoriasis. Tinea is often confused with psoriasis and eczema.
- Acute hair loss is not always due to tinea: consider alopecia areata and hair pulling (trichotillomania).
- Not all warty lesions are warts; epidermal naevi can simulate them.
- Scabies can be very difficult to diagnose and is often mistaken for dermatitis.
- Viral exanthemata are often bilaterally symmetrical and of sudden onset; if the child is taking an antibiotic, it is hard to differentiate viral exanthemata from a drug eruption.
- Viral exanthemata have very varied morphology and come into the differential diagnoses of scabies and dermatitis.
- Not all scaly lesions on hair shafts are nits; sometimes they are just adherent scalp scale (known as hair casts).
- Folliculitis is not always infective. It can be the result of occlusion and overheating. In babies it can be confused with toxic erythema.

trunk associated with scaly burrows on the fingers and wrists. Papular and nodular lesions are often seen around the major flexures in children, and in babies and toddlers there are often vesicles and pustules on the palms and soles and sometimes on the scalp. Secondary bacterial infection may occur.

Confirmation of the diagnosis is made by microscopy of scalpel scrapings from a burrow. Dermoscopy is also useful in visualising burrows. The main pitfall in using this technique is the selection of a suitable burrow, as these are few in number and difficult to identify. Therefore, the most practical diagnostic test may be response to antiscabetic medication.

For the topical treatment of scabies, the treatment of choice in terms of safety and efficacy is permethrin 5% cream. Benzyl benzoate 25% emulsion can be effective in scabies if used correctly, but it is more irritating than permethrin. The treatment is applied from the neck down and left on for a minimum of eight hours for permethrin and 24 hours for benzyl benzoate. If the hands are washed during this time, the treatment should be reapplied. In central and northern Australia, scabies above the neck is common and treatment should also be applied to the face and hair (avoiding eyes and mucous membranes). In babies, the scalp and face should also be treated, and mittens should be worn to stop them sucking their hands. In all cases, the treatment should be repeated after a week.

There is some controversy regarding the treatment of scabies in babies. Although permethrin is not approved for use in those under 6 months of age, this has to be balanced against the high morbidity of untreated scabies, which can be severe in this age group. Although some authors recommend the use of 10% sulfur and 10% crotamiton cream in this age group, there may be no rationale in doing so and, in fact, safety data on these preparations are very sketchy.

For severe infestations in children aged 5 years and over, oral ivermectin may be used. It is given as two doses one to two weeks apart using 200 mcg/kg for each dose.

Head lice

Head lice are common in school children, affect all sections of society and are harmless. They are not a sign of poor hygiene. The infestation is acquired by head to head contact. Although it can be asymptomatic, the scalp and nape of neck can be itchy and nits are noticeable on the hairs. The itching can result in excoriations and lymphadenopathy can follow.

Head lice are crawling mites the size of a sesame seed. They live on the scalp but lay eggs, known as nits, on the hair. Diagnosis of active lice infestation is made by observing a live, moving mite on the scalp. This can be achieved by wet combing the hair with a fine-toothed comb after applying a generous amount of hair conditioner to dry hair. The conditioner stuns the lice and stops them crawling for about 20 minutes. Conditioner from the comb is wiped on to a paper towel and lice and nits are detected. Even when all the lice are dead, nits may remain on the hair. If they are 1.5 cm away from the scalp, they are unlikely to contain live larvae, and just represent old infestation.

Treating head lice can be difficult. Treatment resistance is well documented, and reinfestation is always a risk. Some cases can be cured by wet combing every day for 10 to 14 days until no lice are found. This method has only about a 40% success rate.

Alternatively, topical scabicides can be used. Most contain permethrin and are usually left on for 20 minutes, but maldison is another insecticide that appears to be more effective. Maldison should be left on for eight hours and is not recommended in infants aged under 6 months. Parents and even pharmacists are often worried about using these insecticides on children, and parents may need reassurance before starting treatment. Oral ivermectin may also be used as in scabies.

All head lice treatments should be repeated seven to 10 days later, and the conditioner and combing method (above) should be used the next day to check that there are no further live mites on the scalp. Between treatments, the same combing method should be used twice, with all eggs less than 1.5 cm from the scalp being removed with a head lice comb or pulled off with fingernails. These eggs may contain viable larvae. Wet combing should be repeated weekly for several weeks after cure to detect any recurrence.

Pillowcases, combs and brushes should be washed in hot water (at least 60°C). Family and close physical contacts should be examined and treated if live lice are found. The child's school should be notified, but it is not necessary to exclude children with head lice from school after their initial treatment.

Conclusion

Most children at some time in their childhood have an infectious rash or infestation, some of which can mimic noninfectious rashes (see Practice Points). The key to the diagnosis of infectious rashes or infestations is a positive culture or scraping, which also guides treatment decisions. MT

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Common skin problems in children

Birthmarks

GAYLE FISCHER OAM, MB BS, MD, FACD

Although most birthmarks are small and harmless, some are large and can cause cosmetic or functional impairment. Others can indicate an underlying abnormality, and a few have malignant potential. Some birthmarks can be removed, but others remain a challenge to treat even with present surgical and laser techniques.

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KEY POINTS

- 'Birthmark' is a lay term that encompasses many skin conditions, including malformations, neoplasms and hamartomas; not all are obvious at birth.
- The risk of melanoma in children is low; most melanocytic naevi are removed for cosmetic reasons, not malignancy.
- Naevus sebaceous is the most common epidermal naevus and one of the few with a malignant potential.
- Most haemangiomas are uncomplicated, requiring no intervention; however, facial haemangiomas need to be carefully observed over time as even a modest increase in size can cause substantial problems.
- The 'gold standard' treatment for haemangiomas that are large, ulcerated, likely to cause deformity or affecting an orifice is oral propranolol.
- Children with capillary malformations (port wine stains) may be treated with pulsed dye laser therapy and should be referred for assessment as early as possible.
- Most small birthmarks can be removed; however, the decision should be postponed in most cases until the child can be involved in decision making.

To the layperson, the term 'birthmark' indicates a permanent skin lesion that a child is born with. However, not all congenital lesions are permanent, and not all permanent lesions are present at birth. From a medical viewpoint, birthmarks encompass a wide variety of cutaneous lesions, including malformations, neoplasms and hamartomas. The cells of these lesions include melanocytes, vascular elements, keratinocytes and skin appendages such as sebaceous glands, all of which occur normally in the skin.

This article reviews the most common types of birthmarks occurring in childhood and their presentation and management.

Pigmented naevi

Melanocytic naevi

A melanocytic naevus is a benign collection of pigmented naevus cells. Most melanocytic naevi (often known as 'moles') are not present at birth and are termed acquired. However, they can be congenital, occurring in about one to two in 100 newborns

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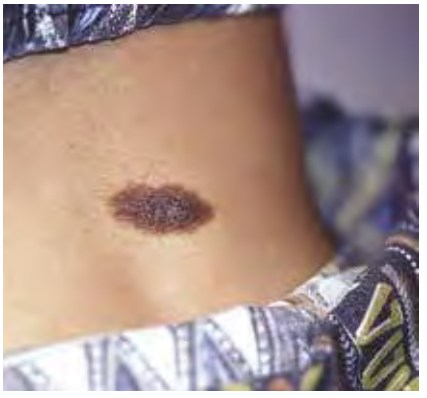
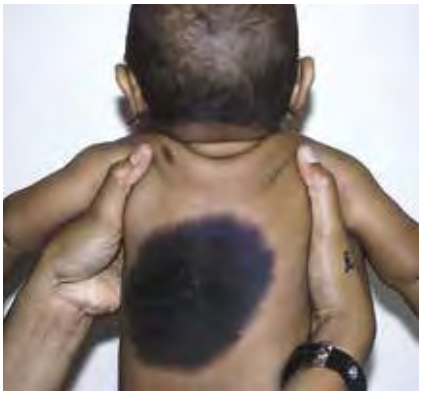
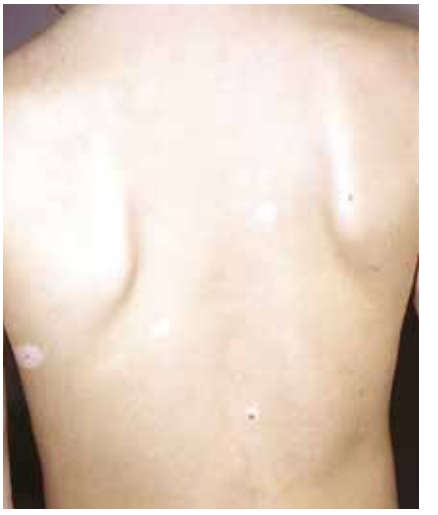




Figures 1a to c. Melanocytic naevi. a (left). Congenital melanocytic naevus. b (centre). Giant melanocytic naevus. c (right). Halo naevi, in which a white ring appears around a melanocytic naevus.

(Figure 1a). The size of melanocytic naevi is highly variable; congenital lesions tend to be larger and can be extensive.

Large congenital melanocytic naevi

Very large melanocytic naevi, with a projected adult size of 20 cm or more, are invariably present at birth, occur in about one in 20,000 births and are often covered in long dark hair (Figure 1b). They may be raised, with a pebbled surface and occasionally nodules. They may occur in a garment distribution on the trunk and may be surrounded by numerous satellite lesions.

Classically, this type of lesion is associated with early onset melanoma; however, this is still very uncommon even in the setting of large naevi. These large lesions, particularly when there are two or more discrete lesions, may also be associated with other abnormalities, including neurological problems. They may present a serious cosmetic issue particularly if located on the face.

Acquired melanocytic naevi

Nearly all children develop at least some melanocytic naevi after the age of 2 years; they can appear at any time up to the age of about the mid-20s. Most are completely harmless and do not require treatment. The number of lesions is determined by a combination of genetic potential and degree of sun exposure. Although melanocytic naevi are most common on sun-exposed sites, they can occur anywhere on the body.

Presentation, life cycle and complications

The appearance of melanocytic naevi is very variable. Colour varies from pink to brown to black, the latter being seen particularly in dark-skinned individuals. Naevi on the scalp often have a pale centre. The lesions may be associated with a variable amount of hair. The shape is usually round to ovoid, but the edge can be somewhat irregular.

It is not always appreciated that melanocytic naevi have a life cycle. It is normal for them to change slowly with time (over years) in size and colour, and eventually they involute so that older people have very few. At puberty they often darken, enlarge and may become hairy, and these normal changes often cause concern about melanoma.

Certain events may complicate all pigmented naevi. Occasionally, they may become itchy or swollen. In some cases, a white halo may appear around the naevus, termed halo naevus (Figure 1c). In children with atopy, dermatitis may localise around naevi. Pedunculated lesions may strangulate, making them look suddenly and temporarily black. All these changes are benign.

The risk of melanoma in children with small melanocytic naevi is negligible; melanoma in prepubertal children is very rare. In those with large congenital naevi, melanoma may uncommonly occur before puberty. In at least half the cases of melanoma occurring in patients with giant melanocytic naevi, the malignancy is found in areas other than the naevus. Therefore, removing the naevus does not completely remove the melanoma risk.

Melanoma risk is correlated with the number of naevi and family history of melanoma. Some families have a history of numerous, unusual-looking naevi that are associated with multiple melanomas. Termed 'dysplastic naevus syndrome', this is quite uncommon. Many patients with multiple naevi never have a melanoma.

Treatment of melanocytic naevi

Most melanocytic naevi do not require treatment. In children, the strongest indication for removal of most melanocytic naevi, particularly small ones, is not concern about their malignant potential but cosmetic embarrassment. Surgical removal of cosmetically distressing lesions can be considered before the child starts school. Sometimes the appearance of the lesion can be improved simply by removal of hair, if present. This may be achieved with hair removal laser therapy. The response to other laser therapy is poor.

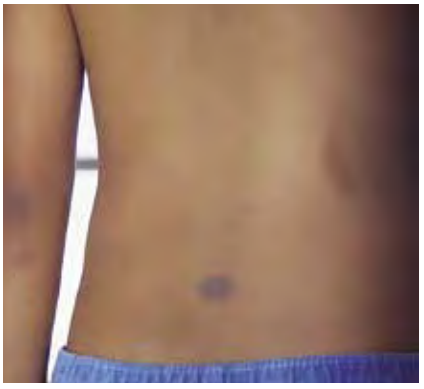


Figure 2. Mongolian spot, showing the typical appearance of a blue-grey macule on the lower back.

Removal of large lesions is rarely easy and may involve numerous complex surgical procedures. Laser treatment may be used, but results are variable. These cosmetic procedures still leave a cancer risk, and the cosmetic effect may not be lasting. Referral to a dermatologist is recommended for any newborns with large lesions.

Mongolian spot

Mongolian spot is a very common congenital lesion in Asian babies and is seen occasionally in children of European background. Typically, a Mongolian spot is a blue-grey macule, occurring most often on the lower back, although they can also be found on the limbs (Figure 2). Some are quite large. These lesions usually resolve spontaneously during the first decade of life.

Café au lait macule

Although most doctors think of neurofibromatosis when they see the flat, well-demarcated, light brown lesions of café au lait macules, these lesions are in fact common as solitary birthmarks and are benign and without significance (Figure 3). They are permanent and darken in summer with sun exposure. Neurofibromatosis needs to be considered only when six or more lesions are present, particularly if there is freckling in the axillae and groin.

Café au lait macules can be lightened with laser treatment but have a tendency to recur.

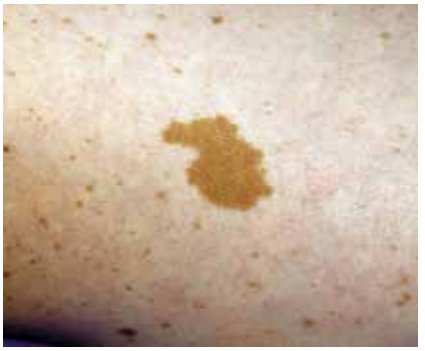


Figure 3. Café au lait macule.

Mosaic disorders of pigmentation

Not all birthmarks involving melanocytes are caused by increased numbers. Naevoid areas of hyper- and hypopigmentation are also part of the naevus spectrum. The patches are usually linear. These lesions may resemble epidermal naevi, but when examined closely are completely macular. Most are harmless. However, syndromes such as McCune-Albright syndrome, where macular hyperpigmentation is associated with bone and endocrine abnormalities, are well known.

Epidermal naevi

Epidermal naevi are the least common and most heterogeneous group of naevi. These lesions are composed of any element present in the epidermis and dermis, including keratinocytes (nonorganoid naevi) and glands (organoid naevi). This results in areas that are darker or lighter than surrounding skin. Epidermal naevi can be part of the epidermal naevus syndrome, which is associated with other abnormalities, particularly of the skeleton, eyes and central nervous system.

Epidermal naevi are not always obvious at birth. They may present for the first time in early childhood and then extend for several years. They can be linear, extending all the way down a limb (Figure 4a). Like melanocytic naevi, they may enlarge and become more problematic at puberty, mainly because of cosmetic and functional concerns. Malignancy is rarely a complication, except in patients with naevus sebaceous.

Genetically, epidermal naevi most often arise from mosaic and somatic postzygotic mutations. The genes involved have been elucidated in many of them.

Epidermal naevus syndrome

In epidermal naevus syndrome, epidermal naevi are associated with abnormalities of the skeleton, eyes and central nervous system. The naevi in this syndrome are often quite extensive, occurring over large areas of the body in linear whorls and streaks (Figure 4b). Children with large epidermal naevi should be referred to a paediatrician for evaluation.

Naevus sebaceous

Naevus sebaceous is the most common type of epidermal naevus, with an incidence of 1 in 1000 live births. Naevus sebaceous consists predominantly of sebaceous glands and usually occurs on the head and neck. It is usually present at birth, appearing as a hairless, orange-yellow plaque (Figure 5).

Unlike most other epidermal naevi, naevus sebaceous has a small malignant potential and may be complicated by carcinoma, most often basal cell carcinoma, in late teenage to adult life. Because of this, it may be wise to surgically remove these lesions prophylactically by the time the patient is 15 years old; they are rarely large or difficult to excise. They are also often removed for cosmetic reasons.

Verrucous epidermal naevi

Naevi composed of keratinocytes (verrucous epidermal naevi) have the appearance of warty linear streaks (Figure 6). They may be mistaken for warts or even lichenified dermatitis.

Treatment of epidermal naevi

If an epidermal naevus is localised, the best treatment is complete full-thickness excision. Laser therapy is helpful, but the lesions usually recur after treatment. In most cases, epidermal naevi are benign and only a cosmetic problem. However, in some locations, such as the genital area or fingers, they may become a functional

problem. These naevi are sufficiently unusual to warrant referral of affected patients to a dermatologist in most cases.

Vascular birthmarks

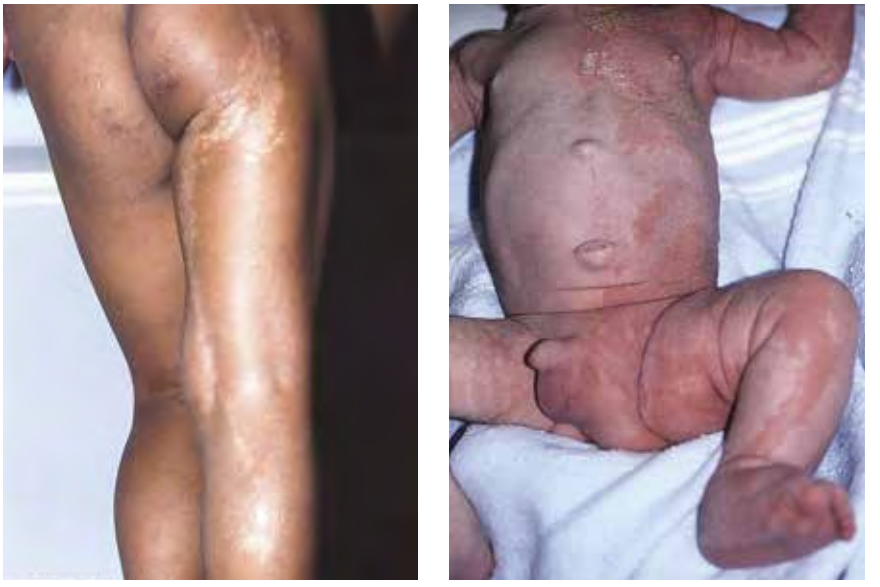
Haemangioma of infancy

Haemangioma of infancy (previously termed capillary haemangioma or strawberry naevus) is a common neoplasm affecting 10% of neonates (Figures 7a and b). It is more common in girls and premature babies. Haemangiomas are not usually present at birth but appear within the first month of life, often initially as an area of pallor, erythema or telangiectasia. There is then a period of growth, usually no longer than about six months. The lesions rarely reach very large proportions. After stabilising, they slowly regress, resolving substantially by the time the child is of school age and completely by the age of 9 years.

Many haemiangiomas of infancy are superficial, but some have a deep component. The characteristic appearance is of a bright red nodule, with the deep part appearing bluish and obviously below the skin surface. Sometimes, the entire lesion is deep, making it difficult to distinguish from a vascular malformation (see below). During regression, grey areas appear on the surface and are a good prognostic sign. The superficial portion usually resolves before the deep one does. In 20% of cases, there is more than one lesion.

Complications of haemangiomas

Most haemangiomas are uncomplicated and never require any form of intervention. When complications do occur, the most common is ulceration. This occurs most often under the nappy and on the lip. It is important to be aware of ulceration as a complication, because affected children are sometimes reported as cases of child abuse. Ulceration is usually treated conservatively with hydrocolloid dressings; however, persistently ulcerated lesions heal with oral propranolol. Referral to a dermatologist is recommended in cases such as these.



Figures 4a and b. Epidermal naevi. a (left). Epidermal naevi may be linear, extending down a limb. b (right). Large epidermal naevus; patients with such lesions should be referred to a paediatrician for evaluation.

Other complications of haemangiomas are unusual. Bleeding and infection occur rarely and, if dermatitis occurs, it may localise to the lesion.

Parents often report that the worst impact of their child's haemangioma, particularly if on a visible area such as the head or neck, is the comments it attracts from strangers in public places, especially supermarkets. Parents often need support and encouragement to develop resilience to this intrusion.

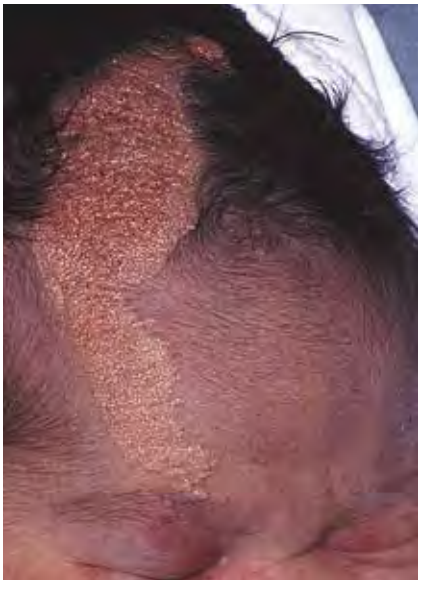


Figure 5. Naevus sebaceous. These lesions have a small malignant potential.

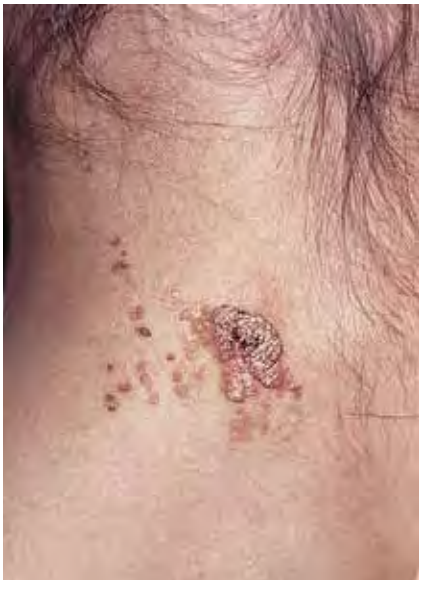
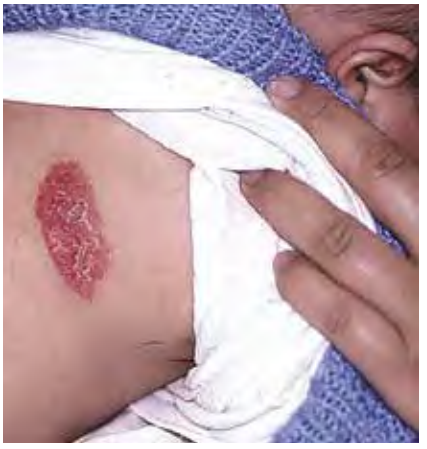
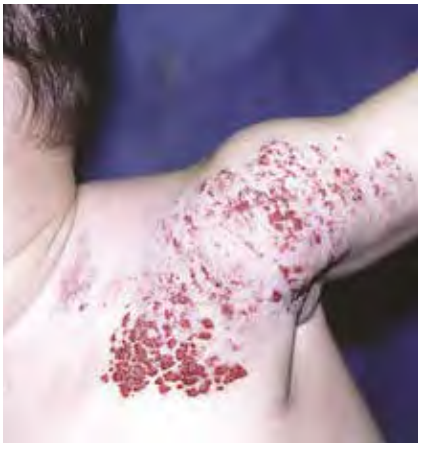


Figure 6. Verrucous epidermal naevus, showing the typical warty appearance.





Figures 7a and b. Haemangioma of infancy. This common neoplasm affects 10% of neonates.

Treatment of haemangiomas

Most haemangiomas do not cause problems and, because of their natural history, no treatment is recommended. However, doctors should beware the following situations:

- facial lesions that may become disfiguring if they enlarge
- lesions that interfere with an orifice (ear, nose, genitals or mouth)
- lesions that may occlude an eye (which may cause blindness)
- rapidly growing large lesions
- large, flat facial lesions around the mouth (these are associated with laryngeal haemangioma that can cause dangerous airway obstruction).

In these cases, treatment with oral propranolol is now gold standard to halt progression and speed up involution. Treatment should be started as early as possible, and urgent referral to a paediatrician or dermatologist is essential. Treatment with propranolol is highly effective, well tolerated and very safe. Smaller, flat lesions may be treated with topical timolol, but this is much less effective than oral therapy.

Any baby with a haemangioma of infancy should be closely followed up. Large haemangiomas of infancy may leave residual stretched tissue and telangiectasia, which may require later surgical intervention and pulsed dye laser therapy.

Naevus flammeus

Naevus flammeus, also known as a 'stork mark', is seen in 50% of newborns. These lesions are seen on the glabella, upper eyelids and nuchal area as irregular red macules that become more obvious when the infant cries.

When these lesions occur on the face, they invariably resolve by 12 months of age. However, those occurring on the nuchal area persist into adult life in many cases and are found in 10 to 20% of adults.

Capillary malformation

Capillary malformation (port wine stain) is encountered in one in 1000 births (Figure 8). Although present at birth, a capillary malformation is not always obvious and may not be diagnosed for several months.

In capillary malformation, the lesion consists of excess superficial capillaries that were formed early in fetal life. Because of this, the cutaneous lesion may be the 'tip of the iceberg', with underlying abnormalities of vessel, soft tissue, nerve and bone. Unlike haemangiomas, capillary malformation lesions are permanent, and do not undergo a growth and resolution phase. Generally, they tend to darken with age and may become thickened, particularly at puberty.

Complications of capillary malformations

Capillary malformations on the face in the distribution of the first trigeminal nerve, particularly if involving the upper eyelid, may be associated with epilepsy and ocular abnormalities (Sturge-Weber syndrome). When found on the midline over the lumbosacral spine, they may be associated with abnormalities of the lower spinal cord.

Lesions on the lower legs may be associated with soft tissue hypertrophy and underlying vascular anomalies (Klippel-Trenaunay syndrome). When such a lesion feels warmer than surrounding skin and is associated with soft tissue hypertrophy or pulsation, it may in fact be an arteriovenous malformation.

Children with these rare lesions should be referred to a paediatrician. They will usually require MRI investigation.

Treatment of capillary malformations

Capillary malformations may be treated by pulsed dye laser, and this is best done as early as possible in the child's life as response is best in the first two years. Children with capillary malformations should be referred to a paediatric dermatology unit at a children's hospital. Treatment may be performed very early, even in the first month of life. This is limited by the pain of the procedure and the risk of general anaesthesia in a baby. The result of laser treatment is usually not perfect; however, a substantial number of children achieve some degree of lightening.

Extensive lesions require imaging to determine whether there are underlying abnormalities. Patients with lesions on the face may require MRI of the brain and ophthalmology referral.

Other vascular malformations

Other types of vascular malformation are rare and consist of a mixture of subcutaneous venous and lymphatic elements. Again, they form early in fetal life. In the past, they were termed cavernous haemangiomas.

These lesions may be small or large, sometimes infiltrating muscles and joints. Their appearance is highly variable, but usually there is obvious hypertrophy and sometimes visible enlarged veins, discolouration or bruising. When the lesion is predominantly lymphatic, it may be

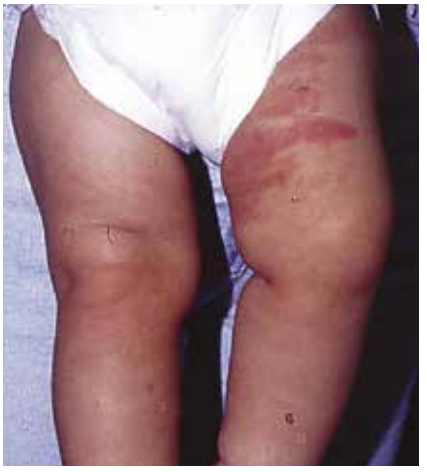


Figure 8. Capillary malformation (port wine stain).

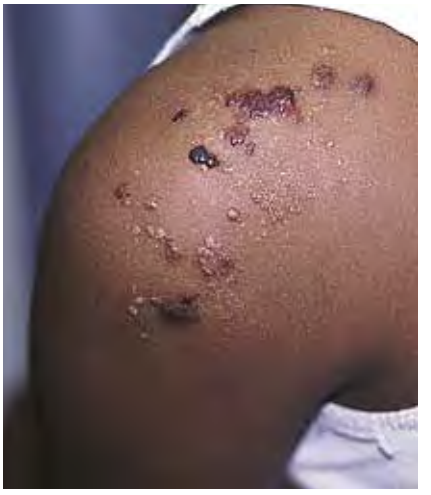


Figure 9. Lymphatic vascular malformation.

repeatedly infected or leak clear fluid (Figure 9). Ultrasound or MRI is required to determine the exact nature of the lesion.

Previously, these lesions were generally treated with surgical excision, often with great difficulty and variable results. More recently, interventional radiologists have used sclerosant therapy under fluoroscopic guidance. Some lasers, such as the long-pulsed neodymium:yttrium-aluminium-garnet (Nd:YAG) laser, have enough penetrance to treat intracutaneous and superficial malformations. These modalities are available at only a few major centres with specialised expertise.

Conclusion

Although it is unusual for babies to be born with skin lesions, nearly all children have at least some melanocytic naevi after the age of 2 years. Birthmarks are rarely dangerous and hardly ever have malignant potential but may be cosmetically embarrassing, particularly if they occur on the face. Some birthmarks, particularly the lesser known epidermal naevi, may be mistaken for other lesions such as warts or even dermatitis. Ulcerated haemangiomas may be mistaken for child abuse. Some lesions can interfere with function.

Most birthmarks do not resolve spontaneously in childhood; however, the common haemangioma of infancy always does and so usually does not need treatment. If they are small, most melanocytic and epidermal birthmarks can be easily removed surgically, with or without the aid of laser therapy, if the child and parents wish it. However, removal of larger lesions can present a significant challenge.

Vascular lesions are often the most difficult to treat because they are too large to be excised, and the visible component on the skin surface may be the tip of the iceberg. A combination of surgery, laser and sclerotherapy is used to treat these.

Although malignant potential and interference with function are important, the most common reason for removal of a congenital lesion is cosmesis. With the exception of laser therapy for capillary malformations, which is most effective early in life, the decision to have treatment can, and should, involve the wishes of the child. MT

Further reading

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Folliculitis

Diagnosis and management of subtypes

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Folliculitis is a common disorder, although the exact prevalence is unknown. It presents as erythematous pustules surrounding hair follicles. It may be due to a superficial or deep infection of the follicle, or may be secondary to trauma. Deeper infections present as sycosis and folliculitis decalvans. Good grooming and hygiene are key to treatment and long-term resolution, alongside pharmacological therapies.

KEY POINTS

- Folliculitis is characterised by inflammation of the hair follicle, with formation of a pustule.
- Superficial folliculitis may arise from mechanical or chemical irritants, such as during hair removal and grooming, or infection with *Staphylococcus aureus*. It is usually self-limiting and requires little pharmacological intervention.
- However, deeper infections can result in more persistent and recurrent subtypes of folliculitis such as sycosis or folliculitis decalvans.
- The mainstays of treatment are hygiene and grooming, alongside antibiotics or antifungals depending on the causative agent and subtype.

Folliculitis is characterised by inflammation of the hair follicle.[1] The result is a tender red papule or nodule, often with a surface pustule (Figure 1) that may be superficial or deep, which may occur in any location where there is a hair follicle.[1] The exact incidence of folliculitis is unknown as most patients do not seek medical attention. However, superficial folliculitis caused by *Staphylococcus aureus* is most common in childhood and usually occurs on the scalp or limbs.[2]

Pathophysiology

Superficial folliculitis is not always primarily infective in origin. Physical or chemical injury to the skin can cause folliculitis, which results in the formation of sterile pustules or papules that contain coagulase-negative staphylococci. *S. aureus* superficial folliculitis is an infection of the follicular ostium.[1]

Occupational contact with mineral oils or tar products can cause folliculitis. A traumatic folliculitis may develop after epilation, and a sterile folliculitis may develop beneath adhesive dressings.[1,3] Other forms of folliculitis are triggered by medications or underlying disease, with a postulated mechanism of a hypersensitivity reaction.

Presentation and prognosis

Follicular lesions present as papules or pustules, and more persistent lesions can occur on the thighs and buttocks of adolescent males.[2] Pustules develop in crops and heal within seven to 10 days, but can become more chronic. Deeper infections may develop as furuncles or sycosis. Recurrent or chronic staphylococcal folliculitis may develop into folliculitis decalvans. Patients who come into close contact with other people may spread the condition, for

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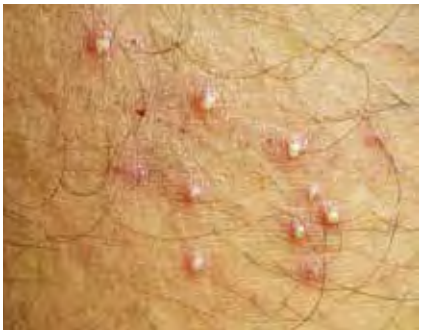

Figure 1. Superficial folliculitis.

example athletes competing in team sports, military personnel and prisoners.[4]

Folliculitis is usually self-limiting and settles within a few days. However, some folliculitis subtypes can be recurrent and persistent, as will be discussed in more depth below and summarised in Table 1.[1]

Differential diagnosis

Differential diagnoses are summarised in Table 2. Pustular miliaria presents as a widespread papulopustular eruption on normal skin in hot and humid conditions and may be confused with folliculitis.

Subcorneal pustular dermatosis develops as follicular pustules grouped around the margins of plaques of erythema and scaling.

Pustular psoriasis or a pustular adverse reaction to medications (such as to epidermal growth factor receptor inhibitors) presents as pustules on a circumscribed red and oedematous or scaling plaque. Follicular graft-versus-host disease can be considered in the context of bone marrow transplantation.

Investigations and management

Investigations are usually not required, although swabs can be taken to assess sensitivity of the causative organism. Most cases of folliculitis can be managed through avoidance of irritants and good hygiene; however, some patients may also benefit from pharmacological treatment. Superficial folliculitis of chemical or physical origin will settle with removal of the irritant. Mild staphylococcal folliculitis is usually

TABLE 1. FOLLICULITIS SUBTYPES

Subtype	Location	Cause	Treatment
Superficial folliculitis	Any area of the body where hair follicles are present	*Staphylococcus aureus*	Antibiotics
Pseudofolliculitis	Areas of shaving/depilation, especially anterior neck/skin folds	Mechanical or chemical irritation	Hygiene and alteration in grooming care
Pityrosporum folliculitis	Shoulders, back	*Malassezia*	Topical antifungals
Gram-negative folliculitis	Perioral, perinasal regional	*Pseudomonas, Klebsiella*	Antibiotics
Viral folliculitis	Any area of the body where hair follicles are present	Herpes simplex virus, herpes zoster virus, molluscum contagiosum virus	Antivirals, curettage/cryotherapy
Sycosis	Upper lip, angle of jaw, chin	*Staphylococcus aureus*	Antibiotic ointment, chronic forms treated with corticosteroid-antibiotic combination
Folliculitis decalvans	Vertex of scalp	*Staphylococcus aureus* implicated	Antiseptic shampoo, topical clindamycin, oral tetracyclines, or oral clindamycin and rifampicin
Eosinophilic pustular folliculitis	Spares mucous membranes	Associated with AIDS, malignancy	Topical corticosteroids, UVB phototherapy
Perioral folliculitis/dermatitis	Perioral region	Topical corticosteroid use	Topical metronidazole, topical calcineurin inhibitors, oral tetracyclines
Folliculitis keloidalis nuchae	Occiput	Chronic irritation	Minimising mechanical trauma, corticosteroids, retinoids and tetracyclines
Dissecting cellulitis/Hoffman disease/perifolliculitis capitis abscedens et suffodiens	Scalp	Unknown	Zinc sulfate, antibiotics, isotretinoin, corticosteroids
Epidermal growth factor receptor inhibitor-associated folliculitis	T-zone of the face	Epidermal growth factor receptor inhibitors	Oral tetracyclines

TABLE 2. DIFFERENTIAL DIAGNOSES OF FOLLICULITIS

Differential diagnosis	Features
Pustular miliaria	Widespread papulopustular eruption on normal skin
Subcorneal pustular dermatosis	Follicular pustules grouped around erythematous plaques
Pustular psoriasis Pustular adverse drug reaction	Pustules on a red, oedematous/scaly plaque
Follicular graft-versus-host disease	Patient history of bone marrow transplantation

self-limiting and may respond to topical antiseptics or cleansing.[1] Hand washing and hygiene are critical in preventing transmission of community-acquired methicillin-resistant *S. aureus* infections.[1]

Subtypes

Pityrosporum folliculitis

Also known as *Malassezia* folliculitis, pityrosporum folliculitis results from the growth of the yeast *Malassezia* within hair follicles and is the most common cause of fungal folliculitis.[5,6] It presents as an itchy monomorphic acneiform eruption with many papules or pustules (Figure 2).[5] Sometimes the rash will settle in a matter of days, but usually it will become chronic unless treated.[6] There is a predisposition to *Malassezia* folliculitis in adolescence due to increased activity of sebaceous glands. The rash is distributed over the shoulders, neck and back. Diagnosis can be confirmed via a potassium hydroxide stain.[6] Treatment with topical antifungals is helpful.[5,7] Some patients require oral antiyeast agents to settle inflammation.[6,7] In this setting itraconazole works well.[7]

Viral folliculitis

This subtype is most commonly caused by the herpes viruses (simplex and zoster), but may also be triggered by the pox virus molluscum contagiosum.[6] A clinical clue to a viral cause of folliculitis is the presence of papulovesicles or plaques rather than pustules. Another clue is that lesions appear in clusters and are often preceded by a burning sensation.[6] Molluscum contagiosum-induced folliculitis more commonly occurs on the face, and is often preceded by hair removal or shaving.[6]

Treatment is via oral aciclovir, valaciclovir and famciclovir.[6] Molluscum contagiosum-induced folliculitis can be treated with curettage or cryotherapy, although it often resolves spontaneously.[6]

Gram-negative folliculitis

Gram-negative folliculitis has the moniker of 'hot tub folliculitis' as it infamously arises from the bacteria *Pseudomonas aeruginosa* after exposure to an improperly treated swimming pool or hot tub.[6,8] Other implicated organisms include *Klebsiella, Proteus, Citrobacter* and *Enterobacter* species, as well as *Escherichia coli*.[6] Patients taking long-term oral antibiotics that inhibit Gram-positive organisms, such as for the treatment of acne, are predisposed to this folliculitis.[1,9]

Clinically, an eruption of multiple small follicular pustules localised to the perioral or perinasal skin occurs (Figure 3), and sometimes these appear on the trunk, mimicking pityrosporum folliculitis.[1,9] Antibiotics that target Gram-negative organisms may clear the folliculitis over a few weeks. Isotretinoin can be used in cases of recalcitrant Gram-negative folliculitis, especially in patients with acne or rosacea, and has a lower relapse rate than other treatment options.[7,10]

Pseudofolliculitis

Pseudofolliculitis is a foreign-body inflammatory follicular and perifollicular reaction.[1,11,12] The described process is of curved hair emerging from the skin surface and then re-entering the skin to form an in-growing hair (extra-follicular penetration).[11,12] Alternatively, closely shaven hair may retract into the hair follicle and then grow into the sides of the follicle (transfollicular penetration).[11,12] Pseudofolliculitis more commonly occurs in people with curly hair and those with Asian and African ancestry.[1,11,12] A genetic defect (a single nucleotide substitution in the hair follicle companion layer-specific keratin gene *K6hf*) has also been implicated, with these patients having six times the risk of developing this condition.[11,12]

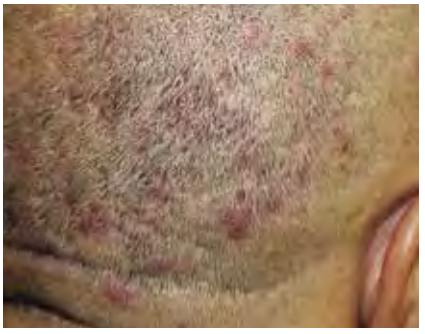

Figure 2. *Malassezia* folliculitis over the scalp.

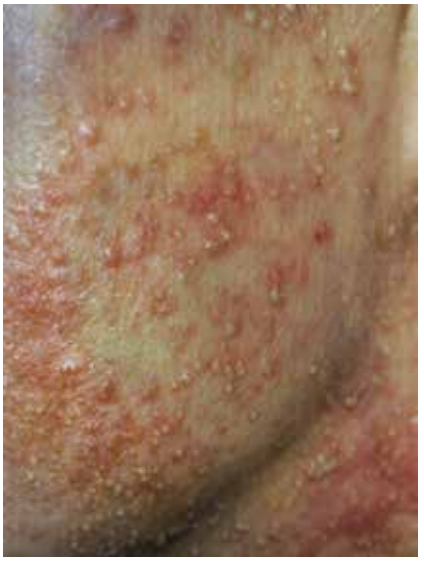

Figure 3. Gram-negative folliculitis. Image reproduced with permission of Professor Gayle Fischer, Sydney, Australia.

FIGURE 2 © DERMATOLOGY11/SHUTTERSTOCK

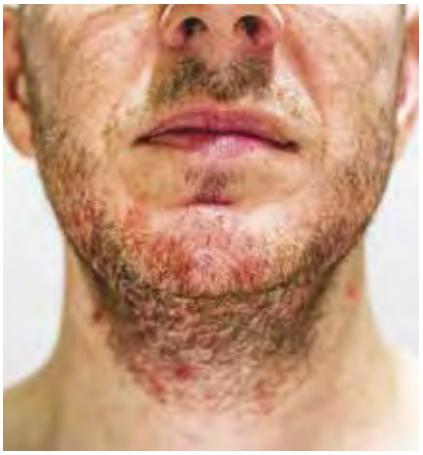

Figure 4. Pseudofolliculitis barbae in the typical neck fold and angle of the jaw areas.

It presents as multiple small papules and pustules on shaven skin (Figure 4). It classically affects the beard area (pseudofolliculitis barbae), especially the neck, which may scar with keloid formation and hyperpigmentation, as well as the lower legs and groin.[1,11,12] Differential diagnoses include bacterial folliculitis such as sycosis barbae and dermatophytosis.

Pseudofolliculitis tends to be a chronic condition with a relapsing and remitting course. Stopping shaving for four to six weeks allows inflammation to settle; however, resumption of shaving or waxing will often lead to relapse. Alternative methods of grooming to avoid pseudofolliculitis include shaving or clipping hair to a length of 1mm, or performing hair removal with chemical depilatories or lasers.[11] Chemical depilatories lyse the disulfide bonds in the hair shaft, thus weakening them and making them less likely to cause extra or transfollicular penetration and thus a foreign-body reaction.[11] Other treatments include topical and oral antibiotics, topical retinoids, chemical peels and electrolysis.[12] The mainstay of treatment traditionally is either to shave the affected area with preshave hair hydration sufficiently close to the skin to prevent extrafollicular penetration and use postshave moisturisation, or to allow the hair to grow out.[12]

Sycosis

Sycosis is a subacute or chronic pyogenic infection involving the whole depth of the follicle.[1] It most commonly affects men in the third or fourth decade of life and usually refers to disease in the beard area (sycosis barbae). The most common infecting organism is *S. aureus*, although infection with other Gram-positive or Gram-negative bacteria (including *Treponema pallidum*, which causes syphilis), as well as fungi, yeasts, parasites and viruses have been reported.[13] Indoor workers are affected more often than outdoor workers.

The affected follicle is packed with polymorphonuclear leukocytes that infiltrate the follicular wall. Around the follicle is chronic granulomatous infiltrate in which lymphocytes, plasma cells, histiocytes and foreign-body giant cells aggregate. The sebaceous gland or whole follicle may be destroyed and replaced by scar tissue.[2]

The primary lesion is an oedematous erythematous follicular papule or pustule centred on a hair. If neighbouring follicles are involved, the perifollicular oedema may coalesce to produce raised plaques studded with pustules (Figure 5). In subacute forms, the lesions are grouped or scattered around the upper lip and below the angles of the jaw. Chronic lesions cluster into plaques, especially on the upper lip and chin. There is often some crusting and scaling but the hairs are retained and there is no evident scarring.[2]

In lupoid sycosis, the follicles are destroyed by scarring and are surrounded by active papules and pustules. Granulomatous inflammatory changes give the papules a lupoid appearance. Lupoid sycosis tends to begin under one ear or under the chin and extend irregularly in any direction. It may extensively involve the scalp. Lupoid sycosis tends to persist indefinitely.[2]

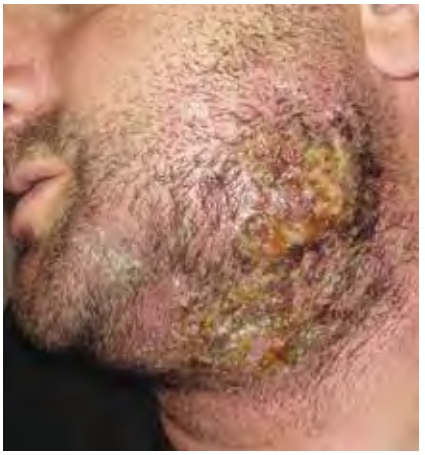

Figure 5. Sycosis barbae.

Differential diagnoses include pseudofolliculitis, although pseudofolliculitis tends to be irregularly scattered over the neck and angles of the jaw and within skin folds. Tinea barbae usually occurs on the chin, mandible or upper lip as an oedematous plaque of grouped pustules.

Bacterial swabs may confirm the presence of *S. aureus* and treatment with antibiotic ointments is typically effective. Chronic forms of sycosis may require a combination of corticosteroids and systemic antibiotics. Other treatment includes fractional radiofrequency microneedle treatment.[13]

Folliculitis decalvans

Although uncommon, folliculitis decalvans is the most common form of neutrophilic scarring alopecia.[14] It causes painful, recurrent exudative folliculitis that most commonly involves the vertex of the scalp.[14,15] Its pathogenesis is not fully understood, although *S. aureus* has been isolated from the pustules.[15,16] The folliculitis penetrates more deeply within the follicle and produces a scarring alopecia that spreads to neighbouring follicles.[15]

Early onset is associated with a more severe form of the disease.[16] Folliculitis decalvans initially presents as painful follicular pustules that become crusted (Figure 6). Tufted hairs are also identified (more than five hairs exiting from the same

follicular opening). It expands outwards with a central area of scarring.[15,16] Men are affected from adolescence onwards and women from their thirties.

Treatment is mainly aimed at eradicating *S. aureus*. Antiseptic shampoos and topical clindamycin are sufficient to treat mild cases. One systematic review of the efficacy of folliculitis decalvans treatments across 20 studies suggested that the most commonly used treatment is a 10-week course of clindamycin and rifampicin.[14] There is some evidence, however, that maximal treatment effect is obtained through rifampicin and clindamycin, tetracyclines and intralesional corticosteroids, although individual clinician treatment choices vary widely.[14,16] Some patients require long-term treatment with oral tetracyclines to control this disease.[17]

Eosinophilic pustular folliculitis

Eosinophilic pustular folliculitis was first described in the 1960s as a case of a woman with recurrent follicular pustules on her face and back, with a peripheral eosinophilia noted on her blood test results and an infiltrate of eosinophils on histology.[18] A syndrome was later defined as pruritic sterile follicular papulopustules with central clearing and peripheral extension and resolution, with residual pigmentation followed by recurrence.[18] The syndrome was also required to be chronic, have an absence of systemic symptoms, and spare the hands, feet and mucous membranes.[18] However, subsequent data suggested that around 20% of cases involve the hands and feet.[18] Associated diseases include AIDS and malignancy (lymphoma, leukaemia and naevoid basal cell carcinoma syndrome).[18] However, some researchers advocate for AIDS-associated folliculitis to be considered a distinct entity.

Eosinophilic pustular folliculitis tends to affect males more than females (4.8:1), with a peak incidence in the third and fourth decades of life. The pathogenesis is thought to relate to an aberrant T helper-2-type immune response to follicular antigens such as contact dermatitis, infections or medications.[18]

There is no strong evidence for any single treatment type, but typical first-line therapy is topical corticosteroids.[18] The optimal treatment might be UVB phototherapy with no reported treatment failures.[18] Other treatments include indomethacin, itraconazole, metronidazole, topical calcineurin inhibitors and oral antihistamines.[18] In cases where HIV AIDS is implicated, antiretroviral therapy has been noted to improve the associated folliculitis.

Although topical corticosteroids provide initial benefit [in treating perioral folliculitis], they lead to rebound flaring and worsening of disease

Perioral dermatitis

A perioral folliculitis or dermatitis is primarily induced by topical corticosteroids.[19,20] Other causes include infections, toothpaste and cosmetic preparations.[20] Perioral dermatitis presents with crops of pruritic, erythematous papules that are usually grouped around the mouth, but can also occur around the eyes and nostrils.

Topical corticosteroids will initially control the disease, with rebound disease when the corticosteroid is ceased. Thus, treatment with corticosteroids should be avoided. Long-term corticosteroid use will result in more severe perioral dermatitis.[19,20]

This condition can be managed through anti-inflammatory antibiotics including topical metronidazole, erythromycin or clindamycin, topical sulfur preparations, or azelaic acid gel.[19,20] Topical calcineurin inhibitors including tacrolimus ointment or pimecrolimus cream have also been used.[19,20] Oral tetracyclines can be used in more moderate and severe disease and taken for up to two to three months.[20] The important point to remember is that although topical corticosteroids provide initial benefit, they lead to rebound flaring and worsening of disease.

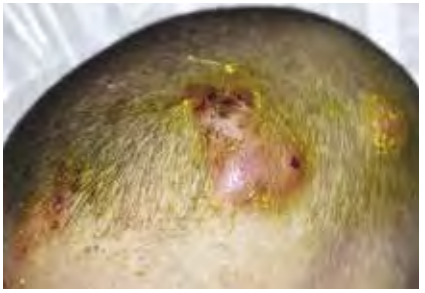

Figure 6. Folliculitis decalvans affecting the scalp.

Folliculitis keloidalis nuchae

Folliculitis keloidalis nuchae, also known as acne keloidalis nuchae, is a chronic inflammatory condition that leads to fibrotic plaques and alopecia over the occiput.[21,22] It is worth noting that, although the term 'keloidalis' is used to describe the condition, there are no keloids associated with it and affected individuals do not have a greater chance of developing keloids.[22] It is also worth noting that the term 'acne keloidalis nuchae' is sometimes preferred, as the condition can extend beyond the occipital area.[22] It is typically seen in postpubertal men of African descent and is rare in women and those aged over 55 years.[21,22] The cause is unknown but is thought to be an aberrant immune response to predisposing factors including androgens, chronic inflammation (such as from shirt collars, helmets and combs), infection, trauma and ingrowing hair.[21,22]

Folliculitis keloidalis nuchae presents as papules, pustules and tumourous masses in the nuchal or occipital regions of the scalp (Figure 7).[21,22] Often these arise within days after a haircut or other irritation. Pruritis, pain and bleeding are associated with active lesions and secondary bacterial infection is common.[21,22] Chronic inflammation leads to fibrosis and large plaques and nodules.[21]

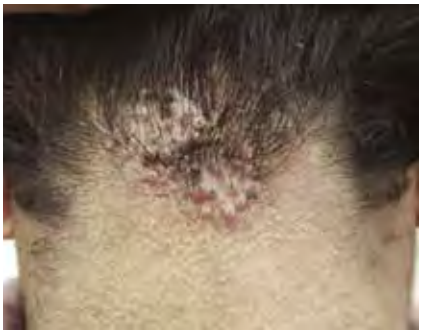

Figure 7. Folliculitis keloidalis nuchae in a typical distribution.

Figure 8. Perifolliculitis capitis abscedens et suffodiens.

The overall goal of management is to prevent the development of further lesions and subsequent alopecia. Nonpharmacological measures include avoidance of close shaving, helmets and tight collars.[21,22] Haircuts should be avoided when lesions are active and bleed easily, because of the risk of blood-borne disease transmission.[21] Antiseptic shampoos may have a role in preventing secondary infections.[21,22] Mild disease can be treated with topical corticosteroids and topical retinoids, whereas moderate disease may require intralesional triamcinolone or systemic corticosteroids.[21,22] More severe or chronic disease will benefit from long-term oral tetracyclines and retinoids. Surgical excision of affected areas can work well, although recovery time is longer and lesions may recur.[21,22] It is thus better used in chronic and refractory disease.[21] Ablative laser therapy is an emerging treatment that has been reported to work well in treating folliculitis keloidalis nuchae, although it is typically less available in Australia.[21]

Perifolliculitis capitis abscedens et suffodiens

Also known as dissecting cellulitis or Hoffman disease, this condition presents with perifollicular pustules, nodules, abscesses and sinuses (Figure 8).[23] The end result is a scarring alopecia.[23] It predominantly affects men of African descent aged 20 to 40 years, and has been associated with arthritis, keratitis, Crohn's disease, pyoderma gangrenosum and keratitis-ichthyosis-deafness syndrome.[23] The aetiology is unknown, although it is classed within the follicular occlusion tetrad, along with acne conglobata, pilonidal cysts and hidradenitis suppurativa.[23]

Treatment includes zinc sulfate or isotretinoin in combination with oral antibiotics for three to 12 months. Oral prednisone can be helpful as an adjunctive agent.[23] Newer therapies include adalimumab and infliximab, and ablative lasers targeting the hair follicle.[23] Surgery can achieve cure, but is not always successful and has higher morbidity than pharmacological treatments.[23] Often bacteria can be isolated on culture of the lesions, but targeting them for treatment has an unclear effect on disease resolution.[23]

Folliculitis secondary to medications

It is well-recognised that certain drugs including protein kinase inhibitors or epidermal growth factor receptor inhibitors (EGFRIs) as well as vemurafenib and dabrafenib for metastatic melanoma, cause a folliculitis. EGFRIs are used for colonic, rectal and pulmonary cancers, and include the monoclonal antibodies cetuximab and panitumumab, and the tyrosine kinase inhibitors erlotinib and gefitinib.[24] More than 50% of patients on EGFRI treatment experience a folliculitis, and this is the most common dermatological side effect.[24] It tends to occur between one and four weeks after commencement of treatment and then slowly resolves. Exacerbating factors include sun exposure, radiotherapy and xerosis.[24] Management is mainly via oral tetracyclines and usually achieves a good result.[24] Starting the tetracycline antibiotic before the development of the folliculitis often reduces the incidence and severity.

Conclusion

Folliculitis is a common dermatological disorder that typically involves inflammation of the hair follicle; however, there are numerous subtypes that vary in their aetiology, presentation and prevalence. In most cases of folliculitis, resolution of disease can be achieved through good grooming and hygiene, and treatment with appropriate antibiotic or antifungal agents. MT

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A list of references is included in the online version of this article (www.medicinetoday.com.au).

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Folliculitis

Diagnosis and management of subtypes

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An acute generalised blistering eruption

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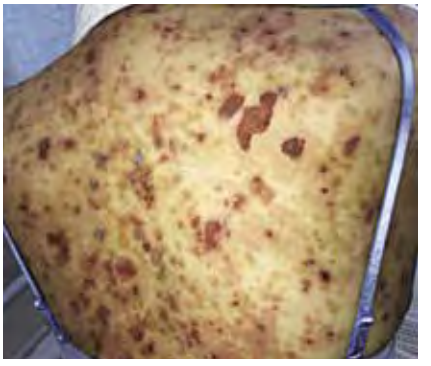
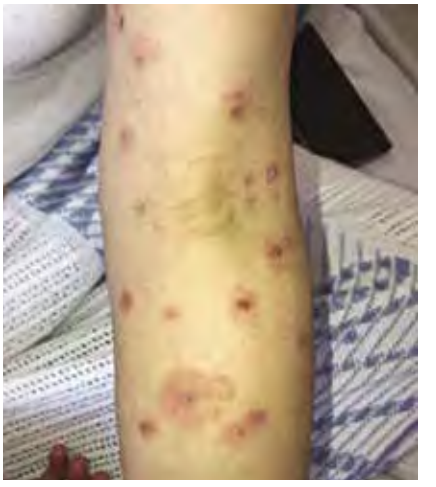
Test your diagnostic skills in our regular dermatology quiz. What is the cause of this gradually progressive skin eruption in an otherwise healthy 54-year-old woman?

Case presentation

A 54-year-old woman presents with a one-month history of a gradually progressive, blistering eruption involving her trunk, arms and legs (Figures 1a and b). She has not started any new medications and has no other medical conditions.





Figures 1a and b. Case patient. a (above). Crusted lesions on the posterior trunk. b (right). Bullae on an erythematous base on the arm.

Differential diagnoses

Conditions to consider among the differential diagnoses for an acute blistering eruption include the following.

- **Erythema multiforme (EM).** This acute self-limiting mucocutaneous eruption most commonly occurs as a result of infection, with herpes simplex virus (HSV) being the most common causative organism.[1] Patients with EM minor are usually well; prodromal fever, myalgia and malaise are seen in EM major, a more severe clinical entity of EM that has far more extensive mucosal involvement.[1] EM initially presents with pruritic and painful urticarial plaques that then develop into the characteristic erythematous concentric targetoid lesions that affect the distal extremities and favour extensor surfaces. The lesions show a large degree of morphological variability, with some atypical EM lesions presenting as haemorrhagic plaques that subsequently develop into blisters with crusting.[1] Biopsy results are dependent on the evolutionary stage of the lesion, with epidermal necrosis a prominent finding in EM bullous lesions.[1]
- **Bullous pemphigoid (BP).** This autoimmune disease presents initially with pruritic urticarial plaques affecting the trunk and limbs that evolve into tense blisters.[2] BP is the most common autoimmune vesiculobullous disease and most commonly affects individuals in the seventh decade of life and over.[2] Disease is caused by circulating antibodies to bullous pemphigoid antigen 2 (BP180), an adhesional structure inherent to hemidesmosomes that tether basal keratinocytes to the basement membrane, resulting in subepidermal blisters.[2] Biopsy is diagnostic and shows subepidermal blistering with a neutrophilic infiltrate; perilesional direct immunofluorescence shows linear deposition of IgG and complement C3 along the basement membrane.[2]
- **Bullous viral exanthem.** HSV, coxsackievirus, varicella-zoster virus, echovirus, enterovirus and adenovirus can result in vesiculobullous eruptions, which are commonly accompanied by systemic signs such as fever and lymphadenopathy.[3] Eczema herpeticum occurs as a result of disseminated HSV-1 infection, resulting in vesiculopustular lesions on an erythematous base in an unwell patient with coexisting skin disease or immunosuppression.[3] Coxsackieviruses A4 and A9 result in an acute onset of yellow vesicular

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crops and blisters affecting the face and trunk with accompanying fever.[3] Varicella should always be suspected in an adult with a widespread, progressive vesiculobullous eruption in varying stages of evolution because of the increased risk of varicella hepatitis, encephalitis and pneumonitis.[3]

- **Bullous impetigo.** This is an acute blistering disease caused by exfoliative toxins (ETs) from *Staphylococcus aureus* infection. Local production of ETs at the site of infection results in acute, erosive, crusted lesions on an erythematous base affecting the face, torso, axillae and extremities in a patient who is otherwise well.[4] Immunosuppressed individuals, such as patients with end-stage renal failure, are at risk of staphylococcal scalded skin syndrome, in which systemic dissemination of ETs cause bullous eruptions distal to the site of infection, and has a mortality rate of 60% in adults.[4] ET-A and ET-B cause proteolysis of desmoglein-1, a desmosomal glycoprotein involved in intercellular adhesion, affecting keratinocytes in the stratum granulosum.[4] Patients are highly contagious because of the subcorneal proliferation of *S. aureus*. Histopathological features of bullous impetigo include subcorneal blisters with an accompanying neutrophilic infiltrate in the superficial epidermis, while Gram staining shows Gram-positive cocci.[4]
- **Pemphigus foliaceous.** This is the correct diagnosis. Pemphigus foliaceous (PF) is a rare autoimmune disease affecting the superficial epidermal layers, in which autoantibodies to desmoglein-1 results in subcorneal acantholysis.[5] PF can affect individuals of all ages, although it most commonly occurs in the sixth and seventh decades of life.[5] Sporadic and endemic forms of PF exist – the latter most commonly occurs in rural Brazil, where it is known as Fogo Selvagem.[5] Penicillins, ACE inhibitors, NSAIDs and barbiturates can also cause a drug-induced form of PF.[5] PF presents acutely with painful, pruritic patches and erosions on an erythematous base affecting the head and trunk but spares the lower limbs.[5] Depending on the extent of disease, these patients are usually well and have a positive Nikolsky sign, which describes induced detachment of the epidermis by mechanical pressure applied parallel to the skin surface (e.g. firmly pressing finger along the skin). Oral mucosal involvement in PF is rare, unlike the more serious form of this disease, pemphigus vulgaris.[5] Pemphigus erythematosus is a clinical variant of PF that results in a facial butterfly-like distributed eruption affecting the malar skin, which may appear similar in morphology to that seen in systemic lupus erythematosus.[5]

Diagnosis

Biopsy is diagnostic for PF and will show acantholysis with an accompanying eosinophilic spongiosis, whereas direct immunofluorescence of perilesional skin shows IgG in the subcorneal epidermis.[5] Serum autoantibodies to desmoglein-1 correlate with disease activity and are useful to diagnose PF and monitor response to therapy.[5]

Management

Pemphigus is a rare disease that tends to be chronic and difficult to treat. The treatment approach to PF has three phases: control (reduce number of new lesions), consolidation (achieve healing in 80% of lesions) and maintenance (prevent new lesions).[5] The mainstay of treatment is oral corticosteroids, with other modalities such as pulsed methylprednisolone or plasmapheresis reserved for cases resistant to oral corticosteroids of 120 mg daily.[5] Off-label use of adjuvant immunosuppressant medications such as azathioprine, cyclosporin, cyclophosphamide, methotrexate and mycophenolate may be used with systemic corticosteroids in refractory cases. Off-label use of rituximab has been described as being useful in halting disease progress.[6] Topical emollients should be used to restore the skin barrier and prevent superimposed skin infections.

Outcome

This patient was admitted to hospital for investigation and management. A skin biopsy for histopathology and direct immunofluorescence was performed, which returned an acantholytic picture with intraepidermal clefting and positive intracellular IgG and C3. Pemphigus antibodies were detected by indirect immunofluorescence in blood. A pre-immunosuppression screen was performed with all parameters normal. She was diagnosed with PF and started on prednisone 50 mg daily, with rapid improvement. Initial treatment with rituximab resulted in a rapid improvement of her existing skin lesions. Treatment was subsequently changed and she remains on a slow taper of prednisone with azathioprine 100 mg daily as a corticosteroid sparer. MT

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COMPETING INTERESTS: None.

Persistent pruritic purple papules

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Test your diagnostic skills in our regular dermatology quiz. What are these itchy lesions, which first began to appear on a patient's back and hands?

Case presentation

A 48-year-old woman presents with a two-month history of intensely itchy papules on her back and hands. More recently, the lesions have begun to appear on her lower limbs and feet (Figure), occurring over bony prominences and after minor trauma. The lesions are hyperpigmented, violaceous, polygonal papules with a flat top.

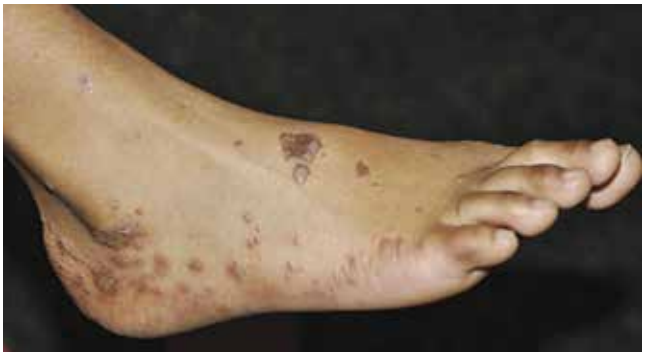

Figure. Lesions on the dorsum and lateral surface of the patient's right foot.

Differential diagnoses

Conditions to consider among the differential diagnoses include the following.

- **Granuloma annulare (GA).** This benign, self-limiting, inflammatory dermatosis of unknown aetiology has a predominance in females and more commonly affects those under the age of 30 years.[1] The lesions are almost always asymptomatic and most commonly involve the upper and lower limbs, particularly the hands, elbows and knees.[1] The most common form is localised GA, which presents with small groups of nonpruritic flesh-coloured dermal papules that spread slowly to form annular lesions; generalised GA is characterised by hundreds of disseminated, small, skin-coloured papules that spare the scalp and acral surfaces.[1] Perforating GA lesions are skin-coloured papules with central umbilication that preferentially involve the upper extremities, whereas subcutaneous GA lesions are larger flesh-coloured dermal nodules that can involve acral surfaces.[1] Biopsy is diagnostic and shows necrobiotic collagen degeneration with surrounding palisading mixed inflammatory infiltrate consisting of monocytes, histiocytes, lymphocytes, fibroblasts and giant cells.[1]
- **Sarcoidosis.** Cutaneous sarcoidosis may be confined to the skin or be the first sign of systemic disease. Sarcoidosis is an idiopathic, noncaseating-granulomatous, multisystem disease. About 30% of patients with cutaneous sarcoid develop systemic sarcoidosis within two years.[2] The most common clinical manifestation of cutaneous sarcoidosis is the maculopapular form, typified by red, brown and violaceous macules/papules that involve the face (periorbital area, eyelids, nasolabial folds). They may be associated with chest radiographic changes, which occur in stage I sarcoidosis (granulomas in lymph nodes only) and II sarcoidosis (granulomas in lymph nodes and lung).[2] Cutaneous sarcoidosis may involve old scars or areas of trauma and this often correlates with degree of visceral sarcoid disease activity. Lupus pernio is a worrisome harbinger of chronic fibrotic pulmonary disease and presents with red to purple indurated lesions affecting the face, ears and nose, with extension into the nasal mucosa sometimes causing septal perforation.[2] Other clinical forms of cutaneous sarcoidosis include pruritic papules in photoexposed areas, scarring alopecia, and lichenoid eruptions, although these are less common.[2] Lofgren's syndrome is a form of sarcoidosis in which there is the triad of erythema nodosum, hilar lymphadenopathy and arthritis or arthralgia. The histopathological features of sarcoidosis, which are diagnostic, include dermal, noncaseating epitheloid histiocytes with surrounding lymphocytic and monocytic infiltrate, also known as sarcoidal noncaseating granulomas.[2]
- **Prurigo nodularis (PN).** This chronic idiopathic pruritic dermatosis of unknown aetiology can affect males and females of all ages but tends to affect atopic individuals at a younger age.[3] Prurigo nodularis can complicate other chronic inflammatory skin diseases such as eczema but can occur in isolation. It can sometimes be associated with hyperthyroidism, chronic kidney disease, coeliac disease, psychiatric disease (e.g. depression, anxiety), haematological malignancy, and viral

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infections such as human immunodeficiency virus, hepatitis C virus and hepatitis B virus infection.[3] The clinical features of prurigo nodularis involve multiple indurated, intensely pruritic papules and nodules that are symmetrically distributed, favouring extensor surfaces, with associated excoriation, crusting and postinflammatory hyperpigmentation.[3] Hyperkeratosis, irregular acanthosis, mixed dermal inflammatory infiltrate, increased mast cells and perineural eosinophils are typical histopathological features.[3]

- **Secondary syphilis.** Caused by the spirochete *Treponema pallidum*, secondary syphilis causes a typically nonpruritic, diffuse papulosquamous eruption that develops between three and 10 weeks after inoculation and may be difficult to distinguish from other dermatoses.[4] Lesions may be macular, papular or follicular, with a copper-coloured surface, that are symmetrically distributed and involve both mucosal and acral surfaces.[4] The painless, indurated chancre of primary syphilis may point to the diagnosis of secondary syphilis. Less than 10% of patients with secondary syphilis will exhibit patchy 'moth-eaten' alopecia, and involvement of the oral mucosa is seen in about 20% of patients.[4] Mucosal lesions vary in morphology from small superficial ulcers to plaques. Condylomatous lesions may be observed in the anogenital region.[4] An accompanying systemic prodrome is common, with symptoms including fever, myalgia, lymphadenopathy, photophobia and headache.[4] Dark field microscopic examination of serous discharge will identify the spirochetes in primary syphilis, whereas serological testing is most useful for the diagnosis of secondary syphilis.[4] The wide morphological spectrum of syphilis lesions is reflected in the highly variable histopathology, with endothelial swelling, epidermal spongiosis, acanthosis and parakeratosis being the most common findings.[4]
- **Lichen planus (LP).** This is the correct diagnosis. LP is an idiopathic inflammatory disease of the skin, nails and mucosal membranes. The characteristic features of classic cutaneous LP are purple/violaceous, planar, polygonal, pruritic papules and/or plaques,[5] which are visible in the Figure. Closer inspection reveals a lacy white surface, known as Wickham's striae, that is often indicative of LP.[6] The disease has a predominance in females, a mean onset in the fifth decade of life for cutaneous LP, and associations with hepatitis C virus seropositivity and autoimmune disorders (e.g. ulcerative colitis, alopecia areata).[5] Cutaneous LP most commonly involves the flexural surfaces of the upper limbs, the extensor surfaces of the hands, legs and the presacral skin.[5] The oral cavity is the most common site of mucosal disease and may often be the only site of LP. Erosive lesions affecting the anogenital area can produce significant morbidity, resulting in destructive and disfiguring scarring that may require surgical correction.[5] Uncommonly, LP may be drug induced. The rash may be indistinguishable from idiopathic LP or present as a nonspecific exanthem or enanthem with an LP-like histopathology. Drugs commonly involved include antimalarials, NSAIDs, thiazides and heavy metals such as gold and mercury (in amalgam). Some supplements and complementary medicines have also been reported to cause LP. Despite the many variants of LP, the histopathological features remain the same: orthokeratosis, thickening of the stratum granulosum, vacuolar degeneration of the stratum basalis and linear band-like lymphocytic infiltration at the dermoepidermal junction.[5]

Management

The appearance of cutaneous LP warrants a full skin examination to ensure that any other mucosal, anogenital, nail, scalp or ophthalmic LP lesions are identified and treated. Topical and intralesional corticosteroids are first-line treatment for both mucosal and cutaneous LP, with treatment intent to reduce pruritus and restore skin appearance.[5] Treatment and monitoring of oral lesions is warranted because there is a 1% risk of development into de novo squamous cell carcinoma.[5] Acute LP usually resolves with treatment within 18 months, but patients should be warned that it may persist in a chronic form.[6] Other treatment options include systemic corticosteroids, oral retinoids and phototherapy.[5]

Outcome

This patient had no extracutaneous or anogenital lesions concerning for LP and hepatitis serological testing returned negative results. The diagnosis of LP was confirmed by biopsy. The patient was initially managed with both topical and systemic corticosteroids, but the results were disappointing. Her LP responded very well to acitretin, which she continued regularly for two years until the LP resolved. MT

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