

Dermatological Emergencies

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Abstract

Acute dermatological problems are seen regularly by physicians. This review aims to provide a framework for the diagnosis and management of urgent dermatological conditions. Cutaneous drug reactions are commonly seen and the different clinical presentations are discussed including the less common but important drug reactions Stevens-Johnson syndrome and toxic epidermal necrolysis. Erythrodermic patients also require prompt evaluation and management. Current guidelines for the management of urticaria and the common acquired autoimmune bullous disorders will be discussed. Patients with complications of eczema and psoriasis frequently require urgent in-patient management and patients with cellulitis and leg ulcers are also seen regularly on acute medical receiving units often requiring both medical and dermatological input.

Keywords

Dermatological emergencies management

Introduction

Cellulitis or dermatitis, pustular drug eruption or pustular psoriasis, erythema multiforme or Steven-Johnsons syndrome? Acute dermatology poses many diagnostic questions and can certainly be challenging even for dermatology trainees. Which topical treatment or dressing to apply can be even more baffling for those with little dermatology experience. This review has been written for physicians who will regularly see patients with acute skin conditions, to provide a framework for the diagnosis and for the implementation of basic dermatological management.

Acneiform
Acral erythema
Acute generalised exanthematous pustulosis
Bullous
Drug hypersensitivity syndrome
Erythema multiforme
Erythema nodosum
Exfoliative dermatitis
Fixed drug eruption
Leukocytoclastic vasculitis
Lichenoid
Exanthematous (maculopapular)
Stevens-Johnsons syndrome
Toxic epidermal necrolysis
Urticaria

Table 1. Drug eruptions.

Acute skin diseases make up a significant proportion of the acute medical workload and, as dermatology on-call advice is not available in all trusts, it is increasingly important for acute physicians to have a basic understanding of the common dermatological emergencies.¹

1. Cutaneous drug reactions

Cutaneous drug eruptions are common, affecting approximately 3% of hospitalised patients.² Many different types of cutaneous reactions are seen (table 1) and a drug-induced eruption should be considered in any patient presenting with an acute onset of a symmetrical rash.

History

A careful history is important to ascertain a complete drug history including over-the-counter drugs, complementary medicines and topical agents. The dosing and duration should be noted as well as the interval between introduction and the onset of the reaction. Drugs commonly associated with cutaneous reactions include:

- antibiotics,
- non-steroidal anti-inflammatory drugs (NSAIDs),
- chemotherapeutic agents,
- anticonvulsants
- psychotropic drugs.

Identifying the culprit drug can be difficult as the reaction is not always simply due to the addition of a new drug. Interactions between drugs can also result in cutaneous reactions. Co-morbidities such as HIV infection should be ascertained as patients with HIV infection are at increased risk for the development of drug eruptions.³

Examination findings

The clinical characteristics of the rash should be sought in addition to features suggesting a severe drug reaction including mucosal erosions, blistering, angio-oedema, erythroderma, skin necrosis and purpura. A number of different types of drug reaction may be seen (Table 1); the clinical features of the more common of these are summarised below.

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Exanthematic reactions are the commonest and can occur with almost any drug, usually within two weeks of administration, although more prolonged administration prior to onset of allergic reaction has been reported.⁴ Typically, confluent erythematous macules and papules develop in a symmetrical distribution with variable pruritus. The trunk and extremities are commonly involved (Figure 1). In children exanthematic rashes are more commonly related to viral infections but in adults are more frequently related to drugs. Viral rashes usually start on the face before progressing to involve the trunk and are more commonly associated with conjunctivitis, lymphadenopathy and fever.⁴ Treatment involves identification and discontinuation of the offending drug. Exanthematic eruptions usually fade with some degree of desquamation (scaling). Symptomatic treatment includes oral antihistamines, emollients and mild to moderately potent topical steroids, especially during the desquamative stage.

Urticaria is the second commonest type of acute cutaneous drug eruption⁵. Wheals appear shortly after administration, usually within 24–36 hours, and resolve rapidly once the drug is withdrawn. Urticarial reactions are commonly due to penicillins, sulphonamides and non steroidal anti-inflammatory drugs (NSAIDs), including aspirin.⁴ Angio-oedema and anaphylaxis may also occur.

Fixed drug eruptions are characterised by more localised lesions which recur at the same sites at each exposure. They appear as well defined, often painful erythematous lesions, sometimes vesicular or bullous which resolve with hyperpigmentation (Figure 2). Lesions occur 30 minutes to 8 hours after drug administration and



Figure 1. Exanthematic reaction (kind permission AG Messenger)



Figure 2. Fixed drug eruption. (kind permission DJ Gawkrödger)

the most common sites include hands, feet, genitalia and perianal areas.⁴ Common causes include antibacterials and NSAIDs.⁶

Exfoliative dermatitis is one of the most serious drug eruptions and frequently presents as erythroderma. The diagnosis and management of this condition is discussed in more detail elsewhere in this article. Common causative drugs include sulphonamides, antimalarials and penicillin.⁴

Acute generalised exanthematous pustulosis (AGEP) presents with multiple small sterile pustules with background erythema and fever followed by desquamation often within days of drug administration. The main differential diagnosis is pustular psoriasis. The presence of eosinophils on biopsy is a useful pointer to a drug cause if diagnosis is uncertain. Antibiotics are the most common culprits.⁷

The drug hypersensitivity syndrome (DHS), also known as drug rash with eosinophilia and systemic symptoms syndrome (DRESS) is most often caused by anticonvulsants. Clinical features include malaise, fever, lymphadenopathy, hypereosinophilia and internal organ involvement such as hepatitis and renal failure. Cutaneous manifestations are variable, ranging from exanthematous to papulopustular, urticaria or even erythema multiforme. Cutaneous features are usually seen after 3–6 weeks of drug therapy.⁴ This highlights the importance of a detailed drug history as patients may have been started on their anticonvulsants many weeks previously. Management is drug withdrawal and supportive therapy. Systemic corticosteroids are frequently used but remain controversial.⁸

Erythema multiforme, Stevens-Johnson syndrome and toxic epidermal necrolysis and leukocytoclastic vasculitis can also be drug related and are discussed in separate sections in this review.

2. Erythema multiforme

Erythema multiforme (EM) is a self-limiting acute eruption characterised by 'target lesions' distributed mainly on the distal sites (acral distribution). It is frequently triggered by infection, probably due to a hypersensitivity reaction. Herpes simplex virus (HSV) infection or mycoplasma are common causes but other bacterial, viral and fungal infections have been implicated. Drug induced erythema multiforme has also been reported. However in up to 50% of cases no triggering factor is identified.⁴

Clinical Features

The lesions of erythema multiforme minor include erythematous papules or urticated lesions in a symmetrical distribution mainly on the distal aspects of the limbs. Target lesions are usually present. These have a central area of dusky erythema surrounded by a ring of paler oedema, which itself is surrounded by a ring of erythema (Figure 3). Patients with erythema multiforme major have more extensive target lesions and mucous membrane involvement with genital and oral mucous membrane erosions and conjunctivitis. The cutaneous eruption

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usually develops over a few days and fades within 1-2 weeks.⁴

Diagnosis

The diagnosis of EM is based on the clinical features and in the presence of target lesions a skin biopsy is not required. Investigations for an underlying cause may include skin swabs for viral cultures, HSV serology, mycoplasma titres and a chest X-ray in patients with features suggestive of a pulmonary infection.

Treatment

Treatment for mild disease is symptomatic only including analgesia, topical steroids. Ophthalmology advice should be sought if there is significant eye involvement. The benefit of antiviral therapy in established cases of erythema multiforme following herpes simplex infections is unclear; however it has a role as long-term prophylaxis in patients with frequently recurring episodes.⁴

Stevens Johnson Syndrome and Toxic Epidermal Necrolysis

Stevens-Johnsons syndrome (SJS) and toxic epidermal necrolysis (TEN) are now considered variants of a single disease process and are classified as a separate disease spectrum from EM. They are uncommon, potentially life threatening cutaneous eruptions which can be triggered by drugs or infection. Common culprits include antiepileptics, antibiotics, allopurinol and NSAIDs.⁴ The onset is usually within four weeks of the introduction of the drug however there is currently no reliable test to identify the causative drug and all possible drug triggers should be withdrawn promptly. HIV infection substantially increases the risk of SJS/TEN. Uncontrolled apoptosis leading to epidermal loss has been proposed as the underlying pathophysiology.

Clinical Features

Typical features include widespread blistering and erosions, predominantly on the trunk and face with mucous membrane involvement.⁹ There is often

widespread erythema, necrosis and bullous detachment of the epidermis mainly on the trunk and face with involvement of at least one mucosal membrane. SJS is defined as cutaneous involvement of less than 10% total body surface area, SJS-TEN overlap as between 10-30% involvement and TEN as greater than 30% involvement.¹⁰ Patients with severe disease are at acute risk of fluid and electrolyte loss, pre-renal failure, bacterial infection and diffuse interstitial pneumonitis.

Diagnosis / Prognosis

Although the diagnosis can usually be made on clinical grounds a biopsy for histology will confirm the diagnosis showing a subepidermal split. The mortality from SJS is approximately 5% whereas the mortality from TEN is around 30%.¹¹ A severity-of-illness (SCORTEN) has been developed and validated to aid assessment of prognosis and is based on seven independent risk factors for death as assessed on the first day of hospitalisation (Table 2 & 3).¹²

Treatment

This involves discontinuation of the offending drug and supportive care in a specialised unit such as a burns or intensive care unit under the supervision of a dermatologist. This is particularly important for patients with TEN.^{13,14} Meticulous attention to fluid and electrolyte balance is essential. Measures to maintain body temperature, nutrition and oral hygiene must be in place. Dermatological care involves pressure-relieving mattresses and nonadhesive dressings. Regular cultures from the skin, mucosal surfaces and blood should be taken as sepsis is a common complication. Ophthalmology and gynaecological assessment are essential to manage the risk of long term complications.

At present there is no consensus on the best management of TEN.¹⁴ Several specific treatments have been advocated including oral steroids, intravenous immunoglobulins, ciclosporin, plasmapheresis and recently infliximab has been reported to control the



Figure 3. Target lesion (kind permission AG Messenger).

Age > 40 years
Heart rate > 120 beats per minute
Presence of cancer or haematologic malignancy
Epidermal detachment involving body surface area > 10% on day one
Blood urea nitrogen > 10 mmol/L
Glucose > 14 mmol/L
Bicarbonate < 20 mEq/L

Table 2. SCORTEN should be computed within the first 24 hours after admission. One point is given for each variable.¹⁴

SCORTEN	Mortality
0-1	3.2%
2	12.1%
3	35.3%
4	58.3%
5 or greater	90.0%

Table 3. SCORTEN level and predicted mortality.¹⁴

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progression of TEN.^{4, 15} Skin sequelae include vaginal, urethral, and anal strictures and pigmentation abnormalities.¹⁴ Ocular complications include synechiae, corneal ulcers, xerophthalmia and blindness.¹⁴ It is extremely important that patients avoid re-exposure to the drug and first-degree relatives should also be warned of their elevated risk of reaction to the same drug.¹⁴

3. Erythroderma

Erythroderma is a term used to describe inflammation of the skin which involves more than 90% of the body surface.

Aetiology

The commonest cause is eczema (40%) followed by psoriasis (25%), cutaneous lymphoma (15%) and drugs (10%).⁴ Rare causes include pityriasis rubra pilaris as well as a number of other inflammatory conditions including pemphigus foliaceus. In a small percentage of cases no cause is established and the erythroderma remains idiopathic.

Clinical Features

Erythroderma is characterised by a widespread, scaling and crythema. Erythrodermic patients are frequently pyrexial and experience lethargy and malaise. The extensive erythema and scaling often obscures primary lesions such as underlying psoriatic plaques. Hair loss and nail changes may be seen if the erythroderma is prolonged.

History of preceding psoriasis or eczema or a drug reaction is often important in ascertaining the underlying diagnosis. However erythroderma may be of sudden onset and the first presentation of skin disease.

Diagnosis and management

A skin biopsy with T cell marker studies may be required to establish the diagnosis.¹⁶ Patients require hospital admission and dermatology advice should be sought early. Patients are at risk of hypothermia, fluid loss, hypoalbuminaemia and high-output cardiac failure. Temperature, electrolyte and fluid balance and haemodynamic status should be monitored. As the skin barrier has been compromised secondary bacterial infection is a threat and skin swabs should be taken. Any clinical deterioration should prompt a search for sepsis. Routine bloods should be performed regularly and all non-essential drugs discontinued. Reactive enlarged lymph nodes may be seen even with erythroderma unrelated to an underlying lymphoma and a lymph node biopsy is only required if an underlying lymphoma is suspected. Patients should be nursed in a warm room. Oral fluids should be encouraged and protein supplements

may be required. Bland emollients should be applied frequently. Once the underlying cause is established appropriate treatment may be started by a dermatologist.

4. Urticaria & angioedema

The urticarias are defined by short-lived swellings of the skin due to plasma leakage from small blood vessels following the mast cell release of histamine and other vasoactive agents. Weals are transient, superficial, itchy, red or pale, well-demarcated swellings of the skin. Angio-oedema is deeper, usually painful, poorly defined, pale or normal colour swellings of the skin and can also affect mucosal membranes. Weals and angio-oedema may occur alone or together.

Anaphylaxis is an acute life-threatening condition due to an immunoglobulin E mediated allergic reaction. Whilst urticaria and angio-oedema may be seen as part of this severe allergic reaction, other symptoms and signs are present such as hypotension and dyspnoea.¹⁷

Classification

Urticaria is classified into physical, contact, acute and chronic ordinary type and urticarial vasculitis. Physical urticarias are weals induced by a physical stimulus (Table 4). Weals generally resolve within a few hours except for those in delayed pressure urticaria which last longer. The weals of contact urticaria develop after biological or chemical contact with the skin (latex, certain foods and plants) and last up to 2 hours.

The lesions of urticarial vasculitis often have similar appearances to ordinary urticaria but they usually last several days¹⁸ and frequently resolve to leave bruising. They are a form of vasculitis and diagnosis is confirmed on biopsy (see section on vasculitis).

The weals of 'ordinary' urticaria typically last from 2 to 24 hours. When the urticaria is present daily or almost daily for less than 6 weeks, it is termed acute but if it occurs for longer than this it is termed chronic.

Acute ordinary urticaria may be due to infections (e.g. upper respiratory tract infections), following administration of drugs (e.g. penicillins, cephalosporins, aspirin, NSAIDs, codeine, radio-contrast media), blood products, food (e.g. fish, milk, nuts, oranges, tomatoes and strawberries) or following bee/wasp stings. However many cases will remain idiopathic after evaluation.⁴

Chronic urticaria is usually idiopathic but can be aggravated by certain drugs such as salicylates and NSAIDs and may flare by intercurrent infections. An increased incidence of thyroid autoantibodies and disturbances of thyroid function have been reported. Many patients probably have an autoimmune basis for their disorder although there is currently no routine laboratory test for histamine-releasing autoantibodies.¹⁸

Angio-oedema can occur with and without weals as part of the chronic idiopathic urticaria angioedema spectrum. Angio-oedema in the absence of weals is well known to be associated with angiotensin-converting enzyme inhibitors. Hereditary angio-oedema is a rare autosomal dominant disease characterised by a qualitative

Aquagenic
Cholinergic urticaria (heat/exercise)
Cold urticaria
Delayed pressure urticaria
Dermographism
Localised heat urticaria
Solar urticaria

Table 4. Physical urticarias.

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or quantitative deficiency of C1 esterase inhibitor.¹⁹ Over 50% of cases present before puberty but the onset may be delayed till later in life.⁴ Patients experience recurrent attacks of skin and mucous membrane swellings, often associated with nausea, vomiting, abdominal pain and urinary symptoms. Pharyngeal, laryngeal and even bronchial involvement are especially significant. An acquired deficiency of the inhibitor may present a similar clinical picture and is associated with lymphoma and SLE.⁴

Management of Urticaria and Angioedema

In acute ordinary urticaria no investigations are required except in cases where an allergic cause is suggested from the history. Skin-prick testing and radioallergosorbent tests (RAST) may be performed if required. Similarly no investigations are required for the majority of patients with chronic ordinary urticaria responding to antihistamines. Patients with angio-oedema without weals should be screened for C1 esterase inhibitor by assaying serum C4. If low, C1 inhibitor deficiency can be confirmed by quantitative and functional C1 inhibitor assays.¹⁸

Management of chronic urticaria includes advice on the avoidance of non-specific aggravating factors, such as stress, alcohol and drugs (e.g. aspirin and codeine). Anti-histamines form the mainstay of treatment. Commonly used non-sedating antihistamines (H1 antagonists) are used in doses up to 4 times normal dosing. H2 antagonists may also be used in addition. The British Association of Dermatologists guidelines recommend that oral corticosteroids should be restricted to short courses for severe acute urticaria or angio-oedema affecting the mouth.¹⁸

Management of C1 esterase inhibitor deficiency includes treatment of acute severe attacks (C1 inhibitor concentrate or fresh frozen plasma should be given with appropriate management of laryngeal oedema). Prophylaxis treatments can be required and include danazol and tranexamic acid.^{18,20}

5. Autoimmune bullous diseases

Patients with blistering skin disorders can be a diagnostic challenge. Blisters can occur secondary to severe oedema, infection and porphyria. Drug reactions can also present with blisters. The more common autoimmune bullous disorders are bullous pemphigoid and pemphigus vulgaris. Less common forms include dermatitis herpetiformis, pemphigus foliaceus, linear IgA disease and epidermolysis bullosa acquisita.

A detailed history of the development of the blisters and their distribution is important. This should be followed by a thorough skin examination including mucosal surfaces.

Biopsy of a fresh blister for routine histology and a sample of perilesional skin are sent for immunofluorescent study. Immunoglobulin and complement deposition within the skin are usually present in autoimmune bullous disorders. Serum can also be sent for pemphigoid and pemphigus antibodies (indirect immunofluorescence).



Figure 4. Bullous pemphigoid (kind permission AG Messenger).

Bullous pemphigoid is the most common bullous autoimmune disease. Elderly patients are usually affected. The disease often starts with itch followed by urticated erythematous lesions (pre-bullous pemphigoid). Subsequently tense blisters develop on both normal and erythematous skin, usually on the trunk and limbs (Figure 4). Mucosal involvement may also be seen. The blisters are subepidermal and hence the condition presents with tense blisters. These are caused by autoantibodies (usually IgG) directed at the hemidesmosomes anchoring the epidermis to the dermis. For localised bullous pemphigoid very potent topical steroid should be implemented in the first instance. For more widespread disease oral corticosteroids are required and should be initiated by a dermatologist who will arrange subsequent follow-up. Azathioprine may be required as a steroid sparing agent.²¹

Pemphigus vulgaris is another common immunobullous disease. It usually affects middle-aged patients who present with flaccid blisters and erosions especially on the scalp, face and flexures although there may be a more widespread distribution (Figure 5). The autoantibodies are directed against the desmosomes which anchor the keratinocytes of the epidermis to each other. Clinically this presents with superficial flaccid blisters. All patients develop mucosal lesions. Treatment involves high dose oral steroids and other immunosuppressive agents which should be initiated by a dermatologist.²²

6. Eczema herpeticum

Patients with active eczema are predisposed to widespread infection with herpes simplex virus.²³ This frequently follows herpes simplex labialis infection. After an incubation period of about 10 days, patients develop vesicles (small <0.5cm raised lesions containing clear fluid) which erupt in large crops.⁴ The vesicles quickly become pustular and often with time will become haemorrhagic and crusted, and may evolve into erosions with a punched-out appearance (Figure 5). Secondary bacterial infection may ensue. The eruption may be confined to the areas originally affected by the eczema but may spread widely. Early specialist advice is required. Fever commonly develops 2 or 3 days after the appearance of the vesicles and constitutional symptoms may be severe.⁴

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To aid diagnosis viral cultures of fresh vesicular fluid can be undertaken. Bacterial swabs for microscopy and culture should also be taken to exclude secondary bacterial infection. Ocular involvement should prompt a referral to ophthalmology as keratitis can lead to scarring. Patients with eczema often use topical steroids and topical

calcineurin inhibitors (topical tacrolimus and pimecrolimus) on a regular basis. These should be temporarily stopped and only emollients should be used. Intravenous acyclovir should be commenced as soon as possible for severe cases but patients with less severe disease may respond to oral acyclovir.⁴ Secondary bacterial infection should be treated with appropriate antibiotics.



Figure 5. Eczema herpeticum (kind permission AG Messenger)



Figure 6. Pustular psoriasis (kind permission from DJ Gawkrödger).

7. Generalised pustular psoriasis

Generalised pustular psoriasis is an uncommon form of psoriasis characterised by widespread pustules and erythema (Figure 6). This may follow a history of chronic plaque psoriasis which develops into a pustular form usually following a severe flare. Occasionally generalised pustular psoriasis can be the presenting form of psoriasis. Aggravating factors include abrupt withdrawal of steroid therapy, “irritating” topical therapies such as dithranol or tar used on unstable psoriasis, infection, pregnancy, hypocalcaemia and certain drugs such as lithium. The differential diagnosis includes pustular drug eruptions and a detailed history is important.²⁴

Initially the skin becomes erythematous and tender. Systemic features are then seen such as pyrexia and malaise. Within hours the psoriatic plaques become inflamed and studded with tiny sterile pustules. Erythema and pustules may develop elsewhere, such as the flexures and genital areas. Eventually the pustules may coalesce into lakes of pus which then dry and peel. Successive crops of pustules may erupt every few days or weeks.

Acute generalised pustular psoriasis is a dermatological emergency with a small risk of mortality. Management involves hospital admission and involvement of dermatology specialists. Patients are at risk of hypoalbuminaemia, hypocalcaemia, acute renal failure and acute respiratory distress syndrome (ARDS). Secondary bacterial and viral skin infections also occur. Investigations include routine haematological and biochemical blood tests, chest X-ray and skin and blood cultures. Topical treatments such as vitamin D analogues, dithranol and tar preparations should be avoided and emollients used instead (e.g. 50/50 white soft paraffin/liquid paraffin). Systemic treatment is generally required and options include oral retinoids such as acitretin or methotrexate and ciclosporin which will be initiated by the dermatology team.

8. Cutaneous small vessel vasculitis

Dermatologists are regularly asked to review medical patients with a rash considered to be vasculitis. Vasculitis represents inflammation of blood vessels and can be classified as large, medium and small vessel vasculitis. Cutaneous involvement occurs frequently.

Cutaneous small vessel vasculitis (also known as cutaneous leukocytoclastic vasculitis or hypersensitivity vasculitis) is inflammation of the cutaneous post-capillary venules.²⁵ Triggers include bacterial and viral infections including hepatitis B and C, drugs, malignancy and autoimmune disease such as systemic lupus erythematosus and rheumatoid arthritis.⁴ A history of recent infection or drug exposure is therefore important. Patients usually have palpable purpura on the legs or

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ankles which are areas prone to stasis but can be more widespread (Figure 7). Other lesions which may be seen include macules, papules/nodules, vesicles/bullae, urticaria and areas of cutaneous necrosis. These lesions may be painful or itchy.

Investigation and Treatment

Confirmation of the diagnosis may require a skin biopsy.²⁶ A full systemic enquiry and medical examination should be performed to look for systemic involvement. Urinalysis and a basic vasculitis screen may include full blood count and film, renal and liver function, anti-nuclear antibody, anti-neutrophilic cytoplasmic antibody, rheumatoid factor, C3/C4, immunoglobulins and serum electrophoresis, cryoglobulins, hepatitis B and C serology and anti-streptococcal antibody titre. Other investigations will be required according to the history. Management includes removing any triggers such as drugs or treating the underlying course. Compression hosiery and elevation of dependent areas to decrease stasis may also be of benefit. Treatments include non-steroidal anti-inflammatory drugs and tapered courses of oral prednisolone in patients with severe, progressive, painful or ulcerative lesions.^{4,27}

9. 'The red leg'

Red legs frequently present to acute medical admissions units and are not infrequently misdiagnosed. Cellulitis is a common bacterial infection of the skin, usually affecting a single limb. The most common infecting organisms are Streptococcal group A organisms and Staphylococcus aureus. Streptococci of other Lancefield groups, mainly G, C and B can also be implicated.⁴ Redness, swelling, heat and pain or tenderness are constant features⁴ (Figure 8). Severe cellulitis can lead to blistering. If there is no increased warmth of the skin the diagnosis is unlikely to be cellulitis. Except in mild cases there is fever and malaise. Predisposing factors should be assessed. These include impaired lymphatic or venous drainage of the area, obesity and portals for the entry of infection such as wounds, ulcers and toe-web tinea.^{28, 29}

The diagnosis is based on clinical features but microbiological specimens should be taken from vesicle fluid or broken areas of skin in addition to blood cultures. Surface swabs from intact skin are unlikely to be helpful.⁴ Appropriate antibiotics should be given in full dosage for at least 10 days. Intravenous antibiotics should be prescribed in the more severe cases although oral treatment may suffice in milder cases. Antibiotics used must cover streptococcal infections.³⁰ Penicillin is the treatment of choice but a macrolide antibiotic or clindamycin are alternatives.⁴ Local antibiotic guidelines are available in most hospitals.

The differential diagnosis includes deep-vein thrombosis and Doppler ultrasound may be useful if this is suspected.

Eczema of the lower legs is also common in middle aged or elderly patients and may be mistaken for cellulitis. Cellulitis usually only affects a single limb; venous eczema is however frequently bilateral. Varicose veins, pigmentation,

oedema, atrophie blanche (white plaques of sclerosis) and ulceration may be present to aid the diagnosis.

Venous eczema is an itchy, red, scaly and often exudative eruption usually of the ankle and lower leg which may develop suddenly or insidiously⁴ (Figure 9). The pathogenesis of venous eczema is venous incompetence leading to venous hypertension causing fibrinogen to leak into the interstitial fluid. This forms a fibrin sheath around the capillaries limiting the diffusion



Figure 7. Cutaneous small vessel vasculitis (kind permission from DJ Gawkrödger).



Figure 8. Cellulitis (kind permission from DJ Gawkrödger)

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of oxygen and nutrients to the skin. The appearance of venous eczema is often modified by secondary contact dermatitis (allergic contact dermatitis to topical treatments and bandages), infection and rubbing. Mild and potent topical steroids may be used to relieve irritation but use of potent steroids should be limited to short intermittent courses. Well-fitted support hosiery can be helpful and patients should be advised to elevate their legs when recumbent.⁴

Acute lipodermatosclerosis may also present with erythema, induration, tenderness and warmth on the lower leg. It appears to be related to venous hypertension, venous incompetence and obesity. Chronic progression results in hyperpigmentation and the 'inverted champagne bottle' appearance.

10. Leg ulcers

The morbidity and associated costs of chronic leg ulceration are very significant. Common causes include venous insufficiency, arterial insufficiency, diabetes and other less common causes such as pyoderma gangrenosum, vasculitic ulceration and malignancy (Figures 10). Factors which contribute to poor healing include old age, malnutrition, anaemia, infection and immunosuppressants. It



Figure 9. Venous eczema (kind permission from DJ Gawkrödger).



Figure 10. Venous leg ulcer (kind permission from DJ Gawkrödger).

is not uncommon for ulcers to have multiple underlying aetiologies such as combined arterial and venous disease.

Clinical Assessment

A full history and examination should be taken including vascular assessment. The length and width of the ulcer should be documented for monitoring purposes. Slough on the ulcer base, irregularity of the ulcer margin (raising the suspicion of malignancy), and any erythema of the surrounding skin should be noted (dermatitis from ulcer exudate or allergy to topical medicaments or dressings or cellulitis). Clinical signs of underlying aetiology may also be seen. For example signs of venous hypertension include varicose veins, hyperpigmentation and atrophie blanche.

Investigation

Investigations will be dictated by the suspicion of the underlying cause. A skin biopsy of the ulcer edge may be required if a skin cancer is suspected. Arterial dopplers are required in most cases and liaison with vascular surgery if an ischaemic cause is identified.

Treatment

Wound management involves debridement of devitalised tissue, usually by topical measures (e.g. de-sloughing agents such as topical hydrogen peroxide). Systemic antibiotics are required for wound infection and should be given for 10-14 days. Choice of dressing depends on the wound. Wounds producing large amounts of exudate require dressings which are absorptive such as foams or alginates. Hydrogels are useful in dressing dry, necrotic or sloughy wounds. The most important part of managing venous leg ulcers is compression. Before compression the Ankle Brachial Pressure Index (ABPI) must be measured to ensure that there is not significant arterial insufficiency compounding the venous disease. If the ABPI is greater than 0.8 then compression bandaging may be applied. An ABPI greater than 1.0 should be interpreted with caution as this may suggest calcification of vessels preventing their compression during measurement. Compression dressings most frequently consist of a multi-layer bandage system.

11. Undiagnosed skin cancer

Patients generally receive a thorough medical examination when admitted to acute medical units and undiagnosed skin cancers can often be detected, especially in elderly patients.

Basal cell carcinomas are the commonest skin cancers and occur most commonly on the face. The nodular-ulcerative type start as a small papule, which becomes nodular and then ulcerates centrally. The margin is rolled and pearly in appearance and telangiectasia are characteristic. Other forms of basal cell carcinoma include pigmented, cystic, morphoeic (scar-like appearance) and superficial (well-defined red, scaly plaque with a very thin rolled margin). These can be referred on a routine basis if a confident diagnosis of basal cell carcinoma can be made.

Squamous cell carcinomas usually present as indurated nodular, keratinous and crusted tumours generally on sun-

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exposed areas. They can also develop in chronic wounds such as scars, burns, ulcers and chronic sores. In view of their potential to metastasize they should be referred for a more urgent assessment.

Assessment of pigmented lesions requires experience. The 'ABCD' guidelines can be useful: Asymmetry, irregularity of Border and Colour and a Diameter greater than 6mm may cause suspicion for a malignant melanoma but early diagnosis is very difficult and any pigmented lesion which you notice and feel is suspicious should be referred urgently.³¹

References

- Kirsner RS, Yang DG, Kerdel FA. Dermatologic disease accounts for a large number of hospital admissions annually. *J.Am.Acad.Dermatol.* 1999 Dec; **41**(6): 970-973.
- Valeyrie-Allanore L, Sassolas B, Roujeau JC. Drug-induced skin, nail and hair disorders. *Drug Safety* 2007; **30**(11): 1011-1030.
- Campos-Fernandez Mdel M, Ponce-De-Leon-Rosales S, Archer-Dubon C, et al. Incidence and risk factors for cutaneous adverse drug reactions in an intensive care unit. *Revista de Investigacion Clinica* 2005 Nov-Dec; **57**(6): 770-774.
- Burns T, Breathnach S, Cox N, et al. Rook's Textbook of Dermatology. seventh edition ed. UK: Blackwell Publishing; 2004.
- Tan EK, Grattan CE. Drug-induced urticaria. *Expert Opinion on Drug Safety* 2004 Sep; **3**(5): 471-484.
- Mahboob A, Haroon TS. Drugs causing fixed eruptions: a study of 450 cases. *Int.J.Dermatol.* 1998 Nov; **37**(11): 833-838.
- Sidoroff A, Dunant A, Viboud C, et al. Risk factors for acute generalized exanthematous pustulosis (AGEP)-results of a multinational case-control study (EuroSCAR). *Br.J.Dermatol.* 2007 Nov; **157**(5): 989-996.
- Tas S, Simonart T. Management of drug rash with eosinophilia and systemic symptoms (DRESS syndrome): an update. *Dermatology* 2003; **206**(4): 353-356.
- Roujeau JC. Stevens-Johnson syndrome and toxic epidermal necrolysis are severity variants of the same disease which differs from erythema multiforme. *J.Dermatol.* 1997 Nov; **24**(11): 726-729.
- Roujeau JC. The spectrum of Stevens-Johnson syndrome and toxic epidermal necrolysis: a clinical classification. *J.Invest.Dermatol.* 1994 Jun; **102**(6): 28S-30S.
- Schulz JT, Sheridan RL, Ryan CM, et al. A 10-year experience with toxic epidermal necrolysis. *J.Burn Care Rehabil.* 2000 May-Jun; **21**(3): 199-204.
- Bastuji-Garin S, Fouchard N, Bertocchi M, et al. A severity-of-illness score for toxic epidermal necrolysis. *J.Invest.Dermatol.* 2000 Aug; **115**(2): 149-153.
- Garcia-Doval I, LeCleach L, Bocquet H, et al. Toxic epidermal necrolysis and Stevens-Johnson syndrome: does early withdrawal of causative drugs decrease the risk of death? [see comment]. *Arch.Dermatol.* 2000 Mar; **136**(3): 323-327.
- Pereira FA, Mudgil AV, Rosmarin DM. Toxic epidermal necrolysis. *J.Am.Acad.Dermatol.* 2007 Feb; **56**(2): 181-200.
- Chia FL & Leong KP. Severe cutaneous adverse reactions to drugs. *Current Opinion in Allergy & Clinical Immunology* 2007; **7**: 304-9
- Rothe MJ, Bernstein ML, Grant-Kels JM. Life-threatening erythroderma: diagnosing and treating the "red man". *Clin.Dermatol.* 2005 Mar-Apr; **23**(2): 206-217.
- Brown SG. Clinical features and severity grading of anaphylaxis. *Journal of Allergy & Clinical Immunology* 2004 Aug; **114**(2): 371-376.
- Grattan CE, Humphreys F, British Association of Dermatologists Therapy Guidelines and Audit, Subcommittee. Guidelines for evaluation and management of urticaria in adults and children. *Br.J.Dermatol.* 2007 Dec; **157**(6): 1116-1123.
- Sachse MM, Khachemoune A, Guldakke KK, et al. Hereditary angioedema. *Journal of Drugs in Dermatology: JDD* 2006 Oct; **5**(9): 848-852.
- Pekdemir M, Ersel M, Aksay E, et al. Effective treatment of hereditary angioedema with fresh frozen plasma in an emergency department. *J.Emerg.Med.* 2007 Aug; **33**(2): 137-139.
- Wojnarowska F, Kirtschig G, Highet AS, et al. British Association of D. Guidelines for the management of bullous pemphigoid. *Br.J.Dermatol.* 2002 Aug; **147**(2): 214-221.
- Harman KE, Albert S, Black MM, British Association of D. Guidelines for the management of pemphigus vulgaris. *Br.J.Dermatol.* 2003 Nov; **149**(5): 926-937.
- Peng WM, Jenneck C, Bussmann C, et al. Risk factors of atopic dermatitis patients for eczema herpeticum. *J.Invest.Dermatol.* 2007 May; **127**(5): 1261-1263.
- Mengesha YM, Bennett ML. Pustular skin disorders: diagnosis and treatment. *American Journal of Clinical Dermatology* 2002; **3**(6): 389-400.
- Lotti T, Ghersetich I, Comacchi C, et al. Cutaneous small-vessel vasculitis. *J.Am.Acad.Dermatol.* 1998 quiz 688-90; Nov; **39**(5 Pt 1): 667-687.
- Carlson JA, Chen KR. Cutaneous vasculitis update: small vessel neutrophilic vasculitis syndromes. *Am.J.Dermatopathol.* 2006 Dec; **28**(6): 486-506.
- Carlson JA, Cavaliere LF, Grant-Kels JM. Cutaneous vasculitis: diagnosis and management. *Clin.Dermatol.* 2006 Sep-Oct; **24**(5): 414-429.
- Dupuy A, Benchikhi H, Roujeau JC, et al. Risk factors for erysipelas of the leg (cellulitis): case-control study. *BMJ* 1999 Jun 12; **318**(7198): 1591-1594.
- Cox NH. Oedema as a risk factor for multiple episodes of cellulitis/erysipelas of the lower leg: a series with community follow-up. *Br.J.Dermatol.* 2006 Nov; **155**(5): 947-950.
- Gabillot-Carre M, Roujeau JC. Acute bacterial skin infections and cellulitis. *Curr.Opin.Infect.Dis.* 2007 Apr; **20**(2): 118-123.
- Abbasi NR, Shaw HM, Rigel DS, et al. Early diagnosis of cutaneous melanoma: revisiting the ABCD criteria.[see comment]. *JAMA* 2004 Dec 8; **292**(22): 2771-2776.