Empowering advanced clinical practitioners in managing acute dermatological emergencies

Hasan Hazim Alsararatee

ABSTRACT

Advanced clinical practitioners (ACPs) encounter patients with acute dermatological presentations ranging from minor to life-threatening conditions in both primary and secondary care settings. However, ACPs often feel unprepared to assess and treat patients with dermatological emergencies. This article aims to provide guidance to trainee and qualified ACPs, whether in acute hospital settings or primary care, in understanding the essential aspects to consider when consulting with patients presenting with acute dermatological emergencies. It also emphasises appropriate referrals to relevant specialties for necessary inpatient or outpatient investigations and ensure prompt treatment.

Key words: Advanced clinical practitioners ■ Dermatology ■ Skin conditions ■ Stevens-Johnson syndrome ■ Toxic epidermal necrolysis ■ DRESS ■ Vasculitis ■ Erythroderma

In recent decades, the role of advanced clinical practitioners (ACPs) has emerged as a crucial component in alleviating the pressure on healthcare systems (Kuczawski et al, 2024). ACPs, unless specialised, should have a broad understanding of diverse medical and surgical domains, enabling them to work autonomously and effectively across primary and secondary care settings (Reynolds and Mortimore, 2021). This article aims to equip ACPs working in acute hospital and primary care settings with the knowledge and skills necessary to identify, manage and appropriately refer patients presenting with acute dermatological emergencies. Despite the complexity of this specialty, ACPs who may lack familiarity with acute dermatological conditions can enhance their expertise and contribute to optimal patient care.

Dermatological emergencies may arise due to exacerbation of primary dermatological conditions, systemic diseases, or reactions to external factors, such as medications or infections (Meltan et al, 2024). Severe rashes can compromise skin function and serve as indicators of critical underlying systemic conditions, necessitating prompt intervention and management (Primary Care Dermatology Society, 2024). In clinical practice, acute skin failure represents a critical condition marked by disruptions in thermoregulation, electrolyte homeostasis and protein equilibrium. This results in malnutrition and compromises dermal barrier function, which enables infiltration by infectious pathogens and foreign substances (Levine et al, 2022).

Any widespread skin eruption has the potential to compromise normal physiological skin functions. When managing such emergencies, meticulous nursing care becomes paramount to maintain skin integrity. The use of emollient ointments, rich in lipids (such as a 50:50 mixture of liquid paraffin and white soft paraffin), aids in preserving the skin barrier function. These emollients prevent heat and water loss, while providing symptomatic relief to patients. Notably, the disruption of the skin barrier can lead to impaired thermoregulation and significant trans–epidermal water loss (up to 2.5 L per day in erythrodermic patients). Consequently, close monitoring of patient temperature, fluid status and electrolyte balance is crucial. Patients with compromised skin barriers are also at risk of secondary bacterial and viral infections, necessitating early identification and prompt intervention.

History taking

If the patient is relatively stable, the clinician should take a comprehensive history: *Table 1* summarises the key points that need to be considered during this process of taking a patient's history. If the patient is critically unwell they will require immediate management.

In an emergency situation, the clinician's priority is to stabilise the patient rather than conduct a comprehensive history. The first step is to use the ABCDE approach (Airway, Breathing, Circulation, Disability and Exposure) to systematically assess and examine critically unwell patients to identify any immediate life-threatening problems and intervene accordingly. Once the patient is stable, a focused history can be obtained to gather relevant information for further management.

Physical examination

Physical examinations should follow the history taken in order to narrow down the differential diagnosis list. Therefore,

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Table 1. Important elements of history for acute dermatological emergencies

Factors	Descriptions
Mode of onset	Acute onset or acute exacerbation of a chronic disorder. Sudden, rapid or explosive onset may indicate severe conditions such as Stevens–Johnson syndrome (SJS) or toxic epidermal necrolysis (TEN)
Distribution/ spread	The pattern of distribution provides valuable diagnostic clues. For example, target lesions may suggest erythema multiforme, while diffuse erythroderma points to severe conditions such as SJS or TEN
Site of onset	The specific site of skin involvement often provides clues about the underlying condition. For instance, SJS often affects mucous membranes
Associated symptoms	It is crucial to distinguish between pruritus associated with rash or pruritus without skin disease, which will require critical evaluation for systemic disease (renal, endocrine, hepatic or haematological) Pain, although less prevalent, can serve as a diagnostic aid in identifying early presentations of herpes zoster (shingles), eczema herpeticum or cellulitis
Any exacerbation/ relieving factors	An understanding of exacerbations and relieving factors aids in making an accurate diagnosis, tailored management and improved patient outcomes in acute dermatological emergencies. This may include specific foods that trigger allergic reactions or certain medications that cause reactive eruptions
Family/contact history	To rule out contagious disease
Travel history	To exclude endemic conditions, such as Rocky Mountain spotted fever
Animal exposure	Animal bites, stings or scratches can trigger allergic reactions in humans, leading to skin manifestations such as urticaria or angioedema
Drug history	To rule out drug reactions such as erythroderma
Any history of allergy	For example, to any medications or chemicals. This also includes skin-contact allergies, for example to lanolin or rubber
Occupational history	Drug-induced SJS/TEN is a well-recognised phenomenon. Occupational exposure to specific medications or chemicals can trigger these severe cutaneous reactions
Vaccination status	Tetanus, a potentially life-threatening condition, can arise from contaminated wounds or burns. Immunisation prevents tetanus infection following such injuries
Sexual history	Immunosuppressed individuals (eg HIV-positive patients) are at higher risk for severe sexually transmitted infections (STIs) Some STIs require specific medications (eg antibiotics, antivirals). These drugs can be potential triggers for SJS/TEN

comprehensive assessments, including general, systemic, and dermatological examinations, are essential. For instance, a thorough evaluation for signs such as pallor, jaundice, oedema, cyanosis, lymphadenopathy, and vital signs is imperative to rule out systemic illnesses or critical conditions such as sepsis or anaphylaxis (McPhillips et al, 2021).

Specific dermatological emergencies

Stevens-Johnson syndrome and toxic epidermal necrolysis

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) represent severe, life-threatening mucocutaneous blistering drug eruptions (Cheung et al, 2024). Although less common, they can also occur as a secondary response to *Mycoplasma pneumoniae* (referred to as Mycoplasma-induced rash with mucositis). SJS and TEN are part of the same spectrum of conditions, distinguished by the extent of body surface area affected: SJS involves less than 10% of body surface area and TEN involves more than 30%. The term SJS/TEN-overlap syndrome describes cases in which between 10% and 30% of body surface area is affected (Lehloenya, 2022). The most common medications that can trigger SJS and TEN are listed in *Table 2*.

Typically, SJS and TEN present with a generalised dusky erythematous macular rash, following flu-like symptoms of 1–3 days (Chen and Jiang, 2024). Patients exhibit systemic symptoms, including fever and dehydration. Notably, atypical targets – rashes with a darker or purpuric centre and a lighter rim – precede the development of flaccid blisters and superficial skin loss (*Figure 1*). Eliciting tenderness and observing a positive Nikolsky's sign (where the epidermis sloughs off with mild fingertip pressure) aid in distinguishing SJS/TEN from other severe cutaneous adverse reactions, such as acute generalised exanthematous pustulosis (AGEP) (Woo et al, 2021).

Diagnostic and management considerations of SJS and TEN

A skin biopsy with direct immunofluorescence is essential for distinguishing SJS and TEN from other blistering or mucocutaneous conditions, such as pemphigus (Kardaun, 2022). SJS/TEN constitutes a multisystem disease, often necessitating management in specialist burns units or intensive care units with access to expert dermatology input.

Early discontinuation of the suspected causative drug plays a pivotal role in halting disease progression. The application of ointment emollients to the skin is crucial for maintaining barrier function. SJS/TEN syndrome can also affect the eyes. Patients might present with red and painful eyes, which can progress to conjunctival erosions, genital erosions, and ulceration of the mouth and lips, often accompanied by overlying haemorrhagic crust (Purnamawati et al, 2016; Kitunzi et al, 2021). Involvement of other mucous membranes may lead to difficulty swallowing, urinary retention, cough and respiratory distress, occasionally necessitating intubation. Therefore, it is important to consult with an ophthalmologist in cases where SJS/TEN affects the eyes to prevent long-term scarring and complications associated with eye involvement.

Despite limited evidence, systemic therapy involving corticosteroids, ciclosporin, intravenous immunoglobulins and anti-tumour necrosis factor (TNF) biological agents is frequently employed. SJS/TEN carries a high mortality risk, which escalates with higher percentage of body surface area involvement (Sood et al, 2021). It is important to note that potential long-term sequelae include cutaneous scarring, blindness, pulmonary fibrosis, chronic kidney disease, and autoimmune conditions (such as Sjögren's disease).

Erythroderma

Erythroderma, characterised by widespread erythema and scaling that affects more than 90% of a patient's body surface

area, can manifest as either idiopathic or secondary to drug reactions or primary dermatological diseases such as eczema or psoriasis (Bettuzzi et al, 2024). Less commonly, conditions such as pityriasis rubra pilaris or cutaneous lymphoma (eg Sezary syndrome) may also present as erythroderma. Clinicians should diligently inquire about preceding rashes and any recent medication that the patient may have been administered to guide accurate diagnosis.

Additional features associated with erythroderma include shivering, peripheral oedema, dermatopathic lymphadenopathy, tachycardia, hypothermia or signs suggestive of high-output cardiac failure.

Diagnostic and management considerations of erythroderma

It is important to discontinue all unnecessary medications to prevent further complications and apply emollients and mild topical steroids, if required. The underlying cause of the erythroderma should be established, if possible. Close monitoring of fluid balance and electrolytes is essential, and consideration of skin biopsy is warranted when the underlying cause remains unclear (Patel and Levell, 2021). The application of ointment emollients every 2 hours will support skin integrity. In addition, moderate potency topical corticosteroids may provide benefit. For some patients, systemic therapy becomes necessary to manage the disease: this approach that should be tailored to the specific underlying cause.

Drug reactions

Drug reactions play a significant role in severe cutaneous adverse reactions that are observed in dermatological emergencies (Godfrey et al, 2024). Although nearly all medication has the potential to cause skin rashes, certain classes of drugs – such as anti-epileptics, antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs), and diuretics – are more frequently associated with severe eruptions. The onset of these rashes often occurs several weeks after commencement of medication. Therefore, taking a meticulous drug history to record all medications taken in the preceding 3 months is crucial for identifying and promptly discontinuing the causative agent.

Table 3 provides a summary of the most common severe cutaneous adverse reactions and corresponding time of onset. The majority of medication-induced rashes do not meet the criteria for severe cutaneous adverse reactions and manifest as maculopapular exanthems without systemic involvement. These eruptions typically arise 1–3 weeks after commencement of a new medication. Although their appearance may cause concern, they are not considered emergencies and generally resolve spontaneously within 1–2 weeks on discontinuation of the causative drug. *Box 1* outlines features that aid in distinguishing a simple maculopapular exanthem from severe cutaneous adverse reactions.

Drug reaction with eosinophilia and systemic symptoms

Drug reaction with eosinophilia and systemic symptoms (DRESS) is a type 4, T-cell-mediated hypersensitivity reaction triggered by drugs. It affects not only the skin, but also other organ systems, including the liver, kidneys, lungs, heart and nerves. DRESS can manifest up to 6 weeks after initiating a new medication. Common presentations include a maculopapular rash (pustules and target lesions may also occur), sometimes accompanied by facial oedema. High fever and lymphadenopathy are prevalent, and about 25% of patients exhibit mucosal involvement. Additional symptoms and signs vary, based on the affected organ systems, and can include hepatomegaly, shortness of breath, diarrhoea and headache.

Table 2. Medications that can trigger Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN)*

Drug	Commonly prescribed for the following conditions
Allopurinol	Gout and hyperuricaemia
Carbamazepine	Epilepsy, bipolar disorder, trigeminal neuralgia
Lamotrigine	Epilepsy, bipolar disorder
Nevirapine	HIV/AIDS
Oxicam-type non-steroidal anti-inflammatory drugs (NSAIDs)	Pain relief, inflammation (eg arthritis)
Phenobarbital	Seizures
Phenytoin	Epilepsy
Sulfonamides	Bacterial infections (eg urinary tract infections)

* See also *Table 3*

Diagnostic considerations for DRESS

A full blood count (FBC) often reveals eosinophilia, with levels exceeding 2.0×10^9/L in about 30% of cases. In addition, a large number of atypical lymphocytes may be detected. Biochemical

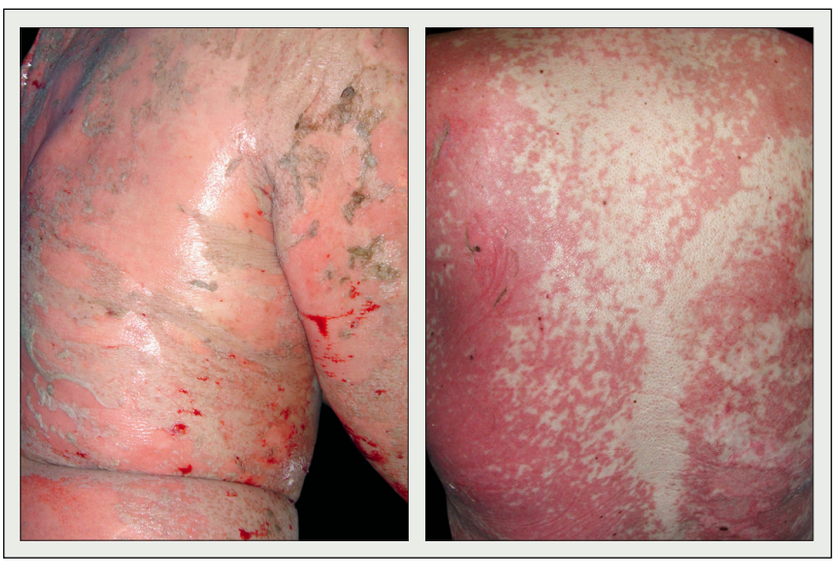

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Figure 1. Manifestations of toxic epidermal necrolysis, with patients experiencing detachment of the epidermis

Table 3. Adverse drug reactions

Adverse drug reaction	Clinical features	Common causative agents	Time from exposure to onset of symptoms
Angioedema	■ Cutaneous: facial swelling, urticaria ■ Other: wheeze/stridor	Antibiotics (especially penicillins), NSAIDs, immunomodulators, contrast agents, neuromuscular agents, nuts, venom	Minutes to hours
AGEP	■ Cutaneous: macular erythematous rash (especially of the proximal flexure), plethora of small pustules (can coalesce) ■ Other: fever, neutrophilia	Antibiotics (especially β-lactams), sulfonamides, antifungals, calcium channel blockers, hydroxychloroquine, carbamazepine, paracetamol	2–5 days
SJS/TEN	■ Cutaneous: mucositis of ≥2 mucous membranes, dusky erythema with epidermal detachment, Nikolsky positive ■ Other: fever, diarrhoea, dyspnoea, cardiovascular instability	Antibiotics, sulfonamides, anticonvulsants (lamotrigine, carbamazepine, phenytoin), allopurinol, paracetamol, NSAIDs	5–28 days
Vasculitis	■ Cutaneous: palpable purpuric papules (especially on the lower legs ■ Other: systemic involvement (kidneys, joints, nerves)	Antibiotics, anti-TNF-α agents, allopurinol, phenytoin, sulfasalazine	7–21 days
DRESS	■ Cutaneous: polymorphic rash (often maculopapular), facial oedema ■ Other: fever, lymphadenopathy, systemic involvement (liver, kidneys)	Anti-epileptics (carbamazepine, lamotrigine, phenytoin, phenobarbital), allopurinol, antibiotics	15–40 days
Maculopapular exanthem	■ Cutaneous: erythematous maculopapular eruption, usually starting on upper trunk before becoming widespread ■ Other: not usually associated with systemic symptoms	Anything, especially antibiotics (β-lactams), sulfonamides, allopurinol, antiepileptics, NSAIDs	7–21 days

AGEP=acute generalised exanthematous pustulosis; DRESS=drug reaction with eosinophilia and systemic symptoms; SJS/TEN=Stevens-Johnson syndrome/toxic epidermal necrolysis; TNF=tumour necrosis factor

NB Severe cutaneous adverse reactions (SCARs) are highlighted in bold

Source: adapted from Coltart and Fityan, 2021

Box 1. Warning signs that might suggest a severe rash

- Fever
- Purpura
- Angioedema
- Blistering or epidermal detachment
- Rash is painful
- Dysfunction of other organ systems (eg liver, kidneys, lungs, heart)
- Lymphadenopathy
- Mucosal involvement (eg conjunctiva, nasopharynx, mouth, urethra, anus)

assessments may reveal hepatic or renal impairment, although this can occur after rash onset.

Infection with, for example, Epstein–Barr virus, cytomegalovirus and human herpesvirus (HHV) – HHV-6 and HHV-7 – can recur. The virus may be reactivated late in the disease course, potentially contributing to relapses in skin symptoms and liver dysfunction, which may in turn trigger SJS/TEN-overlap syndrome and AGEP. The triggers for the reactivation of these viruses vary and the main triggers include a weakened immune system, hormonal changes, immunosuppressive medications, transplantation and advanced age. Other factors are inflammation, an immune response, environmental factors and hormonal changes may also trigger viral reactivation. It is important to note that the specific triggers can vary and depend on the individual and the specific virus involved.

Management approach for DRESS

DRESS frequently resolves spontaneously on discontinuation of the suspected causative medication. It should be noted, however, that DRESS carries a 5–10% mortality risk. In cases that have significant systemic involvement, immunosuppression using systemic corticosteroid or ciclosporin is warranted. Long-term sequelae may include autoimmune and endocrine abnormalities, particularly thyroid disease, which will necessitate monitoring the patient in the year following recovery.

Acute generalised exanthematous pustulosis

AGEP presents as a rapidly progressing erythematous rash, primarily affecting the proximal flexures. It is caused primarily by a drug reaction, for example exposure to aminopenicillins,

macrolides, antifungals, calcium channel blockers, diltiazem and antimalarials (Lee et al, 2015). It gives rise to tens to hundreds of pinhead-sized, superficial, sterile, non-follicular pustules that may coalesce. Fever is typically present, although systemic involvement is not a prominent feature. Neutrophilia (with levels exceeding 7.0×10^9/L) is evident in blood tests, and bacterial culture of pustule fluid yields negative results.

Symptoms generally resolve spontaneously on discontinuation of the causative medication, without necessitating specific treatment. AGEP carries a favourable prognosis with low mortality, typically resolving within 1 week after stopping the suspected agent. Differential diagnosis should consider pustular psoriasis, which can be identified through skin biopsy.

Infections

Viral infections often give rise to pronounced rashes, particularly maculopapular exanthems, which can be challenging to differentiate from maculopapular drug reactions. However, most viral-induced rashes are not dangerous, especially when specific features, including those listed in *Box 1,* are absent. It is beyond the scope of this article to comprehensively cover all potential skin-related emergency presentations associated with viral infections, however, two of the infections described below – SARS-CoV-2 and necrotising fasciitis – should be well known to practitioners.

COVID-19 and its cutaneous manifestations

COVID-19, an infectious disease caused by SARS-CoV-2, exhibits several cutaneous manifestations, including vesicular, urticarial and maculopapular exanthema. Notably, purpura and livedoid skin changes are linked to a more severe disease phenotype. In addition, a chilblain-like rash (popularly referred to as 'COVID toe') may appear on the hands and feet several weeks after COVID-19 has resolved. This phenomenon is thought to be associated with disease-related vasculopathy.

Eczema herpeticum

This represents a potentially severe superimposed herpes simplex virus (HSV) infection in patients with atopic eczema (*Figure 2*). Patients often present with fever and a painful, diffuse eruption characterised by monomorphic (similar shape and size) vesicles and erosions on eczematous skin. Treatment typically involves aciclovir administration and, if necessary, topical corticosteroids for the management of eczema. Urgent ophthalmology assessment is crucial when there is involvement around the eyes to prevent long-term complications associated with HSV infection.

Necrotising fasciitis

This life-threatening bacterial infection (*Figure 3*) primarily affects the subcutaneous fat and fascia (Manovah et al, 2024). It is most commonly caused by haemolytic group A Streptococcus or *Staphylococus aureus*. Early stages can be challenging to differentiate from simple cellulitis.

Key diagnostic clues include pain disproportionate to skin changes, the emergence of purpura, blistering, anaesthesia and palpable crepitus (resulting from gas formation within the tissue).

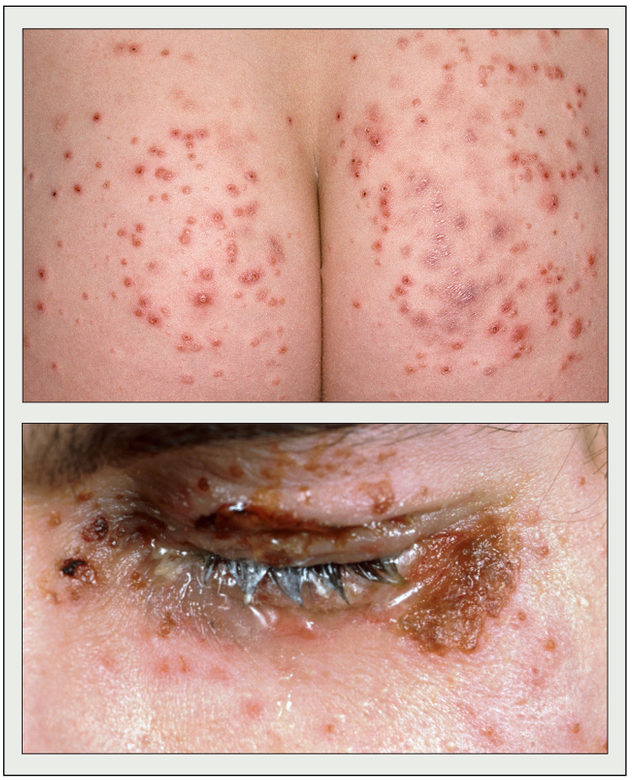

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Figure 2. Eczema herpeticum

Immediate surgical debridement and intravenous antibiotics are essential.

Staphylococcal scalded skin syndrome A

Staphylococcal scalded skin syndrome A is a toxin-mediated blistering condition caused by *S.aureus* (Ekeh et al, 2024). It predominantly affects children. Patients present with painful superficial blisters or erosions on an erythematous base, primarily involving the flexures. Treatment involves antibiotics to eradicate toxin-producing *S.aureus*, along with analgesia and supportive skin care. Mortality rates are low (0.3%) in children, but higher (up to 4%) in adults.

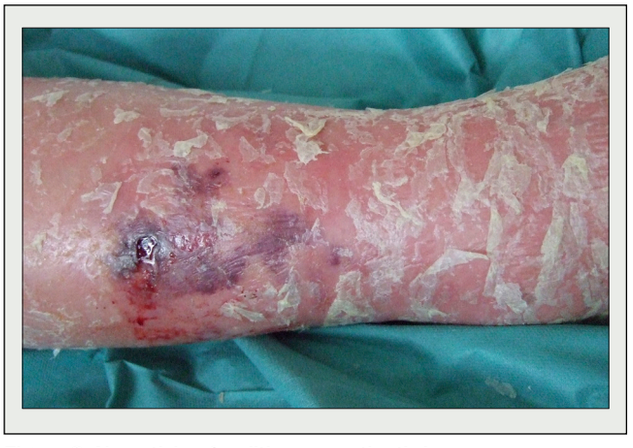

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Figure 3. Necrotising fasciitis on a patient's leg

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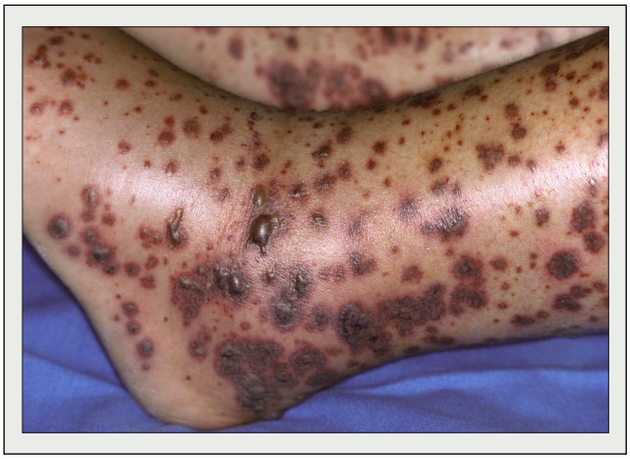

Figure 4. Cutaneous vasculitis: purpura (lesions 4–10 mm in size) and petechiae (<4 mm) on the legs, with haemorrhagic bullae

Purpura and cutaneous vasculitis

Cutaneous vasculitis rarely constitutes an emergency in itself, but it can serve as an indicator of systemic vasculitis that necessitates urgent treatment to prevent organ damage (where the kidneys, lungs, and nerves are affected). The most frequent cause of cutaneous vasculitis is leukocytoclastic vasculitis, often triggered by infection or medication. Removal of the underlying cause typically leads to spontaneous resolution. However, the differential diagnosis encompasses a wide range of conditions, including antineutrophil cytoplasmic antibody (ANCA)-associated vasculitides, immunoglobulin (Ig) A vasculitis, Kawasaki disease, and polyarteritis nodosum.

Vasculitis presents with the '4 Ps':

- Palpable: the lesions are detectable by touch.
- Painful: patients experience discomfort.
- Purpuric: the rash does not blanch on pressure (non-blanching)
- Papules.

Lesions appear as small, raised areas on the skin. These features are often prominent on the lower legs, following a gaiter distribution (*Figure 4*). However, they can also be widespread. A thorough history and examination can provide valuable clues regarding the aetiology. For instance, new-onset asthma may suggest eosinophilic granulomatous polyangiitis, while sinusitis could point to granulomatous polyangiitis.

When evaluating patients with suspected vasculitis, health professionals should conduct a comprehensive assessment to identify systemic involvement. Key investigations include inflammatory markers (such as erythrocyte sedimentation rate and C-reactive protein), FBC, renal function tests, urinalysis (to detect haematuria or proteinuria), and liver function tests. Depending on the clinical presentation, imaging studies may be warranted. A vasculitic screen should encompass hepatitis B/C serology, HIV antibody testing, complement concentrations, antinuclear antibodies (ANA), ANCA, rheumatoid factor, cryoglobulins, immunoglobulins, and serum electrophoresis. Skin biopsy can provide valuable confirmation of the diagnosis. Management strategies primarily focus on addressing the underlying cause.

Purpura fulminans, an acute and life-threatening intravascular thrombosis, often arises in response to infection (most commonly *Neisseria meningitidis*). Patients present with irregular, well-defined purpura that subsequently become necrotic due to micro-occlusion of vessels and subsequent haemorrhage. These patients are typically severely unwell and hypotensive, and may develop disseminated intravascular coagulation.

Blistering: pemphigus vulgaris

Pemphigus vulgaris is an acquired autoimmune condition characterised by widespread flaccid and easily ruptured blisters and erosions affecting both the skin and mucous membranes.

Diagnostic features:

- Skin biopsy: demonstrates intraepithelial separation (acantholysis) and blister formation.
- Direct immunofluorescence biopsy: confirms the diagnosis through the presence of intercellular IgG deposition (specifically targeting desmoglein 1 and 3) within the epidermis.

Management approach:

- Systemic corticosteroids: initial control of the disease often requires systemic corticosteroids (typically at a dose of 0.5-1 mg/kg prednisolone)
- Steroid-sparing immunosuppressants: concurrently, steroid-sparing immunosuppressants such as azathioprine, mycophenolate mofetil, or rituximab may be introduced.

Conclusion

Accessing patients with acute dermatological emergencies requires a high level of skill and knowledge to ensure life-threatening conditions are identified and the patient is referred to the appropriate specialty. Although the role of ACPs is increasingly growing in both primary and secondary care settings, many ACPs may not feel confident in dealing with dermatological emergencies. This clinical review has sought to provide an overview of acute dermatological emergencies and their management plans. Furthermore, it will help ACPs to use a high degree of autonomy and complex decision-making alongside demonstrating the four pillars of advanced practice. **BJN**

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KEY POINTS

- Advanced clinical practitioners, despite their broad scope of practice, may encounter challenges in managing dermatological emergencies due to the complexity of the specialty. Ongoing education and professional development to increase confidence and proficiency in this area is crucial
- Dermatological emergencies can present with a range of clinical features, encompassing both cutaneous and systemic manifestations
- Certain severe drug rashes may emerge as late as 6 weeks following commencement of a medication. Therefore, conducting a comprehensive review of all medications commenced within this time frame is crucial to accurately identifying and discontinuing the suspected causative agent
- Early recognition of dermatological emergencies such as Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) is critical for improved patient outcomes

CPD reflective questions

- How can ACPs proactively address gaps in their knowledge and skills related to acute dermatological emergencies to enhance their confidence and competence in this domain?
- What strategies can healthcare institutions implement to facilitate interdisciplinary collaboration and communication among ACPs, dermatologists and other specialists to optimise patient outcomes in dermatological emergencies?
- In what ways can ACPs integrate reflective practice into their clinical workflows to continuously evaluate and refine their management approaches for acute dermatological conditions?