Dermatologic emergencies

Dermatologic problems represent about 15%–20% of visits to family physicians and emergency departments. It is often a challenge for a primary care provider to differentiate mundane skin ailments from more serious, life-threatening conditions that require immediate intervention. The purpose of this article is to highlight some dermatologic emergencies.

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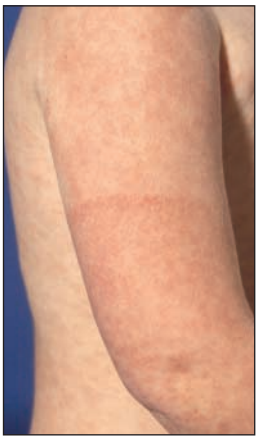
Staphylococcal toxic shock syndrome: This toxin-mediated disease is characterized by rapid onset of generalized erythema with desquamation, fever, hypotension and potential mutisystem failure. The illness occurs in the setting of *Staphylococcus aureus* infection, for example in women using menstrual tampons of high absorbency, or in patients with superficial skin or surgical wound infections. *S. aureus* produces exotoxins, which induce massive cytokine secretion by T cells; fever, hypotension and multi-organ failure result. After a 2–3 day prodrome period of malaise, patients typically present with fever, chills, nausea and abdominal pain. A diffuse erythematous, nonpruritic, maculopapular or petechial rash ensues, with subsequent desquamation. The rash initially appears on the trunk and spreads peripherally to the palms and soles. Multi-system involvement includes arrhythmias, hepatic and renal failure, disseminated intravascular coagulation and acute respiratory distress syndrome.



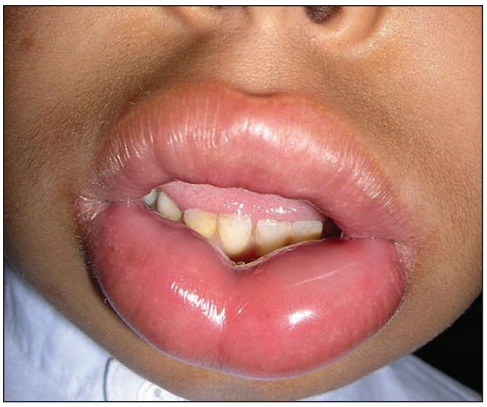
Dr. Brenda Moroz

Management: Aggressive supportive management is required to treat hypotension and potential multi-organ failure. Inciting factors should be removed. Once culture specimens are obtained, treatment with a β-lactamase-resistant anti-staphylococcal antibiotic is recommended.

Angioedema: Characterized by well-circumscribed areas of edema caused by increased vascular permeability, this condition affects mostly the skin, and the gastrointestinal and respiratory tracts. Patients typically present with acute subcutaneous swelling, usually of the face, extremities or genitalia.

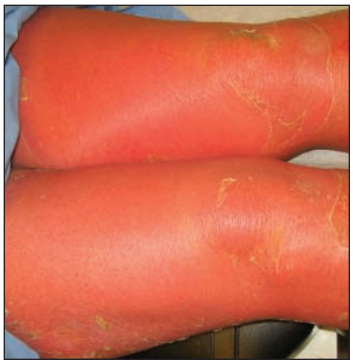
A generalized anaphylactic reaction may occur, which is potentially fatal if the upper airway is compromised. Urticaria can be associated with angioedema in 50% of cases; the angioedema is usually nonpruritic but burning. Although often idiopathic, angioedema can be induced by medications, allergens (e.g., food) or physical agents (e.g., vibration, cold). Typically, 10%–25% of cases are due to angiotensin-converting-enzyme (ACE) inhibitor therapy, occurring in 1–2 per 1000 new users. Penicillins, NSAIDs and radiographic contrast media are other potential triggers. Angioedema can occur as a result of C1 esterase inhibitor (C1INH) deficiency. Two rare but well-described categories exist: hereditary angioedema, which is transmitted in autosomal-dominant fashion, and acquired angioedema, which can be associated with autoimmune disorders and B-cell lymphoproliferative malignant disease.

Management: Treatment is largely supportive. Airway patency must be ensured if the respiratory system is involved. Cool, moist compresses and antihistamines can be used to control local burning. Referral to an allergy specialist for appropriate investigations should be considered. Avoidance of known triggers, such as associated medications, is paramount. ACE inhibitors are contraindicated in patients with C1INH deficiency. Attenuated androgens danazol and stanozolol increase the amount of active C1INH and are used for the prevention of hereditary angioedema. An algorithm for the diagnosis and management of hereditary angioedema was recently published.[1]



Dr. Shabaz Janjua, DermNet NZ

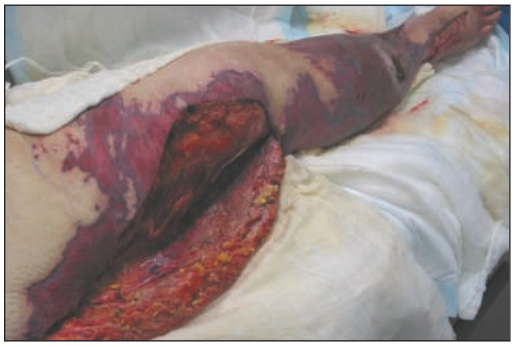
Exfoliative erythroderma: This is a generalized scaly erythematous skin eruption involving more than 90% of the cutaneous surface. Although many cases are idiopathic, it can be associated with a diverse range of underlying dermatoses, including eczema, psoriasis, drug reaction (e.g., allopurinol, calcium-channel blockers, anticonvulsants and lithium), cutaneous T-cell lymphoma or leukemia, pityriasis rubra pilaris, paraneoplastic syndrome and dermatomyositis. Pruritus is usually the initial symptom, and malaise and fever may subsequently develop owing to excessive vasodilation. Fluid and protein loss through the skin can lead to life-threatening hypotension, electrolyte imbalance, congestive heart failure and enteropathy. In a study involving 91 patients with erythroderma, the disease-specific mortality was 18%;[2] 53% of the cases were associated with exacerbation of an existing dermatosis, which underscores the need for a thorough history and skin examination to identify a potential underlying skin condition.



Management: The cause should be determined and, if a drug reaction is suspected, the offending medication stopped. Skin biopsies can be obtained to help establish the diagnosis. Supportive therapy includes hospital admission, proper hydration, nutrition, electrolyte and cardiac monitoring, and temperature support. Skin care involves the use of emollients and compresses as well as topical corticosteroid therapy and antihistamines for pruritus. Antibiotic therapy should be administered if signs of infection develop.

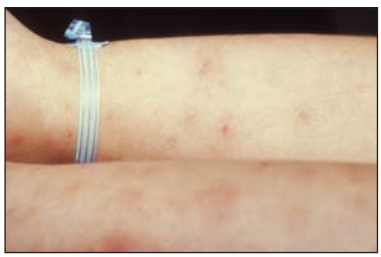
Necrotizing fasciitis: This rapidly spreading infection of the deep fascia can cause necrosis of the subcutaneous tissues. Type II necrotizing fasciitis is caused by group A streptococci, whereas mixed anaerobes, gram-negative aerobic bacilli and enterococci are implicated in type I. The organisms can be introduced through minor cuts, burns, blunt trauma or surgical procedures. Risk factors include diabetes mellitus, peripheral vascular disease and immunosuppression. The first cutaneous manifestation of streptococcal necrotizing fasciitis is diffuse swelling of the affected skin area followed by the development of bullae, which rapidly become burgundy in colour. Without prompt treatment, the infection may develop into frank cutaneous gangrene. Shock and organ failure can ensue and portend a poor prognosis. When skin necrosis is not obvious, diagnosis of necrotizing fasciitis must be suspected if there are signs of severe sepsis or some of the following local symptoms and signs: severe pain, indurated edema, skin hyperesthesia, crepitation, muscle weakness and foul-smelling exudate.

Management: Early recognition, aggressive management of sepsis and surgical débridement of the necrotic tissue are essential for effective treatment of necrotizing fasciitis. Antistreptococcal antimicrobial therapy should be administered; however, if the causal agents cannot be definitively identified, the patient should be given broad-spectrum antibiotics, as dictated by the clinical picture.



Dr. Benjamin Barankin

Meningococcemia: *Neisseria meningitidis* is a leading cause of meningitis and septicemia among North American youth. The incidence of invasive meningococcal disease is about 1.1 cases per 100 000, with the peak incidence at 6–12 months of age, when protective antibodies have not yet developed. The classic presentation of meningococcemia is the abrupt onset of maculopapular or petechial rash and flu-like symptoms, including fever, chills, malaise and disorientation. Over several hours, the disease may rapidly progress to purpura, disseminated intravascular coagulation, shock and death.

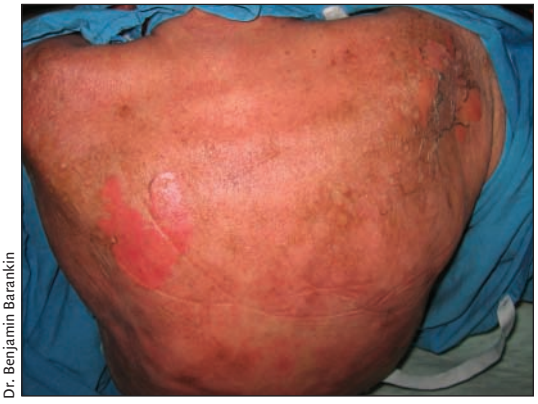


Dr. Brenda Moroz

Management: Any febrile patient with a petechial rash should be suspected of having meningococcemia and treated promptly after blood cultures are obtained. Besides supportive management, therapy with a third-generation cephalosporin (e.g., ceftriaxone) or intravenous penicillin G therapy are the treatments of choice. Chloramphenicol may be used for patients allergic to penicillin.

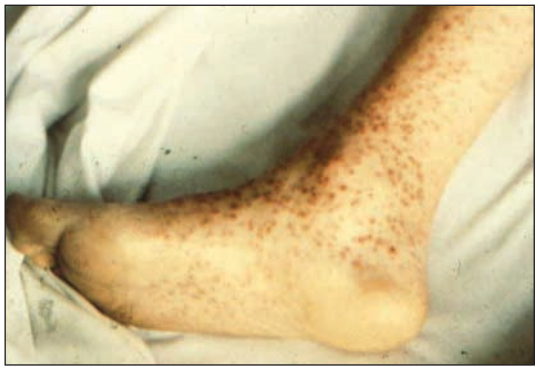
Stevens–Johnson syndrome and toxic epidermal necrolysis: These conditions represent a spectrum of drug-induced or idiopathic mucocutaneous reaction patterns, characterized by skin tenderness, erythema, epidermal necrosis and desquamation. The pathogenesis is thought to involve an impaired capacity to detoxify intermediate drug metabolites and genetic susceptibility. Common medications responsible for Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) include sulfonamides, anticonvulsants, allopurinol and NSAIDs. Drug administration typically precedes the rash by 1–3 weeks. Patients may present with a prodrome of fever, stinging eyes and pain on swallowing, followed by the development of dusky erythematous macules that progress to flaccid blisters. Two or more mucous membranes are usually involved, with erythema and erosions of buccal, genital and ocular mucosa. Severe ophthalmic involvement may lead to permanent scarring and blindness. Epidermal detachment is common, which may lead to massive fluid loss and electrolyte imbalance. Less than 10% of the epidermis sloughs off in SJS and more than 30% in TEN. These conditions are potentially life-threatening because of their multisystem involvement and skin-barrier breakdown. Epithelial loss results in vulnerability to bacterial and fungal infections and predisposes patients to septicemia and severe fluid loss. Mortality ranges from 5% in SJS to 30% in TEN.

Management: SJS and TEN should be managed by an experienced physician. Supportive measures include identification and removal of the offending medication, admission to a burn unit if necessary, intravenous fluid administration, maintenance of electrolyte and temperature homeostasis, and ophthalmologic assessment in case of ocular involvement. Skin care consists of proper wound dressings, oral hygiene (i.e., chlorhexidine rinses), antihistamine and topical corticosteroid therapy for pruritus, and antimicrobial therapy in cases of superinfection due to skin-barrier breakdown. Some centres use high doses of immunoglobulin intravenously as mainstay therapy for TEN; however, more evidence of its effectiveness is needed.



Dr. Benjamin Barankin

Rocky Mountain spotted fever: This condition is potentially life-threatening and is most commonly introduced to humans through tick bites carrying the *Rickettsia rickettsii* parasite. Canadian vectors of *R. rickettsii* include *Dermacentor variabilis* (dog tick) in eastern Canada and *Dermacentor andersoni* (wood tick) in western Canada. Rocky Mounted spotted fever is associated with a mortality of 3%–7% among treated patients and 30%–70% among those not treated promptly or adequately. In about 60% of cases, patients present with a triad of fever, headache and rash following a tick bite. The classic rash with this condition appears within the first 2 weeks on the wrists and ankles and rapidly spreads to the palms and soles and eventually to the trunk and face. Purpuric macules and papules can be observed. Multiorgan involvement may lead to a variety of symptoms, which makes the diagnosis challenging. Complications of Rocky Mounted spotted fever include myocarditis and cardiogenic shock, peripheral edema due to hepatic failure and hypoalbuminemia, acute renal failure, altered mental status, seizures or coma, meningismus and disseminated intravascular coagulation.



Dr. Bernice Krafchik

Management: Besides symptomatic support, appropriate antibiotic treatment should be initiated immediately when this condition is suspected. Doxycycline is the drug of choice and should be continued for at least 3 days after fever subsides and until clinical improvement. The tick should be removed if embedded in the skin at the time of presentation.

This article has been peer reviewed.

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