Acute skin conditions in children

Introduction

This article covers four acute skin conditions seen in children. Early recognition and appropriate prompt treatment is important as significant morbidity may ensue.

Eczema herpeticum

Eczema herpeticum is a disseminated herpes simplex infection (usually herpes simplex virus type 1; HSV1) of the skin occurring in patients with pre-existing dermatitis. The severity varies from mild, transient disease to a fulminating fatal disorder involving the visceral organs. It is commoner in children but is also occurring with increasing frequency in adults (Bork and Brauninger, 1988). Eczema herpeticum can rarely complicate other dermatological conditions such as sebhorroeic dermatitis, Darier's disease and pemphigus (Niimura and Nishikawa, 1988).

Clinical manifestations

The presentation is usually typical, with crops of painful vesicles 2–3 mm in diameter, often with umbilication. There is usually an inflamed erythematous base and the clear fluid contained becomes turbid or purulent. The rash is rapidly evolving and becomes confluent over large areas of skin. Lesions rupture to leave shallow erosions with weeping and crusting that can become secondarily infected (*Figure 1*).

The eruption occurs in areas of pre-existing dermatitis and there is a predilection for the upper body and face. The eyelids may become affected with progression to the eyes themselves; a condition known as ocular keratitis.

The rash continues to spread over 7–10 days and is commonly associated with non-specific systemic symptoms.

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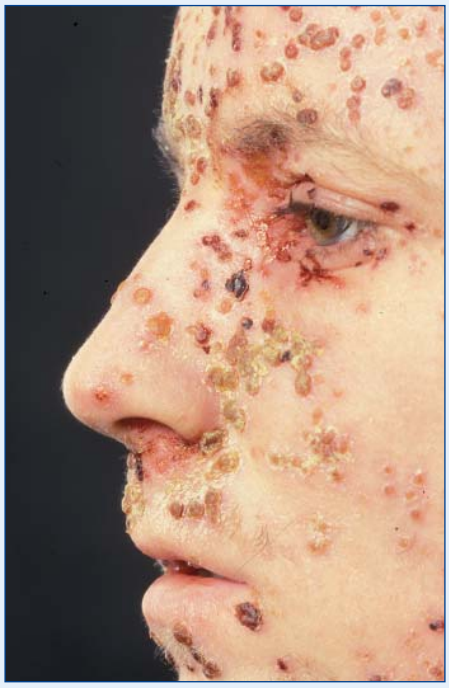


Figure 1. Eczema herpeticum: multiple crops of vesicles with crusting.

Diagnosis

Diagnosis is based on a history of atopic dermatitis, presence of crops of vesicular lesions and rapid progression. Waiting for positive viral culture for HSV is not necessary as this delays treatment. Electron microscopy of fresh vesicular fluid is a more useful and reliable diagnostic test; a result can be available within a few hours. If the lesions are atypical or old, then polymerase chain reaction for HSV can be considered. Skin swabs should also be taken and cultured for evidence of secondary bacterial infection.

Complications

Involvement of the eyes can lead to blepharitis, conjunctivitis, keratitis and uveitis. Herpetic keratitis can lead to blindness as a result of stromal scarring. In these circumstances, regular ophthalmological review is required (Weston et al, 2007).

Treatment

Early antiviral therapy is imperative. If HSV infection is suspected clinically then antiviral therapy should be started before microbiological confirmation. Acyclovir is first-line therapy for eczema herpeticum and is a safe and effective drug (Niimura and Nishikawa, 1988). It is given orally 200–400 mg five times per day. In severe or widespread disease 20–40 mg/kg/day intravenously is indicated (Weston et al, 2007). Disseminated infections require hospitalization as systemic viraemia is the main cause of morbidity and mortality.

In cases with secondary bacterial infection topical or oral antibiotics should be used concurrently.

Patients with severe, recurrent HSV infections are treated prophylactically with a 6-month course of acyclovir 200 mg twice daily. This will reduce recurrences and prevent transfection to other skin sites or family members and playmates (Weston et al, 2007). Education of the patient and his/her family is paramount to reduce spread of this highly contagious infection. Optimal treatment of the underlying skin disease is important in preventing further problems.

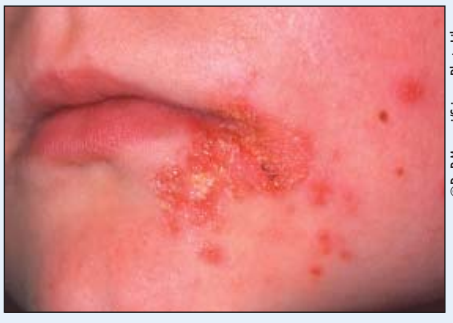
Impetigo

Impetigo is a highly contagious gram-positive bacterial infection of the superficial epidermis. The two causative organisms are *Staphylococcus aureus* and group A beta-haemolytic streptococcus. Both may be present simultaneously at the same affected site.

Clinical manifestations

Impetigo contagiosa begins as small fragile vesicles which are 1–2 mm in diameter. The roof is quickly lost to leave erosions with a typical moist, golden-honey coloured crust (Weston et al, 2007) (*Figure 2*). Multiple lesions are present

Figure 2. Typical impetigo lesion around mouth of young child displaying golden crust.



and occur on exposed areas such as the face, nares and extremities. Lesions can be itchy and spread rapidly as a result of direct inoculation.

Complications

Staphylococcal infection may give rise to other more serious variants. Bullous impetigo is less common than impetigo contagiosa. The causative agent of bullous impetigo is gram-positive, coagulase-positive, group II *S. aureus* (Burns et al, 1998) and the condition is a result of dissemination of bacterial exfoliative toxins. Bullous impetigo is most commonly seen in neonates and infants. Epidemics can occur following transmission in nurseries (Burns et al, 1998).

Bullous impetigo begins as small painful blisters that rapidly enlarge and then rupture, and often found in the flexures. Minor skin traumas such as scratches or insect bites are important risk factors which can trigger the condition (Johnston, 2004). Infection spreads to adjacent and distal sites through direct inoculation.

Clinical manifestations

The characteristic lesion is a vesicle that develops into a superficial, flaccid blister on intact skin. There is minimal or no surrounding erythema. Sites of predilection for the initial lesion are the face, neck, perineum and periumbilical regions (Burns et al, 1998). They may, however, be present anywhere on the body and can be widely scattered. The vesicle initially contains clear fluid that may become turbid or frankly purulent (Burns et al, 1998). The roof of the bulla ruptures, leaving an extending area of exudation and yellow crusting (Johnston, 2004). Intact bullae rupture easily and so may not be seen clinically.

Lesions of a primary underlying skin disease, such as atopic dermatitis or chickenpox, should be looked for. In infants, extensive lesions may be associated with fever, malaise and diarrhoea (Burns et al, 1998).

Differential diagnosis

There is a wide differential diagnosis of acute blistering in infancy. The more important conditions to consider are thermal or chemical burns, viral infections such as HSV or herpes zoster and the staphylococcal scalded skin syndrome (see below) (Burns et al, 1998).

Investigations

Diagnosis is made primarily on clinical findings. Bacterial culture of blister contents is important, however may be negative. Nasal swabs of the patient and immediate relatives should be performed to identify asymptomatic nasal carriers of *S. aureus*.

Treatment

In the treatment of impetigo contagiosa topical antibiotics are used. Topical fusidic acid is first line and topical mupirocin for proven cases of bacterial resistance. Oral antibiotics should be used additionally in more severe cases. First-line systemic therapy is oral flucloxacillin or erythromycin (Johnston, 2004). Gentle debridement of lesional crusts using antibacterial soap and a washcloth is useful. Good hygiene with antibacterial washes, such as chlorhexidine, may prevent the spread of impetigo and prevent recurrences, but the efficacy of this has not been proven.

Untreated bullous impetigo in the infant is associated with significant morbidity and so requires more aggressive treatment from the outset with intravenous antibiotics as outlined above. Failure to treat may lead to complications including pneumonia, septic arthritis and osteomyelitis (Burns et al, 1998).

Any underlying skin condition should be treated aggressively to minimize the chances of further attacks.

Staphylococcal scalded skin syndrome

The staphylococcal scalded skin syndrome and bullous impetigo represent a spectrum of blistering skin disease caused by staphylococcal exfoliative toxins. In bullous impetigo, the exfoliative toxins are restricted to the area of infection and bacteria can be cultured from blister contents. In staphylococcal scalded skin syndrome the exfoliative toxins are spread haematogenously from a localized source causing widespread epidermal damage at distant sites (Johnston, 2004). This is a more severe consequence of staphylococcal infection.

Clinical features

In children the exfoliative toxin-producing strain of *S. aureus* is usually found in a commensal site such as the conjunctiva, perineum, axilla, umbilicus or at an infective site such as a wound (Johnston, 2004).

There is a swift onset of painful, tender and red skin and this is often accentuated in flexural and peri-orifical areas. After 24–48 hours flaccid blisters and erosions develop and large areas of the overlying epidermis loosen and peel like a scald (*Figure 3*). Conjunctival inflammation, perioral erythema and crusting and lip fissuring is characteristic at this stage although mucosal lesions are rare. Important physical signs include skin tenderness, denudation in areas of skin stress and rubbing, and Nikolsky's sign (separation of the outer epidermal layer of the skin from the underlying dermis on gentle rubbing) (Resnick et al, 1991).

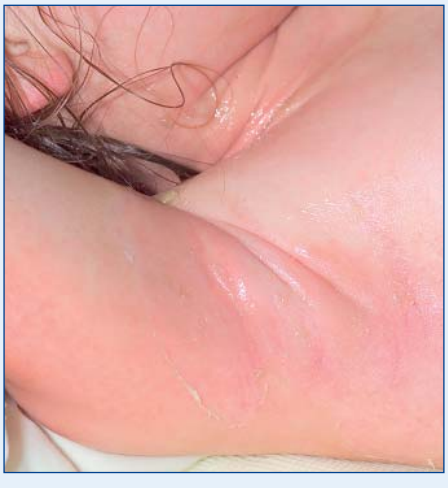
Differential diagnosis

The most important differential diagnosis from staphylococcal scalded skin syndrome is toxic epidermal necrolysis. The latter is rare in infancy and childhood, is usually drug induced, is associated with extensive and severe mucosal involvement, and is often seen in older, previously unwell patients with significant co-morbidity. It has a high mortality (Johnston, 2004).

Investigations

Diagnosis of staphylococcal scalded skin syndrome is usually clinical. Gram stain and culture of blister fluid in staphyloco-

Figure 3. Staphylococcal scalded skin syndrome showing typical peeling lesion in the axilla.



ccal scalded skin syndrome is usually negative as exotoxins produced at a distant site of staphylococcal infection mediate the disease. A skin biopsy is not usually indicated except in cases of diagnostic doubt, for example, differentiating from toxic epidermal necrolysis (Johnston, 2004).

Treatment

Intravenous antibiotic therapy is indicated in extensive, evolving cases of staphylococcal scalded skin syndrome and where the child has evidence of systemic illness or comorbidity such as immunosuppression that will result in more aggressive disease. The treatment of choice is flucloxacillin. Systemic corticosteroids aggravate the disease and are absolutely contraindicated (Burns et al, 1998).

Erythema multiforme

Erythema multiforme is an acute, self-limiting mucocutaneous disorder of variable severity usually occurring in adolescents and young adults. It can be divided into major and minor forms. Erythema multiforme minor represents a localized eruption of the skin with mild or no mucosal involvement. Erythema multiforme major is a more severe mucosal and skin disease and is potentially life-threatening (Johnston et al, 2002).

An infective aetiology caused by HSV is thought to be the cause in the majority of affected children. Epstein–Barr virus, cytomegalovirus and other human herpesviruses have also been implicated (Weston et al, 2007). Antecedent upper respiratory tract infection by *Mycoplasma pneumoniae* has been associated with cases of erythema multiforme major (Johnston et al, 2002).

Clinical features

Erythema multiforme minor is characterized by the onset of oval or round, fixed, erythematous skin lesions. These lesions progress over a period of several days to form concentric zones of colour change with an erythematous border and a central region of crusting or blistering. This is the classical 'target lesion' (*Figure 4*). Each lesion may last up to 3 weeks (Weston et al, 2007). Initial sites of involvement are the dorsal surface of the hands and extensor aspects of the extremities. Palms and soles are also often affected. Erythema multiforme may exhibit the Koebner phenomenon whereby lesions occur in sites of previous trauma (Weston et al, 2007). Oral lesions are present in half of affected patients, but these tend to be few in number. Systemic symptoms are absent in erythema multiforme minor.

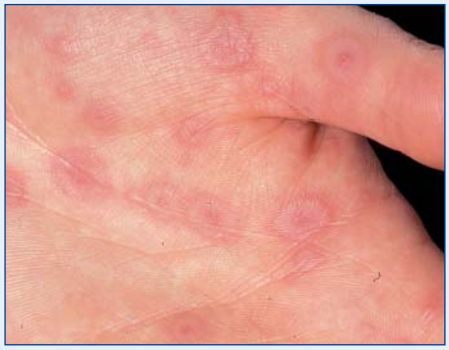


Figure 4. Erythema multiforme: typical target lesions.

Complications

Erythema multiforme major is a more severe variant comprising target lesions, frequent mucous membrane involvement and marked dermal inflammation leading to blister formation (Johnston et al, 2002). Prodromal symptoms are common as are pulmonary symptoms with X-ray changes (Ginsburg, 1982). Systemic symptoms are present and include fever, malaise, respiratory and gastrointestinal upset.

Treatment

The mainstay of treatment is symptomatic relief. This can be achieved by oral antihistamines and wet compresses. In erythema multiforme major the patient is usually systemically unwell and will require hospitalization.

For those with recurrent episodes secondary to proven HSV infection, prophylactic oral acyclovir may be considered.

Education of both the patient and the parents about the role of HSV is important in those with recurrences. Precipitating factors for HSV infection should be avoided as described previously.

Conclusions

These childhood dermatological emergencies cause significant morbidity and mortality. Infection occurs more commonly in pre-existing skin conditions, particularly those that are poorly controlled. Microbiological samples should be taken before commencing antibacterial or antiviral therapy to confirm the diagnosis and identify resistant organisms. Early recognition and treatment is imperative. **BJHM**

Conflict of interest: none.

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KEY POINTS

- Eczema herpeticum is caused by herpes simplex virus 1 infection in those with pre-existing dermatitis and presents with crops of vesicles. Secondary bacterial infection is common.
- Bullous impetigo is caused by *Staphylococcus aureus*. It represents the mild end of a spectrum of blistering infectious skin disease.
- The staphylococcal scalded skin syndrome consists of widespread painful blistering and superficial denudation caused by exfoliative toxins that are spread haematogenously from a localized source.
- Erythema multiforme is usually secondary to infection, often herpes simplex virus 1, and presents as target lesions. It is self limiting.