TODDLERS (1 - 5 YEARS)

Increasing interaction with and exposure to other children predisposes toddlers to a variety of skin problems, especially infections and infestations.

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Crèches, play schools and nursery schools bombard the toddler with air-borne micro-organisms and allergens. Some toddlers may have a genetic predisposition to atopic dermatitis, but apart from this, the toddler years are a relatively quiet period dermatologically, superseded to a large extent by upper respiratory tract infections and gastroenteritis.

ATOPIC DERMATITIS

Atopic dermatitis can occur *de novo* at this stage or continue from infancy. Generally the skin will be very dry, and subacute (inflamed, red, swollen, weeping and crusted) patches of eczema occur, especially on the cheeks and extensor aspects of the limbs, with follicular eczema causing a goose-flesh appearance on the trunk (Fig.1). As the child gets older the sites of maximal involvement can change to include the flexor aspects of the limbs. Atopic children have an increased tendency to urticaria, and direct exposure, by touching, to an allergen like grass will cause contact urticaria, giving the (false) impression that the eczema is caused by the grass. (See article on infants for details of treatment — pp. 493 - 495.) Topical steroid preparations are the mainstay of therapy for active atopic dermatitis, and they should be used liberally and freely until all inflammation subsides. Obviously this treatment is not curative, and recurrences should be similarly managed. Fortunately many toddlers will outgrow their eczema, although these individuals will have a sensitive and slightly dry skin for life. However in some children the condition worsens, and frequent relapses and flares cause considerable morbidity. In these circumstances aggressive treatment is warranted, with specialist referral if necessary. Options for the treatment of resistant eczema include long-term oral steroids, UVB phototherapy, PUVA (psoralen and ultraviolet A) therapy, azathioprine or cyclosporin.

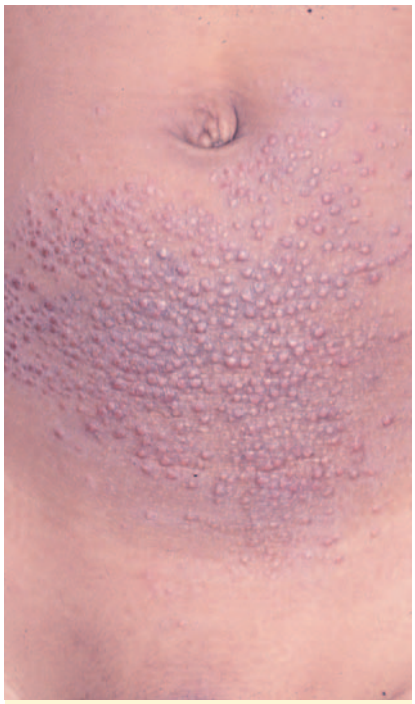

Fig. 1. Follicular eczema.

ERYTHEMA INFECTIOSUM

Also known as fifth disease, this common childhood infection is caused by parvovirus B19. The primary manifestation is a distinctive exanthem, even though there may be mild prodromal symptoms such as fever, malaise and headache. The rash has 3 phases: first a fiery-red macular erythema appears on the cheeks, the so-called 'slapped cheek' appearance. Within a few days the second phase

occurs, with erythematous macules and papules on the proximal extremities and trunk. This can evolve into a lace-like, reticulate pattern of erythema (Fig. 2). The third phase is heralded by the disappearance and subsequent reappearance of the latter rash, a situation that can continue for weeks. Diagnosis can be confirmed on serology, but this is usually not indicated. No treatment is necessary, and infected children need not be isolated, as once the rash occurs the infectious period is over. Parvovirus B19 infection in adulthood can be significant, as complications like arthritis, anaemia, haemolysis and fetal loss can occur in susceptible hosts.

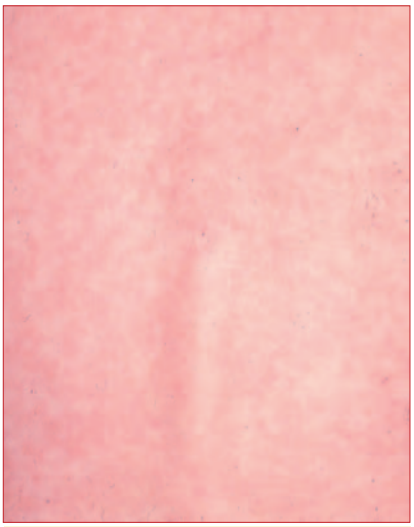

Fig. 2. Erythema infectiosum.

ERYTHEMA MULTIFORME

This reactive dermatosis of uncertain pathogenesis is seen quite commonly in toddlers, but can occur at any age. There is a wide range of severity, from trivial to life-threatening, the latter usually caused by drugs. Most forms are mild and self-limiting, but recurrences can occur. Typically, a symmetrical eruption occurs, with erythematous macules and papules concentrated on the face, ears and extensor aspects of the extremities (Fig. 3). The hands and feet can be involved, including palms and soles. The commonest lesion of erythema multiforme is a rather non-specific papule with a crusted or necrotic surface, but the classic lesion is the so-called target or iris lesion, which is a round plaque with central dusky erythema surrounded by concentric rings of pallor and erythema (Fig. 4). The presence of target lesions makes the diagnosis much easier, but they are frequently absent. Mild erythema multiforme lasts about 2 weeks, and sequelae include postinflammatory pigmentation, especially in dark skins.

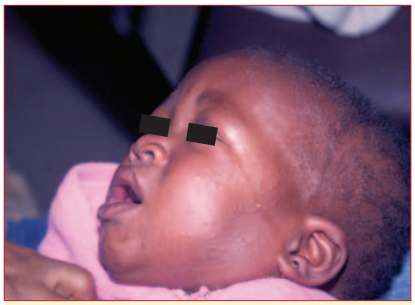

Fig. 3. Erythema multiforme — face.

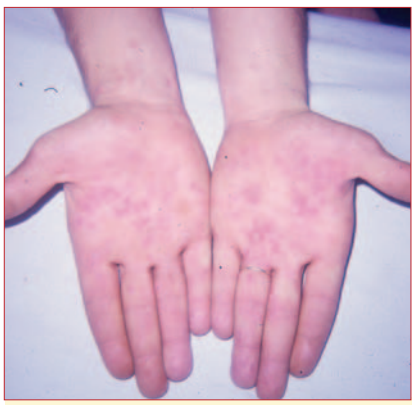

Fig. 4. Target lesions of erythema multiforme.

Severe erythema multiforme, also known as Stevens-Johnson syndrome, is the acute fulminant form, with extensive mucous membrane involvement, skin blistering and systemic toxicity. A prodrome of fever, cough, sore throat, myalgia and malaise precedes the rash, which is highly variable and consists of erythematous macules, papules, vesicles, bullae and sheets of epidermal necrosis (a dusky, violet colour). Blisters on the lips, in the mouth, on the conjunctivae and in the urethra cause severe pain and difficulty with swallowing and voiding. Exuberant haemorrhagic crusts form on these blisters, giving a striking appearance. Eye lesions can result in scarring and blindness. Severe erythema multiforme lasts 4 - 6 weeks and most patients recover fully. However, particularly in the setting of immune compromise or another chronic illness, there may be significant mortality.

Erythema multiforme is a hypersensitivity reaction to foreign antigens. The best-documented causes are herpes simplex virus, *Mycoplasma pneumoniae* infection and drugs. In cases of herpes simplex-induced erythema multiforme, the rash usually occurs 2 weeks after the acute episode of herpes infection, and can recur many times. Mycoplasma-associated erythema multiforme, which can be severe, usually follows symptoms of an upper respiratory tract infection. Drug-associated erythema multiforme is usually severe, and the major offenders include sulphonamides, anticonvulsants, penicillins, allopurinol, and non-steroidal anti-inflammatory drugs (NSAIDs).

Treatment of mild erythema multiforme includes topical steroid creams and possibly a short course of oral corticosteroids. Herpes-associated disease is treated with long-term prophylactic aciclovir or a similar agent. If mycoplasma is suspected, appropriate antibiotics are given. Stevens-Johnson syndrome is best treated in an ICU or burns unit. High-dose oral corticosteroids are usually administered, together with supportive care, antibiotics and ophthalmological attention. A Medic-alert bracelet is imperative in these cases.

GIANOTTI-CROSTI SYNDROME

Also known as papular acrodermatitis of childhood, this self-limiting condition is thought to be an unusual response to viral infections like Epstein-Barr virus, enteroviruses, cytomegalovirus and hepatitis B virus. Toddlers are most frequently affected, with a distinctive and rather chronic exanthem involving the face and limbs only. Erythematous papules resembling insect bites occur symmetrically in these sites, with variable itching (Fig. 5). Depending on the viral cause other features such as lymphadenopathy, hepatomegaly and splenomegaly may be present, and these would warrant

further serological testing. Jaundice is usually absent in the hepatitis B associated type. Specific therapy for Gianotti-Crosti syndrome is not necessary, apart from antihistamines for itch. The condition resolves in 2 - 4 weeks.

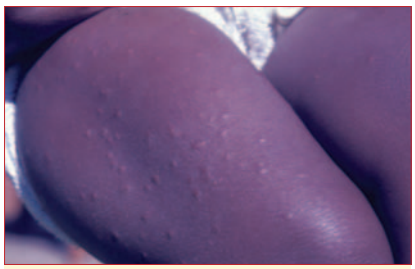

Fig. 5. Gianotti-Crosti syndrome.

HAND, FOOT AND MOUTH DISEASE

This distinctive syndrome is just one presentation of infection by enteroviruses in children, especially Coxsackie virus. After 1 - 2 days of fever and sore throat, small vesicles develop on the gums, tongue and palate, followed by pale oval vesicles with a red halo on the hands and feet. Both dorsal and volar surfaces are involved, and the vesicles often lie in palmar creases (Fig. 6). The face, buttocks and legs are sometimes similarly affected. The disease resolves in a few days. Enteroviruses cause protean dermatological manifestations, including morbilliform eruptions, vesicular, urticarial and petechial rashes.

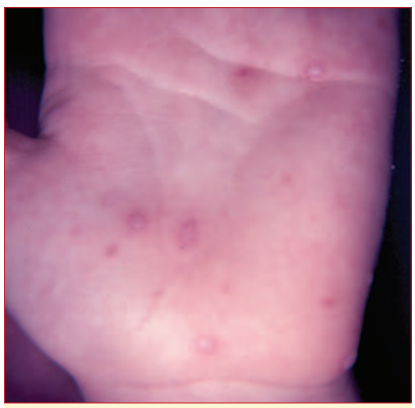

Fig. 6. Hand, foot and mouth disease — palm.

INSECT BITE REACTIONS

Exaggerated insect bite reactions are a hallmark of early childhood, especially in the presence of atopy. The commonest culprits are fleas, mosquitoes and bedbugs. Exposed parts like the face, hands and feet are mostly affected with urticarial papules and plaques, sometimes with central haemorrhagic puncta representing the insertion of the insect mouthparts (Fig. 7). The reaction is caused by type I or IV allergy to the saliva of the insect. Lesions are often grouped in linear clusters of 2 - 4 papules, corresponding to the insect moving from one site to the next for a blood meal. Occasionally bullae are induced. Itching and scratching cause secondary changes like excoriation, impetiginisation and eczematisation. Symptomatic treatment with topical steroid creams, calamine lotion, oral antihistamines and antibiotics is helpful, but the lesions are essentially self-limiting, occurring in crops as further biting occurs. Insect repellants like diethyltoluamide (Tabard, etc.) can be helpful in severe cases to prevent recurrence. A particularly severe hypersensitivity occurs in some children, causing a generalised rash with multiple urticarial papules, excoriations, lichenification and secondary infection. This condition mimics scabies and is termed papular urticaria. Insect bite reactions tend to diminish with age as immune tolerance occurs.

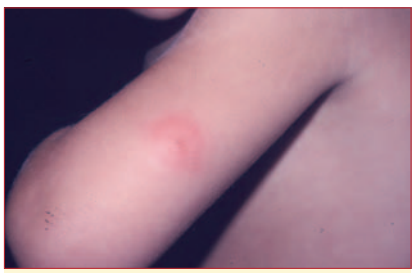

Fig. 7. Insect bite reaction.

MOLLUSCUM CONTAGIOSUM

This extremely common viral infection reaches a peak incidence in the toddler years. Boys are affected more than girls. Transmission is thought to be person-to-person. Genital lesions in adults are probably sexually transmitted. Lesions are often grouped, and start as tiny flesh-coloured or pale papules that grow into dome-shaped whitish papules with central depressions (umbilication). The papule contains a thick whitish material. Molluscum lesions occur at any site, but the face and trunk are predominantly affected (Fig. 8). They are usually asymptomatic but pruritus can occur, and perilesional eczema is common in atopic children. The disease is self-limiting in healthy hosts, resolving in 1 - 2 years, but can become progressive in the immunosuppressed. Treatment is not always necessary, and includes manual expression of the central core, pricking with a sterile needle, liquid nitrogen cryotherapy, curettage or application of cantharidin paint. Prior anaesthesia with EMLA cream is helpful for painful procedures.

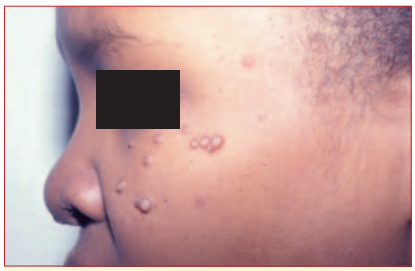

Fig. 8. Molluscum contagiosum — face.

ROSEOLA

Roseola is a common childhood viral infection occurring between 6 months and 3 years. It is caused by human herpesvirus 6, and starts with a high fever for 3 - 5 days, usually with no other symptoms. Febrile convulsions can occur. The rash characteristically occurs only when the fever subsides by crisis, and consists of discrete irregularly shaped pinkish-red macules or papules on the trunk and neck (Fig. 9).

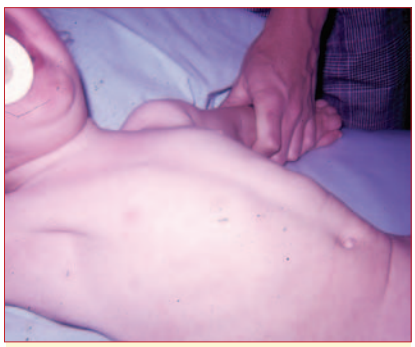

Fig. 9. Roseola.

The rash fades rapidly within hours to a few days, and there is no desquamation. No specific treatment is required.