Clinical review

Common skin infections in children

Michael J Sladden, Graham A Johnston

Most children will have a skin infection at some time. Skin infections are a common reason for consultation in primary care and in dermatology practice.[1–3] We review four common skin infections in children and describe their epidemiology, clinical features, and treatment, focusing on treatments with best evidence.

Sources and selection criteria

We searched Medline and the Cochrane Library using the terms "molluscum," "warts," "impetigo," and "tinea." We included randomised trials, meta-analyses, and clinical guidelines.

Molluscum contagiosum

Molluscum contagiosum is a common, benign, self limiting viral infection of the skin. It generally affects children and is caused by a human specific poxvirus. Infection is rare in children under 1 year of age and typically occurs in the 2-5 year age group.[4] Although the prevalence of molluscum contagiosum is not known, one of six Dutch children have visited their doctor for the condition.[5]

Infection follows autoinoculation or contact with affected people.[6] The incubation period is from two weeks to six months. The condition is more common in young children and in children who swim, who bathe together, and who are immunosuppressed. Little evidence supports the view that lesions (mollusca) are more common in children with atopic dermatitis.

Mollusca present as multiple dome shaped pearly or flesh coloured papules with a central depression (umbilication), which usually appear on the trunk and flexural areas (fig 1). They vary in size from 1 mm to 10 mm, with growth occurring over several weeks.[4] In patients who are immunocompetent, lesions may persist for six to eight weeks. The mean duration is at least eight months when new lesions appear due to continuous autoinoculation.[6] Resolution is often preceded by inflammation. Uncomplicated lesions heal without scarring.

Whether doctors should treat molluscum contagiosum is controversial. As the condition is benign and typically resolves spontaneously, treatment is usually not necessary. Advocates of treatment state that intervention speeds resolution, reduces self inoculation and symptoms, limits spread, and prevents scarring. Often there is pressure from parents to treat their otherwise healthy children because of the stigma of visible lesions.[7]

Summary points

Molluscum contagiosum is a common, self limiting condition

Topical salicylic acid should be regarded as first line treatment for cutaneous viral warts

Mild impetigo is effectively treated with topical mupirocin or fusidic acid for seven days

Oral antibiotics should be reserved for recalcitrant, extensive impetigo with systemic symptoms

Tinea capitis should be considered in every child with a scaly scalp as the infection is common and the presentation diverse

Tinea capitis should be confirmed by mycological analysis before an eight week course of griseofulvin is started

Treatment

Many treatments for molluscum contagiosum have been reported, including physical destruction or manual extrusion of the lesions, cryotherapy, and curettage. Treatments are painful, and there is limited evidence that they are more effective than watchful waiting. One study found no difference in resolution of lesions after extrusion of the umbilicated core compared with destruction of the lesion using phenol, although treatment with phenol produced notably more scarring.[6] Acidified nitrite cream has been reported as effective and painless.[8] Topical imiquimod cream may be useful in widespread or recalcitrant mollusca, but it has not been tested in controlled trials.[9]

A Cochrane review is under way to evaluate treatments for molluscum contagiosum. Until there is clear evidence of safety and efficacy of active intervention, we recommend watchful waiting and reassurance of patients and parents.

Viral warts

Cutaneous viral warts are discrete benign epithelial proliferations caused by the human papillomavirus. Several types occur (box 1).

Viral warts are common.[10] Prevalence increases during childhood, peaks in adolescence, and declines

Department of Dermatology, Leicester Royal Infirmary, University Hospitals of Leicester NHS Trust, Leicester LE1 5WW

Michael J Sladden
specialist registrar

Graham A Johnston
consultant

Correspondence to: M J Sladden m.sladden@ doctors.org.uk

thereafter.[11] In healthy children, warts resolve spontaneously; 93% of children with warts at age 11 showed resolution by age 16.[12] Resolution can be preceded by the appearance of blackened thrombosed capillary loops. Warts may be widespread and persistent in patients who are immunocompromised. The clinical appearance of warts depends on their location. The hands and feet are most commonly affected (fig 2).

Treatment
Although most warts resolve spontaneously within two years, some persist and become large and painful. For this reason many parents present their children for

Fig 1 Typical multiple dome shaped pearly or flesh coloured papules of mollluscum contagiosum, with a central depression (umbilication)

Fig 2 Cutaneous viral wart showing characteristic hyperkeratotic "warty" surface with capillary loops (black dots)

medical treatment. Treatment in children should be simple, cheap, effective, safe, and relatively painless.

Salicylic acid
Topical salicylic acid has been shown to be beneficial in treating viral warts. Data pooled from six randomised trials gave a cure rate of 75% in cases compared with 48% in controls (odds ratio 3.91, 95% confidence interval 2.40 to 6.36).[11 13] Preparations containing salicylic acid include creams, ointments, paints, gels, and colloids, with concentrations of the active ingredient varying from 11% to 50%. Salicylic acid breaks down hyperkeratotic skin but does irritate children's skin. Topical salicylic acid should be regarded as first line treatment.

Cryotherapy
Systematic reviews show that cryotherapy is no better than topical salicylic acid.[11 13] Cryotherapy is best avoided in young children, as parents consider the side effects of pain, swelling, and blistering excessive for a benign self limiting condition. Aggressive cryotherapy scars children's skin.

Other treatments
Although silver nitrate pencils and glutaraldehyde and formaldehyde preparations are licensed in the United Kingdom for treating warts, there is currently insufficient evidence of their benefit. Intralesional bleomycin, topical immunotherapy, photodynamic therapy, and pulsed dye laser treatment are best confined to research centres or resistant cases.

Impetigo

Cutaneous staphylococcal and streptococcal infections are important in children. They cause a wide spectrum of illness depending on the site of infection, the organism, and the host's immunity.[14] Impetigo is a superficial skin infection characterised by golden crusts (fig 3). It is caused by *Staphylococcus aureus* or *Streptococcus pyogenes*.

Impetigo is the third most common skin disease in children, after dermatitis and viral warts, with a peak incidence at 2-6 years of age.[15 16] Lesions are highly contagious and can spread rapidly by direct contact, through a family, nursery, or class.[17] The condition is more common in children with atopic dermatitis, in those living in tropical climates, and in conditions of overcrowding and poor hygiene. Nasal carriage of organisms may predispose to recurrent infection in an individual.

Impetigo can occur either as a primary infection or secondary to another condition, such as atopic dermatitis or scabies, which disrupts the skin barrier. It can be classified clinically as impetigo contagiosa (non-bullous impetigo) or bullous impetigo. Impetigo contagiosa is caused by *S aureus* or *S pyogenes*. Bullous impetigo is invariably caused by toxin-producing *S aureus*.

Impetigo contagiosa
Impetigo contagiosa is the most common form of impetigo. Lesions begin as vesicles or pustules that rapidly evolve into gold-crusted plaques, often 2 cm in diameter. They usually affect the face and extremities and heal without scarring. Constitutional symptoms are absent. Satellite lesions may occur due to autoinoculation.

Fig 3 Impetigo, showing classic golden coloured crust

Bullous impetigo

Bullous impetigo is characterised by flaccid, fluid filled vesicles and blisters (bullae). These are painful, spread rapidly, and produce systemic symptoms. Lesions are often multiple, particularly around the oronasal orifices, and grouped in body folds. To confirm the diagnosis and to target treatment, Gram's stain, culture, and sensitivity testing should be carried out on the exudate from lesions.

Treatment

Treatments for impetigo include topical and systemic antibiotics and topical antiseptics.[18] Good evidence shows that topical mupirocin and fusidic acid are safe and effective treatments for mild impetigo.[18] In mild cases they are probably as effective as oral antibiotics.[18] To minimise the development of resistant organisms, use topical antibiotics that are available in cream form only, which are not available as systemic preparations.

Oral antibiotics

Oral antibiotics may be better than topical preparations for more serious or extensive disease; they are easier to use but have more side effects than topical agents. Flucloxacillin is considered the treatment of choice for impetigo.[19] Macrolides, cephalosporins, and coamoxiclav are also reported to be effective, but evidence is limited because the studies have not been performed.[18] Selection of systemic antibiotic is determined by factors such as local epidemiology of resistance, patients' allergy or intolerance, and proved bacterial sensitivity after microbiological assessment.

If oral antibiotics are needed, we recommend as first line treatment a seven day course of flucloxacillin. In cases of allergy to penicillin, erythromycin (or similar macrolide) is suitable, but in some patients this causes gastrointestinal disturbance, and resistance to erythromycin is increasing. For impetigo caused by erythromycin resistant organisms, cephalosporins such as cephalexin are effective, although 10% of patients who are sensitive to penicillin are also sensitive to cephalosporins. Coamoxiclav (amoxicillin and clavulanic acid) is effective in infections caused by β lactamase producing strains of *S aureus*. Bacteriological culture is important before changing to this drug.

Topical antiseptics

Although no clear evidence supports the role of topical antiseptics in impetigo, they do help to soften crusts and clear exudate in mild disease. In more severe cases they may be a useful adjunct to antibiotics.

We suggest using topical mupirocin or fusidic acid for seven days in clinically mild impetigo. Oral antibiotics should be reserved for recalcitrant, extensive, systemic disease.

Tinea capitis (scalp ringworm)

Tinea capitis (scalp ringworm) is a highly contagious infection of the scalp and hair caused by dermatophyte fungi. It occurs in all age groups, but predominately children. It is endemic in some of the poorest countries.[20] The commonest cause of tinea capitis worldwide is *Microsporum canis*.[21]

The epidemiology of tinea capitis in the United Kingdom has recently changed dramatically,[22] reflecting a similar trend in the United States 20 years ago.[23] In the United Kingdom it is becoming a major public health problem, and Afro-Caribbean children are particularly affected.[24] The predominant organism was *M canis*, but now *Trichophyton tonsurans* causes 90% of cases in the United Kingdom and the United States.[22] *T tonsurans* is an anthropophilic fungus, which spreads from person to person. The reason for this change is unclear, but hairdressing practices such as shaving the scalp, plaiting, and using hair oils may increase spread.[22]

Tinea capitis causes patchy alopecia, but specific clinical patterns can be varied. Six main patterns are recognised (box 2).[25]

Fig 4 Grey type tinea capitis, with inflammation, erythema, scaling, pustules, and crusting

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Box 2: Main patterns of tinea capitis, listed according to occurrence (commonest first)

- *Grey type*—circular patches of alopecia with marked scaling (fig 4)
- *Moth eaten*—patchy alopecia with generalised scale
- *Kerion*—boggy tumour studded with pustules; lymphadenopathy usually present (fig 5)
- *Black dot*—patches of alopecia with broken hairs stubs
- *Diffuse scale*—widespread scaling giving dandruff-like appearance
- *Pustular type*—alopecia with scattered pustules; lymphadenopathy usually present

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Fig 5 Kerion pattern of tinea capitis, showing large boggy nodules with superficial pustules and crusting

cacy in infections caused by *T tonsurans* and *M canis*.[26–28] The recommended dose in children is 10 mg/kg/day, although some authors advocate up to 25 mg/kg/day. Treatment is taken until clinical and mycological cure is documented, usually about eight weeks. Side effects include nausea and rashes (about 10%); griseofulvin is contraindicated in pregnancy.

Good evidence supports the use of terbinafine for treating tinea capitis caused by *T tonsurans*[26–28]; it may be less effective for *M canis*. The dose ranges between 3 and 6 mg/kg/day, given for four weeks. Side effects include gastrointestinal upset and rashes (about 5%). Itraconazole, fluconazole, and ketoconazole are reported to be effective in tinea capitis, but there is less supportive evidence.

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The differential diagnosis for tinea capitis includes seborrhoeic dermatitis, atopic dermatitis, psoriasis, alopecia areata, and alopecia folliculitis. Tinea capitis should be considered in every child with a scaly scalp because the infection is common and the presentation diverse. Only 7% of children receive appropriate treatment for tinea capitis before referral to dermatology practice.[25]

Treatment

If tinea capitis is suspected, specimens of hair and scale should be examined to confirm the diagnosis. The aim of treatment is to provide a quick clinical and mycological cure, with minimal adverse effects and spread of disease. This requires oral antifungal agents, although topical treatment may reduce the risk of transmission at the start of systemic therapy.

Griseofulvin is the only treatment for tinea capitis licensed in the United Kingdom. It has been the treatment of choice for 40 years, with good evidence of effi-

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Lesson of the week

Avulsion fracture of the ischial tuberosity in adolescents—an easily missed diagnosis

Sam Gidwani, Jakub Jagiello, Martin Bircher

Avulsion fracture of the ischial tuberosity is a rare injury in comparison with mid-substance tears of the hamstrings, and its diagnosis is often missed. Such fracture usually occurs between puberty and late adolescence in those who do a lot of sport—it is in puberty that the secondary ossification centre or apophysis appears and in late adolescence that it fuses. In other words, fracture occurs at a time when the apophysis is the weakest link in the chain of muscle, tendon, and bone. The cause is usually forcible contraction of the hamstrings, as in sports such as sprinting and hurdling.

In adolescent patients with a history of proximal hamstring injury and current ischial tenderness, a radiograph of the pelvis should be performed. This is to exclude the presence of an avulsion fracture, which may be substantially displaced.

A prompt diagnosis of a displaced avulsion fracture of the ischial tuberosity will enable early surgery where appropriate. This in turn will prevent the development of chronic pain on sitting and walking and an inability to return to sporting activities.

Case reports

Case 1
A 14 year old boy was sprinting during a 200 m race when he suddenly developed a severe pain posteriorly in the proximal part of his left thigh. He collapsed to the ground in pain and could not complete the race.

His general practitioner advised him to rest to allow the presumed hamstring injury to settle. Despite prolonged physiotherapy, he still had pain when he jogged. Two years after the injury, he was referred to a rheumatologist in case the pain was caused by sciatica. Radiography of the pelvis showed a non-union of the left ischial tuberosity, with marked displacement of the

apophyseal fragment (fig 1). A specialist orthopaedic opinion was urgently sought; clinical examination showed substantial wasting of the hamstrings accompanied by weakness and pain on resisted knee flexion.

Three years after the injury an operation was done using the posterior Kocher-Langenbeck approach. The non-union was mobilised, reduced, and internally fixed using a reconstruction plate and screws. Two months later, he was comfortable apart from some pain on sitting. At five months postoperatively he was walking normally and was able to jog, and at one year he had returned to full sporting activities, including sprinting. Pelvic radiography showed bony union (fig 2).

Case 2
During a football game, a 15 year old boy stretched to kick the ball with his left foot. He instantly felt a severe pain in his left buttock and had to be carried off the pitch. He was told he had "pulled a muscle," and

Fig 1 Case 1: Anteroposterior pelvic radiograph, two years after injury, showing mobile non-union of left ischial tuberosity

Perform pelvic radiography in adolescents with history of proximal hamstring injury and current ischial tenderness

Department of Orthopaedics and Trauma, St George's Hospital, London SW17 0QT
Sam Gidwani *specialist registrar*
Jakub Jagiello *senior house officer*
Martin Bircher *consultant*

Correspondence to: S Gidwani, 122 Durham Road, London SW20 0DG samgidwani@yahoo.com

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