

Diagnosis and management of scalp ringworm

L C Fuller, F J Child, G Midgley, E M Higgins

Scalp ringworm is reaching epidemic proportions in parts of Britain's cities. Prompt diagnosis is needed to stop it spreading from child to child, but the varied presentation makes it easy to miss

Scalp ringworm, or tinea capitis, largely disappeared in Great Britain after oral griseofulvin was introduced in the late 1950s.¹ Over the past few years, however, dermatology departments in London, Bristol, and Birmingham have seen a large increase in cases, with rates of positive scalp isolates up to 20 times higher than previous baseline rates.²⁻⁴ A community point prevalence study from London suggested a disease prevalence of about 2.5% with a carriage rate of between 12% and 47% among schoolchildren.⁵ AfroCaribbean children seem to be particularly vulnerable to infection.³ Scalp ringworm is also increasing in Europe and North America.^{6,7} We discuss the current epidemic, illustrate the clinical presentations of the disease, and describe the methods for diagnosis and management.

Methods

We based this review on our extensive clinical experience in managing patients in a dedicated tinea capitis clinic at a teaching hospital. It also draws on information derived from a study assessing the impact of the problem on the local community and a detailed review of English language publications.

Source of infection

Scalp ringworm is caused by the dermatophyte group of fungi. The fungi are classified into three groups according to where they are normally found. Geophilic organisms live in soil, zoophilic organisms on animals, and anthropophilic organisms on humans. Most cases of childhood ringworm in the past 20 years have been due to the zoophilic organisms *Microsporum canis* (after exposure to an infected puppy or kitten) or *Trichophyton verrucosum* (from cattle). Recently, however, the predominant organism has changed to an anthropophilic one, *T tonsurans*, which spreads directly from child to child at home, school, or the hairdresser. This organism is now responsible for most scalp ringworm in Britain's larger cities.^{3,5} The reason for the change is unclear, but the United Kingdom seems to be repeating a trend observed in the United States 20 years ago.⁸

Clinical appearances of scalp ringworm

Clinical diagnosis of scalp infection with *T tonsurans* can be difficult as presentations are wide ranging and

Summary points

Scalp ringworm is common among innercity children in the United Kingdom

Diagnosis is difficult because of the wide range of clinical presentations

Systemic therapy is required to clear scalp ringworm

The diagnosis should be confirmed by mycological analysis before starting treatment

Antifungal shampoos may reduce the risk of transmission

King's College Hospital, London SE5 9RS

L C Fuller
consultant dermatologist

F J Child
consultant dermatologist

E M Higgins
consultant dermatologist

Medical Mycology Department, St John's Institute of Dermatology, St Thomas's Hospital, London SE1 7EH

G Midgley
medical mycologist

Correspondence to: L C Fuller
claire.fuller@kcl.ac.uk

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variable. It is thus easy for inexperienced clinicians to overlook the diagnosis.⁹ The presenting features can be divided into six main patterns.

Grey type—Circular patches of alopecia with marked scaling (fig 1a).

Black dot—Swollen stubs of broken off hairs are visible within the patch of alopecia (fig 1b). The dermatophytes invade the inside of the hair shaft, making it fragile and vulnerable to fracture.

Kerion—Boggy, localised swelling occurs due to an aggressive inflammatory response to the organism (fig 1c). Patients often also have cervical lymphadenopathy (fig 2).

Diffuse scale—This form looks like dandruff, with widespread scale throughout scalp that can be masked with hair oils.

Moth eaten—Hair loss is patchy, and the underlying scalp may be generally scaly.

Diffuse pustular pattern—Widespread scattered pustules are seen on the scalp (fig 1d). The inflammatory response is brisk but there is only scanty growth of organisms. There may be associated painful lymphadenopathy.

The pustular type is most difficult to diagnose. Pus usually occurs in bacterial infections, and diffuse pustular tinea capitis is often incorrectly treated with antibiotics. Although there may also be bacterial colonisation, the main pathogen is the dermatophyte.

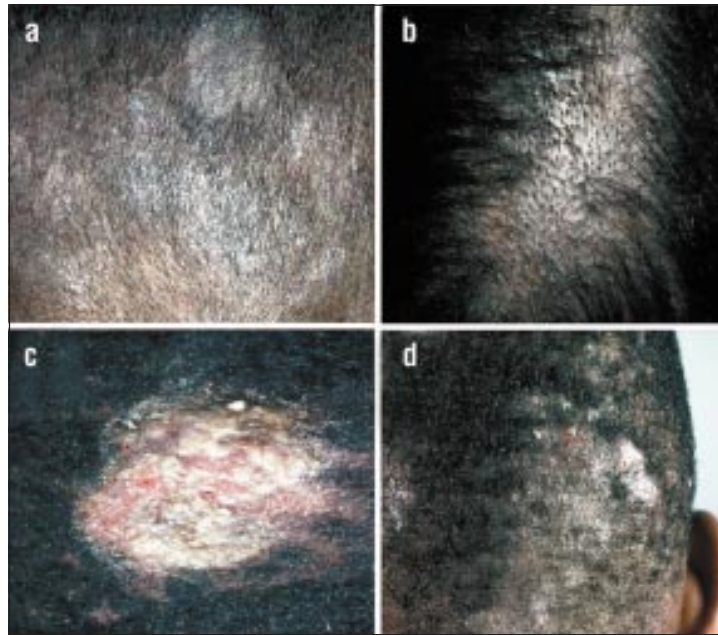


Fig 1 Four types of scalp ringworm: (a) grey type, (b) black dot, (c) kerion, and (d) diffuse pustules

Confirming the diagnosis

The Wood's light, which was traditionally used to diagnose scalp ringworm, can detect infection with only *Microsporum* sp. The greenish fluorescence seen under the light is due to an ectothrix infection of hairs, in which fungal spores form a sheath on the outside of the hair. *T tonsurans* causes an endothrix infection with the spores inside the hair shaft, and there is no fluorescence.

Diagnosis therefore relies on mycological analysis of scalp scale and broken off infected hairs. Samples for analysis can be obtained by scraping the affected area with the blunt side of a scalpel (to avoid slicing through the infected hairs) on to a piece of paper. This technique requires some practice.

Sampling using a soft plastic brush has been shown to be equally effective.¹⁰ The friction from massaging the bristles around the affected areas makes the bristles negatively charged so that they pick up hairs and scalp scales. The brush is then sent to the laboratory and used to inoculate an agar plate. Travel toothbrushes are cheap and ideal for this. If possible, both techniques should be used so that samples can be analysed by both microscopy and culture.¹¹

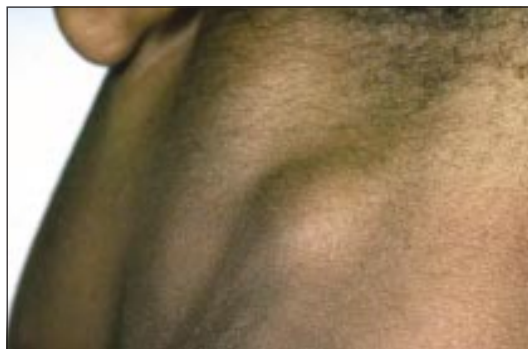


Fig 2 Painful occipital lymphadenopathy associated with kerion scalp ringworm

Management

Tinea capitis requires systemic treatment because antifungal creams are unable to penetrate the hair shaft sufficiently to clear the infection.¹² Furthermore, the use of topical antifungal treatment alone may contribute to the creation of carriers. The concept of carriers is controversial but describes patients whose symptoms and clinical signs are minimal but who are still mycologically positive and presumed capable of transmitting infection.

The only licensed treatment is oral griseofulvin. It is usually given at a dose of 10 mg/kg for six to eight weeks. However, *T tonsurans* seems resistant in some cases, and longer treatment may be required at doses of up to 20-25 mg/kg.¹² Because treatment takes a long time, it is essential to have a positive mycology result before starting. However, patients with the more aggressive clinical variants, pustular tinea capitis and kerion, should use antifungal creams or shampoos to reduce the risk of progression while waiting for the mycology results.¹³ The results of microscopy can take a week and culture up to four weeks, and these variants can be painful.

The newer fungicidal drugs such as oral terbinafine and itraconazole are effective in tinea capitis.^{14 15} Because of their fungicidal action, they require shorter treatments than griseofulvin. However, these drugs are unlicensed for use in children in the United Kingdom.

Patients sometimes develop papules after starting treatment, particularly around the ears and face (fig 3). This is an identity reaction and not an adverse drug reaction. An identity reaction is one that occurs away from the primary lesion and is usually due to an immunological reaction to the causative agent—in this case, the dermatophyte. The treatment should not be stopped.

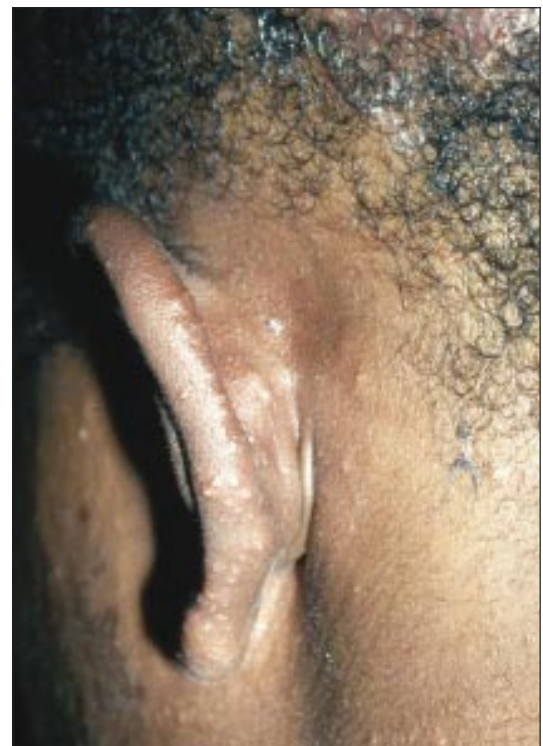


Fig 3 Identity reaction showing papules around the ears

Additional educational resources

British Association of Dermatology (www.bad.org.uk)
Contains clinical guidelines for management
The Skin Site (www.skinsite.com)
The site contains information sheets for patients

Adjuvant therapy

A small randomised trial found that antifungal shampoo (selenium sulphide) increases the rate of eradication, which may reduce the transmissibility of the organism.¹³ Our experience supports this, and we recommend that children use topical treatments as well as oral drugs.^{15 16} It is not clear at what stage during treatment a child is free from spores. However, current advice states that once children are receiving adequate treatment (oral and topical) it is safe for them to return to school.¹⁶

Conclusion

Scalp ringworm seems to be increasing in the United Kingdom and is reaching epidemic proportions in some areas. *T tonsurans* is responsible for most of these cases. Doctors should consider scalp ringworm in any child with a scaly scalp in whom a diagnosis of dandruff or scalp eczema has been suspected.¹⁶

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Lesson of the week**Interfering antibodies affecting immunoassays in woman with pet rabbits**

Adrian Park, Mark Edwards, Mandy Donaldson, Mohammad Ghatei, Karim Meeran

Many antibodies used in diagnostic immunoassays are derived from rabbits. Keeping rabbits as pets is known to be a risk factor for developing heterophilic (or interfering) antibodies.¹ Studies have shown that 30-40% of the population have heterophilic antibodies.² However, only about 0.05-0.5% of immunoassays seem to be affected to the extent that the concentration of interfering antibodies overwhelms the assay system.² We report a case in which the presence of heterophilic antibodies led to unnecessary investigations.

Case history

A 52 year old woman was referred to our hospital in July 2001 for further investigation of persistently raised fasting gut hormones concentrations. She had had irritable bowel syndrome diagnosed 16 years previously. The high concentrations of gut hormones had first been detected nine years ago, when, after an exacerbation of her condition, she had investigations to screen for other possible causes of diarrhoea. Computed tomography of



People who keep rabbits may develop heterophilic antibodies

Interfering antibodies must be considered when the clinical picture and immunoassay results do not match

Correspondence to:
K Meeran
kmeeran@ic.ac.uk
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the abdomen, magnetic resonance imaging of the pancreas, and an octreotide scan at that time all gave normal results. The referring hospital attributed the abnormal blood test results to hyperplasia of pancreatic islet cells.