CME

Follow the link from the online version of this article to obtain certified continuing medical education credits

The diagnosis and management of tinea

Blaithin Moriarty, Roderick Hay, Rachael Morris-Jones

Dermatology Department, King's College Hospital, London SE5 9RS, UK
Correspondence to: R Morris-Jones themojos@doctors.org.uk

Cite this as: *BMJ* 2012;345:e4380
doi: 10.1136/bmj.e4380

bmj.com
Previous articles in this series
- Perioperative management of patients taking treatment for chronic pain (*BMJ* 2012;345:e4148)
- Manifestation, diagnosis, and management of foodborne trematodiasis (*BMJ* 2012;344:e4093)
- Communicating risk (*BMJ* 2012;344:e3996)
- Prepregnancy care (*BMJ* 2012;344:e3467)
- Ventilator associated pneumonia (*BMJ* 2012;344:e3325)

Tinea refers to superficial infection with one of three fungal genera—*Microsporum*, *Epidermophyton*, and *Trichophyton*—collectively known as dermatophytes. These infections are among the most common diseases worldwide and cause serious chronic morbidity. Griseofulvin treatment and school screening programmes almost eradicated tinea capitis (scalp infection) as an endemic condition in the developed world in the 1950s, but it re-emerged as a public health problem in the United Kingdom in the 1990s, with infection rates of at least 12% in school children.[1] Increased mass tourism and mobile populations may have contributed to the changing epidemiological trends.[2] Newly developed polymerase chain reaction based techniques, although useful in rapid diagnosis of dermatophytosis,[3] are still not widely available. Although the number and range of antifungal drugs are limited compared with antibiotics, most are highly effective for fungal disease acquired in temperate climates. This review aims to familiarise the reader with the various clinical presentations of tinea, to outline the steps that should be taken for accurate diagnosis, and to evaluate the most appropriate treatment regimens.

SOURCES AND SELECTION CRITERIA

We based this review on a detailed review of English language publications. We also drew on the British Association of Dermatologists' clinical guidelines for the management of tinea capitis and the management of onychomycosis, Health Protection Agency guidelines, and extensive clinical experience.

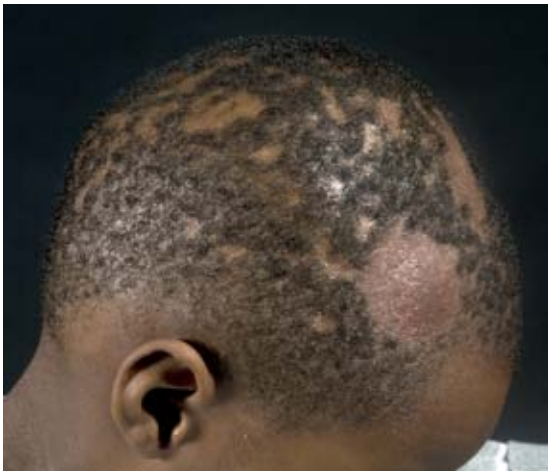

Fig 1 | Tinea capitis. Two of this boy's siblings developed patchy hair loss over three months, whereas three did not. All five children were positive for *Trichophyton tonsurans* on mycological culture from brushings. All were treated simultaneously with oral terbinafine for one month and had complete hair regrowth. Repeat brushings after treatment showed no evidence of ongoing infection

What is tinea?

All three genera of dermatophytes grow in keratinised environments such as hair, skin, and nails.[4] Anthropophilic dermatophytes are restricted to human hosts and produce mild chronic inflammation. The main reservoirs of zoophilic dermatophytes are pets, livestock, and horses; infection with such organisms usually causes marked inflammatory reactions in humans. Geophilic dermatophytes, from soil, only occasionally infect humans and animals.

What are the most common types of dermatophytes?

T rubrum, which takes its name from the underside of cultured colonies being red, is the most common anthropophilic dermatophyte.[5 6] It is the main pathogen implicated in the increasing global incidence of onychomycosis, tinea corporis, tinea cruris, tinea manuum, and tinea pedis in the UK, Europe, and the United States.[7 8]

Infection with *T interdigitale* is a common cause of tinea pedis worldwide, with higher rates seen in some regions such as the Far East[9]; this species is less common at other sites. *T tonsurans* has been the main cause of tinea capitis in London since the mid-1990s,[1 10 11] and it continues to increase in incidence in the US and elsewhere.[8] It has an endothrix pattern of infection—the fungus penetrates into the hair shaft so systemic treatment is necessary. *E floccosum* is the only *Epidermophyton* species known to be pathogenic in humans.[4] It causes a small number of cases of tinea pedis and tinea cruris.[2 6] *M canis* is a zoophilic organism that normally affects household pets.[12] Infections caused by *M canis*, such as tinea capitis, may be highly inflamed and are often accompanied by erythema and pustules. The incidence of this infection is rising in central and southern Europe; it also occurs in the UK.

SUMMARY POINTS

- Tinea is increasingly a problem worldwide
- Tinea may affect any body site and is named according to the anatomical area infected
- Pathogenic organisms have a variable geographical distribution
- Mycology samples should be taken before and after appropriate treatment

Tinea capitis

Dermatophyte infection of the scalp is known as tinea capitis (fig 1). Tinea capitis is almost exclusively a disease of childhood, and current evidence suggests that it occurs more often in children of African or Caribbean extraction.[10 13] It is largely caused by *T tonsurans* in cities in the UK,[1 10 12] by *M canis* in Europe and rural UK,[14] and by *T violaceum* in east Africa and the Indian subcontinent.[11 15 16] Clinical signs may be subtle, with mild flaking of the scalp alone; however, broken-off hairs ("black dot" ringworm), patches of frank alopecia, pustules, large

Box 1 | Presentations of tinea capitis

Diffuse scale: Similar in appearance to dandruff; may initially be masked with hair oils

Grey patches with alopecia: Circular patches with marked scaling may be seen

Black dots: Sites of broken-off hairs within patches of alopecia; seen mainly in endothrix infection

Kerion: Often presents emergently with boggy, localised swelling. It is caused by an aggressive inflammatory response to the organism and may be accompanied by cervical lymphadenopathy

Diffuse pustular lesions: Widespread scattered pustules; may have scanty organisms

inflammatory swellings (kerion), and tender occipital lymphadenopathy may also occur. Five distinct patterns of clinical infection are described (box 1).[17] Infection is often asymptomatic and undiagnosed, with consequent spread to close contacts, especially siblings, who should be screened.[18] *T tonsurans* may be isolated from the personal belongings of children with tinea capitis,[19] and sterilisation of fomites, such as hairbrushes and combs, is recommended.[20] Adults rarely have tinea capitis,[20] although they may develop tinea corporis (of the head or neck) through spread from a child's scalp. Exclusion of infected children from school is no longer advised.[20 21] Disseminated eczema-like ("id") reactions occasionally occur with inflammatory tinea, usually at the start of systemic antifungal treatment.

Tinea unguium

Tinea unguium, a dermatophyte infection of the nails, is also known as onychomycosis (fig 2). In general nail plates are discoloured and may be thickened, brittle, or distorted and show onycholysis (lifting of the nail plate). Five major presentations of tinea unguium are currently recognised[22]: distal and lateral subungual, proximal subungual, superficial white, endonyx, and total dystrophic onychomycosis, although a recent review has proposed refining this classification to help focus treatment according to species.[23] The prevalence of onychomycosis is at least 12.4% in Europe,[24] and the disease is often atypical and aggressive in patients with untreated HIV infection.[25 26] Onychomycosis of the toenails is more common than disease of the fingernails. Patients with psoriasis have an increased risk of onychomycosis because the abnormal psoriatic nail provides a portal for fungal entry.[w1] Because the two conditions may be clinically similar, laboratory confirmation is necessary before the start of oral treatment and should be repeated at the end of treatment. Failure to do this can result in unnecessary or unnecessarily long treatment regimens, because psoriatic nail dystrophy does not respond to antifungals.[w2] HIV may predispose to more frequent and severe infection and failure to respond to conventional doses of antifungals.[w3 w4]

Tinea pedis

Tinea pedis (fig 3), dermatophytosis of the feet, commonly presents with scaling and maceration of the most lateral interdigital spaces extending medially. In addition, hyperkeratosis of the plantar and lateral aspect of the foot may be seen in

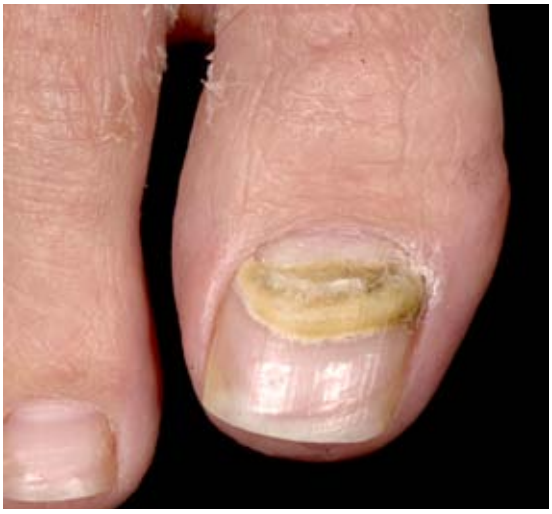

Fig 2 | Tinea unguium, also known as onychomycosis. This patient presented with an 18 month history of worsening abnormality of her right great toenail. On review she was noted to have tinea pedis of the interdigital spaces of both feet

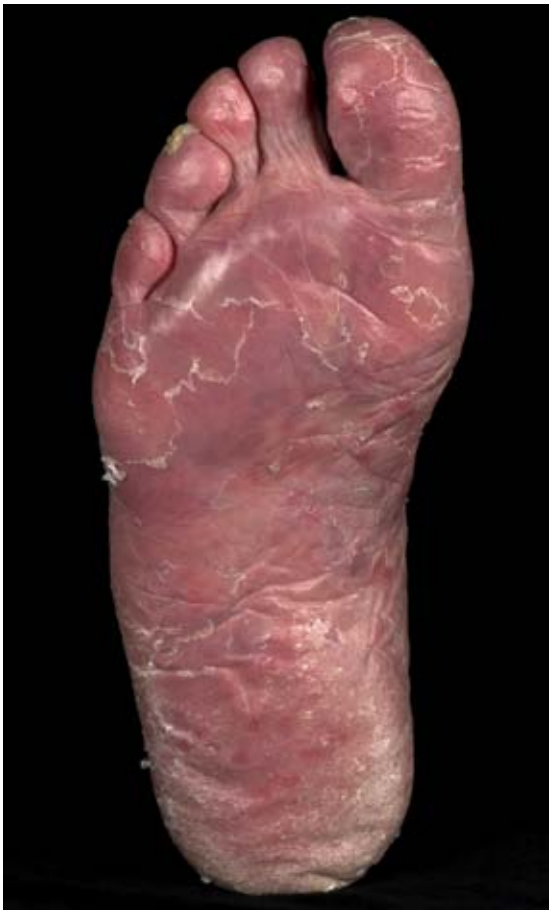

Fig 3 | Tinea pedis in a patient who had initially been suspected to have psoriasis when she developed scaling on both plantar surfaces and the palm of her right hand. On review she was also noted to have nail dystrophy. *Trichophyton rubrum* grew on mycological culture from scrapings, although clippings were negative on two occasions. She declined to take a prolonged course of terbinafine so she received pulsed itraconazole for three months, which led to clearance of both the skin and nail abnormalities

the so called moccasin or dry-type pattern of infection. A less common presentation is with small vesicles and blisters on an erythematous base on the plantar surface of the feet, and these lesions look similar clinically to pompholyx eczema.[w5] The prevalence of tinea pedis is high,[24] particularly in people who wear occlusive footwear, such as athletes.[w6]

Tinea corporis and tinea cruris

Tinea corporis and tinea cruris refer to dermatophytosis of the trunk and groin, respectively. Both present with a pruritic erythematous rash with an active scaly palpable edge within which pustules or vesicles may be seen (fig 4). The infection spreads centrifugally and results in annular patches of varying sizes, usually asymmetrical in distribution. Tinea corporis is seen most commonly in children and young adults, whereas tinea cruris is more common in adult men.[6]

Tinea manuum

Although dermatophytosis of the hand is usually unilateral, it can be bilateral (fig 5). Infection of the dorsum of the hand is similar to tinea corporis in morphology, although infected palmar surfaces usually have a dry scaling appearance. An entity known as "one hand, two foot syndrome" describes tinea in this asymmetrical distribution, with the dominant hand being more commonly affected.

Tinea faciei and tinea barbae

Tinea faciei refers to dermatophytosis anywhere on the face, whereas tinea barbae refers specifically to infection within the beard area. Both entities occur in postpubertal boys and men in a pattern similar to that seen for tinea capitis.[w7] Zoophilic organisms such as *T verrucosum* or *T mentagrophytes*, the most common pathogens implicated, produce an inflammatory pustular eruption with crusting or kerion formation; *T tonsurans* is an increasingly common cause of a similar problem in wrestlers. The term tinea faciei encompasses both this presentation and the classic annular scaly eruption.

Fig 4 | Tinea corporis and tinea cruris in a teenager. He developed a rash similar to that found on his pet dog by the family vet. *Microsporum canis* was cultured from scrapings. Four weeks of treatment with oral terbinafine led to complete resolution of the eruption

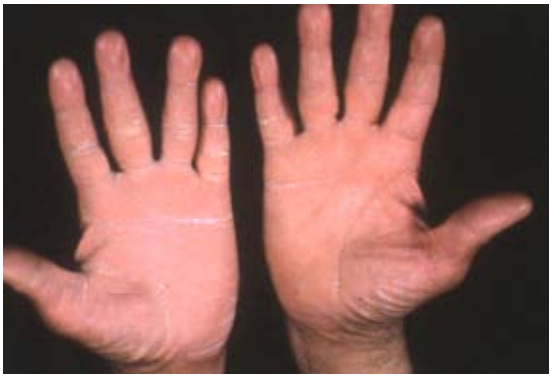

Fig 5 | Tinea manuum caused by *Microsporum canis*. The patient presented with a six month history of itchy hands and the diagnosis was confirmed by culture of scrapings. The symptoms cleared after four weeks of treatment with topical terbinafine twice daily

Tinea incognito

Superficial cutaneous dermatophytosis can be difficult to distinguish from other inflammatory dermatoses listed in table 2. Topical steroids may alter the morphology of tinea, with flattening of the edge and loss of surface scale (fig 6), making the clinical diagnosis difficult (hence the term tinea incognito).[w8] A large retrospective study of tinea incognito in children found that the face or trunk were most commonly affected and that *T mentagrophytes* was the most common organism.[w9]

How is tinea diagnosed?

History

Take a careful history to estimate the duration of symptoms, previous treatments, and affected contacts. Look for a history of exposure to pets—such as dogs, cats, or hamsters—with rashes.

Examination

Perform a full skin examination focusing on the affected sites. Tinea infections are often asymmetrical in distribution. Morphology is usually scaly with erythema, although the characteristic scaly edge is masked in moist flexures. Differing presentations are seen in the scalp, including a diffuse scaling, black dot ringworm (where hairs break close to the scalp skin), alopecia areata-like hair loss, and kerion. Spread of scaly patches to the upper trunk and limbs may be seen in children with tinea capitis as well as occipital or cervical lymphadenopathy. The toenails and interdigital toeweb spaces are often infected in patients who have tinea at other sites, such as the groin, and should be examined.

Diagnostic tools: Wood's light

Wood's light is a lamp filtered by Wood's glass (barium silicate containing 9% nickel oxide that transmits rays of a wavelength >365 nm) which, when directed on tinea

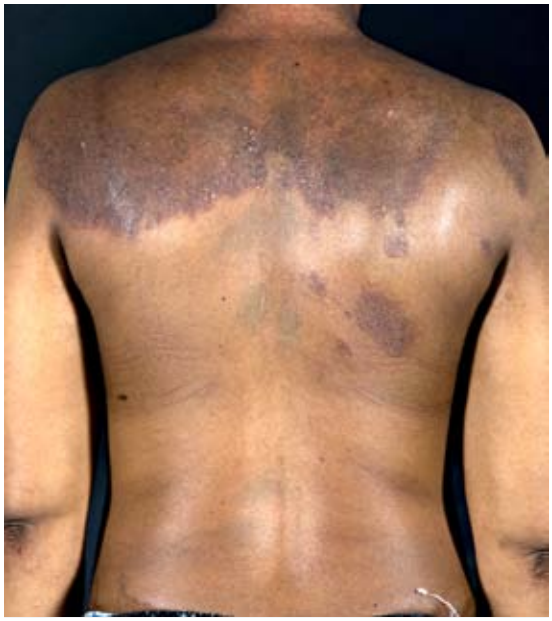

Fig 6 | Tinea incognito in a woman who had been treated for one year with a potent topical steroid for a presumed diagnosis of new atopic eczema. Culture of scrapings confirmed the presence of *Trichophyton rubrum*, and the eruption settled with a four week course of oral terbinafine

Table 1 | Samples taken for different types of tinea

Site	Sample types
Tinea barbae	Swabs, scrapings, brushings
Tinea capitis	Brushings if scaly, swabs if a kerion or pustules
Tinea corporis	Scrapings, swabs if pustules
Tinea cruris	Swabs, scrapings if scaly
Tinea incognito	Scrapings often negative
Tinea manuum	Scrapings, swabs
Tinea pedis	Swabs, scrapings
Tinea unguium (onychomycosis)	Clippings

A PARENT'S PERSPECTIVE

The past seven months have been an emotional rollercoaster for our whole family. One minute our vivacious 2 year old son was running around, then—within the space of a few weeks—his scalp condition deteriorated from dry and flaky to inflamed and oozing (fig 7), and eventually he ended up on a drip in a hospital bed. He developed a large crusty scab, his hair fell out, and he was left with a very sore red scalp, which looked like a burn. We endured emotional turmoil with "looks" from adults who moved their children away saying "eeugh what's on that boys head?" It has been difficult to deal with the alopecia and not knowing whether his hair would regrow.

Despite many frustrations along the way, he has finally been referred to a dermatologist who clearly knows about his condition, which is a fungal kerion. We are relieved that he is now on the correct treatment and on the road to recovery. We have had a daily battle to get him take his drugs, which taste "yucky." The emotional impact on our son and whole family has been immense and is ongoing.

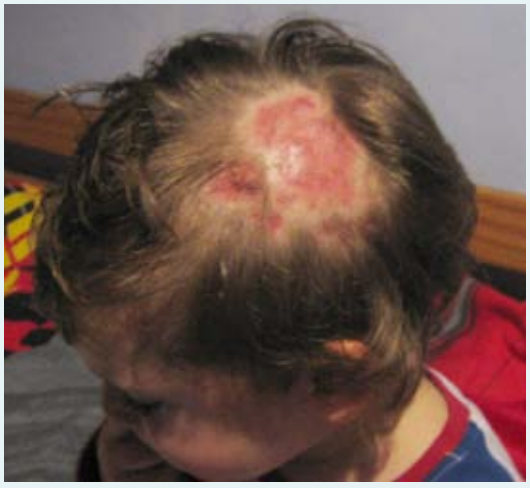

Fig 7 | Fungal kerion in a 2 year old boy

capitis caused by *Microsporum* spp in a darkened room, causes hairs to fluoresce bright green.

How are samples taken for mycological analysis?

Scalp brushings

An excellent way to collect scaly material is with a disposable travel toothbrush that is brushed briskly over visibly affected areas of the scalp. The brush is then sent for mycological culture. Alternatives include dampened swabs or scrapings (table 1).

Scrapings

A blunt instrument such as the back of a scalpel blade is useful for obtaining scales from the active edge of a suspected tinea infection. The material collected should be transported in dark paper to the mycology laboratory for microscopy and culture. Do not use plastic containers, such as sterile specimen jars, because the scale sticks to the plastic (owing to static electricity).

Nail clippings

When nail infection is suspected, include nail clippings and subungual debris in the sample (usually transported in dark folded paper) for microscopy and culture. Because the yield is unpredictable take as much material as possible.

Are swabs useful in tinea?

Swabs for mycology culture are particularly useful when sampling fungal kerions and in pustular or macerated infections.

Interpreting results of traditional mycological tests

Direct microscopy can be performed immediately, and most laboratories report the presence or absence of fungal hyphae or yeasts within 24 hours. Because scalp brushings are usually transported on a brush, microscopy cannot be performed. Regardless of the collection method and microscopy result all samples are cultured on agar for identification, which takes at least two weeks. A negative culture result cannot be confirmed until plates have been incubated for six weeks. Treatment of clinically obvious or severe cases should not be delayed for culture results, although treatment may need to be altered according to the dermatophyte grown. The presence or absence of fungal elements on microscopy is not always predictive of positive culture

Table 2 | Diagnosis and treatment of tinea

Diagnosis	Infectious agents	Clinical manifestations	Differential diagnosis	Treatment
Tinea barbae	*Trichophyton verrucosum*, *T mentagrophytes*	Infection of the beard area in adult men; usually inflammatory and pustular with crusting	Effects of shaving, folliculitis, *Staphylococcus aureus* infection, impetigo	Oral terbinafine 250 mg daily for 4 weeks; itraconazole 200 mg daily for 2 weeks
Tinea capitis	*T tonsurans*, *Microsporum canis*, *M audouinii*, *T verrucosum*, *T violaceum*, *T soudanense*	Infection of the scalp causing scalp scale, itch, pustules and less often kerion; may cause secondary alopecia; eyebrows and eyelashes may also be affected	Atopic eczema, seborrhoeic dermatitis, psoriasis, discoid lupus erythematosus	Oral terbinafine 250 mg daily for 4 weeks in adults; children (depends on weight) 62.5 mg daily if <20 kg, 125 mg daily if 20-40 kg, 250 mg daily if >40 kg (all for 4 weeks)
Tinea corporis	*T rubrum*, *M canis*, *T tonsurans*	Infection of the trunk; pruritic erythematous lesions with central clearing	Annular psoriasis, discoid eczema, annular erythema	Topical terbinafine 1% cream for 4-6 weeks; if extensive use oral terbinafine or itraconazole
Tinea cruris	*Epidermophyton floccosum*, *T rubrum*, *T interdigitale*	Infection of the groin and pubic area; pruritic erythematous lesions with central clearing	Bacterial or candidal intertrigo, erythrasma, psoriasis, seborrhoeic dermatitis	Topical terbinafine 1% cream for 2-4 weeks
Tinea incognito	Potentially any cutaneous dermatophytosis that has been inappropriately treated with topical steroid	Spreading erythema that has lost its raised palpable edge and looks more indistinct	Eczema, psoriasis, subacute cutaneous lupus erythematosus	Stop topical steroids and treat with topical terbinafine 1% cream for 6 weeks
Tinea manuum	*T rubrum*, *T mentagrophytes*	Infection of the hand; pruritic erythematous lesions with central clearing; often one hand (usually the dominant one) only	Psoriasis, eczema, contact dermatitis, keratoderma, keratolysis exfoliativa	Topical or systemic terbinafine for 2-4 weeks
Tinea pedis	*T rubrum*, *T interdigitale*, *E floccosum*	Infection of the feet involving interdigital spaces and, if chronic, soles of the feet; itchy, peeling, maceration, and fissuring of toe webs	Psoriasis, eczema, contact dermatitis, soft corns, candida, pitted keratolysis, keratoderma	Topical terbinafine 1% cream 2-6 weeks
Tinea unguium (onychomycosis)	*E floccosum*, *T interdigitale*, *T rubrum*, *T verrucosum*	Infection of the nail plate; often white or yellow lesions with irregular edges; some degree of subungual hyperkeratosis, which leads to nail detachment, is usually present	Psoriasis, eczema, lichen planus, trauma, candida, bacterial, genetic abnormality	Oral terbinafine 250 mg daily for 3-6 months; itraconazole 200 mg daily for 3 months or pulsed 200 mg twice daily 7 days/month for a total of 2-3 months
Tinea versicolor	*Malassezia* spp	Small scaly annular hypopigmented or hyperpigmented patches on trunk or neck; when treated macular discoloration can persist for months	Psoriasis, eczema, vitiligo	Itraconazole 200 mg daily for 1 week; ketoconazole 200 mg daily for 4 weeks

Box 2 | Reasons for treatment failure

- Poor adherence to treatment: For example, in the UK terbinafine is available only as a crushable tablet, which may be unpalatable for children
- Dermatophyte resistant to drug(s): Consider changing agents if resistance is suspected, but this is very rare
- Reinfection from close contacts: This should always be considered in persistent or recurrent cases and contacts should be screened
- Alternative diagnoses: Tinea may mimic a variety of other conditions
- Rare causes of foot infection: Travel or living abroad may lead to more exotic fungal species that are resistant to treatment being acquired

results, and if a clinician is faced with unexpectedly negative results, repeat the investigations while alternative diagnoses are considered. Collect as much material as possible from the brushings, scrapings, or clippings to ensure that samples are adequate for microscopy and culture; this increases the chance of positive mycology results.

New molecular techniques

Polymerase chain reaction based methods are emerging as useful investigations for the detection of dermatophytes, mainly in nail disease. Currently, however, these are available only occasionally as routine diagnostic procedures. One such commercially available technique, Onychodiag (BioAdvance, France), was designed to detect dermatophytes using a polymerase chain reaction enzyme linked immunosorbent assay in nail samples. In a multicentre study this had a sensitivity of 83.6% and a specificity of 100%.[w10]

How is tinea treated?

Treatment depends on the anatomical location of the infection and the pathogen (table 2).

Tinea capitis

Topical treatments are not effective in tinea capitis because the pathogenic fungi are located within the hair. A 2007 Cochrane review of systemic antifungal treatment for tinea capitis in children concluded that newer treatments, including terbinafine, have equivalent efficacy and safety profiles to griseofulvin and allow treatment to be shorter.[w11] A subsequent randomised controlled trial found that complete cure rates for *T tonsurans* infection were significantly higher for terbinafine than for griseofulvin[w12]; however, griseofulvin remains the only licensed treatment for children in the UK. Nonetheless, many dermatology units in the UK use terbinafine as first line treatment because it is highly effective, well tolerated, and adherence to treatment is high (box 2). Griseofulvin seems to be more effective in *Microsporum* infections,[w13] highlighting the importance of mycological identification in infected people. Similarly, oral itraconazole is effective in treating infections of the scalp. Oral terbinafine is the

TIPS FOR NON-SPECIALISTS

- Patients with tinea cruris should be examined for tinea pedis
- Patients with recurrent cellulitis of the lower leg may have tinea pedis as the portal of entry of bacteria
- Tinea capitis infections and onychomycosis rarely respond to topical treatment
- Investigate the family of a child with tinea capitis for simultaneous infection
- Children with tinea capitis usually have associated occipital lymphadenopathy

ADDITIONAL EDUCATIONAL RESOURCES

Resources for patients

DermNetNZ (www.dermnetnz.org/fungal/onychomycosis.html)—Patient information leaflet on fungal infections of the nails

British Association of Dermatologists (www.bad.org.uk//site/820/default.aspx)—Patient information leaflet on fungal infections of the nails

Resources for healthcare professionals

Higgins EM, Fuller LC, Smith CH. Guidelines for the management of tinea capitis. *Br J Dermatol* 2000;143:53-8. (www.bad.org.uk/Portals/_Bad/Guidelines/Clinical%20Guidelines/Tinea%20Capitis.pdf). Guidelines from the British Association of Dermatologists

Roberts DT, Taylor WD, Boyle J. Summary of guidelines for the management of onchomycosis. www.bad.org.uk/Portals/_Bad/Guidelines/BNF-sponsored%20summary%20guidelines/Onychomycosis%20Summary.pdf

treatment of choice for most kerions, and the addition of a topical steroid reduces inflammation and allows complete hair regrowth.[w14] Children presenting with a kerion may best be managed in secondary care to ensure rapid mycological diagnosis and adequate immediate systemic treatment; additional steroid preparations are indicated to ensure recovery without permanent alopecia.

Tinea unguium

Topical treatment alone is rarely effective in curing onychomycosis.[w15] However, for patients in whom systemic treatment is inappropriate because of comorbidities, or where a single nail is affected, amorolfine nail lacquer can be beneficial when used twice weekly for 6-12 months. Nail infections are treated with a minimum course of systemic terbinafine or itraconazole for six weeks for fingernails and three months for toenails, combined with topical drugs and surgical or chemical nail avulsion, as clinically indicated.[w16] The regimens are terbinafine 250 mg daily or itraconazole 400 mg daily for one week each month. If onychomycosis does not respond to prolonged oral antifungal treatment (at least three months), consider alternative diagnoses for abnormal nail plates including trauma, psoriasis, and lichen planus.

Skin infections

Topical treatment is valuable in managing tinea infections of the skin, although systemic treatment may be needed for more severe or extensive infections. There is no evidence that any specific topical agent is superior; all should be used for two to six weeks (table 2).[w17] The first line topical agent is usually terbinafine 1% cream twice daily for four to six weeks. Creams that contain an antifungal agent plus hydrocortisone are effective for inflamed infections. Tinea pedis and tinea cruris often relapse after successful treatment in susceptible people, who should be advised to keep the affected skin dry to prevent relapse. Advise patients to consider washing or replacing potentially contaminated socks, towels, and footwear (such as training shoes) after successful treatment.

When should referral to hospital be considered?

Fungal skin and hair infections should respond to treatment within a few weeks and nail infections within three months. If symptoms and signs persist beyond these times consider referral to secondary care. The main causes of apparent treatment failure are incorrect diagnosis and suboptimal treatment (table 2).

Contributors: All authors contributed equally to the preparation of this manuscript and helped revise the final draft. RM-J is guarantor.

Competing interests: None declared.

Provenance and peer review: Commissioned; externally peer reviewed.

Patient and parental consent obtained.

1 Hay RJ, Clayton YM, De Silva N, Midgley G, Rossor E. Tinea capitis in south-east London—a new pattern of infection with public health implications. *Br J Dermatol* 1996;135:955-95.
2 Havlickova B, Czaika VA, Friedrich M. Epidemiological trends in skin mycoses worldwide. *Mycoses* 2008;51(suppl 4):2-15.
3 Hay RJ, Jones RM. New molecular tools in the diagnosis of superficial fungal infections. *Clin Dermatol* 2010;28:190-6.
4 Weitzman I, Summerbell RC. The dermatophytes. Clinical microbiology reviews. *Clin Microbiol Rev* 1995;8:240.
5 Macura AB, Krzyściak P, Skóra M, Gniadek A. Dermatophyte species in superficial mycoses in the Kraków district, Poland in the years 1972-2007. *Mycoses* 2010;53:148-52.
6 Welsh O, Welsh E, Ocampo-Candiani J, Gomez M, Vera-Cabrera L. Dermatophytoses in Monterrey, *Mexico Mycoses* 2006;49:119-23.
7 Borman AM, Campbell CK, Fraser M, Johnson EM. Analysis of the dermatophyte species isolated in the British Isles between 1980 and 2005 and review of worldwide dermatophyte trends over the last three decades. *Med Mycol* 2007;45:131-41.
8 Foster KW, Ghannoum MA, Elewski BE. Epidemiologic surveillance of cutaneous fungal infection in the United States from 1999 to 2002. *J Am Dermatol* 2004;50:748-52.
9 Tan HH. Superficial fungal infections seen at the National Skin Centre, Singapore. *Nippon Ishinkin Gakkai Zasshi* 2005;46:77-80.
10 Fuller LC, Child FC, Higgins EM. Tinea capitis in south-east London: an outbreak of Trichophyton tonsurans infection. *Br J Dermatol* 1997;136:139.
11 Fuller LC, Child FC, Midgley G, Higgins EM. Scalp ringworm in south-east London and an analysis of a cohort of patients from a paediatric dermatology department. *Br J Dermatol* 2003;148:985-8.
12 Ginter-Hanselmayer G, Weger W, Ilkit M, Smolle J. Epidemiology of tinea capitis in Europe: current state and changing patterns. *Mycoses* 2007;50:6-13.
13 Bronson DM, Desai DR, Barsky S, Foley SM. An epidemic of infection with Trichophyton tonsurans revealed in a 20-year survey of fungal infection in Chicago. *J Am Acad Dermatol* 1983;8:322-9.
14 Kuklová I, Kucerová H. Dermatophytoses in Prague, Czech Republic, between 1987 and 1998. *Mycoses* 2001;44:493-6.
15 Woldeamanuel Y, Leekassa R, Chryssanthou E, Menghistu Y, Petrini B. Prevalence of tinea capitis in Ethiopian schoolchildren. *Mycoses* 2005;48:137-41.
16 Jahromi SB, Khaksar AA. Aetiological agents of tinea capitis in Tehran, Iran. *Mycoses* 2006;49:65-7.
17 Fuller LC, Child FJ, Midgley G, Higgins EM. Diagnosis and management of scalp ringworm. *BMJ* 2003;326:539-41.
18 White JM, Higgins EM, Fuller LC. Screening for asymptomatic carriage of Trichophyton tonsurans in household contacts of patients with tinea capitis: results of 209 patients from South London. *J Eur Acad Dermatol Venereol* 2007;21:1061-4.
19 Mackenzie DW. "Hairbrush diagnosis" in detection and eradication of non-fluorescent scalp ringworm. *BMJ* 1963;2:363-5.
20 Higgins EM, Fuller LC, Smith CH. Guidelines for the management of tinea capitis. *Br J Dermatol* 2000;143:53-8.
21 Honig PJ. Tinea capitis: recommendations for school attendance. *Pediatr Infect Dis J* 1999;18:211.
22 Faergemann J, Baran R. Epidemiology, clinical presentation and diagnosis of onychomycosis. *Br J Dermatol* 2003;149:1-4.
23 Hay RJ, Baran R. Onychomycosis: a proposed revision of the clinical classification. *J Am Acad Dermatol* 2011;65:1219-27.
24 Ameen M. Epidemiology of superficial fungal infections. *Clin Dermatol* 2010;28:197-201.
25 Gupta AK, Taborda P, Taborda V, Gilmour J, Rachlis A, Salit I, et al. Epidemiology and prevalence of onychomycosis in HIV-positive individuals. *Int J Dermatol* 2000;39:746-53.
26 Rodwell GEJ, Bayles CL, Towersey L, Aly R. The prevalence of dermatophyte infection in patients infected with human immunodeficiency virus. *Int J Dermatol* 2008;47:339-43.