

British Association of Dermatologists' guidelines for the management of tinea capitis 2014

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1.0 Purpose and scope

The overall objective of this guideline is to provide up-to-date, evidence-based recommendations for the management of tinea capitis. This document aims to update and expand on the previous guidelines by (i) offering an appraisal of all relevant literature since January 1999, focusing on any key developments; (ii) addressing important, practical clinical questions relating to the primary guideline objective, i.e. accurate diagnosis and identification of cases; suitable treatment to minimize duration of disease, discomfort and scarring; and limiting spread among other members of the community; (iii) providing guideline recommendations and, where appropriate, some health economic implications (tinea capitis is a common problem in resource-poor settings and therefore treatments that are more easily and cheaply available and applicable to these settings have been factored in); and (iv) discussing potential developments and future directions.

This guideline is presented as a detailed review with highlighted recommendations for practical use in the clinic (see Section Summary), in addition to the production of a patient information leaflet [available on the British Association of Dermatologists' (BAD) website, <http://www.bad.org.uk>].

2.0 Stakeholder involvement and peer review

The guideline development group consisted of consultant, specialist registrar and associate specialist dermatologists, and a mycologist. The draft document was circulated to the BAD membership, British Dermatological Nursing Group (BDNG) and Primary Care Dermatological Society (PCDS) for comments, and was peer reviewed by the Clinical Standards Unit of the BAD (made up of the Therapy & Guidelines Subcommittee) prior to publication.

3.0 Methodology

This set of guidelines has been developed using the BAD's recommended methodology¹ and with reference to the Appraisal of Guidelines for Research and Evaluation (AGREE)

instrument (www.agreetrust.org).² Recommendations were developed for implementation in the National Health Service (NHS) using a process of considered judgement based on the evidence. The PubMed, Medline and Embase databases were searched for meta-analyses, randomized and nonrandomized controlled clinical trials, case series, case reports and open studies involving tinea capitis published in the English language from January 1999 to March 2014; search terms and strategies are detailed in the Supporting Information (see Table S1). Working in pairs, the authors screened the identified titles, and those relevant for first-round inclusion were selected for further scrutiny. The abstracts for the shortlisted references were then reviewed and the full papers of relevant material were obtained; disagreements in the final selections were resolved by discussion among the entire development group. Additional relevant references were also isolated from citations in shortlisted and reviewed literature, as well as (independent) targeted searches carried out by the coauthors. The structure of the 2000 guidelines was then discussed and re-evaluated, and different coauthors were allocated separate subsections. Each coauthor then performed a detailed appraisal of the selected literature with discussions with the entire development group to resolve any issues, for example with the quality of evidence and making the appropriate recommendations. All subsections were subsequently collated and edited to produce the final guideline.

4.0 Limitations of the guideline

This document has been prepared on behalf of the BAD and is based on the best data available when the document was prepared. It is recognized that under certain conditions it may be necessary to deviate from the guidelines and that the results of future studies may require some of the recommendations herein to be changed. Failure to adhere to these guidelines should not necessarily be considered negligent, nor should adherence to these recommendations constitute a defence against a claim of negligence. Limiting the review to English language references was a pragmatic decision, but the authors recognize this may exclude some important information published in other languages.

5.0 Plans for guideline revision

The proposed revision for this set of recommendations is scheduled for 2019; where necessary, important interim changes will be updated on the BAD website.

6.0 Background

6.1 Definition

Tinea capitis is an infection of scalp hair follicles and the surrounding skin, caused by dermatophyte fungi, usually species in the genera *Microsporum* and *Trichophyton*.

6.2 Epidemiology and aetiology

Tinea capitis continues to be predominantly a disorder of prepubertal children, common in inner-city cosmopolitan communities, with no sign of a reduction in incidence. Although across Europe *Microsporum canis* remains the most commonly involved organism, in the U.K., a shift towards anthropophilic species continues to be observed.³ *Trichophyton tonsurans* is reported to account for 50–90% of dermatophyte scalp isolates in the U.K.³ This rise in anthropophilic dermatophyte infections is attributed to immigration and travel patterns.

6.3 Clinical patterns and diagnosis

An accurate diagnosis remains a vital component of management. Clinicians unfamiliar with this condition often misdiagnose tinea capitis, especially inflammatory variants such as boggy kerions, leading to delays in diagnosis and inappropriate management.^{4,5}

Tinea capitis predominates in healthy preadolescent children; infants are less frequently affected.^{6,7} The incidence in adults is generally low, but it is more commonly seen in the immune compromised, where the presentation may be atypical.^{8,9}

The clinical appearance of tinea capitis is highly variable, depending on the causative organism, type of hair invasion and degree of host inflammatory response. Common features are patchy hair loss with varying degrees of scaling and erythema. However, the clinical signs may be subtle and diagnosis can be challenging. A number of clinical patterns exist.

6.3.1 Noninflammatory

Grey patch Small-spored, ectothrix *Microsporum* infection typically produces characteristic fine scaling with patchy circular alopecia, dull grey in colour due to arthrospores coating the affected hairs. Inflammation may be minimal with anthropophilic fungi (e.g. *M. audouinii*, *M. ferrugineum*); however, zoophilic or geophilic species (e.g. *M. canis*, *M. gypseum*) typically demonstrate more intense inflammatory response.

Black dot Endothrix infection with *Trichophyton* species (e.g. *T. tonsurans*, *T. violaceum*, *T. soudanense*) produces relatively noninflammatory patches of alopecia with fine scale, classically studded with broken-off, swollen hair stubs, resulting in a 'black dot' appearance. Patches may be multiple.

Diffuse scale In some cases, alopecia is minimal or absent and infection presents as generalized, diffuse scaling of the scalp, resembling dandruff.

6.3.2 Inflammatory

Diffuse pustular In more inflammatory variants, a diffuse, patchy alopecia may coexist with scattered pustules or low-grade folliculitis. This may be associated with painful regional lymphadenopathy.

Kerion Also known as 'kerion celsi', this is the term given to tinea capitis presenting as a painful, boggy, inflammatory mass with associated alopecia. Plaques may be solitary or multiple, studded with pustules and matted with thick crust. Regional lymphadenopathy is common. This variant represents a delayed host inflammatory response to the causative dermatophyte. Misdiagnosis as bacterial abscess is not uncommon; however, secondary bacterial infection should not be overlooked. Kerion is commonly seen with zoophilic, large-spore ectothrix species (e.g. *T. mentagrophytes*, *T. verrucosum*); however, this has been superseded in recent years by endothrix infections with either *T. tonsurans* or *T. violaceum*, particularly in urban areas.¹⁰

Favus A chronic, inflammatory tinea capitis typically seen in *T. schoenleinii* infection, this variant is most commonly encountered in the Middle East and North Africa. Favus is characterized by yellow, crusted, cup-shaped lesions ('scutula') composed of hyphae and keratin debris, which develop around follicular openings. Favus may result in cicatricial alopecia. Favus infections fluoresce under Wood's lamp.

A pruritic, papular 'id' eruption, also known as 'dermatophytid', particularly around the outer helix of the ear, may accompany treatment initiation, but should not be confused with a drug reaction.^{11,12} These eruptions represent a cell-mediated host response to the dermatophyte after effective therapy has been initiated and do not warrant cessation of systemic antimycotic therapy. Topical (or occasionally, if very severe, oral) corticosteroids may provide symptomatic relief.

6.4 Clinical diagnostic aids

6.4.1 Wood's lamp

Ectothrix *Microsporum* species demonstrate bright green fluorescence of infected hairs under Wood's lamp examination. This may aid clinical distinction from nonfluorescent *Trichophyton* infection (exception: *T. schoenleinii* can fluoresce dull green), although the value of this investigation is limited given the current predominance of nonfluorescing species of *Trichophyton*.

6.4.2 Clinical patterns

The presence of regional lymphadenopathy in combination with alopecia and/or scale in a child suspected of having tinea capitis is an important diagnostic clue and should encourage appropriate investigation with fungal culture.^{13,14}

6.4.3 Dermoscopy

Although the authors have no personal experience of this technique, dermoscopy is being recommended as a useful adjunctive tool in diagnosing tinea capitis. Black dot hair stubs may be visualized more clearly. 'Comma-shaped' hairs have been described in white children with ectothrix infection, whereas corkscrew hairs have been reported in Afro-Caribbean children with tinea capitis.¹⁵

6.5 Differential diagnosis

The differential diagnosis of tinea capitis is extensive, encompassing any condition causing patchy hair loss, scaling or scalp inflammation. Scalp psoriasis, seborrhoeic dermatitis and atopic dermatitis may be difficult to differentiate from non-inflammatory tinea capitis, although these conditions are usually more diffuse, and there may be characteristic signs elsewhere. Alopecia areata is generally nonscaly but may occasionally demonstrate erythema. Exclamation-mark hairs must be distinguished from the broken hairs of tinea capitis. Lupus erythematosus, lichen planopilaris and trichotillomania should also be considered, although they are relatively rare. Inflammatory tinea capitis variants may be misdiagnosed as bacterial folliculitis, folliculitis decalvans or abscesses. Regional lymphadenopathy may be associated with inflammatory variants of tinea capitis.

7.0 Laboratory diagnosis of tinea capitis

Although the clinical diagnosis of tinea capitis is often relatively accurate, when considered, laboratory investigations to confirm the diagnosis are advisable to isolate the causal organism and direct the choice of systemic therapy.^{13,14} Post-treatment samples should be sent to ensure clearance.

7.1 Taking specimens

Suspected tinea capitis lesions should be sampled either by plucking hairs, using a blunt scalpel to remove hair and scalp scale, or by taking scalp brushings. In cases of tinea capitis caused by *M. canis*, affected hairs identified by fluorescence under a Wood's lamp may be plucked and constitute an appropriate specimen. Specimens should be collected in paper or card packs. Bonifaz *et al.*¹⁶ have shown that the cytobrush improves both sensitivity and time to positive culture. Furthermore, this is available as a sterile device and its soft bristles may cause less discomfort to children. (Strength of recommendation D; level of evidence 3; see Appendixes 1 and 2 for explanations of these measures.) A disadvantage of brush sampling is that it does not enable the laboratory to examine the specimen microscopically and permits only culture. Friedlander *et al.*¹⁷ have demonstrated that gauze swabs make an equally effective and often more convenient sampling method. A comparison of sampling methods for the detection of dermatophytes in asymptomatic carriage has been described.^{18,19} This suggests that multiple sampling methods, such as a scalp scraping and a brush, are likely to lead to an increased yield of dermatophyte fungus from infected scalps.²⁰ (Strength of recommendation D; level of evidence 3.) It is considered that sampling the edge of scalp lesions may provide higher yields of causal fungus. Sampling of kerions may be problematic, and culture is often negative. A swab of the lesion may provide the most appropriate specimen.

Scalp lesions in suspected cases of tinea capitis should be sampled by scalpel scraping, hair pluck, brush or swab as appropriate to the lesion. (Strength of recommendation D; level of evidence 3.)

7.2 Laboratory investigations

Microscopy should be carried out on all scalp scrapings and plucked hairs, by mounting in 10–30% potassium hydroxide with or without calcofluor, and examination by light or fluorescence microscopy. The presence of hyphae and/or arthroconidia should be reported. The sensitivity of microscopy is not high.²¹ (Strength of recommendation D; level of evidence 3.) Where possible it should be determined whether the arrangement of arthroconidia is endothrix or ectothrix, but this is often difficult. All specimens should be cultured on Sabouraud agar with at least one agar plate containing cycloheximide to inhibit nondermatophyte mould growth. Plates should be incubated for at least 2 weeks. Where exposure to cattle is documented and an infection caused by *T. verrucosum* is suspected, plates should be incubated for up to 3 weeks and examined very carefully at the end of this period for the presence of the slow-growing and inconspicuous colonies of this species. Any dermatophytes growing should be identified and reported. (Strength of recommendation D; level of evidence 4.) There is no routine indication to test dermatophytes for susceptibility to antifungal agents, as many studies have shown little evidence for the development of resistance.^{22,23} (Strength of recommendation D; level of evidence 3.)

All specimens from cases of tinea capitis should be processed for microscopy and culture where possible, and the causal agent fully identified where isolated. Susceptibility testing is not indicated. (Strength of recommendation D; level of evidence 4.)

8.0 Management

The primary objective of this guideline is to inform dermatologists treating tinea capitis in the U.K.

The aims of treatment are eradication of the organism, resulting in both a clinical and mycological cure as quickly and safely as possible; alleviation of symptoms; prevention of scarring and reduction of transmission to others. Oral therapy is generally required to achieve these goals.²⁴ (Strength of recommendation A; level of evidence 1+.)

8.1 When to start treatment

Ideally one should wait for confirmation of the presence of fungus, either by conducting microscopy at the patient side or waiting for culture. However, in high-risk populations, awaiting results increases delay (as culture results may take 2–4 weeks to be available) and may further increase spread. So, in the presence of a kerion or when the diagnosis of a fungal infection is strongly suspected clinically based on the presence of very typical features of scaling, lymphadenopathy or alopecia, it is reasonable to start therapy immediately, as these are strong predictive factors for tinea capitis.^{13,14} (Strength of recommendation B; level of evidence 2+.)

8.2 Topical therapy

Although a small percentage of patients may clear with topical agents,^{25,26} topical therapy alone is not recommended for the management of tinea capitis.²⁴ (Strength of recommendation B; level of evidence 2++.) However, topical agents are used to reduce transmission of spores,²⁷ and povidone–iodine, ketoconazole 2% and selenium sulfide 1% shampoos have all shown efficacy in this context. (Strength of recommendation C; level of evidence 2+.)

8.3 Oral therapy

It is reasonable to begin treatment on the basis of one or more cardinal signs,¹⁴ while awaiting confirmatory mycology. (Strength of recommendation B; level of evidence 2++.) Clear evidence has now emerged to show that the optimal treatment regimen varies according to the dermatophyte involved (Table 1).^{28–30} Treatment protocols should therefore reflect local epidemiology and be based on the most likely culprit organism.^{31,32} (Strength of recommendation A; level of evidence 1+.) A prolonged course or a change of agent may be required in cases of treatment failures (see Section Treatment failure), or if an unexpected fungus is identified on culture.

Although in the U.K., griseofulvin remains the only licensed treatment for tinea capitis in children, cumulative evidence now demonstrates that newer antifungal agents have higher response rates, and are safe and more cost-effective.^{24,31} This is reflected in recent changes to the licensing and availability of antifungal therapies in parts of Europe and the U.S.A. While we appreciate that the lack of licence in the U.K. may limit the ease of prescribing according to our recommendations, off-licence prescribing processes are well established in most NHS organizations, and the body of evidence supporting this guideline should endorse clinical practice.

8.3.1 Griseofulvin

Griseofulvin is a fungistatic drug that inhibits nucleic acid synthesis, arrests cell division at metaphase and impairs synthesis of the cell wall. There is over 50 years of experience in the use of the drug, and it remains the only licensed product for use in the treatment of tinea capitis in children in the U.K. It is available in several forms (micronized, ultramicronized and suspension), but recently the suspension has become increasingly expensive and not so widely available.²⁴ The suspension is no longer a licensed formulation in the U.K., and griseofulvin tablets are no longer available in some European countries, having been superseded by other agents.³³

Table 1 Choice of drug according to organism isolated

| | |
|---|------------------------------|
| <i>Trichophyton tonsurans</i> | Terbinafine |
| <i>Trichophyton violaceum, soudanense</i> | Terbinafine |
| <i>Microsporum canis</i> | Griseofulvin or itraconazole |
| <i>Microsporum audouinii</i> | Griseofulvin or itraconazole |

The standard licensed treatment protocol for those aged > 1 month is 1 g in children weighing > 50 kg, or 15–20 mg kg⁻¹ daily in single or divided doses for 6–8 weeks if < 50 kg. Taking the drug with fatty food may increase absorption and improve bioavailability. Dosage recommendations vary according to the type of formulation used and how easily it is absorbed. It may be necessary to use doses up to 25 mg kg⁻¹ daily for more prolonged periods in resistant cases.

A meta-analysis of seven studies²⁸ showed that response rates are highly variable depending on the species involved: 88 ± 5% for *Microsporum* species compared with 67.7 ± 9% for *Trichophyton* species. A recent meta-analysis of randomized controlled trials (RCTs) suggests that 8 weeks of griseofulvin treatment is significantly more effective than 4 weeks of terbinafine in confirmed *Microsporum* infection.³² There is no evidence of resistance to griseofulvin *in vitro*, but accumulated evidence suggests that the drug is less effective against *Trichophyton* species in the clinical setting,^{23,31} and higher doses for longer periods (12–18 weeks) may be required in *Trichophyton* infections.³¹

Side-effects occur in 20% of cases, mostly gastrointestinal upset, in particular diarrhoea, rashes and headache.³⁴ The drug is contraindicated in pregnancy and men are cautioned against fathering a child for 6 months after treatment.

Advantages: licensed for use in children in the U.K.; extensive experience; suspension more palatable to children and allows more accurate dosage adjustments.

Disadvantages: increasingly expensive; prolonged treatment required with potential to affect compliance.

Contraindications: lupus erythematosus, porphyria, severe liver disease.

Drug interactions: include warfarin, ciclosporin and the oral contraceptive pill.

8.3.2 Terbinafine

Terbinafine is an allylamine that acts on the cell membrane and is fungicidal. It shows activity against all dermatophytes,²² but has much higher efficacy against *Trichophyton* species than *Microsporum*.^{29,33} At higher doses, terbinafine is more effective against *M. canis* but confers no advantage over griseofulvin,³⁵ and prolonging treatment does not improve efficacy.³⁶ In part, this is because for *M. canis* infection, the minimum inhibitory concentration for terbinafine (and to some extent itraconazole) can exceed the maximum concentration reported in hair, contributing to treatment failures.³⁷ Additionally, terbinafine is not excreted in the sweat or sebum of prepubertal children, and cannot be incorporated into the hair shaft in children, so does not effectively reach the scalp surface where the arthroconidia are located in *Microsporum* infections, accounting for its relative inefficacy.³⁸

In contrast, meta-analysis of RCTs shows that 2–4 weeks of terbinafine is at least as effective as 6–8 weeks of griseofulvin in *T. tonsurans* infections.³² Terbinafine may now be considered the optimal choice, when cost-efficiency and compliance are taken into account.³⁹ Although shorter treatment protocols increase compliance,³⁹ and terbinafine has a clear cost

advantage (Table 2),²⁴ it remains unlicensed for use in children in the U.K. However, its widespread use is reflected in the publication of weight-related dosage schedules in recent editions of the *British National Formulary for Children*.

Although not available in liquid form in the U.K., a new granule formulation of terbinafine (available in 125-mg or 187.5-mg packets to be sprinkled on food) has been licensed for use in children > 4 years of age in the U.S.A.,⁴⁰ and offers significantly higher cure rates than standard griseofulvin suspension, even at higher dosage schedules (Table 3).⁴¹ However, it is not currently available or licensed in the U.K.

Pharmacokinetic studies of terbinafine show that children require significantly weight-normalized doses to approximate the equivalent drug levels needed for efficacy in adults. However, there is no suggestion of any altered safety profile in children compared with adults.⁴²

Overall, terbinafine appears well tolerated in children.^{29,42–44} Side-effects include gastrointestinal disturbances and rashes in < 8%, and very few (0.8%) are required to discontinue treatment.⁴³

Advantages: fungicidal; shorter treatment regimens, so potential to improve compliance; cost; safety.

Disadvantages: no suspension formulation (but in U.S.A., granules provide a palatable alternative); not licensed for treatment of children in the U.K.

Drug interactions: plasma concentration is decreased by rifampicin and increased by cimetidine.

8.3.3 Itraconazole

Itraconazole exhibits both fungicidal and fungistatic activity depending on the tissue concentration of the drug, but, like other azoles, its primary mode of action is fungistatic, through depletion of cell-membrane ergosterols, which interferes with membrane permeability. Doses of 50–100 mg daily for 4 weeks⁴⁵ or 5 mg kg⁻¹ daily for 2–4 weeks have comparable efficacy with griseofulvin or terbinafine.³¹ (Strength of recommendation A; level of evidence 1+.)

Itraconazole is now the preferred agent in the majority of European countries³³ and has activity against both *Microsporum*^{46,47} and *Trichophyton* species.³¹ The drug is well tolerated^{46–48} (Strength of recommendation B; level of evidence 2++) and has been shown to be safe for use in the first year of

Table 2 Comparative costs of antifungal treatment (U.K. 2014)^a

| Drug | Dose | Duration | Cost |
|--------------------------|----------------|----------|--------------------|
| Griseofulvin | 200–300 mg | 8 weeks | £47.04 (tablet) |
| | per day | | £119.00 (syrup) |
| Terbinafine | 125 mg per day | 4 weeks | £2.12 ^b |
| Itraconazole | 100 mg per day | 4 weeks | £11.92 (tablet) |
| | | | £108.90 (liquid) |
| Fluconazole (suspension) | 180 mg | 4 weeks | £265.68 |

^aBased on treatment of a 20-kg child. ^bBased on halving a conventional tablet.

Table 3 Summary of treatment choice

| | |
|----------------------|---|
| Laboratory diagnosis | Scalp lesions in suspected cases should be sampled via scalpel scraping, hair pluck, brush or swab. All specimens should be processed for microscopy and culture, where possible. Susceptibility testing is not indicated (Strength of recommendation D) |
| Treatment | In the presence of a kerion or where one or more of the cardinal clinical signs is present (scale, lymphadenopathy, alopecia) it is reasonable to commence treatment while awaiting confirmatory mycology (Strength of recommendation B) Topical therapy alone is not recommended for the treatment of tinea capitis. Oral therapy is generally indicated to achieve both clinical and mycological cure (Strength of recommendation A) Choice of systemic therapy should be directed by causative dermatophyte and/or local epidemiology (Strength of recommendation A) |
| First-line therapy | Both griseofulvin and terbinafine have good evidence of efficacy and remain the most widely used first-line treatments. As a general rule, terbinafine is more efficacious against <i>Trichophyton</i> species (<i>T. tonsurans</i> , <i>T. violaceum</i> , <i>T. soudanense</i>), and griseofulvin more effective against <i>Microsporum</i> species (<i>M. canis</i> , <i>M. audouinii</i>). In the U.K., griseofulvin remains the only licensed treatment for tinea capitis in children, although the suspension formulation is no longer licensed for use. Terbinafine requires a shorter course of treatment, which may increase compliance (Strength of recommendation A) Griseofulvin dose by body weight < 50 kg 15–20 mg kg ⁻¹ per day (single or divided dose) for 6–8 weeks > 50 kg 1 g per day (single or divided dose) for 6–8 weeks Doses up to 25 mg kg ⁻¹ per day may be required in some cases Terbinafine dose by body weight < 20 kg 62.5 mg per day for 2–4 weeks 20–40 kg 125 mg per day for 2–4 weeks > 40 kg 250 mg per day for 2–4 weeks |
| Treatment failure | Initially consider lack of compliance, suboptimal absorption of drug, relative insensitivity of the organism and reinfection. In cases of clinical improvement but ongoing positive mycology, continue current therapy for a further 2–4 weeks. If there has been no initial clinical improvement, proceed to second-line therapy below |
| Second-line therapy | Itraconazole is safe, effective and has activity against both <i>Trichophyton</i> and <i>Microsporum</i> species. If itraconazole has been selected as first-line therapy, convert to terbinafine second line for <i>Trichophyton</i> infections or griseofulvin for <i>Microsporum</i> species, at standard dosing regimens (Strength of recommendation C) Itraconazole, 50–100 mg per day for 4 weeks, or 5 mg kg ⁻¹ per day for 2–4 weeks |
| Alternative agents | For cases refractory to the above regimens, other modalities to be considered in exceptional circumstances include fluconazole and voriconazole (see main text) |
| Additional measures | Children receiving appropriate therapy should be allowed to attend school or nursery [Strength of recommendation D (GPP)] Index cases due to <i>T. tonsurans</i> warrant screening of all family members and close contacts and treatment for those positive cases (Strength of recommendation B) In asymptomatic carriers (no clinical infection, culture positive) with a high spore load, systemic treatment is generally justified [Strength of recommendation D (GPP)] The end point of treatment is mycological rather than clinical cure; therefore repeat mycology sampling is recommended until mycological clearance is achieved [Strength of recommendation D (GPP)] |

life.⁴⁹ (Strength of recommendation D; level of evidence 3.) Intermittent dosing regimens are effective^{48,50} and may be preferred.^{48,51}

Although licensed in Europe, the drug is not currently licensed for the treatment of tinea capitis in children in the U.K. aged 12 years and under.

Advantages: pulsed regimes; shorter treatment protocols; available in liquid form; has licence for use in children aged > 12 years.

Disadvantages: not licensed in the U.K. for children aged ≤ 12 years with tinea capitis.

Drug interactions: enhanced toxicity of warfarin, some antihistamines (specifically terfenadine, astemizole), antipsychotics

(sertindole), anxiolytics (midazolam), digoxin, cisapride, ciclosporin and simvastatin (increased risk of myopathy); decreased efficacy with concomitant H2 blockers, phenytoin and rifampicin.

8.3.4 Fluconazole

Fluconazole has been used in the treatment of tinea capitis⁵² and has been advocated as an alternative to terbinafine,²⁴ but its use has been relatively limited because of side-effects and because it confers no cost advantage. (Strength of recommendation C; level of evidence 2+.) Comparative efficacy with griseofulvin in a multicentre study of mixed pathogens⁵³ and superior activity in

eradication of *T. violaceum*, *T. verrucosum* and *M. canis* has been shown with fluconazole,⁵⁴ but due its cost and limited availability, griseofulvin remains the treatment of choice in many parts of the world. (Strength of recommendation B; level of evidence 2++.)

Fluconazole is not licensed for the treatment of tinea in children aged < 10 years in the U.K.; however, it is licensed for use in all children for mucosal candidiasis. Furthermore, the drug is licensed for treatment of tinea in children aged > 1 year in Germany.⁵⁵ Once-weekly dosing regimens have been used and appear well tolerated.⁵⁶ (Strength of recommendation B; level of evidence 2++.)

8.3.5 Voriconazole

Voriconazole is more potent against dermatophyte isolates than griseofulvin or fluconazole,⁵⁷ but cost, licensing restrictions and availability limit its current usage. (Strength of recommendation C; level of evidence 2+.)

8.3.6 Ketoconazole

Although the efficacy of ketoconazole in tinea capitis at doses of 3.3–6.6 mg kg⁻¹ daily has been demonstrated in the past, and it has shown comparability with griseofulvin,⁵⁸ resolution of symptoms appears slower and the side-effect profile is sufficiently poor (especially the risk of hepatotoxicity) that oral ketoconazole was withdrawn from use in U.K. and Europe in 2013.

9.0 Additional measures

9.1 Exclusion from school

Although the potential risk of transmission of infection to unaffected classmates has led some authorities to recommend exclusion from school,⁵⁵ most experts consider this impractical and suggest that children receiving appropriate systemic and adjunctive topical therapy should be allowed to attend school or nursery.^{45,59–61} [Strength of recommendation D (GPP); level of evidence 4.]

9.2 Family screening

Index cases due to the anthropophilic *T. tonsurans* are highly infectious.⁶² More than 50% of family members (including adults) may be affected, often with occult disease.⁶³ Failure to treat the whole family will result in high recurrence rates. (Strength of recommendation B; level of evidence 2++.) Therefore we recommend screening of all family members and treating those found positive.

9.3 Cleansing of fomites

Viable spores have been isolated from hairbrushes and combs. For all anthropophilic species, these should be cleansed with

disinfectant.⁶⁴ (Strength of recommendation D; level of evidence 4.) This has particular implications for barbers, who need to ensure that appropriate measures are taken to disinfect multiuser equipment. Proprietary phenolic disinfectants are no longer available, but simple bleach or a 2% aqueous solution of sodium hypochlorite containing 16.5% salt are suitable alternatives. (Strength of recommendation D; level of evidence 3.)

9.4 Steroids

The use of corticosteroids (both oral and topical) for inflammatory varieties of tinea capitis (e.g. kerion and severe id reactions) may reduce itching and general discomfort, but is controversial. Historically, oral steroids were thought to reduce scarring, but studies show that, compared with oral antifungal therapy alone, they do not reduce the time to clearance, and therefore confer no long-term advantage,^{10,65,66} so are not recommended. (Strength of recommendation C; level of evidence 2+.) Scarring is rare in *T. tonsurans* infection, and hair usually fully regrows after effective oral antifungal therapy alone.

9.5 Treatment failure

Some individuals are not clear at follow-up. The reasons for this include (i) lack of compliance – especially in long treatment courses; (ii) suboptimal absorption of drug; (iii) relative insensitivity of the organism; and (iv) reinfection.

If fungi can still be isolated at the end of treatment, but the clinical signs have improved, it is reasonable to continue therapy for a further 2–4 weeks. However, if there has been no clinical response, it is imperative to ensure that the antifungal therapy is appropriate for the causal organism identified on culture. If so, the options then are (i) to increase the dose or duration of the original drug; or (ii) to change to an alternative agent, for example griseofulvin → itraconazole (for *M. canis*); terbinafine → itraconazole (for *T. tonsurans*); or itraconazole → terbinafine (for *T. tonsurans*).

9.6 Carriers

The optimal management of asymptomatic carriers (i.e. those individuals without overt clinical infection who are culture positive) is unclear, but current management practice depends on the spore load.⁶⁷ Asymptomatic carriage is highest in contacts of individuals with *T. tonsurans* infection,⁶⁷ but can occur in *M. audouinii* outbreaks as well.⁶⁸

In asymptomatic carriers with a high spore load, oral therapy is usually justified.⁶⁷ If the spore load is low, carriage may be eradicated with topical treatment alone, but close follow-up is needed, with repeat mycology, to ensure that treatment has been effective. Ideally, the eradication of asymptomatic carriers requires the support and involvement of community healthcare workers, including school nurses, to be comprehensive and effective.⁶⁷ However, although guidelines have been issued, tinea capitis is not considered a public health priority in the U.K. at present, so enforcement is

hampered and widely variable.²⁴ [Strength of recommendation D (GPP); level of evidence 4.]

9.7 Follow-up

The definitive end point for adequate treatment must be mycological cure, rather than clinical response. Therefore, follow-up with repeat mycology sampling is recommended at the end of the standard treatment period and then monthly until mycological clearance is documented. (Strength of Recommendation D; level of evidence 4.) Treatment should therefore be tailored to each individual patient according to response.

10.0 Future directions

Studies continue to look at the emerging and changing epidemiology of tinea capitis across Europe and the rest of the world, and may alter first-line medication recommendations in the future. Additionally, cost-effectiveness considerations are likely to evolve with time. However, diagnostic tools do seem to be emerging that might enable the laboratory diagnosis to be made much more quickly, which will enhance treatment decision making in the U.K.

The future direction for laboratory diagnosis of superficial fungal infections is likely to be a molecular one, and diagnosis of tinea capitis is unlikely to be an exception. Real-time polymerase chain reaction (PCR) tests and PCR reverse-line blot assays, designed for dermatophyte infections, have generally performed well on clinical specimens including hair from patients with tinea capitis.^{69–71} A method for the detection of *T. tonsurans* from hairbrushes has also been described.⁷² Larger-scale comparative studies of PCR, microscopy and culture are required to determine whether DNA detection of dermatophytes will improve the diagnosis of tinea capitis.

11.0 Recommended audit points

- 1 In the last 20 consecutive patients seen with tinea capitis, were specimens taken to confirm the diagnosis (as systemic therapy will be required)?
- 2 In the last 20 consecutive patients seen with tinea capitis, were children allowed to return to school once they had been commenced on appropriate systemic and adjuvant topical therapy and followed up until mycological clearance was documented?
- 3 In the last 20 consecutive patients seen with tinea capitis, had patients been given effective therapy?
- 4 In the last 20 consecutive patients seen with tinea capitis due to *T. tonsurans*, were family members and other close contacts screened (both for tinea capitis and corporis) and appropriate mycological samples taken, preferably using the brush technique, even in the absence of clinical signs?

The audit recommendation of 20 cases per department is to reduce variation in the results due to a single patient, and to allow benchmarking between different units. However,

departments unable to achieve this recommendation may choose to audit all cases seen in the preceding 12 months.

12.0 Summary

Details of evidence are given in the text, and summarized in Table 3. Tinea capitis is a common scalp infection, seen predominantly in childhood. The clinical presentation is highly variable and dependent on the causative organism. The condition is most commonly due to *Microsporum* and *Trichophyton* dermatophyte species, with *T. tonsurans* now accounting for the majority of scalp isolates in the U.K.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Table S1. Literature search strategy.

Appendix 1

Levels of evidence

| Level of evidence | Type of evidence |
|-------------------|---|
| 1++ | High-quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias |
| 1+ | Well-conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias |
| 1– | Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias ^a |
| 2++ | High-quality systematic reviews of case-control or cohort studies. High-quality case-control or cohort studies with a very low risk of confounding, bias or chance and a high probability that the relationship is causal |
| 2+ | Well-conducted case-control or cohort studies with a low risk of confounding, bias or chance and a moderate probability that the relationship is causal |
| 2– | Case-control or cohort studies with a high risk of confounding, bias or chance and a significant risk that the relationship is not causal ^a |
| 3 | Nonanalytical studies (e.g. case reports, case series) |
| 4 | Expert opinion, formal consensus |

RCT, randomized controlled trial. ^aStudies with a level of evidence '–' should not be used as a basis for making a recommendation.

Appendix 2

Strength of recommendation

| Class | Evidence |
|---------|---|
| A | At least one meta-analysis, systematic review or RCT rated 1++, and directly applicable to the target population, or A systematic review of RCTs or a body of evidence consisting principally of studies rated as 1+, directly applicable to the target population and demonstrating overall consistency of results Evidence drawn from a NICE technology appraisal |
| B | A body of evidence including studies rated 2++, directly applicable to the target population and demonstrating overall consistency of results, or Extrapolated evidence from studies rated 1++ or 1+ |
| C | A body of evidence including studies rated 2+, directly applicable to the target population and demonstrating overall consistency of results, or Extrapolated evidence from studies rated 2++ |
| D | Evidence level 3 or 4, or Extrapolated evidence from studies rated 2+, or Formal consensus |
| D (GPP) | A good practice point (GPP) is a recommendation for best practice based on the experience of the guideline development group |

RCT, randomized controlled trial; NICE, National Institute for Health and Care Excellence.