

Guidelines for the management of tinea capitis

E.M.HIGGINS,*† L.C.FULLER* AND C.H.SMITH‡

*King's College Hospital, London SE5 9RS, U.K.

†Orpington Hospital, Kent BR6 9SU, U.K.

‡Lewisham Hospital, London SE13 6LH, U.K.

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Summary

These guidelines for the management of tinea capitis have been prepared for dermatologists on behalf of the British Association of Dermatologists. They present evidence-based guidance for treatment, with identification of the strength of evidence available at the time of preparation of the guidelines, and a brief overview of epidemiological aspects, diagnosis and investigation.

Key words: guidelines, tinea capitis

Disclaimer

These guidelines have been prepared for dermatologists on behalf of the British Association of Dermatologists and reflect the best data available at the time the report was prepared. Caution should be exercised in interpreting the data; the results of future studies may require alteration of the conclusions or recommendations in this report. It may be necessary or even desirable to depart from the guidelines in the interests of specific patients and special circumstances. Just as adherence to the guidelines may not constitute defence against a claim of negligence, so deviation from them should not necessarily be deemed negligent.

Definition

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

Epidemiology

Tinea capitis is predominantly a disease of preadolescent children, adult cases being rare.¹ Although world-wide in distribution, its prevalence in the U.K. has been relatively low in the past. An increase in

prevalence has recently been reported in urban areas, particularly in children of Afro-Caribbean extraction.^{1–3} The main pathogens are anthropophilic organisms with *Trichophyton tonsurans* now accounting for > 90% of cases in the U.K. and North America.^{2,4,5} These infections frequently spread among family members and classmates.^{5,6} Certain hairdressing practices such as shaving of the scalp, plaiting or the use of hair oils may promote disease transmission, but their precise role remains the subject of study. In non-urban communities, sporadic infections acquired from puppies and kittens are due to *M. canis*, which, however, accounts for less than 10% of cases in the U.K. Occasional infection from other animal hosts (e.g. *T. verrucosum* from cattle) occur in rural areas.

Pathogenesis

There are three recognized patterns: endothrix, ectothrix and favus. The latter, a pattern of hair loss caused by *T. schoenleinii*, is rarely seen in the U.K. being largely confined to eastern Europe and Asia and is not considered further here. Endothrix infections are characterized by arthroconidia (spores) within the hair shaft. The cuticle is not destroyed. Ectothrix infections are characterized by hyphal fragments and arthroconidia outside the hair shaft, which leads eventually to cuticle destruction.

Clinical diagnosis of scalp ringworm

A variety of clinical presentations are recognized as being either inflammatory or non-inflammatory and

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Table 1. Summarizing the clinical patterns of tinea capitis

Clinical patterns	Clinical description	Differential diagnosis
Diffuse scale	Generalized diffuse scaling of the scalp	Seborrhoeic and atopic dermatitis, psoriasis
Grey patch	Patchy, scaly alopecia	Seborrhoeic and atopic dermatitis, psoriasis
Black dot	Patches of alopecia studded with broken-off hair stubs	Alopecia areata, trichotillomania
Diffuse pustular	Scattered pustules associated with alopecia scaling \pm associated lymphadenopathy	Bacterial folliculitis, dissecting folliculitis
Kerion	Boggy tumour studded with pustules \pm associated lymphadenopathy	Abscess, neoplasia

are usually associated with patchy alopecia (Table 1). However, the infection is so widespread, and the clinical appearances can be so subtle, that in urban areas, tinea capitis should be considered in the diagnosis of any child over the age of 3 months with a scaly scalp, until dismissed by negative mycology. Infection may also be associated with painful regional lymphadenopathy, particularly in the inflammatory variants. A generalized eruption of itchy papules particularly around the outer helix of the ear may occur as a reactive phenomenon (an 'id' response). This may start with the introduction of systemic therapy and so be mistaken for a drug reaction.

Laboratory diagnosis of scalp ringworm

If tinea capitis is suspected, specimens should be taken to confirm the diagnosis as systemic therapy will be required.

Taking specimens

Affected areas should be scraped with a *blunt* scalpel to harvest affected hairs, broken-off hair stubs and scalp scale. This is preferable to plucking, which may remove uninvolved hairs. Scrapings should be transported in a folded square of paper preferably fastened with a paper clip, but commercial packs are also available (e.g. Dermapak, Dermaco, Toddington, Bedfordshire, U.K. and Mycotrans, Biggar, Lanarkshire, U.K.). It is easier to see affected hairs on white paper rather than black. Alternatively, the area can be rubbed with a moistened gauze swab^{7,8} or brushed gently 10 times with a plastic, sterile, single use toothbrush [BR8 (unpasted), Brushaway Products, Chislehurst Railway Station, Station Approach, Chislehurst, Kent BR7 5NN, U.K.]. The brush can then be sent in the container provided to the laboratory for culture.⁹ (*Strength of recommendation = A; quality of evidence III.*)

Microscopy and culture

Samples should be sent to laboratories with a particular interest and expertise in mycology.

Microscopy provides the most rapid means of diagnosis, but is not always positive. Scalp scales and broken off hair stumps containing the root section (rather than intact hairs) are mounted in a 10–30% potassium hydroxide solution and viewed under the light microscope. Positive microscopy (when the hairs or scales are seen to be invaded by spores or hyphae) confirms the diagnosis and allows treatment to commence at once.

Scales, hairs or samples obtained with a sterile single use brush are often not suitable for microscopy, but are inoculated on to a suitable culture medium, e.g. Sabouraud's. Culture allows accurate identification of the organism involved, and this may alter the treatment schedule. Culture is more sensitive than microscopy; results may be positive even when microscopy is negative, but may take up to 4 weeks to become available.

Conventional sampling of a kerion can be difficult. In these cases negative results are not uncommon and the diagnosis and decision to treat may need to be made clinically. A moistened standard bacteriological swab taken from the pustular areas and inoculated on to the culture plate may yield a positive result.⁸

Wood's light examination

This is useful for certain ectothrix infections, e.g. those caused by *M. canis*, *M. rivalieri* and *M. audouinii*, which cause the hair to fluoresce bright green. However, as most of the current infections in the U.K. are endothrix and so negative under Wood's light, it is of limited value for screening and monitoring these infections.

Therapy of tinea capitis

The aim of treatment is to achieve a clinical and mycological cure as quickly as possible. Oral antifungal

Table 2. Dosing regimen for tinea capitis

Drug	Current standard dose	Duration
Griseofulvin	10–25 mg/kg daily taken with food divided dose	8–10 weeks
Terbinafine	< 20 kg 62.5 mg od: > 20 < 40 kg 125 mg od: > 40 kg 250 mg od	4 weeks ^a
Itraconazole	5 mg/kg per day	1–4 weeks

^a Longer for *Microsporum* infections.

therapy is generally needed.¹⁰ (*Strength of recommendation = A; quality of evidence III.*)

Topical

Topical treatment alone is not recommended for the management of tinea capitis.¹⁰ (*Strength of recommendation = A; quality of evidence = III.*) It may however, reduce the risk of transmission to others in the early stages of systemic treatment. (*Strength of recommendation = B; quality of evidence = III.*) Selenium sulphide¹¹ and povidone iodine¹² shampoos, used twice weekly, reduce the carriage of viable spores and are assumed to reduce infectivity.

Oral

Griseofulvin. This is fungistatic, and inhibits nucleic acid synthesis, arresting cell division at metaphase and impairing fungal cell wall synthesis. It is also anti-inflammatory. It remains the only licensed treatment for scalp ringworm in U.K. It is available in tablet or suspension form. The recommended dose, for those older than 1 month is 10 mg/kg per day. Taking the drug with fatty food increases absorption and aids bioavailability.^{13,14} Dosage recommendations vary according to the formulation used, with higher doses being recommended by some authors for micronized griseofulvin as opposed to ultramicrozoned griseofulvin, but up to 25 mg/kg may be required. The duration of therapy depends on the organism (e.g. *T. tonsurans* infections may require prolonged treatment schedules) but varies between 8 and 10 weeks. (*Strength of recommendation = A; quality of evidence = III.*) Shorter courses may lead to higher relapse rates.

Side-effects include nausea and rashes in 8–15%. The drug is contra-indicated in pregnancy and the manufacturers caution against men fathering a child for 6 months after therapy. (*Strength of recommendation = B; quality of evidence = III.*)

Advantages. Licensed; inexpensive; syrup formulation is more palatable; suspension allows accurate dosage adjustments in children; and extensive experience.

Disadvantages: Prolonged treatment required.

Contra-indicated in lupus erythematosus, porphyria and severe liver disease.

Drug interactions. Include warfarin, cyclosporin and the oral contraceptive pill.

Terbinafine. This acts on fungal cell membranes and is fungicidal. It is effective against all dermatophytes. It is not yet licensed for tinea capitis, although a licence for children of > 2 years is being considered. It is at least as effective as griseofulvin and is safe for the management of scalp ringworm due to *Trichophyton* sp. in children.^{15–17} Its role in management of *Microsporum* sp. is debatable.¹⁸ Early evidence suggests that higher doses or longer therapy (> 4 weeks) may be required in microsporum infections.^{19,20} Dosage depends on the weight of the patient, but lie between 3 and 6 mg/kg per day (see Table 2). Side-effects include; gastrointestinal disturbances and rashes in 5% and 3% of cases, respectively.²¹ (*Strength of recommendation = A/B; quality of evidence = I/III.*)

Advantages. Fungicidal so shorter therapy required (cf. griseofulvin) so increased compliance more likely.

Disadvantages. No suspension formulation and no U.K. licence for tinea capitis.

Drug interactions. Plasma concentrations are reduced by rifampicin and increased by cimetidine.

Itraconazole. Itraconazole exhibits both fungistatic and fungicidal activity depending on the concentration of drug in the tissues, but like other azoles, the primary mode of action is fungistatic, through depletion of cell membrane ergosterol, which interferes with membrane permeability. 100 mg/day for 4 weeks or 5 mg/kg per day in children is as effective as griseofulvin²² and terbinafine.²³ (*Strength of recommendation = B; quality of evidence = I.*) Small studies suggest that shorter or pulsed regimens may also be effective.²⁴ (*Strength of recommendation = B; quality of evidence = I.*) Itraconazole is currently unlicensed in the U.K. for use in tinea capitis and for use in children and there are no plans to change this.

Advantage. Pulsed shorter treatment regimens are possible.

Disadvantage. Lack of U.K. licence to treat tinea capitis and possible side-effects. Potential drug interactions. More studies needed to confirm paediatric requirements.

Drug interactions. Enhanced toxicity of anticoagulants (warfarin), antihistamines (terfenadine and astemizole), antipsychotics (sertindole), anxiolytics (midazolam), digoxin, cisapride, cyclosporin and simvastatin (increased risk of myopathy). Reduced efficacy of itraconazole with concomitant use of H₂-blockers, phenytoin and rifampicin.

Fluconazole. Has occasionally been assessed for tinea capitis but its use has mainly been limited by side-effects. Doses of 3–5 mg/kg per day for 4 weeks are effective in children with tinea capitis.²⁵ There are no large published series of its use and it is not licensed for the treatment of tinea capitis in the U.K. (*Strength of recommendation = C; quality of evidence = I.*)

Ketoconazole. Has occasionally been assessed for tinea capitis but its use has mainly been limited by side-effects. Doses of between 3.3 and 6.6 mg/kg per day. Resolution is comparable with griseofulvin but the response may be slower. However, side-effects are arguably sufficiently significant to lead to the recommendation by some authors that it should not be used in children.²⁶ Studies have not shown it to be consistently superior to griseofulvin and its use in children is limited by hepatotoxicity. Not licensed for the treatment of tinea capitis in the U.K. (*Strength of recommendation = D; quality of evidence = I.*)

Additional measures

Exclusion from school. Although there is a risk of the transmission of infection from patients to unaffected class-mates, for practical reasons children should be allowed to return to school once they have been commenced on appropriate systemic and adjuvant topical therapy.^{3,12} (*Strength of recommendation = B; quality of evidence III.*)

Familial screening. Index cases due to the anthropophilic *T. tonsurans* are highly infectious. Family members²⁷ as well as other close contacts should be screened (both for tinea capitis and corporis) and appropriate mycological samples taken preferably using the brush technique, even in the absence of clinical signs. (*Strength of recommendation = B; quality of evidence = III.*)

Cleansing of fomites. Viable spores have been isolated from hairbrushes and combs. For all anthropophilic species these should be cleansed with disinfectant.²⁸ (*Strength of recommendation = B; quality of evidence = IV.*) Proprietary phenolic disinfectants are no longer available, but simple bleach or Milton should be suitable alternatives. (*Strength of recommendation = B; quality of evidence = III.*)

Soaking off crust from kerions or pustules. This is recommended by some authors. Although this is not necessary, it is often soothing.²⁹ (*Strength of recommendation = C; quality of evidence III.*)

Steroids. The use of corticosteroids (both oral and topical) for inflammatory varieties, e.g. kerions and severe id reactions is controversial, but may help to reduce itching and general discomfort.³⁰ Although in the past steroids have been thought to minimize the risk of permanent alopecia secondary to scarring, current evidence does not suggest any reduction in clearance time compared with griseofulvin alone.^{29,30} (*Strength of recommendation = C; quality of evidence = I.*)

Treatment failures

Some individuals are not clear at follow-up. The reasons for this include:

- 1 Lack of compliance with the long courses of treatment.
- 2 Suboptimal absorption of the drug.
- 3 Relative insensitivity of the organism.
- 4 Reinfection.

T. tonsurans and *Microsporum* sp. are typical culprits in persistently positive cases. If fungi can still be isolated at the end of treatment, but the clinical signs have improved, the authors recommend continuing the original treatment for a further month.

If there has been no clinical response and signs persist at the end of the treatment period, then the options include:

- 1 Increase the dose or duration of the original drug: both griseofulvin (in doses up to 25 mg/kg for 8–10 weeks) and terbinafine have been used successfully and safely at higher doses or for longer courses to clear resistant infections. (*Strength of recommendation = C; quality of evidence = IV.*)
- 2 Change to an alternative antifungal, e.g. switch from

griseofulvin to terbinafine or itraconazole. (*Strength of recommendation = C; quality of evidence = IV*)

Carriers

The optimal management of symptom-free carriers (i.e. individuals without overt clinical infection, but who are culture positive) is unclear. In those with a heavy growth/high spore count on brush culture, systemic antifungal therapy may be justified as these individuals are especially likely to develop an overt clinical infection, are a significant reservoir of infection³ and are unlikely to respond to topical therapy alone.^{5,31} Alternatively, they may represent a missed overt clinical infection. For those with light growth/low spore counts on brush culture, twice weekly selenium sulphide or povidone iodine shampoo is probably adequate.^{11,12} (*Strength of recommendation = B; quality of evidence = IV*)

Follow-up

The definitive end-point for adequate treatment is not clinical response but mycological cure; 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 = A*.) Treatment should, therefore, be tailored for each individual patient according to response.

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Appendix

Strength of recommendations

- A** There is good evidence to support the use of the procedure.
- B** There is fair evidence to support the use of the procedure.
- C** There is poor evidence to support the use of the procedure.
- D** There is fair evidence to support the rejection of the use of the procedure.

- E** There is good evidence to support the rejection of the use of the procedure.

Quality of evidence

- I** Evidence obtained from at least one properly designed, randomized control trial.
- II-i** Evidence obtained from well designed, controlled trials without randomization.
- II-ii** Evidence obtained from well designed cohort or case–control analytical studies, preferably from more than one centre or research group.
- II-iii** Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled experiments (e.g. the introduction of penicillin treatment in the 1940s) could also be regarded as this type of evidence.
- III** Opinions of respected authorities based on clinical experience, descriptive studies or reports of expert committees.
- IV** Evidence inadequate owing to problems of methodology (e.g. sample size, length or comprehensiveness of follow-up or conflicts of evidence)