Chapter 9
Fungal Infections of the Hair

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9.1 Common Fungal Infection of the Hair Shaft: Tinea Capitis

Tinea capitis or scalp ringworm is an infection of the scalp hair caused by dermatophyte fungi. Only certain species appear to have the physiological capacity to invade hair shafts, and one, *Trichophyton schoenleinii*, invades to cause the disease known as favus but does not survive for long within the hair shaft keratin. Conversely, dermatophytes causing tinea capitis can also invade the skin of other sites, mainly the body or the face [1, 2]. Other dermatophyte infections are discussed in Chap. 8

9.2 Introduction

Scalp ringworm has been known for centuries and is well described in the older dermatological literature although often confused with other causes of scalp scaling. Topical treatments, such as mercurials, tars and, later, dyes were used extensively as treatments along with physical methods of epilation. Favus which is a clinically distinct form of infection with crusts and a sharp odour was also recognised for centuries because of its clinical appearances. Control of scalp ringworm was part of a major public health movement in Europe and the USA in the early and middle

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parts of the twentieth century [2]. Children with tinea capitis were often segregated or excluded from school, and adjunctive treatments such as scalp shaving prior to application of topical medications were frequently used, thereby contributing to the stigma. In the UK, for instance, schools or hospital wards originally attached to the Poor Law workhouses were often converted into hospital or clinical treatment facilities and used for specific purposes such as treatment of ringworm which was associated with poverty compounding the stigma that went with it. The newly discovered radiation was employed as a means of depilation using machines specifically designed for the purpose, and many hundreds of children were treated. The long-term consequences of this treatment included radiodermatitis and dysplastic skin lesions or nonmelanoma skin cancer. By the middle of twentieth century, there was also recognition that tinea capitis could reach epidemic proportions, and outbreaks of infection were recorded by community medical officers and also in the dermatological literature.

A massive change in the distribution of tinea capitis followed the introduction of griseofulvin as in a few years a combination of organised treatment regimens, surveillance in schools through the school nursing services and the treatment of contacts resulted in the virtual disappearance of endemic tinea capitis from Europe and the USA by the 1970s [2, 3]. In the succeeding years, tinea capitis was a largely sporadically occurring disease with most new cases attributable to infection from pets or domestic or farm animals. *Microsporum canis* dominated the clinical pattern. The picture is changing again in the USA, parts of Europe and in Latin America with the spread of *Trichophyton tonsurans* originally described by Sabouraud as known to have been present in Europe [3–7]. The source of the most recent outbreak of *Trichophyton tonsurans* infection is not completely understood, but it may well have entered the southern USA with migrants from Mexico and Central America where the infection had remained endemic at a low level. However, the new outbreak of infection travelling through the USA in schools has crossed the Atlantic and is now prevalent in European schools as well as spreading to South America and Africa. The major focus of infection is children with black hair type. With the difficulties and costs inherent in mass treatment and school surveillance, tinea capitis remained an endemic disease in many resource poor countries throughout this period (Figs. 9.1, 9.2, 9.3 and 9.4).

9.3 Epidemiology

Tinea capitis is a disease of childhood although adults may be affected occasionally. Although it is usually seen in children from 2 years old to puberty, it may occur in those younger and even within the first 6 months of life [2]. Infection is usually derived from either a human (anthropophilic) or animal source (zoophilic). Infection in younger children is mainly due to anthropophilic infection. There has been little

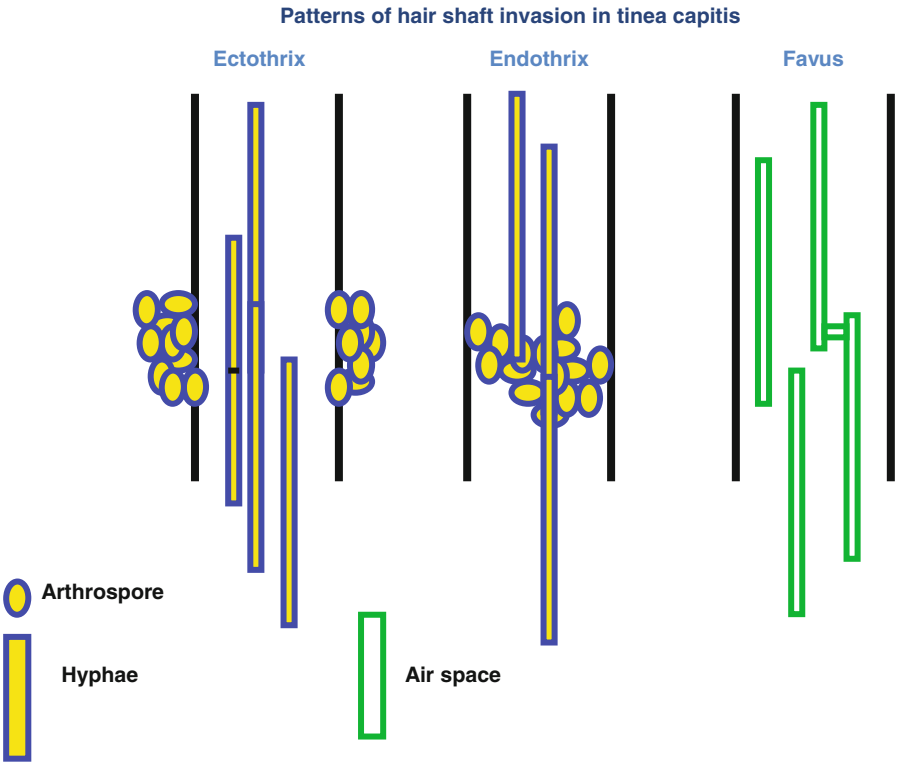

Fig. 9.1 Tinea capitis – patterns of hair shaft invasion

research on susceptibility to this infection although recently polymorphisms within certain genes such as those involved in leucocyte activation and migration, extracellular matrix integrity and remodelling, epidermal maintenance and wound repair, and cutaneous permeability have been associated with increased susceptibility to tinea capitis caused by *Trichophyton tonsurans* in children [8]. Apart from this, there is little information on the basis for susceptibility although infection is clearly associated with the presence of other infected children, and classroom or household spread is a likely factor in transmission. In addition, other sources of exposure may be involved, and sharing the same barber/hairdresser is also associated with increased risk of infection presumably through direct contamination via instruments. The data on gender risk is confusing as some studies have shown increased susceptibility in male children and others with no difference between the two genders.

African-type hair is also more associated with certain infections such as *T. tonsurans* although carriage, e.g. isolation in culture of colonies of organisms from the scalp without clinical signs of infection is seen equally across ethnic groups [9].

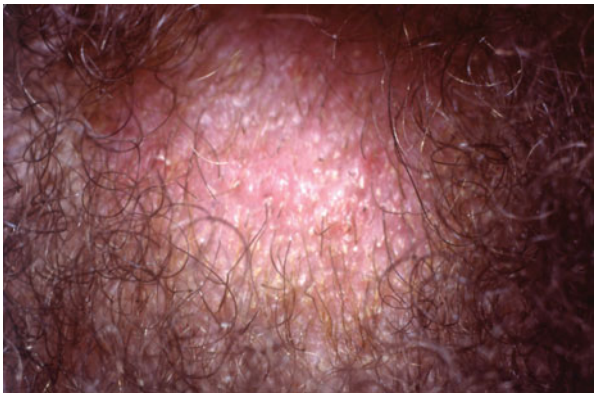

Fig. 9.2 Tinea capitis – ectothrix infection caused by *Microsporum canis*

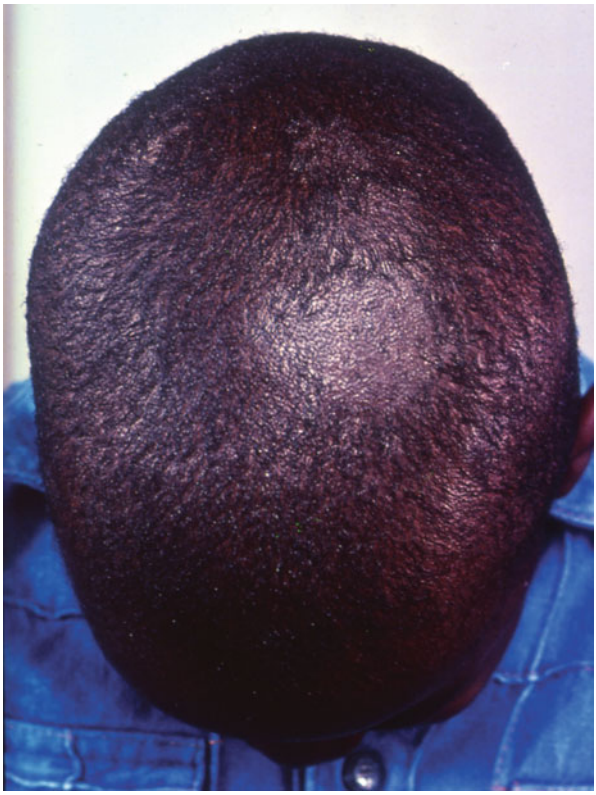

Fig. 9.3 Tinea capitis – endothrix infection caused by *Trichophyton yaoundei (*now reclassified as a variant of *T. violaceum)*

Infection caused by those fungi originating from animals is associated with exposure to the animal although this may not be direct as arthrospores may remain viable in farm building or fences over many months. The main human and animal organisms involved are seen in Table 9.1.

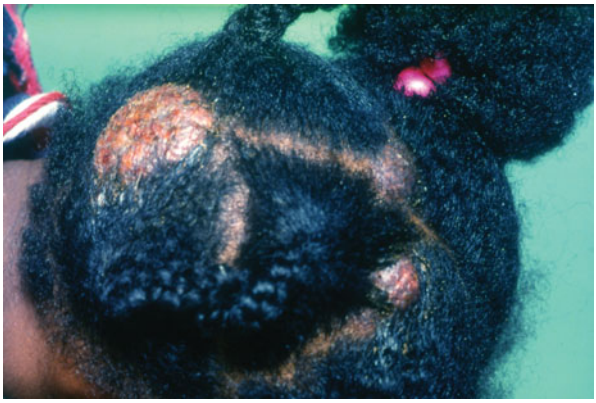

Fig. 9.4 Kerion caused by *Trichophyton tonsurans*

Table 9.1 Common causes of tinea capitis and their distribution

Organisms	Type of infection	Geographic area
Zoophilic		
Microsporum canis	Ectothrix	Global
Trichophyton verrucosum	Endothrix	Europe mainly
Anthropophilic		
Trichophyton tonsurans	Endothrix	USA, Canada, Caribbean, UK, Europe, West and East Africa, Brazil, Mexico
T. violaceum	Endothrix	East Africa, Middle East, Indian subcontinent
M. audouinii	Endothrix	Europe, West Africa
T. schoenleinii	Favus	Rare – Ethiopia, southern and north Africa, Middle East

9.4 Geographic Distribution (Table 9.1)

The worldwide distribution of dermatophytes causing scalp ringworm is determined by (a) the presence of suitable hosts, in the case of animal infection, and (b) geographic location of patients in anthropophilic cases.

Transmission is fairly simple to explain in the case of zoophilic infection as infection occurs where there is a suitable infected animal host [1, 2]. There are underlying variations where there are pockets of hyperendemic infection, for instance, associated occasionally with breeding establishments for cats or dogs where there has been a high incidence of infection. The slow elimination of *T. verrucosum* in cattle in Europe through control measures such as immunisation has resulted in a decline in the case numbers of this infection due to this organism. Although there have been regional changes in the major causes of tinea capitis due to anthropophilic infections, their exact distribution is subject to variation with time where there is movement of populations. The most obvious example is *T. tonsurans* which until the early 1970s was endemic at a low level in many parts of the world,

and it appeared from time to time as cause of sporadic infection. As described above, there has been spread of infection through the USA and Canada particularly in children with black hair type [10]. The infection has more recently spread to the Caribbean islands and also to Europe mainly in inner city areas. In London, for instance, it is currently the dominant cause of tinea capitis [11]. Further, there has been spread to both West and East Africa as well as to South America and Brazil. Other infections have remained more stable such as *T. violaceum* in East Africa and the Indian subcontinent although there is some evidence to suggest that where *T. tonsurans* is introduced into a new area, it may replace the local endemic cause of tinea capitis. In many areas, scalp infection is subject to local changes and, for instance, it may become endemic in a particular school but not in other areas.

Favus, while once common in Europe and the USA, has largely disappeared although it may still be seen in some populations, e.g. Ethiopia. The reason for this change is not clear. But as it is known to result in scarring alopecia and it is clinically distinct, it is likely that patients will present for treatment at an earlier stage. In many countries where it is endemic, there are distinct local words to describe favus different to those used for other types of tinea capitis.

9.5 Pathogenesis

In experimental infection in animals, infection of hair can generally only be initiated if the scalp epidermis is abraded, i.e. there is some form of local epidermal damage. However, in human infection, it is not clear if trauma plays any role in establishing infection. If it is the case, the trauma is likely to be minor as it is seldom recorded by patients. From animal models, it is known that specific dermatophyte genes are switched on in the early phases of hair infection [12–14]. These include protease genes such as subtilisins in *M. canis*, but others such as sulphur transporters appear to be involved. Invading fungal hyphae expand to form a modified structure or hair penetration organ on the cortex of the hair shaft, and beneath the site of this structure, there is a microscopic evidence of loss of keratin. Hyphae penetrate the scalp hair cortex and can proliferate within the central core of the hair shaft. Depending on the organism, arthrospores, which become the future propagules of infection, are formed either within the hair matrix (endothrix) or on the outer surface of the hair (ectothrix). The effect of fungal proliferation is to induce fracture of the hair shaft which is generally closer to the scalp epidermis in the endothrix infection. Clinically in these infection, the hair may break at scalp level leaving clinical appearances often referred to as a black dot or black dot ringworm. Ectothrix infection leads to hair shaft fractures 1–5 mm above the scalp surface.

Immunity to hair infection in experimentally infected animals occurs 2–3 weeks after the onset of infection [12]. However, in naturally acquired infections in both animals such as cats and humans, effective immunity may be delayed. The inflammation caused by the development of an immune response is variable but may result in the development of a severely infiltrated patch of scalp leading to the

formation of massive granulation tissue and infiltration of leucocytes leading to the formation of pustules – a kerion. This is more likely to occur with zoophilic infection in humans, but anthropophilic infections may also lead to severe inflammation in some cases. In the majority of cases of anthropophilic, the inflammation is mild, and in some cases barely visible, the reason for this is not clear although the existence of T cell immunological modification as described with other dermatophyte infections although likely remain unproven [12]. However, the result of this apparent lack of inflammatory response is that naturally occurring infections, again particularly those caused by anthropophilic fungi, are often slow to heal and may last for months without clinical change.

9.6 Clinical Features

The classical features of dermatophyte infection in the scalp result from a combination of hair loss or alopecia and inflammation presenting with scaling and erythema, with or without surrounding infiltration of the scalp [15]. Generally as described above, zoophilic infection leads to more prominent inflammatory changes with marked erythema, itching, hair loss and scaling. These changes are distributed in well-circumscribed patches across the scalp although in some cases the area involved is greater than 10 cm in size. On close inspection, the hair shafts, where the hair has broken, appear slightly swollen and greyish in colour.

In endothrix infection, the same changes occur, but the range of clinical variation is more extensive. In some children, the infection may only present with scaling of the scalp which is then difficult to distinguish from eczema or seborrhoeic dermatitis [15, 16]. The presence of erythema is also variable. Kerion may occur but is less frequent than in ectothrix infections. Hair shafts usually fracture closer to the scalp than in ectothrix infections and may give the appearance of a swollen hair shaft within the follicle – black dot ringworm. Sometimes, one or more patterns dominate, and these have been described as black dot type, seborrhoeic dermatitis-like or grey scale forms of infection. However, these terms are simply describing the main clinical features seen in each case.

In most patients, resolution is not accompanied by any scarring. This is not always the case with kerion as there may be destruction of hair follicles. Even so, there is a surprising degree of recovery in children as the scarred area contracts over succeeding months to leave a smaller area of hair loss. In elderly patients, a pseudopelade reaction has been rarely associated with adult-type tinea capitis, but it is not clear whether this is a reaction to the dermatophyte or whether these patients have pseudopelade with secondary fungal scalp infection.

The clinical appearance of favus is distinct again as here the invading fungus only survives for a short period in the scalp hair and degenerates leaving air channels in the hair shaft which may be weakened but not broken. The second distinctive change is that there is a massive influx of neutrophils at the opening of the hair follicle with keratin debris resulting in the formation of an inflammatory crust which appears as

a grey-coloured mass over the affected area, the scutulum. This has a stale odour. The inflammatory infiltrate also affects the upper dermis and follicle areas, and there is considerable scarring alopecia after the infection leading to permanent hair loss. Infection may also persist for years and, in some cases, into adult life.

In adults, infection of the scalp is rare [17]. However, it has been recorded although it appears to be commoner in females. The inflammatory changes are often minimal, and scaling with patches of hair loss are the usual presentations. There are some reports that suggest that this is also more likely to occur in HIV-positive individuals.

In addition to tinea capitis, patients with scalp infection may also have other lesions of dermatophytosis caused by the same organism usually on the face or upper trunk. Similar changes may be found in their contacts.

9.7 Carriage

Carriage is the term used to describe the presence of the organism in culture from the scalp in individuals who have no signs of infection [16, 17]. It is usually assumed that this occurs because of contamination of scalps by arthrospores shed from other infected patients in the vicinity, e.g. children in the same class. Longitudinal studies have shown that many of those designated as carriers may lose the positive cultures if the scalp is resampled after 6 months suggesting that this is a form of temporary carriage. However, the validity of this definition has been questioned particularly for *T. tonsurans* infections where careful examinations of apparently unaffected children may reveal that a small number of hairs are affected raising the possibility that hair shaft infection occurs but is of a very limited extent in some individuals. The importance of this observation is that if a carriage occurs without hair shaft invasion, then use of topically applied shampoos such as ketoconazole will be effective in preventing subsequent invasion. However, if there is invasion, then oral antifungal therapy is the only viable option for treatment. Ultimately, it is important to examine the siblings or contacts of infected patients very carefully to ensure that there is not a limited infection without significant symptoms.

9.8 Differential Diagnosis

The differential diagnosis of tinea capitis includes other scalp conditions prevalent in children with hair loss. These include seborrhoeic dermatitis or dandruff, eczema, psoriasis, discoid lupus erythematosus and lichen planus. The clinical diagnosis is not easy, and some of these conditions can be confused with tinea capitis which serves to re-enforce the importance of confirming the diagnosis by laboratory methods [18].

9.9 Laboratory Investigations

Treatment of tinea capitis should wherever possible be based on an accurate diagnosis confirmed by laboratory investigation [18, 19].

9.9.1 Wood's Light

Hairs infected with the majority of *Microsporum* species that cause tinea capitis fluoresce with a greenish colour under filtered ultraviolet light or Wood's light. This is important not just as a direct diagnostic clue, but it also serves as a good method to identify infected hair which can then be removed gently for laboratory tests. As usual with this method, it is important to ensure adequate darkness before using the light as the fluorescence may be faint. *Microsporum canis* causes fluorescence as *M. audouinii* does, but it is fainter with *M. gypseum.* The crusts in favus appear yellowish. *Trichophyton* species in hair infections do not produce fluorescence.

9.9.2 Direct Microscopy

This is a very useful diagnostic procedure as direct microscopy of plucked hairs can show the presence of organism and the site of formation of arthrospores in scalp hairs, and it also provides a partial diagnosis on which to base treatment; identification of an endothrix infection on microscopy in many cities in the USA and Europe suggest that the infection is most likely to be due to *T. tonsurans*. It is important to use 5–10% potassium hydroxide solution and to examine the specimen after 10–15 min of incubation. If the examination is delayed, it may be more difficult to discern the relationship between arthrospores and the shaft of the hair and to distinguish between endo- and ectothrix infections. It is not usually necessary to use a fluorescent whitener, such as calcofluor.

Hair samples are taken by gently scraping the scalp of the lesion with a blunt scalpel or the reverse side of the scalpel blade. The samples needed for diagnosis should contain hair rather than skin scales. Removing scalp hair in children is always difficult particularly with a kerion as they may find this painful. Using Wood's light may help as if the infection fluoresces the hair shaft illuminated can be removed simply without exerting pressure. However, if it remains difficult, a moistened swab may be a better approach as this will generally pick up infected hair. A useful alternative is a disposable tooth brush which will both remove loose hair and also can be used to inoculate a culture plate directly. The brush comes with a case, and it can be labelled and sent directly to the laboratory.

9.10 Culture

Cultural examination is carried out as with other dermatophytes on the Sabouraud dextrose agar containing antibiotics such as penicillin and streptomycin as well as cycloheximide.

The organisms generally take 2 weeks to grow sufficiently for identification although *T. verrucosum* may be slower. Dermatophytes are identified using conventional laboratory criteria.

At present, there are no commercially available systems for the diagnosis of scalp infections using molecular tools although there are a number of investigational studies of these methods, and they will become more important over the next few years [20].

9.11 Treatment

Complete treatment of tinea capitis has three main objectives: the treatment of the patient, where appropriate, screening of contacts and prevention of spread of the infection [19]. This involves assessment of the infection and initiation of the correct therapy and in the case of family's careful examination of siblings to check whether they have concurrent infection. It is the best practise in anthropophilic infections to use standard diagnostic tools such as culture and examination of scalp brushes to prove the presence of carriage or infection in family members. If a zoophilic infection is suspected, animal contacts and other children in the household should be screened for similar infection, as they may have been exposed to the same source. Laboratory-confirmed contacts, whether carriers or infected cases, should be treated. Hence, it is important as part of the clinical assessment of the patient with tinea to identify the likely cause as this will show whether the infection is anthropophilic or zoophilic. A second reason for pursuing laboratory confirmation is that it will also help the physician select the optimum antifungal.

9.12 Topical Antifungals

These have no role in the management of tinea capitis. Very little is known about the penetration of topically applied agents applied to hair, and there are no antifungal formulations that have been designed to achieve high hair shaft levels. It is unlikely that any formulation can produce growth inhibition or achieve fungicidal activity. One study compared miconazole with Whitfield's ointment showed that patients receiving topical treatment showed some improvement including some negative cultures and only during therapy, but these were not as great as that expected with oral antifungals, and the patients relapsed subsequently [21].

Topical antifungals are often used as adjunct to oral therapy to reduce the frequency of positive cultures from hair during the early stages of therapy and thereby reduce the risk of dissemination to other children [22]. The antifungals used for this have mainly been shampoo formulations such as selenium sulphide and ketoconazole shampoos.

9.13 Oral Treatments

9.13.1 Griseofulvin

The oldest effective treatment for tinea capitis is griseofulvin given in a dose of 10–15 mg/kg daily, generally for a treatment period of 6–8 weeks [19, 22]. Griseofulvin is fungistatic in vitro against the fungi that cause tinea capitis, and its mode of action is through the inhibition of the formation of intracellular microtubules.

Griseofulvin was one of the earliest antifungal drugs to be introduced, and there are therefore few comparative clinical trials against placebo. However, for most organisms that cause tinea capitis, it is clinically and mycologically effective although there are some patients with infections such as those caused by *T. tonsurans* infections who require longer courses of treatment, e.g. 12 weeks or who may fail to respond. Often, a higher dosage of 20 mg/kg/day is recommended for *T. tonsurans* infections. Clinical trials comparing terbinafine with griseofulvin show equal efficacy in *Microsporum* and *Trichophyton* infections, but responses for *Trichophyton* species appear to be faster [23, 24].

Higher doses of griseofulvin given as single treatments may also be effective as part of a mass drug administration (MDA) for community-based treatments of against certain endothrix infections, e.g. *T. violaceum* [25]. Dosage regimens vary, but 1,000 mg given as a single dose or 500 mg as a stat dose followed by a repeat dose after 2 weeks have been two such regimens employed to reduce population frequency of infection. However, these dosage regimens are not recommended for routine use.

There are both tablet and oral solution formulations of griseofulvin. However, in Europe, the liquid paediatric formulation of griseofulvin is difficult to obtain in many countries. Alternative formulations can be imported, or some pharmacies suspend crushed tablets of griseofulvin in a suitable liquid base.

9.13.2 Terbinafine

Terbinafine is an allylamine drug with broad-spectrum in vitro antifungal activity, including all the dermatophytes that cause tinea capitis in vitro. For tinea capitis, terbinafine is available in a 250 mg tablet form. In some countries, a paediatric tablet is available in 125 mg. The normal daily dose is 250 mg for adults. In children,

the treatment regimen used is based on weight: <20 kg 62.5 mg/day, 20–40 kg 125 mg/day and >40 kg 250 mg/day.

Terbinafine is effective against a range of fungi that cause tinea capitis, and there are a number of studies comparing terbinafine with griseofulvin [23, 24, 26–28]. The recommended treatment period for most infections is usually 4 weeks. Studies have shown that in infections caused by *T. tonsurans*, 4 weeks of treatment is required although shorter periods are sometimes effective. In a study of *T. violaceum* infection, there was no difference in mycological cure rates at follow-up when 1 week of treatment was compared with 2 or 4 weeks therapy [27]. A meta-analysis of studies comparing terbinafine with griseofulvin showed that terbinafine was as effective at treatment durations of up to 2–4 weeks for *Trichophyton* infections compared with griseofulvin for 6–8 weeks [28]. However, the responses of *Microsporum* species were slower than those of *Trichophyton*, and in some patients, there appears to be no response [29–31]. A suggested solution is the use of higher doses of terbinafine, and for *Microsporum* infections, doubling the normal daily dose is therefore generally advised. Unfortunately, the choice of drug cannot be guided by in vitro sensitivity treating as isolates of *Microsporum* are not significantly less sensitive to terbinafine in vitro than those of *Trichophyton* [31].

In some countries, a paediatric terbinafine tablet 125 mg is available. Otherwise, for smaller children, it is necessary to break the 250 mg tablets, which may be scored (depending on source). Other formulations of terbinafine have been developed including a solution but these are not generally available.

9.13.3 Itraconazole

Itraconazole is an orally active triazole antifungal which is fungistatic in vitro against the main causes of tinea capitis. Itraconazole comes in three main formulations: a capsule containing pelleted itraconazole (Sporanox), an oral solution containing itraconazole in cyclodextrin and a newer solid formulation with better absorption (Subacap or Lozanoc). The latter has not been tested specifically for tinea capitis. The currently available cyclodextrin solution is not approved for paediatric use in dermatophytosis.

For tinea capitis, itraconazole is generally given in doses of 3–5 mg/kg daily [32–35]. The regimens used have varied between 3 and 5 mg/kg daily for 4–6 weeks. Efficacy rates have varied from over 80 to 40 % in one study. There is no evidence that *Microsporum* and *Trichophyton* species causing tinea capitis differ in their responses to itraconazole. A pulsed regimen using intermittent treatment with 5 mg/kg/day for 1 week in every 3 weeks has been evaluated in a small number of children. Usually two to three pulses were found to be necessary for most infections [35].

The pelleted capsule formulation is difficult to use in children on a dose per weight basis as it involves opening and dividing the contents of capsules.

9.13.4 Fluconazole

Fluconazole is an orally active triazole antifungal, and there are both capsule and liquid formulations. The drug is active against a range of fungi including those dermatophytes that cause tinea capitis. The recorded doses that have been used in tinea capitis have ranged from 1.5 to 6 mg/kg daily and up to 8 mg/kg weekly [36–38]. Fluconazole appears to be equally effective against a range of different organisms in tinea capitis including both *Trichophyton* and *Microsporum* species. It also appears to be as effective as griseofulvin [39, 40]. The oral solution may be particularly helpful in young children.

9.13.5 Other Azoles

The other azoles in current usage such as posaconazole or voriconazole have not been used in tinea capitis.

9.14 Treatment in Practice

The generally recommended treatment for tinea capitis due to *Trichophyton* species is terbinafine which is given for a 1 month period as the initial treatment of choice. For *Microsporum* infections, there remains a choice which is largely determined by drug availability of griseofulvin for 6–8 weeks; itraconazole or terbinafine at double normal doses are alternatives, and these are also given for 6–8 weeks. Generally children are also treated with a topical agent to reduce the risk of spread in the early phase of oral treatment. Ketoconazole shampoo, twice or three times weekly for the first 2 weeks of oral therapy, is often used.

9.15 Treatment of Carriers

Topically applied ketoconazole and selenium sulphide in shampoos reduce the frequency of positive cultures form both infected children and those carrying the organism without clinical evidence of infection [41–43]. Topical antifungals are recommended for carriers defined as asymptomatic children with positive brush cultures. If scalp brushes produce very heavy growth of fungus, it is likely that the children have a true but asymptomatic infection, and these should be treated as infected patients by using the appropriate oral antifungal.

9.16 Schools

While infected children pose a potential risk to those who are not infected, the method by which the organisms spread from head to head is not known, e.g. aerosol and direct contact. Exclusion from school is not recommended in most countries. Recent work suggests that treating whole classes where there is a risk of infection with ketoconazole shampoo to prevent infection is not effective.

9.17 Kerion

In kerions the same treatment regimens as those used for children with other forms of tinea capitis are used. But it is often necessary to continue antifungal therapy for longer periods, e.g. 12–16 weeks. Few clinical trials have addressed the issue of the use of systemic corticosteroids in kerions, and advice is based on anecdotal experience. However, one trial which examined the value of oral corticosteroids found that they made no difference to clinical and mycological response rates [44].

Removal of surface crusts is often helpful as it relieves itching and secondary infection. It can be painful; therefore, the procedure is best carried out after soaking the crusts with lukewarm water or saline applied topically in the form of moistened dressings. The softened crusts can then be gently teased away. Secondary bacterial infection, usually due to *Staph. aureus,* should be treated with antibiotics such as flucloxacillin; however, pustules in the lesions of kerion are more often a response to the fungus rather than bacteria. The application of an antifungal cream with anti-Gram-positive bacterial activity such as miconazole, clotrimazole or econazole may allow the scalp to heal and prevent formation of new crusts.

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