The aim was to describe the mycological and clinical data in children diagnosed with tinea capitis in a hospital setting in Stockholm. Information concerning demography, symptoms, mycology and treatment were obtained, retrospectively, from medical records of all children up to 15 years of age diagnosed with tinea capitis during two 3-year periods, 1989–1991 and 1999–2001, at the Pediatric Dermatology Unit of the Karolinska Hospital in Stockholm. Between 1989 and 1991, five children were diagnosed with tinea capitis. Between 1999 and 2001, there were 92 children, the vast majority (86%) being of foreign extraction, mostly African (83%). *Trichophyton violaceum* was the most prevalent pathogen, affecting 68% of the children. Of the anthropophilic infections, 62% were linked to relatives. In 71% of all positive cultures, microscopy was positive. The most common clinical findings were scaling of the scalp (80%), itching (54%) and patches of alopecia (52%). The treatment consisted of the oral antimycotics terbinafine (*n* = 48) or griseofulvin (*n* = 49). During the last decade there has been an increase in tinea capitis in Stockholm, most commonly caused by *Trichophyton violaceum*, corresponding with the increased immigration from Africa. Spread within the family seems to be of importance, and family members are preferably screened in an effort to prevent continued transmission. It is important to bear the diagnosis of tinea capitis in mind, especially as, untreated, some cases can develop permanent alopecia and may also cause further spreading of this infection.

**Keywords** dermatophyte infection, mycosis, scalp, *Trichophyton violaceum*

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**Introduction**

Dermatophyte fungi can invade and colonize skin, hair and nails. They are subdivided into three main groups: anthropophilic species that affect only humans, zoophilic species, whose normal hosts are animals but which may also infect humans, and geophilic species, that are found in soil and are only occasionally pathogenic.

Tinea capitis is the term used to describe a dermatophyte infection of the hair follicles in the scalp and surrounding skin. Formerly, tinea capitis in the Stockholm area was caused mainly by zoophilic species, but during the past two decades other dermatophytes causing tinea capitis have been observed [1], probably due to increased immigration from other continents and increased traveling. Worldwide, the main anthropophilic pathogens presently causing tinea capitis show a variable geographic distribution. Thus for example, *Trichophyton tonsurans* is now the dominating fungus in urban areas in the UK and USA [2–4], whereas *Trichophyton violaceum* infection has dominated in North Africa and Asia [4]. Among the zoophilic species, *Microsporum canis* is the dominating pathogen and until recently one of the major causative agent of tinea capitis in western countries.

The present aim was to analyze, retrospectively, the clinical and mycological features of all children up to 15 years of age diagnosed with tinea capitis in a hospital setting in Stockholm. Information concerning demography, symptoms, mycology and treatment were obtained, retrospectively, from medical records of all children up to 15 years of age diagnosed with tinea capitis during two 3-year periods, 1989–1991 and 1999–2001, at the Pediatric Dermatology Unit of the Karolinska Hospital in Stockholm. Between 1989 and 1991, five children were diagnosed with tinea capitis. Between 1999 and 2001, there were 92 children, the vast majority (86%) being of foreign extraction, mostly African (83%). *Trichophyton violaceum* was the most prevalent pathogen, affecting 68% of the children. Of the anthropophilic infections, 62% were linked to relatives. In 71% of all positive cultures, microscopy was positive. The most common clinical findings were scaling of the scalp (80%), itching (54%) and patches of alopecia (52%). The treatment consisted of the oral antimycotics terbinafine (*n* = 48) or griseofulvin (*n* = 49). During the last decade there has been an increase in tinea capitis in Stockholm, most commonly caused by *Trichophyton violaceum*, corresponding with the increased immigration from Africa. Spread within the family seems to be of importance, and family members are preferably screened in an effort to prevent continued transmission. It is important to bear the diagnosis of tinea capitis in mind, especially as, untreated, some cases can develop permanent alopecia and may also cause further spreading of this infection.
years of age diagnosed with tinea capitis during two 3-year-periods at the Pediatric Dermatology Unit of the Karolinska Hospital in Stockholm, Sweden.

Material and methods

Data were collected from patient records of children aged 0–15 years with diagnosed tinea capitis during two 3-year periods, 1989–1991 and 1999–2001. All the patients were seen at the outpatient clinic of the Pediatric Dermatology Unit, Department of Dermatology, Karolinska Hospital, Stockholm, Sweden. The patients were identified with a computerized billing database at our department. This identifies all visits containing either the Herman Classification of Diseases (used 1989–1991) or the International Classification of Diseases (ICD-10) code (used 1999–2001) for tinea capitis. The diagnosis had either been confirmed with a positive dermatophyte culture and microscopy or been assumed to be very probable from the clinical picture combined with epidemiological indications. Data were collected from the patient’s records concerning demography, source of infection, clinical symptoms, results of microscopy and culture for dermatophytes and treatment. Scalp samples included scales taken with either a toothbrush or a curette from affected areas and hairs. In addition, abnormal hairs were also plucked when possible. Dermatophyte hyphae were detected by microscopy using 10% KOH and the fluorescent dye blancophor for staining. Species were identified by microscopy using 10% KOH and the fluorescence. Species were identified on cultures using standard methods. For final identification macroscopic and microscopic observations and biochemical tests were performed [5,6]. Cultures were examined on surface and reverse for color, growth rate, texture and diffusible pigment. Trichophyton violaceum was identified by typical colony morphology and pigmentation, and by microscopic morphology of scotch tape preparations from Sabouraud agar. Casein hydrolysis was observed on Bromcresol purple casein glucose agar. Growth of Trichophyton violaceum was stimulated in vitro by thiamine and inositol. Trichophyton soudanense, Trichophyton mentagrophytes, Trichophyton verrucosum, Trichophyton tonsurans and Microsporum canis were likewise speciated by macro- and micromorphology, biochemistry, vitamin test and casein hydrolysis according to standard methods [5,6].

Results

Patients

Between 1989 and 1991, five children (four girls and one boy) with a median age of 7.5 (range 3–12) years, were identified with tinea capitis. Between 1999 and 2001, 92 children (48 girls and 44 boys) with a median age of 6.1 (range 0.5–14) years were so identified. The majority, 67 of 97 (69%), had been referred, for example, from general practitioners or pediatricians. In 41 of 67 (61%) of these referred patients tinea capitis was suspected. Other suggested diagnoses were ‘unspecified rash of the scalp’ (n = 11), psoriasis (n = 6), eczema (n = 5), defluvium/alopecia (n = 5), infectious rash (n = 2), seborrhoic eczema (n = 2) and infestation/parasite infection (n = 2). The rest of the children came directly to us without referrals, for symptoms shared with siblings whom we had recently diagnosed with tinea capitis.

Ethnicity

In the material from 1989 to 1991, four were children of Eritrean immigrants, and one had Swedish origin. In the children with tinea capitis between 1999 and 2001, the vast majority (86%) were of foreign extraction, most frequently with parents who were immigrants from Africa (83%), especially Somalia (41%) and Eritrea (25%). Only 3% were of non-African foreign extraction (Turkey, Syria and Bangladesh). Whether or not the patients were born in Sweden was rarely documented in the records. Only 9% were of Swedish parentage. In 5%, the parental country of origin was not documented.

Dermatophyte species

In the material from 1989 to 1991, Trichophyton violaceum was identified in three cases, Microsporum canis in one case and in one case the culture was negative. The T. violaceum infections were all seen in children of Eritrean extraction.

Table 1 shows the frequency of different dermatophyte species among children diagnosed with tinea capitis between 1999 and 2001. The most prevalent dermatophyte, T. violaceum, was cultured from 55 of
75 patients (73%) with African origin, most notably Somali, where 87% were infected with this dermatophyte. *T. violaceum* was also found in one of three cases with Asian origin and in two of nine children with Swedish. In the remaining two cases, the parental country of origin was not documented. One of the children with Swedish origin infected with *T. violaceum* also had the diagnosis hereditary keratodermia. The second most prevalent dermatophyte was *Trichophyton soudanense*, which was found in 12 cases (14%), all in children of foreign extraction. Three cases of *Trichophyton tonsurans* were found, one of them in a child of Swedish parentage. The most prevalent zoophilic dermatophyte was *Trichophyton verrucosum*, which was found in eight cases (9%). *Microsporum canis* was isolated in four cases. In three cases cultures were negative.

**Clinical presentation**

In the material from both 3-year-periods, the duration of skin signs and symptoms reported at the first visit to us averaged 6.8 (range 0.5–84) months. The clinical presentation varied. The most common findings were discrete to pronounced scaling of the scalp (80%), followed by itching (54%) and patches of alopecia (52%). The clinical presentation of these symptoms did not differ between the different dermatophytes. Kerion (i.e. a deep purulent inflammatory follicular dermatophyte infection causing a sharply demarcated, painful, indurated, boggy tumefaction) was seen in six patients. It was caused by *Trichophyton soudanense* (*n* = 2), *T. violaceum* (*n* = 1), *T. mentagrophytes* (*n* = 1), *T. verrucosum* (*n* = 1) and *Microsporum canis* (*n* = 1). A persistent cicatrical alopecia occurred in two of the children, and was caused by pronounced inflammatory infections due to *T. mentagrophytes* and *T. verrucosum*. In addition to tinea capitis, some children also had involvement of the face (12%) or other skin areas (24%).

**Source of dermatophyte infection**

Data from both 3-year-periods showed that the origin of the anthropophilic dermatophyte infections was in 62% linked to relatives of the child; the majority of the affected children (57%) having siblings with concomitant or recent tinea capitis. At that time dermatophyte screening was not routinely offered to siblings or relatives without any symptoms of dermatophytosis. Thus the actual number of siblings infected with dermatophytes might in fact have been much higher than observed. In the cases of zoophilic dermatophyte infection, animal contact was documented in three of four children infected with *Microsporum canis*, in one of eight cases with *T. verrucosum* and in the single case with *T. mentagrophytes*.

Twenty-nine patients had a history of staying abroad (less than 2 years previously); 24 of these were infected with anthropophilic dermatophytes, especially *T. violaceum* (*n* = 20).

**Microscopy and culture**

Between 1999 and 2001, 158 scalp skin/hair specimens were obtained for microscopy and/or culture. Combined microscopy on blancophor mounts and cultures was performed in 111/158 (75%) of the samples (Table 2).

**Treatment**

Data concerning treatment were collected from all patient records. Prior to being admitted to our clinic, many children had already received treatment, usually with topical glucocorticoids (*n* = 27), oral antibiotics (*n* = 14), or topical antifungics (*n* = 25), or a combination of these. Antifungal treatment was initiated in our department as soon as the diagnosis was established with positive dermatophyte microscopy and/or culture. Only in three cases was treatment initiated in spite of negative microscopy and culture, as here clinical presentation and epidemiological data very strongly suggested genuine tinea.

In total, 49 patients were treated with griseofulvin and 48 with terbinafine. In addition, ketoconazole shampoo was given in 55 cases (57%). Griseofulvin was given in an average dose of 12.7 mg/kg/day for an average of 3.3 (range 1.5–6) months and terbinafine in an average dosage of 5 mg/kg/day for an average of 1.4 (range 1–2.8) months. In five of 46 children treated with terbinafine, the treatment was extended beyond 4 weeks due to positive dermatophyte culture, in all cases *T. violaceum*, at end of the treatment. In one patient treated with griseofulvin, treatment was switched to terbinafine due to persistently positive culture after 10 weeks. The pathogen in this case was *T. tonsurans*. All the patients were considered cured after the end of treatment. Reinfections were seen after 6–12 months in two of the patients treated with griseofulvin and after 6

<table>
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<th>Table 2</th>
<th>Correlation between dermatophyte cultures and microscopy from samples obtained from children with tinea capitis in our department in a hospital in Stockholm, 1999–2001</th>
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<td><strong>Positive culture</strong></td>
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<td>Positive microscopy</td>
<td><em>n</em> = 54 (48.6%)</td>
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<td>Negative microscopy</td>
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months in one patient treated with terbinafine. Adverse side-effects were seen in three patients after a few days of treatment with terbinafine and consisted of vomiting, diarrhea and stomach-ache (n = 2) and unspecified rash and itch (n = 1). In those treated with griseofulvin, adverse side-effects were seen in four patients: vomiting (n = 2), nausea (n = 2) and stomachache (n = 1). None of these adverse events were considered serious. All patients, apart from those two who developed persistent cicatricial alopecia, healed without residual symptoms.

Discussion

Striking increases in tinea capitis caused by anthropophilic fungi have recently been observed in the western countries [1–4, 7–9]. Today most of these infections, previously rare in this part of the world, seem to occur almost exclusively in families of African–American descent. Tinea capitis is endemic in many African countries, as shown for instance in a recent study in southwestern Ethiopia. Dermatophytes were isolated in 33% of scalp samples from children either asymptomatic or with clinical symptoms, from urban and rural communities. Trichophyton violaceum constituted 97% of these infections [10]. In agreement with these observations, our study showed a striking predominance of scalp infection caused by T. violaceum among children of eastern African extraction. This probably mirrors the increased immigration from such areas to Sweden in recent years. In 1989, for example, 551 persons originally from Somalia were living in Sweden, and in 1999 there were 13 467 (personal communication; Statistics Sweden).

Tinea capitis is seen mainly among pre-adolescent children and is the most frequent manifestation of dermatophyte infections in children, while adult patients seldom have clinical symptoms but may sometimes be carriers [11]. A familiar clinical observation is that spontaneous cure is common at puberty. The reason for this remains unclear, though it is probable that increased fungistatic action of triglycerides in the sebum produced following puberty is of importance [2].

Dermatophytes are transmitted through infected skin scales and hairs shed from infected human or animal hosts. The exact methods of spread of the anthropophilic dermatophytes are unclear. Clustering of cases can be seen among family members, as illustrated in our study, where approximately half of the patients had siblings affected, suggesting spreading either through close person-to-person contact with an infected relative and probably through sharing of combs, towels or bed linen. The asymptomatic carrier state seems to play a role in the transmission of tinea capitis especially within the family. The amount of spore load in the scalp of an asymptomatic individual also seems to be an important vector of transmission [12]. Spreading may occur at kindergartens and schools [1, 2] and the appearance of anthropophilic dermatophytes in children of Swedish parentage in our study supports this hypothesis. Why children of African extraction seem to be at higher risk of infection with these fungi than others remains unclear. One may speculate about factors of importance, such as genetic susceptibility, lower socioeconomic status, social habits and contact with relatives living in areas with endemic tinea capitis. Children of immigrants tend to be infected with dermatophyte species common in their country of origin, irrespective of how long they have lived in their adopted country [13]. Future studies of the pattern of ethnically determined infections will probably elucidate these questions. Further, certain hairdressing practices such as the repeated use of electrical cutters without cleaning them, or the use of hair oil, hypothetically could be important for transmission; although a recent study concluded that hair-grooming practices did not appear to play a major role in the acquisition of tinea capitis [14].

It is important for the physician to bear the diagnosis of tinea capitis in mind, as the clinical symptoms can be vague and easily neglected, or the condition can be misdiagnosed as other dermatological conditions (e.g. eczema, psoriasis or bacterial pyoderma). There is hair loss, alopecia areata may be a differential diagnosis. Diagnoses suggested by referrals from general practitioners and pediatricians in our present study support this notion. A missed correct diagnosis and the use of treatment with topical glucocorticoids can mask the condition (‘tinea incognito’) or worsen it. Also, if tinea capitis goes untreated, scarring alopecia may occur, as well as a risk of further spreading of the infection. The diagnosis of tinea capitis must be based on mycological investigation with microscopy and culture. Even though our study suggests that microscopy is less sensitive than cultivation, if positive it enables the physician to start treatment promptly. To obtain adequate specimens and to perform microscopy requires knowledge and skill and we encourage referral to a dermatologist. Our finding of a high incidence of tinea capitis in siblings suggests that all siblings should be examined. If one culture is positive then asymptomatic siblings should also be treated. Dermatophytes have been cultured from combs and can remain viable for a long time [15], which is why cleaning of combs, brushes and electrical cutters may prevent re-infection.

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Treatment should be initiated only after the diagnosis is established. An oral antifungal drug is required for effective therapy [16,17], but simultaneous use of a topical antifungal is assumed to decrease the contagiousness of the disease [18]. In Sweden, terbinafine is the recommended drug, as griseofulvin has recently been withdrawn from the market. Griseofulvin used to be the mainstay treatment of tinea capitis in Sweden, as it has been for more than four decades in the USA, where now there are some reports of a troublesome decrease in sensitivity to this drug [8]. Terbinafine is still available as a licensed product. The recommended dosage for terbinafine is 62.5 mg/day for weights between 12 and 19 kg, 125 mg/day between 20 and 39 kg and 250 mg/day for 40 kg or more. It is given for 4 weeks [19], although a 2-week terbinafine treatment may suffice as has been shown in some recent studies [19,20]. In certain instances the terbinafine treatment has to be prolonged, especially when infection with *Microsporum canis* occurs, as this dermatophyte seems to be less sensitive to terbinafine [22–24]. Many authors still recommend griseofulvin 10–15 mg/kg/day for 3 months as the treatment of choice, which our study confirms as an effective therapy, although some recent authors recommend a higher dosage of 20–25 mg/kg/day for 8 weeks because of the increase in treatment failures [25]. We found that both terbinafine and griseofulvin were well tolerated. One patient treated with terbinafine developed a rash, and in such cases one should consider the possibility of an id-reaction instead of an adverse cutaneous event.

Today, all children at our clinic treated for tinea capitis are followed-up not only clinically, but also with mycological investigation to ascertain healing of the scalp infection.

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**References**