Outbreak of Tinea capitis caused by Microsporum ferrugineum in Thailand

Wanee Wisuthsarewong¹, Angkana Chaiprasert² & Suchitra Viravan¹

¹Department of Pediatrics; ²Department of Microbiology; Faculty of Medicine, Siriraj Hospital, Mahidol University, Bangkok 10700, Thailand

Received 9 April 1996; accepted in revised form 16 August 1996

Abstract

There was an outbreak of *Tinea capitis* at the Pak-kred Home for Mentally and Physically Handicapped Babies, Bangkok, Thailand in 1993. One hundred and thirty-eight cases were diagnosed as tinea capitis based on clinical signs and positive laboratory investigations. The results of Wood's light examination, KOH preparation and fungal culture were positive in 89.9, 75.9 and 27.4% respectively. The non-inflammatory form had a higher rate of positive KOH and culture than in the inflammatory form. *Microsporum ferrugineum* was the major pathogen (66.7%) and most of its infections (80.4%) caused a non-inflammatory type of tinea capitis. Griseofulvin, in a dosage of 10–15 mg/kg/day and selenium sulfide shampoos, yielded an 84.8% cure rate within 14.9 weeks. No recurrence or obvious adverse reactions were observed.

Key words: diagnosis, Microsporum ferrugineum, tinea capitis, treatment

Introduction

Tinea capitis is a dermatophytosis of the scalp, eyebrows and eyelashes caused by the species of the genera *Microsporum* and *Trichophyton*. It is almost exclusively a disease of children and adolescents. It comprises 16.2% of superficial fungal infection in Thai children of which 16.4% is caused by *M. ferrugineum* [1]. Tinea capitis is highly contagious among children. It is most likely transmitted by fomite spread or by direct contact and it usually occurs among individuals during epidemics [2]. There was an outbreak of endemic tinea capitis in the Pak-kred Home for Mentally and Physically Handicapped Babies, Bangkok, Thailand in 1993. Our study was carried out to determine the causative dermatophytes and to evaluate the diagnostic methods and treatments.

Materials and methods

The study was conducted from March 1993 to August 1995 at the Pak-kred Home. The children with abnormal scalp lesions such as hair loss, scales, crust, erythema or pus were examined. Clinical signs were scored

on a scale from 0 to 4 corresponding to absent, mild, moderate and severe respectively. Wood's light examination, hair extraction by forceps and scalp scraping with sterilized scalpel blades were made from the lesions. The specimens were taken to the laboratory and direct microscopic examination was carried out by using 10% potassium hydroxide mounts in order to check for the presence of hyphae and arthroconidia. A portion of the scrapings and hairs was cultured on Sabouraud dextrose agar supplemented with chloramphenicol (50 mcg/ml) and cycloheximide (500 mcg/ml). They were incubated at room temperature. The fungal colonies that appeared were sequentially identified by staining with lactophenol cotton blue and slide culture. Identification was mainly based on microscopic morphology and cultural characteristics. The cultures were examined once a week for 4 weeks before being recorded as negative. After a diagnosis was established, griseofulvin 10-15 mg/kg/day and 2.5% selenium sulfide shampoos twice a week were started. Physical examinations and laboratory tests were subsequently performed in the next 2 weeks and then every 4 weeks. Ketoconazole (3-6 mg/kg/day) was used in those cases that did not respond to griseofulvin therapy.

Table 1. Scoring of clinical signs.

Score	Hair loss		scale	
	Number	%	Number	%
0	14	10.2	6	4.3
1	8	5.8	7	5.1
2	82	59.4	83	60.1
3	1	0.7	3	2.2
4	33	23.9	39	28.3
Total	138	100	138	100

Results

There were 138 cases diagnosed as tinea capitis out of 437 by clinical signs with, at least, one positive laboratory tests. The tests were the following: (A) the infected area fluoresced a bright green color when exposed to Wood's light, (B) the infected hair and/or skin scraping from the scalp showed a sheath of arthroconidia around the hair shaft and/or hyphal strands within the hair shaft in KOH preparations, and (C) positive isolation of dermatophytes from infected hairs and scalp skin scraping on Sabouraud dextrose agar. The ages of the patients ranged from 9 to 161 months (mean age = 69 months). There were 80 males and 58 females with the male: female ratio = 1.4:1. The mean body weight was 12.8 kg (range 4-25 kg) with 31.9% of cases having a body weight less than 10 kg. Most patients had moderate hair loss and scales (Table 1).

There were 29 cases (21%) of the inflammatory type. More than 90% of the patients had inflammation scores in the mild to moderate range. Four children had associated tinea corporis and one had tinea faciei caused by the same dermatophyte. The sensitivities of Wood's light examinations, KOH preparations and fungal cultures are shown in Table 2. The culture results are shown in Table 3. The majority of the cases (66.7%) were caused by *M. ferrugineum*, most of which (80.4%) were of the non-inflammatory type.

Identifications of *T. rubrum*, *M. gypseum* and *T. mentagrophytes* were based on colonial morphologies and characteristic macroconidia and microconidia. *Microsporum ferrugineum* was identified by its mycological characteristics of slow growth rate and forming glabrous bright yellow colonies with waxy surfaces. It produced neither microconidia nor macroconidia on Sabouraud's dextrose agar (SDA). The old culture was heaped with folded surfaces. All tested isolates failed to sporulate on deficient media such as diluted

Table 2. Results of the laboratory investigations.

	Wood's light	КОН	Culture
All cases (138)			
total examined	542	469	469
positive	487	356	171
%	89.9	75.9	27.4
Microsporum ferru	gineum cases (92	2)	
total examined	436	386	386
positive	402	291	167
%	92.2	75.4	43.3
Non-inflammatory	cases (109)		
total examined	392	335	335
positive	349	261	130
%	89.0	77.9	38.8
Inflammatory cases	: (29)		
total examined	143	128	128
positive	131	89	38
%	91.6	69.5	29.7

Culture results	Number	%	
Microsporum ferrugineum	92	66.7	
Trichopyton rubrum	2	1.5	
M. gypseum	1	0.7	
T. mentagrophytes	1	0.7	
No growth of dermatophytes	42	30.4	
Total	138	100.0	

SDA. Typical faviform, mycelial elements and coarse hyphae with prominent crosswalls (bamboo hyphae) were seen in microscopic examination. *Microsporum ferrugineum* caused an ectothrix type of hair infection with small arthroconidia and infected hairs fluoresced bright green. Hair perforation and urease tests were not performed in our study.

Table 4. Comparison of Wood's light, KOH preparations and culture results.

	Wood's light	
	Positive	Negative
KOH preparations		
positive	315	38
negative	110	6
Cultures		
positive	167	6
negative	259	37

Table 5. Clinical improvement (weeks)

Clinical	Improvement	Cure
Hair loss	6.5 ± 3.6	9.7 ± 7.9
Scale	8.4 ± 6.9	14.9 ± 15.1
Pus	2.2 ± 4.2	$2.2\pm~4.2$
Crust	5.0 ± 1.3	5.0 ± 1.3
Erythema	5.5 ± 2.1	$5.5\pm~2.1$

Direct microscopic examinations and cultures were positive in 75.9 and 27.4% of all treated samples respectively. Wood's light examination was the most sensitive diagnostic method in all of the forms of infection in this study.

One hundred and seventeen patients (84.8%), who had received combined griseofulvin and selenium sulfide shampoos, were cured. In this group, 68.8% had clinical and laboratory cure in 12 weeks and 81.9% were cured in 16 weeks. Mean duration of treatment was 14.9 ± 13.6 weeks. Clinical improvement of pus, crust, erythema, hair loss and scale appeared in 2.2, 5.0, 5.5, 9.7 and 14.9 weeks respectively (Table 5). Seventeen patients, who failed to be cured in the 18week griseofulvin treatment period, were cured by ketoconazole in 21.9 \pm 18.8 weeks. Cases of kerion were successfully treated with topical dressings and oral griseofulvin without corticosteroids. Two patients were dropped because one had died from another disease and the second one was moved to another home for care. No recurrences or obvious adverse reactions were encountered.

Discussions

Microsporum ferrugineum was found to be the most frequent cause (66.7%) of the outbreak of tinea capitis in this study. A similar outbreak of this disease was reported by Kotrajaras et al in 1984 [3]. The non-inflammatory form is more common (80.4%) in *M. ferrugineum* infections. We found that the infections were predominant in boys as found in previous reports [4–6].

The Wood's light is a useful tool for the diagnosis of tinea capitis because the *Microsporum* species produce pteridine, a tryptophane metabolite, that accumulates in infected hairs and fluoresces when exposed to ultraviolet light [7]. Patients having tinea capitis caused by the *Microsporum* species, such as *M. ferrugineum*, can

be rapidly and accurately examined (92.2%) by this test for the presence of infection.

The traditional methods to obtain the specimen involve scraping the scalp and removing hair roots with forceps. Brush culture is an alternative technique that is easy to perform and safe in uncooperative children. It is a reliable method but its primary disadvantage is its cost [8], as a result we selected the traditional method. KOH preparations are also useful for the rapid diagnosis of tinea capitis. Fungal cultures provide definite confirmation of a clinical diagnosis and should be performed in all patients suspected of having this disease [9]. The sensitivity of culture in this study was quite low (27.4%). Its success depends on many factors such as proper specimen collection, preparation, history of previous treatment with antifungal agents and skill of the examiner [10]. There were many cases with positive KOH preparations or fungal cultures from the negative Wood's light cases. The reason for this may be that the disease may have been caused by other dermatophytes. The combination of these 3 procedures should be recommended for the diagnosis of tinea capitis. In concordance with many studies [4, 5, 10], the lesions with an inflammatory form had a lower rate of positive KOH and culture than the non-inflammatory form of tinea capitis.

Griseofulvin remains the mainstay of therapy in tinea capitis because it is safe. It inhibits fungal mitosis by disrupting mitotic spindle formation. Griseofulvin is primarily fungistatic because it does not affect the resting spores [2]. Adverse reactions include disturbance of the gastrointestinal tract, mild pruritus, neutropenia, leukopenia, generalized maculopapular rash, proteinuria, photosensitivity, urticaria and hepatotoxic reaction [4]. The side effects may decrease by taking the drug at mealtime [11]. As a general guideline, 10-15 mg/kg/day of griseofulvin was given in a single dose because of its relatively long half life [7]. Shampoos containing ketoconazole or selenium sulfide is also helpful [12–13]. Most cases (84.8%) were cured with the combined therapy of griseofulvin and 2.5% selenium sulfide shampooing. In clinically persistent cases, inadequate GI absorption of griseofulvin rather than a resistant strain may be the cause [11]. Recalcitrant infections are best treated with ketoconazole or the newer triazoles [13–14]. Ketoconazole is generally fungistatic but may be fungicidal at higher concentrations [15]. Hepatic dysfunctions caused by ketoconazole have been widely reported such as a transient increase in serum aminotransferase and symptomatic hepatitis [16]. It may be used in patients who are

unable to take griseofulvin or those who are unresponsive to griseofulvin therapy but it should not replace griseofulvin as the drug of choice [9]. Ketoconazole is more rapidly absorbed and produces higher concentrations in plasma when administered to infants and children in a suspension form than as crushed tablets [17]. Unfortunately, only 200 mg tablets are available. Patients who did not show clinical improvement, or who showed deterioration or persistence of positive tests were changed to treatment with ketoconazole. Seventeen children were given ketoconazole at a dosage of 3–6 mg/kg/day without any obvious side effects.

There have been many reports about alternative drugs for the treatment of tinea capitis. Itraconazole is an orally active triazole derivative with broad spectrum of activity. It is better tolerated with a lower potential for liver toxicity than ketoconazole. It is more active than ketoconazole but has equal efficacy as compared to griseofulvin for infections caused by M. canis [18-19]. Terbinafine, one of a new class of allylamines, can be used as a treatment for tinea capitis because of its unique fungicidal action. It encompasses the combination of high cure rate, short duration and good tolerability. It is very active against M. ferrugineum with 100% cure in 2 weeks [20]. Terbinafine should be recommended only for children with a body weight of more than 10 kg. Approximately 2-3% of children have adverse effects, However, it is well tolerated and safe for an 8-week treatment [20-21]. Itraconazole and terbinafine were not used in this report because of financial problems and the mean body weight of the patients which was 12.8 kg with 31.9% of the children weighing less than 10 kg.

In kerion, the scalp should be kept clean. Compresses may help in preventing secondary bacterial infection and encourage resolution of the lesions. Short course systemic corticosteroids is indicated in severe inflammatory reaction. Most of the inflammations in patients are mild to moderate so in general treatments are effective without corticosteroids.

Tinea capitis is most common in low socioeconomic groups. The more children there are in a family, the greater the crowding and therefore the disease has a higher chance of spreading. There is higher frequency of fungal transmission among children who have close and prolonged physical contacts. Shed hairs, scales and shared fomites, such as combs, brushes, hats, towels, pillows, beds, sleeping mats, have been implicated in the transmission of fungal elements; and also the effect of playing together [22–24]. Such close direct and indirect contacts accelerate the general diffusion of the disease in homes. Household members should be cultured regardless of the presence or absence of clinical findings or subjective complaints [25]. Asymptomatic carriers should be treated because they may act as reservoirs of the fungus [26–27]. Removal, restriction and disinfection of infected fomites are usually indicated [28]. No positive cultures were obtained from the fomites or the environment of a Home for the Disabled in a previous study [3].

Raising the standard of living in general, avoidance of over-crowding at this Home and using their own fomites would certainly reduce the prevalence of the infection.

References

- Wisuthsarewong W, Kullavanijaya P, Viravan S. Superficial fungal infections in Thai children. Thai J Pediatr 1995; 36: 230–8.
- Krowchuk DP, Lucky AW, Primmer SI, McGuire J. Current status of the identification and management of tinea capitis. Pediatrics 1983; 72: 625–31.
- Kotrajaras R, Chongsathien S, Rojanavanich V. Fungal infection in children in Thailand. 4th International Congress of Pediatric Dermatology Edited by Urabe K, Kimura M, Yamamoto K, Ogawa H. 1987: pp.197–203.
- Laude TA, Shah BR, Lynfield Y. Tinea capitis in Brooklyn. Am J Dis Child 1982; 136: 1047–50.
- Sehgal VN, Saxena AK, Kumari S. Tinea capitis: A clinicoetiologic correlation. Int J Dermatol 1985; 24: 116–9.
- Venugopal PV, Venugopal T. Tinea capitis in Saudi Arabia. Int J Dermatol 1993; 32: 39–40.
- Ginsburg CM. Superficial fungal and mycobacterial infections of the skin. Pediatr Infect Dis J 1985; 4: S19–23.
- Hubbard TW, Triquet JM. Brush-culture method for diagnosing tinea capitis. Pediatrics 1992; 90: 416–8.
- Tanz RR, Hebert AA, Esterly NB. Treating tinea capitis: Should ketoconazole replace griseofulvin ? J Pediatr 1988; 112: 987–91.
- Gan VN, Petruska M, Ginsburg C. Epidemiology and treatment of tinea capitis: ketoconazole vs griseofulvin. Pediatr Infect Dis J 1987; 6: 46–9.
- Rudolph AH. The diagnosis and treatment of tinea capitis due to *Trichophyton tonsurans*. Int J Dermatol 1985; 24: 426–31.
- Allen HB, Honig PJ, Leyden JJ, McGinley KJ. Selenium sulfide: Adjunctive therapy for tinea capitis. Pediatrics 1982; 69: 81–3.
- 13. Odom R. Pathophysiology of dermatophyte infections. J Am Acad Dermatol 1993; 28: S2–7.
- Lukacs A, Korting HC, Lindner A. Successful treatment of griseofulvin-resistant tinea capitis in infants. Mycoses 1994; 37: 451–3.
- Legendre R, Steltz M. A multi-center, double-blind comparison of ketoconazole and griseofulvin in the treatment of infections due to dermatophytes. Rev Infect Dis 1980; 2: 586–91.

- Martinez-Roig A, Torres-Rodriguez JM, Bartlett-Coma A. Double-blind study of ketoconazole and griseofulvin in dermatophytoses. Pediatr Infect Dis J 1988; 7: 37–40.
- Ginsburg CM, McCracken GH, Olsen K. Pharmacology of ketoconazole suspension in infants and children. Antimicrob Agents Chemother 1983; 23: 787–9.
- Elewski BE, Tinea capitis: Itraconazole in *Trichophyton ton-surans* infection. J Am Acad Dermatol 1994; 31: 65–7.
- Lopez-Gomez S, Palacio A, Cutsem JV, et al. Itraconazole versus griseofulvin in the treatment of tinea capitis: A doubleblind randomized study in children. Int J Dermatol 1994; 33: 743–7.
- Jones TC. Overview of the use of terbinafine (Lamisil[®]) in children. Br J Dermatol 1995; 132: 683–9.
- Nejjam F, Zagula M, Cabiac MD, Guessous N, Humbert H, Lakhdar H. Pilot study of terbinafine in children suffering from tinea capitis: evaluation of efficacy, safety and pharmacokinetics. Br J Dermatol 1995; 132: 98–105.
- Ajao AO, Akintunde C. Studies on the prevalence of tinea capitis infection in Ile-Ife, Nigeria. Mycopathologia 1985; 89: 43–8.

- Schwinn A, Ebert J, Muller I, Brocker EB. *Trichophyton rubrum* as the causative agent of tinea capitis in three children. Mycoses 1995; 38: 9–11.
- Snowden MS, Loder L, Alexander WJ. Infectious alopecia in a child day-care center. JAMA 1985; 254: 3038.
- Hebert AA, Head ES, MacDonald EM. Tinea capitis caused by Trichophyton tonsurans. Pediatr Dermatol 1985; 2: 219–23.
- Vargo K, Cohen BA. Prevalence of undetected tinea capitis in household members of children with disease. Pediatrics 1993; 92: 155–7.
- Williams JV, Honig PJ, McGinley KJ, Leyden JJ. Semiquantitative study of tinea capitis and the asymptomatic carrier state in inner-city school children. Pediatrics 1995; 96: 265–7.
- Shtayeh MS, Arda HM. Incidence of dermatophytosis in Jordan with special reference to tinea capitis. Mycopathologia 1985; 92: 59–62.

Address for correspondence: Dr. Suchitra Viravan Department of Pediatrics, Faculty of Medicine, Siriraj Hospital Bangkok 10700, Thailand.

Phone: 662-4197008-9; Fax: 662-4121871