JOURNAL OF CLINICAL AND DIAGNOSTIC RESEARCH

How to cite this article:
AL SAMARAI A G M. TINEA CAPITIS AMONG IRAQI CHILDREN: PUBLIC HEALTH IMPLICATION. Journal of Clinical and Diagnostic Research [serial online] 2007 February [cited: 2007 Dec 3]; 6:476-482.
Available from
http://www.jcdr.net/back_issues.asp?issn=0973-709x&year=2007&month=December&volume=1&issue=6&page=476-482&id=127

ORIGINAL ARTICLE / RESEARCH

Tinea Capitis among Iraqi Children: Public Health Implication

Al SAMARAI A G M

ABSTRACT

Background: Recent observations on tinea capitis cases in Iraq suggest that there has been a change in the pattern of infection, with a recent and significant rise in the incidence of infection due to anthropophilic dermatophytes.

Objectives: The purpose of this study was to investigate the incidence rate and identify factors that might affect the spread of infection of tinea capitis in Iraq.

Patients and Methods: There were 40,384 children from 6 to 16 years of age included in the study. The children were from two different geographical areas.

Results: The prevalence rate of clinically diagnosed tinea capitis was 1.25%, while the mycological prevalence rate was 0.86%. The above difference between clinical and mycological prevalence rates was statistically significant ($P < 0.001$). In addition, the prevalence rate was significantly higher ($P < 0.05$) in males (1.08%) as compared to females (0.96%) and in rural areas (2.06%; $P < 0.0001$) as compared to urban (0.62%) areas. Animal contact and family history of tinea capitis were important risk factors in disease transmission. The prevalence of tinea capitis was 86.3% in age group of 6–10 years ($P < 0.0001$). The non-inflammatory type of infection was found in 87.5% of cases ($P < 0.0001$). The frequency of isolation of anthropophilic dermatophytes was reported in 73.3% of cases ($P < 0.001$). The predominant causative agent was *Trichophyton tonsurans* (26%), followed by *T. verrucosum* (25.1%). The isolated species was distributed in urban and rural areas.

Conclusion: This study indicated a shift in the epidemiologic patterns of tinea capitis in Iraq. *T. tonsurans* was the most prevalent cause of tinea capitis in Iraq. There was a significant role for influence of infection for sex, residence, animal contact, and hair type.

Keywords: Tinea capitis, Iraq, children

Introduction

Tinea capitis has a worldwide distribution [1] and tends to be epidemic and endemic in areas where crowding and imperfect conditions for personal hygiene seem to favour its spread and persistence within the community [2]. The prevalence of infection varies considerably with geographical location and climatic condition [3].

Corresponding author: Prof. Dr. Abdul Ghani Mohamed Al Samarai. Department of Medicine, College of Medicine, Tikrit University, P O Box 45, Al Yarmouk ST., Tikrit, Iraq. Mobile: 07901755258, 07705871455; e-mail: galsamarrai@yahoo.com

Many studies that have been conducted throughout the world revealed the increased prevalence and shift in the epidemiology of tinea capitis [4–10]. Geographic locations are important factors in the type of infection seen [3–15].

Objectives

The objectives of this study are to

1. determine the demographic and clinical characteristics of tinea capitis in Iraq;
2. identify the etiological dermatophytes for tinea capitis in Iraq.

Study Population

A cross-sectional community study was performed, and two groups of schools were chosen. The first group was distributed in urban areas, and the second group was selected from different rural areas in Tikrit and Kirkuk governorates. For Tikrit survey two groups of schools were chosen: the first group from urban areas and the second from differing rural areas in the vicinity of Tikrit. Three boys' schools, three girls' schools, and two large mixed schools were randomly selected from five urban areas of Tikrit. Six schools from four rural villages were chosen: two boys', two girls', and two mixed schools. Tikrit survey was conducted between September 1994 and April 1995, and a total of 4461 children were included in the survey. The second survey was carried out in Kirkuk from September 1996 to May 1997. In urban areas 131 schools were selected, of them 24 were girl's schools, 20 boys' schools, and 87 mixed schools. In rural areas 20 schools were selected: eight boys' schools, three girl's schools, and nine mixed schools. A total number of 35,923 children were included in the survey. The criteria for their selection were the same as described previously [14],[15]. The children were all preadolescent school-age children. Their ages range from 6 to 16 years. Since both surveys were with the same design, the data of both were combined together and reanalyzed to get an idea about the pattern of tinea capitis in Iraq. All the students in the school were examined, so a total of 40,384 students were included in the study. The scalp of each child was thoroughly examined in all areas for evidence of scaling, crusting, follicular inflammation, hair loss, and erythema. Clinical diagnosis of tinea capitis was done according to criteria of Arnold et al. [11].

Sample Collection

In all suspected cases, hairs and scales were collected for mycological examination by a conventional technique. Scale scrapings were collected from at least two areas with a number 15 sterile surgical blade, and approximately 12 hair stumps (roots) were pulled out with sterile epilator forceps. Both hairs and scales were placed in a clean, labelled envelope and sent to the laboratory for investigation.

Laboratory Procedures and Identification

Media used for primary isolation of dermatophytes, maintenance, and identification were according to conventional reported procedures [16]. Species were identified using conventional method, which emphasised colony morphology, microscopy, and other miscellaneous tests. Cultures were examined macroscopically for morphology, texture, and colour from the top and reverse sides of the plate. Then using a sterile straight loop the colony was examined by placing a sample on a drop of lactophenol solution on a clean glass slide. The matted mycelial mass was teased or separated with dissecting needles to facilitate microscopical observation. The preparation was then covered by a cover slip and examined under the microscope for the presence of microconidia, macroconidia, and other structures. Every positive growth obtained was subcultured on two Sabouraud plates, one with added yeast and the other with added sodium chloride. The inoculated plates were incubated for 2 weeks at 28°C to further stimulate the chlamydospores. After identification was completed, the plates were kept refrigerated at 4°C for a maximum period of 1 month.

In vitro hair perforation by certain dermatophytes was used for further species identification. This test was used to differentiate certain species of *T. mentagrophytes*, which can penetrate hair in vitro, from *T. rubrum*, which cannot. This simple procedure involved the use of baby hair in Petri dishes to which 25 ml of sterile distilled water and 2–3 drops of 10% sterilised yeast extract were added. Several colony fragments served as inoculums, and the inoculated dishes were incubated in the dark at 25°C. After 3 weeks of incubation, hair segments overgrown with mycelium were removed from the dishes with sterile forceps,

placed in a drop of lactophenol cotton blue mounting fluid, and examined under the microscope. Penetrated hair segments were identified by wedge-shaped perforations.

Corn meal agar tests and potato dextrose agar tests were used to differentiate *T. rubrum* and *T. mentagrophytes*, the first showing deep pigmentation and the second no pigmentation. *Microsporum auduoinii* was differentiated by absence of growth on polished rice grains, inability to penetrate hair in vitro, and culture characteristics.

Statistical Analysis

Chi-square test was used for estimation of significance.

Results

The prevalence rate of clinically diagnosed tinea capitis was 1.25%, whereas the mycological prevalence rate was 0.86%. The above difference between clinical and mycological prevalence rate was statistically significant ($P < 0.001$). In addition, the prevalence rate was significantly higher ($P < 0.05$) in males (1.08%) as compared to females (0.96%) and in rural areas (2.06%; $P < 0.0001$) as compared to urban (0.62%) areas ([Table/Fig 1]).

[Table/Fig 1] Prevalence rates

Variable	Total number tested	Number of positive cases	Prevalence rate (%)
Clinical	40,384	504	1.25
Mycological	40,384	350	0.86
Female	14,298	104	0.96
Male	26,086	246	1.08
Urban	33,402	206	0.62
Rural	6982	144	2.06

Tinea capitis cases were more common in urban (59%) than in rural (41%) areas ($P < 0.0001$) and in males (70%) than in females ($P < 0.05$) ([Table/Fig 2]). The case frequency was 93.5% in children with history of animal contact, whereas the corresponding value was 31.3% in children without history of animal contact ($P < 0.0001$).

Normally, the frequency of infection was reported in all cases with a family history of tinea capitis; however, the infection was reported in 63% of children without any family history of disease ($P < 0.0001$) ([Table/Fig 2]).

[Table/Fig 2] Demographic and exposure characteristics of cases

Character	Cases (%) [No.]
Urban	59 [206]
Rural	41 [144]
Male	70 [246]
Female	30 [104]
Patients in contact with animals	93.5 [289]
Patients with no contact with animals	31.3 [61]
Patients with positive family of infection	100 [62]
Patients with no family of infection	63 [288]
Age in years:	
6–10	86.3 [302]
11–16	13.7 [48]

The prevalence of tinea capitis was 86.3% in age group of 6–10 years ($P < 0.0001$). The prevalence of tinea capitis was lower in straight hair (33%), as compared to curly hair (67%, $P < 0.001$) ([Table/Fig 3]). Frontal site was the predominant (46%) site involved. The non-inflammatory type of infection was found in 87.5% of cases ($P < 0.0001$). The scaling was found to be the predominant clinical characteristic (59.1%). Seborrhoid was the most common clinical form (80.8%). Favus presented in 1.7% of cases (six cases); five of them were caused by *T. violaceum* and in one case the causative dermatophytes were unidentified.

The frequency of isolation of anthropophilic dermatophytes was reported in 68.1% of cases ($P < 0.001$). The predominant causative agent was *T. tonsurans* (27.6%), followed by *T. verrucosum* (26.7%). The isolated species was distributed in urban and rural areas.

In rural areas, the predominant isolate was *T. verrucosum* (33.3%), followed by *T. tonsurans* (21%) ([Table/Fig 4]). In urban areas, *T. tonsurans* forms the predominant (32.5%) isolate, followed by *T. mentagrophytes var interdigitale* (26.8%) ([Table/Fig 4]).

[Table/Fig 3] Clinical feature of cases

	Case percent	Case number
Hair type		
Straight	33.0	115
Curly	67.0	235
Infection site		
Frontal	46.0	161
Occipital	24.3	85
Parital	19.4	68
Diffuse	6.6	23
Vertex	3.7	13
Infection type		
Scaling	59.1	207
Crusting	8.9	31
Alopecia	20.3	71
Oedema	1.4	5
Inflammation	10.3	36
Clinical forms		
Seborrhoid	80.8	283
Gray patch	4.3	15
Kerion	10.3	36
Black dot	2.8	10
Favus	1.7	6
Ecology*		
Zoophilic	31.9	108
Anthropophilic	68.1	224
Non-inflammatory	88.3	309
Inflammatory	11.7	41

*The only identified dermatophytes numbers are = 329.

The isolated species were distributed in both sexes. In male, the predominantly isolated species was *T. tonsurans* (27%) followed by *T. verrucosum* (25.2%), whereas in female the predominantly isolated species was *T. verrucosum* (30.5%), followed by *T. tonsurans* (29.5%) ([Table/Fig 4]).

Discussion

This study has shown that the clinical prevalence rate of tinea capitis, in a large sample of primary school children in two different Iraqi governorates, was 1.25%. However, the mycological prevalence rate was significantly lower than clinical prevalence rate. Sex seems to play a role in infection, as the prevalence rate was more in male as compared to female. This male predominance of infection was also reported for other geographical areas [9],[17–21].

The high rate of tinea capitis in male may be attributed to the easy implantation of spores because of short hair [22] and frequency of sharing comb, brushes, and cups [23]. This study shows that the prevalence of tinea capitis was more in rural than in urban areas. This finding is in agreement with that reported before for different geographical areas in Iraq [24–26]. The high prevalence rate of the disease in rural areas may be due to low standard of living, low standard of health education, overcrowding, poor hygiene, close personal contact, animal contact, and no separation between animal house and their houses [26]. Contact with animals seems to be an important risk factor that increases prevalence rate, as this study indicated higher significant rate in children with animal contacts than in children without animal contacts. This was also reported for other geographical areas outside Iraq [18],[19],[22].

[Table/Fig 4] The fungal isolation frequency

Organism	Frequency percent				
	Male	Female	Urban	Rural	Total
T. tonsurans	27.0	29.5	32.5	21.0	27.6
T. mentagrophytes var interdigitale	22.6	20.0	26.8	14.8	23.4
T. verrucosum	25.2	30.5	22.2	33.3	26.7
T. rubrum	7.3	5.3	3.6	9.6	6.7
T. violaceum	7.3	9.5	8.2	7.4	7.9
T. mentagrophytes var mentagrophytes	8.0	3.2	4.1	10.4	5.2
M. auduoinii	2.6	2.1	1.5	3.7	2.4

T. = *Tricophyton*; M. = *Microsporum*.

Presence of tinea capitis in other family members also acts as an important source of infection. This is due to intra-familial transmission, and the home might be more likely a source of infection than the school [26],[27]. These results are reported by others [17],[27–30]. Hair type influences the infection rate, and thus the cases were more in children with curly hair. This could be due to difficulty in hair washing and cleaning, which provides stable and less hazardous environment for dermatophytes [1],[26],[31],[32]. The predominant clinical form was seborrhoid, and the mostly affected site was frontal area of the scalp.

Concerning aetiology, the zoophilic dermatophytes were found as the cause of infection in 31.9% of cases. These zoophilic organisms are acquired through contact with infected animals, since a large number of families in the study areas were in contact with animals. Although human infections with zoophiles are of the supportive type, animal infection may be clinically silent and thus the animals serve as asymptomatic carriers [33]. Anthropophilic dermatophytes were more predominant cause of tinea capitis. This may be due to the fact that anthropophilic infections are often epidemic in nature [33], and children are population at risk of anthropohilic tinea capitis [33],[34]. Thus, prevention control programs for tinea capitis in children may need to take this in consideration when designing and performing their program. Primary school health programs for tinea capitis control must concentrate on primary school students who demonstrate higher prevalence rate (ages 6–10 years).

The predominantly isolated dermatophyte was ere *T. tonsurans*, and this finding indicated a shift of pattern in the aetiology of tinea capitis in Iraq. It is well known that the types of dermatophytoses present may vary depending on geographical areas, and multiple factors are responsible for this fact [2],[33]. Certain strains of dermatophytes are endemic to specific geographical areas [33]. Because of patients travelling to and from these areas, resident dermatophytes may remain restricted geographically or become more cosmopolitan [12],[33]. In addition, it appears that dermatophytes endogenous to certain areas of the world have adapted themselves to their human hosts in these areas and vice versa [12],[33].

The location of dermatophytosis is partially dependent on climatic conditions of the area and the customs of the resident population [2],[12],[33]. Tinea capitis is impeded in areas where the population is using hair oils [33]. There is some evidence to suggest that certain human population may be genetically more susceptible to particular dermatophyte infections [26],[35]. This community-based prospective survey indicated that there is a change in the causative fungi of tinea capitis. During the period from 1948 to 1980, *T. schoenleinii* was the primary cause of tinea capitis in Iraq [24],[36–38]. However, other dermatophytes such as *T. violaceum* [39] and *M. canis* [40] found to be the causative agents of tinea capitis in Baghdad in 1962 and Basrah in 1983, respectively. Furthermore, a reported study in 1999 for Basrah indicated that *T. violaceum* was the predominant cause of tinea capitis [41].

The shift in the cause of tinea capitis in Iraq from *T. schoenleinii* to *T. tonsurans* leads to a change in typical clinical presentation and epidemiology of the disease. Thus, tinea capitis due to *T. tonsurans* becomes endemic within Iraqi community, and several factors may favour its spread. *T. tonsurans* is able to exist as an asymptomatic carrier state in children [32]. Similarly, the atypical presentation of some infections may lead to misdiagnosis, allowing further spread to occur. Other factors such as short hair, tight braiding, and oil application have been associated with the spread [32]. In addition, *T. tonsurans* tinea capitis was found in individuals with low socioeconomic group and crowded living conditions [14],[33]. An interesting finding of this study is that favus was caused by *T. violaceum* and not by *T. schoenleinii*.

Dermatophytes causing tinea capitis have a geographic variation, and the most common cause of the disease varies according to the governorate studied. In rural areas, *T. verrucosum* was the common cause of tinea capitis. In urban areas, the most common cause is *T. tonsurans*. So it is important for the clinicians to be cognizant of the prevalent organism responsible for tinea capitis in his or her geographical areas. Although, these trends are relatively stable, they are not absolutely so and may change with time [42].

Sex seems to have an influence on the predominant cause of tinea capitis in Iraq nowadays. However, many studies have demonstrated a significant male predominance in infection caused by microorganisms [2],[4],[9],[10],[33], whereas with *T. tonsurans* the male/female infection rate is equal in childhood [33],[42]. In addition to host and geographic factors, the virulence of the infecting organisms must be considered, and the important point is that strain to strain differences may exist for the same dermatophyte such as in case of *T. mentagrophytes* and *T. rubrum* strains [33].

In conclusion, there were significant differences in prevalence rate in regards to gender, residence, animal contact, hair type, clinical characteristics, and clinical forms. The significant differences were detected in frequency distribution of dermatophytes as a cause of tinea capitis and their distribution in rural and urban areas and indicate a shift in epidemiologic patterns. However, the conclusion emerged from this study demonstrate the demographic and clinical characteristics of the disease in Iraq as a whole country.

References

[1] Rippon JW. The changing epidemiology and emerging patterns of dermatophytes species. Curr Top Med Mycol 1985;1:208–34.

[2] Al-Sogair S, Hay RJ. Fungal infection in children: tinea capitis. Clin Dermatol 2000;18:679–85.

[3] Shah HG, Amin AG, Kavinde MS, Patel GD. An analysis of 2000 cases of dermatophytoses. Indian J Pathol Bacteriol 1975;18:32–7.

[4] Kradjen S. Dermatophytes: epidemiological and clinical features. In: Kane J, Summerbell R, Kradjken S, Sigler L, Land G, editors. Laboratory handbook of dermatophytes: a clinical guide and laboratory handbook in dermatophytes. Belmont: Star Publishing Company; 1997. p. 5–32.

[5] Ghannoum M, Isham N, Hajjeh F, et al. Tinea capitis in Cleveland: survey of elementary school students. J Am Acad Dermatol 2003;48:189–93.

[6] Chinelli P, Sofiatti A, Nunes R. Dermatophyte agents in the city of Sao Paulo from 1992–2002. Rev Inst Med Trop Sao Paulo 2003;Sep/Oct:1–8.

[7] Labato MN, Vugia DJ, Frieden IJ, Tinea capitis in California children: a population based study of growing epidemics. Pediatrics 1997;99:551–4.

[8] Gargoom AM, Elyazachi MB, Maki S, Duweb GA. Tinea capitis in Benghazi, Libya. Int J Dermatol 2000;39:263–5.

[9] Popoola TOS, Ojo DA, Alabi RO. Prevalence of dermatophytosis in junior secondary school children in Ogum State, Nigeria. Mycoses 2006;49:499–503.

[10] Gumusay T, Ilkit M. Epidemiology of tinea capitis in Ceyhan district, Adana in Cukurova region, Turkey. Mycoses 2006;49:346–9.

[11] Arnold HL, Odom RB, James WD. Andrews diseases of the skin. 8th ed. Philadelphia: WB Saunders Company; 1990. p. 319–24.

[12] Champion RH, Burton JL, Burns DA, Breathnach SM, eds. Rook/ Wilkinson/Ebling textbook of dermatology. 6th ed. Edinburgh: Blackwell Science; 1998. p. 1277–376.

[13] Al-Samarai AGM, Fathi HI. Tinea capitis in Iraq: clinical types. J Islamic Med Assoc [North Am] 1999;5:236–8.

[14] Fathi HI, Al-Samarai AGM. Tinea capitis in Iraq: laboratory results. East Mediterr Health J 2000;6:138–48.

[15] Al Samarai AGM, Al Kadi ZF, Fathi HI. Epidemiological characteristics of tinea capitis in Kirkuk, Iraq: a community based study. Med J T U 2001;7:1–8.

[16] Al-Doory Y. Laboratory medical mycology. Philadelphia: Lea and Fabiger; 1980. p. 229–45, 360–75.

[17] Hussain I, Aman S, Haoon TS. Tinea capitis in Lahore, Pakistan. Int J Dermatol 1994;33:355–7.

[18] Venugopal PV, Venugopal TV. Tinea capitis in Saudi Arabia. Int J Dermatol 1993;32:39–40.

[19] Al-Fozan AS, Nanda A. Dermatophytosis of children in Kuwait. Int J Dermatol 1994;33:690–3.

[20] Woodgger A. *Trichophyton tonsurans* infections in New Zealand. Mycosis 1993;3:1–15.

[21] Hay RJ, Clyton YM, Silva NDE, Midgley G, Rossor E. Tinea capitis in South East London. A new pattern of infection with public health implications. Br J Dermatol 1996;135:955–8.

[22] Sehgal VN, Saxena AKK, Kumari S. Tinea capitis: a clinic etiologic correlation. Int J Dermatol 1985;24:116–9.

[23] Reid BJ, Shimkin MB, Blank F. Study of tinea capitis in Philadelphia using case and control groups. Public Health Rep 1986;83:497–501.

[24] Rahim GF. A survey of fungi causing tinea capitis in Iraq. Br J Dermatol 1966;78:213–8.

[25] Ghani HM, Yahya M. Incidence of dermatophyte infection in Nineveh province. Ann Coll Med Mosul 1997;10(1):43–8.

[26] Fathi HI, Al-Samarai AGM. Incidence of tinea capitis in Iraq among primary school children. Eastern Med Health J 2000;6:128–37.

[27] Maleville J, Moulinier C, Taieb A, et al. Course of the dermatophytic spectrum in tinea capitis. A propos of 124 cases seen in Bordeaux. Ann Dermatol Venereol 1986;113(1):25–9.
[28] Malhotra YK, Garg MP, Kanwar AJ. A school survey of tinea capitis in Benghazi, Libya. J Trop Med Hyg 1979;82(3):59–61.
[29] Bhakhtariziam C, Shafi M, Mehta MC, Kumar R, Nagah M, Crew AS. Tinea capitis in Tripoli. Clin Exp Dermatol 1984;9(1):84–8.
[30] Stiller MJ, Klein WP, Dorman RI, et al. Tinea corporis gladiatorum: an epidemic of *Trichophyton tonsurans* in student wrestlers. J Am Acad Dermatol 1992;27:632–3.
[31] Howard R, Frieden IJ. Tinea capitis: new perspectives on an old disease. Semin Dermatol 1995;14:2–8.
[32] Sharma V, Hall JC, Knapp JF, et al. Scalp colonization by *Trichophyton tonsurans* in an urban pediatric clinic. Asymptomatic carrier state. Arch Dermatol 1988;124:1411–3.
[33] Martin AJ, Kobayashi GS. Fungal diseases with cutaneous involvement. In: Fitzpatrick TB, Eisen AZ, Wolff K, Freedberg IM, Austen KF, editors. Dermatology in general medicine. Vol 2, 4th ed. New York: McGraw-Hill; 1993. p. 2421.
[34] Malhotra YK, et al. A study of tinea capitis in Libya (Benghazi). Sabouraudia 1979;17:181–3.
[35] Ravine D, et al. Genetic inheritance of susceptibility to tinea imbricata. J Med Genet 1980;17:342.
[36] Shaikh Gumar AW, Guirges SY. Survey of etiological agents of fungal infections of skin. J Fac Med 1978;20:19–29.
[37] Junaid AJ, Rassam KH. Species of fungi causing dermatomycosis in Iraq. Iraqi Med J 1974;22:38–40.
[38] Yehia MM. Studies on dermatophytes in Mosul and vicinity. Mosul University of Mosul. College of Medicine, M Sc Thesis, 1980.
[39] Akrawi F, Rassam KH. Species of fungi which cause ringworm of the scalp in Iraq with a study of the action of griseofulvin on them in vitro. J Fac Med 1962;4:23–5.
[40] Al-Hashimi JM, Hussain I, Nair BK. Tinea capitis in Basrah, an exploratory study. Med J Basrah Univ 1980;2:107–19.
[41] Al-Duboon AH, Muhsin TM, Al-Rubaiy KK. Tinea capitis in Basrah, Iraq. Mycoses 1999;42:331–3.
[42] Laude TA. Tinea capitis in Brooklyn. Am J Dis Child 1982;136:1047.