Tinea Capitis

Maha Al Dayel, *
Iqbal Bukhari **

Abstract:

Tinea capitis is a common superficial fungal infection of the scalp, with a propensity for attacking hair shafts and follicles. The disease is considered to be a form of dermatophytosis caused predominantly by Trichophyton or Microsporum species. It is observed generally but not exclusively in prepubertal children. Clinical presentations are variable, but the major clinical patterns are the gray patch, the seborrheic-like, the "black dot", the favic type, and the inflammatory tinea capitis with kerion and tiny pustules in the scalp. Diagnosis is aided by wood's light and confirmed by microscopic examination and culture. Treatment of tinea capitis requires the use of oral antifungal agents since topical treatment usually is ineffective. This review summarizes all data mentioned in the literature about tinea capitis with some clinical implications that would be useful for us as dermatologists.

Key Words:

Tinea Capitis, Dermatophyte infections, hair disorders

Introduction

Tinea capitis is a common skin disease caused by superficial fungal infection of the scalp, with a propensity for attacking hair shafts and follicles.[1-3] The disease is a form of dermatophytosis. Several synonyms are used, including ringworm of the scalp and *Tinea tonsurans*.[2-5] Dermatphytosis are classified into three genera namely *Trichophyton, Microsporum* and *Epidermophyton*. Tinea capitis is caused predominantly by *Trichophyton or Microsporum* species[5-7] On the basis of host preference and natural habitat, dermatophytes are also classified as anthropophilic, geophilic and zoophilic. The etiological agents of tinea capitis usually fall in the anthropophilic and zoophilic group.[4, 8] Tinea capitis is observed generally but not exclusively in prepubertal children.[8-10] Clinical presentations of tinea capitis are variable, but the major clinical patterns are the gray patch, seborrheic-like, "black dot", favic type, and inflammatory tinea capitis with kerion and tiny pustules in the scalp.[4, 5, 11, 12] The clinical diagnosis is aided by wood's light examination and confirmed by mycological examination for which hair stumps and scales are collected and examined microscopically using 10% potassium hydroxide solution, and cultured on modified Sabouraud's dextrose agar with cyclohexamide and chloramphenicol.[1, 4, 5, 12, 13] Treatment of tinea capitis requires the use of oral antifungal agents since topical treatment usually is ineffective.[5]

Historical background:

The disease was described in the earliest historic accounts along with other dermatophyte infections of the skin. The word "Tinea" literally refers to an insect larvae that feed on clothes and books that were thought by the Romans to be the cause of the infection.[8] Subsequently, it meant parasitic infestation of the skin. By the mid 16th century, the term was used to describe all diseases of the hairy scalp.[2,4] During the 1830's the work of Remak and Schonlein, followed by Gruby resulted in the culture of the causative agent of tinea infections of the beard and scalp.[2,4] Approximately 50 years later Sabouraud published his dissertation in classifications of dermatophytes and made other clinical and therapeutic observations.[16] For this work, Sabouraud is fairly considered the father of modern mycology. In the 1920s, the scientific studies of the dermatophytes by Benham and Hopkins formed the foundations of modern day medical mycology,[4,8] and in the 1950s, effective treatment of tinea capitis by griseofulvin became available.[17]

Epidemiology:

Incidence and Prevalence:

Tinea capitis is a worldwide problem. Its epidemiology is in a constant state of flux and varies considerably with respect to population and geography.[13] Generally, the disease is wide spread in some urban areas in North, Central, and South America. It is also common in parts

* *Specialist, Dermatology Dept., Dammam Central Hospital, Dammam, Saudi Arabia*

** *Assistant Professor, Dermatology Dept., College of Medicine, King Faisal University, Dammam Saudi Arabia*

King Fahad Hospital of the University

P.O.Box 40189 - Alkhobar 31952, Saudi Arabia

Tel No.+96638957886 - Fax No. +96638949209

Email: consultant@dermatologyclinics.net

of Africa and India. In South East Asia the rate of infection has decreased dramatically in the last fifty years and in Northern Europe, the disease is sporadic. [10] In the United Kingdom its prevalence has been relatively low in the past but an increased prevalence has recently been reported in urban areas, particularly in children of Afro-Caribbean extraction. [9, 11, 18] A recent epidemiologic observation in the United States showed a striking increase in the incidence of tinea capitis, particularly among African-Americans. [4]

Age and Sex predilection:

Tinea Capitis is overwhelmingly an affliction of childhood, since adult cases are rare. [8] It is seen in children younger than 10 years and the predominant age range is 3-7 years. [10, 9, 19-24] The age predilection is believed to result from the presence of pityrosporum orbiculare (pit.ovale), which is part of normal flora, and from the fungistatic properties of fatty acids of short and medium chains in post pubertal sebum .The incidence of specific fungal species in tinea capitis may also vary by sex, such as when the causative organism is *Microsporum audouinii*, boys are affected more commonly than girls and the male to female ratio is 5:1. However the reverse is true after puberty, possibly due to increased exposure to infected children by women who take care of them. [25-26] Also when the causative organism is of *Trichophyton* species adult women are affected more frequently than men, although, they are equally affected during childhood. [25, 27]

Etiology:

Dermatophytes are classified into three genera: *Microsporum, Trichophyton, and Epidermophyton.* Tinea capitis is caused by the former two but no known literature stated *Epidermophyton* as a cause. [2, 5-7] Besides, dermatophytes have three major reservoirs and hence classified ecologically into anthropophilic (found in humans), Zoophilic (found in animals), and geophilic (found in soil) [13, 28] (Table 1.)

The geophilic species is found world wide, while anthoropophilic and some Zoophilic species may be geographically restricted. The predominant organism varies with geographical area, and it is often difficult to know the precise distribution of a particular dermatophyte. In addition, the etiologic agents of tinea capitis in a given geographic region can also change over time. Prior to the 20th century, the most common causative organism worldwide was *Microsporum canis.* [12] Later on *Microsporum audouinii* became the prominent cause of tinea capitis in North America and in Western Europe. In contrast, in the US, the incidence of infection with *M audouinii* became significantly less common, while that due to *Trichophyton tonsurans* continued to increase, as demonstrated by surveys of the San Francisco Bay area over a 20-year period. A massive increase in the percentage of Tinea capitis cases caused by *T. tonsurans* from 60% to 91% was recorded. [29]

Pathogenesis:

Tinea capitis is a communicable fungal infection. Invasion by dermatophytes follows a common pattern beginning with adherence between arthroconidia and keratinocytes, followed by germination of arthroconidia and penetration of the hair keratin. [30]

The spores can be demonstrated in the air in close proximity to patients with the condition. It is highly likely that scalp hair acts as trapping device. [31] Actual hair infection starts with invasion of the stratum corneum of the scalp skin. Trauma assists inoculation, which is followed by hair shaft infection. Spread to other follicles proceeds, then for a period of variable duration the infection persists, but does not spread further. Finally there is a period of regression with or without inflammatory phase. [2,32]

There are three patterns of hair invasion: Ectothrix, Endothrix, and Favus. Ectothrix hair invasion is frequently caused by *M.audouinii, M.canis, M.gypseum, M.nanum, T.verrucosum T.mentagrophyte;* and, rarely by *T.rubrum.* Hair appears to be susceptible to ectothrix dermatophyte during mid to late anagen. The infection begins in the perifollicular stratum corneum, following a period of incubation. Hyphae generally spread into and around the hair shaft. They descend into the follicle and penetrate the mid portion of the hair until they reach the border of keratogenous zone. Here they continue to grow in balance with the keratinization process, so that they proceed no deeper than the upper limit of the keratogenous zone. In this location the terminal tuft of hyphae is termed Adamson's fringe, above which is the weakest point of hair shaft. Intrapilary hyphae proliferate within the hair shaft. There are two types of ectothrix arthrospores:

1. The small-spored ectothrix is caused by *M. audouinii*

and *M canis* mainly. Secondary extrapilary hyphae burst out and grow in a tortuous manner over the surface of the hair shaft. These secondary extrapilary hyphae segment to produce a mass of small and spherical arthroconidia. Fluorescence under wood's lamp is characteristically present in this type of hair invasion.

2. Large-spored ectothrix is caused by *T. verrucosum* and *T. mentagrophyte*. The arthroconidia are large, spherical, arranged in chains and again confined to the external surface of the hair shaft. They arise from straight primary extrapilary hyphae rather than from the hyphae inside the hair. There is no fluorescence. (2,26,32,33)

Endothrix pattern is caused predominantly by *T.Tonsurans, T.Soudanese, T.violaceum, T.yaoundei* and, occasionally *T.rubrum*. The infection develops in a similar manner as ectothrix until the hair is penetrated. The arthrospores are formed rapidly and in time replace much of the intrapilary keratin, while leaving the cortex intact. The hair is fragile and, with trauma, breaks at its weakest point which is the surface of the scalp where it loses the supporting follicular wall. A final important difference between endothrix and ectothrix infection is that endothrix infection may continue past the anagen phase of the hair cycle and into telogen. Therefore, these infections tend to be more chronic than those caused by the ectothrix organism. (3,34) The favic type is caused by *T.schoenleinii*. In the early stages of infection, hyphae invade the hair follicle and gradually distend the follicular opening. The affected hair is less damaged than in other types, and may continue to grow to considerable lengths. The favus hair shows hyphae coursing lengthwise and no arthrospores. (8) Because of autolysis, vacant tunnels are found within the hair and these may appear as airspaces within the hair shaft. (35)

Sources of Infection:

In the United States, surveys showed that large family size, crowded living conditions and low socioeconomic status contributed to the increased incidence of tinea capitis. (8, 21) It can be transmitted from an infected person by fomites, like contaminated hairbrushes, combs, hats, pillows, and toys or from an infected animal. (4, 28) Anthropophilic fungi like *T.violaceum* and *T.schoenleinii* can be transmitted from an infected person or asymptomatic carrier by direct contact, or by sharing clothes, towels or fomites. Furthermore asymptomatic carriage, in which the person has normal skin yet positive fungal element, is considered a major reservoir of infection with the above-mentioned organisms. (36,37) Zoophilic fungi are transmitted by direct contact with pets or wild animals, (5) such as cats and dogs, which are considered the major sources of M. Canis. In rural areas, farmers may acquire infection from the cattle. Geophilic dermatophyte is transmitted when working with contaminated soil. (5, 21, 36, 37)

Clinical Features:

Tinea capitis may present with several clinical patterns, (4,5,11,12) depending on the type of organism, the type of hair invasion, level of host resistance and the degree of inflammatory host response. (2,38) Generally there are a wide variety of clinical manifestations, including the asymptomatic carrier state. It may vary from few dull, broken-off hairs with mild scaling to severe, painful, inflammatory mass. In all types the cardinal feature is a partial alopecia with some degree of inflammation. (36,39,40,41) A prominent cervical or occipital lymphadenopathy may occur in all types of tinea capitis. Moreover, a wide spread dermatophytide (id) reaction may occur. It is a non-fungal cutaneous eruption, representing an allergic response to a distant focus. It usually presents with symmetrical pruritic follicular papules or vesicles that start on face then spread to the trunk. (2,4,7)

The major clinical types include:

A. Non inflammatory or gray patch:

This clinical pattern is caused mainly by *M.audouinii* and *M. ferrugineum*. The disease is produced by ectothrix hair invasion. (19, 21) The lesion begins as a small erythematous papule surrounding a hair shaft; eventually it spreads centrifugally, involving the neighboring hair follicle. Typically, the lesions consist of patches of partial alopecia, which is circular in shape, showing numerous broken-off hairs, grey, and lusterless due to their coating of arthrospores. There is also fine scaling with minimal inflammation. (2,3,7) In *M.canis* the picture is similar but there are more inflammatory changes. (2, 3)

B. Black dot and seborrhoic like type:

This pattern is caused by endothrix organisms such as *T. tonsurans* and *T.violaceum*. [5, 19] The location of the arthrospores is inside the hair shaft making the hair extremely brittle and breaks at the level of the scalp. The remnant of the hair shaft appears as black dot on clinical examination. [34,36,42] There may be diffuse scaling with minimal hair loss and inflammation, resembling seborrhoic dermatitis or psoriasis. Hence the name seborrhoic type is used. The lesions in black dot type are multiple with angular outline. [2,3] This is in contrast to grey patch, which is annular and well defined. Black dot infections are often inflammatory, where inflammation ranges from folliculitis to frank kerion. Nail and glabrous skin involvement have been seen in some patients. [7] Rarely "Black dot" tinea capitis may present without black dots making clinical diagnosis difficult.[4]

C. Kerion:

This is the inflammatory type of tinea capitis; it is caused by zoophilic organisms usually *T.verrucosum* and *T.mentogrophyte* or geophilic dermatophytes such as M.gypseum. [19,39] Anthropophilic tinea capitis may suddenly become inflammatory and develop into Kerion when a high degree of hypersensitivity develops. The disease presents with painful, boggy mass in which hair is loose and broken-off. Follicles may discharge pus and there may be sinus formation. [5,42-44] The reaction is thought to be a delayed type of hypersensitivity to fungal element. [2,4] The affected area may be limited but occasionally a large lesion may involve the whole scalp. Regional lymphadenopathy with fever and pain may occur when lesions are extensive. Bacterial copathogen may have some role in this type of tinea capitis. [5] Kerions have also been associated with erythema nodosum. [45]

D. Favus:

This pattern is a rare type of tinea capitis. It is caused by *T.schoenleinii*. [28,40] It is seen sporadically in the Middle East, South Africa, Pakistan, and rarely in the United States, Canada, and Australia. [46,47] The organism may affect the skin and nails as well. It is characterized by the presence of yellowish, cup-shaped crusts known as scutula, formed around the hairs. Confluent scutula may form a mass of yellow crust. [4,48] In addition the scutula have a distinctive mousy odor, and hair may be extensively lost ending with cicatricial alopecia and atrophy. [2-5]

Diagnosis:

Since the manifestation of tinea capitis is variable, a definitive diagnosis cannot be made on the clinical appearance alone. [13] Patients should undergo woods light examination and specimens should be collected for microscopy and culture. [1,13]

1. Wood's light examination:

The infected, broken-off hairs and the intrafollicular portion when the hair is plucked will have bright green fluorescence in case of *Microsporum* infection. [49-51] While in *Trichophyton schoenleinii* infections the fluorescence is a faint blue color. But *T. tonsurans* and *T.verucosum* do not fluoresce. [7, 12, 34] The chemical responsible for positive fluorescence is a petridine. [3, 49] Thus the fluorescence of these infected hairs indicates the presence of infection but does not differentiate the causative organisms. Besides, there are some organisms that do not fluoresce. So Wood's light examination is of limited value and is used as a screening test only. [14]

2. Microscopy and Culture:

a) Collecting Specimens:

There are several methods of obtaining specimens suitable for microscopy and culture. The affected areas should be cleaned with 70% alcohol then scraped with blunt scalpel to harvest affected hairs, broken-off hair stubs and scalp scale. [1,2,15] The scrapings should be transported in a folded square of paper fastened with a paper clip. [1] Another method of collecting samples is by moistening the affected area with wet gauze, [52] and then brushed gently with sterile tooth-brush. In this maneuver, infected scales and hair are recovered; the brush is then sent in a container to the laboratory. [53,54]

b) Microscopy:

Microscopy provides the most rapid means of diagnosis, but is not always positive. Scalp scales and broken off hair stumps are mounted in a 10-30% solution of potassium hydroxide (KOH). Then a cover slip is applied. The specimen is then warmed over a flame until

the hairs are macerated. It is then examined with low power and high-power objective for details. Xylol may be used as a mounting medium and it is as good as KOH and does not need warming. [55] Examination of properly mounted specimen will demonstrate the type of hair invasion involved. In ectothrix infections, arthrospores are seen outside the hair shaft, while in endothrix infections the arthrospores are intrapillar, but hyphae can be seen within the hair in both types of infection. [1,2,4,40]

c) Culture:

The final and exact identification of the causative organism could be determined by culture. [13,41,56] Specimens are collected using the toothbrush method or scraping method and placed on suitable fungal medium such as Sabouraud's dextrose agar mycosel (mycobiotic) agar or Dermatophyte test Medium (DTM) containing cyclohexamide and chloramphenicol to suppress the growth of common saprophytic and bacterial contaminants.

DTM contains a color indicator that changes from yellow to red in the presence of dermatophyte fungi. [4,11,15,19,57]

Cultures are incubated at 25-30C for 4 weeks, but positive cultures will show signs of growth within 10 days. The organism is identified by the characteristics of the colony and microscopy.[19,38,58]

Pathology:

In tinea capitis, hyphae are present within and around the hair shaft. Special stains are used to identify or emphasize the presence of dermatophytes such as Periodic Acid-Schiff (PAS) or Methenamine silver. In the dermis there will be a perifollicular mixed inflammatory infiltrate of lymphocytes, histiocytes, plasma cells and eosinophils. An adjacent foreign-body giant cell reaction is seen when there is follicular disruption. In Kerion there is dense dermal infiltrate with polymorphonuclear leukocytes forming abscesses in the dermis as well as in the follicle. [59]

Differential Diagnosis: [1- 4, 33]

Seborrheic dermatitis, Atopic dermatitis ,Psoriasis, Alopecia areata, Trichotillomania, Secondary syphilis, Pseudopelade, Furunculosis, Impetigo, Folliculitis decalvans, Discoid lupus erythematosus, Lichen planopilaris, Pediculosis capitis, Lichen simplex chronicus

Treatment:

The principles of management of Tinea Capitis consist of systemic therapy, topical therapy and preventive measures. [5]

1) Topical Therapy:

Topical antifungal agents are not recommended as the sole therapy for the management of Tinea Capitis. [40,60] It may however, decrease the risk of spread of the infection to others by reducing the shedding of fungal spores. [1,5] Selenium sulfide, [61] ketoconazole shampoo [40,62] and povidone-iodine [63] shampoos, used twice weekly, decrease the carriage of fungal spores and thus reduce infectivity.

2) Oral Therapy:

Oral antimycotic agents are needed to penetrate the hair follicle. [40, 46,60] The gold standard of oral therapy for Tinea Capitis for the past four decades has been griseofulvin.[4, 46] In The 1990s, a variety of therapeutic options were introduced for the treatment of Tinea Capitis. [36] The new agents available now for the treatment of Tinea Capitis are Fluconazole, Ketaconazole, Itraconazole, and Terbinafine. They offer alternative approaches to the treatment of Tinea Capitis. [64-69]

A. Griseofulvin:

It is an antibiotic derived from several different species of the penicillium mold. [4,70] It has a fungistatic [1,4,5] which produces its effects by inhibition of the fungal RNA, DNA, microtubular assembly, and cell wall synthesis. [1,65,70] It is is poorly absorbed on an empty stomach. However, ingestion after a fatty meal results in abrupt rise in serum levels. [70-72] The recommended dose for Tinea Capitis is 20mg/kg/day for the micronized form and 15mg/kg/day for the ultramicronized form. [71] The duration of therapy is generally 6-12 weeks. [5]

B. Azoles antifungal drugs:

These include the following drugs: ketoconazole, itraconazole and fluconazole. They share the

same mechanism of action which involve the inhibition of cytochrome p450-dependent enzymes (particularly C14-demethylase) acting in the biosynthesis of ergosterol; an essential component of fungal cell membrane. [70]

1. Ketoconazole:
   It reaches the skin via excretion of the sebum and of eccrine sweat, [73] binds strongly to the keratinocytes and may get attached to the hair matrix cells.
2. Itraconazole:
   It binds strongly to the keratinocytes in the basal cell layer and is excreted by sebaceous glands, where it reaches the hair. It may also be incorporated into hair follicle. Excretion of the drug by sweat gland is minimal. [5,74]
   The recommended dose is 5mg/kg/day given for 4 weeks. [1,5] That may be given as continuous therapy or as a pulse therapy for 1 week followed by 2-3 week period off treatment. This cycle is repeated three times. [75]
3. Fluconazole:
   It is the newest triazole given at a dose of 3-5mg/kg/day for 2-4 weeks, or 8mg/kg once weekly for 4-8 weeks. [5]

C. Terbinafine:

It is an allylamine which act by inhibiting fungal ergosterol biosynthesis required for fungal membrane integrity and growth. [5] and it is given in a dose of 250 mg/day Given for 4 weeks [76]

References:

1. *Higgins EM, Fuller LC, Smith CH. Guidelines for the management of tinea capitis. Br J dermatol 2000; 143(1): 53-58.*
2. *Hay RJ, Moore M. Mycology in: Rook, Wilkinson, Ebling, et al, eds. Textbook of dermatology. Sixth edition. Oxford: Blackwell scientific publication.1998: 1303-1305.*
3. *Martin AG, Kobayshi GS. Superficial fungal infection. In: Fitzpatrick TB, Eisen AZ, Wolff K, et al. eds. Dermatology in General Medicine. New York: McGraw-Hill, 1993:2421-2432.*
4. *Elewski BE. Tinea capitis: a current perspective. J Am Acad Dermatol 2000; 42: 1-20.*
5. *Gupta AK, Hofstadter SL, Adam p, Summerbel RC. Tinea capitis: An overview with emphasis on management. Pediatr Dermatol 1999; 16(3): 171-189.*
6. *Gargoom AM, Elyazachi MB, Anis M, Duwebg A. Tinea capitis in Benghazi, Libya. Int J Dermatol 2000; 39(4): 263-5*
7. *Gupta AK, Summerbel RC. Tinea capitis. Med Mycol 2000; 38 (4): 255-287.*
8. *Rippon JW. Dermatophytosis and dermatomycosis in medical mycology: The pathogenic fungi and the pathogenic actinomycetes. 3rd ed. Philadelphia, Saunders, 1988:169.*
9. *Hay RJ, Clayton YM, Desilva N, et al. Tinea capitis in southeast London – a new pattern of infection with public health implication. Br J Dermatol 1996; 135: 955-8.*
10. *Kemna ME, Elewski BE. A U.S epidemiologic survey of superficial fungal diseases. Int J Dermatol 1996; 35: 539-542.*
11. *Bronson DM, Desai DR, Barsky S, Foley SM. An epidemic of infection with trichophyton tonsurans revealed in a 20-year survey of fungal infections in Chicago. J Am Acad Dermatol 1983; 8: 322-30.*
12. *Prevost E. The rise and fall of fluorescent tinea capitis. Pediatr Dermatol 1983; 1: 127-123.*
13. *Chen BK, Friedlander SF. Tinea capitis update: A continuing conflict with an old adversary. Curr Opin Pediatr 2001; 13(4): 331-335.*
14. *Ayaya SO, Kamar KK, Kakai R. Aetiology of tinea capitis in school children. East Afr Med J 2001; 78(10): 532-535.*
15. *Rubio-Calvo C. The aetiological agents of tinea capitis in Zaragoza (Spain). Mycosis 2001; 44(1-2): 55-58.*
16. *Sabouraud R. Les Teignes. Paris, Masson, 1910.*
17. *Blank H, Roth FJ. The treatment of dermatomycosis with orally administered griseofulvin. Arch Dermatol 1959; 79: 259-263.*
18. *Fuller LC, Child LC, Higgins EM. Tinea capitis in southeast London: An outbreak of trichophyton tonsurans infection. Br J Dermatol 1997; 136:139.*
19. *Elewski BE, Hazen P. The superficial mycoses and dermatophytes. J Am Acad Dermatol 1989; 21: 655-73.*
20. *Kwon-Chung KJ, Bennett J. Medical Mycology. Philadelphia, Lea & Febiger; 1992:105-161.*
21. *Babel DE, Baughman SA. Evaluation of the adult carrier state in juvenile tinea capitis. J Am Acad Dermatol 1989; 21: 1209-1212.*
22. *Gan VN, Petruska M, Ginsburg CM. Epidemiology and treatment of tinea capitis: Ketoconazole vs. Griseofulvin. Pediatr Infect Dis J 1987; 6: 49-59.*
23. *Leeming JG, Elliot TS. The emergence of Trichophyton tonsurans tinea capitis in Birmingham, UK. Br J Dermatol 1995; 133: 929-931.*
24. *Williams JV, Honig PJ, McGinley KJ, Leyden JJ. Semi quantitative study of tinea capitis and the asymptomatic carrier state in inner-city school children. Pediatrics 1995; 96: 265-7.*
25. *Elewski BE. Tinea capitis. In: Demis DJ. ed. Tinea capitis in clinical dermatology. Philadelphia: JB Lippincott, 1999: 1-23.*
26. *Klingman AM. Tinea capitis due to M.audouinii and M.canis Arch Dermatol 1955; 71: 313-348.*

27. Vidimos AT, Camisa C, Tomecki KJ. Tinea capitis in three adults. Int J Dermatol 1991; 30(3): 206-207.
28. Aly R. Ecology, epidemiology and diagnosis of tinea capitis. Pediatr Infect Dis J 1999; 18: 180-185.
29. Wilmington M, Aly R, Frieden IJ. Trichophyton tonsurans tinea capitis in the San Francisco bay area: increased infection demonstrated in a 20-year survey of fungal infections from 1974 to 1994. J Med Vet Mycol 1996; 34: 285-287.
30. Aljabre SHM, Richardson MD, Scott EM, Shankland GS. Germination of Trichophyton mentogrophyte on human stratum corneum in vitro. J Med Vet Mycol 1992; 30: 145-52.
31. Mackenzie DWR. Hairbrush technique in detection and eradication of non-fluorescent scalp ringwarm. Br Med J 1963; ii: 363-5.
32. Klingman AM. The pathogenesis of tinea capitis due to M.audouinii and M.canis. J Invest Dermatol 1952; 18: 231-246.
33. Odom RB, James WD, Berger TG. Diseases resulting from fungi and yeasts. In: Andrew's Diseases of the skin: Clinical dermatology. 9th ed. Philadelphia, Saunders, 2000: 359-364.
34. Hernandez AD. An approach to the diagnosis and therapy of dermatophytosis. Int J Dermatol 1980; 19: 540-47.
35. Dvoretzky I et al: Favus. Int J Dermatol 1980: 19: 89.
36. Friedlander SF: Tinea Capitis-Past, Present, and future. Curr Prob Dermatol 2000; 11: 126-129.
37. Ali-Shtayeh MS, Salameh A, Abu-Ghdeib SI, et al. Prevalence of tinea capitis as well as of asymptomatic carriers in school children in Nablus area (Palestine). Mycosis 2002; 45(5-6): 188-194.
38. Jones HE. A clinical, Mycological and immunological survey of dermatophytosis. Arch Dermatol 1973; 108:61:65
39. Jahangir M, Hussain I, Khurshid K, and Haroon TS: A clinico-etiologic correlation in tinea capitis. Int J Dermatol 1999; 38: 275-278.
40. Elewski B. Tinea Capitis. Dermatol clinics 1996; 14: 23-31
41. Singal A, Rawat S, Bhattacharya SN, et al. Clinico-mycological profile of Tinea Capitis in North India and response to griseofulvin. J Dermatol 2001; 28(1): 22-26.
42. Rudolph A. The clinical recognition of Tinea Capitis from Trichophyton Tonsurans. JAMA 1979; 242(16): 1770.
43. Laude TA. Epidemiology of Tinea Capitis. Pediatr Dermatol 1985; 2: 234-236.
44. Padilha-Goncalves A. Inflammatory tinea capitis (kerion) mimicking dissecting cellulites [letter]. Int J Dermatol 1992; 31: 66.
45. Calista D, Schianchi S, Massimo M. Erythema nodosum induced by kerion celsi of the scalp. Pediatr Dermatol 2001; 18(2): 114-116.
46. Elewski BE. Treatment of Tinea Capitis: beyond griseofulvin. J Am Acad Dermatol 1999; 40: 27-30.
47. Aly R, Hay RJ, Delpalacio A, Galimberti R. Epidemiology of tinea capitis. Med Mycol 2000; 38(s1): 138-188.
48. Schwartz RA, Janniger CK. Tinea capitis. Cutis 1995; 55: 29-33.
49. Aswanonda P, Taylor CR. Wood's light in dermatology. Int J Dermatol 1999; 38(11): 801-807.
50. Leffell DJ, Stetz ML, Milstone LM, Decklbaum LI. In vivo fluorescence of human skin. A potential marker of photoaging. Arch Dermatol 1988; 124: 1514-1518.
51. Halprin Km. Diagnosis with woods light. Tinea capitis and erythrasma. JAMA 1967; 199:177.
52. Brochers SW. Moistened gauze technique to aid diagnosis of tinea capitis. J Am Acad Dermatol 1985; 13: 672-3
53. Head ES, Henry JC, Macdonald EM. The cotton swab technique for the culture of dermatophyte infections—its efficacy and merit. J Am Acad Dermatol 1984; 11: 797-801.
54. Fuller LC, child FJ, Midgely G. A practical method for mycological diagnosis of the tinea capitis: validation of the toothbrush technique. J Eur Acad Dermatol Venereol 1997; 9(s1): 209.
55. Shelley WB, Wood MG. New technic for instant visualization of fungi in hair. J Am Acad Dermatol 1980; 2: 69-71
56. Fathi HI, Alsamarai AM. Tinea capitis in Iraq: laboratory results. East Mediterr Health J 2000; 6(1): 138-48.
57. Romano C. tinea capitis in Siena, Italy. An 18- year survey. Mycosis 1999; 42: 559-562.
58. Elbenhawi MO. Mycologic study of tinea capitis in Qatar. Int J Dermatol 1991; 30(3): 204-205.
59. Lever WF, Schaumburg-Lever G. Fungal diseases. In: Histopathology of the skin. 8th ed. Philadelphia, Lippincott 1997: 517-551.
60. Elewski BE. Cutaneous mycoses in children. Br J Dermatol 1996; 134(s46): 7-11.
61. Allen HB, Honig PJ, Leyden JJ, McGinley KJ. Selenium sulphide: adjunctive therapy for tinea capitis. Pediatrics 1982; 69: 81-83.
62. Nesbitt LT. Treatment of tinea capitis. Int J Dermatol 2000; 39: 261-262.
63. Neil G, Hanslo D. Control of the carrier state of the scalp dermatophytes. Pediatr Infect Dis J 1990; 9: 57-58.
64. Hay RJ. New development in antifungals. Int J Dermatol 1999; 38(s2): 65-69.
65. Elewski BE. Tinea Capitis: Itraconazole in Trichophyton tonsurans infection. J Am Acad Dermatol 1994; 31: 65-67.
66. Haroon TS, Hussain I, Mahmood A, et al. An open clinical study of the efficacy and safety of oral terbinafine in dry non-inflammatory tinea capitis. Br J Dermatol 1992; 126(S39): 47-50.
67. Tanz RR, Herbert AA, Esterly NB. Treating tinea capitis: should ketoconazole replace griseofulvin? J Pediatr 1988; 112: 987-991.
68. Bennett ML, Fleischer AB, Loveless JW, Feldman SR. Oral griseofulvin remains the treatment of choice for tinea capitis in children. Pediatr Dermatol 2000; 17(4): 304-309.
69. Friedlander SF. The evolving role of itraconazole, fluconazole

and terbinafine in the treatment of tinea capitis. Pediatr Infect Dis J 1999; 18: 205-210.

70. *Millikan LE, Shrum JP. Antifungal agents. In: Wolverton, Wilkon. eds. Systemic drugs for skin diseases. 1st ed. WB. Saunders Company. 1991:25-40.*
71. *Ginsburg CM, McCracken GH, Petruska M, et al. Effect of feeding on bioavailability of griseofulvin in children. J Pediatr 1983; 102: 309-311.*
72. *Mercurio MG, Elewski B. Tinea capitis treatment. Dermatol Ther 1997; 3: 79-83.*
73. *Dedoneker P. Pharmacokinetics of oral antifungal agents. Dermatol Ther 1997; 3: 46-57.*
74. *Cauwenbergh G, Degreef H, Heykants J, et al. Pharmacokinetic profile of orally administered itraconazole in human skin. J Am Acad Dermatol 1988; 18:263-268.*
75. *Gupta AK, Alexis ME, Roboobee N, et al. Itraconazole pulse therapy is effective in the treatment of tinea capitis in children: an open multicentre study. Br J Dermatol 1997; 137: 251-254.*
76. *Schwinn A, Hamm H, Brautigam M, et al. What is the best approach to tinea capitis with terbinafine? J Eur Acad Dermatol Venereol 1998; 11(S2): 232.*

Table 1 Ecological classification of dermatophytes

Anthropophilic	Zoophilic	Geophilic
M. audouinii	*M.* Canis	*M* gypseum
T. tonsurans	*T.* mentogrophyte	
T. violaceum	*T.* verrucosum	
M. ferrogineum	*M.* distortum	
T. mentogrophyte		
T. schoenleinii		
T. rubrum		
T. soudanese		
T. yaondei		

CORRECTION

In the October issue of the Gulf Journal of Dermatology and Venereology 2003 the name of Doctor AL-Fouzan A., as first author of the article "vitiligo treatment update review article" page 1-13 was missed as a typing error. The correction is **"vitiligo treatment update review article"**

Al-Fouzan A., *MD; PhD.*
Nawwaf al Mutairi; *MD; FRCPC*
Osama Nour-El-Din; *MD*