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REVIEW ARTICLE

Tinea Capitis

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Abstract. Tinea capitis is a widespread scalp infection in children caused by dermatophytes. In fact, it is the most common cutaneous mycosis in children but is uncommon in adults. The disease has been a major public health concern for decades. Some factors implicated in infection include poor personal hygiene, crowded living conditions, and low socioeconomic status. It can be caused by any pathogenic dermatophyte except for *Epidermophyton floccosum* and *Trichophyton concentricum*. *Trichophyton rubrum*, the most commonly isolated dermatophyte worldwide, is rarely the causative agent of this infection. Tinea capitis is a classic example of the changing geographic patterns of dermatophytosis. In developed countries, *Trichophyton tonsurans* is the most common causative agent, whereas in developing countries such as Mexico, the most common agent is *Microsporum canis* followed by *T tonsurans.* The increasing incidence of tinea capitis warranted a review of the current literature and treatment strategies.

Key words: tinea capitis, children, *Trichophyton tonsurans, Microsporum canis*.

TIÑA DE LA CABEZA

Resumen. La tiña de la cabeza es una dermatofitosis del cuero cabelludo frecuente en niños, de hecho es la más común de todas las micosis cutáneas en este grupo de edad, siendo muy rara en adultos. Se ha considerado un problema de salud pública importante durante décadas, algunos de los factores asociados son: higiene personal deficiente, hacinamiento y bajo nivel socioeconómico. Está causada por cualquier dermatofito patógeno, excepto *Epidermophyton floccosum* y *Trichophyton concentricum. Trichophyton rubrum*, el dermatofito más comúnmente aislado en el mundo, es excepcional como causa de *tinea capitis.* La tiña de la cabeza es un ejemplo clásico del cambio de los patrones geográficos de las dermatofitosis. En los países desarrollados hay un incremento de las tiñas tricofíticas y en países en vías de desarrollo como México el agente causal más común es *Microsporum canis*, seguido de *Trichophyton tonsurans.* El incremento de la incidencia de las dermatofitosis de la cabeza hace necesaria una revisión de la literatura, así como de las medidas terapéuticas actuales.

Palabras clave: *tinea capitis*, niños, *Trichophyton tonsurans, Microsporum canis*.

Introduction

Tinea capitis is a dermatophytosis of the scalp that is common in children. It can be caused by any dermatophyte pathogen with the exception of *Epidermophyton floccosum* and *Trichophyton concentricum*. *Trichophyton rubrum*, the most commonly isolated dermatophyte worldwide is only responsible for tinea capitis in exceptional cases. The causative agent varies between continents and from one country to another. For instance, *Microsporum canis* is the predominant pathogen worldwide, while *Trichophyton tonsurans* is the main causative agent in the USA.[1,2]

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Historical Considerations

The description of an acute inflammatory disease of the scalp with production of purulent material is attributed to Celsus around 30 BCE in Rome, leading to the name kerion celsi. During the 19th century, tinea

capitis was a very serious public health problem that reached epidemic proportions; it was apparently introduced to the American continent by the Europeans.[3]

Epidemiology

The incidence of tinea capitis is unknown. It has been considered a serious public health problem for decades and appears most often in children between 3 and 7 years of age; in fact, it is the most common of all cutaneous mycoses in this age group. It is rare in adults and more often affects women around the menopause and elderly women; in these patients the clinical presentation may be different, presenting problems for diagnosis. Conerly and Green stated that "folliculitis" was the diagnosis most commonly confused with tinea capitis in adults.[4] Tinea capitis is considered to be almost exclusive to children and rarely occurs after puberty, probably due to changes in the pH of the scalp and an increase in fatty acids serving a protective role. Consequently, most cases occurring in adults involve women with hormonal disorders resulting in carryover of tinea capitis from childhood or in patients with severe immunodepression due to leukemia, lymphoma, or treatment with immunosuppressant drugs. In a multicenter retrospective study undertaken in Mexico City to determine the epidemiologic and clinical characteristics of tinea capitis in adults, out of a total of 1028 cases, 30 occurred in adults, with an overall frequency of 2.9%. Most of the patients were women, with a 3:1 ratio compared to men, and the most common etiologic agents were *M canis* followed by *T tonsurans*.[5-8] One of us has reported 2 cases, an 87-year-old woman who was infected via onychomycosis of the hands and a 75-year-old woman with diffuse alopecia of the scalp and multiple erythematous scaly plaques on glabrous skin, both of which were caused by *T tonsurans*; in the second case, the patient suffered a relapse upon treatment with systemic corticosteroids.

Tinea capitis occurs predominantly in rural or suburban areas and some of the factors associated with this increased frequency include poor personal hygiene, overcrowding, and low socioeconomic level. The organism responsible for tinea capitis has been cultured from fomites such as combs, hats, pillows, and theater seats, where the spores can live for long periods of time, contributing to spread of the disease.[9,10] Contact in schools is probably the most important independent factor affecting the rapid spread of tinea capitis. Infection in children of school age is usually followed by infection of younger siblings. Family epidemics are common and the existence of asymptomatic carriers hinders eradication of the disease.[1,11]

Geographic Distribution

Dermatophytes are keratinophilic fungi that can invade the stratum corneum of the skin and other keratin-containing tissues. They have 3 main reservoirs: humans (anthropophilic), animals (zoophilic), and the earth (geophilic or telluric). Although geophilic dermatophytes are found throughout the world, the anthropophilic and some zoophilic species may be geographically restricted. The predominant organisms vary according to the geographic region and it is often difficult to determine the precise distribution of a particular dermatophyte. Consequently, the causative agents in tinea capitis have not been investigated in many parts of the world, and the agents responsible for dermatophytosis tend only to be identified in regions where there are laboratories to perform the mycologic studies.[12]

Tinea capitis is a classic example of the changing geographic patterns of dermatophytosis during the late 19th and early 20th century. *Microsporum audouinii* and *M canis* were the main causative agents of the disease in Western Europe and the Mediterranean, while *Trichophyton schoenleinii* predominated in Eastern Europe, and now both have almost disappeared. The geographic distribution and prevalence vary as a result of factors such as climate and migration, among others.[11-13]

Europe

The spread of dermatophytes as causative agents of scalp infections in Europe is highly heterogeneous. *M canis* has been most often detected, although its prevalence is only high in southern Europe, where it has been isolated in more than 80% of cases.

Studies undertaken in Spain have identified other dominant pathogens that vary according to the region; for instance, *Trichophyton mentagrophytes* in Barcelona and *Trichophyton verrucosum* in Salamanca. In Madrid, where the causative agent was usually *M canis*, an increase in the incidence of cases due to *T tonsurans* has been reported (similar to the phenomenon observed in the USA). In Western and Central Europe, the situation is different. In these regions, it has been demonstrated that *M canis* is responsible for approximately half of all cases of dermatomycosis of the scalp and the other half is predominantly due to *T verrucosum*, *T mentagrophytes*, and *T violaceum*, similar to findings in other studies in the United Kingdom and France. In Italy, the main causative agent is *M canis*, although in that country and in Holland it has been reported that there has been a resurgence in the number of cases caused by *M audounii* and *T violaceum* as a result of the increased number of immigrants from African countries.

The introduction of griseofulvin for the treatment of tinea capitis along with the introduction of school-based screening programs around the end of the 1950s and the beginning of the 1960s led to a marked reduction in the incidence of tinea capitis due to *Microsporum* species in Western Europe.[12-16] In the United Kingdom, a significant increase has been observed in the incidence of tinea capitis caused by *T tonsurans*.[17]

North America

In the USA, unlike in Europe, a significantly lower percentage is caused by *M canis*. In the last 30 to 40 years, the agents responsible for tinea capitis in the USA have changed, and this has been attributed to immigration from Mexico, the Caribbean, and other countries of Central and South America. Up to 1950, *M audouinii* was responsible for most epidemics of tinea capitis in the USA. This organism has almost disappeared, but the causes are not well understood. The existence of regions of the USA such as Arizona that have had similar patterns of immigration but in which cases due to *T tonsurans* are not observed suggests that the explanation for the change in distribution of causative agents may be more complex.

Pipkin was one of the first authors to report an increase in the frequency of cases due to *T tonsurans* along the southeast border of the USA, and from the beginning of the 1980s, *T tonsurans* was responsible for 90% of cases of tinea capitis in New York, Charleston, South Carolina, and Chicago[18]; in a study of 937 children in Cleveland, Ohio in 2002, there were 122 cases with positive culture and, with the exception of a single case in which *M canis* was isolated, the only causative agent isolated was *T tonsurans*. In Canada, the predominant causative agents are *T mentagrophytes* and, to a lesser extent, *M canis*.[19]

Mexico

In Mexico, tinea capitis accounts for between 4% and 10% of dermatophytoses and corresponds to the dry type in 90% of cases and the inflammatory type in 10%.[8] In a clinical and mycologic study of 125 cases in Mexico City, the age range was found to be predominantly between 6 and 10 years (63.2%), with a mean of 7.2 years, corresponding essentially to the schooling period, and in the classification of the causative agents, the majority of cases were found to be due to *M canis* (77.6%) followed by *T tonsurans* (16.8%). These findings reveal a change in the epidemiologic profile, since 30 years ago the most common causative agent was *T tonsurans*, and this change is attributed to the majority of patients now living in urban areas, where *M canis* is predominant. In rural areas, *T tonsurans* continues to be isolated, although to a lesser extent than 30 years ago. Nevertheless, it is curious that the frequency of cases involving *T tonsurans* continues to be high around the northern border of Mexico.[6,20] Ten years later, in a retrospective study undertaken by the same authors in 2 dermatology departments in Mexico City (Hospital General de Mexico and Hospital Infantil de Mexico), the following results were obtained. In a total of 122 cases confirmed clinically and by mycology, the mean age was 6.1 years, 71 patients (58.1%) were girls, and 44 (36%) involved ectothrix infection. In terms of the causative agents, *M canis* was isolated in 75 (61.5%) and *T tonsurans* in 36 (29.5%); thus, although *M canis* continued to be the main causative agent, the proportion was reduced by 15% compared with the previous study. In terms of the clinical variants, the dry type was most common (87%), similar to the findings of the National Consensus Conference on Superficial Mycosis in Mexico (*Consenso Nacional de Micosis Superficiales en México*).[6,20,21] *T verrucosum*, also known as *Trichophyton ochraceum*, is common in cattle but has only been isolated in humans once in Mexico, in a case of inflammatory tinea capitis.[22]

Caribbean

In Puerto Rico, around two-thirds of all cases of tinea capitis are caused by *T tonsurans* and a third by *M canis*. In the Dominican Republic, tinea capitis is a significant public health problem, and changes in the prevalence of the causative agents have also been documented. Between 1966 and 1972, *M audouinii* represented the causative agent in 76% of cases, followed by *T tonsurans* and *M canis* in 11%; between 1972 and 1980, *M canis* and *M audouinii* predominated, and after 1980, *M canis* was the predominant causative agent. In a study undertaken in the first 5 months of 1996, 481 cases were documented and *M canis* was isolated in 89.6%.[15,23]

Asia

T violaceum is predominant in India and Pakistan. *M canis* is predominant in Iran, although there are reports of a large number of cases caused by *T violaceum*, *T schoenleinii*, and *T verrucosum* (*T ochraceum*).[24] In Israel, *T schoenleinii* is the predominant causative agent. In studies undertaken in Saudi Arabia and Kuwait, *M canis* was the most common causative agent.[15]

Africa

As in other parts of the world, the prevalence of different types of dermatophytes in Africa varies from region to

Table 1. Ecto-endothrix Invasion

Megaspore	Large spores (3 to 5 m), disorganized
Microsporic	Small spores (2 to 3 m), disorganized
Microid	Small spores (2 to 3 m), organized

region. In Senegal and the western part of Zaire, the predominant dermatophytes are *M audouinii* and *T soudanense*, while *Trichophyton yaoundei* is predominant in Cameroon and the eastern part of Zaire. In Ethiopia and Somalia, *T schoenleinii* is the predominant causative agent, and in the tropical region around the equator, it is *Trichophyton ferrugineum*.[15]

In a study undertaken in Tunisia between 1985 and 1998, out of 1222 cases, 55.8% involved *Trichophytum* species, 41.7% *Microsporum* species, and the remainder corresponded to inflammatory tinea and tinea favosa (1.8% and 0.6%, respectively).[25] In that study, the predominant causative agents were *T violaceum* (53%) and *M canis* (44.7%). Similar data were reported in a study undertaken in Mozambique in 2001, in which there was a clear prevalence of *M audouinii*, which was responsible for 21 of the 29 cases studied.[26]

Types of Infection in Tinea Capitis

Tinea capitis can be classified according to the microscopic pattern of fungal invasion as endothrix, with 2 varieties (trichophytic and favic), and ecto-endothrix, with 3 varieties.

Endothrix invasion is mainly produced by *T tonsurans*, *T soudanense*, and *T violaceum*, and other causative agents that act through endothrix invasion are *T gourvilli*, *T yaoundei*, and occasionally, *T rubrum*. The hyphae grow downwards along the hair and penetrate the shaft. The fungus grows exclusively inside the hair shaft and the cuticle remains intact. The hyphae within the hair fragment into arthroconidia. Direct microscopic examination of the hair reveals arthroconidia inside the hair shaft without destruction of the cuticle. In the USA, most infections of this type are due to *T tonsurans*, while *T violaceum* is more common in Europe.

Favus, which is mainly caused by *T schoenleinii*, is a type of inflammatory tinea that is characterized by the presence of raised yellow cup-shaped crusts around the hair follicles, with keratotic crusts that contain hyphae and are highly infectious.

Tinea favosa is characterized by the production of hyphae in the hair that run parallel to the long axis of the hair follicle. When the hyphae degenerate they leave long tunnels inside the hair, which usually does not break. These can be seen alongside air bubbles during microscopic examination of the parasitized hairs with potassium hydroxide (KOH). This is why the disease is called favus, from the Latin for honeycomb. Tinea favosa can also be caused by *Microsporum gypseum* and *T violaceum*.[3,12]

Ecto-endothrix invasion of the hair is often associated with *M audouinii*, *M canis*, *M distortum*, *M ferrugineum*, *M gypseum*, *M nanum*, and *T verrucosum*. Some of these cause fluorescence under Wood light.

The hyphae invade the medial part of the hair follicle, grow outwards, and cover the surface of the hair; they can develop arthroconidia inside or outside the hair. The spores surrounding the hair give the impression of a sheath. The process of ecto-endothrix invasion is similar to endothrix invasion, with the exception that the hyphae destroy the hair cuticle and grow around the external sheath of the hair. The hyphae then convert into infectious arthroconidia. This type of parasitization is subdivided into megaspore, microspore, and microid, depending on the size and distribution of the spores (Table 1).

Clinical Manifestations

The clinical presentation of tinea capitis is determined by the form of invasion of the hair by the pathogenic fungi (ectothrix or ecto-endothrix), the size of the inoculum, and the immune status of the host (Table 2).

A wide variety of presentations have been described, ranging from asymptomatic carriers, diffuse scaling similar to seborrheic dermatitis, areas of alopecia without inflammation, alopecia with black dots, and if the causative agent is zoophilic or geophilic, a variable inflammatory response is triggered in the host and is clinically manifested as folliculitis or kerion. It is also common to encounter enlargement of the auricular and posterior occipital lymph nodes, and this can be the primary manifestation of the disease.[2,12]

The predominant clinical presentation of tinea capitis has varied as a consequence of the changing geographic patterns of the agents responsible for the disease. Thus, the area of alopecia that spreads slowly with little or no inflammation characteristic of parasitization by *M audouinii* has been replaced by the appearance of black dots attributed to *T tonsurans* in some regions such as the USA.[13] *T tonsurans* essentially does not cause inflammatory tinea and generally manifests as dry tinea with the appearance of seborrheic dermatitis with mild inflammation and little hair loss; however, it can have a wide variety of clinical manifestations, and the type of presentation would not correctly indicate the causative agent, or vice versa, and consequently, tinea due to

Table 2. Clinical Characteristics according to Causative Agent

Organisms Associated With Different Clinical Types of Tinea Capitis			
Inflammatory	*Noninflammatory*	*Black Dots*	*Favus*
Microsporum audouinii	*M audouinii*	*Trichophyton tonsurans*	*Microsporum gypseum*
Microsporum canis	*M canis*	*Trichophyton violaceum*	*Trichophyton schoenleinii*
M gypseum	*Microsporum ferrugineum*	*T tonsurans*	
T violaceum			
Microsporum nanum	*T tonsurans*		
Trichophyton mentagrophytes			
T schoenleinii			
T tonsurans			
Trichophyton verrucos			

T tonsurans is more difficult to diagnose than tinea due to *Microsporum* species.[4,27]

Dry and Inflammatory Tinea Capitis

The dry variety of tinea capitis manifests as scaling and characteristic changes of the hair, namely, short (2-3 mm), thick, friable, and deformed hairs that sometimes have a whitish sheath. Tinea due to *Trichophytum* species causes diffuse alopecia with small irregular plaques interspersed with healthy hairs. The diseased hairs have an appearance of grains of dust (black dots) and there may be very small lesions involving 2 to 3 hairs. Disease due to *Microsporum* species, on the other hand, causes 1 or a few well-delimited, rounded areas (plaques) of pseudoalopecia that are larger than those seen in trichophytic tinea, almost always various centimeters in diameter, with the affected hairs cut off at the same level. They give the impression of having been cut with a lawnmower. Pruritus is minimal in both clinical varieties.

The inflammatory forms are divided into 2 groups: the most common is the hypersensitivity type or kerion celsi, which may be clinically indistinguishable from Majocchi granuloma of the head, a rare manifestation that is regularly seen in patients with immunologic disorders and is caused by *Trichophytum* species, shows little or no response to intradermal antigens, and unlike kerion, shows little tendency towards spontaneous recession; although the first cases described affected the head, the more common site is currently the legs.[4,6] Kerion celsi is characterized by an inflammatory mass that is painful on palpation and accompanied by regional adenopathy. It is generally isolated and with a limited size, but can be large and also multiple. It initiates as a dry tinea involving 1 or more pseudoalopecia plaques with scaling and short hairs; subsequently, there is erythema and inflammation, generating a painful lesion with clear borders that is covered with numerous pustules producing abundant pus; it is this clinical appearance that leads to the name kerion, meaning honeycomb. The most notable symptom of this variety of tinea capitis is pain, and there may be enlarged postauricular and satellite lymph nodes. If the disease continues, the short hairs are gradually expelled or remain below the inflammation. Within around 8 weeks, the tissue response and, in particular, cell-mediated immunity leads to complete elimination of the parasite, but as a consequence leaves behind areas of definitive alopecia with fibrosis, due to the sustained attack on the hair follicle. Diagnosis and appropriate treatment are therefore of particular importance in this variety.

While all of the strains that invade the hair can lead to inflammation similar to kerion, this is particularly the case in zoophilic dermatophytes such as *M canis*, *T verrucosum*, and *T tonsurans*, or geophilic species such as *M gypseum*. Cases have been described involving *T tonsurans* and in a report of 9 cases of inflammatory dermatophytosis (5 of trichophytic granuloma and 4 of kerion), 3 of the inflammatory cases were due to *T tonsurans* and 1 to *T rubrum*.

On rare occasions, kerion can be associated with erythema nodosum or hypersensitivity reactions at a distance. The latter can present in the form of a small number of papules with a lichenoid appearance that extend from the scalp to the trunk and limbs. Such reactions can also occur as a consequence of antifungal treatment, generally around the ears and on the face. In those cases, treatment should not be suspended.[27]

In cases of inflammatory alopecia, the possibility of tinea capitis should be excluded. If direct examination and culture are negative, a biopsy should be performed and divided in order to perform cultures in specific media for fungi.

Carrier Status

Transmission of *T tonsurans* via adult carriers was reported by Raimer et al, and in a study of 50 children with clinical diagnosis of tinea capitis and from whom *T tonsurans* was cultured in all cases, cultures were also obtained from 46 adults who lived with them, 14 (30.4%) had positive cultures for *T tonsurans* and none had clinical evidence of tinea capitis.[28] Herbert et al evaluated the contacts and environment of 2 children with tinea capitis due to *T tonsurans* and isolated the same dermatophyte from the scalp of the mother, grandmother, and 2 aunts living in the same home; they also obtained positive cultures for *T tonsurans* from rugs, cushions, furniture, brushes, combs, toys, and earpieces of telephones. Prevost examined a grandmother, mother, and 5 children who lived in the same home, and found that all were infected with *T tonsurans*.[18]

Parasitization by *T tonsurans* appears to be an endemic disease with a relatively stable incidence in the black population of the USA, and with a similar distribution in girls and boys. If this noninflammatory form of tinea capitis is not treated it can persist for long periods of time, in contrast to inflammatory tinea, which tends to display an acute self-limiting course. These characteristics of *T tonsurans* infection lead to the hypothesis that there is a disease stage that is clinically undetectable by physicians and does not present symptoms in the patient. To address this possibility, studies were undertaken in the USA in which cultures were obtained from the scalp of 200 healthy children (100 white and 100 black) and 8 (4%) were found to be positive for *T tonsurans* var *sulfureum*, all from black children. Those infections were not clinically apparent and were asymptomatic, and it was therefore concluded that the agent was in an asymptomatic state in this well-defined population of children, and although it is not definitive evidence that this subpopulation of children represents a group of carriers, the absence of symptoms and clinical signs would make them a potential reservoir.[29] In addition, this colonization (carrier status) could explain the increased prevalence of tinea capitis in urban areas.

A study undertaken in a school in Paris in 2001 during an epidemic of tinea due to *T tonsurans* evaluated 129 children and 15 adults and detected 10 cases of tinea and 25 asymptomatic carriers; the majority of positive cases came from the same class. Thirteen families were also studied and it was found that 2 of them had various affected members. That study highlighted various important points: the high level of contagiousness of *T tonsurans* and the detection of 2 mechanisms of indirect contamination—a doll that was the mascot of the class with the largest number of cases and the asymptomatic carriers in the families.[30]

There are currently no established criteria for the treatment of these asymptomatic carriers.

Diagnosis

Clinical Diagnosis

The presence of alopecia, scaling, follicular inflammation, and enlarged auricular and posterior cervical lymph nodes are suggestive of parasitization by dermatophytes and the presence of lesions consistent with tinea on other parts of the body oblige careful examination of the scalp. Children with scaling of the scalp accompanied by pruritus and any child with alopecia should be carefully assessed. Examination with Wood light is a useful technique, since it is inexpensive, accessible, and practical. Its importance centers on its ability to act as a guide for the identification of the microorganism involved—it will be negative in cases of tinea involving *Trichophyton* species since these usually involve endothrix infection, which does not produce the characteristic yellowish-green fluorescence observed with organisms that involve ecto-endothrix infection such as *M audouinii* and *M canis*. Examination can also be done with dermatoscopy.[11,12]

Direct Examination and Culture

Examination of the scalp should include a search for broken hairs or black dots, which can be collected with tweezers for mycologic analysis.[3,11] Direct examination is an indispensable mycologic test performed with KOH and dimethylsulfoxide or chlorazol black. If a fluorescence microscope is available it can be used for observation of calcofluor white. Material can be easily obtained with a scalpel blade or the edge of a microscope slide; it is then place on a slide and examined for hyphae or spores in or around the hair. The sample should also be inoculated in fungal culture medium such as Sabouraud agar or medium containing antibiotics (Mycosel); growth of colonies is observed in 1 to 6 weeks. It is important to be familiar with nontraumatic techniques for obtaining samples in children. Rubbing the affected area of scalp with gauze moistened with sterile saline is one such method that is also simple and effective; alternatively, a sterile toothbrush or even an agar plate can be used.[11,13] There have been reports of sterile swabs used to obtain samples in cases of inflammatory tinea, since in those cases it is common for no material to be

obtained and this approach allows samples to be obtained from a larger surface area.[26,32]

Immunologic Study

The immune response to dermatophytes has been studied. However, no parameters have been defined for its behavior. In general, trichophytin extracted from *T mentagrophytes* is used as the antigen. Its antigenic determinant is a galactomannan peptide that is cross-reactive with other dermatophytes. When applied intradermally it generates 2 responses: an immediate type I response and a delayed type IV response. The first is positive in chronic cases or atopic individuals and is more associated with the polysaccharide fraction, while the second occurs in acute cases such as kerion and is associated with the peptide fraction.[6]

Histopathology

Histopathology usually reveals the presence of arthrospores in the hair follicles or stratum corneum. There may be growth of hyphae and formation of arthroconidia on the surface of the hair (ectothrix) or inside the hair shaft (endothrix). In tinea favosa, the hyphae are located in the stratum corneum, in the hair shaft, and in the cup-shaped crusts. Follicular atrophy occurs along with chronic inflammatory infiltration in the dermis. The histopathology findings in kerion celsi can be classified according to the pattern of inflammation as follows: perifolliculitis (PF), suppurative folliculitis (SF), SF with suppurative dermatitis (SD), SF with granulomatous suppurative dermatitis (GSD), and GSD with fibrosing dermatitis (FD). In a study undertaken in 19 cases of kerion celsi, the following were observed: SF in 11%, SF with DS in 37%, SF with GSD in 26%, and GSD with FD in 26%. The following dermatophytes were isolated: *M canis* in 6 cases (32%), *T mentagrophytes* in 5 (27%), *T tonsurans* in 4 (21%), *M gypseum* in 1 (5%), and *T rubrum* in 2 (10%). The 2 cases in which only SF with minimal dermal infiltrate was observed were both due to *M canis*. In that study, the 4 controls with noninflammatory tinea displayed minimal primarily lymphocytic perifollicular and perivascular infiltration; of these 4 cases, 3 were due to *M canis*.[33,34]

Differential Diagnosis

The differential diagnosis for tinea capitis should include various conditions, depending on the clinical presentation, such as alopecia areata, bacterial folliculitis, seborrheic dermatitis, atopic dermatitis, psoriasis, trichotillomania, and trichorrhexis nodosa (Table 3).[3,5,12] For instance, inflammatory tinea should be distinguished from conditions that manifest with vesicles and pustules on the scalp, such as folliculitis decalvans and erosive pustular dermatitis of the scalp, and in cases of scarring with alopecia areata.[26]

Table 3. Differential Diagnosis According to Clinical Presentation

Clinical Appearance	*Differential Diagnosis*
Diffuse scaling (noninflammatory)	Seborrheic and atopic dermatitis, psoriasis
Alopecia plaque (noninflammatory)	Seborrheic and atopic dermatitis, psoriasis
Black dots	Alopecia areata, trichotillomania
Diffuse pustular (inflammatory)	Bacterial folliculitis
kerion celsi (inflammatory)	Abscess, tumor

Treatment

The aim of treatment is to achieve clinical and mycologic cure as soon as possible, and in the majority of cases treatment with oral antifungals is necessary.

Topical Treatment

Topical treatment alone is not usually recommended because preparations of this type do not penetrate the hair shaft adequately; an exception is only made to eliminate the parasite in breastfeeding mothers, mainly in recent infections. However, topical treatment can reduce the risk of transmission at the beginning of systemic treatment. In 1982, Allen et al[35] reported that the use of a shampoo containing 2% selenium sulfide was effective in reducing the number of viable spores in the scalp of pediatric patients treated in parallel with griseofulvin, and more recently, shampoos containing 2% ketoconazole have been used with similar results. Patients should be told to use the shampoo 3 times a week and to leave it in contact with the scalp for at least 5 minutes before rinsing. It should be used until the patient is clinically and mycologically cured.[2]

Griseofulvin

In 1958, Williams and Marten documented the effectiveness of oral treatment with griseofulvin for tinea

capitis, and the use of this drug was responsible for a significant reduction in epidemics. Thanks to the introduction of griseofulvin, the use of X-rays for depilation, which had been used since it was first proposed by Sabouraud in the late 19th century, could also be abandoned. The use of thallium acetate could also be abandoned. Nevertheless, tinea capitis continues to be a common pediatric disease affecting 10% to 20% of the population during epidemic outbreaks.

Since the end of the 1950s, griseofulvin has represented the treatment of choice for tinea capitis, although the dose and duration of treatment may vary according to the patient. It is generally used at 10 to 20 mg/kg/d for 8 to 12 weeks (Table 4).

Griseofulvin is fungistatic and inhibits the synthesis of nucleic acids and interrupts cell division in metaphase, thereby preventing synthesis of the fungal cell wall. It also has an anti-inflammatory action. The drug is available in tablet form or as a suspension and it is recommended that it be taken with fatty foods, since this increases the absorption of the drug and improves its bioavailability. The recommended dose varies according to the formula used, and some authors recommend higher doses for micronized griseofulvin. The duration of treatment depends on the causative agent (*T tonsurans* may require longer treatment regimens). The adverse effects include nausea and exanthematous eruptions in 8% to 15% of cases. It is contraindicated during pregnancy.

Various studies have compared griseofulvin with ketoconazole for the treatment of tinea capitis in children, and it has been found that ketoconazole is safe and effective, although it has not been shown to be better than griseofulvin, which has been demonstrated to have a more rapid effect. Griseofulvin is relatively safe and effective in children when administered at appropriate doses.[11,18]

Terbinafine

Terbinafine is an allylamine fungicide with a high affinity for keratin and acts on the cell membrane of the fungus.

Table 4. Recommended Treatment[a]

Drug	*Recommended Dose*	*Duration*
Griseofulvin	10-20 mg/kg/d	8-12 wk
Terbinafine	<20 kg, 62.5 mg; <40 kg, 125 mg; >40 kg, 250 mg	4- wk[b]
Itraconazole	5 mg/kg/d	1-4 wk

[a]Source: Jones[38]
[b]Longer for tinea due to *Microsporum* species

It is effective against all dermatophytes. It is at least as effective as griseofulvin and is safe for the management of tinea due to *Trichophyton* species in children, while its role in the management of tinea caused by *Microsporum* species is debatable; it has been suggested that higher doses are required for longer periods (more than 4 weeks) in these cases. The dose depends on the weight of the patient and is generally recommended at between 3 and 6 mg/kg/d (Table 4). In terms of adverse effects, gastrointestinal complaints and eruptions can occur in 5% and 3% of cases, respectively. In a study undertaken in 50 children, of which 49 had tinea due to *Trichophyton* species and only 1 due to *Microsporum* species, clinical and mycologic cure was obtained in more than 86% after 2 weeks of treatment; the authors of that study suggested 2 additional weeks of treatment in cases involving *Microsporum* species. In another study evaluating treatment with terbinafine in 152 children, the clinical and mycologic efficacy was very good, with a rate of cure of 96% at the end of the treatment; in that study the authors recommended treatment for 4 weeks for tinea capitis in children.[36-38]

Itraconazole

Itraconazole has both fungistatic and fungicidal activity, depending on its tissue concentration, but as for other azoles, its principal mechanism of action is fungistatic, through depletion of ergosterol in the cell membrane, leading to alteration of membrane permeability. A dose of 100 mg/d for 4 weeks is recommended or 5 mg/kg/d in children, in which it is as effective as griseofulvin and terbinafine (Table 4). It is highly lipophilic and keratinophilic, and it persists in the stratum corneum for 3 to 4 weeks after suspension of treatment, allowing it to be used in pulses of 1 week separated by periods of 2 weeks without treatment.[39,40]

Fluconazole

Fluconazole is an antifungal with a broad spectrum of activity against a variety of dermatophytes and *Candida* species. It has good bioavailability, low protein binding, and a long half-life. In a study performed in children with tinea capitis due to *T tonsurans* the drug was found to be effective and safe at a dose of 6 mg/kg/d for 20 days. In that study, 89% of the patients displayed continued clinical and mycologic cure 4 months after completion of treatment.[41]

Treatment of Inflammatory Tinea

Due to the high risk of scarring alopecia the timely initiation of treatment is very important. In kerion, the same

treatments have proven effective at similar doses; therefore, once a diagnosis has been made, administration of griseofulvin or ketoconazole should be initiated immediately, and after a month significant clinical improvement is observed. The use of corticosteroids should be carefully considered in these patients and contraindications for their use should be ruled out. Intralesional corticosteroids can be used in localized disease but systemic treatment should be used when the condition is diffuse; prednisone is generally used at a dose of 1 mg/kg/d for 1 or 2 weeks.[4,27]

Conclusions

Tinea capitis is the most common cutaneous mycosis in children and represents a public health problem in many parts of the world; in addition, it is probably underdiagnosed as a result of the wide range of clinical presentations that can be observed. Systemic treatment is required to achieve clinical cure and the diagnosis should be confirmed by mycology prior to initiation of treatment.

Conflicts of Interest
The authors declare no conflicts of interest.

References

1. Fitzpatrick's Dermatology in General Medicine. 6th ed. New York: Mc Graw Hill; 2003;II:1994-6.
2. Elewski BE. Treatment of tinea capitis: beyond griseofulvin. J Am Acad Dermatol. 1999;40:S27-30.
3. Schwartz RA, Janniger CK. Tinea Capitis. Cutis. 1995,55: 29-33.
4. Sperling LC. Major. Inflammatory Tinea Capitis (Kerion) Mimicking Dissecting Cellulitis. Int J Dermatol. 1999;30: 190-2.
5. Bronson DB, Foley MC. 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.
6. Bonifaz A, Perusquía AM, Saúl A. Estudio clínico-micológico de 125 casos de tiña de la cabeza. Bol Med Hosp Infant Mex. 1996;53:72-8.
7. Medina M, Padilla M, Fernández R, Arenas R, Bonifaz A. Tiña de la cabeza en adultos: estudio clínico, micológico y epidemiológico de 30 casos en la ciudad de México. Piel. 2003;18:403-8.
8. Bonifaz A. Micología médica básica. 2nd ed. México: Méndez-Cervantes; 2005. p. 44-9.
9. Navarrete O, Vázquez H, Arenas R. Tiña de la cabeza en el anciano. Dermatología Rev Mex. 1999;43:123-6.
10. Arenas R. Dermatofitosis en México. Rev Iberoam Micol. 2002;19:63-7.
11. Elewski BE. Cutaneous mycoses in children. Br J Dermatol. 1996:134 Suppl 46:7-11.
12. Elewski BE. Tinea capitis: A current perpspective. J Am Acad Dermatol. 2000;42 1 Pt 1:1-20.
13. Raza A. Ecology and epidemiology of dermatophyte infections. J Am Acad Dermatol. 1994;31 3 Pt 2:S21-S4.
14. Cuétara MS, del Palacio A, Pereiro M, Noriega AR. Prevalence of undetected tinea capitis in a prospective school survey in Madrid: emergence of new causative fungi. Br J Dermatol. 1998;138:658-60.
15. Schwinn A, Ebert J, Brocker EB. Frequency of Trichophyton rubrum in tinea capitis. Mycoses. 1995;38:1-7.
16. Chouaki T, Peguet C, Teillac-Hamel D, Raccurt CP, Hennequin C. Épidémies scolaires de teignes á dermatophytes zoophiles. J Mycol Med. 1999;9:233-4.
17. Fuller LC, Child FC, Higgins EM. Tinea capitis in southeast London: an outbreak of Trichophyton tonsurans infection. Br J Dermatol. 1997;136:132-48.
18. Hebert AA. Tinea Capitis. Arch Dermatol. 1988:124: 1554-7.
19. Ghannoum M, Isham N, Hajjeh R, Cano M, Al-Hasawi F, Yearick D, et al. Tinea capitis in Cleveland: Survey of elementary school students. J Am Acad Dermatol. 2003;48: 189-93.
20. Arenas R, Bonifaz A, Padilla MC. Micosis Superficiales. Tercera Revisión del Consenso Nacional de Prevención, Diagnóstico y Tratamiento. México; 2006.
21. Martínez-Suárez H, Guevara-Cabrera N, Mena C, Valencia A, Araiza J, Bonifaz A. Tiña de la cabeza. Reporte de 122 casos. Dermatología Cosmética, Médica y Quirúrgica. 2007; 5:9-14.
22. Ibarra G, Arenas R, Reynoso S. Tiña inflamatoria por Trichophyton ochraceum. Primer caso humano en México. Dermatol Rev Mex. 1983;27:27-38.
23. Isa-Isa R, Reyes A, Cruz AC, Bencosme I. Tinea Capitis. Características Clínicas y Epidemiológicas IDCP Enero-Mayo 1996. Rev Dom Dermatol. 1998;25:27-30.
24. Jhangir M, Hussain I, Khushid K, Haroon TS. A clinicoetiologic correlation in tinea capitis. Int J Dermatol. 1999;38:275-8.
25. El Euch D, Mokni M, Sellami A, Cherif F, Azaiz MI, Osman BD. Les teignes du cuir chevelu observées á Tunis de 1985 á 1998: á propos de 1222 cas. J Mycol Med. 2001;11:87-91.
26. Amorim F, Sidta M, Beatriz C. Tinea capitis in street-children of Maputo, Mozambique. J Mycol Med. 2001;11: 92-4.
27. Bojórquez MA, Arenas R, Molina de Soschin D, Veja-Memije ME. Granuloma tricofítico y querion de Celso: datos clínicos, micológicos e histopatológicos de nueve casos. Dermatología Rev Mex. 2002;46:15-22.
28. Babel DE, Baughman SA. Evaluation of the adult carrier state in juvenile tinea capitis caused by Trichophyton tonsurans. J Am Acad Dermatol. 1989;21:1209-12.
29. Hall S, Sarai K, Babel G. Scalp Colonization by Trichophyton tonsurans in an Urban Pediatric Clinic. Arch Dermatol. 1988;124:1511-3.
30. Viguié-Vallanet C, Serre M, Masliah L, Tourte-Schaefer C. Épidemie de teignes á Trichophyton tonsurans dans una école maternelle de la région parisenne. Ann Dermatol Venereol. 2005;132:432-8.
31. Borchers Samia W. Moistened gauze technic to aid in diagnosis of tinea capitis. J Am Acad Dermatol. 1985;13: 672-3.
32. Head ES, Henry JC, Macdonald EM. The cotton swab technic for the cultura of dermatophyte infections-its efficacy and merit. J Am Acad Dermatol. 1984;11 5 Pt I:797-801.

33. Barnhill RL, Crowson AN. Textbook of dermatopathology. 2nd ed. Miami Lakes, Florida: McGraw Hill; 2004. p. 481-2.
34. Arenas R, Toussaint S, Isa-Isa R. Kerion and dermatophytic granuloma. Mycological and histopathological findings in 19 children with inflammatory tinea capitis of the scalp. Int J Dermatol. 2006;45:215-9.
35. Allen HB, Honig PJ, Leyden JJ, McGinley KJ. Selenium sulfide: adjunctive therapy for tinea capitis. Pediatrics. 1982;69:81-3.
36. Baudrez-Rosselet F, Monod M, Jaccoud S, Frenk E. Efficacy of terbinafine treatment of tinea capitis in children varies according to the dermatophyte species. Br J Dermatol. 1996;135:1011-2.
37. Krafchik B, Pelletier J. An open study of tinea capitis in 50 children treated with a 2-week course of oral terbinafine. J Am Acad Dermatol. 1999;41:60-3.
38. Jones TC. Overview of the use of terbinafine in children. Br J Dermatol. 1995;132:683-9.
39. Higgins L, Fuller C, Smith CH. Guidelines for the management of tinea capitis. Br J Dermatol. 2000;143:53-8.
40. Gupta AK, Alexis ME, Raboobee N, Hofstader SLR, Lynde CW, Adam P, et al. Itraconazole pulse therapy is effective in the teatment of tinea capitis in children: an open multicentre study. Br J Dermatol. 1997;137:251-4.
41. Solomon BA, Collins R, Sharma R, Silverberg N, Jain AR, Sedgh J, et al. Fluconazole for the treatment of tinea capitis in children. J Am Acad Dermatol. 1997;37:274-5.