

# **Tinea Capitis: A Practical Approach**

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#### **Mini Review**

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#### Abstract

Tinea capitis is a common superficial fungal infection of the scalp in children. It is caused by dermatophytes mainly *Microsporum canis* and *Trichophyton tonsurans* and can be acquired from the environment or infected living beings. Clinical manifestations go from mild scaling with little hair loss to large inflammatory and alopecic pustular plaques. Trichoscopic patterns such as comma hairs, corkscrew or code-like hairs can suggest and support the diagnosis as well as clinical follow up. Treatment must be chosen due to causal agent for a successful outcome.

**Keywords:** *Trichophyton spp; Microsporum spp;* Dermatophytes; *Trichophyton Tonsurans* 

# Introduction

Tinea capitis is a superficial fungal infection of the scalp, hair shaft, eyebrows and eyelashes, also referred as ringworm and it remains a major public health concern in urban areas. It is caused by dermatophytes of Trichophyton and Microsporum genera [1,2].

It remains the most common superficial fungal infection in children. The causal agent's distribution has diverse geographical variations depending on several factors such as socio-economic level, population, migration among others.

Infection can be acquired from the environment, wild animals, pets or people with this dermatophytes infection. Also, there are specific conditions that favor the growth of the fungus such as humidity, heath, extended use of corticosteroids and lack of hygiene [3].

Clinical manifestations are varied from mild scaling with little hair loss to large inflammatory and pustular plaques with extensive alopecia. A simple non-invasive technique for diagnosis and follow-up is trichoscopy. Specific aspects of the hair have been described like: comma hair, corkscrew hair, bar code-like hair and zigzag hair [4,5].

Diagnosis is achieved by focused clinical history, clinical examination, mycological and trichoscopical study. Treatment will depend on causal agent, age and weight.

Our goal is to analyze current data within the last years about this infection.

# **Epidemiology**

Principal changes related to epidemiology of tinea capitis in latest years has been the rise of *M. canis* as the dominant fungal agent in some parts of Europe and the spread of *T. tonsurans* in urban communities in USA, Western Europe, UK and France [6,7].

*T. tonsurans* has spread to both South America and West Africa [8,9]. In Far Eastern countries has mainly been associated with infection in older children and wrestling in adults [10]. Trends in tinea capitis and migration, has also increase infections due to *Trichophyton violaceum* and *Microsporum audouinii* in countries that were previously not common [11,12].

However, the rise in tinea capitis due to *T. tonsurans* in any country has presented a challenge to both treatment and community infection control.

# **Clinical Features**

Tinea capitis is caused by many species of dermatophytes which infect hair follicles and contiguous skin. It affects children from 2to12 years old, but predominantly between 3-7 and it is more commonly observed in a low socioeconomic population [2,13].

Even though this fungal infection it's considered an "only child's" disease, a growing number of tinea capitis cases have been reported in the past years among adults, particularly menopausal, elderly women and immunocompromised patients [13].

The clinical appearance of tinea capitis will in part depend on the type of invasion affecting our patient [14].

- Ectothrix Form: The hair shaft is infected at midfollicle level and hyphae grow towards hair bulb. Fluorescence under Wood's light its characteristically present in infections caused by Microsporum species. There is hair loss with hair shafts breaking 2-3 mm above the scalp level.
- Endothermic Form: Almost all hairs break at scalp level, leaving swollen hair stubs inside the follicles appearing "black dots". Trichophyton species does not fluorescence under Wood's light [15].

Another clinical feature will depend on level of host resistance and inflammatory host response; it will vary from undetectable with a few broken hairs, little scaling and inflammation to a severe, painful, boggy mass.



Figure 1: Trichophytic tinea due to *T. tonsurans.* 



Figure 2: Microsporic tinea due to M. canis.



Figure 3: Kerion Clesi.

Tinea capitis can be classified in two types: Dry variety (conformed by "*microsporic tinea*" and "*trichophytic tinea*"), mainly cause by *Mycrosporum canis* and *Trichophyton tonsurans*. Inflammatory variety also called Kerion Celsi, mainly caused by *T. mentagrophytes*, *M. canis* and rarely *T. verrucosum*. In all types partial hair loss is present [15] (Figures 1-3).

# Dry, Non-Inflammatory Variety

- Microsporum specie: Lesions can be alopecia patches mostly circular, broken hairs at same height and characteristic fine scaling but little inflammation
- Trichophyton specie: Lesions can be diffuse alopecia with little and irregular plaques interspersed with healthy hairs.

#### **Trichocospic Features**

Trichoscopy allows in vivo visualization infected hair patterns and epidermal portion of hair follicles, which cannot be seen by the naked eye [13,16].

Typical trichoscopic findings can support the diagnosis but mycological examination remains the gold standard for confirmation of tinea capitis [17,18].

One of the main dermoscopic markers of tinea capitis is comma hairs, which appear as short C-shaped hairs due to disintegrated, cracked, and bent hairs caused by multiple hyphae within the hair shaft. Corkscrew hairs were proposed as new trichoscopy finding of tinea capitis in patients of African descent due the shape of the broken hair shaft. Nevertheless, Hughes et al. declared that comma hair and corkscrew are the main trichoscopic findings; they pointed out that corkscrew hair is a variant of comma hair in African patients and is a marker for endothrix tinea capitis [17-19]. But in our experience both patterns can be found in Caucasians, blacks and mestizo patients (Figures 4 & 5).



**Figure 4:** Dermoscopic findings, comma and corkscrew hairs.



Figure 5: Kerion Celsi, zig zag hairs.

The recently described bar code-like hairs also called Morse code hair is a specific dermoscopic patterns of fungal infection of the scalp. The features of the bar codelike hair are horizontal white bands which can be better observed by polarized dermoscopic. Under ultraviolet dermoscopy bright-green fluorescence in hair shaft is present and bright-white in accumulated a scale, that disappears at the bands of the bar code-like hairs [20]. Campos S et al performed a non-randomized prospective clinical study with fifty patients under 13 years of age and the most frequent dermoscopic findings were perifollicular scaling in 94%, diffuse scaling 90%, short broken hairs 74%, black dots 34%, corkscrew hairs 30%, comma hairs 20%, pustules 18% and zigzag hairs 12% [21]. After treatment a progressive disappearance of hair alterations it's observed and at follow-up all patients had a negative mycological examination and dystrophic hairs were not observed [21].

Richarz N, et al. [22] proposed trichoscopy as a fast, non-invasive method for monitoring treatment; they reported the resolution of corkscrew hair after an antifungal therapy. It predicts a clinical cure accurately, allowing us to decide to stop antifungal oral therapy.

#### Treatment

Tinea capitis treatment should be started once the clinical diagnosis and the direct examination with KOH-Chlorazol black are confirmative. It is not recommended to wait for the fungal culture to grow because delaying treatment increases the risk for disease progression, permanent hair loss, and transmission [3,23].

There is very low or none clinical improvement with antifungals such as voriconazole or posaconazole. Tinea capitis require clinical follow-up to determine if oral antifungal treatment should be discontinued, changed or if additional treatment is needed [23,24].

Topical therapy alone is not recommended, they have an inadequate penetration of the hair follicles and may miss areas of subclinical infection. Povidone-iodine, ketoconazole 2%, selenium sulfide 1-2.5% and ciclopirox shampoo have shown some efficacy to reduce the risk of spreading, transmission to other children and decreasing the risk for reinfection [25,26].

As a general rule in tinea capitis, terbinafine is more effective against Trichophyton spp (*T. tonsurans, T. violaceum, T. soudanense*), as opposed to griseofulvin and itraconazole which are more effective against Microsporum spp (*M. canis, M. audouinii*) [3].

Griseofulvin and terbinafine are the two drugs approved by the US Food and Drug Administration (FDA) and the mainly used by the same reason. Ketoconazole should not be used for tinea capitis, because of the risk of severe liver injury and adrenal insufficiency [3,19,23,24,27].

#### **First Line Treatment**

Optimal treatment must be chosen according to the etiological dermatophyte involved [3,23,24,27] (Table 1).

Trichophyton spp.	Terbinafine
Trichophyton tonsurans	
<i>Trichophyton violaceum / soudanense</i>	
Microsporum spp.	Griseofulvine or Itraconazole
Trichophyton spp.	Terbinafine
Trichophyton tonsurans	

 Table 1: Treatment in tinea capitis depending causal agent.

**Griseofulvin:** It is a fungi static drug with micronized, ultra-micronized and suspension forms, not always available in some countries [28]. The recommended dose in children is 15to20mg/kg per day in single or divided doses for 6to8 weeks. In resistant cases doses up to 25mg/kg can be necessary. Griseofulvin effectiveness is low against *Trichophyton spp.* [29]. A meta-analysis comparing griseofulvin and terbinafine has shown that griseofulvin should be the first-line treatment for children in whom the etiological agent is unclear [30].

Griseofulvin is contraindicated in lupus erythematosus, porphyria, liver disease and pregnancy [29,31].

**Terbinafine:** Terbinafine is an alternative first-line treatment and considered the optimal choice, due to shorter courses, cost-efficiency, and compliance [29]. A meta-analysis showed that the effectiveness of 2to4 week's treatment with terbinafine is similar to the 6to8 week's treatment with griseofulvin for *T. tonsurans* [32-35]. A new granule formulation available in 125-mg or 187.5-mg packets to be sprinkled on food has been licensed in the USA for children over four years of age [23,30,36].

#### Second Line Treatment

**Itraconazole:** It has both fungicidal and fungistatic activity. Doses of 50 to 100 daily mg for four weeks have comparable efficacy with griseofulvin or terbinafine [23,31].

Pulse intermittent dosing regimens are effective. This is recommended at 5 mg/kg per day for one week each month during two to three months. In the U.K. it is not recommended for the treatment of tinea capitis in children aged 12 years and under [23,39]. Liver function tests are indicated in patients treated with azolic derivates for over a month or before starting treatment in patients with pre-existing hepatic dysfunction [3,39,40].

**Fluconazole:** It is considered an alternative to terbinafine. It can be administered as a pulse therapy of 6mg/kg once a week for 6to12 weeks. Serious side effects including hepatoxicity, severe drug hypersensitivity reactions (Stevens-Johnson syndrome), anaphylaxis, torsade de pointes, and prolongation of the QT interval, are rare [23,41-43].

**Infants under the Age of 1 Year:** Tinea capitis is uncommon in infants under the age of one year and there is no a consensus about treatment although Fluconazole, griseofulvin, and terbinafine have shown efficacy in this population [44,45].

**Treatment in Adults:** Drug selection and treatment duration criteria in adult tinea capitis are similar to the ones used for children; however, optimal regimens still unclear. The recommended doses of antifungals in adults are: Terbinafine 250mg per day, Itraconazole 5mg/kg per day (maximum 400mg/day), fluconazole 6mg/kg per day, and ultra-micronized griseofulvin 10 to 15mg/kg per day (maximum 750mg/day) [23,41-43].

**Kerion Celsi:** It is recommended to combine antifungal therapy with oral prednisone in order to prevent scaring alopecia in 0.5mg/kg/day for two weeks. Wet compresses are useful to remove exudates and crusts when bacterial infection its suspected. In small cases, topical or systemic antibiotics can be prescribed [23,44,45].

# Comments

Tinea capitis is a fungal infection caused by dermatophytes of the genera *Microsporum spp* and *Trichophyton spp*. The hair shaft is infected as *Ectothrix form* in the first one and *Endothrix form* in the second one. Clinical manifestations can be classified in two types: the dry variety (microsporic and trichophytic), and the inflammatory form (kerion Celsi). Trichoscopic findings as comma hairs, corkscrew and code-like hairs can support clinical diagnosis. Treatment must be chosen according with causal agents, terbinafine is more effective against *Trichophyton spp* and griseofulvin and itraconazole are more effective against *Microsporum spp*.

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