# Tinea capitis: a review

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Tinea capitis (TC) is a dermatophyte infection of the scalp hair follicles and intervening skin, mainly caused by anthropophilic and zoophilic species of the genera *Trichophyton* and *Microsporum*.

**Key Words**
Tinea capitis, dermatophyte infection, management of tinea capitis infection

## *EPIDEMIOLOGY*

It is said to be the most common fungal infection in childhood. Its incidence in children is 8% (1). Blacks are affected more than whites (1,2).
It mostly affects children between 6 and 10 years of age and is less common after age 16 or in infants. In adults it is rare and men are almost never affected (2).

## *ETIOLOGY/ DEMOGRAPHY*

It varies from country to country; species tend to change in time due to immigration (2). In Latin America, the principal agents of tinea capitis are *M. canis* and *T. tonsurans*; in Mexico *M. canis* is the commonest agent (91%), followed by *T. tonsurans* (9%). In India, *T. violaceum* is the most common agent. In Western Europe and the United States *T. tonsurans* causes 90% of infections. Cats are its main reservoir of infection. (1-4).

## *TRANSMISSION*

Person-to-person, animal-to-person, via fomites transmission can occur. Spores may be present on asymptomatic carriers, animals or inanimate objects (2).

## *PATHOGENESIS*

Scalp hair traps fungi from the environment or fomites. Dermatophytes initially invade the scalp stratum corneum, which is followed by hair shaft infection and spread to other hair follicles ensues.
Fungi release enzymes (keratinases, metalloproteases, and serine proteases) and also produce lipases and ceramides. The released enzymes, aside from breaking the bonds of the keratinized tissue, behave as antigens and induce various degrees of inflammation. As a result, tissue damage due to a combination of dermatophyte enzymatic action and the defense mechanisms activated during the inflammatory processes occurs.
Infections caused by dermatophytes generally induce a mainly Th1-type adaptive immune response with production of proinflammatory cytokines like interleukin (IL)-2 and interferon (IFN)-γ. The immunologic response varies among the different species of dermatophytes. It is more intense when the infection is caused by zoophilic (e.g. *M. canis*) or geophilic dermatophytes (e.g. *M. gypseum* complex) and is weak when the infection is caused by anthropophilic dermatophytes (e.g. *T. tonsurans*) (5).

## *CLINICAL MANIFESTATION*

### History

Two to four days or sometimes weeks after exposure, scaly pruritic patches appear in the scalp together with hair loss. If left untreated, the lesions enlarge. Systemic symptoms may include cervical lymphadenopathy, malaise or fever. Lesions last from weeks to months (1).
Skin symptoms and signs in inflammatory tinea capitis are pain, tenderness, ± alopecia, adenopathy and in non inflammatory infection; scaling, scalp pruritus, diffuse or circumscribed alopecia, occipital or posterior auricular adenopathy (2)

### Physical examination

Clinical appearance varies with type of hair invasion, level of host resistance, degree of inflammatory host response (2).
1. Ectothrix[3] (Infection is on the outside of the hair shaft. Hyphae fragment into arthroconidia, leading to cuticle destruction)
a. Gray patch tinea capitis [4] is dull gray due to the arthrospore coating. Hair becomes brittle; shafts break off close to the scalp surface. Fine scaling with a fairly sharp margin can be observed. It is usually caused by *M. audouinii* and *M. canis* (1,2)
2. Endothrix[4] (Infection is on the inside of the hair shaft without cuticle destruction. Arthroconidia are found within hair shaft)
a."Black dot" tinea capitis[4] shows broken-off hairs near surface that give appearance of black dots (swollen hair shafts) in dark-haired patients, caused by *T. tonsurans* and

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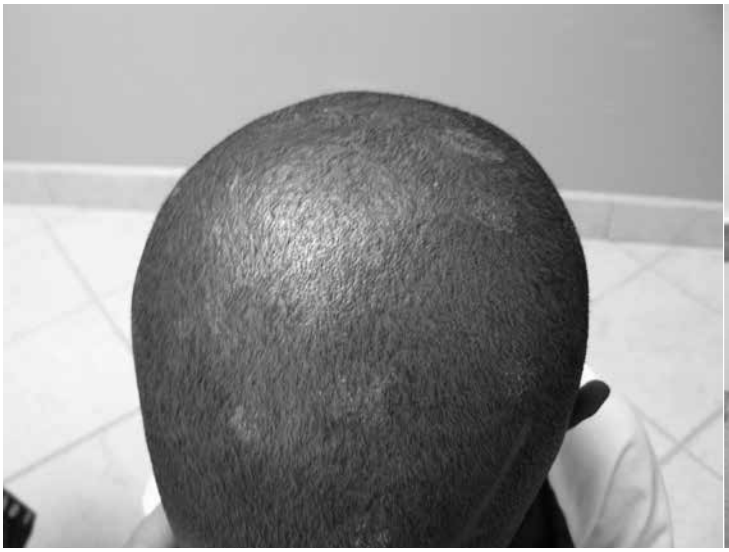
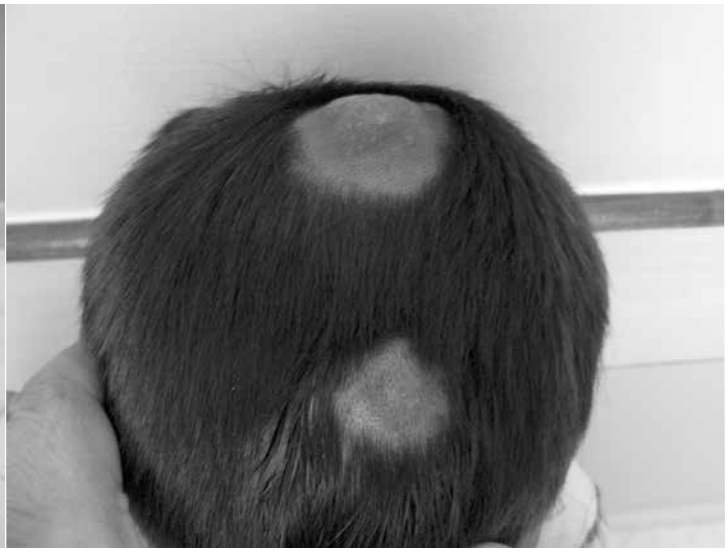


*Tinea capitis "grey patch"*



*Tinea capitis inflammatory type (Kerion celsi)*

*T. violaceum* (1,2)

b. Kerion[4]. A boggy, inflamed painful nodule drains pus. Hairs do not break; but fall out easily and can be pulled out without pain. There are two clinical presentations, kerion celsi and dermatophytic folliculitis. The latter is usually an initial lesion and the former a localized matted mass, but multiple plaques may also be found. If diagnosis and treatment are delayed, kerion causes scarring alopecia. A report from Mexico documented 14 of 19 cases of kerion were caused by *M. canis, T. mentagrophytes* was isolated in 3 cases, and *T. tonsurans* in 2 (1,2, 6,7).

c. **Favus**. Early cases show perifollicular erythema and matting of hair. Late, scutula (yellowish, thick adherent crusts composed of skin debris and hyphae) are present on the scalp infected with *T. schoenleinii*. It has a fetid odor and is endemic in the Middle East and South Africa (2,6)

## *DIAGNOSIS*

### Differential Diagnosis

Differential diagnosis of the various types of tinea capitis infection must be established with respect to different entities:

"Gray patch" tinea capitis: Seborrheic dermatitis, psoriasis, atopic dermatitis.

"Black dot" tinea capitis: seborrheic dermatitis, psoriasis, alopecia areata, chronic cutaneous lupus erythematosus

Kerion: Cellulitis, furuncle, carbuncle

Favus: Impetigo, echtyma, crusted scabies.

### Laboratory examinations

WOOD'S LAMP

Wood's lamp examination should be performed in any patient with scaling scalp lesions or hair loss of undetermined origin.

Wood's lamp reveals bright green hair shafts in scalp infections caused by *M. audouinii* and *M. canis*. *T. tonsurans* does not exhibit fluorescence (2,8).

DIRECT MICROSCOPY

Specimens should include hair roots and skin scales. Examination with 10%-20% KOH shows spores within (endothrix) or surrounding (ectothrix) the hair shaft. Loose chains of arthrospores and airspaces in hair shafts are visible in favus. Cotton swab and toothbrush methods of specimen collection are reliable, quick, and painless (2).

FUNGAL CULTURE

Infected hair or scalp scale can be inoculated into Sabouraud's agar or other DTM media (Dermatophyte test medium) and the causative organism can be identified in 10-14 days. It is still important to identify the causative organism via culture, as this may affect choice of treatment regimen (1, 8).

In diagnosing tinea capitis, clinical symptoms should ideally be confirmed by positive culture, as it will reveal the presence or absence of fungus in addition to the specific species. Specimen samples can also be treated with either potassium hydroxide (KOH) or sodium hydroxide (NaOH) for direct microscopy.

*T. tonsurans* and *Microsporum canis* are the most prevalent pathogens causing tinea capitis in North America and Europe, respectively. Clinical diagnosis can be challenging because of a wide variety of presentations. Infection should be confirmed with mycological examination before initiation of systemic antifungals (8).

## *MANAGEMENT*

Tinea spreads on brushes, combs, towels, pillowcases, and hats. Children with tinea capitis should not share these items. Tinea can also be spread by close contact with infected children. In addition, *T. tonsurans* can exist in an asymptomatic carrier state in children, acting as a reservoir of infection. Therefore it is important to examine home and school contacts of affected children. The current recommendation is that infected children may attend school once treatment has begun (1- 3).

Adjunctive topical therapies such as selenium sulfide (grade

of recommendation B; strength of evidence II a) or ketoconazole (Grade of recommendation B; strength
of evidence III); shampoos, as well as fungicidal creams or lotions have been shown to decrease the carriage of viable spores responsible for disease contagion and reinfection and may shorten the cure time with oral antifungals. The topical fungicidal shampoo or lotion should be applied to the lesions once daily for a week (grade of recommendation C, strength of evidence IV). Patients should be instructed to lather the shampoo and let it sit on the scalp for 5 to 10 minutes before rinsing (1, 2, 9, 10).

Tinea capitis always requires systemic treatment because topical antifungal agents do not penetrate the hair follicle. Despite recommendations that tinea capitis be treated with an oral antifungal, according to a study performed in the U.S.A, only 56% of the diagnosed patients were treated with an oral antifungal agent, and just 17% of these patients used griseofulvin for at least 6 weeks. The short duration of oral antifungal use may imply that patients diagnosed with tinea capitis are still being undertreated or comply poorly with the therapy because of the inconvenient dosage and duration. A delay in diagnosis sometimes leads to severe scarring and inappropriate invasive therapies, such as surgical excisions of presumed abscesses, which are actually inflammatory tinea kerions (11).

Griseofulvin is the only drug approved by the Food and Drug Administration of the U.S. for the treatment of tinea capitis, although randomized controlled trials indicate that terbinafine, itraconazole, and fluconazole are also effective. The main disadvantage of griseofulvin is the long duration of treatment required (6–12 weeks or longer) which may lead to reduced compliance. Also in some countries of Europe, griseofulvin is no longer available. (8,10).

In a metaanalysis conducted in 2006, which reviewed all the studies to that date comparing griseofulvin with terbinafine in the treatment of childhood tinea capitis, results indicated that 2 to 4 weeks of terbinafine is at least as effective as 6 to 8 weeks of griseofulvin in cases of tinea capitis caused by Trichophyton spp. The metaanalysis also indicated that griseofulvin is more effective at treating tinea capitis caused by Microsporum spp. Its efficacy and treatment duration for these cases is matched by that of fluconazole and itraconazole; griseofulvin is cheaper (8-10).

Kerion and other forms of inflammatory tinea capitis and dermatophytic granuloma have an excellent response to griseofulvin, itraconazole, and terbinafine. To prevent alopecia in kerion, an oral corticosteroid should be added early (6). The question of griseofulvin resistance has been mentioned and may be clinically very important in some locales. As a result, the allylamines may be the best approach to therapy, but the cost may be a limitation for its use. If a 6-week course of griseofulvin therapy is not curative, then the next step is to use an imidazole or an allylamine—the dose varying with the different agents (Table 1) (9,10).

Clinical and mycological examinations of the children should be conducted at regular intervals (2–4 weeks). Treatment may be stopped when the culture becomes negative or when hair regrowth is clinically evident; consequently, the duration of treatment must be individualized according to the response (10).

It may be concluded that if the clinical findings suggest that Microsporum species is the pathogenic dermatophyte, such

**TABLE 1.** *Dosing Regimens for the Treatment of Tinea Capitis*

| Antifungal agent | Dosage | Duration of treatment |
|---|---|---|
| Griseofulvin Microsize | 20–25 mg/kg/day | 6–12 weeks or longer until fungal cultures are negative |
| Griseofulvin Ultramicrosize | 10–15 mg/kg/day | |
| Terbinafine | 10–20 kg: 62.5 mg/day<br>20–40 kg: 125 mg/day<br>>40 kg: 250 mg/day<br>Or<br>4–5 mg/kg/day | *Trichophyton* spp.: 2–4 weeks<br>*Microsporum* spp.: 8–12 weeks |
| Itraconazole | Capsules: 5 mg/kg/day<br>Oral solution: 3 mg/kg/day | Daily dosing: 2–6 weeks<br>Pulse regimen (1 week with 2 weeks off between the first 2 pulses and 3 weeks between the 2nd and 3rd): 2–3 pulses (range: 1–5) |
| Fluconazole | Daily dosing: 5–6 mg/kg/day<br>Weekly dosing: 8 mg/kg once weekly | 3–6 weeks<br>8–12 weeks |

*Source: Kakourou T, Uksal U. Guidelines for the management of tinea capitis in children. Pediatr Dermatol. 2010 May-Jun 27;3:226-8*

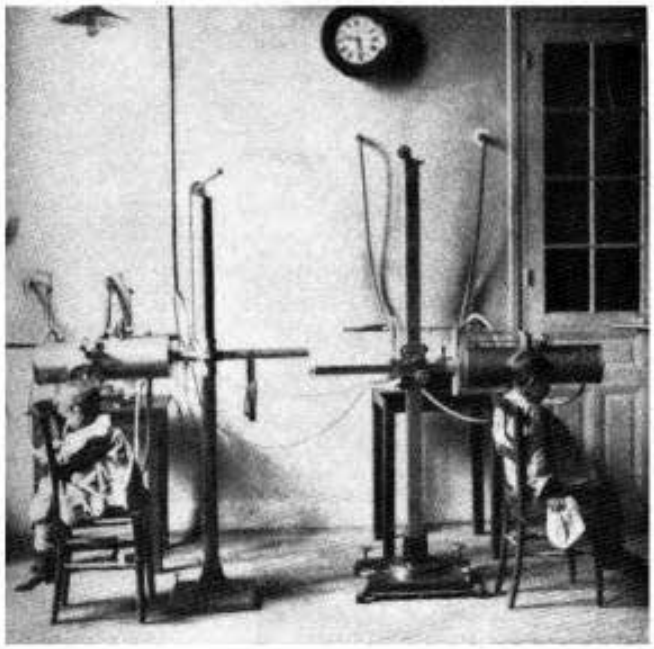


**Fig. 7** Tinea capitis treated with radiotherapy. *Les Teignes*, 1910.

*Source: Negroni R. Historical aspects of dermatomycoses. Clin Dermatol 2010 Mar-Apr 28: 125-132*

as the presence of fluorescence under Wood's lamp examination and contact with an infected cat, dog or rabbit is possible; empirical use of griseofulvin is recommended. In the absence of these features and in populations in which Trichophyton species are the predominant pathogen causing tinea capitis, empirical use of terbinafine is recommended if cost is not an issue (12, 13).

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