



# Adult Tinea Capitis: a Clinical Entity in Increasing Frequency

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## Abstract

**Purpose of Review** This review summarizes the fungal literature currently available for adult tinea capitis (ATC), as well as providing data for clinical practice.

**Recent Findings** Available studies in ATC are scarce; however, they provide essential information for treating ATC.

**Summary** Treatment of TC is effective; however, it needs a minimum of 1 month to watch clinical efficacy. Systemic treatment is often needed to favor drug penetration to the deepest part of the hair follicle. The newest oral antifungal has higher efficacy rates than conventional therapy, as well as much shorter duration of treatment but at higher costs. We review the literature concerning ATC, including treatment schemes.

**Keywords** Adult tinea capitis · Epidemiology · Incidence · Treatment

## Introduction

Superficial mycoses caused by dermatophytes comprise four principal genera: *Trichophyton*, *Microsporum*, *Nannizzia*, and *Epidermophyton*. None of these is part of the microbiota of the skin and may provoke hair diseases, nail thickening or plaques with erythema, and scaling with active borders. The first involves tinea capitis (TC), an infection or parasitization of the hair, scalp, eyebrows, and eyelashes. It is commonly observed in scalp hair related to dermatophytic fungi (*Trichophyton*, *Microsporum*, and *Nannizzia*) and more often in children, although there is a growing number of reports in adults (Table 1) [12•, 13•].

The global epidemiology of TC is varied; *Trichophyton tonsurans* is the most common agent in America, Europe, and Africa, followed by *Microsporum canis*, mostly in

Mediterranean countries. The most important carriers of dermatophytes are cats, dogs, and rabbits [14].

## Epidemiology

Tineas are cosmopolitan mycoses that predominate in tropical environments; these are considered the most frequent fungal diseases, appearing in subjects of any age, race, or sex, as well as any socioeconomic level or occupation [15]

In epidemics caused by *T. tonsurans* affecting the head, infections are usually subclinical and related to fomites; epidemics due to *M. canis* are related to contact with dogs or cats. Some factors are commonly associated with TC, including socioeconomic status (rural and suburban regions), poor hygiene, and overcrowding, usually schools, where the infection spreads rapidly [1•, 16–19].

Shaving hair is one of the most commonly related habits. Immunosuppression is also an increased risk factor for TC, mainly among adults [10, 11].

Although TC is rarely reported in adults, as shown in several studies [1•, 2, 3–5, 12•, 13•, 20], it is far from being exceptional. In a study carried out in the Afro-American population in the USA, the incidence of TC in adults was 11.4% [21]. In a study, Park et al. reported a rate of up to 44% of TC in adults [1•]. However, most studies report an incidence ranging from 2 to 5.8% [2–5].

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In Mexico, TC predominates in rural or suburban areas, and it is more frequent in rural workers and people of low socioeconomic status. It is almost exclusive among children (98%); it often affects women after puberty and around the menopause or the elderly in a frequency ranging from 2 to 2.5% of cases [13•].

It can also occur in immunosuppressed people (HIV/AIDS infection, posttransplant, immunosuppressant, etc.). The asymptomatic carrier status, usually in adults of the same family of the patients, is related to flares in children [12••]. Previous studies have documented a higher incidence of adult TC among menopausal women [6•], explained by the involution of sebaceous glands following a decreased blood estrogen level during menopause [22, 23].

## Etiopathogenesis

The most often associated agents in adult TC include *T. tonsurans*, *T. violaceum*, *T. verrucosum*, *M. canis*, and *N. gypsea* [5].

Dermatophytes reproduce in the stratum corneum, penetrating at the follicular infundibulum, and invades the hair sheath; it extends to the depth without exceeding the keratogenic zone (Adamson's stripe) (the growth of hyphae and arthroconidia occurs in the opposite direction of hair growth) [13•]. Simultaneously, it extends towards the distal part of the hair developing a thick, fragile, and easily breaking hair [12••, 13•].

Typically, the growth of the fungus takes place inside the hair bulb, generating spores without affecting the cuticle (endothrix) or affecting it (ectothrix). These infections develop in parallel with hair growth [12••, 13•]. The keratinocyte fungal adhesion favors the infection from one host to another, which eventually progresses into an active infection [24].

Fungal proteolytic enzymes favor invasion even in hostile media (acid or alkaline) [24]. Protease genes are variably expressed with different enzyme patterns in human infection or in vitro cultures [25].

In some patients, an intense inflammatory reaction occurs; in others, it is minimal, and there may be asymptomatic commensalism between fungus and host [1•]. The degree of response relies on two aspects: (1) the causal species; strains of granular morphology have high production of enzymes; it is also probable that some strains produce substances that expel near bacteria; arthroconidia production is related to the parasitic form (the zoophilic and geophilic strains generate intense inflammation), and (2) the degree of host hypersensitivity [12••, 13•].

Increased cellular immunity and a delayed hypersensitivity reaction contribute to clinical and mycological cure; while defects of cellular immunity and the hypersensitivity reaction

as occurring among adults, may predispose to chronic and recurrent dermatophytosis [6•, 13•].

The relative resistance to TC after puberty is probably due to the presence of long-chain fatty acids; however, in adults, mainly in postmenopausal women, there are quantitative and qualitative variations of cutaneous sebum, in particular of medium-chain fatty acids [26].

## Clinical Manifestations

TC is commonly seen in children, and rarely seen among adults. *M. canis* and *T. tonsurans* are the causal agents identified in Mexico and many Latin American countries. TC can be dry or inflammatory, although the state of asymptomatic carrier and a clinical presentation with diffuse flake pityriasis capitis-type are present when the causative agent is *T. tonsurans* [13•].

The clinical appearance of TC is determined by the ability of the fungus to invade the hair, and the host immune status. The clinical expressions vary from an asymptomatic carrier to severe inflammatory presentations that develop scarring alopecia. Most affected patients are children from 6 months to 12 years [27, 28]. The typical clinical pattern includes broken hairs with scaling; however, this image has changed due to the geographical distribution of the infectious species [29].

Adult TC (ATC) has a different presentation than in children. In a study by Park et al [1•], they found that the most common clinical type of ATC was pustular and similar to seborrheic dermatitis (26.8% and 25.5%, respectively), initially diagnosed in about 65.9% of cases such as folliculitis or dermatitis seborrheic, which contributed to the delay in diagnosis in up to 7–8 months; usually in children's TC the duration in confirmation of the diagnosis is less than 1 month. The authors observed that more than half of patients with ATC (62.2%) had no obvious alopecia. The low frequency of the disease as well as the atypical presentation can contribute to delayed diagnosis, so ATC requires a high index of suspicion, and a mycological study should be performed on adult patients, mainly the elderly with inflammatory changes in the scalp (Fig. 1a) even without an alopecia patch.

The carrier status is considered in non-clinically asymptomatic infections. This status is usually evident in children or parents, becoming potential reservoirs of fungi and thus spreading the infection [11, 13•].

## Diagnosis

### Direct Examination

Direct examination shows long, tabicated, and arthrospored hyphae. In the trichoscopic study, when viewing with the

**Table 1** Characteristics of the studies of adult tinea capitis

Region/country	Period	Adult cases (%)	Main dermatophyte	Mean age (range)	Sex ratio (M:F)	Other site of involvement (%)	Infection related to animals (%)	Chronic underlying disease	Treatment	Ref
Jeonju/Korea	2000–17	82 (44.3)	<i>M. canis</i>	66.9 ± 15.8 (20–90)	1:3.1	46.2	12.8	74.4	Terbinafine 250 mg/day, itraconazole 200 mg/day, topical antifungal agent	[1•]
Daegu/Korea	1978–88	41 (2)	<i>M. canis</i>	58.5 (21–85)	1:19.5	14.6	31.7	N/A	Griseofulvin 500 mg/day 1–30 weeks	[2]
Cagliari/Italy	1973–94	17 (N/A)	<i>M. canis</i>	N/A (17–76)	All female	N/A	N/A	29.4	Griseofulvin 25 mg/kg/day or terbinafine 250 mg/day, topical antifungal 40–50 days	[3]
Sousse/Tunisia	1990–2005	60 (5.27)	<i>T. violaceum</i>	34.5 (18–75)	N/A	N/A	6.6	20 (Menopausal)	Griseofulvin 20–25 mg/kg/day 6–8 weeks, topical antifungal agent	[4]
Cairo/Egypt	2002–12	58 (4.2)	<i>T. violaceum</i>	42.9 ± 8.54	1:5.4	29.3	17.2	34.4	Griseofulvin 500 mg/day, itraconazole 200 mg/day, terbinafine 250 mg/day	[5]
Malaga/Spain	1995–2012	33 (11.4)	<i>T. violaceum</i> (immunocompetent), <i>T. tonsurans</i> (immunosuppressed)	56.9 ± 18.46 (18–80)	1:10	9	N/A	29	Terbinafine (Trichophyton), Griseofulvine (Microsporum)	[6•]
Thessaloniki/Greece (Northern)	1981–95	35 (5.8)	<i>T. violaceum</i>	N/A (19–89)	1:16.5	28.6	N/A	20	N/A	[7]
Turin/Italy	1997–2012	13 (2.6)	<i>M. canis</i>	56.5 (29–80)	1:2.2	38.5	46.2	23.1	Griseofulvin 500 mg/day, terbinafine 250 mg/day	[8]
Lisboa/Portugal (Southern Europe)	2008–18	15 (1.5)	<i>M. audouinii</i>	43.3 (18–86)	1:1.5	40	N/A	46	Terbinafine 250 mg/day	[9]
Dakar/Senegal	2015–6	121 (N/A)	<i>T. soudanense</i>	36.1 (19–70)	1:11.1	N/A	0	9	Griseofulvine, terbinafine 4–8 weeks	[10]
Tehran/Iran	2010–5	25 (20.6)	<i>T. violaceum</i>	45.28 (25–62)	1:1.5	60	24	80	Terbinafine 250 mg/day 2–6 weeks (Trichophyton), itraconazole 250 mg/day 3 weeks (Microsporum)	[11]

**Fig. 1** **a** A 78 year-old female with an 8-month history of scaling, patchy hair loss, and mild perifollicular erythema. **b** Dermoscopy of the scalp showing comma hairs (white arrow) and corkscrew hairs (black arrow). **c** Spore ectothrix fungus typical of *M. canis* surrounds and fills the hair shafts (Grocott, 60×)



dermatoscopy, the pseudo-alopecia patch of the microsporic TC, short hair, “coma” hairs, corkscrew, and peripilar chainstays is found (Fig. 1b). The hair can present five types of parasitism, two endothrix, and three ecto-endothrix. The trichophytic parasitism (*T. tonsurans*) with many spores clustered densely inside the hair, and a phallic form with few spores and hyphae as well as with air vesicles inside the hair (*T. schoenleinii*) is evident in the endothrix type [13•].

The parasitoid ectoendothrix (classic ectothrix) has a microscopic modality (*M. canis*) with small spores that form a cuff around the hair, a microid form with spores with loose disposition around the hair (*T. mentagrophytes*), and a megasporated presentation (*T. verrucosum*) with large spores around the hair (Fig. 1c) [13•].

## Culture

Macroscopic and microscopic characteristics of the colonies are useful for diagnosis; *Trichophyton* colonies have smooth walls, in *Microsporum* and *Nannizzia equinulata* and *Epidermophyton* colonies are also smooth, the latter without microconidia [13•].

## Molecular Studies and Dermatoscopy

Molecular tools with enough specificity using different techniques are available: standard polymerase chain reaction [30, 31], as well as the newest diagnostic methods, such as multiplex-dependent probe amplification and rolling circle amplification [32], allowing faster and more accurate results for identifying dermatophytes. Trichoscopy (dermatoscopy) is a diagnostic technique widely used in dermatology [33, 34]. In infections by *T. tonsurans*, multiple “coma”, as well as corkscrew hairs, can be seen [35]; while in *M. canis* infection, we

can observe dystrophic and elbow-shaped hairs, and different levels of broken hair height [36]. Campos et al. [37], in a prospective study, found that trichoscopy is also useful for recording the response to treatment; they observed that 8 weeks after starting treatment, all the dermatoscopic signs evaluated with TC decreased at the end of follow-up; there was no dystrophic hair. The authors inferred that disappearing dystrophic hair could be a marker of therapeutic success.

In a study by Arrazola-Guerrero et al. [38], which included 37 Latin American patients, mostly children, the following dermatoscopic patterns were confirmed: “coma hairs” (41%), corkscrew hairs (22%), short hairs (49%), and black dots (33%). They also observed the presence of scales (89%), peripilarial cylinders (46%), alopecia (65%), pustules (8%), and meliceric crusts (16%).

## Histopathological Study

A biopsy is not usually necessary in superficial TC since there are no specific images; however, hyperkeratosis with parakeratosis and the presence of hyphae between the corneous cells may be seen. In TC, parasitized hair is detected, with rounded arthrospores into the follicles or the stratum corneum [39].

## Wood’s Light

It is carried out in a dark room, and an ultraviolet light lamp of 320 to 400 nm is used and gives a green fluorescence in microscopic hairs, white-bluish in the favus, and does not generate any in trichophytic tinea [40]. Wood’s light shows fluorescence in microsporic and favic hairs; also, Wood’s light may be useful for therapy follow-up and carrier status [41].

## Differential Diagnosis

TC in adults often present an atypical clinical pattern, which can easily lead to confusion with other dermatoses, resembling bacterial folliculitis, folliculitis decalvans, dissecting cellulitis, or the scarring related to lupus erythematosus. Thus, the correct diagnosis and treatment are delayed often, increasing the risk of a new infection. Other diagnoses include seborrhoeic dermatitis, alopecia areata, trichotillomania, psoriasis, discoid lupus erythematosus, impetigo, folliculitis, and erosive pustular dermatosis [1•, 12••, 13•].

## Treatment

Treatment of ATC is similar to that of TC in children; however, it is necessary to take into account the age of the patient by association with different comorbidities and interactions with other treatments. The most suitable treatment for TC is griseofulvin, 10 to 20 mg/kg/day, and in resistant cases, up to 30 mg for 8 to 12 weeks. In older than 12 years old, 500 mg/day is usually prescribed (although some authors recommend higher doses for micronized griseofulvin) [42, 43•]. Fatty meals, milk, or ice cream increase the bioavailability of griseofulvin [13•].

It is possible to add a topical antifungal or 2.5% Selenium disulphur or azoles in shampoo to remove spores from the surface of the scalp; after a week, there is no possible transmission. It is advisable to lightly rub the affected areas during the bath to remove parasitized hairs or scales. The treatment lasts 2 to 3 months (it is advisable to prolong the treatment 1 month after the clinical cure). It is advisable to check for infected animals or relatives and use bleaches to disinfect fomites [12••, 13•].

In a recent meta-analysis about the efficacy of oral antifungals for TC [44], it was stated that terbinafine is “more efficacious than” griseofulvin for TC caused by *Trichophyton* sp., while griseofulvin is “more efficacious than” terbinafine for *Microsporum* sp. In a recent Cochrane summary review, Chen et al. [45••] included 25 randomized controlled trials (4449 study subjects). The authors found that terbinafine and griseofulvin had similar effects in children with both microscopic and trichophytic infections. Terbinafine showed a higher rate of complete cure compared with griseofulvin in *T. tonsurans* infections; however, griseofulvin was better for *Microsporum* sp. Itraconazole and fluconazole show similar efficacy against *Trichophyton* infections but are not optimal choices.

Terbinafine is active at a dose of 250 mg or 10 mg/kg, and a dose of 3 to 6 mg/kg/day, in case of trichophytic tinea for at least 1 week and in microsporic TC for 4 weeks. It is often required to prolong the treatment or double the dose. An additional alternative for microsporic TC is itraconazole 100 mg/day for 4 weeks [44].

Both terbinafine and itraconazole can also be given as intermittent therapy (“pulses”) of 1 week each month, for at least 3 months [46–48].

For patients without a clinical cure, it is advisable to consider lack of adherence; suboptimal drug absorption; fungal resistance; biofilm formation [49]; as well as reinfection. When clinical improvement is observed, although mycological cure has not been achieved, treatment should be continued for an additional month. If there has been no clinical response and signs persist at the end of the treatment period, we can increase the dose or the treatment duration [45••].

The most commonly used oral antifungal is safe for long periods, or even at higher doses. In resistant cases, we recommend changing the treatment for another oral antifungal, for example, in cases of lack of response to griseofulvin, change to terbinafine or itraconazole [50–52].

The choice of the oral antifungal drug depends on the characteristics of the patient; griseofulvin has been widely used and, more recently, terbinafine [47]. Ketoconazole, itraconazole, and fluconazole are used to a lesser extent. In AIDS patients, some authors recommend terbinafine [53, 54]; however, it can be ineffective in cases of TC due to *M. canis*, and thus, griseofulvin is preferred [47]. Frequent and significant adverse reactions have been reported with griseofulvin and terbinafine, mainly in transplant patients due to its interaction with cyclosporine [55, 56]. Also, several studies have shown a faster efficacy of terbinafine when compared with griseofulvin (4 weeks vs. 8 weeks) in TC related to *Trichophyton* [57, 58].

## Prevention

There is no effective preventive measure for TC; a vaccine that prevents infection in animals has been developed; however, it is not suitable for humans [59].

## Prognosis

TC usually presents a benign course; some forms heal without treatment, while others evolve to chronicity; some modalities decrease the patient’s quality of life due to pruritus or esthetic deformation and scarring alopecia [12••, 13•].

## Complications

A secondary bacterial infection is not common. Erythema nodosum has been described in association with inflammatory TC [60]. This reaction can be associated with immune complex deposition and is considered a hypersensitivity response [61].

## Conclusion

TC is a common disease throughout the world, with a wide variety of clinical presentations leading to many differential diagnoses. It is often seen in adults, mostly postmenopausal women, and immunosuppressed patients. The use of oral antifungal medications for clinical and mycological cures is commonly required in order to prevent epidemics and carrier status.

## Compliance with Ethical Standards

**Conflict of Interest** The authors declare that they have no conflicts of interest.

**Human and Animal Rights and Informed Consent** This article does not contain any studies with human or animal subjects performed by any of the authors.

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