Tinea Capitis: Current Concepts in Clinical Practice

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GOAL

To understand tinea capitis to better treat patients with the condition

OBJECTIVES

Upon completion of this activity, dermatologists and general practitioners should be able to:

1. Describe the etiology of tinea capitis.
2. Recognize and diagnose tinea capitis.
3. Effectively treat tinea capitis.

CME Test on page 88.

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Tinea capitis is a common infection, particularly among young children in urban regions. The infection often is seen in a form with mild scaling and little hair loss, a result of the prominence of Trichophyton tonsurans *(the most frequent cause of tinea capitis in the United States).* T tonsurans *does not fluoresce under Wood light, unlike the common tinea capitis–causing fungal organisms seen in Europe and many other countries, which emit a green fluorescence. However,* T tonsurans*, like other fungi, also may less often produce an intense inflammatory reaction, which is suggestive of an acute bacterial infection.*

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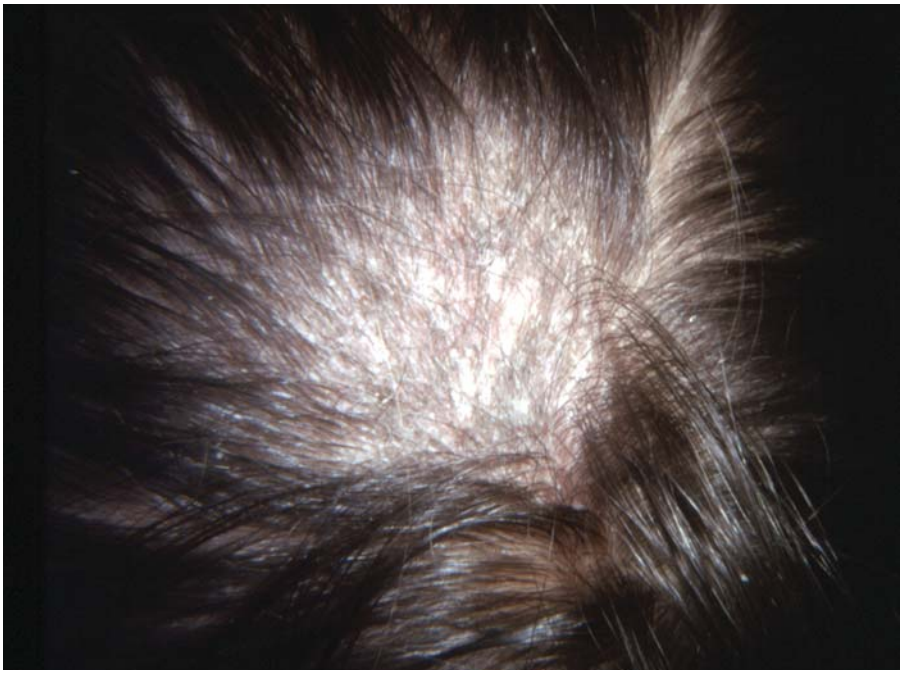
Tinea capitis, or ringworm of the scalp, is most common in preschool and school-age children and often is associated with crowded living conditions.[1-5] In the United States, *Trichophyton tonsurans* causes most cases of tinea capitis and at times produces widespread scaling with minimal hair loss, prompting suspicion of seborrheic dermatitis. *T tonsurans* also may induce violent tissue

reactions, including inflammation and pustulation, which is suggestive of an acute bacterial infection.[2] Alternatively, childhood infections with this fungus may be asymptomatic. A central European study showed a peak incidence of scalp fungal infections in children aged 4 to 6 years.[5] Another survey conducted in an urban pediatric clinic in the United States reported an overall incidence of scalp fungal infections of 4%, with the highest incidence, 12.7%, occurring among black girls.[1] This higher incidence was thought to be related to the use of occlusive pomades and tight braiding of the girls' hair. However, a race-matched case-controlled study showed that use of oils or grease, hairstyling, frequency of washing, and other hair care practices were not associated with the presence of tinea capitis.[6] The presence of an adult carrier state in juvenile tinea capitis warrants a thorough evaluation of the patient's immediate and extended families.[2]

Etiology

Tinea capitis is caused by dermatophytic infections that belong to 3 genera of fungi: *Trichophyton*, *Microsporum*, and *Epidermophyton*.[5-16] Ecologically, these fungi can be classified by host preference as either anthropophilic (humans), geophilic (soil), or zoophilic (animals). Clinically, the patterns of infection of these fungi are classified by anatomic preference, such as tinea capitis (scalp), tinea pedis (foot), or tinea corporis (body). These fungi characteristically produce infections with an active circinate margin, hence the term *ringworm*. Although most species of dermatophyte are capable of producing tinea capitis, some species have a greater tendency than others, and a few (namely *Epidermophyton floccosum*) are noted for no involvement of the scalp hair. When molds other than the 3 dermatophytic fungi described above attack the hair, nails, or skin, infection by these nondermatophytic fungi is called *dermatomycosis* rather than *dermatophytosis*.

Tinea capitis is characterized by broken hair and often produces alopecia (Figure). Previously, *Microsporum audouinii* and *Microsporum canis* were the most common causes of tinea capitis in prepubescent children because of the children's contact with cats and dogs.[17] *M audouinii* and *M canis* emit a green fluorescence under a Wood light; however, nonfluorescent *T tonsurans* has replaced these 2 organisms as the most common cause of tinea capitis in North America.[8-13] This development may be due to shifting immigration patterns from Mexico, Central America, and South America.[18] In Africa, Pakistan/India, South America, and eastern Europe, the most common cause of tinea capitis is *Trichophyton violaceum*, whereas in western Europe, it is *M canis*,[5,8] with cats serving as a common vector.[13] *Trichophyton soudanense* is a significant cause of tinea capitis in Africa but is a rare cause elsewhere in the world. *M audouinii* often produces a noninflammatory infection that is almost asymptomatic. At the other end of the spectrum, a severe, inflammatory kerion may be produced that is most often because of either *T tonsurans* or *M canis*. Another severe, inflammatory type of tinea capitis,



Tinea capitis displaying a patchy alopecia with some evidence of matting, which is suggestive of kerion development.

called *favus*, is due to *Trichophyton schoenleinii*.[15] Granulomatous perifolliculitis also may be seen, usually due to *Trichophyton rubrum*, manifesting on shaved legs rather than on the scalp.

When hair is involved, fungal infections are divided into 2 types: ectothrix (*M audouinii*, *M canis*), whereby spores are present on the hair surface only, and endothrix (*T tonsurans*, *T violaceum*, *T soudanense*), whereby spores are present within the hair follicle; endothrix infection spores also are present on the skin surface, hence the ability to perform a fungal culture of this infection. Fungi enter the proximal cortex where the cuticle is immature; the fungi then colonize the proximal keratinized cortex and generate septate hyphae that become arthrospores and replace the cortex; this may cause the weakened hair to coil up inside the infundibulum and form the black dots of black dot ringworm. *T rubrum* rarely affects the hair shaft; nevertheless, when it does, the infection may be both nonfluorescent ectothrix and endothrix.[11,16]

Historical Considerations

In Rome around 30 AD, an acute inflammatory scalp condition with purulent drainage was first described by Celsus; thus, the kerion is some times referred to as *kerion celsi*.[11,14] During the turn of the 20th century, tinea capitis was a plague.[14] Accordingly, Europeans attempting to immigrate to America who had tinea capitis, especially the favus type, were barred at Ellis Island. The history of this infection has had a predictable effect on its epidemiology in the United States, which differs from that in Europe. *M audouinii* was responsible for causing most tinea capitis infections in the United States in 1954, with *T tonsurans* playing a small role. In the 1960s, the incidence of infection caused by both organisms became about equal; however, *T tonsurans* now causes the majority of tinea capitis infections.[11]

Clinical Characteristics

The most common manifestation of tinea capitis is an incomplete alopecia of the scalp that often is prominent on the crown, occipital and parietal regions, and easily accessible areas, with a tendency for contralateral patches corresponding with the dominant hand.[3] The eyelashes, eyebrows, pubic regions, and bearded areas of the face also may be affected. The alopecia may appear either in solitary or multiple patches that are well defined and irregularly shaped, and the patches may be linear in configuration in a few patients. The patches often contain both short broken hairs and long, apparently normal hairs. Careful scrutiny may reveal black dots at the site of the broken hairs. The alopecia characteristically is asymptomatic without evidence of scarring, atrophy, or erythema. *T tonsurans* often produces dry, seborrheic dermatitis–like scaling without inflammation and with only slight loss of scalp hair.[13] This virtually asymptomatic infection may persist throughout childhood. However, a more serious reaction may occur, with the formation of black-dot stubs in areas of marked alopecia. Pruritus with lichenification and secondary excoriation may occur in some patients. The pruritus is thought to be psychogenic in nature.[19] Other patients experience a severe inflammatory reaction.

The 2 severe types of tinea capitis are kerion and favus. A kerion is a painfully inflamed, crusty, matted mass that often is associated with purulent drainage from the sinuses. Regional lymphadenopathy is characteristic.[20,21] Kerions tend to be small and solitary, though multiple plaques or one giant mass involving most of the scalp may be seen. Favus produces inflammation and scarring, and is characterized by yellow cup-shaped crusts called *scutula* that are found around a hair.[15] These crusts contain infectious hyphal masses that coalesce into a yellow hyperkeratotic mass.

Laboratory Characteristics

A 10% potassium hydroxide preparation of skin scrapings provides an adequate cytologic specimen for examination and diagnosis. Smearing the specimen on a glass slide using a scalpel blade or the edge of the slide, and then applying the 10% potassium hydroxide, yields consistent diagnostic results.[22] Results of microscopic examinations reveal hyphal forms of fungi that generally are considered pathogenic. When examining a child, 2 effective initial approaches are to rub off several scalp hairs using a moist piece of gauze[23] or to use the toothbrush culture technique, which can collect scales and debris without upsetting the child.[18] Running a sterile cotton swab over the scalp surface of a child rather than scraping the scalp with a blade is a simpler technique to inoculate the specimen onto the fungal media; additionally, the results of this technique have been validated against the results of the toothbrush technique.[13] Material that is inoculated onto the fungal media requires approximately 6 weeks for colony growth on Mycosel or Sabouraud agar plates. To determine which actual fungus is the cause of infection, various characteristics must be considered.

Histopathologic Features

Occasionally, confirmation of a diagnosis of tinea capitis may require examination of a skin biopsy

specimen. Specimens often show collections of polymorphonuclear leukocytes within the upper portion of the stratum corneum.[21,22] A periodic acid–Schiff stain or methenamine silver stain should be performed to elucidate the fungal elements. Traumatic avulsion of hair by itching results in characteristic histologic changes. As heavily pigmented, soft, keratinous material is deposited by the matrix cells into the vacant follicular infundibula, follicular plugging becomes evident. Varying degrees of atrophy characterize these empty follicles. No inflammatory response to the insult is seen. In the perivascular regions of the superficial dermis, a mild lymphocytic infiltration may occur. Follicular hemorrhage often is detected microscopically as the follicular epithelium separates from the surrounding connective tissue.[21] The remaining hairs transition to the catagen phase of the hair cycle, with a greatly thickened and convoluted basement membrane.

Diagnosis

Key clues in the diagnosis of tinea capitis include irregular or linear configuration of the patches of alopecia and the incomplete nature of the hair loss. Histologic confirmation should follow, usually by cytologic examination and fungal culture. A false-negative reading of the potassium hydroxide preparation results may occur in early or inflammatory tinea capitis.[12] Therefore, a routine fungal culture is warranted for suspected scalp patches. As mentioned previously, running a sterile cotton swab over the scalp surface to inoculate the specimen onto the fungal media is a technique that is simpler than scraping the scalp with a blade, particularly in children.[13] Also noted previously, a skin biopsy specimen may be necessary to make or confirm the diagnosis of tinea capitis.

Differential Diagnosis

In addition to tinea capitis, other common entities that lead to alopecia in children are trichotillomania, alopecia areata, and traction alopecia.[24-29] A complete patient history should precede any physical examination for alopecia. Duration of alopecia, medications, overall health over the past year, hair care and cosmetics products, diet, and family history are all key issues in the taking of a focused history for alopecia.[26] Family members also may be a valuable resource because they may witness any acts of hair pulling by the patient. However, if the trichotillomania takes place when the child is going to sleep, and the patient's behavior is normal in other respects, it may be difficult to convince relatives that the disorder is self-induced.[3] Occasional formation of black dots in trichotillomania can suggest black dot ringworm.[25] Alopecia areata, on the other hand, produces the well-defined, smooth, oval patches seen in total alopecia. The margins display short "exclamation mark" hairs that easily are pulled out. As in trichotillomania, alopecia areata usually appears on the scalp; however, it also may be seen on the eyebrows and chin. The patches often regrow spontaneously, only to appear later in other areas.[26] Traction alopecia, which often has a higher prevalence rate in young girls of sub-Saharan African lineage, produces noninflammatory linear areas of hair loss in regions where tight ponytails, braids, or cornrows are found; the hair loss often is reversible after the traction is relaxed.[25] Tinea capitis due to *T tonsurans* may manifest similar to seborrheic dermatitis, recurrent folliculitis of the scalp, psoriasis, and lupus erythematosus.[13]

Androgenic alopecia, scarring alopecia, defects in the hair shaft, Pohl-Pinkus mark, monilethrix, pili torti, trichorrhexis nodosa, trichorrhexis invaginata, pili annulati, pili multigemini, "spun-glass" hair, trichothiodystrophy, and syphilis are other congenital anomalies that occasionally may display the same clinical appearance as tinea capitis.[7,25] "Moth-eaten" alopecia of secondary syphilis is of particular concern.[29]

Therapy

Although several therapeutic options are available for treating tinea capitis, griseofulvin has been the mainstay of the infection's management for the past 40 years.[30-51] Griseofulvin is a fungistatic metabolic product of *Penicillium griseofulvin* that disrupts the microtubule-associated proteins necessary for cell division. Both the micronized (15–25 mg/kg daily) and ultramicronized (10 mg/kg daily) forms of griseofulvin are given as a single dose with a fat-containing meal for 6 to 8 weeks. If the child is healthy and the therapy lasts for less than 3 months, blood tests (eg, complete blood count, liver function test) may not be necessary. Although follow-up cultures sometimes may be helpful, it should be noted that culture results may suggest continued spore shedding, even after the infection has been treated adequately. Griseofulvin does not affect *Candida* species or saprophytic fungi. *M canis* and *T rubrum* may have decreased in vitro sensitivity.

Clinical studies over the past decade have suggested a decrease in sensitivity to griseofulvin and have prompted the investigation of newer oral antifungal agents.[43,47] Because there often is resistance to griseofulvin in *M canis*, for example, ketoconazole may be used at 5 mg/kg daily. Ketoconazole is a broad-spectrum antifungal agent because it impairs ergosterol synthesis. However, due to the resistance

of *M canis* to ketoconazole and the concerns about its long-term safety, a 4- to 6-week therapeutic regimen of the broad-spectrum azole antifungal agent itraconazole (5 mg/kg daily) appears to be a better choice and to match griseofulvin for the treatment of tinea capitis in children. Itraconazole has been shown to have a more favorable side effects profile, as well.[41,42,44] A 4-week therapeutic regimen of terbinafine (10 mg/kg daily) also has been used with success. Terbinafine is a nafitine analogue that accumulates in the skin and hair because it is highly lipophilic and keratinophilic. Terbinafine also is fungicidal against dermatophytes and fungistatic against yeasts, and has been used successfully against *T tonsurans*.[45,50] Fluconazole, a synthetic triazole that acts like other azoles, inhibits fungal cytochrome P450–dependent enzymes, thereby blocking ergosterol synthesis. Fluconazole has been suggested as a treatment of *T tonsurans*, *M gypsum*, and *M canis* in children.[46] Terbinafine and the azoles have yet to receive approval by the US Food and Drug Administration for use in childhood tinea capitis, though controlled evaluations are underway. Other antifungal options are on the horizon.[36-50] Regardless of the chosen therapy, clinicians may want to perform follow-up cultures until a negative result is obtained; this is especially valid for infections caused by *T tonsurans* to prevent a chronic carrier state.[13]

The question whether to shave the hair on a patient's scalp remains open to debate in central European countries such as Lithuania, where shaving commonly is performed at 3 weekly intervals during the course of treatment.[17] The cure rate experienced with this technique reportedly reaches 95% to 98%. In the United States, shaving the scalp hair is unpopular because of its stigmatizing effect; however, with dry, noninflammatory tinea capitis, small lesions on the scalp easily can be overlooked, which are known to be the principal cause of relapse.[17]

When a child has tinea capitis, household contacts should be evaluated. Knowing the fungal etiologic agent is good for planning effective therapy.[1-6,30,31] Asymptomatic cats and dogs often harbor dermatophytes.[19,51,52] Transmission occurs directly from person to person or from animal to person, as well as through combs, clothing, bedding, vinyl chairs, couches, stuffed toys, dolls, and telephone receivers. Occasionally, cases of transmission have been reported to occur at the hairdresser.[53] *M canis* is one example of a zoophilic fungus that commonly gets transmitted through contact with cats.[54] When an infection by *T tonsurans* occurs, all family members may be treated, and the home environment should be evaluated.[13] If necessary, floors should be mopped with a strong disinfectant, all washable objects should be laundered, and all nonwashable objects should be vacuumed. Patients with positive fungal culture results should treat their scalps with selenium sulfide 2.5% or ketoconazole shampoo for 5 minutes prior to rinsing to eliminate superficial infectious spores.[55] As previously mentioned, culture results may suggest continued spore shedding, even after adequate antifungal therapy.

A kerion with regional lymphadenopathy and a culture specimen that reveals pathogenic bacteria warrants antibiotic therapy. In fact, combination therapy with prednisone 1 mg/kg daily and erythromycin may be used in addition to antifungal agents to manage a severe inflammatory kerion.[32,56,57] Although this triple regimen was not shown to accelerate the resolution of kerions compared with griseofulvin alone in one study, it did note that it hastened the reduction of scaling and pruritus.[58] Studies also have shown that both the lymphadenopathy and pus formation seen in kerions are unrelated to the presence or absence of bacterial colonization.[59-61] Because colonization with pathogenic bacteria is common in a kerion, it may in fact play a minor role in its signs and symptoms.

Follow-up

Follow-up visits every 2 to 4 weeks that incorporate Wood lamp testing, microscopic examination, and culture tests to monitor the efficacy of tinea capitis treatment are recommended. Individual hairs with persistent fluorescence under Wood light should be removed from patients. Discontinuation of topical treatments 2 days before the follow-up visit is recommended to avoid false-negative results. Continuation of both topical and systemic treatment also is recommended for at least one week after a negative culture result is obtained. Three weeks of treatment usually cures tinea capitis.[17] To facilitate effective management, patient and parental knowledge of cutaneous fungal disease should be emphasized and evaluated.[62]

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