Consultant360 Multidisciplinary Medical Information Network

WHAT'S YOUR DIAGNOSIS?

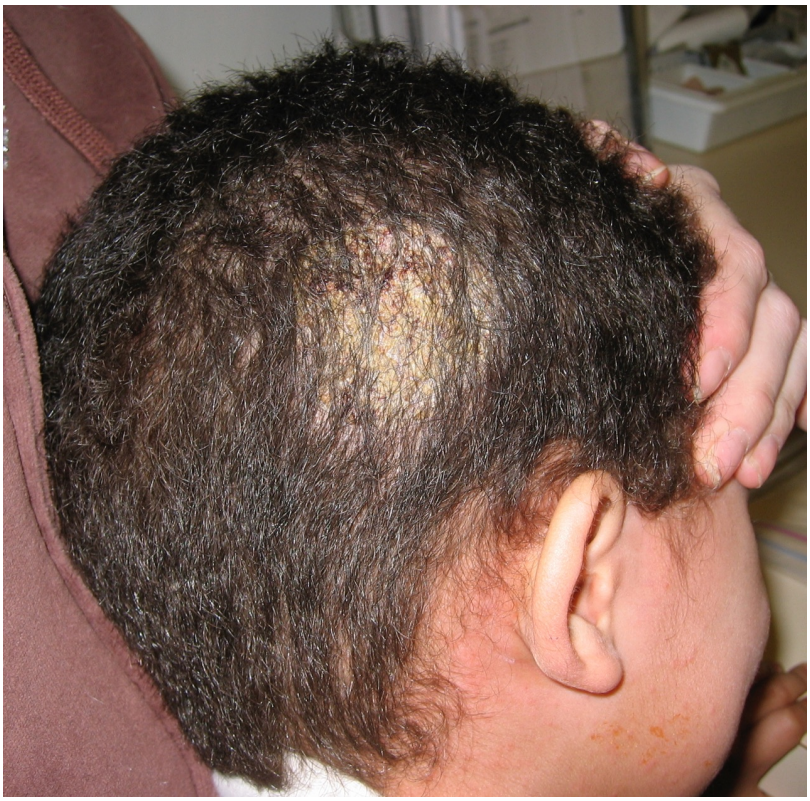
PEER REVIEWED

Why Does this Boy Have an Itchy, Scaly Scalp With Hair Loss?

Alexander K.C. Leung, MD, MBBS, FRCPC, FRCP
Benjamin Barankin, MD, FRCPC

A 5-year-old boy presented with itching, scaling, and an area of hair loss with a duration of 6 months in the right parietal area on the scalp. There was no history of plucking, pulling, or twisting of hair. Review of systems was negative. The family had acquired a dog a year ago, and the dog had appeared healthy and had normal fur. There was no family history of similar hair loss or skin lesions.

Examination of the right parietal scalp revealed fine scaling, patchy hair loss with broken hair shafts, and a black dot appearance. The hair in the affected area was of irregular length. The results of the hair-pull test were negative, and there was no cervical, occipital, or postauricular lymphadenopathy. The rest of the examination results were unremarkable.



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ANSWER: TINEA CAPITIS

Tinea capitis, also known as scalp ringworm, refers to a dermatophyte infection of the scalp and hair shaft, most often caused by any of the dermatophytes belonging to 2 genera: *Trichophyton* and *Microsporum*.

ETIOLOGY

Dermatophytes are grouped as either anthropophilic (human), zoophilic (animal), or geophilic (soil).1 Examples of anthropophilic fungi are *Trichophyton tonsurans*, *T mentagrophytes var interdigitale, T violaceum, T soundanense*, *T schoenleini,* and *Microsporum audouinii*.1 Examples of zoophilic fungi are *M canis, M nanum, M distortum,* and *T verrucosum*.1 *M fulvin* and *M gypseum* are geophilic fungi that are rare causes of tinea capitis.1 The fungal species responsible vary according to the geographical region, and may change over time. Currently, *T tonsurans* is the most common cause of tinea capitis in the United States (US), United Kingdom, and in parts of Western Europe, whereas *M canis* is the most common cause in South America, Southern and Central Europe, Africa, and the Middle East.1-4

EPIDEMIOLOGY

Tinea capitis is usually found in preadolescent children with a peak incidence between 3 and 9 years of age, and it is especially common in black children.5,6 In the US, the prevalence in children ranges from 3% to 8%.4 In the pediatric age group, tinea capitis is more common in males than females.4,6 In adults, the reverse is true. Predisposing factors include poor hygiene; crowded living environment; lower socioeconomic status; concomitant fungal infection; sharing of contaminated fomites (eg, combs, brushes, pillows, or hats); warm, moist environment; and

immunodeficiency.7,8

PATHOGENESIS

Humans may become infected through close contact with infected persons, animals (in particular, house pets), soil, or contaminated fomites. Transmission of fungal spores among family members is the most common route; children often become infected by spores shed by a household contact.9

Infections of the hair can be classified into 3 forms, namely, endothrix infection, ectothrix infection, and favus.1 Endothrix infection is characterized by the development of arthroconidia (fungal spores) within the hair shaft only without destroying the cuticle, often appearing like "a bag of marbles."9 All endothrix infections result from anthropophilic fungi, notably *T tonsurans*, and affected hairs do not fluoresce with a Wood lamp. In ectothrix infections, arthroconidia are found on the surface of the hair shaft. Infected hairs usually fluoresce with a Wood lamp. Causative agents include *M canis*, *T verrucosum*, *M audouinii*, and *M gypseum*. Favus (also known as tinea favosa) is most often caused by *T schoenleinii*. In favus, hyphae are arranged parallel to the hair shaft and arthroconidia within the hair shafts.1,9 Infected hairs become brittle and can be broken off easily. The fungus can invade the scalp because of the enzymes produced, such as proteases that digest keratin, and keratinases that penetrate keratinized tissue. Scaling results from increased epidermal replacement following inflammation.

HISTOPATHOLOGY

Histopathologic features include arthroconidia and/or hyphae within or surrounding hair shafts, fungi sparsely distributed in the stratum corneum, and a perifollicular mononuclear infiltrate in the dermis.1

CLINICAL MANIFESTATIONS

Noninflammatory tinea capitis often presents as fine scaling with single or multiple patches of circular alopecia; diffuse or patchy, fine, white, adherent scaling of the scalp resembling generalized dandruff with subtle hair loss; or single or multiple patches of well-demarcated area(s) of alopecia with fine scale, studded with broken-off hairs at the skin line, resulting in a “black dot” appearance.1 Erythema of the scalp may be present.7,10,11 Itching of the scalp is common in the various presentations of tinea capitis, and early disease can be limited to itching and scaling.5 Patients with tinea capitis may have erythematous papules, scaling, or plaques over the helix, antihelix, and retroauricular region, referred to as the “ear sign.”12

Inflammatory tinea capitis may present as diffuse, patchy alopecia with scattered pustules or low-grade folliculitis; painful and suppurative boggy edematous plaques or nodules often associated with purulent drainage and alopecia (kerion); and yellow or honey-colored, cup-shaped, follicular crusts grouped in patches that resemble a honeycomb, known as scutula

(favus), as well as matted hair on the scalp.1,7 In favus, infected hairs appear yellow. Regional (cervical/suboccipital) lymphadenopathy is often present in many cases of tinea capitis, especially the inflammatory variants.7,9

DIAGNOSIS

The diagnosis of tinea capitis is often clinical. Wood lamp examination is not very useful because hairs infected by *T tonsurans*, the most common cause of tinea capitis, do not fluoresce.5 On the other hand, Wood lamp examination will show bright-green to yellow-green fluorescence of hairs infected by *M canis* and *M audouinii,* and dull or gray-green fluorescence of hairs infected by *T schoenleinii*.5,9 Dermoscopy is more helpful. Typical dermoscopic findings include white perifollicular scales, broken hairs, dystrophic hairs, corkscrew hairs, zigzag hairs, comma hairs, barcode-like hairs, and black dots.10,13-15

If necessary, the diagnosis can be confirmed by potassium hydroxide wet-mount examination of scalp scrapings of the active border of the lesion, or black dots, as well as stubbly, broken hairs to increase the sensitivity of the test.5 A drop of 10% to 20% potassium hydroxide, with or without dimethyl sulfoxide, is added to the specimen. The specimen is then gently heated to accelerate the destruction of the squamous cells if no dimethyl sulfoxide is added. The potassium hydroxide dissolves the epithelial tissue, leaving behind easily visualized septate hyphae and fungal spores. The spores of *M canis* will be seen coating the hair shaft, while the spores of *T tonsurans* will be seen within the hair shaft.5

Although fungal culture is the gold standard to diagnose dermatophytosis, culture is rarely needed unless the diagnosis is in doubt or the infection is severe, widespread, or resistant to treatment. Culture is more expensive and takes 7 to 14 days for results. The most common culture medium is Sabouraud peptone-glucose agar.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes seborrheic dermatitis, alopecia areata, pityriasis amiantacea, trichotillomania, traction alopecia, loose anagen syndrome, plaque psoriasis, pediculosis capitis, discoid lupus erythematosus, lichen simplex chronicus, lichen planopilaris, pyoderma, bacterial folliculitis, folliculitis decalvans, and dissecting cellulitis.1,5,7

COMPLICATIONS

Untreated or inappropriately treated favus and kerion may result in permanent scarring and alopecia, which may have a significant impact on the child's self-esteem and an adverse effect on the quality of life.7,11 Dermatophytid reaction, commonly referred to as id reaction or autoeczematization, may also occur, especially just after starting antifungal treatment.2,16 Rarely, erythema nodosum may occur, especially in association with kerion.1 Very rarely, disseminated systemic disease has been reported in immunocompromised individuals.1

disseminated systemic disease has been reported in immunocompromised individuals.1

TREATMENT

All family members should be examined and treated simultaneously if tinea capitis is found. Parents of classmates and playmates should be informed so that their children may be examined and treated if necessary. Sharing of fomites and participation in contact sports should be discouraged. Cleaning of fomites such as combs, brushes, and hats are warranted.

Oral antifungal agents such as griseofulvin (not available in Canada), terbinafine, itraconazole, and fluconazole, should be used; the duration of treatment should be 4 to 6 weeks.2 Topical antifungal agents alone are not recommended for the treatment of tinea capitis because these agents do not penetrate to the root of the hair follicles deep within the dermis.2,5,16 However, topical antifungal therapy can be used to reduce transmission of spores.

Combined therapy with topical and oral antifungals may increase the cure rate. Using an antifungal shampoo (eg, selenium sulfide, ketoconazole, ciclopirox) twice per week for 1 month for the entire family during the treatment of the patient in question can prevent “ping pong” transmission between family members.5 With appropriate treatment, patients diagnosed as having tinea capitis have a good prognosis.

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