Tinea Capitis Caused by *Trichophyton rubrum* Mimicking Favus

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PRACTICE **POINTS**

- Although favus is uncommonly seen in developed countries, it still exists and can mimick other conditions, notably cutaneous malignancies.
- Favus may affect the skin and nails in addition to the hair.
- The lesions of favus may persist for many years.

Favus is an uncommon form of tinea capitis (TC) currently seen in geographic areas with poor sanitation and limited access to health care such as emerging nations. Several variants of this condition have been described including one exhibiting a plaque composed of parchmentlike material. The makeup of this plaque has not been described. Tinea capitis is rare in adults, particularly when the infectious agent is Trichophyton rubrum*, and affected patients often exhibit comorbidities associated with diminished immune surveillance. This case report describes an elderly woman with TC due to* T rubrum *mimicking a rare form of favus.*

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In 1909, Sabouraud[1] published a report delineating the clinical subsets of a chronic fungal infection of the scalp known as favus. The rarest subset was termed *favus papyroide* and consisted of a thin, dry, gray, parchmentlike crust up to 5 cm in diameter. Hair shafts were described as piercing the crust, with the underlying skin exhibiting erythema, moisture, and erosions. Children were reported to be affected more often than adults.[1] Subsequent descriptions of patients with similar presentations have not appeared in the medical literature. In this case, an elderly woman with tinea capitis (TC) due to *Trichophyton rubrum* exhibited features of favus papyroide.

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Case Report

An 87-year-old woman with a long history of actinic keratoses and nonmelanoma skin cancers presented to our dermatology clinic with numerous growths on the head, neck, and arms. The patient resided in a nursing home and had a history of hypertension, osteoarthritis, and mild to moderate dementia. Physical examination revealed a frail elderly woman in a wheelchair. Numerous actinic keratoses were noted on the arms and face. Examination of the scalp revealed a large, white-gray, palm-sized plaque on the crown (Figure 1) with 2 yellow, quarter-sized, hyperkeratotic nodules on the left temple and left parietal scalp. The differential diagnosis for the nodules on the temple and scalp included squamous cell carcinoma and hyperkeratotic actinic keratosis, and

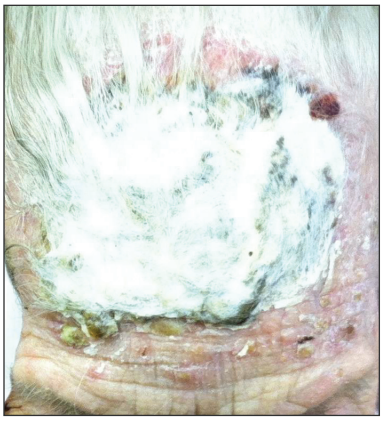

Figure 1. A white-gray plaque of tinea capitis on the crown with erythema and alopecia at the back edge of the plaque.

both lesions were biopsied. Histologically, they demonstrated pronounced hyperkeratosis and parakeratosis with numerous infiltrating neutrophils. The stratum malpighii exhibited focal atypia consistent with an actinic keratosis with areas of spongiosis and pustular folliculitis but no evidence of an invasive cutaneous malignancy. Periodic acid–Schiff stains were performed on both specimens and revealed numerous fungal hyphae within the stratum corneum (Figure 2) as well as evidence of a fungal folliculitis.

At a follow-up visit 2 weeks later, a portion of the hyperkeratotic material on the crown of the scalp was lifted free from the skin surface, removed with scissors, and submitted for histologic analysis and culture. The underlying skin exhibited substantial erythema and diffuse alopecia. The specimen consisted entirely of masses of hyperkeratotic and parakeratotic stratum corneum with numerous infiltrating neutrophils, cellular debris, and focal secondary bacterial colonization (Figure 3). Fungal hyphae and spores were readily demonstrated on Gomori methenamine-silver stain (Figure 4). A fungal culture from this material failed to demonstrate growth at 28 days. The organism was molecularly identified as *T rubrum* using the Sanger sequencing assay. The patient was treated with fluconazole 150 mg once daily for 3 weeks with eventual resolution of the plaque. The patient died approximately 3 months later (unrelated to her scalp infection).

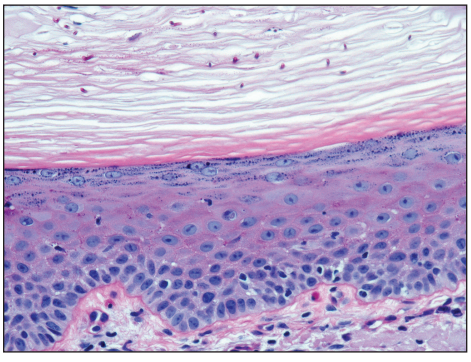

Figure 2. One of the initial biopsies from the left temple demonstrated mild keratinocyte atypia and numerous fungal hyphae within the stratum corneum (periodic acid–Schiff, original magnification ×400).

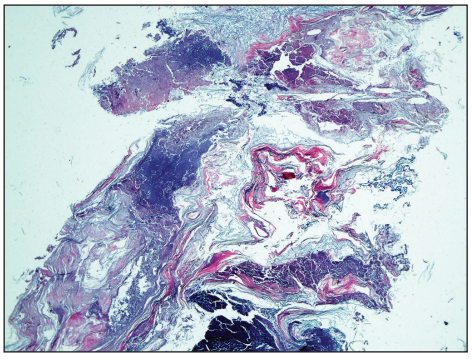

Figure 3. Low-power view of the parchmentlike plaque atop the scalp exhibited occasional hair shaft fragments with massive hyperkeratosis and infiltrating inflammatory cells (H&E, original magnification ×4).

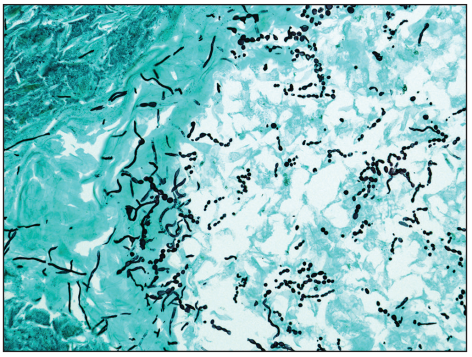

Figure 4. Gomori methenamine-silver stain of the scalp plaque demonstrated numerous fungal hyphae and spores (original magnification ×200).

Comment

Favus, or tinea favosa, is a chronic inflammatory dermatophyte infection of the scalp, less commonly involving the skin and nails.[2] The classic lesion is termed a *scutulum* or *godet* consisting of concave, cup-shaped, yellow crusts typically pierced by a single hair shaft.[1] With an increase in size, the scutula may become confluent. Alopecia commonly results and infected patients may exude a "cheesy" or "mousy" odor from the lesions.[3] Sabouraud[1] delineated 3 clinical presentations of favus: (1) favus pityroide, the most common type consisting of a seborrheic dermatitis–like picture and scutula; (2) favus impetigoide, exhibiting honey-colored crusts reminiscent of impetigo but without appreciable scutula; and (3) favus papyroide, the rarest variant, demonstrating a dry, gray, parchmentlike crust pierced by hair shafts overlying an eroded erythematous scalp.

Favus usually is acquired in childhood or adolescence and often persists into adulthood.[3] It is transmitted directly by hairs, infected keratinocytes, and fomites. Child-to-child transmission is much less common than other forms of TC.[4] The responsible organism is almost always *Trichophyton schoenleinii*, with rare cases of *Trichophyton violaceum*, *Trichophyton verrucosum*, *Trichophyton mentagrophytes* var *quinckeanum*, *Microsporum canis*, and *Microsporum gypseum* having been reported.[2,5,6] This anthropophilic dermatophyte infects only humans, is capable of surviving in the same dwelling space for generations, and is believed to require prolonged exposure for transmission. *Trichophyton schoenleinii* was the predominant infectious cause of TC in eastern Europe in the 19th and early 20th centuries, but its incidence has dramatically declined in the last 50 years.[7] A survey conducted in 1997

and published in 2001 of TC that was culture-positive for *T schoenleinii* in 19 European countries found only 3 cases among 3671 isolates (0.08%).[8] Between 1980 and 2005, no cases were reported in the British Isles.[9] Currently, favus generally is found in impoverished geographic regions with poor hygiene, malnutrition, and limited access to health care; however, endemic foci in Kentucky, Quebec, and Montreal have been reported in North America.[10] Although favus rarely resolves spontaneously, *T schoenleinii* was eradicated in most of the world with the introduction of griseofulvin in 1958.[7] Terbinafine and itraconazole are currently the drugs of choice for therapy.[10]

Tinea capitis is the most common fungal infection in children, with 1 in 20 US children displaying evidence of overt infection.[11] Infection in adults is rare and most affected patients typically display serious illnesses with concomitant immune compromise.[12] Only 3% to 5% of cases arise in patients older than 20 years.[13] Adult hair appears to be relatively resistant to dermatophyte infection, probably from the fungistatic properties of long-chain fatty acids found in sebum.[13] Tinea capitis in adults usually occurs in postmenopausal women, presumably from involution of sebaceous glands associated with declining estrogen levels. Patients typically exhibit erythematous scaly patches with central clearing, alopecia, varying degrees of inflammation, and few pustules, though exudative and heavily inflammatory lesions also have been described.[14]

In the current case, TC was not raised in the differential diagnosis. Regardless, given that scaly red patches and papules of the scalp may represent a dermatophyte infection in this patient population, clinicians are encouraged to consider this possibility. Transmission is by direct human-to-human contact and contact with objects containing fomites including brushes, combs, bedding, clothing, toys, furniture, and telephones.[15] It is frequently spread among family members and classmates.[16]

Prior to World War II, most cases of TC in the United States were due to M *canis*, with *Microsporum audouinii* becoming more prevalent until the 1960s and 1970s when *Trichophyton tonsurans* began surging in incidence.[12,17] Currently, the latter organism is responsible for more than 95% of TC cases in the United States.[18] *Microsporum canis* is the main causative species in Europe but varies widely by country. In the Middle East and Africa, *T violaceum* is responsible for many infections.

Trichophyton rubrum–associated TC appears to be a rare occurrence. A global study in 1995 noted that less than 1% of TC cases were due to *T rubrum* infection, most having been described in emerging nations.[12] A meta-analysis of 9 studies from developed countries found only 9 of 10,145 cases of TC with a culture positive for *T rubrum*.[14] In adults, infected patients typically exhibit either evidence of a concomitant fungal infection of the skin and/or nails or health conditions with impaired immunity, whereas in children, interfamilial spread appears more common.[11]

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