Kerion Kept at Bay with Use of Ketoconazole Shampoo

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Case Presentation

A 10-year-old male patient with a chromosomal translocation presented to the pediatric dermatology clinic with significant hair loss and multiple boggy nodules on his scalp. The mother of the patient notes that the patient had a small round patch of hair loss when he was adopted at age three and was subsequently given 2% ketoconazole shampoo. She notes that the patient's focal patch of hair loss was maintained with 2% ketoconazole shampoo for seven years without progression until two months prior to his presentation to our clinic when he significantly worsened.

On presentation, the patient demonstrated greater than 50% hair loss with a shiny erythematous patch of alopecia affecting his vertex scalp extending to his frontal and parietal scalp. On palpation multiple boggy nodules were present with sinus tracts and drainage of serosanguinous and purulent material. The patient did not exhibit lymphadenopathy but noted scalp tenderness. A 6mm punch biopsy was obtained from the patient's right parietal scalp.

Differential Diagnosis

The differential diagnoses for a boggy, fluctuant mass with significant alopecia includes primary or secondary infection, dissecting cellulitis of the scalp, severe discoid lupus erythematosus (DLE), and a kerion. Dissecting cellulitis of the scalp presents as boggy, fluctuant, suppurative nodules often with purulent drainage and hair loss, however, it occurs predominately in the post-pubertal population making it a much less favored diagnosis for our patient.[1,2] Severe DLE can present in children with a scalp rash and alopecia but is much more common in adults and women and boggy, fluctuace would be atypical.[3] Primary or secondary bacterial infection is considered in the setting of uncertain primary diagnoses of psoriasis and seborrheic dermatitis, as seen in our patient.

Scalp psoriasis occurs commonly in children and presents as a silvery scale and occasionally with alopecia. However, psoriasis is unlikely to be secondarily infected by bacteria due to increased production of anti-microbial peptides.[4] We also had a low index of suspicion for seborrheic dermatitis due to its bimodal distribution, occurring in infants up to 3 months of age and in adults in the fourth to sixth decade of life.[5] Irritant or allergic contact dermatitis were considered but the patient's mother endorsed no correlation of symptoms with use of hair care products or other irritants. Our highest index of suspicion was for kerion.

Histology

A 6mm punch biopsy and culture are obtained from the patient's right parietal scalp which reveal the definitive diagnosis.

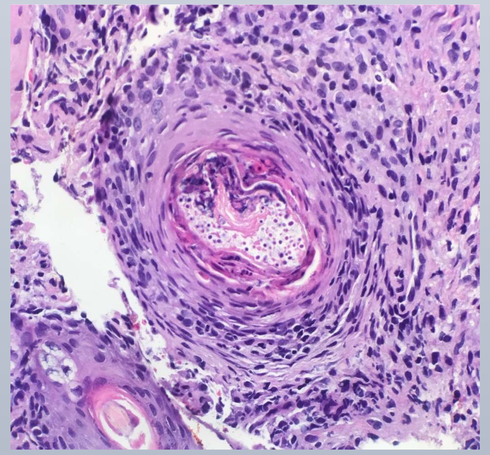

Figure 1: The biopsy revealed many hyphae and spores in the hair shaft bulb with acute, chronic, and focally granulomatous inflammation. Tissue culture identified T. tonsurans..

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Clinical Presentation

Figure 2: Vertex scalp with numerous erythematou s tender boggy nodules and associated alopecia.

Figure 3: Lateral view of alopecia and focal drainage of serosanguino us and purulent material.

Figure 4: Patient 8 weeks of therapy, with resolution of the boggy, erythematous plaques and some terminal hair regrowth.

Discussion

When there is a suspicion for tinea capitis, diagnostic procedures include both direct microscopy and culture. In office potassium hydroxide (KOH) preparations are not always available but are often negative in kerion because it mostly represents the inflammatory response to tinea.[7] Likewise, KOH scrapings from patients treated with topical antifungals such as ketoconazole 2% shampoo may yield a false negative. Cultures can be invaluable in the diagnosis of tinea capitis and are acquired by swabbing the infected area with a cotton-tipped applicator.[13] Wood's lamp can reveal fluorescent bright green to yellow-green for diagnosis of *Microsporum* species but is less helpful in the United States where *Trichophyton* is more commonly isolated.[7]

In any child with alopecia and scaling on the scalp, the diagnosis of tinea capitis should always be excluded.[7] Physical exam and clinical suspicion is helpful in determining the likelihood of the diagnosis. A convenience survey of 100 children with at least one sign or symptom of scalp pruritis, scaling, alopecia, or occipital adenopathy looked at which sign or symptom best predicts diagnosis of tinea capitis. Positive likelihood ratios were calculated to be 7.5, 3.3, 1.4, and 1.1 for the presence of adenopathy, alopecia, pruritus, and scaling, respectively. 55 of 55 children in the study with both adenopathy and alopecia and 60 of the 62 children with both adenopathy and scaling had cultures positive for tinea.[12] Our patient did not display adenopathy on presentation but it should be noted that this is a finding, most notably posterior cervical lymphadenopathy, that is highly suggestive of tinea capitis.

Once the diagnosis of tinea capitis is made, first-line treatment is oral griseofulvin.[14] Systemic treatment of tinea capitis is required as the drug must penetrate the hair follicle.[10] A 2017 systemic review of 25 randomized controlled trials with 4449 participants found griseofulvin to be more effective in *Microsporum* species, an ectothrix organism, whereas terbinafine was found to be slightly more effective in *Trichophyton* species, an endothrix organism. This difference in effectiveness is attributed to fact that terbinafine preferentially accumulates within the hair shaft where endothrix organisms reside. Although some sources recommend griseofulvin dosage of 10-20 mg/kg/day with the liquid microsize solution, many experts recommend 20 to 25 mg/kg/day due to relative resistance with extended treatment duration and possibly lower cure rates. Griseofulvin regimen is recommended for greater than or equal to 6 weeks, until clinically clear. It is FDA approved for children older than 2 years in liquid or tablet form and should be taken with fatty foods. Terbinafine is FDA approved for children older than 4 years with standard regimen consisting of 4 weeks dosed daily by body weight: 10-20 kg, 62.5 mg; 20–40 kg, 125 mg; >40 kg, 250 mg.[13] Antifungal shampoo (selenium sulfide 1 or 2.5%, ciclopirox 1%, or ketoconazole 2%) should be used as adjunctive treatment twice a week to prevent shedding of spores.

In patients with kerion, some have recommended the addition of oral or intravenous corticosteroids but combination with oral antifungals has not been shown to be superior to antifungal alone.[15] All individuals residing with the patient should be examined for signs of tinea and appropriately treated. Carriers may not show signs of disease so the clinician should consider treating suspected asymptomatic carriers with antifungal shampoo in the event of patient recurrence. Combs, brushes, clippers, and headwear should also be disinfected or discarded as dermatophyte organisms remain viable for prolonged periods.[10, 13]

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