CASE REPORT

A Five-Year-Old with a Unique Bald Spot

Lindsay Taylor Ber, MD[1]; Aneeb Mohideen, MD[1]; Zachary M. Gohsman, MD[2]
[1]Pediatric Resident Physician, Department of Pediatrics, University of Florida, Gainesville, FL
[2]Assistant Professor, Department of Pediatrics, University of Florida, Gainesville, FL

ABSTRACT

A kerion is a common but treatable cause of alopecia for children in the United States. With proper clinical diagnosis and follow-up, the lesion can be cleared, and the risk of permanent alopecia can be minimized. This case involves a five-year-old African American male who presented to the clinic with a pruritic bald spot on his scalp for two months. The lesion was determined to be a kerion caused by tinea capitis. The kerion improved after an eight-week course of griseofulvin. However, the patient still had some scaling and crusting of the scalp and no hair regrowth. During his follow-up appointment, the patient endorsed headaches, a known side effect of the medication. His treatment course was extended, and he was placed on four additional weeks of terbinafine with ultimate clearing of the infection. Prompt recognition by the team allowed for preliminary hair regrowth at the completion of the treatment courses. This report discusses the primary pathology, current treatment guidelines, and emerging kerion and tinea capitis trends.

Background

Tinea capitis and kerion are common causes of scalp lesions in pediatric patients; however, they are not commonly recognized by parents. Many parents will try to treat the lesion with topical products, such as shampoo and various creams, before seeking assistance from a physician. Delays in seeking care and in pediatricians recognizing the lesion can increase the risk of permanent alopecia.

Primary objective

Ability to identify a kerion, distinguish it from tinea capitis, and understand current treatment regimens.

PRESENTATION

A five-year-old African American male presented to the clinic with a pruritic bald spot on the scalp for two months. The patient had his first haircut at a barber shop three months prior to the presentation, where his hair was styled into an "afro." After the haircut, the patient began touching the head and scratching at the apex of his scalp. The mother noticed a bald spot forming and began treating the area with tea tree oil without improvement. Approximately one month after the haircut, the patient's mother shaved his head. She noticed the pruritic area had become absent of hair follicles, leaving a cleared area of erythema and yellow pustules. She began

Figure 1: *Photographs of Scalp Lesion on Presentation to Clinic*

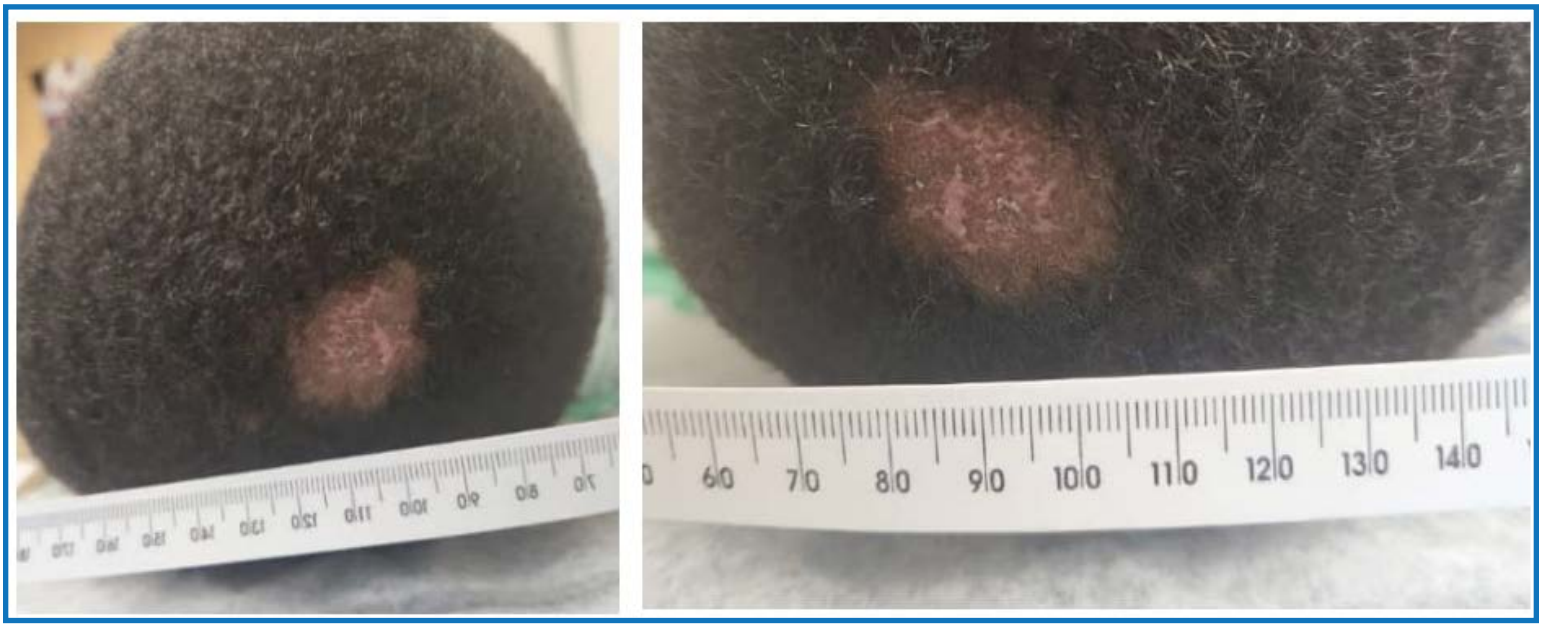

Figure 2: *Photographs of Scalp Lesion After Eight Weeks of Griseofulvin*

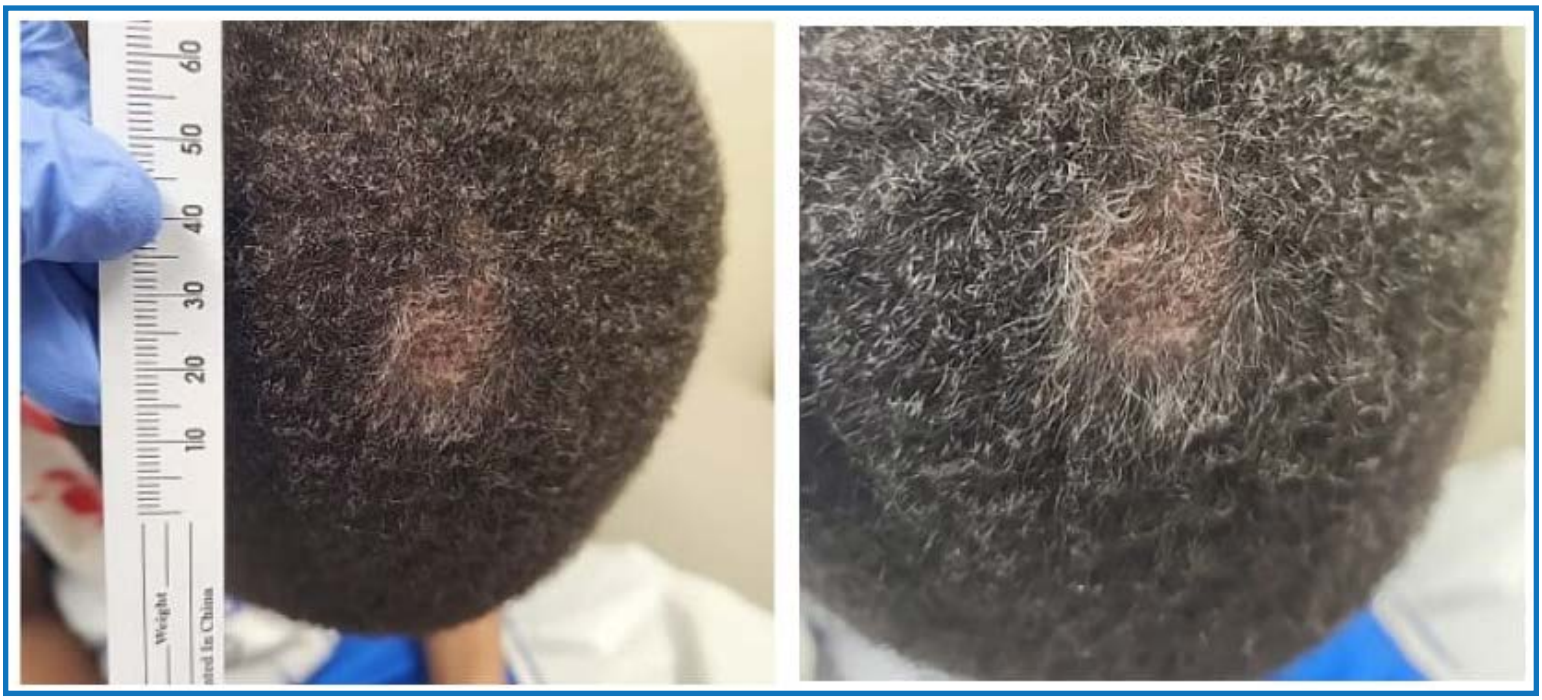

Figure 3: *Photographs of Scalp Lesion After Four Additional Weeks of Terbinafine*

washing his hair with Selsun Blue® shampoo but noticed no improvement. One week before presentation, the scalp pustules burst, leaking pus, and the skin started to weep. After two months of topical over-the-counter treatments, the patient's mother was most concerned about the lack of hair growth.

On presentation, the area was non-erythematous, and the weeping portion of the scalp was very tender to touch. No yellow pustules were present. The plaque was 2 x 2 cm (Figure 1). The area was boggy and had black dots throughout. According to his mom, the lesion was previously "shedding skin" and peeling when the child touched it. The patient had flaking skin surrounding the perimeter of the lesion and slight scarring at the center of the lesion where the pustules were previously located. The patient had no lymphadenopathy of the head or neck, scalp lesions, or rashes on the body.

The patient had no significant past medical history, no past surgical history, or allergies. His mother had a history of seasonal allergies, and his father had eczema. There was no family history of skin disorders, systemic lupus erythematous, or diabetes. No one else at home or school had similar-looking lesions.

The patient was started on an eight-week course of griseofulvin and advised to return to the clinic for follow-up. At the eight-week follow-up, the lesion had decreased in size (Figure 2). The patient was still without erythema or pustules. The patient continued to complain of pruritus in the area, and more black dots appeared in the center of the lesion. Due to incomplete improvement of the scaling, the length of treatment was extended. The patient complained of twice-weekly bilateral frontal headache, a known side effect of griseofulvin, which self-resolved. As a result, the patient was switched to oral terbinafine, which he took for four additional weeks. Liver function tests were ordered, but the family did not follow up with the lab.

The child returned to the clinic four weeks later for follow-up, and the kerion had resolved (Figure 3). Significant hair regrowth was noted on the periphery of the previous lesion, and the beginnings of hair growth were seen in the central area of the lesion. The area was no longer boggy, tender, or pruritic.

FINAL DIAGNOSIS

Kerion

DISCUSSION:

Tinea capitis is a fungal scalp infection that presents with pruritis and scaling areas of hair loss. It is most often caused by *Trichophyton* and *Microsporum* species of dermatophyte fungi.[1] In the United States, peak incidence occurs in African American children between three and seven years of age.[2,3] Children most commonly acquire the infection via contact with a dermatophyte-infected individual or with a contaminated object (comb, hairbrush, hat, or pillows)[4]. In the case discussed above, the patient likely acquired the infection from clippers or a comb that was not cleaned properly between customers at the barbershop.

It is not well understood why children are more commonly infected than adults. The leading theory is that post-pubertal sebum contains short and medium-chain fatty acids with innate fungal-static properties. With lower amounts of fatty acid in the sebum of children, the pH of the scalp increases, leading to a more hospitable environment for dermatophytes.[4,5] Commensal yeast colonizes the adult skin/hair, further inhibiting the infection and overgrowth of dermatophytes.[4]

Tinea capitis typically presents as a flat, scaly, circular patch of scalp with a raised border and central clearing. Each lesion is typically a few centimeters in diameter and enlarges circumferentially over time.[5] Infection can also present as a patch of alopecia with black dots. Patients can also have widespread scaling with surrounding erythema and areas of minimal hair loss, mimicking the appearance of seborrheic dermatitis.[5,6,7]

Dermatophytes use various virulence factors to inhabit the skin and hair. Specific glycoproteins in the cell wall attach to keratin-containing cells in the host tissue. Dermatophytes produce a proteolytic enzyme known as keratinase, allowing it to penetrate the epidermis's stratum corneum, penetrate the skin's deeper layers, and extend downward into the hair follicle.[6]

A kerion is a severe manifestation of tinea capitis that results from an intense immune response to the infection. Histologically, they are characterized by neutrophilic or granulomatous infiltrates, which result in a fibrotic scar.[5,8] As in the case above, they typically present as a solitary lesion in the occipital area of the scalp, although they can present as multiple lesions.[9] Clinically, a kerion can be classified into two categories: inflammatory and non-inflammatory. The non-inflammatory presentation predominantly involves scaling of the skin and hair loss. Lesions are well-circumscribed patches of alopecia caused by destroyed cuticles. Non-inflammatory kerion is characterized as an ectothrix infection pattern as the fungal spores remain outside the hair shaft. In the endothrix infectious pattern, the kerion presents as black dots, which are exposed hair follicles caused by breakage of the hair shaft at the scalp. The diffuse scale presentation is characterized by dandruff-like scaling of the scalp.[5,10] The inflammatory category consists of tender plaques covered with broken hairs and pustules. [5,11] The child above presented with an inflammatory kerion where the erythema, skin weeping, and pustules had resolved, but thick crusting, scaling, and a boggy tender plaque were still present.

There are currently no well-established clinical guidelines for the treatment of a kerion. Dermatologists have recently developed a major and minor criterion and a grading system for kerions; however, the criteria do not change the standard treatment regimen and are more for educational/research purposes.[7]

Treatment should begin once a clinical diagnosis is made. Delaying treatment while awaiting fungal culture can lead to disease progression, disease transmission, and permanent hair loss. Fungal culture is not necessary for diagnosis. Topical anti-fungal treatment is ineffective as it cannot penetrate deep enough into the hair follicle and can lead to subclinical infection. First-line treatment is with a long course of oral anti-fungal agents, which in the United States is typically 6 to 12 weeks of griseofulvin.[7,9,11]

Current treatment guidelines specify first-line treatment with griseofulvin micronized formulation of 20 to 25 mg/kg per day for 6 to 12 weeks or ultra-micronized formulation of 10 to 15 mg/kg per day for 6 to 12 weeks.[12] Treatment failure is usually attributed to poor absorption, so it is recommended that patients take the medication with fatty foods. Initially, the patient in the case above was improving after eight weeks of treatment; however, due to side effects, he was switched to oral terbinafine for four weeks. Liver function tests may be obtained prior to the start of terbinafine therapy, as it can be liver toxic, but this is not a requirement for treatment due to the short course of medication.[7,12]

Despite the difference in appearance, treating a kerion is the same as treating tinea capitis. Kerion requires an early diagnosis to prevent bacterial superinfection, folliculitis, and permanent alopecia. Although the case above was not concerning for bacterial infection, if superinfection is present, an oral antibiotic should be added to the treatment regimen. Antibiotic options include oral cephalexin, dicloxacillin, or clindamycin.[13] Due to its boggy appearance and drainage, kerion can often be misdiagnosed as cellulitis, folliculitis, or an abscess in the scalp; however, none of these would be accompanied by alopecia. It is not well documented in the literature if treating severe infection with systemic glucocorticoids accelerates the improvement of lesions or improves outcomes in terms of alopecia.[1,7,12] Surgical drainage of a kerion is not helpful and should be avoided.

With better recognition and proper treatment of the lesion by both parents and clinicians, the risk of permanent alopecia can be minimized.

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