

Scalp Mass Is Painful and Oozes Pus

Four weeks ago, an 8-year-old boy developed a lesion in his scalp that manifested rather quickly and caused pain. Treatment with both topical medications (triple-antibiotic cream and mupirocin cream) and oral antibiotics (cephalexin and trimethoprim/sulfa) has failed to resolve the problem, so his mother brings him to dermatology for evaluation.

The patient is afebrile but complains of fatigue. His mother denies any other health problems for the child. There is no history of foreign travel, and the patient's brother is healthy.

The boy is in no acute distress but complains of tenderness on palpation of the lesion. The mass in his left nuchal scalp, which measures 4 cm, is impressively swollen, boggy, wet, and inflamed. Numerous red folliculocentric papules—many oozing pus—are seen on the surface. Located inferiorly to the lesion on the neck is a firm, palpable subcutaneous mass. Examination of the rest of the scalp reveals nothing of note.

The clinical presentation and lack of response to oral antibiotics yield a presumptive diagnosis of kerion. A fungal culture is taken,



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with plans to prescribe appropriate medication.

The standard treatment for a fungal kerion includes

- Oral terbinafine
- Oral griseofulvin
- Oral itraconazole
- Oral terbinafine or griseofulvin, plus a two-week taper of prednisone

ANSWER

The correct answer is oral terbinafine or griseofulvin, plus a two-week taper of prednisone (choice “d”); further discussion follows.

Most authorities recommend the use of oral steroids with terbinafine (choice “a”) or griseofulvin (choice “b”), to dampen the acute inflammatory reaction to the fungal antigen. Some sources advise the use of itraconazole (choice “c”) but not as a single agent.

DISCUSSION

This case is a classic representation of kerion, a type of tinea capitis. This distinctive presentation results from not only active localized fungal infection with one of the dermatophytes, but

also an allergic response to the fungal antigen. (This antigen can also trigger a widespread eczematous rash called an *id reaction*.) The resulting boggy, tender mass often oozes pus and usually provokes significant localized adenopathy.

A more common type of tinea capitis is uncomplicated dermatophytic infection of the scalp, presenting as mild localized scaling and modest hair loss, with no edema or redness to speak of. Kerion, by contrast, is far more acute and involves an impressive amount of localized redness and edema, along with modest hair loss, purulence, bloody drainage, and marked tenderness. Untreated, kerion can result in permanent scarring alopecia.

Several different dermatophytes have been isolated from kerions, including *Trichophyton tonsurans*, *T violaceum*, and various members of the *Microsporum* family. These zoophilic or geo-

philic organisms affect children far more than adults. Distinguishing the causative type is significant, because different drugs are required to effectively treat each. This is why a fungal culture is done at the outset.

When the diagnosis is in doubt, a punch biopsy may be necessary, with the sample divided for processing of default H&E stains and for fungal culture. Other information can be obtained by plucking a few hairs from the mass and examining them under 10x power to see if fungal hyphae are confined to the insides of the hair shafts (endothrix) or the outside of the shafts (ectothrix).

This is one situation in which KOH is not helpful for diagnosis: The organisms are too deep to obtain with a superficial scrape.

A number of scalp conditions can mimic a kerion, including lichen planopilaris and folliculitis decalvans. Several years ago, I had a patient who presented with

a similar lesion that turned out to be squamous cell carcinoma—which eventually metastasized and led to his death.

TREATMENT

Treatment of more severe types of tinea capitis can be trying, even when the diagnosis is nailed down. The challenge becomes treating the problem long and strong enough to produce a cure.

In this case, I started the patient (who, at age 8, weighed 110 pounds) on a month-long course of terbinafine (250 mg/d) with a two-week taper of prednisone (40 mg). The expectation was that this would rapidly diminish the edema and pain while we waited for the culture results.

If the results showed the expected *T tonsurans*, treatment would continue as planned. If the cause turned out to be one of the *Microsporum* species, a switch to griseofulvin, at relatively high doses, would be considered. **CR**

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