Granuloma Gluteale Infantum and Kerion

March 01, 2006
By Bskirk Barber, MD, FRCPC [1]

I was asked to see this child by her physician who was concerned that these lesions were a neoplastic event. The physician wanted a dermatologist's opinion and a biopsy to guide treatment decisions. Needless to say, the child's parents were distraught. The child was happy, playful, and not at all disturbed by the rash.

What are your thoughts--and your next step?

Case 1: This is **granuloma gluteale infantum**--a condition seen during the diaper years and considered by most experts to be related to the use of mid-strength topical corticosteroids for diaper dermatitis that may be secondarily infected with *Candida*. Occlusive plastic pants or hours of exposure to urine-soaked diapers intensifies the reaction.

There are cases of granuloma gluteale infantum that occur in the absence of a history of corticosteroid use. In this setting, it is argued that this condition is an abnormal host response to *Candida*. In any event, granuloma gluteale infantum is benign and self-limited once the inciting factors are removed.

The most common clinical presentation is that of reddish brown ovoid papules and nodules that may ulcerate. These lesions occur within the diaper area and, in my experience, are most common in the anogenital area. Although the lesions appear "granulomatous," there are no granulomas on histopathologic examination, and the biopsy shows inflammation.

The diagnosis is most often clinical. Because of the "serious" appearance of the lesions, however, a biopsy is usually considered appropriate rather than waiting the few months that resolution will most likely take. Unfortunately, the lesions may leave scars if the ulcerations are severe enough.
This 14-year-old boy presented with an ulceration that had developed on his scalp over the past 3 weeks. According to his parents, the ulcer began with multiple pustules grouped in the involved area; these then coalesced to form a tender mass, which eventually ulcerated. The boy's physicians in a rural hospital had treated him with intravenous antibiotics, with only marginal improvement. There is one important question to ask the patient--and one simple test that may confirm the diagnosis for you. What are these?

**Case 2:** This teenager, who lives on a farm, has a kerion--a highly inflammatory tinea capitis that is produced by an infection with a zoophilic fungus. (A zoophilic infection spreads from animal to human; an anthropophilic infection spreads from person to person). Zoophilic fungi produce more inflammatory reactions in humans than do anthropophilic fungi; in some circumstances, this inflammatory reaction may resolve spontaneously if left untreated. There is significant variation in species that infect humans from one geographic region to another. Your regional laboratory will be able to provide you with information about the most common fungi in your area.

This young boy's fungal cultures grew *Trichophyton verrucosum*--which is a zoophilic species found in cattle in the region of Alberta, Canada, where this patient lived. Examination showed an ulceration of the scalp that was erythematous and "boggy" at the margins and necrotic centrally. There was an obvious scarring alopecia (hair was destroyed within the ulcer). The hairs at the periphery of the ulcer were loosely attached and falling out spontaneously. The patient had slightly tender occipital lymphadenopathy. He was without fever or pain, however, and looked surprisingly well.

The key historic detail relates to the boy's work with cattle. The simple "bedside" test that needs to be performed is a 10% potassium hydroxide (KOH) preparation on hairs taken from the border of the ulceration.

The KOH preparation in this patient's case showed an ectothrix, in which spores and hyphae are present outside the hair shaft. (In endothrix, spores and hyphae stuff the hair shaft.) Given the patient's exposure to cattle, these findings suggested *T verrucosum* infection even in the absence of culture results.

Unfortunately, in my experience, the more inflammatory the skin reaction, the less likely it is that KOH test results will be positive. Fungal cultures need to be sent to the laboratory. Results, however, will be of little help in directing your therapy, because they may not be available for 3 to 4 weeks. Nevertheless, they may give you some idea of the source of infection once the organism is identified. A bacterial culture should also be obtained, since these lesions are occasionally secondarily infected with staphylococci or streptococci.

The entity that most often produces these clinical findings is a kerion. With a good history of exposure to animals and failure of systemic antibiotic therapy, I advise starting antifungal therapy. I see no point in waiting for culture results, and a positive KOH test result is notoriously difficult to obtain. I resort to a biopsy of these lesions when I do not see a response to therapy or when there is an underlying medical problem that might result in a noninfectious ulceration of the scalp.

In children, the gold standard for the treatment of tinea capitis--including a kerion--is griseofulvin. This therapy has stood the test of time, although resistance appears to be developing. Consequently, newer antifungal agents, such as terbinafine and itraconazole, are being used more commonly; these agents remain off-label in the United States.

This child was treated successfully with terbinafine. It is not within the scope of this short presentation to review the controversy about griseofulvin versus the newer antifungals or their proper dosing. No matter which agent you choose, however, treatment often has to last at least 4 to 8 weeks with adequate doses.

In this patient's case, I also prescribed a short course of systemic corticosteroids with the hope of reducing the inflammation and saving hair. One never knows if this has been useful, but because a kerion causes significant destruction of hair in the involved area, I believe that this intervention may be helpful.