

Kerion Mimicking Erosive Pustular Dermatitis in Elderly Patients

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Erosive pustular dermatosis of the scalp typically occurs in elderly patients; a diagnosis of fungal kerion infection in this patient population may seem unlikely. We present 3 elderly patients who developed pustular eruptions on the scalp that were suggestive of erosive pustular dermatosis. Culture and/or biopsy findings initially excluded kerion fungal infections. Later, cultures isolated Trichophyton species from 1 patient and Microsporum species from 2 patients, and the correct diagnosis of kerion was made. All patients were treated successfully with oral terbinafine hydrochloride. Fungal infection can be suspected in some elderly patients with erosive pustular dermatoses of the scalp. Repeated cultures and biopsies of hair-bearing skin, scale, and cut hair samples may be required to establish the correct diagnosis of kerion.

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Erosive pustular dermatosis can develop on the scalp after trauma or treatment of neoplasms.¹⁻⁶ Pustules may appear within or at the edge of the erosion. Cultures for bacteria usually isolate *Staphylococcus aureus*. Erosive pustular dermatosis typically affects patients older than 65 years.⁷ Sometimes the disorder seems to be an autoaggressive inflammatory process because treatment with calcineurin inhibitors⁸ or topical or systemic glucocorticosteroids often heals the erosions.

Kerion is an infection of the skin caused by dermatophytic fungi. Infection, particularly by zoophilic or geophilic fungi, may lead to an aggressive immune response with excessive tissue destruction and inflammation.^{9,10} Similar to erosive pustular dermatosis,

the infection causes enlarging boggy pyoderma with plaques, erosions, and pustules. Kerions are most common in younger patients.

We present 3 elderly patients who developed boggy erosions that mimicked erosive pustular dermatosis but were actually attributable to fungal infections. The intense inflammatory response apparently suppressed fungal growth, as fungi were not seen in initial biopsies and were not isolated from pus. The correct diagnoses ultimately were established by tissue culture. All 3 patients were successfully treated with oral terbinafine hydrochloride.

Case Reports

Patient 1—An 84-year-old woman presented with an asymptomatic, 3- to 4-cm, red, scaling, atrophic plaque with areas of hemorrhagic crust and scattered 1- to 2-mm perifollicular pustules (Figure 1A). She previously had been treated by her dermatologist for presumed actinic keratoses with a regimen of 5-fluorouracil cream for 2 weeks. Potassium hydroxide (KOH) preparation of the scales and pustule roof showed no branching hyphae, and tissue homogenates failed to isolate fungi or bacteria. A punch biopsy specimen of the affected skin showed epidermal atrophy, dermal fibrosis, and superficial and deep dermal granulomas with a mixed infiltrate. Gomori methenamine-silver and Fite stains were negative for microorganisms. Because an active infectious process could not be identified, a tentative diagnosis of erosive pustular dermatosis of the scalp was made, and the patient was treated with clobetasol propionate ointment 0.05% twice daily.

One month later the plaque and area of hair loss had slightly enlarged; it was associated with shallow ulcerations and persistent 1- to 2-mm pustules. The area remained asymptomatic. A swab sample from the pustules again failed to isolate any fungi. Treatment with clobetasol ointment was continued, and minocycline hydrochloride (100 mg) administered twice daily was added for increased anti-inflammatory and antimicrobial effects.

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Figure 1. Patient 1 before (A) and 8 weeks after treatment with terbinafine hydrochloride and prednisone (B).

The patient returned with symptoms of tenderness and burning 1 month later. The lesion had enlarged and became more inflamed. In the center of the lesion the epidermis was atrophic. A KOH preparation of the scale at the border of the plaque showed branching hyphae. An additional biopsy specimen was submitted for fungal culture and *Trichophyton* species were isolated. Treatment with clobetasol and minocycline was discontinued; instead, the patient was prescribed a regimen of terbinafine hydrochloride (250 mg daily) and prednisone (40 mg daily).

One week later the erythema, burning, and tenderness had markedly improved and the pustules had resolved. Treatment with terbinafine was continued, but the dose of prednisone was tapered and eventually discontinued. At 8 weeks the infection had completely resolved, leaving scarring alopecia with some hair regrowth (Figure 1B).

Patient 2—A 75-year-old woman presented with multiple areas of scaling on her scalp of 2 years'

duration. In the year prior to presentation, these areas had become increasingly inflamed, pustular, painful, and confluent, covering more than 50% of her scalp (Figure 2A). Six months prior to presentation, large, erosive, pustular lakes and scarring alopecia developed. The patient previously had been treated by her dermatologist for actinic keratoses and squamous cell carcinoma in situ using 5-fluorouracil cream and cryotherapy. In the last year, the patient also had applied various topical corticosteroid preparations and tar shampoo for treatment of suspected scalp psoriasis. At presentation she reported daily application of fluocinonide solution 0.05%. The patient reported that numerous cultures performed in the past had yielded no growth of fungi or bacteria. In addition to pustular erosions and alopecia, multiple round, firm, 2- to 3-mm, mobile nodules also were present on the scalp. Because of the patient's history of negative cultures, erosive pustular dermatosis was suspected. Four 4-mm punch biopsy specimens were obtained. Two specimens that were taken from scaling nodules showed numerous fungal hyphae, both superficially and within hair follicles. The other 2 biopsy specimens were taken from pustules and pustular erosions and failed to isolate fungi or yeast. A smear of the pustule contents also did not show fungi, but *Microsporum* species were isolated from tissue homogenate culture. The patient was prescribed terbinafine hydrochloride (250 mg daily), and within 8 weeks the lesions had nearly resolved and left scarring alopecia (Figure 2B).

Patient 3—A 93-year-old woman presented with an inflammatory scalp dermatosis of 3 years' duration that was previously diagnosed as erosive pustular dermatosis. She had been seen by several physicians and was treated with various agents, including intralesional and oral glucocorticosteroids, oral cyclosporine, sulfasalazine, isotretinoin, clobetasol propionate ointment 0.05% under occlusion at bedtime, and tacrolimus ointment 0.1% twice daily, but her condition did not remit. Biopsy specimens of skin showed epidermal acantholysis, abundant plasma cells, scattered neutrophils, and alopecia. Histologic stains for fungi were negative. During physical examination it was noted that tender pustules and pus-filled bullae on red indurated bases covered two-thirds of the scalp (Figure 3A). Punch biopsy specimens of skin from the scalp showed an atrophic epidermis with neutrophilic spongiosis, dermal suppuration, and granulomatous inflammation. Gomori methenamine-silver stain showed numerous fungal hyphae. *Microsporum* species were isolated from tissue homogenate culture. A diagnosis of kerion was made, and the patient was administered terbinafine hydrochloride (250 mg daily), prednisone (20 mg daily), and ketoconazole



Figure 2. Patient 2 before (A) and 8 weeks after treatment with terbinafine hydrochloride (B).

shampoo 2%. Within 3 weeks the boggy purulent plaques had completely resolved, but residual scarring remained (Figure 3B).

Comment

The response to infection with dermatophytic fungi can vary.¹⁰⁻¹² The degree of inflammation is affected



Figure 3. Patient 3 before (A) and 3 weeks after treatment with terbinafine hydrochloride, prednisone, and ketoconazole shampoo 2% (B).

by the nature of the infective species and the intensity of the host immune response. Most dermatophyte infections induce cell-mediated immunity to polysaccharides (mannans) in the fungal cell wall. Development of immunity often is associated with successful ridding of fungus from the skin.¹¹

Some organisms such as *Trichophyton rubrum* tend to elicit minimal or no cell-mediated immunity. Affected individuals may have chronic dermatophytosis with indolent infections over a large portion of the body surface and chronic or recurrent infections of the groin and feet.^{12,13} The infections often elicit little to no itching or pain because inflammation is scanty. The stratum corneum usually is crowded with branching fungal hyphae, which are easily seen in scales cleared with aqueous KOH.

Other organisms elicit aggressive, cell-mediated immunity to infecting fungi, particularly when the

fungal species is uncommon in humans (eg, zoophilic and geophilic fungi).^{14,15} Cytokines released by lymphocytes responding to the infection cause intense inflammation and edema associated with marked pain and itching. Often this host response successfully eliminates fungus from the skin. In other situations, the immune system can keep fungal numbers and invasion in check but cannot kill the fungus, which is especially true when the fungus hides in hair and hair follicles, often on the scalp and beard areas. The inflammation keeps fungal numbers low so that samples of scale and/or tissue may fail to include organisms.^{16,17} Cultures from the purulent exudate often isolate only colonizing bacteria; infecting fungi usually are not detected because neutrophils kill fungi or at least keep their growth in check.¹⁷ In severe unremitting cases, the inflammation becomes chronic and aggressive, producing large erosions, pustules, and alopecia, as evidenced by our 3 cases.

Similar to kerion, erosive pustular dermatosis is a pyoderma; unlike kerion, however, its cause often is unknown. Erosive pustular dermatosis can develop after trauma, including surgery or radiotherapy for treatment of skin cancer, and it can follow nonfungal infections such as herpes zoster. Often, a seemingly ordinary wound can simply fail to heal, remaining as an open and purulent wound that may enlarge despite wound care treatment. Cultures may isolate bacteria, but usually the lesions persist even after administration of antibiotic agents and other antibacterial therapies. Erosive pustular dermatosis, similar to kerion, may result from an overaggressive immune response with autodestructive inflammation.¹⁸ Sometimes treatment with inflammation stimulators such as topical imiquimod can elicit this condition, and anti-inflammatory medications such as topical or systemic corticosteroids allow healing to occur. Isotretinoin also may be effective in some patients.

Our cases show that dermatophytic infections can produce a clinical and histologic picture that resembles erosive pustular dermatosis. When pustular or purulent eruptions occur in an elderly individual who resides in a city, a misdiagnosis of erosive pustular dermatosis is more likely because a kerion is not suspected. Culture from the purulent exudate is unlikely to isolate fungi, though colonizing bacteria may be plentiful. Fungi are more likely to be isolated from hairs within the boggy plaques, from scales at the edges of erosions, or from hair or skin in biopsy samples. Biopsies taken from various sites at various times may be necessary, as demonstrated by our patients. Cultures may be more sensitive than KOH preparations in the detection of infecting fungi.

We suggest several steps for evaluating patients with scalp erosions, pustules, purulent exudate, and hair loss. The major differential diagnoses are squamous cell carcinoma, kerion fungal infection, erosive pustular dermatosis, and pemphigus vulgaris. Biopsy specimens for histologic examination can be obtained from involved skin at the edge of the erosion; they can be stained with Gomori methenamine-silver to detect fungi. The pathologist should specifically look for fungal hyphae in the stratum corneum and within hairs from the affected area. If pemphigus is suspected, a skin biopsy from nearby normal skin can be stained (direct immunofluorescence) for intercellular IgG autoantibodies. If histologic findings are inconclusive, more biopsy specimens from the scaling edge and involved nodular or erosive skin can be cultured for dermatophytic fungi, along with roofs of pustules, scales, and hair in involved areas, if present. Cultures for fungi from purulent surface exudates and from pustule contents rarely are positive and therefore often are misleading. Treatment with potent topical corticosteroids can reduce inflammation and exudate, which may cure erosive pustular dermatosis. It also may increase the number of fungal hyphae in kerion, subsequently making the fungi easier to isolate or observe. Nonresponse or recurrence of the erosive pustular dermatosis suggests the need for additional cultures of hair and skin, and perhaps even empiric treatment with terbinafine hydrochloride.

For our patients, the differential diagnosis of kerion was initially discarded when initial biopsies and cultures failed to show fungi. Nonresponse to treatment with systemic antibiotics and topical glucocorticosteroids prompted a second, more thorough search. Fungi were then detected. Patients were treated with oral terbinafine hydrochloride, resulting in cure.

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