

Folliculitis Decalvans Treated With Radiation Therapy

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A 33-year-old black man presented with folliculitis decalvans resistant to multiple oral and topical therapies. The patient ultimately responded to radiation therapy.

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Folliculitis decalvans is a rare, chronically progressive, purulent folliculitis that causes follicular atrophy and scarring alopecia. Clinically, numerous rounded or irregular atrophic areas of scarring alopecia are surrounded by inflammatory folliculitis or purulent miliary abscesses. We report a case of folliculitis decalvans refractory to oral and topical therapies but responsive to radiation therapy.

Case Report

A 33-year-old black man presented to a branch dermatology clinic in December 1998 with a 12-year history of acne keloidalis nuchae. Results of a physical examination revealed extensive occipital scarring alopecia with deep and inflammatory papulopustules noted to be distributed frontally to occipitally. Results of a scalp biopsy revealed changes consistent with a chronic scarring folliculitis. The epidermis demonstrated hyperkeratosis and acanthosis. Extensive dermal fibrosis and foreign body granulomas were intermixed with neutrophilic abscesses and the presence of naked hair shafts in the deep dermis. Results of special stains for bacteria and fungi were negative. Repeat cultures of the involved scalp frequently grew out

methicillin-sensitive *Staphylococcus aureus*. Based on the clinicopathologic findings, a diagnosis of folliculitis decalvans was made.

The patient was treated with a variety of medications prior to being evaluated in our clinic, with progressive worsening of his condition. He first presented to our clinic in April 1999 for further evaluation. During the subsequent 15 months, the patient underwent multiple therapeutic interventions that included antistaphylococcal antibiotics (oral: cephadrine, dicloxacillin, levofloxacin, minocycline, rifampin, trimethoprim-sulfamethoxazole; topical: clindamycin, mupirocin) and steroid therapy, such as multiple prednisone tapers, intralesional triamcinolone injections, topical clobetasol, and fluocinolone. Other trials included tazarotene cream, oral isotretinoin, and dapsone. Shampoos included chloroxine and nizoral. Oral zinc sulfate was used. Laser hair reduction with a long-pulsed 1064-nm Nd:YAG laser was attempted, but the therapy was not tolerated by the patient. None of these interventions yielded persistent beneficial results.

Despite close supervision and extensive therapeutic trials, the inflammation continued to advance in groups of pustules with subsequent scarring of the occipital and parietal scalp (Figure 1). In July 2000, we initiated radiation therapy using 50 kilovolt (peak) x-rays in 5 defined fields covering the occipital and posterior parietal scalp. Each field was treated once in a single setting with a dose of 440 cGy. The patient did not receive therapy to the uninvolved frontal scalp. The goal of the therapy was to give a dose that would cause temporary epilation and thus reduce the presumed inflammatory response to the hair follicles. The dose needed for permanent epilation varies with fractionation; a single dose of 700 cGy may cause permanent epilation.¹ Alopecia began 3 weeks postradiation and was associated with an inflammatory flare that responded to oral levofloxacin and topical clindamycin gel. At 12 weeks postradiation, there was total alopecia of the irradiated areas of the scalp, with no increased scarring in the radiated area, a few scattered

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Figure 1. Extensive areas of scarring alopecia with deep inflammatory papulopustules.



Figure 2. Smooth scar with cigarette paper texture 6 months postradiation therapy.

inflammatory papules, and substantial pruritus. The patient was in full remission 6 months postradiation, with a smooth scar with cigarette paper texture (Figure 2). Rare tufts of terminal hairs were present. Sixteen months posttreatment, a therapeutic regimen of monthly intralesional injections of triamcinolone acetonide 40 mg/mL was started along the hypertrophic borders of the tufted hair regrowth areas. At 23 months posttreatment, the patient still noted scalp pruritus and sensitivity to extreme temperatures, especially cold.

The patient had no recurrence of inflammatory folliculitis, though he self-treated with short courses of dicloxacillin when he sensed a flare. The patient was highly satisfied with the results of his treatment. Our next goal was to reduce hair density using a 1064-nm laser after the thickness of the scar was reduced.

Comment

Folliculitis decalvans has been described in men and women aged 18 to 70 years² and is more common in black individuals. *S aureus* may act as a cofactor and is present in most cases, possibly as

a superinfection. The host may have an abnormal response to superantigens released by the bacteria or to breakdown products of the degenerating follicle. The true etiology is unknown. Multiple treatment modalities have been advocated. Treatment generally is only suppressive and rarely is curative.

Folliculitis decalvans is resistant to treatment. The literature is replete with anecdotal case reports. Bogg³ described success with fusidic acid. Suter⁴ also reported success with oral and topical fusidic acid. Brozena et al⁵ had success using oral rifampin for 10 weeks in a patient. Abeck et al² demonstrated improvement in 3 patients using oral and topical fusidic acid and zinc sulfate for more than one year. Powell et al⁶ had success using a combination of rifampin and clindamycin for 10 weeks.

Radiation therapy is an option that has been used in the past in selected patients with dissecting cellulitis of the scalp, a related form of scarring alopecia.^{7,8} Treatments were given in doses adequate to cause either temporary epilation or permanent alopecia in the treated areas. The patients treated with lower doses (300 cGy) experienced delayed hair regrowth and substantial reduction in disease

activity. The dose needed for permanent epilation usually is approximately 700 cGy.¹ X-ray therapy was once widely used in dermatology for many conditions, including acne, folliculitis, and tinea infections. Because of the risk of subsequent local neoplastic transformation,⁷ the development of more effective systemic and topical medications replaced x-ray therapy for treating acne, folliculitis, and tinea infections. Accepted therapy today remains limited to oral and topical antibiotics.

We present a case of severe, debilitating, progressive folliculitis decalvans unresponsive to numerous conventional therapies. We treated it in an unconventional manner. Our patient was in constant pain. He had profuse purulent discharge on his bedding and clothing, was unable to wear prescribed military head cover, and was socially stigmatized. The application of radiation therapy was a last resort in an attempt to halt the progression of the patient's disorder. Although not widely used today, x-ray therapy has been proven to be effective in turning off the inflammatory and subsequently scarring course of skin diseases. Although we do not recommend x-ray therapy as a primary means of managing folliculitis decalvans, it may be considered in severe cases unresponsive to other systemic and topical immunomodulatory agents.

Our patient's prognosis was guarded. We recommended close follow-up of symptoms, with local laser hair reduction, if needed, and symptomatic

treatment of his pruritic scalp, which could herald a recurrence of his folliculitis.

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