BRIEF ARTICLES

Extensive Primary Anetoderma Refractory to Erbium YAG Fractionally Ablative Laser

James C. Prezzano, MD[1], Christopher T. Richardson, MD, PhD[1], Glynis A. Scott, MD[1], Sherrif F. Ibrahim, MD, PhD[1]

[1]Dermatology Department at the University of Rochester Medical Center, Rochester, NY

ABSTRACT

Primary anetoderma is a rare elastolytic disorder characterized by well-circumscribed flaccid, atrophic macules and patches caused by focal loss of elastic fibers. Anetoderma is divided into two forms: primary, which is idiopathic and occurs on clinically normal skin, and secondary, which follows a prior dermatosis. Although it is indolent, the lesions of anetoderma persist and may be associated with significant aesthetic changes causing potential psychosocial difficulties. Anetoderma has been successfully treated with ablative, pulsed dye and non-ablative fractionated lasers. Patients with secondary anetoderma and anetoderma limited to a relatively small body surface area may be more amenable to laser treatment than patients with extensive involvement.

INTRODUCTION

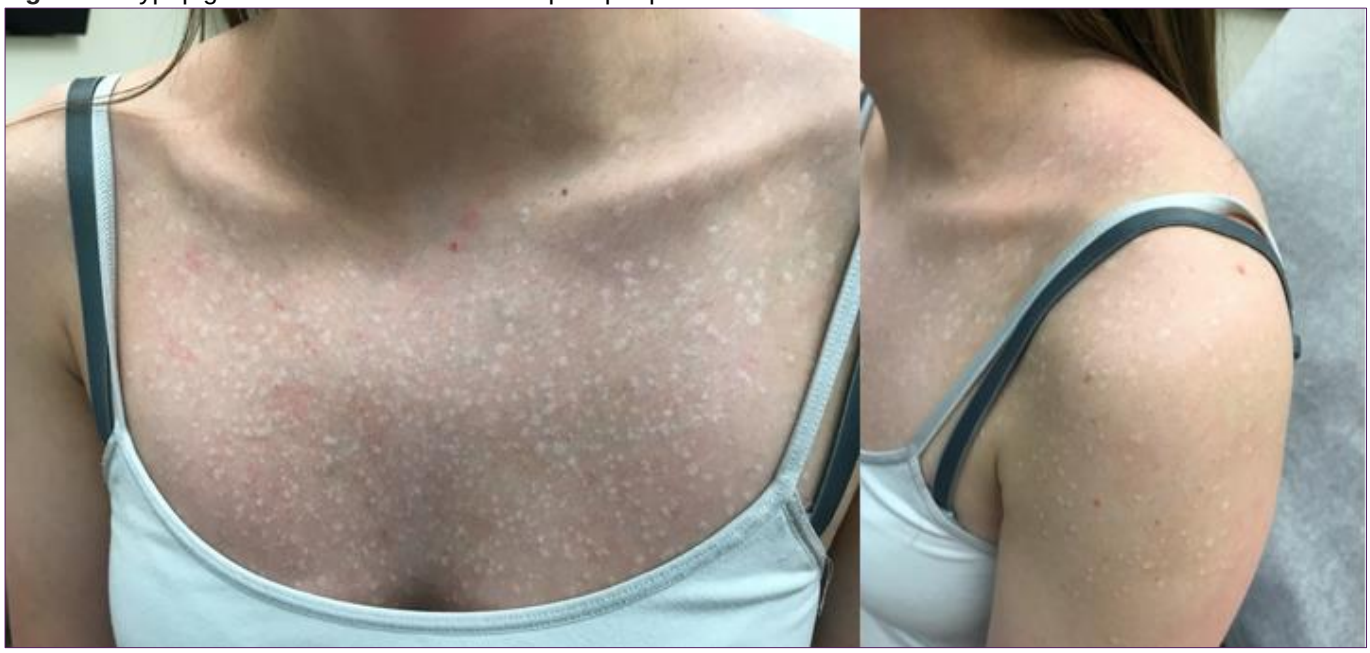
Primary anetoderma is a rare elastolytic disorder characterized by well-circumscribed flaccid, atrophic macules and patches caused by focal loss of elastic fibers.[1] Lesions herniate or bulge with palpation or pressure and are described as having a “sac-like” appearance.[1] Anetoderma is divided into two forms: primary, which is idiopathic and occurs on clinically normal skin, and secondary, which follows a prior dermatosis. Primary anetoderma has been described in association with autoimmune conditions such as systemic lupus erythematosus, systemic sclerosis, antiphospholipid antibody syndrome and thyroiditis, as well as in association with HIV.[2,3]

CASE PRESENTATION

A 23 year old woman with no significant past medical history presented to the clinic for evaluation of “spots” that gradually appeared over 2-3 years. The lesions erupted without associated redness, itching, scaling, or burning. She stated that the lesions had stabilized over the two years prior to presentation. The patient previously reported a history of mild acne vulgaris in a small area that was involved, though she stated her acne was not extensive. A prior biopsy was consistent with dermal fibrosis consistent with scar.

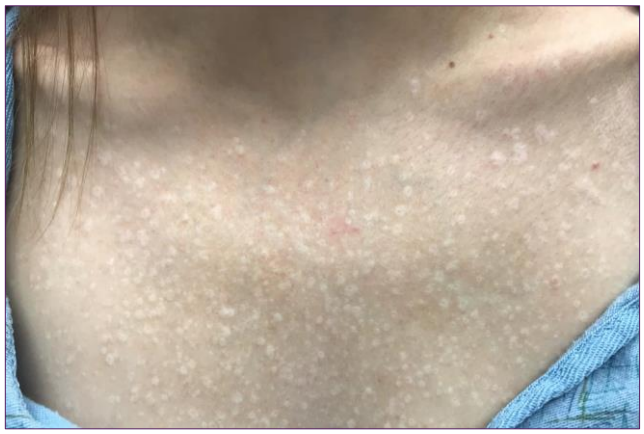
On physical exam, the patient’s trunk and bilateral proximal extremities were covered by innumerable hypopigmented to flesh colored atrophic plaques that herniated with slight palpation (Figure 1). The face, neck

Figure 1. Hypopigmented to flesh colored atrophic plaques



and distal extremities were spared. A test area was chosen to treat with a fractionally ablative erbium YAG laser (2940-nm) to a 1 mm depth, density of 11%, with three passes and a clinical end point of pinpoint bleeding. The patient was seen in follow up about three months after treatment with equivocal response (Figure 2). A second lesion was treated in fully ablative mode with the Er:YAG laser, but also did not result in significant improvement. Given her lack of response, repeat biopsy was performed at eight months after treatment, which showed superficial and focal loss of elastin, consistent with anetoderma (Figure 3). An autoimmune workup including ANA, lupus anticoagulant, and antibodies against cardiolipin, beta-2 glycoprotein, thyroglobulin and thyroid peroxidase, as well as comprehensive metabolic panel, complete blood count, thyroid tests, and HIV were performed and were unremarkable.

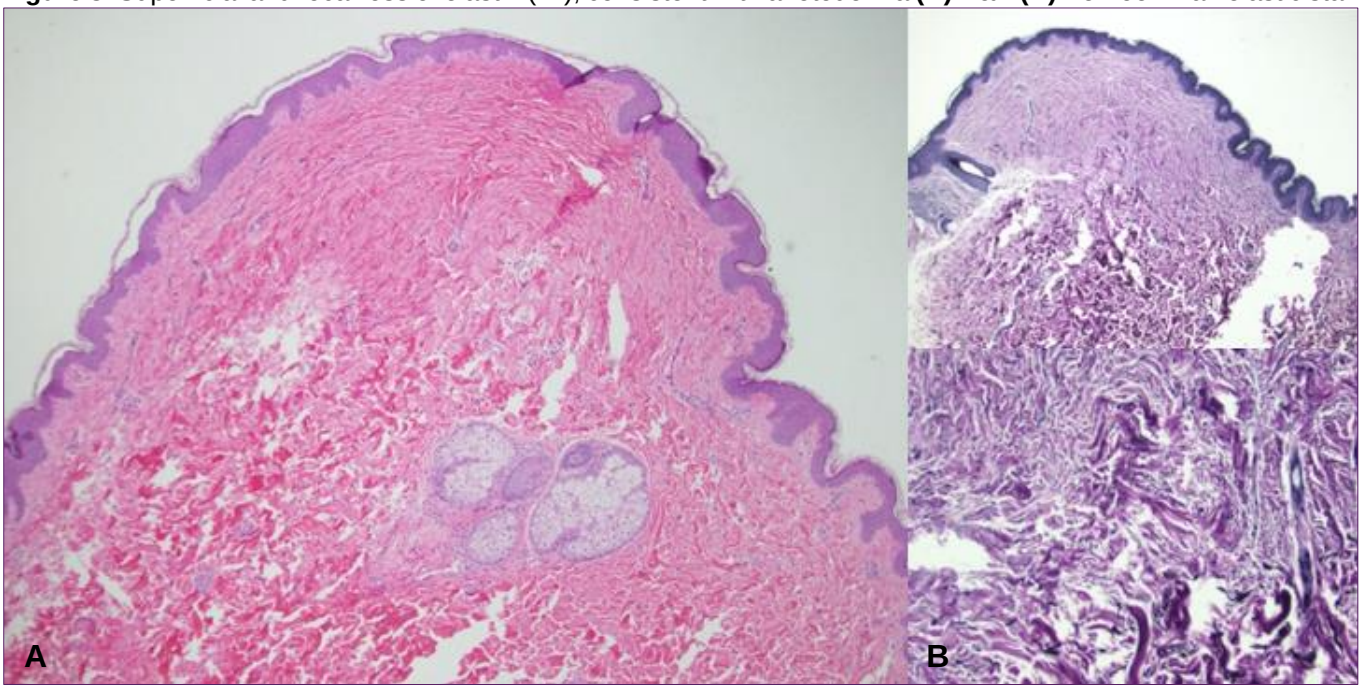
Figure 2. Equivocal response 3 months after treatment



DISCUSSION

Primary anetoderma has historically been subdivided into two subtypes: the Jadassohn-Pellizzari type, with preceding inflammation, and the Schweninger-Buzzi type, which appears spontaneously.[4] The two types have a similar course so the division is primarily considered academic. The trunk and extremities are the most commonly

Figure 3. Superficial and focal loss of elastin (4x), consistent with anetoderma **(A)** H&E **(B)** Verhoeff-Van elastic stain



involved sites.[4] Due to the above associations, primary anetoderma should prompt an autoimmune workup including ANA, HIV, and antiphospholipid panel (lupus anticoagulant, anticardiolipin antibody and anti-beta-2-glycoprotein).[3] The current case is best classified as idiopathic primary anetoderma of the Schweninger-Buzzi subtype with no associated autoimmunity markers, thyroid abnormalities or HIV.

There are no established treatment recommendations for anetoderma. Topical corticosteroids and systemic medications such as hydroxychloroquine have been tried with variable success. Several case reports suggest laser therapy may be helpful for anetoderma. A 10,600-nm CO_2 laser using the pinhole method was used successfully to treat lesions on the ear of an eight-year-old boy with secondary anetoderma following juvenile xanthogranuloma.[5] Anetoderma secondary to a severe sun burn was successfully treated with 595-nm pulsed-dye laser combined with 1550-nm non-ablative fractionated laser.[6] A 10,600-nm CO_2 laser was used to successfully treat anetoderma secondary to Steven-Johnson syndrome.[7] These cases show promise for energy based treatments for secondary anetoderma. Unfortunately, ablative laser treatment was not as successful in our patient with primary anetoderma, likely given the depth and number of lesions.

Although it is indolent, the lesions of anetoderma persist and may be associated with significant aesthetic changes causing potential psychosocial difficulties. Anetoderma has been successfully treated with ablative, pulsed dye and non-ablative fractionated lasers. Patients with secondary anetoderma and anetoderma limited to a relatively small body surface area may be more amenable to treatment than patients with extensive involvement.

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Corresponding Author:
James C. Prezzano, MD
University of Rochester Dermatology at College Town
40 Celebration Drive
Rochester, NY 14620
Phone: 585-275-7546
Fax: 585-461-3509
Email: James_Prezzano@URMC.Rochester.edu

References:

1. Emer J, Roberts D, Sidhu H, et al. Generalized anetoderma after intravenous penicillin therapy for secondary syphillus in a HIV patient. *J Clin Aesthet Dermatol.* 2003;6:23-28.
2. Göebel-Pinto JB, de Almeida HL Jr, de Castro LAS, Rocha NM. Ultrastructural aspects of primary anetoderma. *J Cutan Pathol*. 2017;44:786-789.
3. Tong LX, Beasley J, Meehan S, et al. Primary anetoderma with undifferentiated connective tissue disease. *Dermatol Online J*. 2017;23.
4. Kineston DP, Xia Y, Turiansky GW. Anetoderma: a case report and review of the literature. *Cutis*. 2008;81:501-6.
5. Lee SM, Kim YJ, Chang SE. Pinhole carbon dioxide laser treatment of secondary anetoderma associated with juvenile xanthogranuloma. *Dermatol Surg*. 2012 Oct;38:1741-3.
6. Wang K, Ross NA, Saedi N. Anetoderma treated with combined 595-nm pulsed-dye laser and 1550-nm non-ablative fractionated laser. *J Cosmet Laser Ther*. 2016;18:38-40.
7. Cho S, Jung JY, Lee JH. Treatment of anetoderma occurring after resolution of Stevens-Johnson syndrome using an ablative 10,600-nm carbon dioxide fractional laser. *Dermatol Surg*. 2012;38:677-9.