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Frontotemporal hairline recession in a postmenopausal woman

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The patient

A 69-year-old postmenopausal woman consulted for frontal hair loss for two years. She had started menopause at the age of 50 years old and had been taking bisphosphonates for her osteoporosis for two years. Her clinical history, including gynecological data, was otherwise negative. Anamnestic data ruled out the possibility of traction alopecia. Dermatological examination revealed a Fitzpatrick skin type III. She had a linear frontotemporal recession with perifollicular erythema, lonely hairs on the frontal region, and scarring alopecia (Figure 1). The patient had a total loss of eyebrows but she did not have body hair loss. There were no other skin or mucosal abnormalities. Thyroid hormone function was also normal. Dermoscopy with a non-contact polarizing FotoFinder dermatoscope x20 (FotoFinder Systems, Inc, Bad Birnbach, Germany) revealed perifollicular erythema and very mild perifollicular scaling in addition to hair shaft dystrophy and broken hair. Furthermore, dermoscopy noted the presence of white dots coexisting with irregular white and pink areas devoid of hair follicular openings (Figure 2). No prior topical treatment was used before our consultation. A 4 mm scalp punch biopsy from the frontal hairline was performed. His-

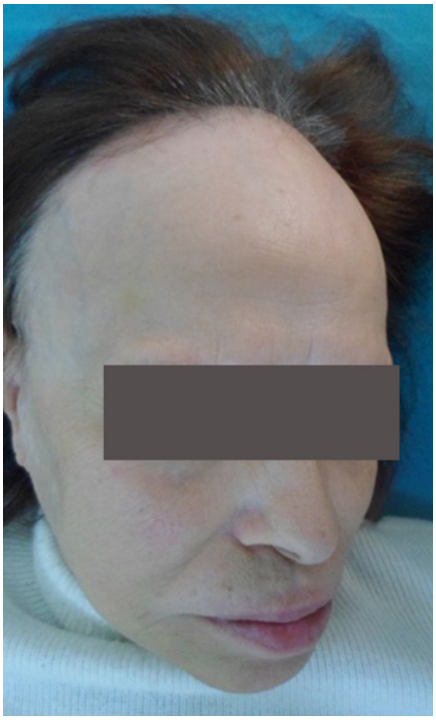

Figure 1. Scarring alopecia affecting the frontotemporal hairline. [Copyright: ©2015 Zaouak et al.]

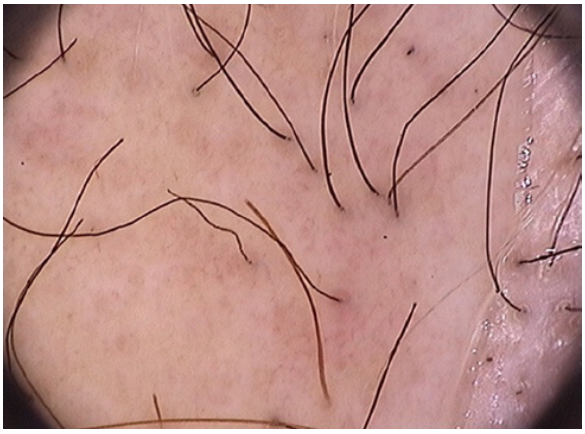

Figure 2A. Perifollicular erythema, very mild perifollicular scaling, acquired hair shaft dystrophy and broken hair. [Copyright: ©2015 Zaouak et al.]

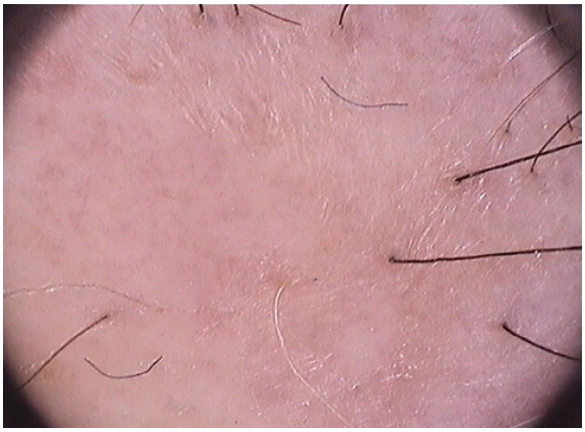

Figure 2B. White dots with irregular white and pink areas devoid of hair follicular openings. [Copyright: ©2015 Zaouak et al.]

topathological examination revealed perifollicular lamellar fibrosis, loss of sebaceous glands and a lichenoid lymphocytic infiltrate targeting the infundibulum and isthmus (Figure 3).

What is your diagnosis?

Diagnosis

Frontal fibrosing alopecia

Clinical course

The patient was treated with minoxidil 2% with a slight improvement of her scarring alopecia.

Discussion

Frontal fibrosing alopecia (FFA) is a relatively recently recognized condition of unknown origin and was first described in 1994 [1]. It is generally considered as a variant of lichen planopilaris primarily affecting postmenopausal women.

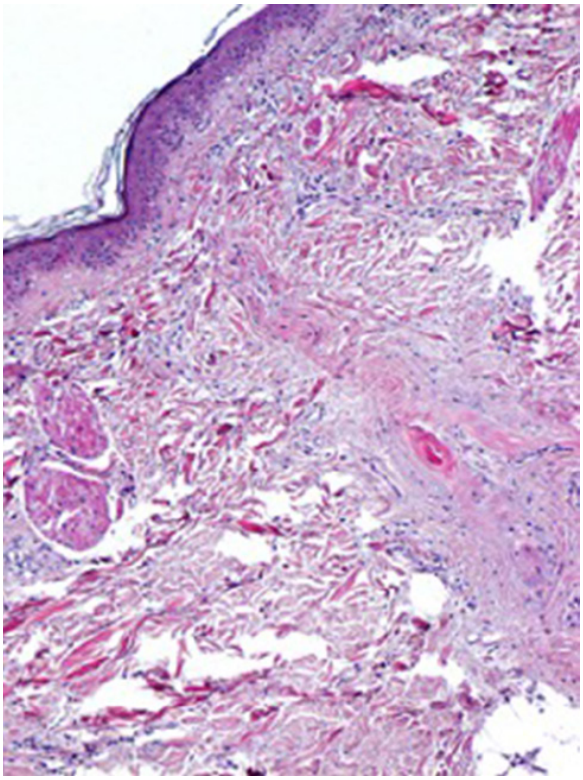

Figure 3. Perifollicular lamellar fibrosis, loss of sebaceous glands and a lichenoid lymphocytic infiltrate targeting the infundibulum and isthmus (H&E X40). [Copyright: ©2015 Zaouak et al.]

The hormonal imbalance caused by the decrease of estrogens associated with menopause could be the main trigger that creates the inflammatory scarring reaction of FFA in predisposed patients [2]. It is a disease that is diagnosed clinically in most cases. The progressive recession of the frontotemporal hairline is the most constant and characteristic clinical manifestation of FFA. It occurs symmetrically and bilaterally giving rise to a band of alopecia between 0.5 cm and 8 cm from the original hairline. Hair loss from the lateral third of the eyebrows is also characteristic of FFA [3]. Histologic features of FFA and lichen planopilaris are similar: both demonstrate a follicular lichenoid inflammatory infiltrate involving the isthmus and infundibulum, perifollicular fibrosis and fibrous tracts as seen in our patient [4]. Typical dermoscopic findings, as seen in our patient, include mainly the absence of follicular openings, perifollicular scaling and perifollicular erythema [5,6]. Trichoscopy appears to be a non-invasive diagnostic tool for the diagnosis and follow-up of FFA. In fact, in a recent study including 79 patients [5], the authors concluded that perifollicular erythema may represent a direct trichoscopic marker of disease activity in FFA.

Our patient had a scarring alopecia of the scalp margin and FFA was diagnosed mainly on clinical appreciations. However, in front of an early stage of FFA, dermoscopy appears to be helpful to establish differential diagnosis

between traction alopecia, alopecia areata and cicatricial marginal alopecia. In fact, our patient had a cicatricial alopecia with the absence of yellow dots and dystrophic hairs, which are the most relevant dermoscopic findings in alopecia areata. Anamnestic data ruled out the possibility of traction alopecia characterized by the absence of miniaturized hairs, white dots and fractured hair shafts at dermoscopic examination [7-9]. As for cicatricial marginal alopecia (CMA), this entity is characterized by an area of permanent hair loss that involves mainly the crown and vertex and spreads centrifugally. CMA is characterized dermoscopically by low hair density, loss of follicular ostia with a peripilar white gray halo around the emergence of hairs that were absent in our patient [10,11].

Currently, no treatment protocols exist for FFA. Stabilization of hair loss is occasionally observed with various topical or systemic therapies such as oral 5-α-reductase inhibitors, hydroxychloroquine, minoxidil and topical or intralesional corticosteroids. The aim of the treatment is to arrest hair loss. Improvement of FFA was most often seen when treated with oral finasteride or dutasteride, but a spontaneous stabilization of the disease may also occur. The regrowth of hair is usually minimal and always located at the hairline [12]. Some treatments may reduce inflammation, but the impact on progression of alopecia is uncertain.

We report this case not only for the rarity of the disease but also to underline the role of dermoscopy as a very useful tool in the diagnosis of frontal fibrosing alopecia. In fact, the characteristic clinical presentation together with typical dermoscopic features could help in avoiding unnecessary biopsies in patients with frontal fibrosing alopecia. Hence, dermoscopy could improve diagnostic accuracy of hair and scalp disorders.

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