Frontal Fibrosing Alopecia: Dermoscopic Features☆

Alopecia frontal fibrosante. Hallazgos dermatoscópicos

Dear Editor:

First described by Kossard[1] in 1994, frontal fibrosing alopecia is a type of cicatricial alopecia that is clinically characterized by progressive and symmetrical recession of the frontotemporal hairline. It is accompanied by partial alopecia of the eyebrows in most cases[2] and loss of body hair in a variable number of patients.[3] It is more frequent in postmenopausal women, although there have been reports of cases in men and premenopausal women.[2,4] Histopathology shows it to be a variant of lichen planopilaris, which is characterized by a reduced number of hair follicles, a perifollicular lymphocytic inflammatory infiltrate, and fibrosis.[5] Few published studies describe the dermoscopic features of frontal fibrosing alopecia.[6,7]

We report the case of a 51-year-old woman who consulted with a 12-month history of hair loss at the frontal hairline. No other symptoms or associated diseases were present. Physical examination revealed a bald patch in the form of a band that delimited the hairline of the frontal region (Fig. 1). She also had partial alopecia of the eyebrows. The remaining findings were normal. The patient had previously taken high-potency topical corticosteroids and 2% minoxidil with no signs of improvement.

Dermoscopy (Dermlite 00 multispectral) highlighted a marked reduction in the number of follicular ostia in the central area and erythema, perifollicular desquamation, and perifollicular blue-gray dots at the border of the patch (Fig. 2). A biopsy specimen was taken, and histopathology revealed a marked reduction in the density of the pilosebaceous units, as well as laminar fibrosis and a perifollicular lymphocytic inflammatory infiltrate around the isthmus and infundibulum (Fig. 3). The clinical, dermoscopic, and histopathologic findings confirmed a diagnosis of frontal fibrosing alopecia.

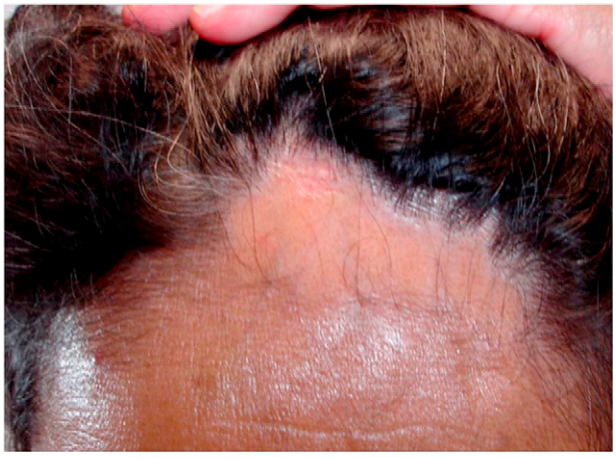

Figure 1 Woman aged 51 years with a bald patch in the form of a band on the frontotemporal region. Note the receding hairline.

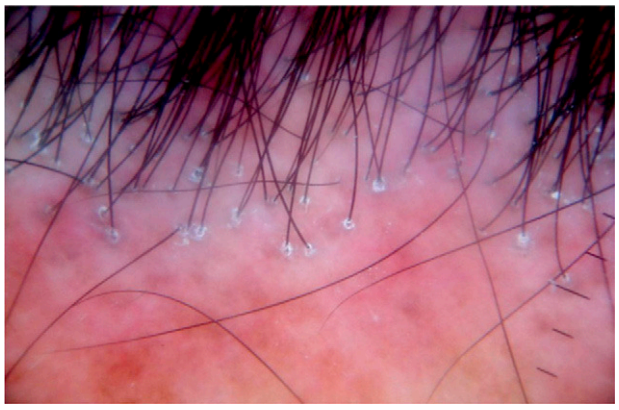

Figure 2 Dermoscopy (Dermlite 00 multispectral) revealed markedly reduced follicular ostia, scale, erythema, and perifollicular blue-gray dots.

Since it was first described in 1994, just under 200 cases of frontal fibrosing alopecia have been reported throughout the world.[1,2,4] Although initially considered an uncommon entity, this condition is believed to be a much more common cause of cicatricial alopecia than previously thought.[2] There are few published reports of dermoscopic findings in patients with this condition. Several dermoscopic findings are characteristic of frontal fibrosing alopecia, whereas others also appear in other types of cicatricial alopecia, such as discoid lupus erythematosus or lichen planopilaris. Inui et al.[6] reported 3 dermoscopic findings in 4 patients with frontal fibrosing alopecia, namely, reduced follicular ostia, erythema, and perifollicular scale, which were also found in the case we report. Other dermoscopic findings also reported in patients with this condition include ramified capillaries, honeycomb-like pigment network, white patches, white dots, and vellus hair.[7] In the present case, we also identified blue-gray dots around some follicles, a finding that has previously been reported in discoid lupus erythematosus, but not in frontal fibrosing alopecia.[7] Although no studies have established a correlation between dermoscopic findings and histopathologic findings in cicatricial alopecia, it has been suggested that the white patches correspond to fibrosis and that they are more commonly found in long-standing bald patches.[7] The blue-gray dots correspond to melanophages in the papillary dermis and the white dots to pigment incontinence[7]; the honeycomb pattern is the result of exposure of the bald patches to sunlight.[8] Erythema and hyperkeratosis are the results of inflammation in the follicle and the hyperkeratosis that develops in lichen planopilaris and frontal fibrosing alopecia.[8]

Dermoscopic criteria are proving useful in the diagnosis of alopecia.[9] In the present case, dermoscopic findings enabled us to rule out other causes of alopecia, such as alopecia areata. The correlation between clinical findings, dermoscopic findings, and histopathologic findings enabled us to establish a diagnosis of frontal fibrosing alopecia.

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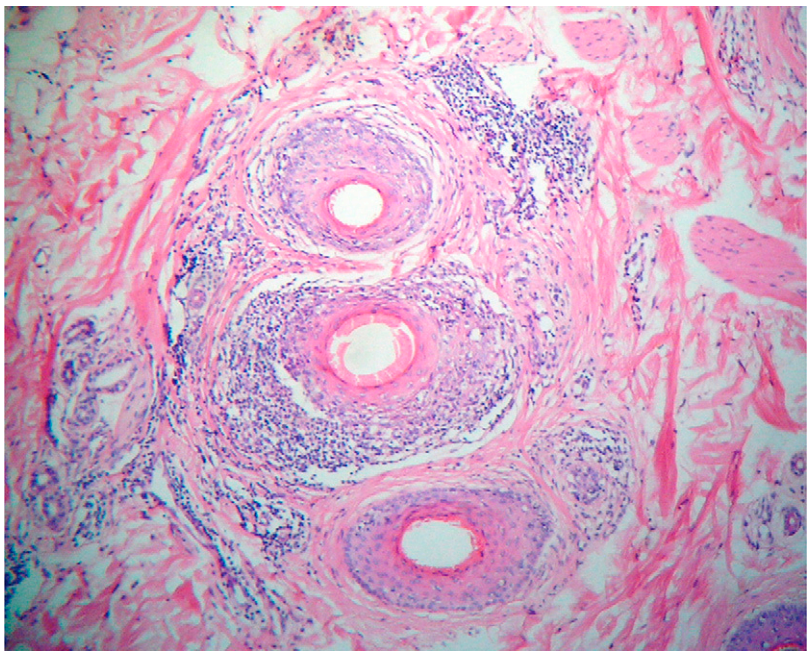

Figure 3 Histopathology revealed a reduction in the number of hair follicles, lymphocytic inflammatory infiltrate, and laminated perifollicular fibrosis (hematoxylin–eosin, original magnification 40×).

To date, no cases of frontal fibrosing alopecia have been reported in Latin America. Although this condition has characteristic manifestations, dermoscopy provides more detailed information. The present case underlines the role of dermoscopy for evaluating cicatricial alopecia and establishing a differential diagnosis.

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H. Mireles-Rocha,[a] L.E. Sánchez-Dueñas,[b,*]
M. Hernández-Torres[c]

[a] *Dermatología, Colima Col., Mexico*
[b] *Instituto Dermatológico de Jalisco Dr. José Barba Rubio, Mexico*
[c] *Departamento de Dermatopatología, Instituto Dermatológico de Jalisco Dr. José Barba Rubio, Mexico*

* Corresponding author.
E-mail address: dr_lesd@hotmail.com
(L.E. Sánchez-Dueñas).

Good Response of Hyperkeratotic Palmoplantar Psoriasis to Ustekinumab☆

Psoriasis hiperqueratósica palmoplantar con excelente respuesta a ustekinumab

To the editor:

Although only a small area of the body is affected in palmoplantar psoriasis or psoriasis of the palms and soles, this condition causes considerable functional impairment due to hyperkeratosis, fissures, and erythema, and occasionally inflammation and pustules. These symptoms may seriously interfere with the patient's quality of life and may be disabling.[1] Moreover, the lack of a standard treatment hinders the therapeutic management of this clinical variant. We report a case of hyperkeratotic palmoplantar psoriasis that had not responded to several conventional therapies but responded well to treatment with ustekinumab.

The patient was a 56-year-old man with a 1.5-year history of palmoplantar psoriasis whose treatment history at another hospital was as follows: high potency topical corticosteroids and calcipotriol (no improvement); topical psoralen UV-A therapy 3 times a week for 6 months, (poor response); and methotrexate 15 mg/wk associated with elevated transaminase values (5 times baseline) and marked gastrointestinal symptoms that led to withdrawal of treatment after 2 months.

The patient presented at our hospital with severe palmoplantar hyperkeratosis, fissuring, and 100% involvement of the palms and soles; it was difficult for him to walk and carry out his daily activities (Fig. 1). There were no other lesions or joint involvement. Treatment with acitretin 50 mg/d (weight 76 kg, 0.66 mg/kg) resulted in some improvement, but was poorly tolerated because of dry skin, cheilitis, joint pain, gynecomastia, alopecia, and hypertriglyceridemia (352 mg/dL). Reduction of the dose to 35 mg yielded little improvement in the side effects and worsening of the lesions. Consequently, treatment was suspended after 9 months.

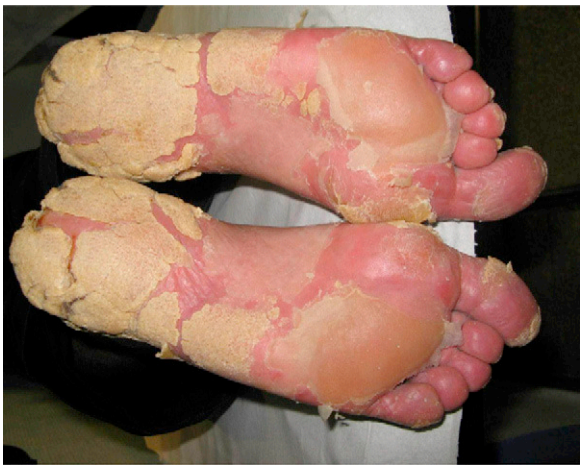

Figure 1 Soles of the feet with marked hyperkeratosis prior to treatment.

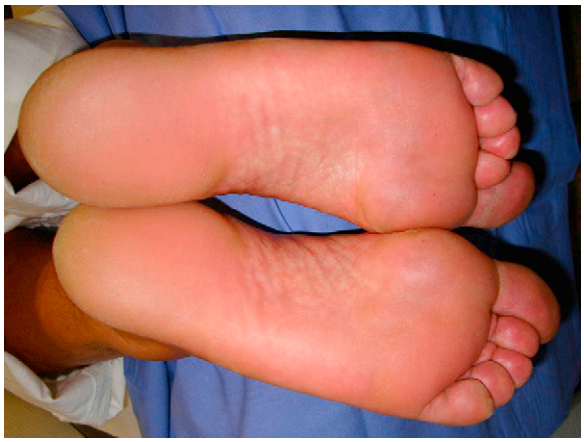

Figure 2 Excellent response after 16 weeks' treatment with ustekinumab.

Owing to the failure of conventional therapies, we decided to begin treatment with a biologic agent. As the patient was a frequent traveler who spent long periods away from home, ustekinumab 45 mg was prescribed and administered according to the conventional regimen (initial dose followed by another 4 weeks later and every 3 months thereafter). We successfully applied for a compassionate use permit and the patient duly signed informed consent. At 16 weeks, after receiving 2 doses of ustekinumab 45 mg, the patient reported complete resolution of his disease (Fig. 2). Clinical findings and tests showed no drug-related side effects. The patient has continued with the treatment for the last 12 months with excellent results and no adverse events.

Hyperkeratotic palmoplantar psoriasis has traditionally been treated with the drugs used for psoriasis vulgaris (with varying, but generally poor, results) and often represents a challenge to dermatologists.[1–4] In cases where conventional therapies have failed, anti-tumor necrosis factor agents have proved effective.[2,5] However, cases have also been reported in which these agents have paradoxically been associated with new onset or worsening of palmoplantar psoriasis or pustulosis. The mechanism is poorly understood and biologic drugs should therefore be used with caution in this setting.[6] Efalizumab is another biologic agent that has proved to be effective in palmoplantar psoriasis,[3] although international sales of this drug have now been discontinued because of an increased risk of progressive multifocal leukocytopenia.

Ustekinumab – the biologic agent most recently approved for use in psoriasis – is a fully human monoclonal antibody that binds to the shared p40 subunit of the interleukins (IL) 12 and IL-23 and blocks their action. To date, ustekinumab has only been studied in plaque psoriasis, and pustular, erythrodermic, and palmoplantar psoriasis are not, therefore, included in the summary of product characteristics; the only evidence available on its use in these variants comes from case series and isolated case reports. Since ustekinumab is a new drug, experience of its use

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