Frontal Fibrosing Alopecia: A Retrospective Analysis of 72 Patients from a German Academic Center

Markus V. Heppt, MD, MSc[1] Valerie Letulé, MD[1] Ieva Laniauskaite, MD[2] Markus Reinholz, MD[1] Julia K. Tietze, MD[1] Hans Wolff, MD[1] Thomas Ruzicka, MD[1] Elke C. Sattler, MD[1]

[1] Department of Dermatology and Allergy, Ludwig-Maximilian-University Munich, Munich, Germany
[2] Centre of Dermatovenereology, Vilnius University, Vilnius, Lithuania

Facial Plast Surg 2018;34:88–94.

Address for correspondence Markus V. Heppt, MD, MSc, Department of Dermatology and Allergy, Ludwig-Maximilian-University Munich, Frauenlobstraße 9-11, 80337 Munich, Germany (e-mail: Markus.Heppt@med.uni-muenchen.de).

Abstract

Frontal fibrosing alopecia (FFA) describes the scarring, band-like recession of the frontotemporal hairline. Treatment is difficult, and currently, no evidence-based therapy exists. The purpose of this study is to report clinical features and treatment responses in a large cohort of patients with FFA. The authors analyzed a series of 72 patients with a clinical or histologic diagnosis of FFA. A total of 70 patients were female (97.2%), and 2 were male (2.8%). In females, the first onset of FFA was postmenopausal in 81.4% ($n = 57$). Documented eyebrow loss was present in 61.1% ($n = 44$), whereas involvement of eyelashes and body hair was reported in only 4.2% ($n = 3$) and 5.6% ($n = 4$), respectively. Clinical symptoms were itching (40.3%, $n = 29$) and trichodynia (4.2%, $n = 3$) in the alopecic area. Virtually all patients were treated with topical high-potency steroids. Additional treatments were topical tacrolimus, systemic retinoids, and hydroxychloroquine. A total of 48 patients (66.7%) received a combination of high-potency steroids with topical pimecrolimus. In this subgroup, subjective improvement or disease stabilization was reported by 64.6% ($n = 31$), and the hairline was stabilized on average after 9 to 12 months of therapy. The combination therapy of topical high-potency steroids with pimecrolimus may be an effective and steroid-saving treatment for FFA.

Keywords
- calcineurin inhibitor
- frontal fibrosing alopecia
- lichen planopilaris
- steroid-saving therapy

Frontal fibrosing alopecia (FFA) was first described by Kossard in 1994 as a progressive, frontal, scarring recession of the hair line.[1] As this condition was initially observed in postmenopausal women, it was formerly referred to as postmenopausal FFA.[1–3] However, several reports demonstrated its occurrence in young men and premenopausal women, although the precise incidence and prevalence remains unclear.[4–6] Typical clinical features are cicatricial alopecia with an irreversible recession of the frontotemporal hairline.[7–9] FFA may involve eyebrows, eyelashes, body hair, or beard.[4] Furthermore, facial papules have been described as part of the disease in some patients.[4]

The etiology and pathogenesis of FFA are increasingly being understood.[10] It is considered a variant of lichen planopilaris (LPP) because both conditions share clinical and pathologic features.[11,12] The histological image is characterized by a marked reduction of hair follicles with a dense lichenoid perifollicular infiltrate and follicular fibrosis.[13,14] Thus, management strategies for FFA have been adopted from LPP with topical corticosteroids being a mainstay of treatment.[15] Also, systemic corticosteroids, antimalarial agents, or oral finasteride have been reported previously with varying rates of success.[4,15,16] Since FFA is a chronic and progressive condition, most patients require long-term treatment. Although topical

published online December 26, 2017

Issue Theme Managing the Thick Skin in Facial Plastic Surgery; Guest Editor, Roxana Cobo, MD

DOI https://doi.org/10.1055/s-0037-1615281.
ISSN 0736-6825.

corticosteroids have minimal side effects, risks can include skin atrophy and telangiectasia formation. Therefore, steroid-saving regimens are required. Substituting or alternating steroids with topical calcineurin inhibitors is well established in other inflammatory skin diseases, such as atopic eczema.[17] However, such therapy has not been evaluated in a larger cohort of FFA long-term.

This retrospective analysis summarizes the clinical manifestations, relevant comorbidities, and treatment outcomes in 72 patients with FFA, who were treated at a large German academic trichology unit. Furthermore, it investigates a subgroup of 48 patients who received a combination therapy of topical high-potency steroids and pimecrolimus cream.

Patients and Methods

This is a retrospective monocentric analysis of patients with a diagnosis of FFA treated at the trichology unit of the Department of Dermatology of the Ludwig-Maximilian-University Munich between 2010 and 2015. The study was approved by the Institutional Review Board of the Ludwig-Maximilian-University (approval number: 157–16) and followed the principles of the Declaration of Helsinki.

The diagnosis was based on the typical clinical and dermoscopic features of FFA such as irregular recession of the frontotemporal hairline with eyebrow loss or perifollicular erythema, follicular hyperkeratosis, and loss of follicular orifices. The disease activity was assessed based on perifollicular erythema and perifollicular scaling. A skin biopsy was performed to confirm the diagnosis of FFA in 19 cases (26.4%). However, histological examination was not required for inclusion in the study.

Data were collected and extracted from standardized patient records. Patient demographics included: age at first presentation and onset of FFA, gender, race, menopausal status in females, and relevant medical comorbidities. Other skin disorders possibly related to FFA, such as lichen planus, vitiligo, androgenetic alopecia (AGA), and lichen sclerosus et atrophicus (LSA) were reported.

The success of the respective treatments was assessed by the recession of the hairline in the patients. It was measured from mid-eyebrow, upper rim level of both sides (right and left) as well as from the center point between both eyebrows (middle) to the macroscopically visible recessed hairline and indicated in centimeters. To obtain a single representative value, the mean of these three measurements (right, left, and middle) was calculated. If the hairline was not consistently documented in the records, digital macroscopic and dermoscopic photographs served as additional evaluation of treatment success. The subjective treatment success and patient satisfaction were assessed on a three-tier scale (worsening, stable disease, improvement) along with the improvement of symptoms such as trichodynia and itching. Also, the use of a wig was reported.

Descriptive statistics were applied for patient demographics and comorbidities. Frequencies were calculated for categorical variables. All analyses were performed with the GraphPad Prism software (version 5.01).

Results

Patient Characteristics and Comorbidities

A total of 72 patients with a clinical or histological diagnosis of FFA were included. Most patients were Caucasian (94.4%, $n = 68$), one was Latin American (1.4%). The majority of patients were female (97.2%, $n = 70$) and only two were male (2.8%). Most women were postmenopausal at the onset of FFA (81.4%, $n = 57$). Of the remaining 18 females, 6 were premenopausal (8.6%) and 7 could not recollect time of hair loss onset (10.0%). The median age of symptom onset was 62 years (range: 31–85). The median age of the first presentation to the clinic, however, was 64 years (range: 34–86).

Hypothyroidism and arterial hypertension were each observed in 21 cases (29.2%) as coexistent diseases. Seven individuals (9.7%) suffered from concurrent rheumatic diseases, such as rheumatoid arthritis (8.3%, $n = 6$) and giant cell temporal arteritis (1.4%, $n = 1$). Coexistent skin diseases were psoriasis (2.8%, $n = 2$), mucosal lichen planus (4.2%, $n = 3$), and alopecia areata (1.4%, $n = 1$). In the three patients with mucosal lichen planus, the mucosal lesions preceded the onset of FFA symptoms. A total of 25 (34.7%) cases presented with either concurrent or a reported history of AGA. Neither vitiligo nor LSA which were both associated with FFA in other studies were observed in any patient (►**Table 1**).[18]

Clinical Features

All patients presented with scarring recession of the frontal and temporal hairline. The median hairline recession at initial presentation was 7.8 cm (range: 5.0–10.2 cm). Perifollicular erythema and scaling were observed in 57 (79.2%) and 36 (50.0%) patients, respectively. Eyebrow loss was observed in 44 subjects (61.1%). In contrast, loss of eyelashes was documented in only three patients (4.2%). Involvement of body hair was evident in four cases (5.6%). The most common clinical symptoms of FFA were itching and burning of the alopecic area (40.3%, $n = 29$), while trichodynia was rare (4.2%, $n = 3$). Features of lichen planopilaris were found in 11 patients (15.3%). There were no cases of cutaneous lichen planus. However, three individuals had signs of mucosal, that is, oral or genital, involvement (4.2%). One patient (1.4%) had signs of nail disease (►**Table 2**). Nine women reported wearing a wig due to the condition (12.5%).

Treatment

Virtually all patients were treated with topical high-potency steroids. The majority received several different preparations in the course of the disease. The most common agents were clobetasol propionate (81.9%, $n = 59$) and betamethasone valerate (27.8%, $n = 20$). Medium to lower-mid potency steroids, such as mometasone furoate, methylprednisolone aceponate, or hydrocortisone butyrate, were applied in only a few patients (4.2%, $n = 3$). Steroid-induced local side effects, such as the formation of telangiectasia and marked skin atrophy were detected in seven cases (9.7%, ►**Fig. 1**). Other FFA-relevant topical and systemic therapies which were used adjunct with steroids are shown in ►**Table 3**. Four patients (5.6%) received systemic retinoids but were lost to follow-up

Table 1 Patient characteristics and comorbidities

Demographics	
Gender	
Female	70 (97.2%)
Male	2 (2.8%)
Age at onset of FFA	
Median	62 y
Range	31–85 y
Age at first presentation	
Median	64 y
Range	34–86 y
Menopausal status	
Postmenopausal	57 (81.4%)
Premenopausal	6 (8.6%)
Unknown	7 (10.0%)
Ethnicity	
Caucasian	68 (94.4%)
Latin American	1 (1.4%)
Unknown	3 (4.2%)
Comorbidities	
Endocrine disorders	
Hypothyroidism	21 (29.2%)
Diabetes mellitus type II	2 (2.8%)
Hyperparathyroidism	1 (1.4%)
Rheumatic disorders	
Polyarthritis	6 (8.3%)
Giant cell arteritis	1 (1.4%)
Arterial hypertension	21 (29.2%)
Skin diseases	
Psoriasis	2 (2.8%)
Mucosal lichen planus	3 (4.2%)
Alopecia areata	1 (1.4%)
Melanoma	4 (5.6%)
Non-melanoma skin cancer	2 (2.8%)
Androgenetic alopecia	25 (34.7%)
Malignancies	
Leukemia	1 (1.4%)
Breast cancer	3 (4.2%)
Lung cancer	1 (1.4%)

Table 2 Clinical features and symptoms

Cutaneous and mucosal manifestations of frontal fibrosing alopecia and the lichen planus complex	
Loss of eyebrows	
Yes	44 (61.1%)
No	18 (25.0%)
Unknown	10 (13.9%)
Loss of eyelashes	
Yes	3 (4.2%)
No	39 (54.2%)
Unknown	30 (41.7%)
Itching	
Yes	29 (40.3%)
No	15 (20.8%)
Unknown	28 (38.9%)
Trichodynia	
Yes	3 (4.2%)
No	35 (48.6%)
Unknown	34 (47.2%)
Body hair involvement	
Yes	4 (5.6%)
No	38 (52.8%)
Unknown	30 (41.7%)
Features of lichen planopilaris	
Yes	11 (15.3%)
No	32 (44.4%)
Unknown	29 (40.2%)
Oral and genital lichen planus	
Yes	3 (4.1%)
No	40 (55.6%)
Unknown	29 (40.3%)
Nail involvement	
Yes	1 (51.4%)
No	40 (55.5%)
Unknown	31 (43.1%)

early, and the assessment of the treatment response was not possible. One patient (1.4%) received 200 to 400 mg hydroxychloroquine daily as systemic therapy. She was followed up for 13 months and showed a hairline recession of 0.7 cm.

To spare topical steroids and reduce steroid-induced adverse events, 48 patients (66.7%) received an alternating therapy of external high-potency steroids (clobetasol propionate, betamethasone valerate) and pimecrolimus 1% cream once daily. The application frequencies of the combination depended on disease activity, starting from three times per week of each agent for high disease activity, which was then lowered to two applications of steroid alternating with the calcineurin inhibitor twice weekly. With further reduction in the disease activity, the topical steroid was reduced to once weekly and discontinued finally. The application of pimecrolimus twice weekly was kept for long-term maintenance.

We further analyzed the subgroup of patients receiving this combination therapy, because topical calcineurin inhibitors have an established steroid-sparing capacity and high

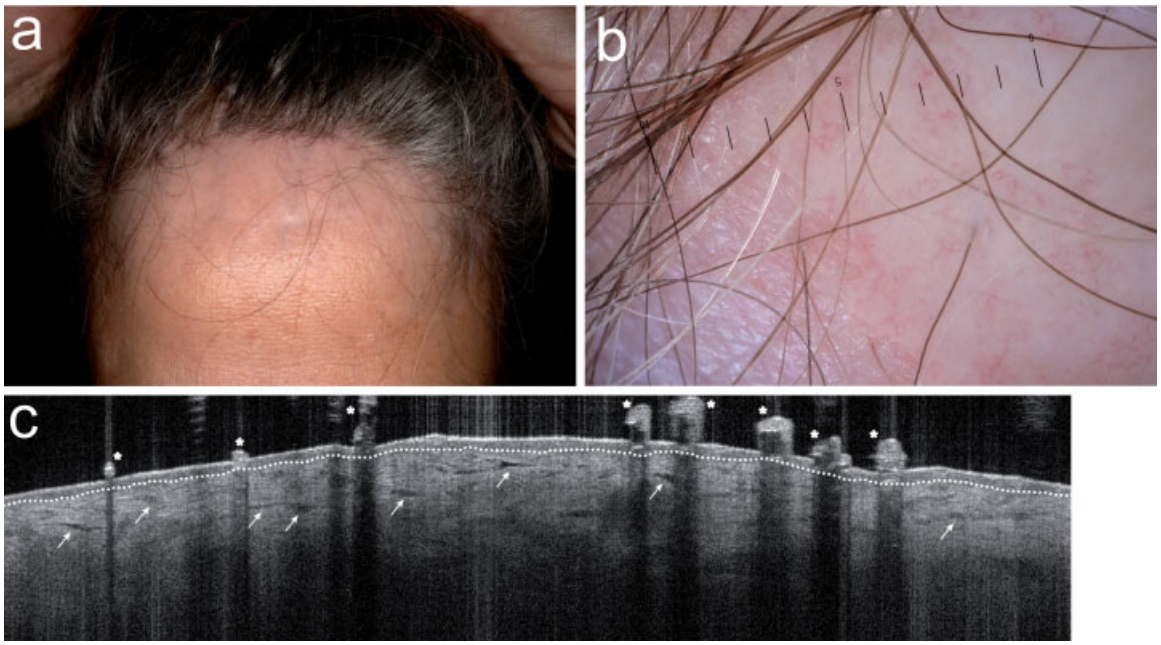

Fig. 1 Frontal fibrosing alopecia in a female patient who received high doses of topical clobetasol propionate and showed steroid-induced side effects. A steroid-saving combination therapy with pimecrolimus 1% cream was applied rather late in her case. (**a**) Photographs showing the macroscopic image with an irregular recession of the frontotemporal hairline. (**b**) Dermoscopic image revealing the formation of telangiectasia in steroid-treated areas (DermoGenius [DermoScan GmbH], scale bars as indicated in millimeters). (**c**) Exemplary images with optical coherence tomography (OCT) demonstrated multiple dilated vessels (white arrows) in the upper dermis and marked thinning of the epidermal layer (white dotted line). Asterisks show single floating hair with a dorsal shade in OCT (Vivosight).

Table 3 Treatment agents for frontal fibrosing alopecia

Topical agents[a]	
Glucocorticosteroids	
Clobetasol propionate	59 (81.9%)
Betamethasone valerate	20 (27.8%)
Mometasone furoate	1 (1.4%)
Methylprednisolone aceponate	1 (1.4%)
Hydrocortisone butyrate	1 (1.4%)
Topical calcineurin inhibitors	
Pimecrolimus	53 (73.6%)
Tacrolimus	13 (18.1%)
Systemic agents	
Hydroxychloroquine	1 (1.4%)
Retinoids	4 (5.6%)
Zinc supplementation	5 (6.9%)
Biotin supplementation	2 (2.8%)

[a]Many patients received several topical agents in the course of the disease. Thus, the percentages indicated here do not amount to 100%.

efficacy in other inflammatory skin conditions, such as atopic eczema.[17] Furthermore, this subgroup was large, well-documented, and more homogeneous regarding the therapy regimen compared with the patients who were not treated with this particular combination therapy.

The median duration of observation for the 48 patients receiving the combination therapy was 20 months (range: 3–62). Twenty-five patients (52.1%) initially reported itching, which resolved in 11 cases with therapy. Three patients (6.3%) complained of trichodynia with marked improvement in one case with therapy. Subjective improvement and stabilization of disease were reported by 19 (39.6%) and 12 (25.0%) patients receiving the combination therapy, respectively. In contrast, subjective worsening with therapy occurred in 11 cases (22.9%). Seven patients (14.6%) reported the use of wigs during the disease. Erythema and scaling were observed as adverse events in two patients (4.2%). However, steroid-induced skin atrophy and telangiectasia formation were rarely seen (►**Fig. 2**).

Data on hairline recession was available for up to 62 months in this subgroup. During the initial 9 months after the primary presentation, the hairline receded with a recession rate of approximately 0.4 to 0.5 cm per 6 months. However, the slope of the recession flattened from 9 months on and reached a plateau at around 12 months of treatment. The mean recession values compared with baseline are shown in ►**Fig. 3**.

Discussion

In concordance with previous reports, most patients were female, and the onset of disease symptoms occurred postmenopausally in more than 80% of cases.[1,2] We also identified two men within our population. This rate of 2.8% is comparable to other large-scale investigations reporting prevalence in males of 3 to 5% or less.[4,8,19] The most common

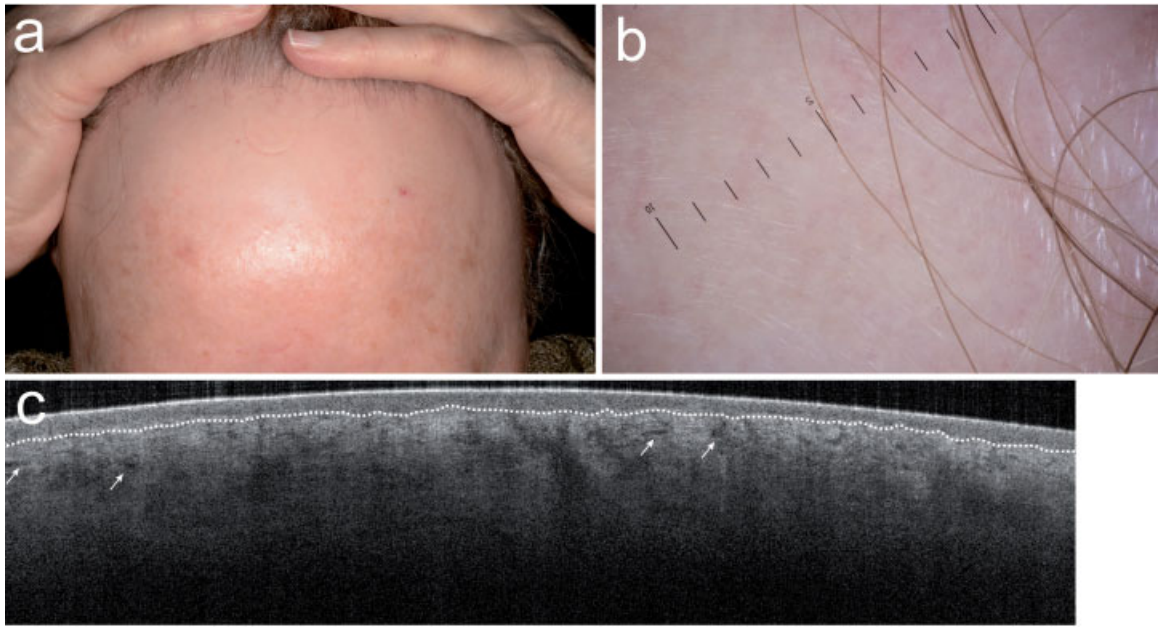

Fig. 2 Frontal fibrosing alopecia in another female patient. Although she initially presented with a hairline which was widely recessed, the topical treatment could be reduced to pimecrolimus 1% cream only for maintenance quite quickly with a stable response and controlled disease activity. (**a**) Photographs and (**b**) dermoscopic evaluation reveal little atrophy and telangiectasia formation (DermoGenius [DermoScan GmbH], scale bars as indicated in millimeters). (**c**) Exemplary optical coherence tomography images demonstrate only a few dilated vessels (white arrows) in the upper dermis and little epidermal atrophy (white dotted line, Vivosight).

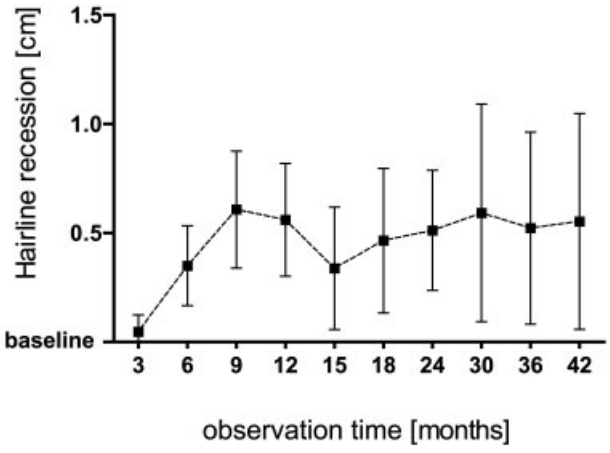

Fig. 3 The recession of the hairline in the subgroup of patients ($n = 48$) receiving a combination therapy of high-potency steroids and pimecrolimus 1% cream. The recession of the hairline is shown as a mean ± standard error of mean relative to the baseline value of the hairline (y-axis). During the initial 9 months of therapy, the recession followed a recession rate of approximately 0.4 to 0.5 cm per 6 months. The hairline stabilized from month 9 to 12 on and reached a plateau lasting up to 42 months.

comorbidities in our population were hypothyroidism and arterial hypertension. A survey conducted from 2005 to 2008 estimated that 29 to 31% of adults in the United States suffer from hypertension.[20] Thus, the prevalence of hypertension in our cohort appears consistent with figures in the general population. In contrast, the prevalence of subclinical or overt hypothyroidism ranges from 4 to 10% of adults in the general population.[21] We observed a high percentage of hypothyroidism (29.2%) in our collective.[22] This rate is higher than proposed recently in a large Spanish multicenter study where hypothyroidism was detected in 15% of patients with FFA.[4] Also, concomitant rheumatic diseases were observed in our study, strengthening the hypothesis that autoimmune processes may be involved in the pathogenesis of FFA.[23]

Due to the lack of well-designed, adequately powered, randomized controlled trials, there is currently no gold standard therapy for FFA.[15] Several agents have been tested for clinical efficacy with only limited success.[4,15] Superpotent steroids, delivered intralesionally or topically, are most commonly used in clinical practice. Virtually all of our patients received high-potency steroids at least once, albeit previous reports have failed to show a clear benefit for steroids.[1,2,15,24,25] To minimize side effects, 48 patients were treated with a combination of steroids and topical pimecrolimus. To our knowledge, this is the largest cohort receiving this regimen to date. More than half of the patients receiving this combination subjectively reported improvement or stabilization of the condition, indicating a high degree of patient satisfaction.

MacDonald et al recently reviewed a series of 60 cases out of whom 22 received topical calcineurin inhibitors revealing a mean hairline regression of 0.95 to 1.4 cm/year.[26] Others proposed a mean recession of approximately 1.05 cm/year on the basis of retrospective estimates.[4,18,27] In our study, the velocity of the hairline recession followed a similar rate within the initial 9 months of follow-up. However, we observed a disease stabilization beginning from months 9 to 12 after the onset of the tandem therapy. The recession of the hairline reached a plateau after 12 months of therapy. Interestingly, a

similar treatment effect was observed in patients treated with oral finasteride for FFA where four out of eight patients demonstrated an arrest of the alopecic band after 12 to 18 months on treatment.[24]

With regards to safety, the combination of steroid and pimecrolimus was well tolerated. Among all patients, the formation of telangiectasia was observed in 9.7% in the frontal and temporal area. This rate may be inflated because patients were regularly examined with dermoscopy and not only with the naked eye. Furthermore, some patients with FFA may show skin atrophy even without external steroid application after stabilization. Treatment-related side effects have rarely been assessed in previous studies, although steroids can induce skin atrophy and hence worsen the clinical image of cicatricial alopecia that is seen in late stage FFA.[15]

A major limitation of the present study is its retrospective design and lack of a control cohort. A direct comparison between topical and systemic agents was not possible because of the heterogeneity of the study population. A systematic literature review on the treatment in FFA and LPP did not identify any randomized controlled clinical trials for FFA.[15] Most investigations are retrospective case series and reports without control groups; only two prospective studies were found.[28,29] However, uncontrolled studies are at risk of overestimating the true treatment effect. Thus, we cannot exclude that results in the subgroup receiving high-potency steroids and pimecrolimus may reflect the natural disease course without a control cohort. This is of particular importance because Kossard and coworkers noted that 5 out of 16 women in his initial case series had spontaneously stabilized and, among those with active disease, some showed a rapid rate of hair loss and some were more slowly progressive.[1,2] Moreover, the assumption of a linear trend defined by a constant recession rate does not necessarily reflect the natural course of FFA as some studies demonstrated that disease progression could be self-limited and spontaneous stabilization may occur regardless of treatment. Some authors have suggested that the recession of the hairline follows a periodic or irregular rhythm.[7] Other limitations are the retrospective design with potential recall bias of the study participants and the unknown period between the first onset of the disease and treatment initiation which may confound the treatment effect.

Nevertheless, a combination therapy of high-potency topical steroids with pimecrolimus 1% cream may represent a promising, steroid-sparing treatment in FFA. Future prospective randomized trials with adequate control groups will be warranted to fully evaluate the safety and efficacy of the tandem treatment and to ultimately establish an evidence-based standard therapy for FFA.

Conflict of Interest

The authors declare no conflict of interest.

Funding

None.

Acknowledgments

The authors would like to thank Undine Wolf, Rehab Alharbi, and Diana Wittmann for their support with ►**Figs. 1** and **2**. They would also like to thank Lauren Metterle for critically reading and editing the article.

References

1 Kossard S. Postmenopausal frontal fibrosing alopecia. Scarring alopecia in a pattern distribution. Arch Dermatol 1994;130(06):770–774
2 Kossard S, Lee MS, Wilkinson B. Postmenopausal frontal fibrosing alopecia: a frontal variant of lichen planopilaris. J Am Acad Dermatol 1997;36(01):59–66
3 Camacho Martínez F, García-Hernández MJ, Mazuecos Blanca J. Postmenopausal frontal fibrosing alopecia. Br J Dermatol 1999; 140(06):1181–1182
4 Vañó-Galván S, Molina-Ruiz AM, Serrano-Falcón C, et al. Frontal fibrosing alopecia: a multicenter review of 355 patients. J Am Acad Dermatol 2014;70(04):670–678
5 Nusbaum BP, Nusbaum AG. Frontal fibrosing alopecia in a man: results of follicular unit test grafting. Dermatol Surg 2010;36(06):959–962
6 Tziotzios C, Fenton DA, Stefanato CM, McGrath JA. Familial frontal fibrosing alopecia. J Am Acad Dermatol 2015;73(01):e37
7 Ladizinski B, Bazakas A, Selim MA, Olsen EA. Frontal fibrosing alopecia: a retrospective review of 19 patients seen at Duke University. J Am Acad Dermatol 2013;68(05):749–755
8 Samrao A, Chew AL, Price V. Frontal fibrosing alopecia: a clinical review of 36 patients. Br J Dermatol 2010;163(06):1296–1300
9 Ceballos C, Priego C, Méndez C, Hoffner MV, García-Hernández MJ, Camacho FM. Study of frontal hairline patterns in Spanish Caucasian women. Actas Dermosifiliogr 2013;104(04):311–315
10 Tziotzios C, Stefanato CM, Fenton DA, Simpson MA, McGrath JA. Frontal fibrosing alopecia: reflections and hypotheses on aetiology and pathogenesis. Exp Dermatol 2016;25(11):847–852
11 Chew AL, Bashir SJ, Wain EM, Fenton DA, Stefanato CM. Expanding the spectrum of frontal fibrosing alopecia: a unifying concept. J Am Acad Dermatol 2010;63(04):653–660
12 Miteva M, Tosti A. The follicular triad: a pathological clue to the diagnosis of early frontal fibrosing alopecia. Br J Dermatol 2012; 166(02):440–442
13 Mirmirani P, Willey A, Headington JT, Stenn K, McCalmont TH, Price VH. Primary cicatricial alopecia: histopathologic findings do not distinguish clinical variants. J Am Acad Dermatol 2005;52 (04):637–643
14 Stefanato CM. Histopathology of alopecia: a clinicopathological approach to diagnosis. Histopathology 2010;56(01):24–38
15 Rácz E, Gho C, Moorman PW, Noordhoek Hegt V, Neumann HA. Treatment of frontal fibrosing alopecia and lichen planopilaris: a systematic review. J Eur Acad Dermatol Venereol 2013;27(12): 1461–1470
16 Tziotzios C, Fenton DA, Stefanato CM, McGrath JA. Finasteride is of uncertain utility in treating frontal fibrosing alopecia. J Am Acad Dermatol 2016;74(04):e73–e74
17 Wollenberg A, Bieber T. Proactive therapy of atopic dermatitis–an emerging concept. Allergy 2009;64(02):276–278
18 Miteva M, Aber C, Torres F, Tosti A. Frontal fibrosing alopecia occurring on scalp vitiligo: report of four cases. Br J Dermatol 2011;165(02):445–447
19 Dlova NC, Jordaan HF, Skenjane A, Khoza N, Tosti A. Frontal fibrosing alopecia: a clinical review of 20 black patients from South Africa. Br J Dermatol 2013;169(04):939–941
20 Egan BM, Zhao Y, Axon RN. US trends in prevalence, awareness, treatment, and control of hypertension, 1988-2008. JAMA 2010; 303(20):2043–2050
21 Walsh JP, Bremner AP, Feddema P, Leedman PJ, Brown SJ, O'Leary P. Thyrotropin and thyroid antibodies as predictors of hypothyroidism:

a 13-year, longitudinal study of a community-based cohort using current immunoassay techniques. J Clin Endocrinol Metab 2010;95 (03):1095–1104
22 Kajantie E, Phillips DI, Osmond C, Barker DJ, Forsén T, Eriksson JG. Spontaneous hypothyroidism in adult women is predicted by small body size at birth and during childhood. J Clin Endocrinol Metab 2006;91(12):4953–4956
23 Vaisse V, Matard B, Assouly P, Jouannique C, Reygagne P. Postmenopausal frontal fibrosing alopecia: 20 cases [in French]. Ann Dermatol Venereol 2003;130(6-7):607–610
24 Tosti A, Piraccini BM, Iorizzo M, Misciali C. Frontal fibrosing alopecia in postmenopausal women. J Am Acad Dermatol 2005; 52(01):55–60
25 Moreno-Ramírez D, Camacho Martínez F. Frontal fibrosing alopecia: a survey in 16 patients. J Eur Acad Dermatol Venereol 2005; 19(06):700–705
26 MacDonald A, Clark C, Holmes S. Frontal fibrosing alopecia: a review of 60 cases. J Am Acad Dermatol 2012;67(05):955–961
27 Tan KT, Messenger AG. Frontal fibrosing alopecia: clinical presentations and prognosis. Br J Dermatol 2009;160(01):75–79
28 Georgala S, Katoulis AC, Befon A, Danopoulou I, Georgala C. Treatment of postmenopausal frontal fibrosing alopecia with oral dutasteride. J Am Acad Dermatol 2009;61(01):157–158
29 Rallis E, Gregoriou S, Christofidou E, Rigopoulos D. Frontal fibrosing alopecia: to treat or not to treat? J Cutan Med Surg 2010;14 (04):161–166