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Clinicopathological characteristics and treatment outcomes of fibrosing alopecia in a pattern distribution: A retrospective cohort study

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RJ, JP, BB have nothing to disclose.
RS is a member of the international advisory board (paid consultancy) and principal investigator of sponsored clinical trials for Pfizer and Eli Lilly. He is principal investigator of sponsored clinical trials for Jaansen, Sun Pharma, Arena, Demira, AstraZenica, Novartis, Merck, Sanofi, Abbvie, Galderma, Prinicipia, Reistone Pharma, Aclaris and Samson Clinical. None of the above is relevant to the submitted work.

ABSTRACT

Background: Fibrosing alopecia in a pattern distribution (FAPD) is a primary cicatricial alopecia considered a subtype of lichen planopilaris (LPP).

Objective: The clinical and histopathological features, and treatment response of 24 female patients with FAPD were evaluated.

Methods: Demographic, clinical, histopathological and treatment data of patients with FAPD were retrospectively collected.

Results: Twenty-four women were included (mean age 60.7 years). The mean Lichen Planopilaris Activity Index score was 1.50 and the median Sinclair grade was 3. Twelve patients had diffuse thinning of the centroparietal region, and four had frontal accentuation and eight had vertex accentuation of hair loss. Eight had loss of facial hair. Predominant trichoscopic features included hair shaft diameter variability

(100%), perifollicular erythema and/or scaling (95%) and loss of follicular ostia (95%). Histopathology revealed a scarring alopecia with interface changes in follicular epithelia targeting terminal and intermediate and, occasionally, vellus hairs. Treatment with hair growth-promoting, anti-inflammatory and anti-androgen agents arrested disease progression in 14 patients and resulted in hair regrowth in two patients. Six patients had progression of their hair loss in spite of treatment.

Limitations: Referral bias to a specialist hair clinic, retrospective design and small sample size.

Conclusions: We present clinicopathological features of FAPD which can aid in the diagnosis of this insidious scarring alopecia. Our findings suggest a more favourable outcome if treatment is initiated in early stages of the disease.

INTRODUCTION

Fibrosing alopecia in a pattern distribution (FAPD) is a rare form of cicatricial alopecia first reported in 2000 by Zinkernagel and Trüeb.[1] FAPD is currently considered by some as a subtype of lichen planopilaris (LPP) with clinical and histopathological features of both LPP and androgenetic alopecia (AGA).[1] LPP and its clinical variant, frontal fibrosing alopecia (FFA), are primary cicatricial alopecias of the lymphocyte-predominant subgroup. The underlying pathogenic process in LPP and FFA is collapse of the hair follicle (HF) bulge immune privilege, resulting in cytotoxic lymphocyte-mediated HF destruction and replacement with fibrous tissue.[2] Irreversible damage to epithelial stem cells of the HF is central to scarring alopecias such as LPP and FFA, and causes permanent hair loss.[3] In contrast, AGA is characterised by progressive miniaturisation, leading to vellus transformation of terminal HFs. The authors of the original paper acknowledged the difficulty in determining if FAPD represents AGA with a lichenoid tissue reaction or patterned LPP.

To date, the FAPD literature remains limited to case reports and small case series. There is insufficient data on effective therapy, with current treatments aimed at suppressing inflammation or promoting hair regrowth.[4] Our objective was to describe the clinical, trichoscopic and histopathological features, and treatment response of 26 patients with FAPD.

METHODS

Selection criteria

The electronic records of patients diagnosed with FAPD at a specialist hair clinic in Melbourne, Australia, between 2011 and 2020 were reviewed. To identify potential patients with FAPD who were misdiagnosed as LPP, we reviewed the records of patients who received concurrent diagnoses of AGA and LPP. Diagnosis was confirmed based on the clinical (slowly progressive, diffuse rarefaction of hair in a centroparietal distribution), trichoscopic (perifollicular erythema and/or scaling, loss of follicular ostia and hair shaft diameter variability) and histopathological (perifollicular lymphocytic infiltrate, concentric lamellar fibrosis and HF miniaturisation) features,[4] and verified by a dermatologist with expertise in hair and scalp disorders. Patients were excluded if clinical and trichoscopy images were not available.

Data collection

The clinical pattern of loss of scalp hair, eyebrows and eyelashes was evaluated based on clinical photographs. The five-point Sinclair scale was used to assess the severity of hair loss pre- and post-treatment. Although this classification was originally developed for female pattern hair loss (FPHL),[5] it allowed for an objective assessment of hair loss in our patients. Disease activity was measured using the Lichen Planopilaris Activity Index (LPPAI), a standardised quantitative scoring system originally developed to measure LPP activity.[6]

Trichoscopy was performed on the mid-frontal scalp and vertex using a hand-held dermoscope (10-fold-magnification) and the findings were recorded. In patients who had a (dermoscopy-guided) biopsy, examination of horizontally and vertically oriented haematoxylin and eosin-stained histologic sections from 4.0-mm punch biopsy specimens of the scalp were studied. Our dermatopathologist reported histological findings in a synoptic format (Supplementary Table 1).

RESULTS

Demographics

Twenty-four women were diagnosed with FAPD during the 9-year period. The mean age at diagnosis was 60.7 years (range 40-82). The baseline characteristics are summarised in Table 1. Eight patients had autoimmune diseases, of whom three had a past history of alopecia areata (AA) which was in complete remission at the time of diagnosis of FAPD. Other associated hair and scalp conditions included FFA (n=2), psoriasis (n=1) and traction alopecia affecting the post-auricular areas (n = 1). Fourteen patients had a family history of AGA. Laboratory investigations were available for all but four patients and abnormalities included low ferritin (n=3) and free testosterone (n=1) levels.

Clinical features

All patients had diffuse hair loss in the absence of discrete patches. Of the 24 women, 12 had uniform rarefaction of hair extending from the mid-frontal scalp to the vertex, and four had frontal accentuation and eight had vertex accentuation of hair loss (Figure 1). The median (interquartile range, IQR) hair loss severity was Sinclair grade 3 (3-5). Ten patients reported scalp symptoms, namely pruritus (n=8), pain (n=1) and burning (n=2). The mean LPPAI score was 1.50.

Eight patients had loss of facial hair, including eyebrows in seven patients, two of whom had known FFA and one had AA, and eyelashes in one patient who did not have associated FFA or AA. None of the patients had lichen planus (LP) affecting the skin, nails, oral mucosa or genital mucosa on examination.

Notably, two men with biopsy-proven FAPD were also identified. Of these, one had vertex hair loss whilst the other had frontal scalp hair thinning. Interestingly, both men had preservation of their anterior hairline. These patients were not included in the overall analysis.

Trichoscopy

Twenty-two patients had documented trichoscopy findings. The most common features were hair shaft diameter variability (n=22), vellus hairs (n=22), increased single-hair follicular units (n=19), perifollicular erythema (n=21), perifollicular scaling (n=21) and loss of follicular ostia (n=21) (Figure 2). Additional trichoscopic features

included perifollicular white fibrotic patches (n=12), reticular pigmentation (n=4), broken hairs (n=2) and black dots (n=1).

Histopathology

Histopathological evaluation of affected scalp in 20 patients showed a mild-to-moderate perifollicular lymphocytic infiltrate at the level of the infundibulum or isthmus. The inflammation surrounded terminal hairs in all cases, as well as intermediate and vellus hairs in 18 and six cases respectively (not recorded in one case). All cases were characterised by an interface dermatitis with vacuolar degeneration or apoptosis of basal follicular keratinocytes, perifollicular concentric lamellar fibrosis and reduced or absent sebaceous glands (Figure 3). A decrease in terminal hair density (mean terminal hair density 159/cm^2, normal: 175-300/cm^2)[7] with a reciprocal increase in vellus-like hairs (mean terminal-to-vellus ratio 1.8:1, normal ≥7:1)[8] and underlying fibrous streamers were observed. Four patients declined scalp biopsies.

Treatment response

Only one patient declined treatment. Treatments and outcomes for each patient are outlined in Table 2. Systemic treatments used included low-dose oral minoxidil (LDOM) (n=23), spironolactone (n=19), bicalutamide (n=5), flutamide (n=8), dutasteride (n=2), finasteride (n=9) and hydroxychloroquine (n=3). Topical treatments included high potency corticosteroids (n=17), tacrolimus 0.1% ointment (n=1) and minoxidil 5% lotion (n=2). Intralesional corticosteroid injections were administered in four cases.

An overall reduction in global hair density was observed after a mean treatment duration of 4.1 years [median (IQR) Sinclair grade 4 (2-5), representing a 1-point increase from baseline]. One patient was not evaluated for treatment response as duration of treatment was less than six months. Six patients had worsening hair loss, with a 1-point increase in their Sinclair grades, while 14 patients had stable disease with no discernible change in their hair density. Notably, a 1-point reduction in Sinclair grade (from 3 to 2) and improvement in symptoms were seen in two patients (Figure 4). Both patients were treated with a combination of LDOM, an anti-androgen (finasteride, bicalutamide or spironolactone) and clobetasol propionate lotion 0.05%.

One patient with severe hair loss demonstrated an improvement in global hair density with platelet-rich plasma (PRP) treatment (three treatments, one month apart), although this improvement was not sufficient to result in a reduction in her Sinclair grade (5).

Side effects reported with LDOM included facial hypertrichosis (n=13), postural hypotension (n=5), palpitations (n=3) and fluid retention (n=1), resulting in discontinuation in the latter patient. Adverse effects of spironolactone included hyponatraemia (n=1) and breast tenderness (n=1). One patient on hydroxychloroquine developed bull's eye maculopathy, which necessitated cessation of the medication. No side effects attributable to bicalutamide, flutamide, dutasteride or finasteride were reported.

DISCUSSION

FAPD is a variant of primary cicatricial alopecia which was first described six years after Steven Kossard's first report of FFA in six post-menopausal women in 1994.[9] As with FFA, this raises the question of whether FAPD did not exist or was simply not recognised prior to 2000. If it is the former, the rising incidence would invite speculation for environmental triggers. While the role of contributory factors such as sunscreens, leave-on cosmetic products, contact allergens, chemical exposures and foods in FFA is still the subject of controversy,[10–12] the aetiology of FAPD remains obscure.

Our study shows that FAPD predominantly affects Caucasian women (like FFA) and those with a family history of AGA, reflecting previous findings.[1,4,13–15] However, this may in part be due to our patient demographics, with Europeans representing 68.5% of the Greater Melbourne population. Hair loss in all our patients was diffuse with no discernible bald patches. We identified three distinct patterns of alopecia, all of which affected the androgen-dependent areas of the scalp, including uniform hair thinning from the frontal scalp to the crown, frontal accentuation (described as the "Christmas tree pattern" in FPHL) and vertex accentuation. Limited evidence suggests that the pattern of hair loss in men with FAPD mimics classical AGA with bitemporal recession and vertex balding.[1,16] Interestingly, one of the men identified but not

included in our analysis had vertex hair loss in the absence of temporal involvement whereas the other had diffuse alopecia in a distribution similar to FPHL with a "Christmas tree pattern". We do know that a subset of men with AGA have diffuse hair thinning with retention of the anterior hairline.[17] Our observations therefore suggest that FAPD causes diffuse cicatricial alopecia which affects the centroparietal scalp but does not necessarily follow the classical Ludwig pattern in women or Hamilton-Norwood pattern in men. In all cases, hair loss was slowly progressive and symptoms, if present, were often mild. This is reflected in the relatively low LPPAI scores of our patients (mean 1.50), which is significantly lower than the LPPAI scores reported in LPP and FFA,[6] highlighting the insidious nature of FAPD. Eyebrow involvement in FAPD is uncommon,[18] as opposed to FFA which is associated with eyebrow loss in up to 80% of cases.[11] Sparse eyebrows and eyelashes were reported in a minority of our patients in the absence of any systemic or other active hair disorder. Loss of eyelash, beard and other body hair has not previously been reported in FAPD.[4] It is not clear whether the loss seen in these patients was related to FAPD. Biopsies of affected non-scalp sites in future studies will be pertinent to elucidate the pathology. None of our patients had mucocutaneous LP. Unlike LPP or FFA, LP involving the skin or mucosal surfaces is not known to occur in patients with FAPD. Trichoscopically, perifollicular erythema and hyperkeratosis, and loss of follicular orifices, found in the majority of our patients, allows distinction of FAPD from AGA and cicatricial pattern hair loss.[19,20] Perifollicular white patches, similar to those described in central centrifugal cicatricial alopecia (CCCA)[21] and Afro-Hispanic patients with FAPD,[16] were seen in a few of our cases. The presence of broken hairs in a small number of our patients is not typical of FAPD but has been described in a diffuse variant of LPP.[22]

The histopathology of FAPD typically shows features of LPP and AGA in the same biopsy specimen, including interface changes involving follicular epithelia with a lymphocytic infiltrate surrounding the HF, concentric lamellar fibrosis, reduced sebaceous glands and HF miniaturisation.[1,4,16,22,23] A folliculocentric interface dermatitis was present in all but one of our cases, reaffirming the characterisation of FAPD as a variant of LPP. However, the inflammation seen in all our cases was mild to moderate, in contrast to the dense perifollicular lymphocytic infiltrate typically seen in LPP or FFA.[24,25] This perhaps explains the "low-grade" nature of FAPD. The

patient whose biopsy lacked an interface dermatitis was on tofacitinib for concomitant AA. Tofacitinib has shown promising results in patients with LPP,[26,27] but whether it may have a role in the treatment of FAPD is yet to be determined. Acutely affected vellus hairs, which may be difficult to identify in later stages of FAPD,[4,16] were noted in 32% of our cases. AGA-like features, including HF miniaturisation and underlying fibrous stellae, were present in all cases. It has been suggested that CCCA may represent FAPD in women of African descent.[28] Indeed, premature desquamation of the inner root sheath, considered a defining histopathological feature of CCCA,[21,29,30] was found in 68.8% of biopsy specimens in a series of 16 dark-skinned patients with FAPD.[16] The lack of premature desquamation of the inner root sheath in our cohort, which consisted mainly of Caucasians, as well as a ubiquitous perifollicular interface dermatitis (absent in CCCA), suggests that FAPD is distinct from CCCA, at least in fair-skinned patients. Granulomatous inflammation associated with retained hair shaft fragments, a less specific feature of CCCA,[4,29,31]was present in six patients with late-stage disease in our study.

Starace *et al.* recently described a diffuse form of LPP, which they termed “LPP Diffuse Pattern”.[22] Although diffuse LPP and FAPD are both characterised by diffuse scarring hair loss, there are some key distinguishing features. Classical LPP is often symptomatic, with all patients with diffuse LPP in Starace *et al.*’s study reporting scalp dysaesthesia. On the other hand, 58.3% of our patients with FAPD were asymptomatic. The hair loss in FAPD is confined to the androgen-dependent region whereas diffuse LPP can affect any area including the occipital scalp. Trichoscopically, hair shaft diameter variability is unique to FAPD in the lymphocyte-predominant primary cicatricial alopecia subgroup.[32] Histologically, FAPD can be distinguished from diffuse LPP by the presence of HF miniaturisation and a relatively discrete inflammatory infiltrate.

Turegano and Sperling proposed the umbrella term “lichenoid folliculitis” for the constellation of skin conditions characterised by keratotic papules, atrophy, cicatricial alopecia and lichenoid inflammation.[33] The lichenoid folliculitis family contains (1) the keratosis pilaris atrophicans subgroup, and (2) the LPP subgroup, including LPP

itself, FFA, Graham-Little-Piccardi-Lassueur syndrome and FAPD. Later, Du, *et al.* proposed the unifying concept "fibrosing alopecia" to refer to the LPP subgroup of diseases, which share the histological features of lichenoid inflammation and fibrosis involving the distal HF.[34] Their rationale for lumping rather than splitting is that the aetiology and pathogenesis of these individual cicatricial entities have not been defined. Whilst we agree that there may be little utility in separate classifications at this stage, greater awareness of FAPD, either as a clinical entity in its own right or a variant of lichenoid folliculitis/fibrosing alopecia, will enhance distinction from its non-scarring mimics, AGA or AGA and concomitant seborrheic dermatitis.[4,16,32]

The natural history of FAPD has not been investigated; in particular, it is not known whether it eventually 'burns out'. Sparse preliminary evidence suggests that topical and intralesional corticosteroids, 5α-reductase inhibitors and hydroxychloroquine may arrest the progression of hair loss in FAPD.[1,14,22,35] However, to our knowledge, no therapy has been shown to induce hair regrowth. The treatments used in our study included hair growth-promoting, anti-inflammatory and anti-androgen agents. LDOM and bicalutamide have recently emerged as an effective and safe treatment for AGA,[36–40] but our study represents the first report of their use in FAPD. Fourteen patients had no progression of their hair loss after commencing treatment. It needs to be acknowledged that many of these patients had advanced hair loss (Sinclair grades 4 and 5) at diagnosis. It is possible that, in these cases, treatment arrested disease progression but, unsurprisingly, did not stimulate hair regrowth. Interestingly, an increase in hair density was observed in two patients with clinically early-stage disease (Sinclair grade 3), as demonstrated by a 1-point reduction in their Sinclair grades. Therefore, as with other primary cicatricial alopecias, early treatment is probably associated with a better outcome. One major benefit of our study is the use of validated scoring systems to measure the baseline severity (Sinclair grade and LPPAI) and response to treatment (Sinclair grade). However, given the retrospective design and use of concurrent therapies, we acknowledge it is difficult to determine the direct clinical response to individual agents. Based on our experience, we propose that FAPD is best treated with a combination of anti-inflammatory and hair growth-promoting agents. Topical or intralesional corticosteroids should be considered first-line, with systemic immunomodulatory therapies reserved for

refractory cases. As HF miniaturisation is a prominent feature, we suggest the addition of topical or low-dose oral minoxidil and anti-androgen therapy such as finasteride. Patients with late-stage, irreversible alopecia should be offered camouflage techniques e.g. wigs.

Limitations of our study include the small sample size, retrospective design, referral bias to a specialist hair clinic and lack of a control group to assess the impact of treatment on the natural history of FAPD. Finally, a few of our patients had been labelled as having AGA by other healthcare professionals before they were eventually diagnosed with FAPD. That patients with FAPD are more often than not diagnosed late or misdiagnosed altogether underscores its insidious nature.

To the best of our knowledge, this series reports the largest collection of FAPD cases to date. We present clinical and histopathological observations which will help to better characterise this subtle scarring alopecia. The clinical, trichoscopic and histopathological features of FAPD are such that its inclusion in the lymphocyte-predominant subgroup of primary cicatricial alopecias may be justified. Our findings suggest that prompt diagnosis and intervention in patients with early-stage FAPD can result in a more favourable outcome. Controlled studies are required to evaluate the effectiveness of treatment modalities for FAPD.

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FIGURE LEGENDS

Figure 1: Patterns of scalp hair loss in patients with biopsy-proven fibrosing alopecia in a pattern distribution (a) uniform rarefaction of hair extending from the mid-frontal scalp to the vertex; (b) frontal accentuation, and (c) vertex accentuation.

Figure 2: Dermoscopic examination of scalp of patients with fibrosing alopecia in a pattern distribution showing hair shaft diameter variability, vellus hairs, increased single-hair follicular units, perifollicular erythema and scaling, and loss of follicular ostia.

Figure 3: Histopathological examination of horizontally oriented scalp biopsies from patients with fibrosing alopecia in a pattern distribution showing (a) hair follicles of varying sizes with interface changes in the follicular epithelium, perifollicular fibrosis and almost complete loss of sebaceous glands (haematoxylin and eosin, original magnification: x40), and (b) perifollicular fibrosis with prominent vacuolar changes in the follicular epithelium and occasional apoptotic keratinocytes. Bland appearing lymphocytes are also present in the follicular epithelium in this example (haematoxylin and eosin, original magnification: x400).

Figure 4: (a) 52-year-old woman with fibrosing alopecia in a pattern distribution, Sinclair grade 3 at baseline; (b) improvement in hair density to Sinclair grade 2 after one year of treatment with low dose oral minoxidil, spironolactone, finasteride and topical clobetasol propionate 0.05%.

Table 1. Demographics and clinical features of 24 patients with fibrosing alopecia in a pattern distribution.

Demographics		
Mean age at diagnosis (median, range), years		60.7 (61.5, 40-82)
Mean age of onset of hair loss (median, range), years		50.9 (51, 23-80)
Mean interval period between onset of hair loss and diagnosis of FAPD (median, range), years		9.8 (9, 1-44)
Post-menopausal		18
Ethnicity		
	European	21
	South Asian	1
	Middle Eastern	1
	Mixed	1
Family history of androgenetic alopecia		14
Autoimmune disease		8
	Pernicious anaemia	1
	Alopecia areata	3*
	Rheumatoid arthritis	2
	Coeliac disease	2
Concomitant scalp condition		8
	Frontal fibrosing alopecia	2
	Scalp psoriasis	1
	Traction alopecia	1
Pattern of scalp hair loss		
	Diffuse centroparietal	12

	Frontal accentuation	4
	Vertex accentuation	8
Median Sinclair grade at presentation (range)		3 (2-5)
Mean LPPAI score at presentation (range) *n=22*		1.50 (0.33-3.17)
Other body hair loss		8
	Eyebrows	7
	Eyelashes	1

*All 4 patients were in complete remission from their alopecia areata.

Abbreviations: LPPAI, Lichen Planopilaris Activity Index

Table 2: Treatment outcomes in 24 patients with fibrosing alopecia in a pattern distribution.

Patient/ Sex/Age at diagnosis	Diagnosis prior to diagnosis of FAPD	Treatment prior to diagnosis of FAPD	Sinclair grade pre-treatment	Treatment following diagnosis of FAPD^	Total treatment duration (years)	Sinclair grade post-treatment	Change in global hair density	Side effects of current treatment
1/F/40	FPHL	Flutamide, finasteride, LDOM, spironolactone TCS	3	Flutamide, LDOM, spironolactone, TCS	2	3	Stabilisation	Palpitations, postural hypotension
2/F/82	FFA, FPHL	Bicalutamide, dutasteride, HCQ, LDOM, spironolactone, TCS,	4	Dutasteride, HCQ, LDOM, spironolactone, TCS	1.3	4	Stabilisation	Postural hypotension
3/F/56	FPHL	Bicalutamide, dutasteride, LDOM, spironolactone, TCS	3	Dutasteride, LDOM, spironolactone, TCS	1.2	4	Worsening	Postural hypotension
4/F/57	FPHL, LPP	Flutamide, HCQ, ICS, LDOM, minocycline, spironolactone, TCS	5	Flutamide, HCQ, ICS, LDOM, PRP, spironolactone, TCS, hair transplant	7.5	5	Stabilisation	Facial hypertrichosis
5/F/56	FPHL	None	5	Finasteride, LDOM, spironolactone, TCS	0.1	Too early to evaluate	N/A	None
6/F/41	FPHL, LPP	Finasteride, flutamide ICS, LDOM, spironolactone	2	Finasteride, LDOM	8	2	Stabilisation	Facial hypertrichosis
7/F/41	FPHL	Finasteride, LDOM, spironolactone TCS	2	Finasteride, LDOM, spironolactone, TCS	8	2	Stabilisation	Facial hypertrichosis

8/F/63	FPHL	LDOM, prednisolone, spironolactone, TCS	3	LDOM, spironolactone, TCS	1	4	Worsening	Hyponatremia
9/F/59	FPHL	Finasteride, LDOM, spironolactone, topical minoxidil	3	Finasteride, LDOM, spironolactone, topical minoxidil	1	4	Worsening	Breast tenderness, facial hypertrichosis, fluid retention
10/F/40	FPHL	Bicalutamide, finasteride, LDOM, spironolactone, TCS	5	Finasteride, LDOM, TCS	2.2	5	Stabilisation	None
11/F/51	FFA, FPHL	Bicalutamide, finasteride, LDOM, spironolactone, TCS, tolbutamide	3	Finasteride, LDOM, spironolactone, TCS	4	2	Improvement	Facial hypertrichosis
12/F/68	FPHL	Finasteride, flutamide, LDOM, spironolactone	5	Finasteride, LDOM, spironolactone	16	5	Stabilisation	Facial hypertrichosis
13/F/60	FPHL	Bicalutamide, finasteride, flutamide, LDOM, spironolactone, TCS	3	Finasteride, LDOM, spironolactone, TCS	2.2	4	Worsening	Facial hypertrichosis, palpitations, postural hypotension
14/F/58	FPHL, LPP	Flutamide, ICS, LDOM, spironolactone, minocycline	3	Flutamide, ICS, LDOM, spironolactone	1.3	4	Worsening	Facial hypertrichosis
15/F/66	FPHL, LPP	Flutamide, spironolactone, LDOM, TCS	5	LDOM, TCS	9	5	Stabilisation	Facial hypertrichosis
16/F/70	FPHL, LPP	HCQ, ICS, LDOM, spironolactone, TCS	5	HCQ, ICS, LDOM, spironolactone, TCS	6	5	Stabilisation	Bull's eye maculopathy, facial

								hypertrichosis
17/F/64	FPHL	Bimatoprost 0.03% solution, topical minoxidil, TCS	2	Bimatoprost 0.03% solution, topical minoxidil, TCS	0.9	2	Stabilisation	None
18/F/81	FFA, LPP	Dutasteride, LDOM, tacrolimus 0.1% ointment	4	Dutasteride, LDOM, tacrolimus 0.1% ointment	1.2	4	Stabilisation	None
19/F/78	FFA, FPHL, LPP	LDOM, TCS	3	LDOM, TCS	9	4	Worsening	Facial hypertrichosis, palpitations, postural hypotension
20/F/67	FPHL	Flutamide, LDOM, spironolactone	5	Flutamide, LDOM, spironolactone	3.6	5	Stabilisation	Facial hypertrichosis
21/F/42	FPHL	LDOM, spironolactone, TCS	2	LDOM, spironolactone, TCS	2.5	2	Stabilisation	Facial hypertrichosis
22/F/71	FPHL	None	5	None	N/A	No follow-up	N/A	N/A
23/F/77	FFA, FPHL	Bicalutamide, finasteride, ICS, LDOM, TCS	3	Finasteride, LDOM, TCS	1.3	2	Improvement	None
24/F/69	FFA, FPHL	Bicalutamide, bimatoprost 0.03% solution, finasteride, LDOM, TCS	4	Bicalutamide, LDOM, spironolactone, TCS	6	4	Stabilisation	Facial hypertrichosis
Median		**-**	**3**	**-**	**4.1***	**4**		**-**

*Refers to mean duration (not median).

^ **Dosage ranges**: Bicalutamide: 10-20 mg/day; dutasteride: 0.5 mg/day; finasteride: 1-2.5 mg/day; flutamide: 50-200 mg/day; HCQ: 200-400 mg/day; LDOM: 0.25-12.5 mg/day; minocycline: 50 mg/day; spironolactone: 50-200 mg/day.

Abbreviations: FFA, frontal fibrosing alopecia; FPHL, female pattern hair loss; HCQ, hydroxychloroquine; LDOM, low dose oral minoxidil; LPP, lichen planopilaris; PRP, Platelet-rich-plasma; TCS, topical corticosteroids; F, female; M, male; ICS, intralesional corticosteroids

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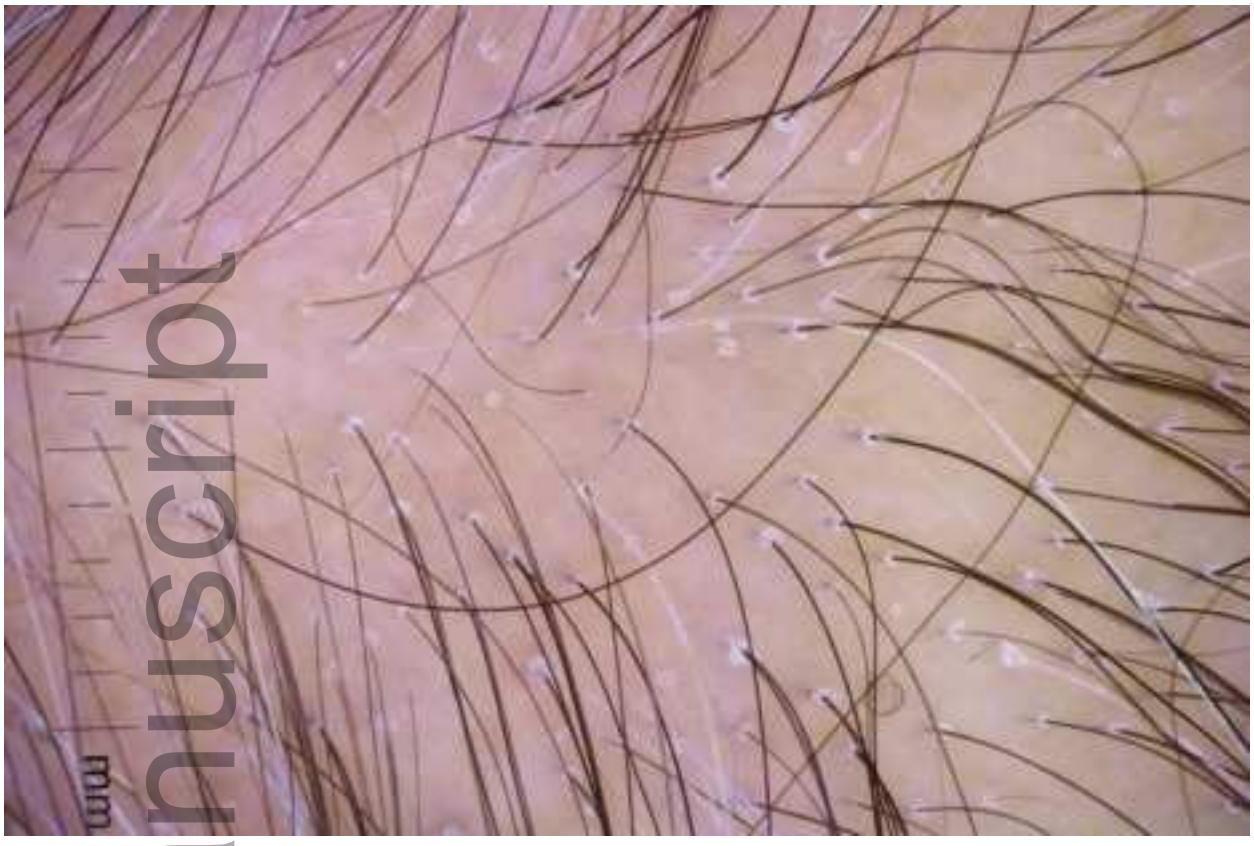

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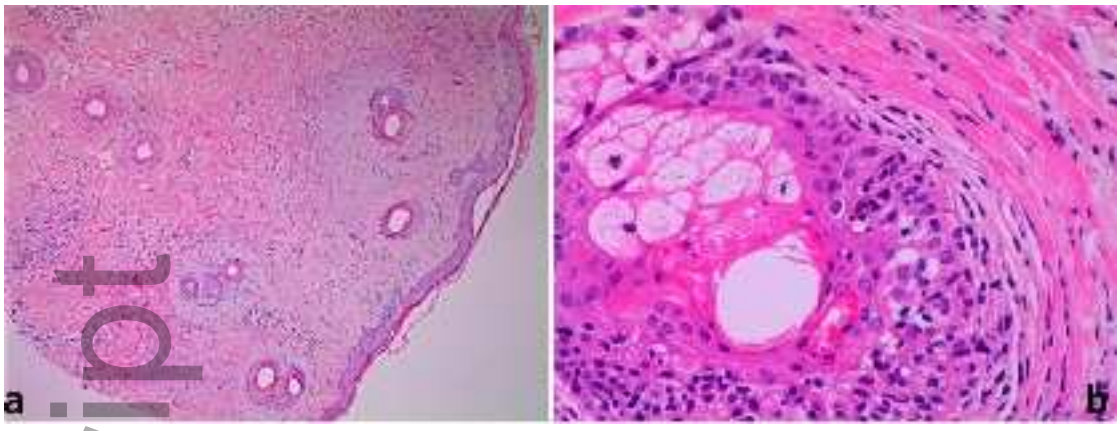

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