Diffuse variants of scalp lichen planopilaris: Clinical, trichoscopic, and histopathologic features of 40 patients

Michela Starace, MD,[a] Gloria Orlando, MD,[b] Aurora Alessandrini, MD,[a] Carlotta Baraldi, MD,[a] Francesca Bruni, MD,[a] and Bianca Maria Piraccini, MD, PhD[a]
Bologna and Padova, Italy

Background: Fibrosing alopecia in a pattern distribution and cicatricial pattern hair loss are poorly recognized diffuse variants of lichen planopilaris (LPP).

Objectives: The medical features of 40 patients affected by a diffuse hair thinning associated with a long-lasting history of pruritus and erythema of the scalp and a histopathologic diagnosis of LPP were reviewed.

Methods: Clinical data, results of trichoscopy and histopathology, response to treatment, and follow-up were analyzed.

Results: There were 18 patients diagnosed with fibrosing alopecia in pattern distribution and 2 patients with cicatricial pattern hair loss. A new variant of diffuse LPP, named "lichen planopilaris diffuse pattern," was described in 20 individuals.

Limitations: Low number of cases due to rarity of the diseases.

Conclusion: In patients complaining of a long-lasting history of scalp erythema, itching/dysesthesia, and diffuse hair thinning, it is advisable to consider diffuse variants of LPP. (J Am Acad Dermatol 2020;83:1659-67.)

Key words: cicatricial alopecia; fibrosing alopecia; fibrosing alopecia in pattern distribution; histopathology; lichen planopilaris; scalp itching; trichoscopy.

Lichen planopilaris (LPP) is a primary lymphocytic cicatricial alopecia defined as a follicular form of lichen planus.[1] The etiology is unknown, even if it is commonly assumed to have a hair-specific autoimmune pathogenesis.[2]

On the basis of the clinical distribution of the lesions, several variants have been described. The classic form presents with patches of scarring alopecia that can occur anywhere on the scalp, especially at the vertex, with peripheral perifollicular erythema and hyperkeratosis, associated with itching.[3] The Graham-Little-Piccardi-Lassueur syndrome is characterized by the triad of cicatricial alopecia of the scalp, noncicatricial alopecia in the armpit and pubis, and lichenoid papules on the trunk and extremities.[4] Frontal fibrosing alopecia (FFA) is characterized by a progressive recession of the frontotemporal hairline associated with loss of eyebrows, eyelashes, and peripheral body hair, affecting mainly postmenopausal women.[5-7]

Moreover, 2 other forms of scarring alopecia have been reported with histologic features of LPP but clinically presenting without well-defined alopecic patches and with an androgenetic pattern of hair loss.[8] Fibrosing alopecia in pattern distribution (FAPD), described in 2000 by Zinkernagel and

From the Division of Dermatology, Department of Experimental, Diagnostic and Specialty Medicine, University of Bologna[a]; and the Unit of Dermatology, Department of Medicine-DIMED, University of Padova.[b]
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Correspondence to: Gloria Orlando, MD, Unit of Dermatology, Department of Medicine-DIMED, University of Padova, Via Gallucci 4, 35121 Padova, Italy. E-mail: gloriaorlando@gmail.com.
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Truëb,[8] presents as central scarring hair loss with perifollicular erythema and follicular hyperkeratosis along with histologic features of LPP and androgenetic alopecia. Cicatricial pattern hair loss (CPHL), described by Olsen in 2005,[9] is characterized by histologic features similar to FAPD and clinical features of female pattern hair loss with the presence of focal atrichia, described as "pencil eraser–sized" areas of patchy scarring, but lacking the clinical signs of follicular erythema and hyperkeratosis seen in FAPD.

Our study describes the epidemiologic, clinical, trichoscopic features, treatment outcome, and long-term follow-up of 40 patients affected by diffuse hair thinning associated with a long-lasting history of itching and erythema of the scalp and a histopathologic diagnosis of LPP.

CAPSULE SUMMARY

- Fibrosing alopecia in a pattern distribution and cicatricial pattern hair loss are recently described forms of lichen planopilaris, characterized by pattern hair loss and destruction of miniaturized/intermediate hair follicles. A new variant of lichen planopilaris is described with small alopecic areas appearing diffusely throughout the scalp due to destruction of terminal follicles.
- In patients with diffuse hair thinning and a long-lasting history of scalp erythema, itching, or dysesthesia, it is strongly recommended to perform a trichoscopy-guided biopsy.

MATERIALS AND METHODS

This retrospective study included 40 patients with histologic diagnosis of scalp LPP associated with diffuse hair loss referred to the Outpatient Consultation for Hair Disease of the Dermatology Unit of the Department of Experimental, Diagnostic and Specialty Medicine of the University of Bologna from April 2015 to April 2018.

All patients signed informed consent for the use of the clinical documentation and photographs for scientific purposes. Patients were excluded if they were diagnosed with scalp diseases other than LPP or if complete clinical data were not available. The medical data collected included age, areas of scalp involvement, subjective symptoms, clinical and trichoscopic signs, histopathologic features, and prescribed treatments and their efficacy.

RESULTS

FAPD was diagnosed in 18 patients and CPHL in 2 patients. In the other 20 patients, we identified a new clinical variant called "lichen planopilaris diffuse pattern" (LPPDP).

Clinical features

We analyzed data for 40 white patients, 15 men and 25 women, with a mean age of 54.9 years (range, 34-76 years), complaining of a long-lasting history of itching/burning sensation on the scalp associated with diffuse hair thinning. Patches of scarring alopecia were absent.

FAPD was diagnosed in 18 patients with a mean age of 54.9 years (range, 38-75 years): 12 women (mean age, 58.9 years) and 6 men (mean age, 47 years). They experienced an accelerated hair loss in a female or male pattern distribution. All patients complained of dysesthesia of the scalp (pruritus or pain) correlated with clinically evident erythema (Fig 1, *A*). Four women also had cicatricial recession of the frontal hairline, consistent with a diagnosis of FFA.

Two women (aged 70 and 76 years) were diagnosed with CPHL because they clinically presented pencil eraser–sized areas of focal atrichia, without scalp erythema, in a female pattern distribution. They both reported a previous history of scalp itching, although they were asymptomatic at the moment of the evaluation.

The diagnosis of LPPDP was made in 20 patients, with a mean age of 53 years (range, 34-71 years). Of these, 11 patients (55%) were women (mean age, 56 years), and 9 were men (mean age, 49.3 years). All patients had a diffuse scalp itching. Clinical examination showed widespread scalp hair thinning in 11 patients (55%) and evident scalp erythema in 13 (65%) (Fig 2, *A*). Clinical features are presented in Tables I and II.

Pull test was positive in 15 patients (83.3%) with FAPD and in 19 (95%) with LPPDP, with anagen roots with thick sheaths, indicating active disease. The test was negative in the CPHL group.

Trichoscopy

In FAPD and CPHL groups (Table I), trichoscopic features were correlated to subjective symptoms. All patients with FAPD complaining of scalp itching or pain showed trichoscopic inflammatory signs, such as perifollicular erythema and follicular hyperkeratosis (Fig 1, *B*). By contrast, these inflammatory signs were absent in the 2 patients with asymptomatic disease and diagnosed with CPHL. All patients with FAPD and CPHL showed loss of follicular ostia and white fibrotic patches

Abbreviations used:

CPHL: cicatricial pattern hair loss
FAPD: Fibrosing alopecia in pattern distribution
LPP: lichen planopilaris
LPPD: lichen planopilaris diffuse pattern

(Fig 1, *B*) limited to the area of androgenetic hair loss. Tufted or broken hair was not found.

In all patients affected by LPPDP (Table II), trichoscopy showed perifollicular erythema, follicle hair loss, and white patches widely diffused all over the scalp (Fig 2, *B*). Moreover perifollicular hyperkeratosis was seen in 14 patients (70%) and tufted hair in 9 (45%) (Fig 2, *B*). Four patients showed broken hair.

Pathology

All patients underwent a trichoscopy-guided scalp biopsy, in which the specimen was collected in the most active part of the affected area. All patients showed histologic features consistent with LPP.

In the patients with FAPD and CPHL (Table I), histopathology showed a reduced number of hair follicles with decreased or absent sebaceous glands. The most striking histopathologic finding was the presence of a mild lichenoid infiltrate around the isthmus and infundibular region and perifollicular lamellar fibrosis that mainly affected miniaturized follicles (Fig 1, *C* and *D*). In 6 patients (33.3%) with FAPD, terminal hair follicles were completely spared by the inflammatory infiltrate. The presence of fibrotic collagen tracts and streamers was prevalent in patients with a longer history of the disease, especially in the CPHL group, whereas a lymphocytic interface dermatitis with destruction of basal keratinocytes was evident in patients with FAPD and acute symptoms.

All scalp biopsy specimens taken from the 20 patients affected by LPPDP showed similar pathologic changes (Table II). As in FAPD, we observed the presence of lymphohistiocytic infiltrate around the isthmus and infundibular region with a reduction in the number of hair follicles, concentric perifollicular lamellar fibrosis, sebaceous gland reduction, and lymphocytic interface dermatitis (Fig 2, *C* and *D*). The most relevant difference between the 2 diseases was that in LPPDP, the inflammatory infiltrate spared the miniaturized follicles and involved terminal (100% of patients) and intermediate follicles (65% of patients), but with a milder intensity (Fig 2, *D*). Perifollicular lamellar fibrosis was evident around the same follicles and was a prevalent feature in 7 patients. The reduction of hair follicles correlated with the severity of perifollicular lamellar fibrosis and was less pronounced than in FAPD and CPHL.

Treatment and outcome

Among the treatments administrated to the FAPD group (Table I), 9 patients were prescribed with finasteride (2.5 mg/d) or dutasteride (0.5 mg/d), and 4 patients were prescribed hydroxychloroquine (400 mg/d). Five patients with subjective symptoms or evident inflammatory signs, or both, were also treated with a short cycle of systemic steroids (intramuscular triamcinolone acetonide, 3-4 injections of 40 mg every 4 weeks). Topical therapy was always associated and included local application of clobetasol propionate 0.05% cream, tacrolimus 0.1% ointment, or pimecrolimus cream. All patients also applied daily 2% or 5% topical minoxidil solution. The 2 patients with CPHL were treated with finasteride (2.5 mg/d) associated with topical tacrolimus 0.1% ointment and 5% minoxidil solution.

Topical calcineurin inhibitors and minoxidil solution were used as maintenance therapy in both groups.

In the LPPDP group (Table II), the therapeutic approach differed from FAPD and CPHL in the administration of a longer cycle of systemic intramuscular triamcinolone acetonide (up to 6-8 months) in 14 patients and hydroxychloroquine (400 mg/d) in 8 patients. As for FAPD, topical therapy with clobetasol propionate 0.05% cream was recommended in 15 patients (75%), and all patients received also 2% or 5% topical minoxidil solution. Minoxidil solution was used as maintenance therapy.

Concomitant therapy with topical and systemic agents halted the progression of FAPD, CPHL, and LPPDP in 95% of the patients after 1 year.

DISCUSSION

LPP is an inflammatory scalp disorder that is considered the most common cause of scarring alopecia.[2] The North American Hair Research Society[10] divides it into 3 clinical variants, mainly distinguished by the clinical pattern of hair loss: classical LPP, FFA, and Graham-Little-Piccardi-Lassueur syndrome.[11] Less common subtypes include FAPD and CPHL. FAPD is a progressive scarring alopecia histopathologically indistinguishable from LPP but limited to the area of androgen-sensitive hair follicles, showing perifollicular erythema, follicular keratosis and loss of follicular orifices in the central scalp.[8] CPHL is a variety of lymphocytic cicatricial alopecia in a female pattern hair loss without clinically evident inflammatory signs but characterized by small pencil

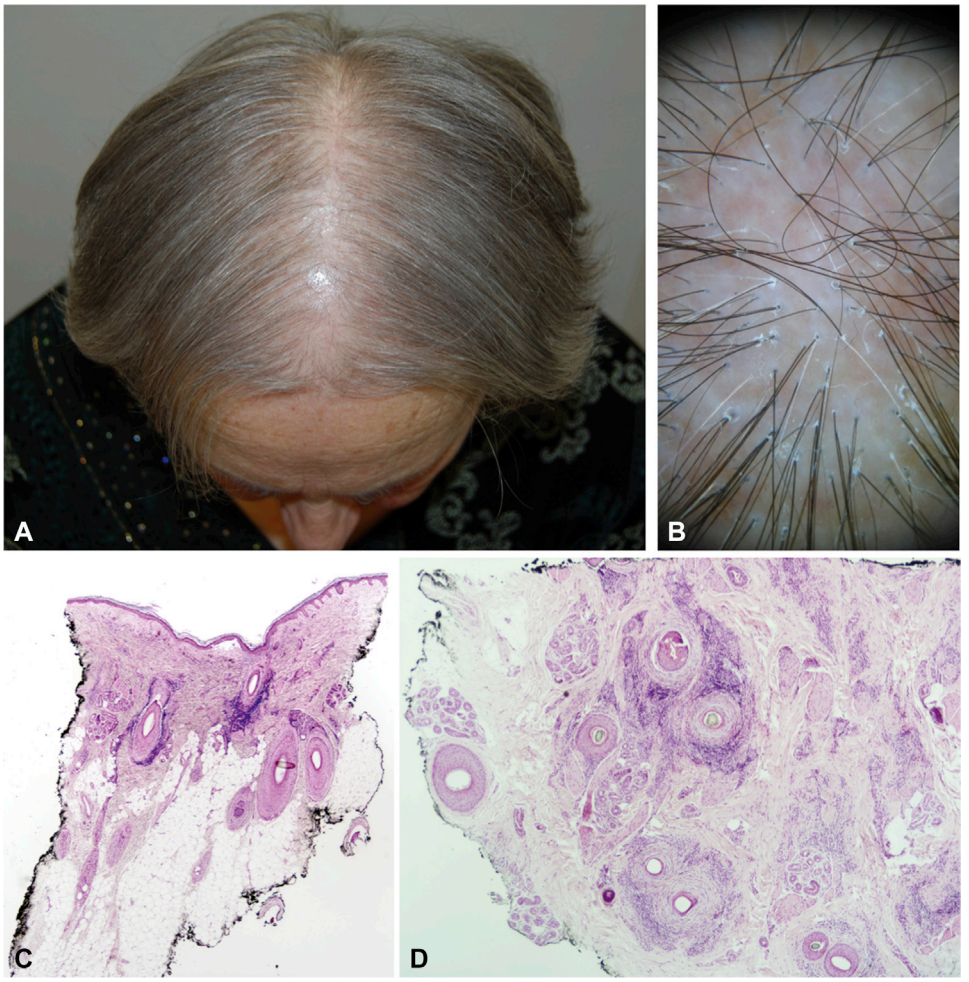

Fig 1. **A**, Mild erythema of the scalp and clinically evident hair thinning in the crown area in a patient with fibrosing alopecia in pattern distribution. **B**, Trichoscopy shows miniaturized follicles with mild perifollicular erythema and hyperkeratosis. White fibrotic areas and loss of follicular ostia are also present. **C**, Longitudinal section of the scalp biopsy specimen shows loss of sebaceous glands, fibrotic collagen tracts, dermal melanophages, and mild lymphocytic infiltrate around the isthmus of miniaturized hair follicles. Terminal hair follicles are spared. **D**, Transverse section of the scalp biopsy specimen shows loss of sebaceous glands, perifollicular lymphocytic infiltrate, and lamellar fibrosis involving mainly the miniaturized hair follicles and fibrotic collagen tracts (**C** and **D**, hematoxylin-eosin stain; original magnifications: ×4).

eraser–sized areas of focal atrichia.[9] LPP can therefore present itself not only as a localized disease with defined patches of scarring alopecia but also with a diffuse involvement of the scalp.

The 40 white patients analyzed in our study complained of a long-lasting history of itching/burning sensations on the scalp associated with diffuse hair thinning. Clinically and trichoscopically, they all showed widespread lichenoid alterations in absence of defined scarring alopecia patches. All patients underwent a trichoscopy-guided scalp biopsy, and the specimens showed histopathologic features coherent with a perifollicular lichenoid reaction. A careful analysis of clinical, dermoscopic, and histologic data allowed us to identify 3 different variants of diffuse LPP (Table III).

There were 18 patients diagnosed with FAPD and 2 patients with CPHL. Furthermore, we describe a new variant of diffuse LPP, named LPPDP, which, to our knowledge, has not been previously reported.

Our series of 18 patients with FAPD confirmed that the condition mostly targets postmenopausal women (mean age, 58.9 years), although it can also affect men, who are generally younger (mean age, 47 years). Clinically, patients showed a hair thinning on the central scalp associated with itching.

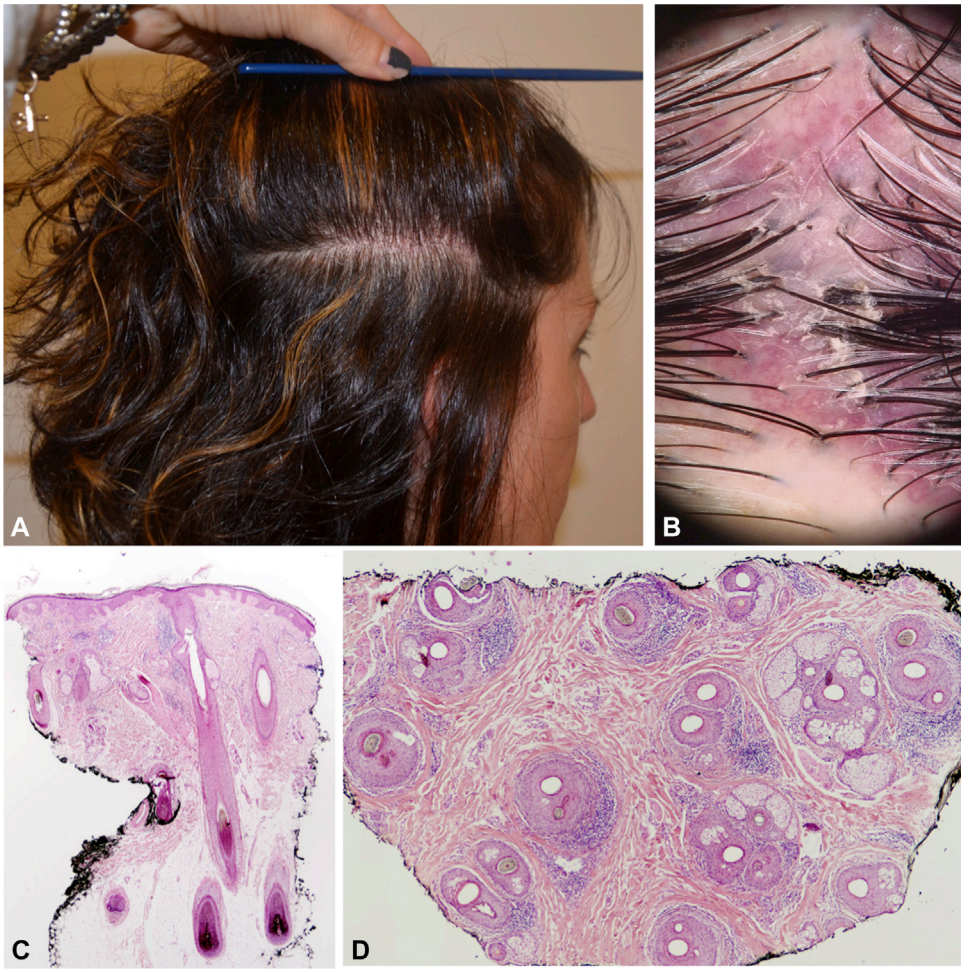

Fig 2. A, Clinically evident erythema of the scalp in a patient diagnosed with lichen planopilaris diffuse pattern, complaining of a 2-year history of diffuse hair thinning and severe scalp itching. **B**, Trichoscopy shows perifollicular erythema and hyperkeratosis associated with white fibrotic areas and tufted hairs. **C**, Longitudinal section of the scalp biopsy specimen shows lymphocytic infiltrate around the isthmus of a terminal hair follicle and mild concentric perifollicular lamellar fibrosis. **D**, Transverse section of the scalp biopsy specimen shows reduction of sebaceous glands, mild lymphocytic infiltrate around the isthmus and infundibular region of the terminal hair follicles, and concentric perifollicular lamellar fibrosis. Some tufted hairs and fibrotic collagen tracts are seen (**C** and **D**, hematoxylin and eosin stain; original magnifications: ×4).

Trichoscopy detected small fibrotic areas and perifollicular inflammatory signs.

CPHL was found in elderly people (mean age, 73 years), who experienced a long-lasting progressive hair thinning and a decreasing itching over time. There was no clinical evidence of scalp inflammation, but only small areas of focal atrichia classified as pencil eraser–sized zones.[9] Histopathology showed a pronounced perifollicular lamellar fibrosis, typical of longstanding disease,[8] suggesting that CPHL might represent a postinflammatory variant of FAPD.

The 20 cases identified as a new variant of LPP, named LPPDP, showed distinctive features compared with the 2 latter forms. These patients, aged from 34 to 71 years with no sex predilection (9 men, 11 women), had a long-lasting history of scalp itching or pain associated with erythema and mild hair thinning, often misdiagnosed as seborrheic dermatitis. They clinically presented with mild hair thinning widespread all over the scalp, without a specific female or male pattern distribution. Trichoscopy revealed scarring alopecia, showing very small areas of follicular ostia loss and fibrotic tracts spread all over the scalp, associated with follicular erythema and perifollicular hyperkeratosis.

In FAPD and CPHL, histopathology revealed a typical lichenoid perifollicular lymphocytic infiltrate

Table I. Clinical data of patients with fibrosing alopecia in pattern distribution and cicatricial pattern hair loss (*CPHL*)

Demography			Symptoms		Signs		Trichoscopy						Pathology				Treatments			
Patient	Age, y	Sex	Itch	Pain	Thinning	Erythema	Perifollicular erythema	Perifollicular hyperkeratosis	Loss of follicular ostia	Hair tufted	White patches	Broken hair	Reduction sebaceous glands	Lichenoid infiltrate	Perifollicular fibrosis	Tufted hairs	Systemic	Topical	Outcome	Duration
1	65	F PM	+	+	++	+	+	+	++	–	++	–	++	+	+	–	Finasteride	Clobetasol minoxidil	Arrested	9 mo
2	50	F PM	++	–	++	+	+	++	++	–	++	–	+	++*	+	–	Finasteride	Tacrolimus minoxidil	Arrested	12 mo
3	38	M	++	+	++	+	+	++	++	–	++	–	+	++	+	–	Triamcinolone	Clobetasol minoxidil	Arrested	6 mo
4 CPHL	76	F PM	–	–	++	–	–	–	++	–	+	–	++	+	++	–	Finasteride	Tacrolimus minoxidil	Arrested	6 mo
5	53	F PM	+	+	+	+	+	+	+	–	+	–	++	+	+	+	Dutasteride	Pimecrolimus minoxidil	Arrested	12 mo
6†	72	F PM	+	–	++	+	+	+	++	–	++	–	+++	+*	++	–	Finasteride	Tacrolimus minoxidil	Arrested	12 mo
7	55	M	++	+	++	++	++	++	+	–	++	–	++	+*	+	–	Triamcinolone	Clobetasol minoxidil	Arrested	6 mo
8	49	F PM	++	++	+	+	+	+	+	–	+	–	++	+	+	–	Triamcinolone	Clobetasol minoxidil	Arrested	12 mo
9	44	M	++	+	+	+	+	+	+	–	+	–	+	+	+	–	HCQ	Clobetasol minoxidil	Arrested	9 mo
10	50	M	+	+	++	+	+	+	++	–	++	–	++	+	+	–	HCQ	Tacrolimus minoxidil	Arrested	9 mo
11	40	F	++	++	+	++	++	++	+	–	+	–	+	++*	+	–	Triamcinolone	Tacrolimus minoxidil	Arrested	6 mo
12	56	F PM	++	+	+	++	++	++	+	–	+	–	++	+	+	–	Triamcinolone	Clobetasol minoxidil	Arrested	6 mo
13	55	F PM	+	+	++	+	+	+	++	–	++	–	++	+	+	–	HCQ	Clobetasol minoxidil	Arrested	12 mo
14†	68	F PM	+	+	++	+	+	+	++	–	++	–	+++	+*	+	–	Finasteride	Pimecrolimus minoxidil	Arrested	9 mo
15	47	M	++	+	+	+	+	+	+	–	+	–	+	++	+	+	HCQ	Clobetasol minoxidil	Arrested	7 mo
16†	75	F PM	+	+	++	+	+	+	+	–	+	–	+	+	++	–	Finasteride	Tacrolimus minoxidil	Arrested	9 mo
17	48	M	+	+	+	+	+	+	+	–	+	–	++	+	+	–	Finasteride	Clobetasol minoxidil	Arrested	12 mo
18 CPHL	70	F PM	–	–	+	–	–	–	–	–	+	–	++	+	++	–	Dutasteride	Tacrolimus minoxidil	Arrested	9 mo
19	59	F PM	++	++	+	+	++	++	+	–	+	–	++	++*	+	+	Finasteride	Clobetasol minoxidil	Arrested	12 mo
20†	67	F PM	+	+	++	+	+	+	++	–	++	–	++	+	+	–	Finasteride	Tacrolimus minoxidil	Arrested	10 mo

–, Absent; +, mild; ++, moderate; +++, severe; *F*, female; *HCQ*, hydroxychloroquine; *M*, male; *PM*, postmenopausal.
*Terminal hair follicles spared.
†Association with frontal fibrosing alopecia.

Table II. Clinical data of patients with lichen planopilaris diffuse pattern

Demography			Symptoms		Signs		Trichoscopy						Pathology				Treatments			
Patient	Age, y	Sex	Itch	Pain	Thinning	Erythema	Perifollicular erythema	Perifollicular hyperkeratosis	Loss of follicular ostia	Hair tufted	White patches	Broken hair	Reduction sebaceous glands	Lichenoid infiltrate	Perifollicular fibrosis	Tufted hairs	Systemic	Topical	Outcome	Duration
1	48	F	+	+	+	+	+	+	+	–	+	–	++	+	+	+	Triamcinolone	Minoxidil	Arrested	5 mo
2	71	F PM	++	+	+	+	+	++	+	+	+	+	+	+	++	–	Triamcinolone HCQ	Minoxidil	Slowly progressive	still in treatment
3	34	M	++	+	–	+	+	++	+	+	+	–	+	+	+	+	Triamcinolone	Clobetasol minoxidil	Arrested	6 mo
4	56	M	+	+	–	–	+	–	+	+	+	–	++	+	+	+	HCQ	Clobetasol minoxidil	Arrested	9 mo
5	44	M	++	+	+	+	+	+	+	–	+	–	++	+	+	+	Triamcinolone	Clobetasol minoxidil	Arrested	4 mo
6	51	F PM	+	+	–	–	+	–	+	–	+	–	++	+	+	–	HCQ	Clobetasol minoxidil	Arrested	12 mo
7	50	M	++	+	–	++	+	–	+	–	+	+	+	+	+	+	Triamcinolone HCQ	Clobetasol minoxidil	Arrested	15 mo
8	48	F	++	++	+	–	+	+	+	+	+	–	++	+	+	+	Triamcinolone	Clobetasol minoxidil	Arrested	5 mo
9	43	M	++	+	+	+	+	+	+	+	+	–	+	++	++	+	Triamcinolone	minoxidil	Arrested	6 mo
10	46	M	++	+	–	+	+	+	+	+	+	–	+	+	+	–	HCQ	Clobetasol minoxidil	Arrested	12 mo
11	64	F	++	++	+	++	+	++	+	+	+	–	+	+	++	–	Triamcinolone	Clobetasol minoxidil	Arrested	4 mo
12	50	F	++	+	+	++	+	++	+	–	+	+	++	+	++	+	Triamcinolone	Clobetasol minoxidil	Arrested	4 mo
13	55	F PM	+	+	–	+	+	–	+	–	+	–	++	+	+	+	HCQ	Clobetasol minoxidil	Arrested	12 mo
14	68	F PM	+	+	–	–	+	–	+	–	+	–	+	++	++	+	HCQ	Clobetasol minoxidil	Arrested	9 mo
15	47	M	++	+	+	–	+	+	+	+	+	–	+	+	+	+	HCQ	Clobetasol minoxidil	Arrested	12 mo
16	60	F PM	++	+	–	+	+	+	+	+	+	–	+	+	+	–	Triamcinolone	Minoxidil	Arrested	6 mo
17	59	M	+	+	+	+	+	–	+	–	+	–	++	++	+	+	Triamcinolone	Clobetasol minoxidil	Arrested	12 mo
18	52	F PM	++	++	+	–	+	+	+	–	+	+	+	+	++	+	Triamcinolone	Clobetasol minoxidil	Arrested	8 mo
19	49	F PM	++	++	+	+	+	++	+	–	+	–	+	++	++	+	Triamcinolone	Clobetasol minoxidil	Arrested	12 mo
20	65	M	+	+	–	–	+	+	+	–	+	–	++	+	+	–	Triamcinolone	Minoxidil	Arrested	4 mo

–, Absent; +, mild; ++, moderate; +++, severe; *F*, female; *HCQ*, hydroxychloroquine; *M*, male; *PM* postmenopausal.

Table III. Distinguishing clues between fibrosing alopecia in pattern distribution (*FAPD*), cicatricial pattern hair loss (*CPHL*), and lichen planopilaris diffuse pattern (*LPPDP*)

Features	FAPD	CPHL	LPPDP
Clinical features	Mostly targets postmenopausal women	Mostly targets postmenopausal/ elderly women	No sex predilection
	Involvement of the central scalp	Involvement of the central scalp	Diffuse involvement of the scalp
	Moderate hair thinning	Moderate/severe hair thinning	Mild hair thinning
	Moderate hitching/burning sensation	Absent/mild hitching/burning sensation	Moderate/severe hitching/burning sensation
	Often misdiagnosed with androgenetic alopecia	Often misdiagnosed with androgenetic alopecia	Often misdiagnosed with seborrheic dermatitis
	Clinically evident erythema	"Pencil eraser–sized" areas of focal atrichia	Clinically evident erythema
Trichoscopic features	Follicular erythema	Absence of follicular erythema	Follicular erythema
	Perifollicular hyperkeratosis	Absence perifollicular hyperkeratosis	Perifollicular hyperkeratosis
	Loss of follicular ostia	Loss of follicular ostia	Loss of follicular ostia
	White fibrotic patches	White fibrotic patches	White fibrotic patches
	Absence of tufted hair	Absence of tufted hair	Tufted/broken hair
Histologic features	Lichenoid infiltrate of miniaturized follicles	Lichenoid infiltrate of miniaturized follicles	Lichenoid infiltrate of terminal follicles
	Perifollicular lamellar fibrosis of miniaturized follicles	Perifollicular lamellar fibrosis of miniaturized follicles	Perifollicular lamellar fibrosis of terminal follicles
	Sparing of terminal hair	Sparing of terminal hair	Sparing of miniaturized follicles
	Prevalent lymphocytic interface dermatitis	Prevalent fibrotic collagen tracts and streamers	Prevalent lymphocytic interface dermatitis
	Reduced number of hair follicles and sebaceous glands	Reduced number of hair follicles and sebaceous glands	Reduced number of hair follicles and sebaceous glands

associated with perifollicular lamellar fibrosis involving predominantly the miniaturized hair follicles, whereas the lymphocytic infiltrate in LPPDP involved mainly the terminal follicles, as in classical LPP, sparing miniaturized follicles. Compared with LPP, however, the number of targeted hair follicles in LPPDP and the intensity of the infiltrate in each microscopic field of view were milder than in classical LPP.

From the clinical presentation, trichoscopic images, histologic features, and response to treatment, it could be assumed that these lichenoid alopecias could represent a variable pattern of 2 main diseases: FFA and LPP. Although FAPD and CPHL could be comparable to FFA, LPPDP is more likely closer to the classical LPP. This confirms that LPP and FFA may be generalized processes affecting the scalp.[12]

Although this sample is too small to make conclusions about the concomitant occurrence of FAPD and FFA, we noticed 4 patients who were diagnosed with both diseases.

All the details mentioned above support the absence of clear boundaries separating FFA, FAPD, and CPHL and support the hypothesis they could represent a spectrum of the same disease. Because the clinical, trichoscopic, and histopathologic examinations show similar features, some studies have suggested an overlap or a progression between FFA, FAPD, and CPHL.[9,13,14]

Finally, the question of whether central centrifugal cicatricial alopecia may represent FAPD in African patients has been recently raised because they share clinical and histologic aspects.[15] Central centrifugal cicatricial alopecia is a lymphocytic cicatricial alopecia that has been associated with hair care practices, such as hot combs, relaxers, and occlusive ointments. It clinically begins in the central midline scalp with a gradual centrifugal spread, and its histologic appearance presents no differences from LPP.[16,17]

CONCLUSION

LPP variants are classically described as localized diseases. As reported in the recent literature, our study confirms that they can occur diffusely on the scalp. These widespread forms are often misdiagnosed as seborrheic dermatitis or androgenetic alopecia, with consequent delay in the diagnosis and a progression of the irreversible fibrosis. In clinical

practice, it is advisable to consider lichenoid alopecias in patients with a long-standing history of erythema and dysesthesia of the scalp associated with trichoscopic signs suggesting LPP. A specimen from a trichoscopy-guided biopsy is mandatory to confirm the diagnosis in these patients and begin the proper treatment as soon as possible. This study provides strong evidence that early diagnosis and treatment are decisive for outcome, because all patients showed cessation of disease progression.

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