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

Title: Diffuse variants of scalp Lichen Planopilaris: clinical, trichoscopic and histopathologic features of 40 patients.

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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.

Abstract

Background: Fibrosing Alopecia in a Pattern Distribution (FAPD) and Cicatricial Pattern Hair Loss (CPHL) 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 histopathological diagnosis of LPP were reviewed.

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

Results: Eighteen patients were diagnosed with FAPD and two patients with CPHL. Furthermore, a new variant of diffuse LPP named “Lichen Plano-Pilaris Diffuse Pattern” was described in 20 subjects.

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

Conclusions: In patients complaining of a long-lasting history of scalp erythema, itching/dysesthesia and diffuse hair thinning it’s advisable to consider diffuse variants of LPP.

Introduction

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]

Based on 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 (GLPLS) is characterized by the triad: cicatricial alopecia of the scalp, non-cicatricial 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 fronto-temporal hairline associated with loss of eyebrows, eyelashes, and peripheral body hair, affecting mainly postmenopausal women. [5-7]

Moreover, two other forms of scarring alopecia have been reported with histological 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 Truëb, presents as central scarring hair loss with perifollicular erythema and follicular hyperkeratosis and histological features of LPP and androgenetic alopecia (AGA). Cicatricial Pattern Hair Loss (CPHL), described by Olsen in 2005, [9] is characterized by histological features similar to FAPD and clinical features of female pattern hair loss (FPHL) 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.

The aim of our study was to describe the epidemiological, 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 histopathological diagnosis of LPP.

Materials and methods

This is a retrospective study including 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 (DIMES) of the University of Bologna, from April 2015 to April 2018.

All patients signed informed consent for the use of the clinical documentation and photos for scientific purposes. Patients were excluded if they were diagnosed with scalp diseases other than LPP or if complete clinical data was not available.

The medical data collected included: age, areas of scalp involvement, subjective symptoms, clinical and trichoscopical signs, histopathological features, prescribed treatments and their efficacy.

Results

Eighteen patients received the diagnosis of FAPD and two patients of CPHL. In the other 20 cases, we identified a new clinical variant called “Lichen Plano-Pilaris Diffuse Pattern” (LPPDP).

Clinical Features

40 Caucasian patients, 15 males and 25 females, aged between 34 to 76 years (mean age: 54.9 years), complaining of a long-lasting history of itching/burning sensation on the scalp associated with diffuse hair thinning were analyzed. Patches of scarring alopecia were absent.

FAPD was diagnosed in 18 patients, aged 38 to 75 years (mean age: 54.9 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, consisting with a diagnosis of FFA.

Two female patients (aged 72 and 76 years) were diagnosed with CPHL, as 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 aged 34 to 71 years (mean age: 53 years). Eleven (55%) patients were females (mean age: 56 years) and 9 patients were males (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 patients (65%) (Fig. 2, A). Clinical features are resumed in Table I and II.

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

Trichoscopy

In FAPD and CPHL groups (Table I) trichoscopical features were correlated to subjective symptoms. All patients with FAPD complaining of scalp itching or pain showed trichoscopical inflammatory signs, such as perifollicular erythema and follicular hyperkeratosis (Fig. 1, B). By contrast, these inflammatory signs were absent in the two patients with asymptomatic disease and diagnosed with CPHL. All patients with FAPD and CPHL showed loss of follicular ostia and white fibrotic patches (Fig. 1, B) limited to the area of androgenetic hair loss. Tufted or broken hair were 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 and tufted hair were seen in 14 (70%) and 9 patients (45%) respectively (Fig. 2, B). Four patients showed broken hair.

Pathology

All patients underwent a trichoscopy-guided scalp biopsy, where the specimen was collected in the most active part of the affected area. All patients showed histological 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 histopathological 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 with FAPD (33.3%), 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, while a lymphocytic interface dermatitis with destruction of basal keratinocytes was evident in subjects with FAPD and acute symptoms.

All scalp biopsy specimens taken from the 20 patients affected by LPPDP showed similar pathological changes (Table II). Like in FAPD, we observed the presence of lymphohistiocytic infiltrate around the isthmus and infundibular region with 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 two 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/day or dutasteride 0.5 mg/day, and 4 patients with hydroxychloroquine 400 mg/day. Five patients with subjective symptoms and/or evident inflammatory signs 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 two patients with CPHL were treated with finasteride 2.5 mg/day 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) and hydroxychloroquine 400 mg/day, in 14 and 8 patients, respectively. As for FAPD, topical therapy with clobetasol propionate 0.05% cream was the most recommended (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 almost all of the patients (95%) after 1 year.

Discussion

LPP is an inflammatory scalp disorder that is considered the most common cause of scarring alopecia. [2] According to the North American Hair Research Society, [10] it is divided into three clinical variants, mainly distinguished by the clinical pattern of hair loss: classical LPP, FFA and GLPLS. [11] Less common subtypes include FAPD, 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] and CPHL, a variety of lymphocytic cicatricial alopecia in a FPHL without clinically evident inflammatory signs, but characterized by small "pencil-eraser-sized" areas of focal atrichia. [9]

Therefore, LPP can present itself not only as a localized disease with defined patches of scarring alopecia, but also with a diffuse involvement of the scalp.

In our study we analyzed 40 Caucasian patients complaining 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 that showed histopathological features coherent with a perifollicular lichenoid reaction. A careful analysis of clinical, dermoscopic and histological data allowed us to identify three different variants of diffuse LPP (Table III).

Eighteen patients were diagnosed with FAPD and two patients with CPHL. Furthermore, we describe a new variant of diffuse LPP named LPPDP that, 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 affect also male patients, who are generally younger (mean age 47 years). Clinically, patients showed a hair thinning on the central scalp associated with itching. Trichoscopy detected small fibrotic areas and perifollicular

inflammatory signs. CPHL was found in elderly people (mean age: 74 years) who experienced a long-lasting progressive hair thinning and a decreasing itching over time. Clinically, there was no 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 post-inflammatory variant of FAPD.

The 20 cases identified as a new variant of LPP, named LPPDP, showed distinctive features compared to the two 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, and 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 associated with perifollicular lamellar fibrosis involving predominantly the miniaturized hair follicles, while in LPPDP the lymphocytic infiltrate involved mainly the terminal follicles, as in classical LPP, sparing miniaturized follicles. Compared to LPP, however, in LPPDP the number of targeted hair follicles and the intensity of the infiltrate in each microscopic field of view were milder than in classical LPP.

Based on the clinical presentation, trichoscopical images, histological features and response to treatment, it could be assumed that these lichenoid alopecias could represent a variable pattern of two main diseases: FFA and LPP. While 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 process affecting the scalp. [12]

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

All the abovementioned details support the absence of clear boundaries separating FFA, FAPD and CPHL, supporting the hypothesis they could represent a spectrum of the same disease. Since the clinical, trichoscopical and histopathological exams 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 (CCCA) may represent FAPD in African patients has been recently raised, as they share clinical and histological aspects.[15] CCCA 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 histological appearance presents no differences from LPP. [16, 17]

Conclusions

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 AGA, with consequent delay in the diagnosis and a progression of the irreversible fibrosis. In the 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 trichoscopical signs suggesting LPP. In these patients a trichoscopy-guided biopsy is mandatory to confirm the diagnosis and begin the proper treatment as soon as possible. This study provides strong evidence that early diagnosis and treatment are decisive for outcome, since all cases showed cessation of disease progression.

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Figure Legend

Fig. 1: A, Mild erythema of the scalp and clinically evident hair thinning in the crown area in a subject 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: loss of sebaceous glands, fibrotic collagen tracts, dermal melanofages and mild lymphocytic infiltrate around the isthmus of miniaturized hair follicles. Terminal hair follicles are spared. (H&E stain, 4x). **D**, Transverse section of the scalp biopsy specimen: loss of sebaceous glands, perifollicular lymphocytic infiltrate and lamellar fibrosis involving mainly the miniaturized hair follicles and fibrotic collagen tracts. (H&E stain, 4x).

Fig. 2: A, Clinically evident erythema of the scalp in a patient diagnosed with Lichen Planopilaris Diffuse Pattern, complaining of two years history of diffuse hair thinning and scalp severe itching. **B**, Trichoscopy shows perifollicular erythema and hyperkeratosis associated with white fibrotic areas and tufted hairs. **C**, Longitudinal section of the scalp biopsy specimen: lymphocytic infiltrate around the isthmus of a terminal hair follicle and mild concentric perifollicular lamellar fibrosis (H&E stain, 4x). **D**, Transverse section of the scalp biopsy specimen: 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 (H&E stain, 4x).

Table Legend

Table I: Clinical data of patients with Fibrosing Alopecia in Pattern Distribution and Cicatricial Pattern Hair Loss (CPHL).

Table II: Clinical data of patients with Lichen Planopilaris Diffuse Pattern.

Table III: Distinguish clues between Fibrosing alopecia in pattern distribution (FAPD), Cicatricial pattern hair loss (CPHL) and Lichen planopilaris diffuse pattern (LPPDP).

Table I: Clinical data of patients with Fibrosing Alopecia in Pattern Distribution and Cicatricial Pattern Hair Loss (CPHL).

DEMOGRAPHY			SINTOMS		SIGNS		TRICOSCOPY						PATHOLOGY				TREATMENTS			
Case	Age,y	Sex	Itch	Pain	Thinning	Erythema	Perifollicular Erythema	Perifollicular Hyperkeratosis	Loss of follicular ostia	Hair Tufted	White Patches	Broken Hair	Reduction Sebaceus Glands	Lichenoid Infiltrate	Perifollicular Fibrosis	Tafted Hairs	Systemic	Topical	Outcome	Duration
1	65	F PM	+	+	++	+	+	+	++	-	++	-	++	+	+	-	finasteride	clobetasol minoxidil	Arrested	9 months
2	50	F PM	++	-	++	+	+	++	++	-	++	-	+	++**	+	-	finasteride	tacrolimus minoxidil	Arrested	12 months
3	38	M	++	+	++	+	+	++	++	-	++	-	+	++	+	-	triamcinolone	clobetasol minoxidil	Arrested	6 months
4 CPHL	76	F PM	-	-	++	-	-	-	++	-	+	-	++	+	++	-	finsteride	tacrolimus minoxidil	Arrested	6 months
5	53	F PM	+	+	+	+	+	+	+	-	+	-	++	+	+	+	dutasteride	pinecrolimus minoxidil	Arrested	12 months
6*	72	F PM	+	-	++	+	+	+	++	-	++	-	+++	+**	++	-	finasteride	tacrolimus minoxidil	Arrested	12 months
7	55	M	++	+	++	++	++	++	+	-	++	-	++	+**	+	-	triamcinolone	clobetasol minoxidil	Arrested	6 months
8	49	F PM	++	++	+	+	+	+	+	-	+	-	++	+	+	-	triamcinolone	clobetasol minoxidil	Arrested	12 months
9	44	M	++	+	+	+	+	+	+	-	+	-	+	+	+	-	HCQ	clobetasol minoxidil	Arrested	9 months
10	50	M	+	+	++	+	+	+	++	-	++	-	++	+	+	-	HCQ	tacrolimus minoxidil	Arrested	9 months
11	40	F	++	++	+	++	++	++	+	-	+	-	+	++**	+	-	triamcinolone	tacrolimus minoxidil	Arrested	6 months
12	56	F PM	++	+	+	++	++	++	+	-	+	-	++	+	+	-	triamcinolone	clobetasol minoxidil	Arrested	6 months
13	55	F PM	+	+	++	+	+	+	++	-	++	-	++	+	+	-	HCQ	clobetasol minoxidil	Arrested	12 months
14*	68	F PM	+	+	++	+	+	+	++	-	++	-	+++	+**	+	-	finasteride	pinecrolimus minoxidil	Arrested	9 months
15	47	M	++	+	+	+	+	+	+	-	+	-	+	++	+	+	HCQ	clobetasol minoxidil	Arrested	7 months
16*	75	F PM	+	+	++	+	+	+	+	-	+	-	+	+	++	-	finasteride	tacrolimus minoxidil	Arrested	9 months
17	48	M	+	+	+	+	+	+	+	-	+	-	++	+	+	-	finastreride	clobetasol minoxidil	Arrested	12 months
18 CPHL	70	F PM	-	-	+	-	-	-	-	-	+	-	++	+	++	-	dutasteride	tacrolimus minoxidil	Arrested	9 months
19	59	F PM	++	++	+	+	++	++	+	-	+	-	++	++**	+	+	finasteride	clobetasol minoxidil	Arrested	12 months
20*	67	F PM	+	+	++	+	+	+	++	-	++	-	++	+	+	-	finasteride	tacrolimus minoxidil	Arrested	10 months

- absent, + mild, ++ moderate, +++ severe

PM postmenopausal, HCQ hydroxychloroquine

* association with FFA

** terminal hair follicles spared

Table II: Clinical data of patients with Lichen Planopilaris Diffuse Pattern.

DEMOGRAPHY			SINTOMS		SIGNS		TRICOSCOPY						PATHOLOGY				TREATMENTS			
Case	Age,y	Sex	Itch	Pain	Thinning	Erythema	Perifollicular Erythema	Perifollicular Hyperkeratosis	Loss of follicular ostia	Hair Tufted	White Patches	Broken Hair	Reduction Sebaceus Glands	Lichenoid Infiltrate	Perifollicular Fibrosis	Tafted Hairs	Systemic	Topical	Outcome	Duration
1	48	F	+	+	+	+	+	+	+	-	+	-	++	+	+	+	triamcinolone	minoxidil	Arrested	5 months
2	71	F PM	++	+	+	+	+	++	+	+	+	+	+	+	++	-	triamcinolone HCQ	minoxidil	Slowly progressive	still in treatment
3	34	M	++	+	-	+	+	++	+	+	+	-	+	+	+	+	triamcinolone	clobetasol minoxidil	Arrested	6 months
4	56	M	+	+	-	-	+	-	+	+	+	-	++	+	+	+	HCQ	clobetasol minoxidil	Arrested	9 months
5	44	M	++	+	+	+	+	+	+	-	+	-	++	+	+	+	triamcinolone	clobetasol minoxidil	Arrested	4 months
6	51	F PM	+	+	-	-	+	-	+	-	+	-	++	+	+	-	HCQ	clobetasol minoxidil	Arrested	12 months
7	50	M	++	+	-	++	+	-	+	-	+	+	+	+	+	+	triamcinolone HCQ	clobetasol minoxidil	Arrested	15 months
8	48	F	++	++	+	-	+	+	+	+	+	-	++	+	+	+	triamcinolone	clobetasol minoxidil	Arrested	5 months
9	43	M	++	+	+	+	+	+	+	+	+	-	+	++	++	+	triamcinolone	minoxidil	Arrested	6 months
10	46	M	++	+	-	+	+	+	+	+	+	-	+	+	+	-	HCQ	clobetasol minoxidil	Arrested	12 months
11	64	F	++	++	+	++	+	++	+	+	+	-	+	+	++	-	triamcinolone	clobetasol minoxidil	Arrested	4 months
12	50	F	++	+	+	++	+	++	+	-	+	+	++	+	++	+	triamcinolone	clobetasol minoxidil	Arrested	4 months
13	55	F PM	+	+	-	+	+	-	+	-	+	-	++	+	+	+	HCQ	clobetasol minoxidil	Arrested	12 months
14	68	F PM	+	+	-	-	+	-	+	-	+	-	+	++	++	+	HCQ	clobetasol minoxidil	Arrested	9 months
15	47	M	++	+	+	-	+	+	+	+	+	-	+	+	+	+	HCQ	clobetasol minoxidil	Arrested	12 months
16	60	F PM	++	+	-	+	+	+	+	+	+	-	+	+	+	-	triamcinolone	minoxidil	Arrested	6 months
17	59	M	+	+	+	+	+	-	+	-	+	-	++	++	+	+	triamcinolone	clobetasol minoxidil	Arrested	12 months
18	52	F PM	++	++	+	-	+	+	+	-	+	+	+	+	++	+	triamcinolone	clobetasol minoxidil	Arrested	8 months
19	49	F PM	++	++	+	+	+	++	+	-	+	-	+	++	++	+	triamcinolone	clobetasol minoxidil	Arrested	12 months
20	65	M	+	+	-	-	+	+	+	-	+	-	++	+	+	-	triamcinolone	minoxidil	Arrested	4 months

- absent, + mild, ++ moderate, +++ severe

PM postmenopausal, HCQ hydroxychloroquine

Table III: Distinguish clues between Fibrosing alopecia in pattern distribution (FAPD), Cicatricial pattern hair loss (CPHL) and Lichen planopilaris diffuse pattern (LPPDP).

	FAPD	CPHL	LPPDP
CLINICAL FEATURES	Mostly targets post-menopausal women	Mostly targets post-menopausal/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 AGA	Often misdiagnosed with AGA	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
HISTOLOGICAL 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 sebaceus glands	Reduced number of hair follicles and sebaceus glands	Reduced number of hair follicles and sebaceus glands

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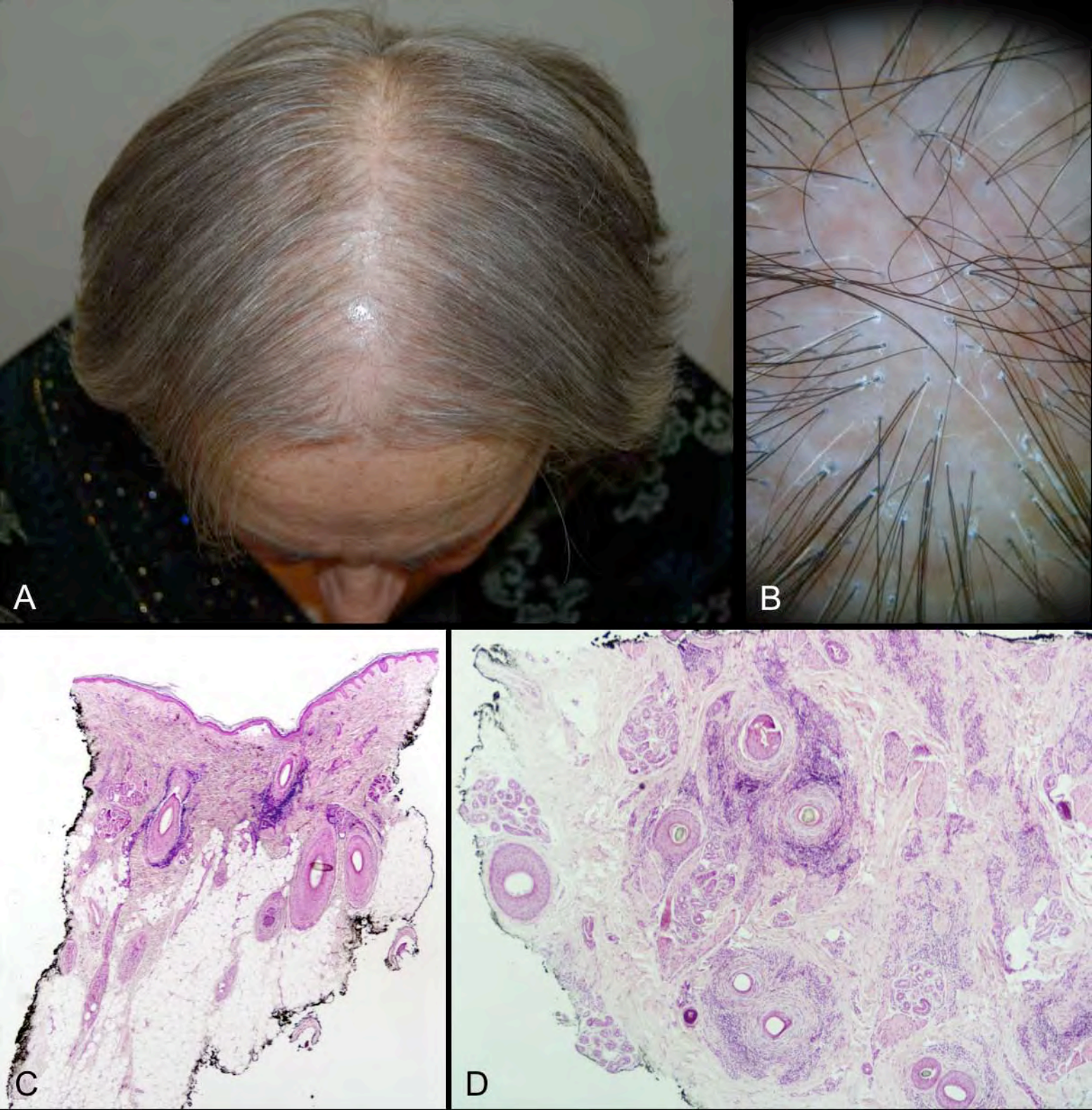
Capsule Summary

- 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.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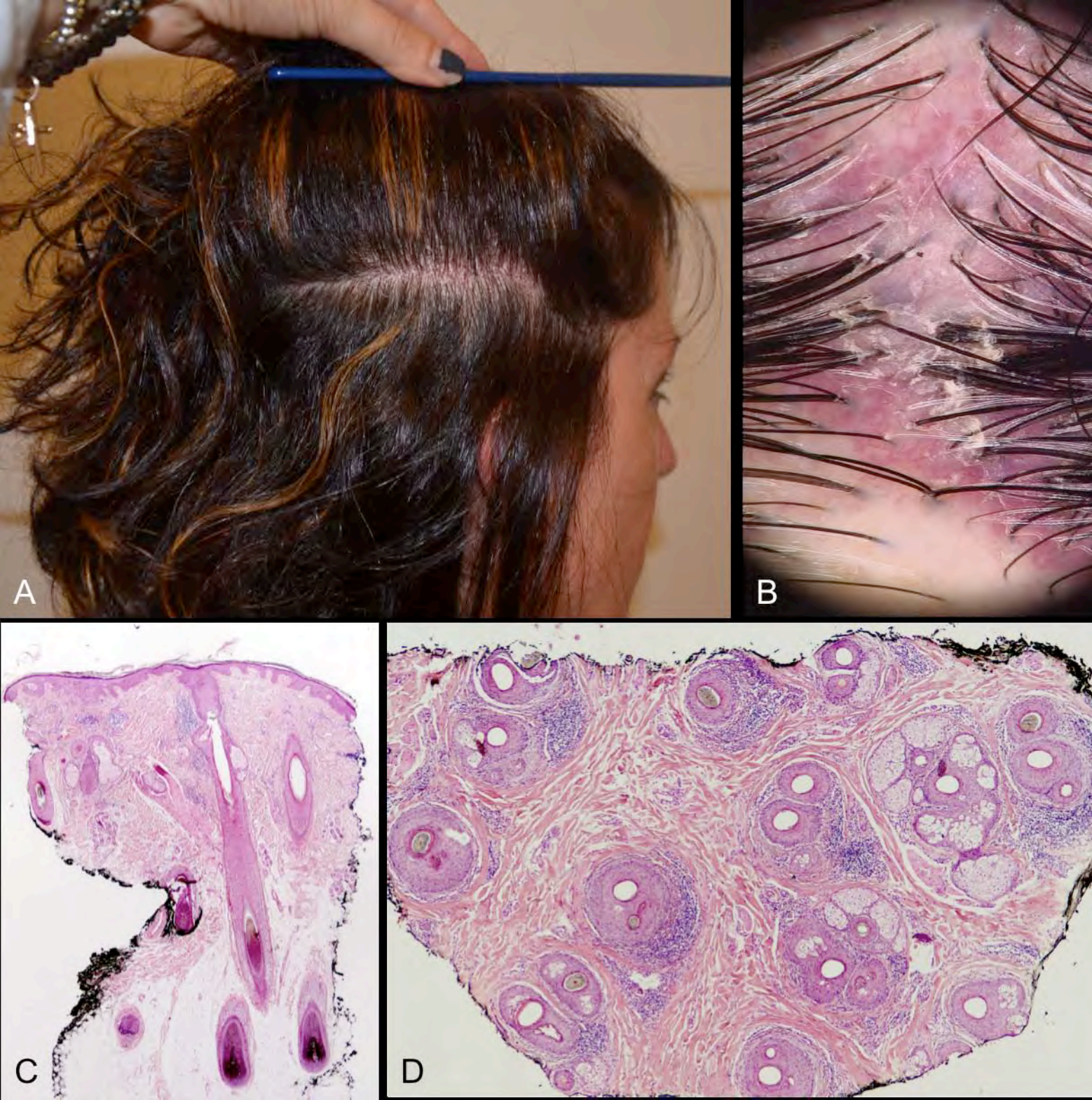
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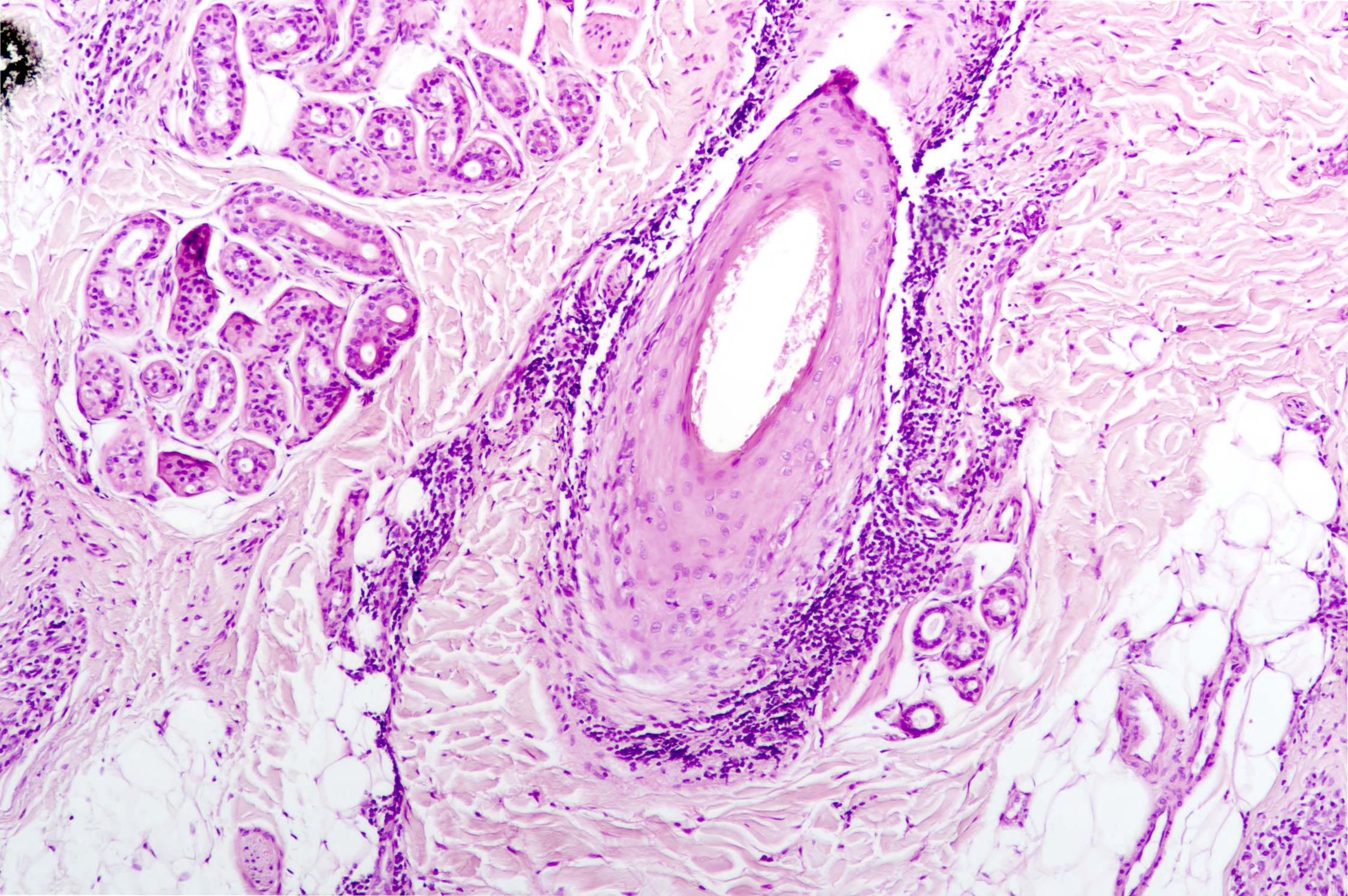

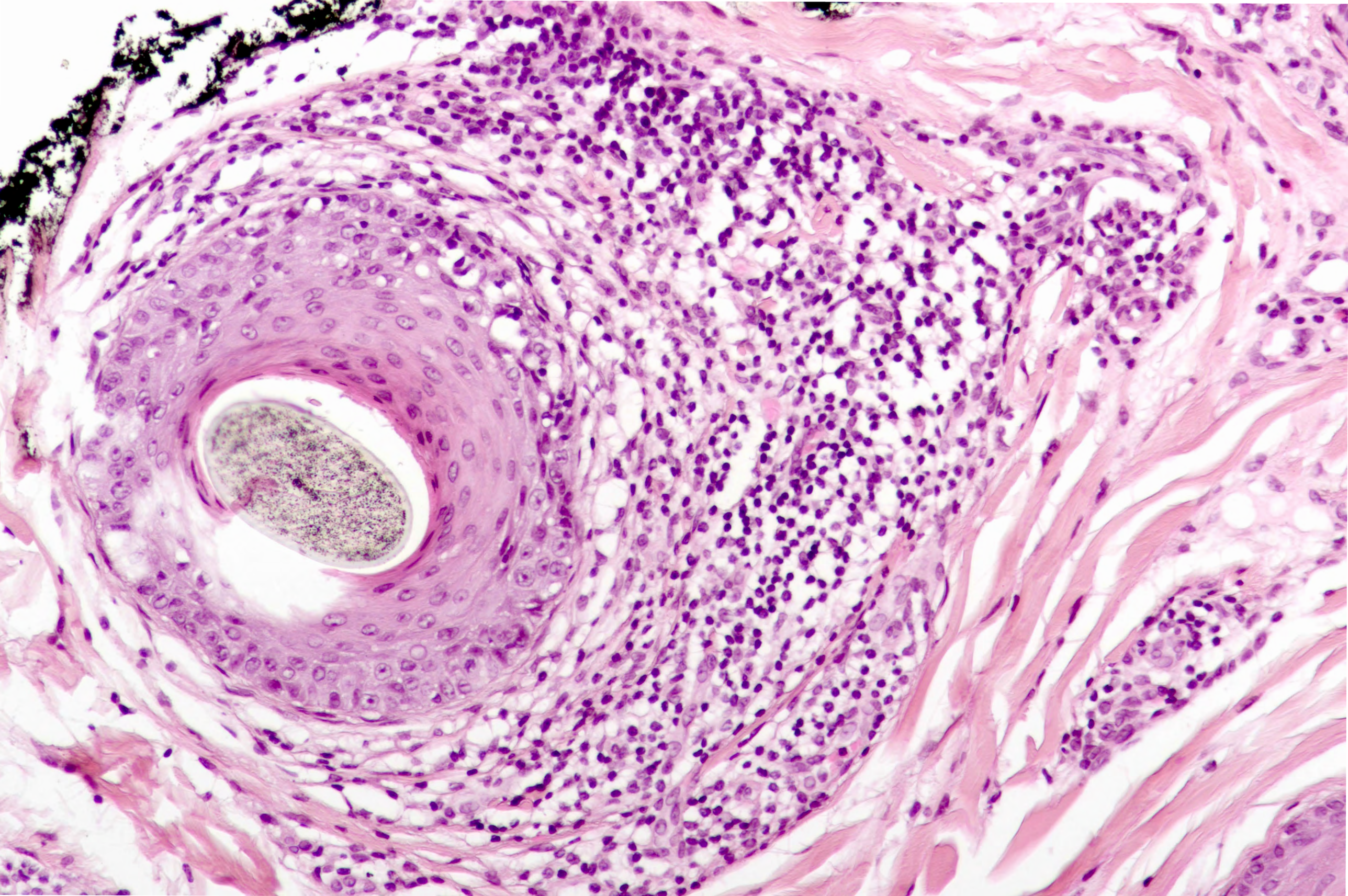

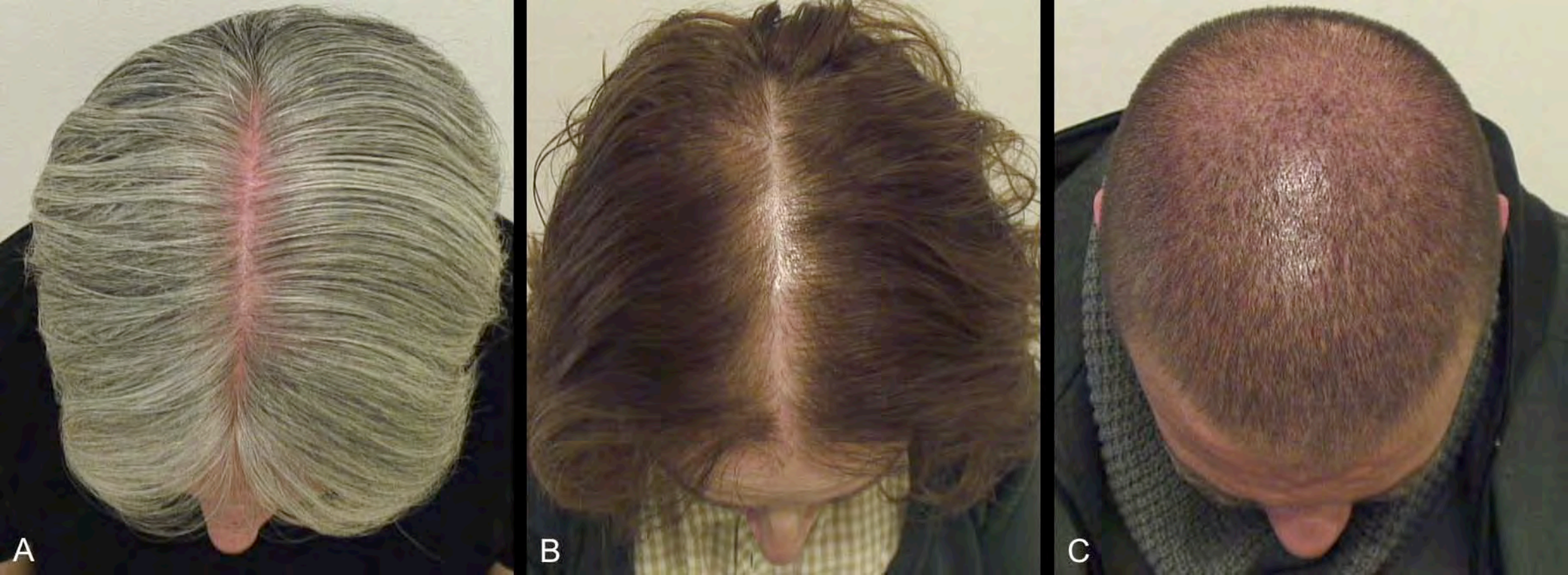
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