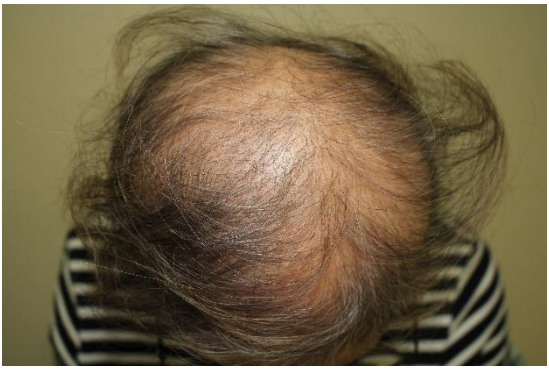
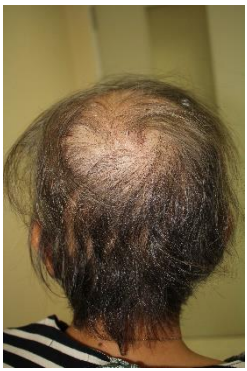
Case 2
A 58-year-old Thai woman from Bangkok
Chief complaint: Progressive hair loss for 10 years

Present illness: The patient gradually developed progressive hair loss for 10 years. The lesion started from central scalp and spread centrifugally with no symptom. She denied previous illness before onset of alopecia, also history of significant weight loss and family history of androgenetic alopecia.

Past history

There was no underlying disease or any current medication

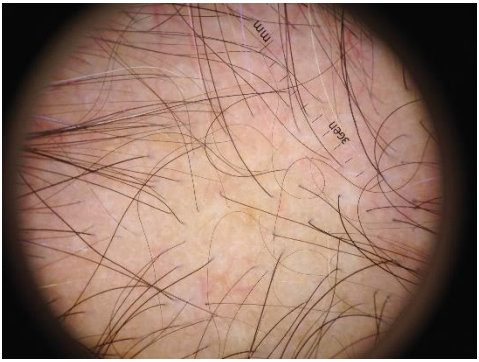
Physical examination

HEENT: not pale, no jaundice, thyroid gland not enlarge
Heart and lung: normal
Abdomen: no hepatosplenomegaly

Skin examination

Scalp: Diffuse alopecia predominately on vertex, frontal and parietal area. Perifollicular scale, mild perifollicular erythema and follicular dropout were generally observed within alopecic area. Dermoscopic examination revealed

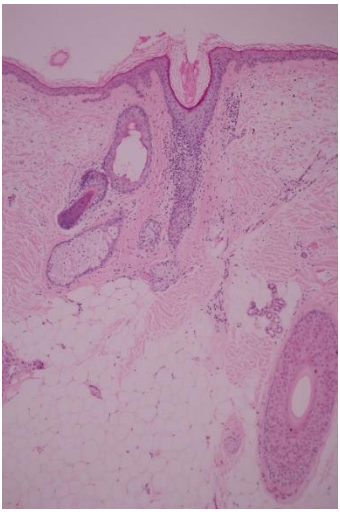
diversity of hair shaft diameter, focal atrichia, whitish spots, interfollicular erythema and arborizing blood vessels.

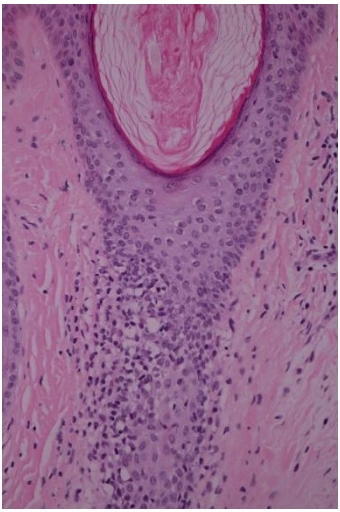


Skin: otherwise unremakable

Histopathology (S15-020542)

Dense inflammatory cell infiltrate of lymphocytes around upper portion of terminal hair follicle. Miniaturized terminal hair follicle in the scalp.





Diagnosis: Fibrosing alopecia in a pattern distribution
Treatment: Hydroxychloroquine 200 mg twice daily, Finasteride 2.5 mg once daily, Desoximetasone 0.25% apply twice daily

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Discussion:

Fibrosing alopecia in a pattern distribution (FAPD), was first described by Zinkernagel and Trueb in 2000.[1] FAPD was considered to be a variant of lichen planopilaris (LPP).[2] It is a distinct form of cicatricial alopecia characterized by inflammation and fibrosis with accelerated hair loss in the distribution of typical male androgenetic alopecia (AGA) or female pattern hair loss (FPHL).[1]

The etiology and pathogenesis of this condition have not been elucidated. A benefit of antiandrogen therapy or oral finasteride in stabilizing the progression of hair loss and decrease the scalp inflammation suggested that androgen may play a role in the development of the inflammatory reaction of the scalp[1], although further studies are warranted.

Clinical presentation reveals features of an inflammatory scarring alopecia, perifollicular erythema, loss of follicular ostia and perifollicular hyperkeratosis confined to the area involved by pattern hair loss.

Histopathology characterized by lichenoid inflammation selectively targeting the miniaturizing follicles. Terminal to vellus hair ratio was significantly

reduced in 70% of patients and fibrous tracts were observed. A lymphohistiocytic infiltration around isthmus and infundibular region of hair follicles was found in all cases. Follicular interface dermatitis found in 57% of patients in the early phase. The overlying interfollicular epidermis and lower portions of the follicles including the hair bulbs were spared. Concentric perifollicular lamellar fibrosis was found in 93%. Neither inflammation nor fibrosis was observed around the sweat glands. Fibrous tracts in subcutis extended through the reticular dermis at the sites of destroyed follicles, naked hair shaft fragments and orphaned arrector pili muscles occasionally were found.[1]

The histological differential diagnosis of lymphocytic inflammation involving the upper follicle and presence of completely fibrosed follicular tracts includes lichen planopilaris, frontal fibrosing alopecia, pseudopelade of Brocq and central centrifugal cicatricial alopecia. The differential diagnosis and their histological features was summarized in table 1.[3]

Currently there was no standard treatment for FAPD due to limited of case reports. The choices of treatment include combination of topical minoxidil and clobetasol propionate. Antiandrogen therapy, cyproterone or oral finasteride, significantly stabilized the progression of disease and reduced clinical signs of inflammation.[1] Because FAPD was classified as a variant of LPP, non-aggressive disease may be treated by moderate- or high- potency topical steroids, intralesional triamcinolone acetonide or combination of these agents.[4] Systemic medications indicated for local steroid-refractory, rapid progressive, active and

symptomatic case. Short courses of systemic steroids, retinoids, or antimalarials could be considered.

References

1. Zinkernagel MS , Trueb RM. Fibrosing alopecia in a pattern distribution: patterned lichen planopilaris or androgenetic alopecia with a lichenoid tissue reaction pattern? Arch Dermatol 2000;136:205-11.
2. Sperling LC, Solomon AR , Whiting DA. A new look at scarring alopecia. Arch Dermatology 2000;136:235-42.
3. Chiu HY , Lin SJ. Fibrosing alopecia in a pattern distribution. J Eur Acad Dermatol Venereol 2010;24:1113-4.
4. Ross EK, Tan E , Shapiro J. Update on primary cicatricial alopecias. J Am Acad Dermatol 2005;53:1-37; quiz 8-40.

Table 1.The clinical and histopathological differential diagnosis

Condition	Characteristics	Histology
Fibrosing alopecia in a pattern distribution	Alopecia in the distribution of typical male AGA or FPHL Perifollicular erythema and hyperkeratosis	Miniaturization of hair follicles Lichenoid inflammatory infiltrate at isthmus and infundibular region perifollicular lamellar fibrosis
Androgenetic alopecia/female pattern hair loss	Pattern baldness on the bitemporal areas and vertex of the scalp (male) Diffuse hair thinning particularly on crown with hair line sparring (female)	Miniaturized vellus follicles Increased telogen hairs in late stage
Frontal fibrosing alopecia	Cicatricial frontotemporal hair line recession Almost exclusively in postmenopausal women Perifollicular erythema and hyperkeratosis	Perifollicular fibrosis Lymphocytic infiltration at the isthmus and infundibulum
Central centrifugal cicatricial alopecia	Cicatricial alopecia of the central scalp and enlarges centrifugally	Premature inner root sheath desquamation Lymphocytic infiltration at the upper follicle Perifollicular concentric fibrosis
Pseudopelade of Brocq	Multiple round or irregularly shaped, hairless, cicatrial patches	Early stage: perifollicular lymphocytic infiltration Late stage: follicular longitudinal fibrous tract extended into subcutis