

## Frontal fibrosing alopecia, just lichen planopilaris?



*To the Editor:* In the comprehensive review of lichen planus and lichenoid dermatoses by Tziotzios et al,<sup>1</sup> the authors suggest that frontal fibrosing alopecia (FFA) is a misnomer and a restrictive diagnosis. The central issue is whether FFA is only a distinct form of lichen planopilaris (LPP) or a more complex disorder.

The clinical presentation of FFA, a symmetric progressive recession of the frontal hairline representing a patterned alopecia occurring mainly in postmenopausal women, is distinctive.<sup>2,3</sup> Scalp biopsy reveals LPP. Cases have rarely been reported in men. Although LPP is classified as a scarring alopecia, gross scarring is not present. Lymphocytes specifically target and destroy follicles producing thin fibrous tracts with loss of follicular ostia. The subtle skin signs might be mistaken for a nonscarring alopecia, particularly alopecia areata and androgenic alopecia in the case of FFA. The term fibrosing included in FFA reflects this clinical aspect as well as the histopathology. Prominent concentric perifollicular fibrosis and lymphocytic inflammation without gross interfollicular scarring is found indistinguishable from LPP.

I consider this distinctive presentation and course of LPP to be linked to sex-based differences in androgenic male- and female-pattern alopecia and ultimately to the hormonal changes of menopause. One variant of male-pattern alopecia features marked recession of the frontal hairline. This contrasts with female-pattern hair loss, which is characterized by retention of the frontal hairline, although temporal recession might occur.

The basis for the variation in patterned hair loss in androgenetic alopecia in men and women remains unknown. Ultimately, these differences might hinge on the variation in type and density of hormonal and other follicular receptors that are genetically controlled and differ within the areas of patterning. Clearly, FFA cannot be equated to the distinct form of male-pattern alopecia affecting the frontal hairline. This has an earlier onset and is not driven by lichenoid destruction of follicles but by progressive miniaturization and loss of hair density.

It is possible that women with FFA are gene carriers of this male-pattern subtype. At the time of menopause, changes in the sex hormone profile and relative levels could affect the binding and saturation

of these receptors distributed in a patterned distribution. This in turn might lead to conformational changes in the follicular receptors. In the frontal hairline, this menopause-related change in conformation could trigger a targeted lymphocytic response that was previously protected by the premenopausal hormonal milieu.

Once the lymphocytes have attained the capacity to eliminate follicles, epitope spreading and more extensive symmetric follicular destruction might ensue at other sites, particularly the eyebrows. This aspect could precede clinically obvious frontal hairline recession and be observed by patients.

This hypothetical model of FFA could open further directions in the proposed investigations for the pathogenesis of this subset of LPP.<sup>4</sup> The potential key pathogenic element of hormonal change captured postmenopause in the initial designation of FFA has been dropped, and it appears premature to propose a further change in terminology for this complex form of alopecia that might not be just LPP.

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*Funding sources: None.*

*Conflicts of interest: None disclosed.*

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<https://doi.org/10.1016/j.jaad.2019.02.072>