Commentary on: "Cicatricial pattern hair loss is not a variant of lichen planopilaris"

To the Editor: We thank Dr Bhoyrul[1] for his letter in response to our original article "Diffuse variants of scalp lichen planopilaris: Clinical, trichoscopic, and histopathologic features of 40 patients."[2]

Cicatricial pattern hair loss (CPHL) is a form of cicatricial alopecia in a female pattern hair loss, first described by Olsen in 2005.[3] Nowadays, some authors refer to CPHL not as a single disease but rather as a clinical phenotype of different subtypes of cicatricial alopecias.[4]

According to the description included in Olsen's original article,[3] all of our patients showed small "pencil-eraser-sized" areas of focal atrichia in the crown area, in absence of perifollicular erythema and follicular hyperkeratosis. Histologically, they manifested lichenoid perifollicular lymphocytic infiltrate surrounding the isthmus and infundibular region, sebaceous gland loss, and concentric lamellar fibrosis.

Based on the clinical and histologic aspect of our cases, we are convinced that we were not simply observing an advanced form of androgenetic alopecia (AGA). Indeed, the end stages of AGA show upper dermal perifollicular inflammation and mild perifollicular fibrosis involving both telogen and anagen hair follicles.[5] Contrarily, the lichenoid infiltrate in CPHL attacks and destroys the hair follicles before they enter the telogen phase. Although the perifollicular inflammation is also described in classical AGA, finding an inflammatory infiltrate limited to the upper part of the follicle associated with perifollicular fibrosis is mandatory to make the diagnosis of lichenoid alopecia.[6]

We agree with the author's opinion that our patients with CPHL "may have had FAPD that had 'burnt out' by the time of presentation" and "end-stage disease (as in burnt-out LPP), making the distinction between end-stage FAPD and CPHL virtually impossible."[1] Indeed, in our article, we asserted that CPHL might represent a postinflammatory variant of FAPD.[2]

Nevertheless, AGA could play a role in the development of FAPD and CPHL. The lichenoid infiltrate in FAPD occurs primarily around miniaturized hair follicles, possibly representing an exaggerated inflammatory response to damaged hair follicles typically seen in AGA. A possible pathogenetic mechanism could involve the damaged hair follicles, where the expression of cytokines may initiate an inflammatory response or an apoptosis-mediated organ deletion. The same process can be seen in healthy murine skin, where damaged follicles are removed by immunologically driven programmed organ deletion. Another theory suggests that in immunogenetically susceptible patients, an unknown antigenic stimulus could trigger a lichenoid reaction on hair follicles that are altered in the course of AGA.[3]

In regard to the therapeutic approaches, as in FAPD, the goal is to stop the progression of the disease and to achieve a cosmetically acceptable improvement. We recommend caution in performing a hair transplant in all lichenoid alopecias, because to succeed, this option should be considered exclusively in well-controlled and stable disease.

In conclusion, because CPHL shows the histologic distinctive features of LPP, we suggest that it represents a residual form of FAPD. However, we do not exclude the hypothesis that FAPD could be triggered by an underlying AGA.

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