Cicatricial pattern hair loss is not a variant of lichen planopilaris

To the Editor: We read with interest the paper by Starace et al[1] describing the clinical, trichoscopic, and histopathologic features of diffuse lichen planopilaris (LPP). There is a paucity of data on the diffuse forms of cicatricial alopecia, including fibrosing alopecia in a pattern distribution (FAPD) and cicatricial pattern hair loss (CPHL), and this case series will certainly contribute to a better understanding of these conditions. However, although FAPD probably represents a subtype of LPP,[2] we take issue with the authors' description of CPHL as a variant of diffuse LPP.

We know that permanent follicular dropout can be seen in late-stage androgenetic alopecia (AGA), alopecia areata, and traction alopecia, all of which represent alopecias that are generally considered to be nonscarring.[3] Histopathologically, upper dermal perifollicular inflammation and mild perifollicular fibrosis have been shown to be more common in AGA than in controls.[4] In his evaluation of transverse sections of scalp biopsy specimens of patients with male pattern hair loss, Whiting found moderate to severe inflammation or fibrosis, or both, in 36% of the patients.[4] Therefore, CPHL, characterized clinically by focal atrichia[5] and histologically by the above features, likely represents end-stage AGA.

In our experience, there are clinical and histologic clues to the diagnosis of CPHL. Like AGA, it tends to be asymptomatic, so it is possible that the history of pruritus in the 2 patients with CPHL in the current study may have been related to other causes (eg, seborrheic dermatitis). Alternatively, they may have had FAPD that had "burnt out" by the time of presentation. We agree that loss of follicular ostia can be observed in both FAPD and CPHL but that perifollicular erythema and scaling are absent in CPHL (Fig 1).

We concur with the authors that FAPD and CPHL are both histologically characterized by a mild peri-infundibular or peri-isthmic lymphocytic infiltrate, concentric lamellar fibrosis, and loss of sebaceous glands.[2,5] Interface changes in the follicular epithelium may be present in FAPD[2] but not in CPHL (Fig 2). However, this folliculocentric interface dermatitis may disappear in end-stage disease (as in burnt-out LPP), making the distinction between end-stage FAPD and CPHL virtually impossible.

The distinction between FAPD and CPHL, albeit challenging, is essential for therapeutic decision making. The evidence for the treatment of FAPD is limited, but the current approach is to stop disease progression with anti-inflammatory agents or 5α-reductase inhibitors, or both.[1,5] Patients with LPP and its variants are not suitable candidates for hair transplantation, whereas those with AGA are, providing they have an adequate donor supply. Whether CPHL is amenable to hair transplantation is yet to be determined. If hair transplantation is not

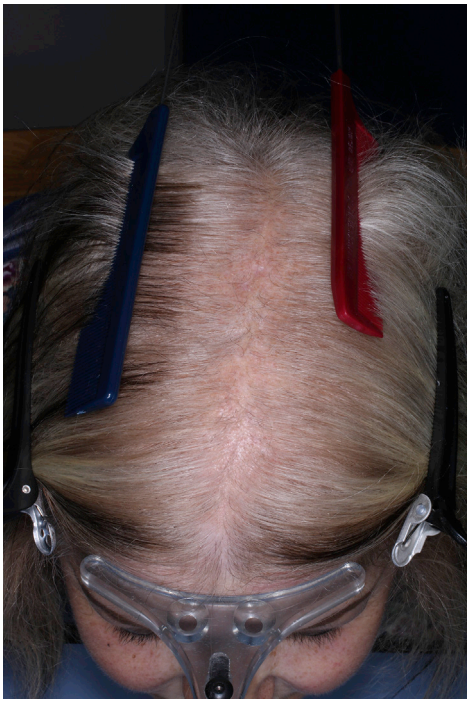

Fig 1. A 69-year-old woman with diffuse alopecia over the midfrontal scalp characterized by small areas of focal atrichia. Note the absence of perifollicular erythema or scaling.

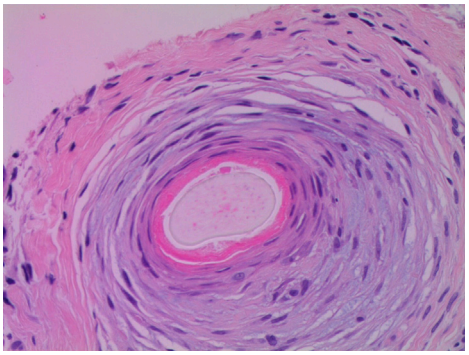

Fig 2. Horizontal section of a scalp biopsy specimen of a patient with cicatricial pattern hair loss showing a hair follicle with attenuation of the follicular epithelium and surrounding lamellar fibrosis. Interface changes (basal vacuolar changes or apoptotic keratinocytes) are not observed (Hematoxylin and eosin, original magnification: ×400).

an option, camouflage techniques should be offered to patients at an early stage.

In summary, we suggest that CPHL represents end-stage AGA with follicular dropout and fibrosis. We do not consider it a primary cicatricial alopecia, and therefore, not a variant of LPP.

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