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Telogen Effluvium: Is There a Need for a New Classification? A Reply

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Dear Editor,

I thank Prof. Trüeb for his precise update on the telogen effluvium (TE) story [1], and for having mentioned my work so vastly. Nonetheless, there are points that I would like to discuss.

The first point concerns the concept of alopecia areata incognita (AAI). As I have repeatedly commented upon [2, 3], AAI is by no means the so-called diffuse AA [4, 5]. When I proposed AAI [6], I did not describe any particular clinical manifestation. I only suggested the hypothesis that AA could not only be characterized by dystrophic shedding, but also by a diffuse shedding of telogen hairs, without developing any glabrous areas, unless the process lasts for years. Instead, diffuse AA shows patches with indistinct borders and a tendency to merge, resulting in alopecia totalis in a few weeks. I had been inclined to call my hypothetical entity AA *telogenica*, but I preferred *incognita* just to emphasize the absence of any sign of thinning of the scalp. Later I found, with the late Quercetani [7], that in some TEs a minimum of dystrophic component (3–4%) is also present. I am now prone to believe that the concept of AAI overlaps that of “inflammatory/autoimmune” TE, since their clinical presentations are alike. Finding dystrophic hairs requires examination under the microscope of a huge amount of shed hairs per patient. This is really a time-consuming test, while the commonplace modified wash test (MWT) is simple and well accepted by the patients [8].

As for the autoimmune nature (alopecia areata-like), I admit it as being a long shot of mine. As in AA, there is a history of emotional stress, the strikingly common coexistence (in women) with Hashimoto thyroiditis, and the presence of dystrophic hairs. As for the scarcity of histopathologic signs of inflammation, Whiting [9],

who studied them, did not mention if his TEs were active or not at the moment of the biopsy, which is essential to catch the possible peribulbar inflammation. The typical 3-month lag, in fact, is often misleading. Patients may well be observed when the noxious agency of their TE has already ceased to be active while hairs keep shedding. Biopsies of that moment may provide deceptive information.

The third point concerns the need for a new classification of TE. My classification is not in contrast with existing ones (Headington's classification), the correspondences between the two classifications being mentioned. There is still a need for a friendlier terminology. Not for nothing Headington's classification is mentioned in textbooks, but hardly, if ever, in the literature just because of the difficulty to apply it in the office. Whiting's case stands as an example. He discussed the Headington's classification, but did not adopt it. His patients could belong to almost any of the different subtypes of TE and not necessarily to the one I labelled inflammatory/autoimmune.

My aim was to stress the existence, within the various entities that are encompassed by the term TE, of a distinctive clinical entity which must be recognized and somehow baptized. The disorder is the most common to be observed in the office, is characterized by a profuse and distressing shedding, and is easily distinguished simply by the history from other less common and/or less alarming TEs, like for example the postfebrile TE and the postpartum TE. It is typically, but not exclusively, complained of by a frightened lady, who claims having always had “a full head of hair which now are coming off by the handful”, can time the onset of shedding precisely, wakes up in the middle of the night thinking of her hair and, in about 30% of cases, reports (if specifically asked for) trichodynia. There is a striking association (in women) with Hashimoto thyroiditis (5 of 7 patients [10]) and less commonly with other autoimmune diseases. Nonetheless, there are also women who report having always observed a hair shedding that has gained their worrisome attention only recently. I guess that they have a telogen effluvium-androgenetic alopecia (TE-AGA) overlap. I agree with Prof. Trüeb that the latter is a challenging diagnosis, but I am relying on MWT [11], which confirms such diagnosis and assesses the relative severity of the two conditions, which is essential for a rational treatment. I tried to label this disorder inflammatory/autoimmune, but any other more appropriate title is welcome.

Disclosure Statement

The author has no conflict of interest.

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