 DOI:10.1590/S1807-59322011000800028

LETTER TO THE EDITOR

# Alopecia areata incognita: a comment

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I read the article by Dr. Molina et al. on alopecia areata incognita (AAI)[1] with great interest. As the author of the original 1987 article in which the existence of AAI was hypothesized,[2] I feel entitled to present my dissenting opinion with respect to some of statements made by Dr. Molina et al.

The case Dr. Molina et al. described and successfully cured is not a case of AAI but rather of diffuse alopecia areata (AA). Contrary to the claims of Dr. Molina et al., AA is not ''identical'' to AAI but is a different entity. Of course, the distinction between AAI and AA is a matter of minute differences that, nonetheless, should matter to dermatologists.

AAI has a typical clinical presentation: it looks like an acute telogen effluvium with an intensely positive pull test and trichodynia. There is no rarefaction of hair, unless the hair is shed for months or years, and there are rarely exclamation point hairs. More than 350 hairs are usually collected with the modified wash test (MWT),[3] and, sometimes, as many as 1000 hairs are collected. Dystrophic hairs can be observed in about 3-4% of patients and represent 2-7% of all hairs collected at each MWT.[4] This observation is possible, however, only if the hairs are collected by means of the MWT. Because the dystrophic hairs are notably rare and are scattered randomly throughout the scalp, finding them with the traditional trichogram is impossible. If patients are followed up, patches of alopecia areata are detected between the sixth and ninth week in approximately 50% of patients with dystrophic hairs at the MWT.[4] The patches are small and have no tendency to enlarge.[4]

Diffuse AA, in contrast, presents with patches of hair rarefaction, which differ from those of classical AA because of the indistinctness of their borders and their tendency to merge. Often, other areas are affected, such as the eyebrows and limbs, an occurrence that is not observed in AAI. Diffuse AA easily results in alopecia totalis in a few weeks.

Of course, AAI shares with both classical and diffuse AA some dermoscopic and histopathological features, such as the ''yellow dots'' and the telogen germinal units reported by Tosti et al.[5]

The most important difference between AAI and AA, however, involves the different ways in which hair sheds. In classical AA, hairs shed in dystrophy, and the glabrous patch is formed because a group of hairs in the same early subphase of anagen is stricken simultaneously by the anti-mitotic insult,[2] hence the neatly designed borders of the patch. In diffuse AA, hairs are shed in a mixed fashion, with both dystrophic and telogenic characteristics. In AAI, however, the hair loss is practically only telogenic, hence the absence of rarefaction or patches.

Finally, I cannot agree with the statement that AAI is more common in young people. Were this the case, AAI would not differ from classical AA, which rarely occurs after the fifth decade. One of the possible explanations for this curious epidemiological behavior is the higher prevalence of androgenetic alopecia (AGA) at that age. In other words, as I postulated in 1987, the shortening of the hair cycles in AGA may be the crucial factor that prevents the formation of dystrophy and favors the ''telogen escape'' from AA.[1]

## REFERENCES

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