Clinicodermoscopic Pattern of Beard Alopecia Areata: A Cross-Sectional Study

Sir,

We welcome the comments raised by some of the esteemed readers on the original study published in this journal titled "Clinicodermoscopic pattern of beard alopecia areata: A cross-sectional study".[1] We would like to take this opportunity to issue clarifications on some of the points raised by them.

The correspondents seem to have totally missed the points acknowledged under "Limitations of the study" including the one where the Fitzpatrick skin type of our patients was stated as belonging to type IV and V.

At the outset, we would like to clarify to the correspondents that this study aimed to document all the observable dermoscopic findings within patches of alopecia areata (AA) affecting the beard as the existing literature on the normal pigmented skin is limited. Some of the findings such as hem-like pigmentation, nonfollicular white dots, and vascular pattern are considered normal.

1. In fact, as the correspondents have pointed out, nonfollicular white dots, which they agree are surface ductal openings of eccrine glands, are indeed observed beyond the alopecic patches too in some of the patients. The presumption that we have concluded all the observed features as findings specific to the disease process of AA alone is not correct, especially when histopathological confirmation has not been carried out. We would also like to make it clear that nowhere have we claimed in the paper that nonfollicular white dots are a diagnostic finding of beard alopecia areata (BAA)
2. The interpretation of yellow dots versus white dots is mostly subjective, especially on pigmented skin. We are of the opinion that those were yellow dots.

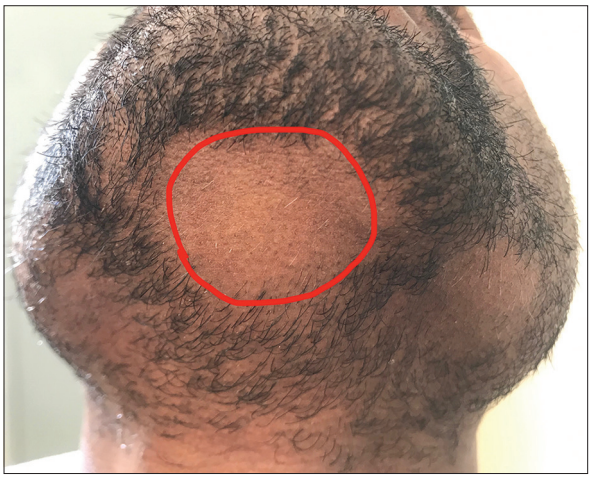

Figure 1: Beard alopecia areata under the chin. Representative lesion is encircled in red

3. The following sentence in our original study – "Peripilar sign, which was previously described only in lichen planus pigmentosus was also seen in 17.39% of our patients" should have read "Peripilar sign, which was previously described in androgenetic alopecia (AGA), was also seen in 17.39% of our patients". We sincerely regret the error, and the confusion it has caused.

The peripilar sign, also known as brown perifollicular discoloration or perifollicular hyperpigmentation, is seen in AGA, telogen effluvium, and also in healthy individuals.[2] Previously it was thought that "peripilar sign" was difficult

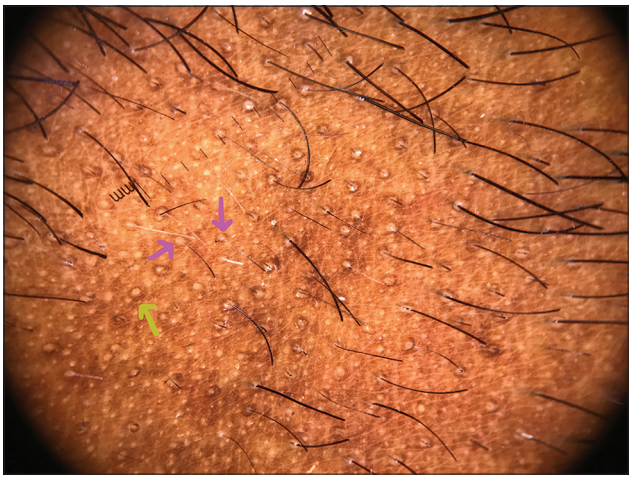

Figure 2: Dermoscopic image (×10 magnification polarized mode) from the representative lesion encircled in red in Figure 1. Note the increased perifollicular pigmentation in a tapering hair (pink arrow) and empty follicles (green arrow)

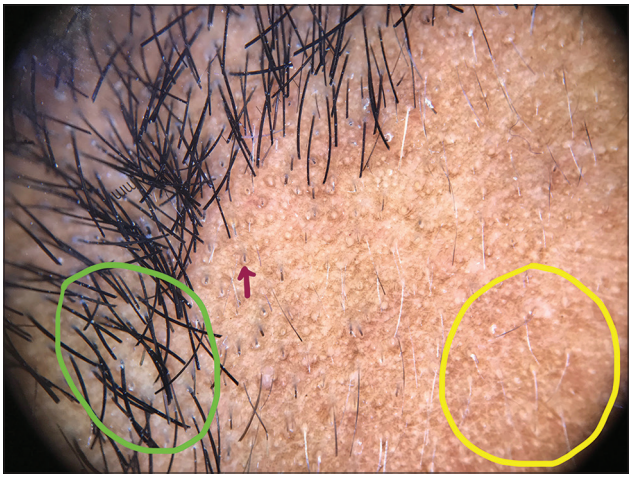

Figure 3: Dermoscopic image (×10 magnification polarized mode) from the representative lesion encircled in red in Figure 1. Note the increased perifollicular pigmentation in a black dot (maroon arrow). Such a feature is absent in the surrounding hairy margin (green circle) and the center of the alopecic patch (yellow circle)

Table 1: Dermoscopic findings based on activity

Features		Active disease	Nonactive disease	*P*
Black dots	Present	10	8	0.4
	Absent	12	16	
	Total	22	24	
Short vellus hair	Present	7	11	0.331
	Absent	15	13	
	Total	22	24	
Tapering hair	Present	3	11	0.018
	Absent	19	13	
	Total	22	24	
Regrowing hair	Present	4	10	0.084
	Absent	18	14	
	Total	22	24	
Yellow dots	Present	7	6	0.608
	Absent	15	18	
	Total	22	24	
White dots (follicular)	Present	0	9	
	Absent	22	15	
	Total	22	24	
Broken hair	Present	4	5	0.821
	Absent	18	19	
	Total	22	24	
Blotchy erythema	Present	0	8	
	Absent	22	16	
	Total	22	24	
Peripilar sign	Present	0	8	
	Absent	22	16	
	Total	22	24	
Vascular network	Present	0	3	
	Absent	22	21	
	Total	22	24	
Honeycomb pigment pattern	Present	0	2	
	Absent	22	22	
	Total	22	24	
Coudability sign	Present	0	2	
	Absent	22	22	
	Total	22	24	
I-hair	Present	0	1	
	Absent	22	23	
	Total	22	24	
Perifollicular hemorrhage	Present	0	1	
	Absent	22	23	
	Total	22	24	
Tulip hair	Present	0	1	
	Absent	22	23	
	Total	22	24	

to visualize in Indian skin, but a recent article from South India has reported peripilar sign in 89.4% of male patients of AGA and 40% of women with female pattern hair loss.[3]"Peripilar sign" corresponds to the perifollicular presence of inflammatory infiltrate. As AA is a disease characterized by peribulbar lymphocytic infiltration, the peripilar sign (annoted by purple arrow in Figure 4 of original published article)[1] is possibly due to this very same reason! The scalp and the beard hair not only differ morphologically and chemically, but there also exists differences in their dermal papilla, specific biomarkers, and response to androgens.[4-6] This combined effect may be the reason for the thinning of hair shaft, and their eventual loss from the follicles where this sign was observed.[7,8]

We also bring to the notice of the readers that findings classically noted in sun-exposed scalp cannot all be extrapolated to the beard region. Case in point, the area under the chin which is anatomically spared in photo distributed dermatitis. This is illustrated in the following images sourced from the same patient. [Figures 1-3].

Therefore, in view of 1) lack of similar findings around the follicles at the center of the alopecic patch and 2) lack of histopathological confirmation, we disagree with the correspondents that the perifollicular pigmentation is secondary to prolonged sunlight exposure.

4. We would like to reiterate again that the "hem-like" dermoscopic morphology described in our study has not been projected as a feature specific to BAA alone.
5. For the benefit of readers, Table 1 provides the dermoscopic findings based on activity.

No doubt dermoscope is a handy tool in our day-to-day practice but the gold standard for diagnosis of AA remains histopathology. Dermoscopy at best remains corroborative. Thus, in the absence of large-scale studies from multiple researchers from different parts of the world that conclusively attributes each and every dermoscopic finding to corresponding presence or absence of underlying histopathological features, it is detrimental to have fixed ideas.

Medical science is continuously advancing. What is established as standard practice today, may not necessarily be so in the future. The basis of understanding of a disease entity evolves over time only when we constantly seek to update the existing knowledge database. We sincerely hope our article stimulates others to take up large-scale studies on this and add to the existing literature.

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Conflicts of interest

There are no conflicts of interest.

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