Sir, in the previous issue of the Indian Journal of Dermatopathology and diagnostic dermatology, Joshi[1] discusses the usefulness of semiquantitatively assessing dermal melanophages, in inflammatory skin diseases. The proposed scale showed a high concordance between melanophage scores and lichen inflammatory diseases that present with hyperpigmentation clinically.

Dermal melanophages are ubiquitous, present in a number of inflammatory and noninflammatory dermatoses. Dermal pigment incontinence is a result of effete keratinocytes or pigment-laden melanocytes (either naturally senescent or destroyed as a result of inflammation), dropping down into the papillary dermis. Subsequent mopping up of the melanin by dermal macrophages results in melanophages. The identification of the occasional melanophage in the dermis is nondiagnostic, as they represent mopping up of incidental senescent epidermal keratinocytes or melanocytes. A number of inflammatory dermatoses (including psoriasiform and spongiotic) result in dermal melanophages, though this is not well documented. This paper is the first to have attempted to confirm and quantify the presence of melanophages in diseases traditionally not associated with clinical pigmentation[1] (such as psoriasis, etc.). It is however common knowledge that psoriasis in Indian skin is usually pigmented.

Melanophages can be cited in both hypo as well as hyperpigmentary disorders though the identification of melanophages as such does not allow one to postulate on the nature of the clinical presentation. There is a trend apparent from this paper that in general hypopigmentary diseases have low melanophage scores when compared to the others. Although it is well known, this is the first study to quantify the presence of melanophages in hypopigmented dermatoses. This phenomenon may be specific to dark-skinned races. It would be interesting to do a blinded study, where melanophage scores are compared to clinical pigmentation scores.

It is not uncommon to find that a single disease (say cutaneous lupus, inflammatory vitiligo, or lichen sclerosus) may sometimes present with both hypo as well as hyperpigmented lesions (occasionally, a single patch displaying both variants), indistinguishable histologically.[2] A number of reasons can be postulated; however, no one has specifically studied this in detail. Such a phenomenon may either be due to the fact that melanocytes may be the primary target (vitiligo) or that the melanocytes are destroyed before the transfer of melanin to keratinocytes has taken place. Whether melanophages are qualitatively and quantitatively different in pigmentary variants of the same disease is a question that can only be answered by histopathologically scoring these pigmentary variants.

The authors in this study have set out to find the answer to an interesting question – Can a melanophage scoring system be used to predict the nature of an inflammatory disease? Unsurprisingly, high melanophage scores were observed in lichenoid dermatoses of the lichen planus spectrum. It is well known that the phenomenon of pigment incontinence is exaggerated in interface dermatoses, where melanophages are usually found in abundance. Interface dermatoses usually result in clinical hyperpigmentation (lichen planus, etc.), probably as a result of excitation of melanogenesis due to the inflammatory milieu within the interface; and subsequent destruction of the basal layer resulting in melanin incontinence. It would have been interesting to combine melanophage scores with a clinical pigmentation score like what is used in melasma area and severity index or melasma severity index scoring systems for melasma.[3] This may have helped correlate their findings better. A number of other questions also need answers – How does the presence of inflammation affect clinical pigmentation scores, in cases where melanophage scores are alike? Is the melanophage score affected by the duration/stage of disease, place of biopsy (margins vs. central), recent treatment, etc.? Future studies need to take these factors into consideration.

One of the main drawbacks of this study is that the scoring system has not been validated, and interobserver reliability has not been tested. Further, melanophage score by itself has no diagnostic utility, as the diagnosis of hyperpigmented lichenoid dermatoses is not made on the basis of finding or not finding melanophages, but on the basis of finding a lichenoid infiltrate. One of the main issues that troubles the dermatopathologist is where late stage (non/postinflammatory) lesions are sent for biopsy and a specific diagnosis (e.g.,: melasma vs. ashy dermatoses/lichen planus pigmentosus) may not be possible. Future studies may be able to answer this question in light of the melanophage scores presented in this study, particularly the sensitivity and specificity of melanophage scores in this scenario and whether one may be able to make a diagnosis by quantitatively and qualitatively assessing the melanophages alone!

Further, some of the conditions presented were represented by only 1–2 biopsies making it difficult to generalize the results. Since this retrospective study only picked up cases, where the dermatopathologist “mentioned” melanophages in the report; by default, only cases with “significant” (enough to worth a mention) melanophage counts were studied. This would have led to significant bias in case selection. A study...
of 100 consecutive inflammatory biopsies, looking for melanophage scores within and correlation with the clinical diagnoses would have been more interesting. Quantification of melanophages though interesting as a research tool may not help in the diagnosis of diseases, as this largely depends on identifying the nature of the inflammation.

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Conflicts of interest
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