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LETTER TO THE EDITOR

Seborrheic macular hypopigmentation: a case series proposing a new pigmentary disorder

Editor

In 2014, a published case series highlighting a unique presentation of patterned hypopigmentation introduced an entity coined *hypochromic vitiligo*. This was described as near symmetric, scattered hypopigmented macules in a seborrheic distribution in patients with darker skin types that may be a form of vitiligo.¹ This patterned presentation of hypopigmentation may have also appeared in the literature under a different name, *vitiligo minor*, which was described as scattered, hypopigmented oval-shaped macules and patches in darker skinned individuals.²

We have similarly identified a cohort of 14 patients, all Black and majority middle-aged, with an identical presentation of hypopigmented macules and patches in a seborrheic distribution affecting the scalp, nose, face, chest, and back. We feel this condition is an under-recognized, difficult-to-treat, and distressing disorder of hypopigmentation in skin of colour patients without a clear understanding of the aetiology and pathogenesis (Figures 1 and 2).

To better characterize this disorder, we performed a multicentric retrospective chart review involving the Departments of Dermatology at New York University, the Henri Mondor University Hospital, and the Henry Ford Hospital. Fourteen patients with the unique clinical phenotype of hypopigmented macules and small patches in a seborrheic distribution were identified and included. Eleven patients were male. The average age at the time of consultation was 51 years old (range: 29–73 years old) and the disease was present on average 9.15 years at the time of the first visit. All patients were Black with Fitzpatrick skin types IV–VI. Hypopigmented macules and patches were on the face, neck, scalp, upper chest, and/or upper back in a seborrheic distribution. Thirteen of the 14 patients were asymptomatic. One endorsed a history of autoimmune disease, specifically autoimmune thyroiditis, and no patients had a personal or family history of vitiligo. Response to treatment, regardless of modality, was poor. Topical treatments included calcineurin inhibitors, glucocorticoids, antifungals, and antibiotics. Some patients also underwent narrow-band ultraviolet B (nbUVB). No patients achieved complete resolution with any treatment.

Histopathological specimens of affected areas were obtained for all 14 patients. When available, tissue was stained for haematoxylin and eosin (H&E), Periodic-acid Schiff (PAS), Fontana-Masson/Melan-A, microphthalmia transcription factor (MITF), and/or SOX-10. All specimens stained with PAS were negative.

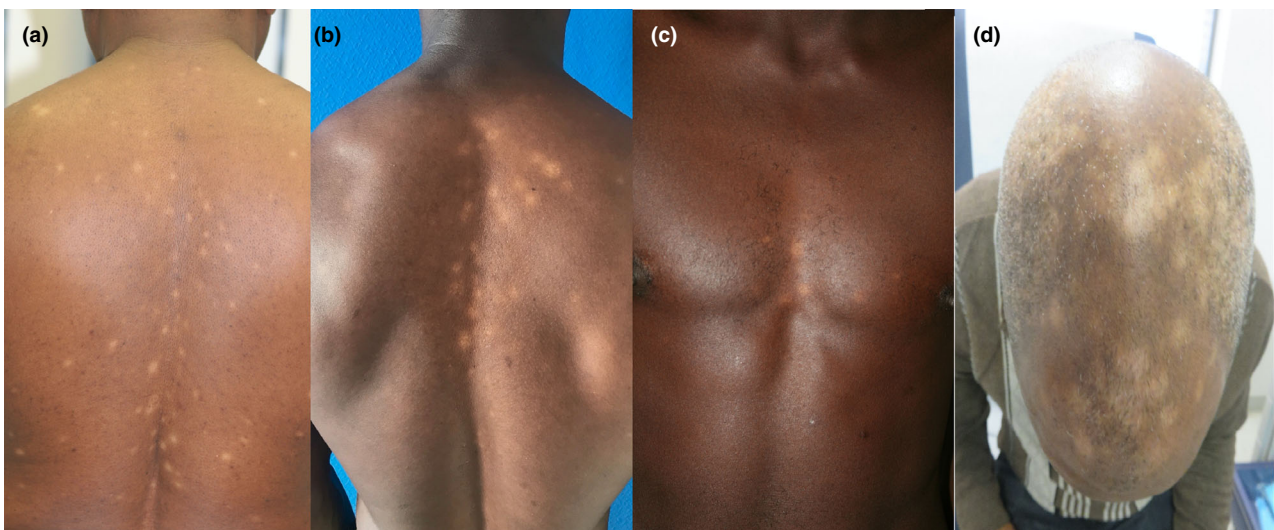


Figure 1 (a–d) Hypopigmented patches in a seborrheic distribution along the trunk (a–c), and scalp (d) demonstrated in four Fitzpatrick skin type IV or higher individuals.

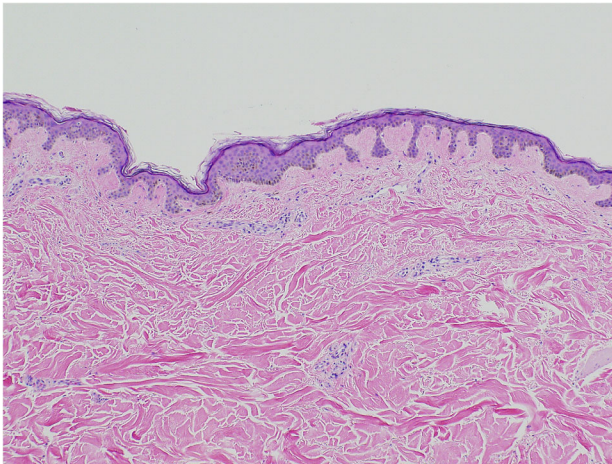


Figure 2 Haematoxylin and eosin stain of lesional (hypopigmented) skin at 10x demonstrating sparse superficial perivascular infiltrate with melanophages in the papillary dermis. Melanocytes are visualized at the basal layer, without evidence of infiltrate at the dermal–epidermal junction (DEJ).

Fite staining was performed in one sample and was negative. Biopsies involving both affected and unaffected skin were performed in 11/14 cases. All 11 specimens stained with H&E and/or MITF demonstrated, to some degree, the presence of melanocytes. Six lesional samples stained with Fontana Mason demonstrated scattered dermal melanophages. SOX-10 performed on three scalp biopsies revealed intact melanocytes along the dermo–epidermal interface. These features, including the retained presence of melanocytes and the presence of dermal melanophages suggests against the diagnosis of vitiligo. Other disorders of hypopigmentation were considered, including those of inflammatory, infectious, or neoplastic aetiologies. Proposed differential diagnoses were effectively deemed less likely based on clinical presentation, lack of treatment response, and/or histopathological presentation.

Potential aetiologies for this condition include melanocyte senescence or low-grade inflammation of unknown aetiology leading to loss of pigmentation. Involvement of photodistributed areas of the face and scalp indicate that sun exposure and actinic damage may play a role as well. Lastly, the occurrence of this condition exclusively in patients of colour, particularly Black individuals, suggests genetic factors and pigmentary differences may also play a role.

We propose this entity be renamed *Seborrheic Patterned Macular Hypopigmentation (SPMH)*. This study is limited in its retrospective nature and size. Larger studies with additional diagnostic testing including electron microscopy are needed to better evaluate this condition. We hope this case series will promote further recognition and understanding of this condition, including the discovery of its aetiology and effective treatment options.

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The patients in this manuscript have given written informed consent to publication of their case details.

Conflicts of interest

The authors of this manuscript have no financial or personal conflicts of interest to report.

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Data availability statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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