



Acral speckled hypomelanosis: Is there an autoimmune origin?

Akral benekli hipomelanoz: otoimmün bir köken var mı?

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To Editor,

Acral speckled hypomelanosis, a relatively new entity, is characterized by confetti-like, speckled hypopigmented macules on the dorsal or lateral parts of acral regions that usually arise in childhood¹. The etiopathogenesis of the disease is unknown. We herein report a case of acral speckled hypomelanosis and elevated thyroid autoantibodies.

A 15-year-old boy presented with a one-month history of multiple asymptomatic white dots on the dorsum of his hands and feet. He was otherwise healthy and had no systemic or cutaneous disease. He did not report any inflammatory dermatoses before the pigmentation change in the lesional areas. There was no history of drug intake, chemical exposure, or phototherapy. There was no family history of similar lesions. The mother had psoriasis, and the grandmother had vitiligo.

Dermatological examination revealed multiple symmetrically distributed confetti-like hypopigmented macules on the dorsum of both hands and feet with a normal-colored background skin without atrophy. The lesions were more intense on the distal parts, especially over the fingers,

and in a speckled pattern. The margins of the hands were also affected. We did not observe any accentuation of lesions under a Wood lamp (Figures 1, 2). Other systemic evaluations found the nails, mucosal membranes, and the other cutaneous regions to be normal.

A blood evaluation showed elevated thyroid autoantibodies [anti-thyroid peroxidase: 274 IU/mL (reference: 0-26) and anti-thyroglobulin: 230 IU/mL (reference: 0-64)]. Thyroid functional tests [thyroid stimulating hormone, serum triiodothyronine (sT3), and sT4] were within normal limits. Complete blood count, renal and liver function tests, serum glucose, erythrocyte sedimentation rate, C reactive protein, vitamin B12, and folate were all within normal range.

Histopathological examination of the lesional skin and immunohistochemical staining with S100 and HMB45 revealed a reduction in the number of melanocytes and reduced melanin pigment compared to the normal perilesional area. There were sparse perivascular mononuclear inflammatory infiltrates in the dermis (Figure 3).

Acral speckled hypomelanosis is characterized by multiple, confetti-like, asymptomatic hypopigmented macules with a

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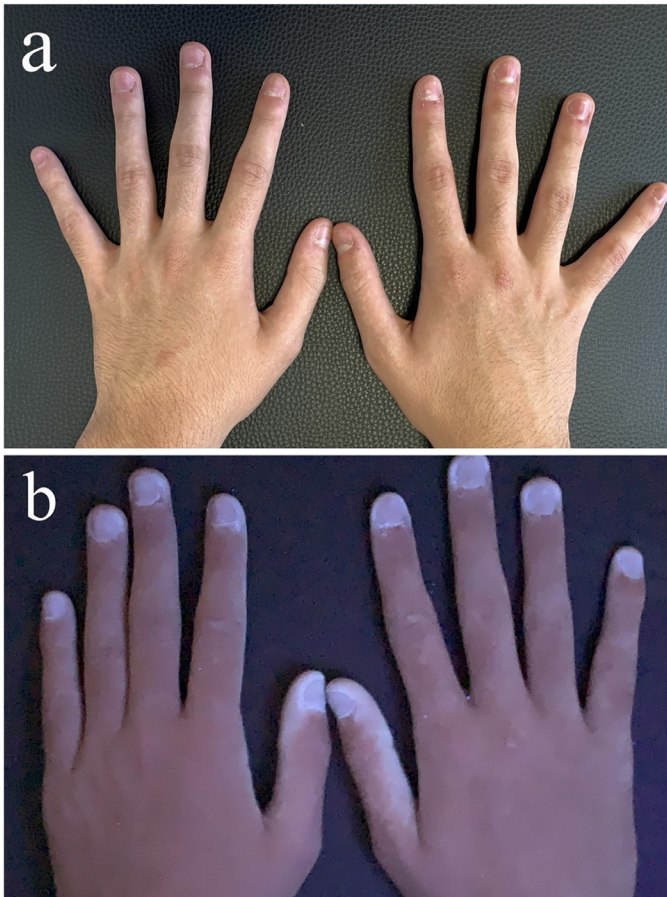


Figure 1. Multiple, symmetrically distributed, hypopigmented guttate macules on the dorsum of both hands (a), especially on the fingers. Wood light reveals no accentuation of hypopigmented lesions (b)

speckled or reticulated appearance primarily on the dorsal or lateral parts of the hands and feet. The background skin is of normal color. Wood light reveals no accentuation of hypopigmented lesions. Ten cases (6 females and 4 males), whose ages range between 9-25 years, have been reported since their first description¹⁻⁴.

Differential diagnosis of acral speckled hypomelanosis includes various disorders¹. The differentiating features of acral speckled hypomelanosis are the distribution of hypopigmented, non-atrophic, guttate macules over the dorsum of both hands and feet without accentuation under a Wood lamp, the absence of concurrent hyperpigmented lesions, no lesions on other mucocutaneous regions, normal skin appendages, no accompanying systemic disorders, no family history of similar lesions, and a reduced or normal number of melanocytes, unlike the reduced number seen in vitiligo.

The etiopathogenesis of the disease is unknown. Of the reported 10 cases, family history is present in only two¹. Because most cases present in early adolescence, mutations in pigmentary genes, and dysregulations in pigment production or distribution, may have a role in the pathogenesis³.

The association of vitiligo with autoimmune disorders, such as type 1 diabetes, psoriasis, pernicious anemia, and especially autoimmune thyroid disorders, is well known⁵. Among 10 cases with acral speckled hypomelanosis, accompanying systemic disorders (type 1 diabetes and congenital dyserythropoietic anemia type 2) were reported in only one

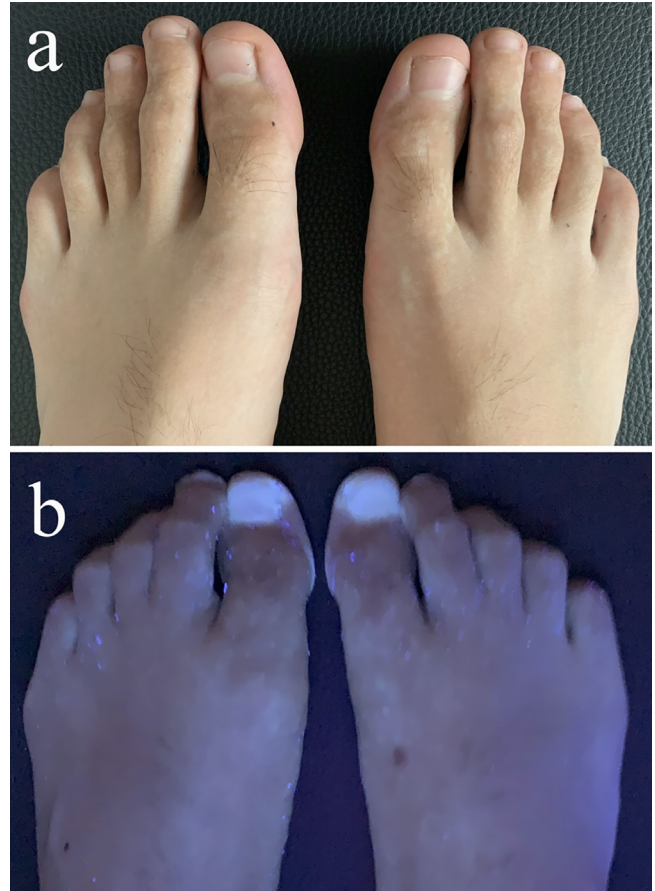


Figure 2. Multiple, symmetrically distributed, hypopigmented guttate macules on the dorsum of both feet (a), especially on the fingers. Wood light reveals no accentuation of hypopigmented lesions (b)

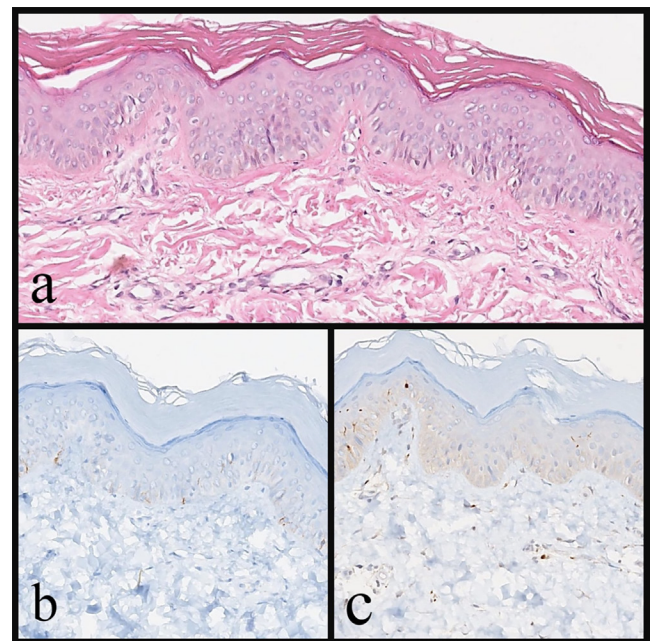


Figure 3. Histopathological sections of lesional skin show a reduced number of melanocytes and melanin pigment compared to the normal perilesional area. (a) Hematoxylin and eosin, x200; (b) HMB45, x200; (c) S100, x200

case². However, the previous reports did not include any laboratory data. Our case had elevated thyroid autoantibody levels as well as a family history of psoriasis in his mother and vitiligo in his grandmother, respectively. Thus, autoimmunity may also be an additive factor in the pathogenesis of acral speckled hypomelanosis, besides genetics.

Topical treatments, including corticosteroids, tacrolimus, and calcipotriol, were reported as unsuccessful^{3,4}. In one patient, six months of narrowband ultraviolet B treatment resulted in significant improvement. However, a relapse occurred within a few weeks after the cessation of the phototherapy⁴.

Because of the age at the onset of lesions, clinical and histopathological features, absence of systemic or cutaneous disorders, no history of chemical exposure or drug history, and no family history of similar lesions, other diseases in the differential diagnosis¹, including idiopathic guttate hypomelanosis, idiopathic confetti-like leukoderma, leukoderma punctata, lichen sclerosus, vitiligo, symmetric acroleukopathy, Darier disease, Cole disease, tuberous sclerosis complex, progressive macular hypomelanosis, familial white lentiginosis, acropigmentation of Dohi, acromelanosis albopunctata, and dyschromatosis universalis hereditaria, were excluded, and the patient was diagnosed with acral speckled hypomelanosis. He was treated with topical tacrolimus 0.1% ointment for 5 months, yet no improvement was observed, and the condition remained stable.

The exact etiopathogenesis of acral speckled hypomelanosis is not known. Our case had elevated thyroid autoantibodies, as well as a family history of psoriasis and vitiligo suggesting a pathogenetic role of autoimmunity. Further genetic and immunological studies are needed.

Ethics

Informed Consent: The patient discussed above gave consent for their photographs and medical information to be published in print and online and with the understanding that this information may be publicly available.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Ö.Z., S.K., Concept: Ö.Z., S.K., Design: Ö.Z., S.K., Data Collection or Processing: Ö.Z., S.K., Analysis or Interpretation: Ö.Z., S.K., Literature Search: Ö.Z., S.K., Writing: Ö.Z., S.K.

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