Segmental vitiligo and twenty-nail dystrophy: An unusual association

Sir,

Localized depigmented patches in a dermatomal distribution that do not cross the midline are called segmental vitiligo. The course of the segmental type tends to be earlier in onset and more stable than generalized vitiligo and is not familial.

Twenty-nail dystrophy (TND) presents as rough surfaced nail plates and involving up to 20 nails. Two types of nail changes have been described. In the first type, the entire nail appears to have been sandpapered (sandpaper nails) in a longitudinal direction and shows excessive ridging and roughness. In the second type, the nail plate is shiny (shiny nails). Here we report a case of an unusual association of segmental vitiligo and twenty-nail dystrophy.

A male patient aged 20 years presented with depigmented skin lesions since 5 years and also with nail changes of 2 years’ duration.

On cutaneous examination, localized, multiple, round-to-oval–shaped, achromic macules were seen in a segmental distribution over the abdomen (corresponding to T9,10 dermatome). Examination of the nails revealed longitudinal ridging and roughness over the nail plates (sandpaper nails) in all the nails. Histopathology of an achromic macule showed marked absence of melanin granules, and Wood’s lamp examination revealed amelanotic macules. Nail biopsy showed spongiosis in the nail matrix with mononuclear inflammatory infiltrate.

Association of cutaneous and systemic autoimmune diseases with vitiligo occurs more significantly in the non-segmental type than in the segmental type. Though rarely, segmental vitiligo has been reported in association with a few skin disorders like poliosis, halo nevus, nevoid basal cell carcinoma syndrome, Parry-Romberg syndrome and linear scleroderma.

The association of vitiligo and TND is very rare and has been sparsely reported and vitiligo in most of such associations was of non-segmental type, like generalized vitiligo, scalp vitiligo (localized), and acrofacial vitiligo. However, association of segmental vitiligo with TND has been described in 2 patients.

The association of segmental vitiligo with TND in the present case can be explained by the autoimmune origin of these disorders. Although the etiology of segmental vitiligo is based primarily on the neurogenic theory of melanocyte destruction, an immune mechanism cannot be completely ruled out, because autoimmune disorders have been described in approximately 3.4% to 9.5% of cases of segmental vitiligo. Further, systemic and topical steroids and psoralen with ultraviolet A (PUVA) therapy have shown encouraging responses in early lesions. The strong association of TND with dermatoses which have autoimmune etiopathogenesis has led some to speculate that the nail changes are primarily due to an autoimmune process. Thus our case emphasizes a common autoimmune insult to the melanocytes and nail matrix as the logical explanation for this rare association.

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Letters to Editor