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Becker's nevus and lichen planus: A rare coexistence

Femina Azeez[1], Kunjumani Sobhanakumari[2], Meriya Zacharia[1], Seena Palakkal[1], Kiran Shaj[1]

[1]Department of Dermatology and Venereology, Government Medical College, Kottayam, [2]Department of Dermatology, Al Azhar Medical College and Super Specialty Hospital, Thodupuzha, Kerala, India.

***Corresponding author:**
Femina Azeez,
Department of Dermatology and Venereology, Government Medical College, Kottayam, Kerala, India.

femina.azeez@gmail.com

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ABSTRACT

Becker's nevus, an epidermal nevus, may occur alone or may be associated with musculoskeletal anomalies. There are reports of certain inflammatory and neoplastic diseases showing a predilection for Becker's nevus. We report a patient who manifested lesions of lichen planus. Interestingly, the lesions showed a predilection for a cutaneous area that harbored a Becker's nevus. We reviewed similar cases where a Becker's nevus was reported as a site of predilection for other dermatoses and suggest that the possibility of the former acting as an immunocompromised district of Ruocco (an area that shows less resistance to a disease process in comparison to other body areas) may be considered.

Keywords: Becker's nevus, Lichen planus, Immunocompromised district of Ruocco, Site of predilection, Resistance

INTRODUCTION

Becker's nevus is an epidermal nevus that usually manifests in adolescence, though an earlier onset has also been reported.[1-3] There are occasional reports of inflammatory or neoplastic dermatoses that remain confined to a Becker's nevus or show a predilection for a cutaneous area harboring a Becker's nevus.[1-9] Various authors have put forward different explanations for the same.[1-9] We report a patient in whom the lesions of lichen planus predominantly affected the skin harboring a Becker's nevus.

CASE REPORT

A 38-year-old male presented with multiple, pruritic lesions distributed over the limbs and the back of trunk of 3 months duration. He did not give any history of drug intake prior to the onset of these lesions. He had no other comorbidities or skin lesions, except for a well-defined, hyperpigmented patch with irregular borders and hypertrichosis that involved the posterior aspect of the right shoulder and the right upper arm. The hyperpigmented patch started as spotty, hyperpigmented macules in adolescence. The spotty macules gradually increased in size and number over a few years and coalesced to reach the current size and remained asymptomatic.

The patient denied any history of trauma, excessive sun exposure, vaccination, or cutaneous viral infection anywhere on the hyperpigmented skin lesion.

Clinical examination showed multiple, violaceous, papules, and plaques (ranging from 0.5 × 0.5 cm to 4 × 3 cm) distributed over the right shoulder and upper arm, both forearms

[Figure 1a], dorsal aspects of hands, buttocks, thighs, legs, and feet [Figure 1b] with koebnerization. Lesions were more prominent over the hyperpigmented patch [Figure 2]. Oral mucosa showed white and reticulate lesions. Nails were normal. Systemic examination was unremarkable.

We made a clinical diagnosis of lichen planus with Becker's nevus. Incision biopsies were taken from a violaceous papule overlying the hyperpigmented skin lesion and also from an area on the hyperpigmented patch devoid of violaceous papules. Skeletal radiography of the right upper arm and shoulder did not reveal any bony abnormality. The patient was treated with emollient (liquid paraffin) and cetirizine 10 mg per orally once a day as we waited for the histopathological confirmation.

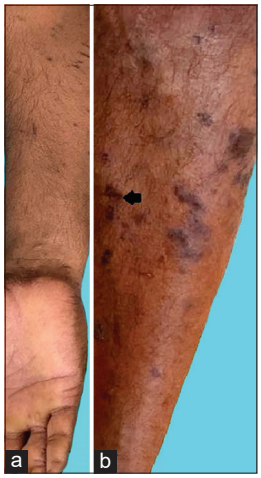

Figure 1 (a): Violaceous, flat topped papules of lichen planus on the flexor aspect of forearm; (b): Violaceous, flat topped papules and plaques (black arrow) of lichen planus on the leg of the same patient.

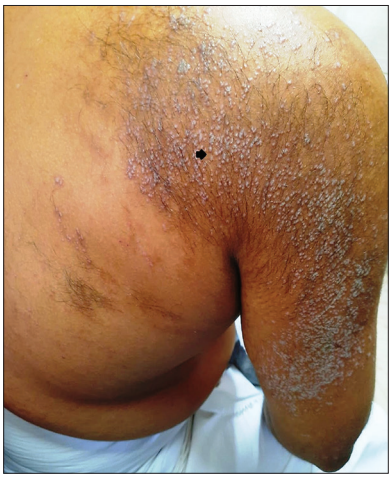

Figure 2: Violaceous, flat topped papules (black arrow) of lichen planus overlying the Becker's nevus.

Biopsy from a violaceous papule that overlied the hyperpigmented patch showed hyperkeratosis, wedge-shaped hypergranulosis, basal cell degeneration, and band-like lymphocytic inflammatory infiltrate along the basement membrane zone [Figure 3a], confirming the diagnosis of lichen planus. Biopsy from the hyperpigmented patch without overlying violaceous papules revealed hyperpigmentation of basal layer and melanophages in dermis consistent with the clinical diagnosis [Figure 3b] of Becker's nevus. We made a final diagnosis of lichen planus with Becker's nevus.

When the patient returned for the histopathology report 2 weeks later, we found that the lesions of lichen planus which were superimposed on the Becker's nevus had already subsided (within 2 weeks of biopsy), without any specific treatment [Figure 4]; but the lesions on the other body sites persisted. The patient was advised topical clobetasol propionate ointment 0.5% and continuation of cetirizine, for lichen planus on other body sites.

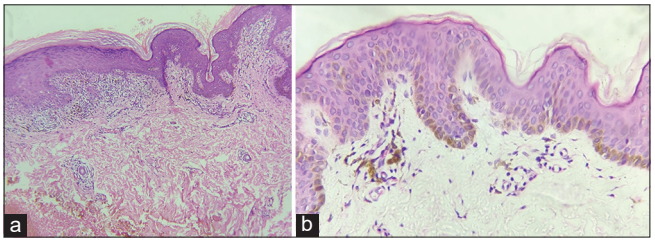

Figure 3 (a): Skin biopsy of a violaceous papule overlying the Becker's nevus showing hyperkeratosis, wedge-shaped hypergranulosis, basal cell degeneration, and band-like inflammatory infiltrate in dermoepidermal junction (H and E, ×100); (b): Skin biopsy from the Becker's nevus on the posterior aspect of the right shoulder and upper arm (biopsy was taken from a site devoid of lesions suggestive of lichen planus) showing hyperpigmentation of basal layer and melanophages in dermis (H and E, ×400).

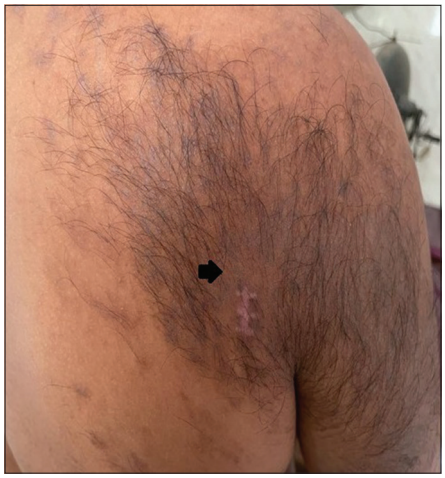

Figure 4: Complete clearance of lesions of lichen planus superimposed on the Becker's nevus within 2 weeks of biopsy (black arrow).

DISCUSSION

Becker's nevus, an epidermal nevus, manifests as an acquired hyperpigmentation (often accompanied by hypertrichosis) and usually appears in adolescence.[1-3] Rarely, it may manifest at birth or in the initial few years of life.[3] Male predilection, appearance in puberty, and accompanying hypertrichosis, and acneiform lesions suggest a causative role for androgens, which was supported by the observed increase in androgen receptors in the nevus.[10] Many associations have been reported for the nevus such as smooth muscle hamartomas, hypoplasia of the pectoral muscles and breast, limb hypertrophy, and scoliosis.[3]

Lichen planus is considered as an autoimmune and T-cell-mediated disease. The characteristic lesions are produced by the interface dermatitis evoked by the cytotoxic CD8+ T-cells, which may involve the skin, mucosae, or both.[11]

Terheyden *et al.* had described a patient, who had lichen planus lesions restricted to a Becker's nevus and suggested the need to evaluate the existence of a specific cellular interaction between the two.[2]

Puri *et al.* reported an 11-year-old boy, who manifested coexistence of Becker's nevus and lichen planus.[3] The Becker's nevus had an early onset from birth and the lesions of lichen planus first appeared within the nevus and then extended to involve other body sites. The authors proposed that the keratinocyte hyperplasia and the increased number of epidermal CD1a+ dendritic cells in Becker's nevus could elicit a cytotoxic T-cell response, leading to the evolution of lichen planus within the nevus. The subsequent dissemination to other body sites was attributed to koebnerization.[3]

Reviewing the literature, we came across reports of certain other dermatoses within the lesions of Becker's nevus, such as pityriasis versicolor, morphea, granuloma annulare, Bowen's disease, basal cell carcinoma, and malignant melanoma.[1,4-9] Different authors have proposed different mechanisms for the coexistence of these lesions.

We suggest that "Ruocco's immunocompromised district model" could probably explain the preference shown by the lesions of lichen planus for Becker's nevus.[12] Ruocco *et al.* defined "immunocompromised district in dermatology as an area of skin that shows local dysregulation of immune control, which often favors (but at times hinders) the local onset of immunity-related eruptions or skin disorders."[12] The terms "locus minoris resistentiae" and "locus maioris resistentiae" were used, respectively, to refer to a site of body that showed less and more resistance to the onset of a new disease.[12]

The authors had mentioned certain predisposing factors which can lead to the formation of cutaneous areas with immune dysregulation and they included vaccination, minor injuries, thermal burns, radiation dermatitis, dermatome infection by herpes zoster, chronic lymph stasis, and skin mosaicism.[12] The absence of precipitating factors such as a history of physical trauma, chronic lymph stasis, and infections involving the site of nevus in our patient points to the possibility of Becker's nevus itself acting as an area of "immunocompromised district." The pathomechanism that underlies the probable immune dysregulation in a Becker's nevus needs further analysis. A cutaneous mosaicism could be the probable explanation. Weinberg *et al.,* while describing a 25-year-old man who presented with granuloma annulare restricted to a Becker's nevus, suggested the presence of mosaicism within an individual as a possible explanation, for the restriction of an inflammatory process (granuloma annulare) to the area of a Becker's nevus.[4] The authors drew attention to a previous report of Becker's nevus associated with chromosomal mosaicism and congenital adrenal hyperplasia.[13] Afsar *et al.* described a young girl who manifested Becker's nevus at the age of 12 years in close proximity to three macules of nevus depigmentosus which were present since birth.[14] The authors proposed "allelic twin spotting" as the genetic mechanism to explain this unusual coexistence.[14]

However, the rapid clearance of the lesions of lichen planus (following biopsy), those were superimposed on the Becker's nevus, cannot be explained by the "immunocompromised district model" since "locus maioris resistentiae" denotes resistance to the onset of a new disease, and not to the clearance of pre-existing lesions following trauma.[12] The clearance of lichen planus lesions from the biopsied area could possibly be a reverse Koebner phenomenon, the exact mechanism of which remains unclear.[15] Whether reverse koebnerization itself is a 'reflection of trauma to a particular body site resulting in reversal of a pre-existing immune dysregulation' remains unclear.

CONCLUSION

There are reports of inflammatory, infective, and neoplastic dermatoses, which remain confined to or preferentially affect a Becker's nevus. We suggest that the possibility of a Becker's nevus acting as an "immunocompromised district of skin" that favors the onset of a new disease needs further analysis.[12]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

Dr. Kunjumani Sobhanakumari is on the editorial board of the Journal.

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