Received: 3 September 2020 | Revised: 9 November 2020 | Accepted: 23 November 2020

DOI: 10.1111/dth.14597

DERMATOLOGIC THERAPY WILEY

LETTER

Lichen planopilaris restricted to the vitiliginous skin on the leg

Dear Editor,

Lichen planus (LP) and vitiligo are inflammatory skin disorders caused by the autoimmune response. Lichen planopilaris (LPP) is subtype of LP, which may cause scarring alopecia by permanently destroying hair follicles. To our knowledge, the co-localization of LPP and vitiligo has not been reported in the literature.

A 26-year-old man presented to the Dermatology Department with erythema and localized hair loss on a depigmented large macule on his right leg (Figure 1A). He was diagnosed with vitiligo 3 years ago. The periorificial areas on the face, extensor surfaces of lower limbs, upper limbs, and the axillary regions were symmetrically involved (Figure 1B). Wood's light examination showed the reflection of the well-defined borders of depigmented macules. The patient had used antioxidants and topical corticosteroids to treat vitiligo, previously. After a few months, he discontinued the application of topicals because of lack of response.

Skin biopsy specimen from the erythematous area with hair loss and depigmentation was obtained. Histopathologically, lesion displayed perifollicular lichenoid infiltration and widening of the hair follicle orifices accompanied by hydropic degeneration and dense perifollicular infiltration of lymphocytes and histiocytes and sparse apoptotic cells in the basal cell layer of the follicular epithelium. EVG stain showed partial loss and fragmentation of elastic fibers in the reticular dermis. Loss of pigmentation and loss of melanocytes were seen with SOX-10 and Masson Fontana stains (Figure 2). The patient was diagnosed with LPP in vitiliginous skin based on clinical and histopathological findings.

The term "immunocompromised cutaneous district" was introduced to explain the occurrence of new dermatoses on previously damaged vulnerable skin sites.[1] Any obstacle changing the natural trafficking of immune cells either by the lymphatic obstruction or by disruption of chemokines/neurotransmitters may cause local immune aberrations. In the previous reports of lichen planus (LP) and vitiligo associations, vitiligo precedes the LP lesions (3-10 years) suggesting the immune dysregulations in vitiligo may provide a suitable environment for the development of LP.[2-6] Lichen planus arising on vitiliginous areas reported in a mother and daughter indicate a strong genetic susceptibility for the two diseases.[7]

Vitiligo is a multifactorial disorder associated with genetic susceptibility as well as autoimmune alterations of the cellular and humoral immune response. The role of cellular immunity is supported by the infiltration of mainly $CD8^+$ T cells in perilesional dermo-epidermal junction. This infiltration results in both melanocyte and keratinocyte apoptosis. Experimental depletion of $CD8^+$ T cells prevent melanocyte destruction supporting the role of cytotoxic T cells.[8] Upregulation of INF- γ-induced genes and the subsequent increased expression of the T-cell chemokine receptor CXCR3 and its ligands (CXCL9, CXCL10, and CXCL11) and high levels of TNF-α, IL-10, and IL-17 vitiligo[9] act in synergy to maintain the immunological alterations.

Lichen planus is a T-cell mediated autoimmune dermatosis in which the major insult is by Fas ligand expressing $CD8^+$ T cells to the basal keratinocytes. In LPP, a significant elevation in the number of $CD8^+$ T cells in the infundibulum, bulge epithelium, and the perifollicular mesenchymal area have been reported in patients with LPP.[7] In LPP, autoreactive lymphocytes are attacking the follicular antigens. The development of LP on vitiligo lesions may potentially be explained by acquired immunological cross-reactivity of $CD8^+$ T cells. $CD8^+$ T cells which are autoreactive to melanocytes in vitiligo may undergo epitope spreading and become cross-reactive with self-

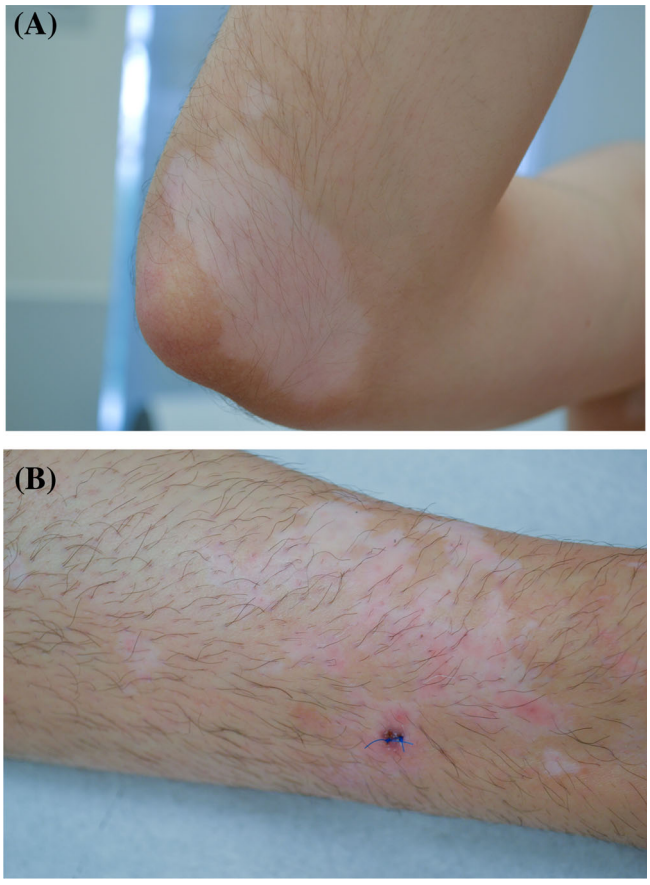

FIGURE 1 A, depigmented vitiligo macule on the elbow; B, areas of alopecia and erythematous macules on a depigmented area on the leg

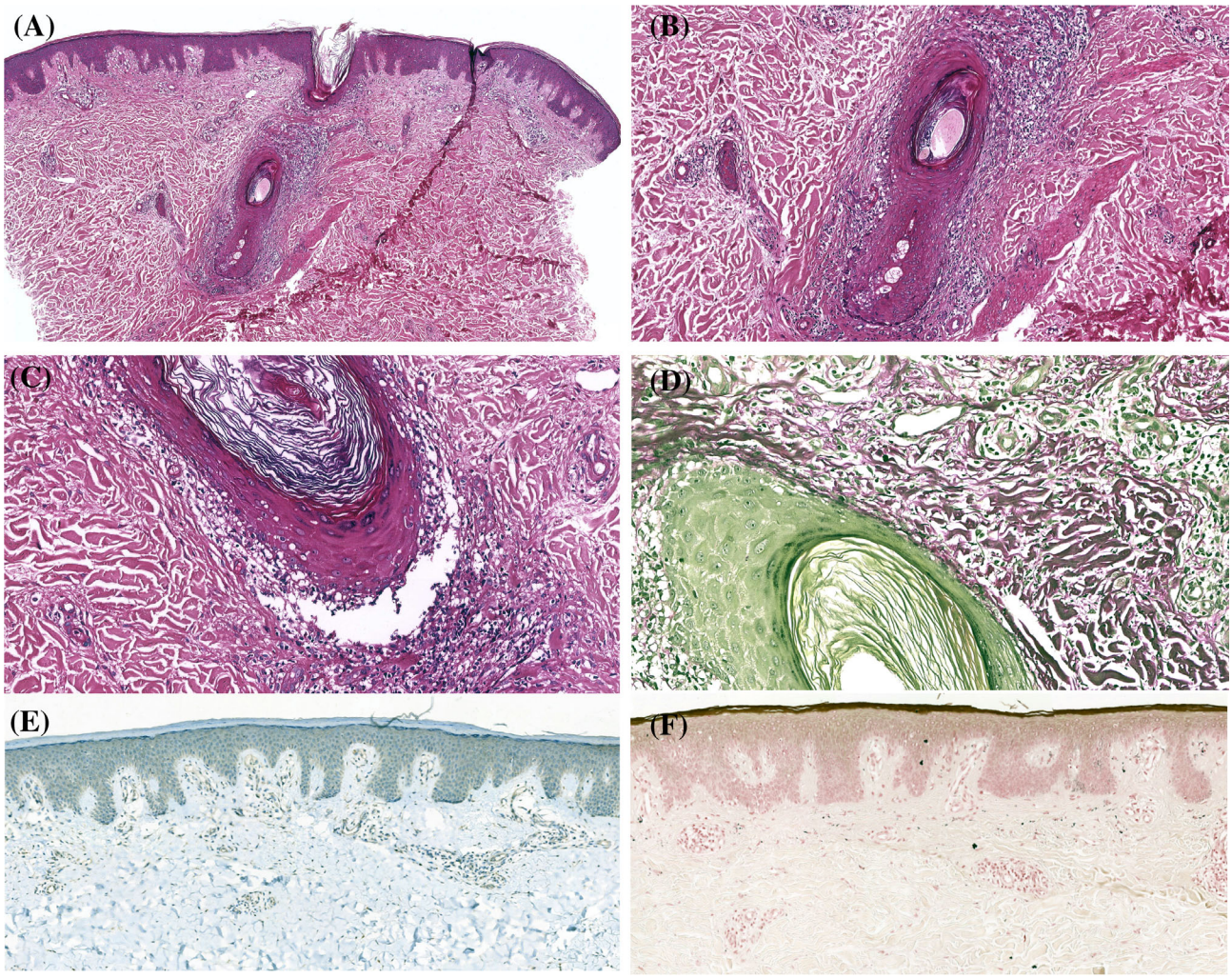

FIGURE 2 A, Perifollicular lichenoid infiltration and widening of the hair follicle orifice (HE ×10); B) Hydropic degeneration at the basal cell layer of the follicular epithelium with sparse apoptotic cells, and dense perifollicular infiltration of lymphocytes and histiocytes (HE ×40); C, Hydropic degeneration at the basal cell layer of the follicular epithelium (HE ×200); D, Partial lost and fragmentation of elastic fibers in reticular dermis (EVG ×200); E, Loss of pigmentation in the epidermis highlighted with Masson-Fontana stain (×100); F, Lack of immune-expression of SOX-10, due to loss of melanocytes (×100)

epitopes of keratinocytes or follicular epithelium initiating the immunologic attack in LP. Hereditary factors may enhance this shift in genetically prone individuals.

In conclusion, this case provides another brick in the wall, suggesting in a chronic immunocompromised district, local tissue factors, and immunological stimulation can contribute to the association of vitiligo and lichen planopilaris.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

AUTHORS CONTRIBUTION

G.E. took the lead in writing manuscript with S.V.'s supervision and support. B.M., C.D., S.A., and S.V. were responsible from diagnosis and patient follow-up. All authors discussed the results and contributed to the final manuscript.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Gizem Erbaş[1]
Cüyan Demirkesen[2]
Sibel Alper[3]
Bachar Memet[3]
Seçil Vural[3]

[1]*School of Medicine, Technical University of Munich, München, Germany*
[2]*Department of Pathology, Acıbadem Mehmet Ali Aydınlar University, School of Medicine, İstanbul, Turkey*

[3]*Department of Dermatology and Venereology, Koç University School of Medicine, İstanbul, Turkey*

Correspondence

Seçil Vural, MD, Koç University, School of Medicine, Department of Dermatology, and Venereology, Address: Topkapı, Koç Üniversitesi Hastanesi, Davutpaşa Cd. No:4, 34010 Zeytinburnu/İstanbul, Turkey.
Email: sevural@ku.edu.tr

ORCID

Gizem Erbaş https://orcid.org/0000-0001-5852-776X

REFERENCES

1. Ruocco V, Ruocco E, Brunetti G, Sangiuliano S, Wolf R. Opportunistic localization of skin lesions on vulnerable areas. *Clin Dermatol*. 2011;29(5):483-488.
2. Veitch D, Kravvas G, Hughes S, Bunker C. A rare Colocalization of lichen planus and vitiligo. *Case Rep Dermatol Med*. 2015;2015:840193.
3. Sardana K, Sharma RC, Koranne RV, Mahajan S. An interesting case of colocalization of segmental lichen planus and vitiligo in a 14-year-old boy. *Int J Dermatol*. 2002;41(8):508-509.
4. Goktay F, Mansur AT, Aydingoz IE. Colocalization of vitiligo and lichen planus on scrotal skin: a finding contrary to the actinic damage theory. *Dermatology*. 2006;212(4):390-392.
5. Baghestani S, Moosavi A, Eftekhari T. Familial colocalization of lichen planus and vitiligo on sun exposed areas. *Ann Dermatol*. 2013;25(2):223-225.
6. Ujiie H, Sawamura D, Shimizu H. Development of lichen planus and psoriasis on lesions of vitiligo vulgaris. *Clin Exp Dermatol*. 2006;31(3):375-377.
7. Harries MJ, Meyer K, Chaudhry I, et al. Lichen planopilaris is characterized by immune privilege collapse of the hair follicle's epithelial stem cell niche. *J Pathol*. 2013;231(2):236-247.
8. van den Boorn JG, Konijnenberg D, Dellemijn TA, et al. Autoimmune destruction of skin melanocytes by perilesional T cells from vitiligo patients. *J Invest Dermatol*. 2009;129(9):2220-2232.
9. Abdel-Malek ZA, Jordan C, Ho T, Upadhyay PR, Fleischer A, Hamzavi I. The enigma and challenges of vitiligo pathophysiology and treatment. *Pigment Cell Melanoma Res*. 2020;33(6):778-787.