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A coexistence case of psoriasis and vitiligo: insights into possible pathomechanisms

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Psoriasis and vitiligo are common immune-mediated skin disorders that rarely coexist at the same anatomical site [1, 2]. In such cases, interpretation of their relationship, including the possibility of a Köbner phenomenon, can be challenging. Here, we report a case of co-localized psoriasis and vitiligo, analyze its histopathological and immunological features, and discuss possible interpretations of their relationship.

A 47-year-old man had been diagnosed with psoriasis vulgaris 15 years earlier. After topical treatment, depigmented patches developed at sites overlapping with psoriatic lesions and expanded in parallel with disease activity. Eight years ago, the patient visited our department and was diagnosed with psoriasis and vitiligo. Oral cyclosporine (2.5 mg/kg/day, tapered to 1 mg/kg/day) was ineffective for psoriasis, and the vitiligo further expanded (Figure 1A). Because his psoriatic lesions were refractory (Psoriasis Area and Severity Index [PASI] score 23.2, Body Surface Area 20%) and he had no joint symptoms suggestive of psoriatic arthritis, the patient received anti–IL-23p19 antibody therapy, subcutaneous risankizumab (150 mg) at baseline, 1 month later, and every 3 months. Thereafter, marked clinical improvement of psoriasis was observed after 4 months of treatment (Supplementary Material 1), whereas the vitiligo persisted and mildly expanded without new psoriatic lesions during 5 years of continuous risankizumab treatment.

Skin biopsy samples were obtained before anti–IL-23p19 antibody treatment from two sites: a lesion containing both psoriasis and vitiligo (PVL) and a vitiligo without psoriatic features (VL; Figure 1B). PVL showed typical psoriatic features such as parakeratosis, elongated rete ridges, Munro's microabscesses, and marked dermal inflammatory infiltration (Figure 1C). In contrast, VL exhibited mild epidermal hyperplasia with limited superficial dermal infiltration (Figure 1D). Neither lesion showed melanin or melanocytes on Fontana–Masson or Melan-A staining (Supplementary Material 2).

Immunohistochemical analysis was performed focusing on inflammatory cell markers (CD3, CD4, CD8, CD123, and Foxp3; Figure 1E). In PVL, dense infiltration of $CD3^+$ and $CD4^+$ cells was observed in the dermis, while $CD8^+$ cells were present in both the dermis and, sparsely, in the epidermis. $CD123^+$ cells were distributed throughout the dermis, and $Foxp3^+$ cells were sparsely detected. In contrast, VL showed only small numbers of $CD3^+$, $CD4^+$, $CD8^+$, $CD123^+$, and $Foxp3^+$ cells in both the epidermis and dermis. Quantitative analysis, in which positively stained cells were manually counted in three representative microscopic fields, and the results are presented as mean ± standard error, demonstrated markedly higher numbers of $CD3^+$ T cells in PVL than in VL (335 ± 211 vs. 42 ± 36 cells per ×100 field). Similarly, $CD123^+$ cells, considered plasmacytoid dendritic cells, were more abundant in PVL than in VL (32 ± 19.2 vs. 5.6 ± 4.6 cells per ×100 field). The proportion of regulatory T cells, defined by the Foxp3/CD3 ratio, was 45.4% in PVL and 37.0% in VL.

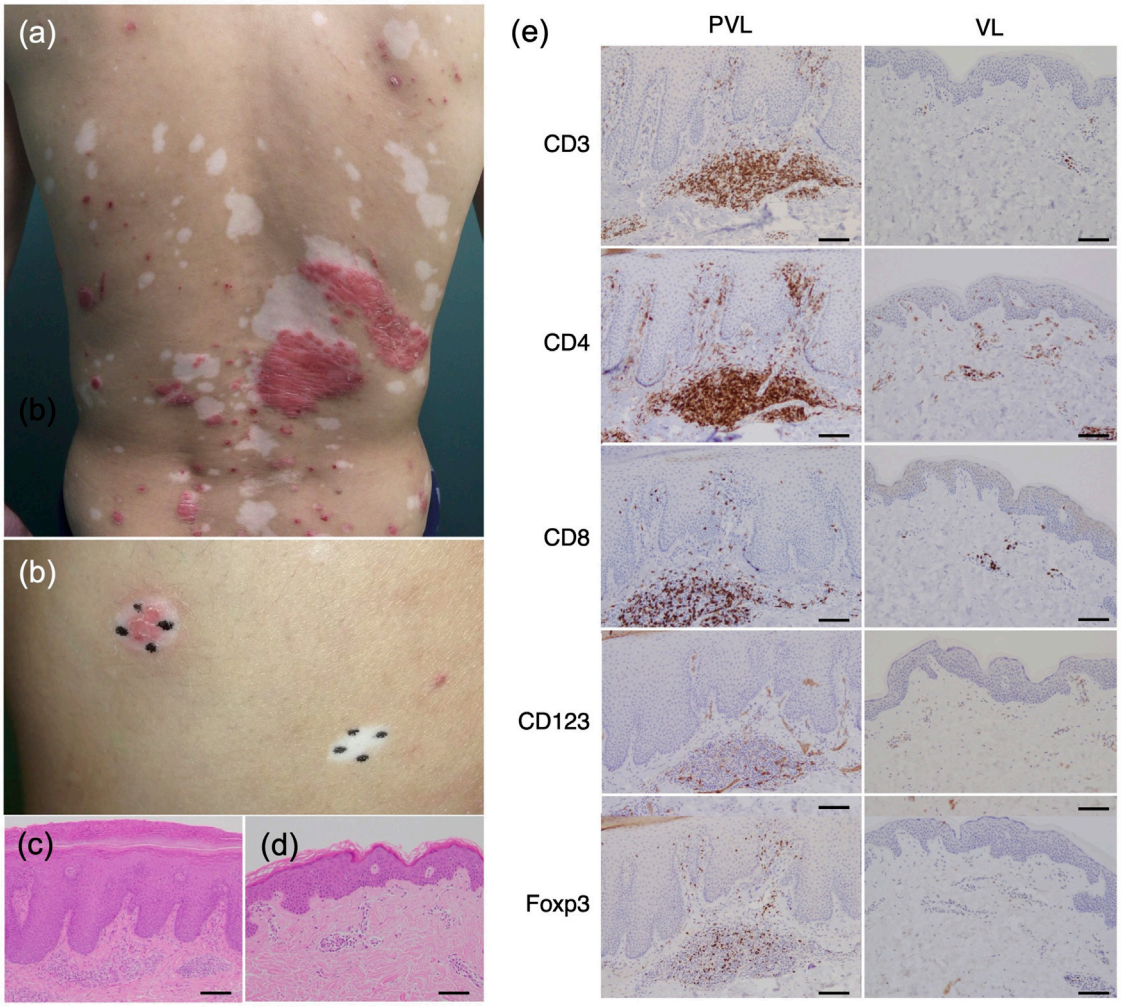

FIGURE 1
(A) Clinical presentation of a co-localized lesion of psoriasis and vitiligo. **(B)** Skin biopsies were obtained from a lesion exhibiting both psoriasis and vitiligo (PVL), and from a lesion with vitiligo alone (VL). Hematoxylin and eosin staining of **(C)** PVL and **(D)** VL. **(E)** Immunohistochemical staining for CD3, CD4, CD8, CD123, and Foxp3 in PVL and VL. Scale bars: 100 μm.

These findings indicate that PVL showed markedly greater inflammatory cell infiltration than VL, while the overall cellular composition was broadly similar between the lesions. This difference likely reflects the intrinsic inflammatory nature of psoriasis rather than a disease-specific interaction. Ono et al. reported coexisting vitiligo and psoriasis lesions and demonstrated that the proportion of Tregs among $CD3^+$ T cells increased from vitiligo to psoriasis [2], which is consistent with our findings. In addition, increased infiltration of $CD123^+$ cells in PVL suggests a role for plasmacytoid dendritic cells in psoriatic inflammation [3]. Notably, pDCs have also been implicated in progressive vitiligo through local type I interferon production [4].

The relationship between psoriasis and vitiligo in this case remains uncertain, and several interpretations should be considered. Psoriasis may have induced secondary melanocyte loss through chronic inflammation. The clinical course, in which psoriasis preceded vitiligo, and the absence of melanocytes in inflamed areas support the possibility of inflammation-induced melanocyte damage. However, psoriatic changes were observed mainly in the central regions of vitiligo rather than at their active edges, which does not fully support a simple Köbner phenomenon–based explanation. The increased infiltration of CD3-positive T cells and CD123-positive cells in the psoriasis-containing lesion may primarily reflect active psoriatic inflammation rather than a psoriasis-specific interaction with depigmentation.

In this case, an IL-23p19 inhibitor therapy succeeded to treat the psoriasis but not vitiligo. A previous case report demonstrated that *de novo* vitiligo developed during IL-23p19 inhibitor therapy [5], suggesting that IL-23p19 inhibitor therapies may not suppress vitiligo-associated inflammation and, in some cased, may even contribute its development. This study is a single case report and has limitations, including restricted biopsy sites and the lack of detailed immunological analyses. The present findings neither confirm nor exclude a Köbner phenomenon. Overall, this case highlights the complexity of interactions between psoriasis and vitiligo and underscores the need for further investigation.

Data availability statement

The original contributions presented in the study are included in the article/Supplementary Material, further inquiries can be directed to the corresponding author.

Ethics statement

Ethical approval was not required for this single case report in accordance with the applicable institutional guidelines. Written informed consent was obtained from the patient for the publication of this case report and any potentially identifiable images or data. The study was conducted in accordance with the Declaration of Helsinki and local institutional requirements.

Author contributions

All authors listed have made a substantial, direct, and intellectual contribution to the work and approved it for publication.

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Conflict of interest

The author(s) declared that this work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Generative AI statement

The author(s) declared that generative AI was used in the creation of this manuscript. The authors used ChatGPT (OpenAI) for English language editing and improving readability. All scientific content, interpretation, and conclusions were reviewed and verified by the authors, who take full responsibility for the manuscript.

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Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontierspartnerships.org/articles/10.3389/jcia.2026.17495/full#supplementary-material

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