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LETTER TO THE EDITOR

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Chronic actinic dermatitis developing after remission of malignant lymphoma

Dear Editor,

We herein report a 74-year-old man, who presented with pruritic erythema on his face and nuchal region 2 years after achieving remission from non-Hodgkin's lymphoma ($CD20^+$ diffuse large B-cell lymphoma; DLBCL) treated with six cycles of R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone) chemotherapy. Pruritic erythema occurred repeatedly, became indurated, and gradually spread to the face, neck, and dorsal aspects of both hands and fingers. It was exacerbated from spring to summer and often relapsed during therapy using topical corticosteroids, oral antihistamines, and prednisolone (2.5-5 mg/d). After 4 more years, he finally was referred to our department for further evaluation of the skin lesions, which were possibly photosensitive (Figure 1A-B). At the time, he had not been taking any medicines for 6 months. Phototesting showed hypersensitivity to both UVB (minimal erythema dose, 20 mJ/cm^2) and UVA (minimal response dose, 4 J/cm^2) and no hypersensitivity to visible light. Histopathological examination revealed acanthosis, liquefaction, and epidermal and dermal infiltration of lymphocytes with a few eosinophils (Figure 1C-D). The infiltrating lymphocytes in the dermis were mainly positive for CD8 (Figure 1E–G). The results of blood tests, including antinuclear antibodies, and urinary and porphyrin derivatives, were normal or negative, while marked eosinophilia (14.5% in 7420/mm^3 WBC) was observed. A flow cytometric analysis revealed a decreased frequency of $CD4^+$ cells (10.1%) with a decreased $CD4^+$ cell count (164/μL), a normal frequency of $CD8^+$ cells (12.3%) and a low CD4/CD8 ratio (0.82). Measurement of blood cytokines showed normal levels of IFN-γ (≦0.1 IU/mL), TNF-α (0.9 pg/mL), IL-5 (≦3.9 pg/mL), and IL-10 (≦2 pg/mL), but the level of IL-4 (11.1 pg/mL) was increased (reference range, ≦6.0 pg/mL). Based on these findings, the patient was diagnosed with chronic actinic dermatitis (CAD), which appeared during the remission period of DLBCL. We provided guidance about

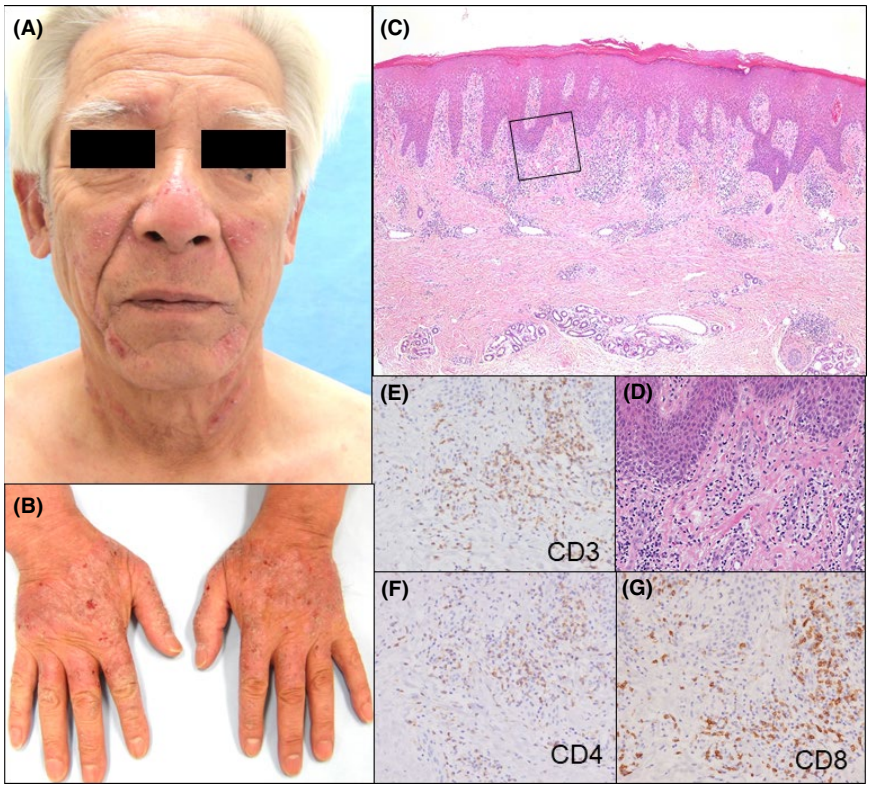

FIGURE 1 At his first visit, well-demarcated, indurated erythematous lesions with scratch marks were observed on A, his face, neck, and B, dorsal aspects of both hands. A histopathological examination revealed epidermal thickening with parakeratosis, partial spongiosis of the epidermis, and liquefaction. C and D, Intraepidermal and dermal infiltration of lymphocytes with a small number of eosinophils was observed with minimal atypia. Immunohistochemistry revealed that the lymphocytes in the dermis were positive for CD3, CD4, and CD8 (E-G). $CD8^+$ cells outnumbered $CD4^+$ cells

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light shielding, administration of oral prednisolone (15 mg/d) and bepotastine besilate, and topical corticosteroids and clobetasol propionate ester, with therapeutic effects. Four years later, the patient is still hypersensitive to sunlight unless sunscreens were used. Oral and topical corticosteroids are required to treat recurrent skin lesions on the sun-exposed areas.

As CAD is likely to complicate human immunodeficiency virus infection and blood disorders such as adult T-cell lymphoma/leukemia, some kind of immune compromise may be involved in the onset of CAD.[1–3] In our case, $CD8^+$ T-cell predominance was observed in the skin lesions, which might have been induced by decreased number of $CD4^+$ cells. We hypothesized that DLBCL and sequential chemotherapy might have caused an immunomodulated state that activated $CD8^+$ T cells, thereby triggering CAD. Curiously, the patient showed an increased serum IL-4 level with eosinophilia. The serum level of TARC, a Th2 chemokine, was reported to be increased in CAD patients, and we assume that a systemic shift toward Th2 immunity in our case might have occurred secondarily after the long duration of CAD.[4,5]

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

1. Sugita K, Shimauchi T, Tokura Y. Chronic actinic dermatitis associated with adult T-cell leukemia. J Am Acad Dermatol. 2005;52(Suppl 1):38–40.
2. Agar N, Morris S, Russel-Jones R, Hawk J, Whittaker S. Case report of four patients with erythrodermic cutaneous T-cell lymphoma and a severe photosensitivity mimicking chronic actinic dermatitis. Br J Dermatol. 2009;160(3):698–706.
3. Koch K. Photosensitive disorders in HIV. South Afr J HIV Med. 2017;18(1):676.
4. Ma Y, Lu Z. Treatment with topical tacrolimus favors chronic actinic dermatitis: a clinical and immunopathological study. J Dermatol Treat. 2010;21(3):171–7.
5. Ko DY, Choi SH, Ha SM, et al. The clinical severity score of chronic actinic dermatitis correlates with in vivo photoallergic reactions and the immunologic parameters related to a shift towards Th2 immunity from the Th2/Th1 balanced status in patients with chronic actinic dermatitis. Photodermatol Photoimmunol Photomed. 2016;32(4):199–206.