



A Case of Primary Cutaneous Gamma-Delta T-Cell Lymphoma with Pautrier Microabscess

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Dear Editor:

Primary cutaneous gamma-delta T-cell lymphoma (PCGD-TCL) accounts for less than 1% of primary cutaneous T-cell lymphomas (CTCLs). PCGD-TCL is characterized by a highly aggressive clinical course, while mycosis fungoides (MF) has a relatively favorable prognosis^{1,2}. PCGD-TCL is recognized for its complexity and heterogeneity³. Here we present a case of PCGD-TCL with Pautrier microabscess.

A 69-year-old female presented with erythematous tumors and plaques on her trunk and extremities. Initially, eryth-

emas and plaques had been noticed at her lower extremities and had spread to her trunk and upper extremities in the past year and a half. Physical examination revealed multiple erythematous tumors and plaques on her trunk and extremities (Fig. 1). Histopathological examination from an erythematous plaque showed focal infiltrates of atypical lymphocytes in the dermis (Fig. 2A, B). Epidermotropic infiltrates of atypical lymphocytes and aggregations of atypical lymphocytes as Pautrier's microabscess in the epidermis were recognized (Fig. 2C). Flow cytometric analysis and immunohistochemistry both showed



Fig. 1. (A) Multiple erythematous tumors and plaques on the patient's trunk. The largest tumor was observed at the right lower abdomen. (B) Multiple erythematous tumors on the lower extremities.

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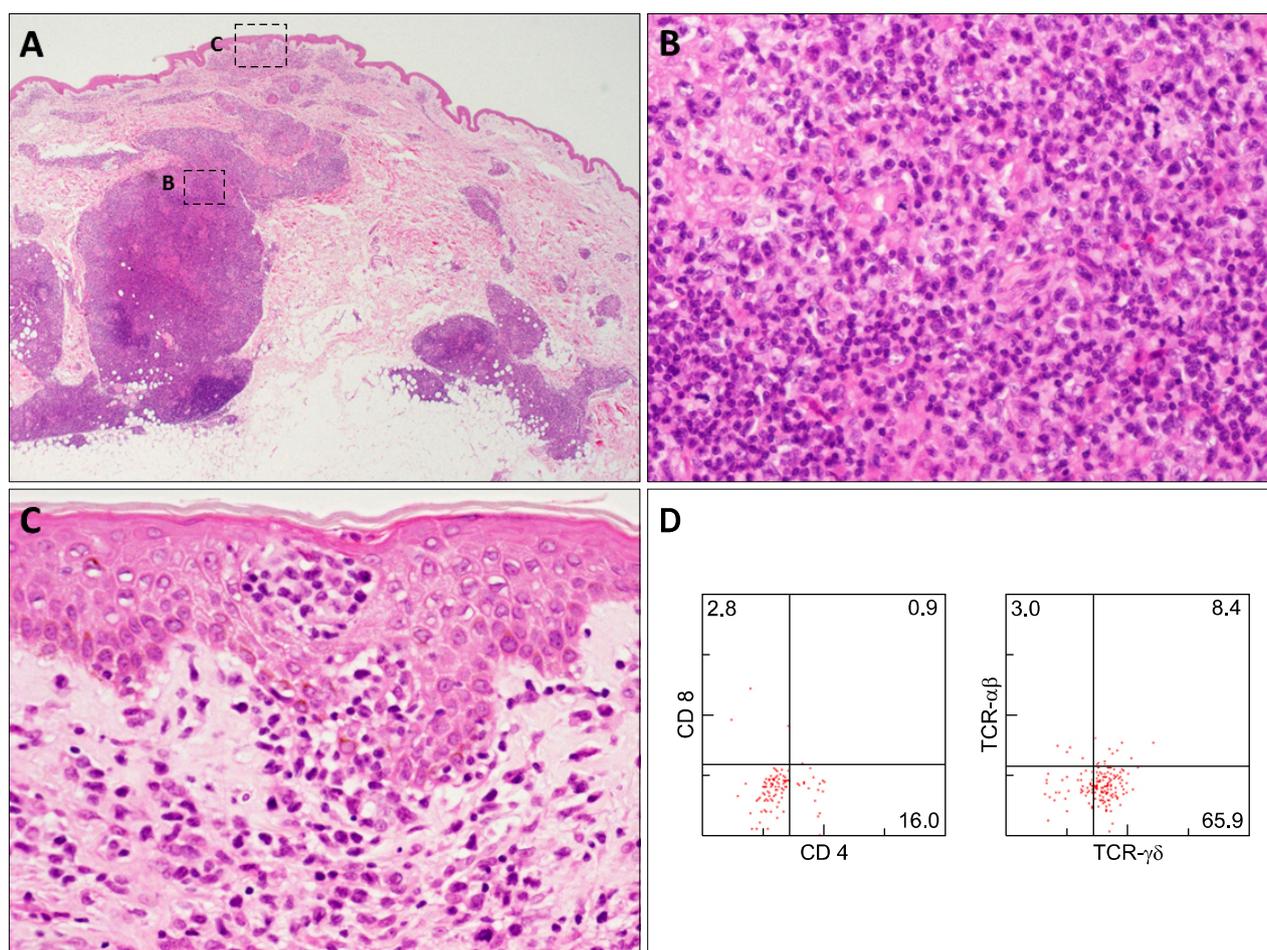


Fig. 2. (A) Histology of a biopsy specimen of an erythematous plaque showing focal infiltrates of atypical lymphocytes in the dermis. The dotted boxes indicate the locations shown at higher magnification in Fig. 1B and 1C (H&E, $\times 20$). (B) Infiltrates of atypical lymphocytes intermingled with histiocytes in the dermis (H&E, $\times 400$). (C) Aggregations of atypical lymphocytes as Pautrier's microabscesses in the epidermis (H&E, $\times 400$). (D) Flow cytometry analyses of a biopsy specimen from the skin using a CD45/SSC gating procedure. CD45/SSC gated cells with various surface antigens of CD4, CD8, T-cell receptor (TCR)- $\alpha\beta$, TCR- $\gamma\delta$ shown in two dot plots. (E) Immunohistochemistry of a biopsy specimen from the skin showing a positive reaction for C γ M1 ($\times 400$). (F) Immunohistochemistry of a biopsy specimen from the skin showing a negative reaction for β F1 ($\times 400$). (G) Immunohistochemistry from a lymph node showing a positive reaction for C γ M1 ($\times 400$).

positive reactions for CD2, CD3 and CD5, a partially positive reaction for CD4, and negative reactions for CD7, CD8, CD20 and CD56 in atypical lymphocytes (Fig. 2D). A positive reaction for C γ M1 and negative reactions for T-cell restricted intracellular antigen-1 and granzyme B were demonstrated by immunohistochemistry (Fig. 2E). Atypical lymphocytes in the epidermis were positive for C γ M1 and negative for β F1 (Fig. 2F). Positron emission tomography revealed multiple accumulation sites with fludeoxyglucose in the skin and inguinal lymph nodes. Histopathological examination of a lymph node showed medium to large sized atypical lymphocytes. Flow cytometric analysis and immunohistochemistry of the lymph node both showed identical results with the skin in addition to a

positive reaction for C γ M1 by immunohistochemistry (Fig. 2G). Southern blots showed TCR gene rearrangements by probes of J γ . A diagnosis of PCGD-TCL was made. Treatments with radiation and cyclophosphamide-hydroxydaunorubicin-oncovin-prednisone (CHOP) had diminished most tumors and plaques, but the tumors recurred. CHOP therapy excluding oncovin due to peripheral neuropathies was effective, and most tumors and plaques have been under control for two years since the start of the initial therapy.

Although PCGD-TCL and MF should be distinct clinical entities in terms of their histogenesis, an accurate diagnosis of PCGD-TCL is sometimes challenging due to the aberrant expression of surface markers in neoplastic cells

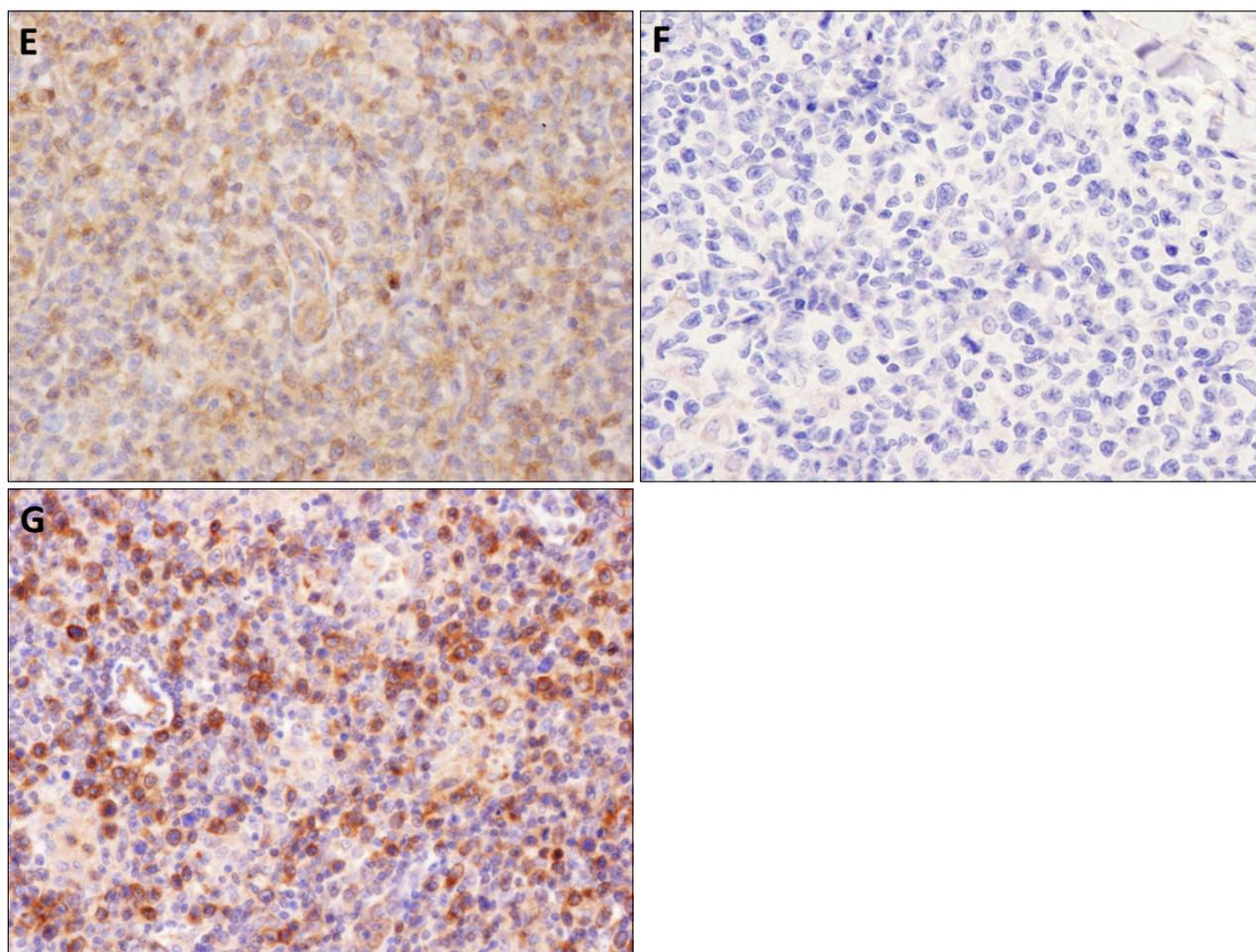


Fig. 2. Continued.

and technical complexities such as insufficient antibodies that work in paraffin-embedded sections. A previous study of 146 CTCLs showed that 5 PCGD-TCLs and 2 MFs were included in 12 CTCLs with T-cell receptor- γ expression³. Another study of 53 PCGD-TCLs using the same antibody showed that the clinical and histopathological features of 6 cases were similar to those of MF⁴. Further, PCGD-TCL following MF has also been reported⁵. Those studies and reports may suggest the heterogeneity of PCGD-TCL. In our case, erythematous plaques were observed in addition to tumors and an epidermotropism as Pautrier microabscess was recognized. Those clinical and histopathological findings may confuse an accurate diagnosis. Detailed analyses of such cases will lead to an accurate diagnosis and the elucidation of the heterogeneity of PCGD-TCL.

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CONFLICTS OF INTEREST

The authors have nothing to disclose.

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Granulocyte Colony-Stimulating Factor-Induced Psoriasiform Dermatitis Improved by Narrowband Ultraviolet B

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Dear Editor:

Granulocyte colony-stimulating factor (G-CSF) is a hematopoietic growth factor that stimulates the proliferation and differentiation of hematopoietic cells in a bone marrow, and activates neutrophils. There are possible systemic reactions associated with G-CSF, including Sweet's syndrome, bullous pyoderma gangrenosum, leukocytoclastic vasculitis, and panniculitis¹. Furthermore, G-CSF may induce psoriasiform dermatitis, or exacerbate pre-existing psoriasis².

A 73-year-old man presented with one-month history of pruritic erythematous papules and patches on the whole body. He was diagnosed with gastric cancer and myelodysplasia 3 years ago. Cancer treatment included chemotherapy using decitabine. G-CSF therapy was initiated when the peripheral blood neutrophil level decreased below 1,000/mm³. After two treatments with G-CSF (1 and 3 weeks prior to presentation), the skin lesion developed. At 4 weeks after discontinuation of G-CSF, however, the lesions partially improved. And further G-CSF therapy exacerbated the lesions. The patient had no history of der-

matologic diseases. The physical examination revealed erythematous variable sized scaly papules and patches on the whole body (Fig. 1A). At the time of skin lesion onset, a blood cell count showed a white blood cell count of 1,330/mm³ (normal: 4,700~9,600/mm³), neutrophil count of 150/mm³ (normal: 1,880~6,640/mm³). Histopathologic findings showed epidermal acanthosis, confluent parakeratosis, neutrophils in the stratum corneum (Munro's microabscess), and dilated capillaries in the dermal papillae (Fig. 1C). Based on the clinical course, drug history, and findings of physical and histologic examination, the diagnosis of G-CSF-induced psoriasiform dermatitis was made. As no significant improvement was seen after stopping G-CSF, he was treated with 19 cycles of narrowband ultraviolet B (NB-UVB) (3 times weekly), which resulted in significant improvement (Fig. 1B). The lesions improved gradually without aggravation and side effect during NB-UVB treatment. In follow-up period, he died of sepsis after ophthalmologic surgery for orbital mucormycosis.

Psoriasiform dermatitis associated with G-CSF is known to be induced by increase in peripheral blood neutrophils,

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