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

Min Soo Jang, Jong Bin Park, Joon Hee Kim, Myeong Hyeon Yang, Kang Hoon Lee, Sang Hwa Han, Kee Suck Suh

Department of Dermatology, Kosin University College of Medicine, Busan, Korea

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 panniculitis[1]. Furthermore, G-CSF may induce psoriasiform dermatitis, or exacerbate pre-existing psoriasis[2].

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/mm^3. 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 dermatologic 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/mm^3 (normal: 4,700～9,600/mm^3), neutrophil count of 150/mm^3 (normal: 1,880～6,640/mm^3). 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 (NBUVB) (3 times weekly), which resulted in significant improvement (Fig. 1B). The lesions improved gradually without aggravation and side effect during NBUVB 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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Corresponding author: Kee Suck Suh, Department of Dermatology, Kosin University College of Medicine, 262 Gamcheon-ro, Seo-gu, Busan 49267, Korea. Tel: 82-51-990-6145, Fax: 82-51-990-3041, E-mail: ksderm77@unitel.co.kr

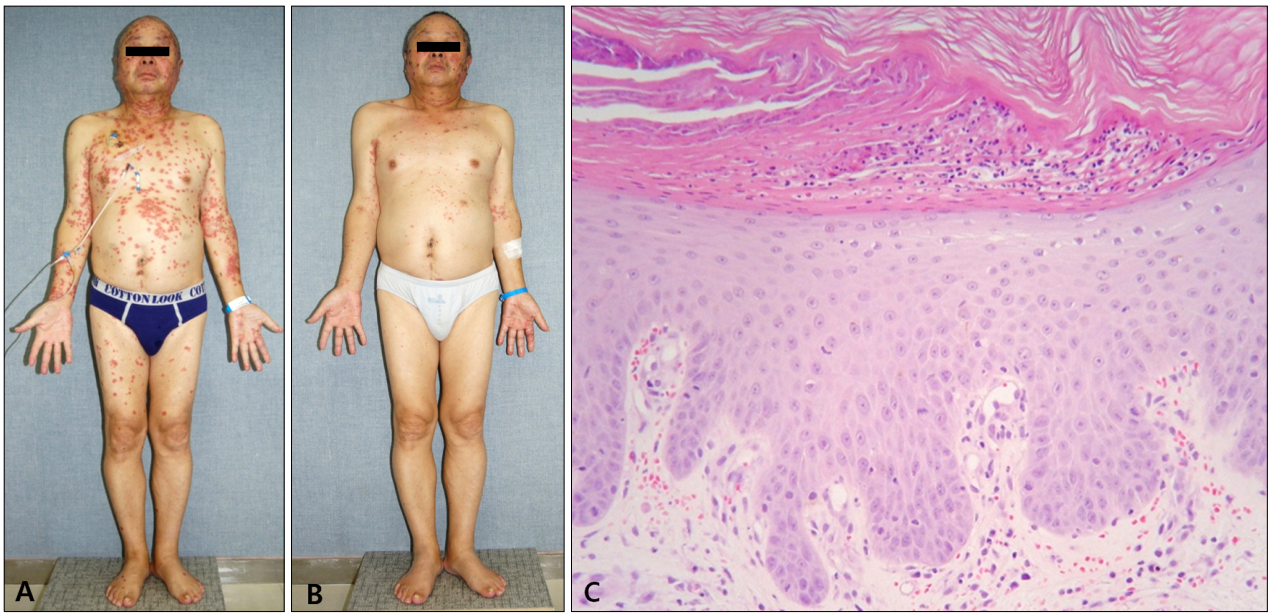

Fig. 1. (A) Generalized erythematous scaly papules and patches on the whole body. (B) Clinical photographs taken one week after 19 cycles of narrowband ultraviolet B phototherapy. There is significant improvement in the skin lesions from the initial presentation, especially on the trunk. (C) Histopathologic findings showed epidermal acanthosis, confluent parakeratosis, neutrophils in the stratum corneum (Munro's microabscess), and dilated capillaries in the dermal papillae (H&E, ×200).

neutrophil activation, promotion of endothelial neutrophils adhesion, and increase in production of the neutrophil chemotactic factor interleukin-8[2,3]. In this case, as neutrophil count increased with G-CSF therapy, the skin lesions exacerbated. When the G-CSF was stopped, the neutrophil count decreased, and the skin lesions partially improved.

Rácz et al.[4] described improvements in erythema and scales in patients with imiquimod-induced psoriasiform dermatitis after NBUVB irradiation. The group with NBUVB irradiation experienced a greater decrease in epidermal neutrophil count, and angiogensis compared to that of the group without NBUVB irradiation. Ozawa et al.[5] suggested that induction of T cell apoptosis could be the major mechanism by which 312-nm UVB resolves psoriatic skin lesions. Our patient was treated with 19 cycles of NBUVB, which resulted in significant improvement. The authors think that NBUVB may show therapeutic effect by inducing T cell apoptosis and decreasing the epidermal neutrophil count and angiogenesis.

We have shown a case of G-CSF-induced psoriasiform dermatitis in a patient with myelodysplasia who had been treated with chemotherapy. Based on this case, it is important to consider G-CSF as the cause of psoriasiform dermatitis with consistent histopathologic findings in chemotherapy patients. And we report the NBUVB to be effective treatment.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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