should consider the possibility of *M. marinum* infection even if the history and location are atypical.

ACKNOWLEDGMENT

This study was supported by the Laboratory Equipment and Research Fund of Chosun University, in 2015.

REFERENCES

1. Runyon EH. Anonymous mycobacteria in pulmonary disease. *Med Clin North Am* 1959;43:273-290.
2. Adhikesavan LG, Harrington TM. Local and disseminated infections caused by *Mycobacterium marinum*: an unusual cause of subcutaneous nodules. *J Clin Rheumatol* 2008;14:156-160.
3. Ko DY, Song KH. *Mycobacterium marinum* infection occurring on the face. *J Dermatol* 2013;40:773-774.
4. Chia JH, Wu TL, Su LH, Kuo AJ, Lai HC. Direct identification of mycobacteria from smear-positive sputum samples using an improved multiplex polymerase chain reaction assay. *Diagn Microbiol Infect Dis* 2012;72:340-349.

<http://dx.doi.org/10.5021/ad.2015.27.5.641>

Imatinib Mesylate-Induced Erythema Multiforme: Recurrence after Rechallenge with 200 mg/day Imatinib

Min Kyung Lee, Won Joo Kwon, Eun Byul Cho, Eun Joo Park, Kwang Ho Kim, Kwang Joong Kim

Department of Dermatology, Hallym University Sacred Heart Hospital, Anyang, Korea

Dear Editor:

Imatinib mesylate (Gleevec; Novartis AG, Basel, Switzerland), a selective tyrosine receptor kinase inhibitor, is increasingly used for treating chronic myeloid leukemia, Philadelphia chromosome-positive acute lymphoblastic leukemia, and high-grade gastrointestinal stromal tumors (GISTs)¹. Several cases of cutaneous reactions after imatinib use have been reported¹. We report a case of EM after imatinib administration for the treatment of a GIST.

A 66-year-old woman was referred for pruritus from the department of oncology. She received a diagnosis of a

GIST, for which she received adjuvant imatinib therapy after gastric wedge resection. She noticed a pruritic rash on her trunk after 5 weeks of 400 mg/day imatinib therapy. Physical examination revealed generalized variable-sized erythematous wheal-like patches with some targetoid lesions on the trunk, face, and extremities (Fig. 1). Immunoglobulin (Ig) G and IgM antibodies to the herpes simplex virus were not detected. A skin biopsy from the trunk revealed vacuolar degeneration, tagging of lymphocytes along the dermal-epidermal junction, and perivascular lymphocytic and some eosinophilic infiltrations in the upper dermis (Fig. 2A). Some dyskeratotic and necrotic keratinocytes were obvious in the epidermis (Fig. 2B); therefore, EM was diagnosed. As imatinib was the only medication administered to the patient, it was considered the most probable cause. Imatinib was discontinued, and oral steroid and antihistamine were prescribed. For 2 weeks, 30 mg/day steroid, tapered to 5 mg/day, was administered. One month after the discontinuation of imatinib therapy, the rash was fully cured. Imatinib treatment was restarted at a lower dose of 100 mg/day without steroids; no skin lesion developed for 2 months. However, when the dose

Received August 9, 2014, Revised November 10, 2014, Accepted for publication February 4, 2015

Corresponding author: Eun Joo Park, Department of Dermatology, Hallym University Sacred Heart Hospital, 22 Gwanpyeong-ro 170 beon-gil, Dongan-gu, Anyang 14068, Korea. Tel: 82-31-380-3765, Fax: 82-31-386-3761, E-mail: anpark7770@hanmail.net

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

Fig. 1. Clinical manifestations of the patient. (A) Generalized erythematous variable-sized wheal-like patches on the trunk. (B) Several lesions showing the typical targetoid appearance.

Fig. 2. Histopathological findings of the lesion. (A) Vacuolar degeneration, tagging of lymphocytes along the dermal-epidermal junction, and perivascular lymphocytic and a few eosinophilic infiltrations in the upper dermis (H&E, $\times 200$). (B) Spongiosis and necrotic keratinocytes in the epidermis (H&E, $\times 400$).

was increased to 200 mg/day without oral steroids, a similar rash developed. The patient could continue imatinib therapy with a gradual dose escalation from 100 to 200 mg/day with concomitant 5 mg/day oral steroids. No additional skin lesions were detected during 5 months of follow-up.

It is estimated that approximately 7%~21% of patients treated with imatinib experience variable degrees of skin eruptions². There are a few reports of imatinib-induced EM. Park et al.³ reviewed patients with cutaneous eruptions after imatinib therapy, and 2 of 10 patients (20%) had EM-like drug eruptions. The cutaneous adverse effects of imatinib are dose dependent and seemingly related to a pharmacological effect of the drug^{1,2}. The most common skin reactions are maculopapular rashes^{1,2}, and these are spontaneously relieved with a minimal dose of antihistamine or topical steroids. Several uncommon or severe re-

actions such as Stevens-Johnson syndrome have also been reported; however, they seem to be an idiosyncratic adverse reaction of hypersensitivity⁴. These reactions require the concomitant use of oral steroids, and immediate discontinuation of imatinib therapy is imperative^{4,5}.

Here, we observed a case of imatinib-induced EM. We noted a dose-dependent relation; thus, we consider that imatinib-induced EM is related to a pharmacological effect of imatinib. The skin lesion subsided and did not recur only with the concomitant use of low-dose oral steroids. As imatinib is a very effective therapeutic agent and alternative treatment options are limited, recognition of adverse cutaneous reactions after imatinib therapy and the appropriate management required is helpful.

REFERENCES

1. Valeyrie L, Bastuji-Garin S, Revuz J, Bachot N, Wechsler J, Berthaud P, et al. Adverse cutaneous reactions to imatinib (STI571) in Philadelphia chromosome-positive leukemias: a prospective study of 54 patients. *J Am Acad Dermatol* 2003; 48:201-206.
2. Brouard M, Saurat JH. Cutaneous reactions to STI571. *N Engl J Med* 2001;345:618-619.
3. Park HJ, Kim HS, Kim HJ, Lee JY, Cho BK, Lee AW, et al. Immunohistochemical characterization of cutaneous drug eruptions by STI571. *J Dermatol Sci* 2005;38:9-15.
4. Hsiao LT, Chung HM, Lin JT, Chiou TJ, Liu JH, Fan FS, et al. Stevens-Johnson syndrome after treatment with STI571: a case report. *Br J Haematol* 2002;117:620-622.
5. Lee JH, Chung JY, Jung MY, Kim CR, Park JH, Park JH, et al. Lichenoid drug eruption after low-dose imatinib mesylate treatment. *Ann Dermatol* 2013;25:500-502.

<http://dx.doi.org/10.5021/ad.2015.27.5.643>

A Giant, Deep, Benign Fibrous Histiocytoma with a Palisading Pattern

Jeong Nan Kang, Wonkyung Lee, So Young Jung¹, Se Won Jung, Jung Eun Seol, Hyojin Kim, Ho Suk Sung

Department of Dermatology, Busan Paik Hospital, ¹Department of Dermatology, Haeundae Paik Hospital, Inje University College of Medicine, Busan, Korea

Dear Editor:

A 42-year-old male patient presented to our clinic with a skin-colored mass that had been present for 2 years; his medical and family histories were otherwise unremarkable. Physical examination revealed a soft, movable, skin-colored mass on his upper left back, with a brown, bean-sized pedunculated nodule on the overlying skin (Fig. 1A). Excisional biopsy was performed. The tumor, which was well demarcated and localized in the subcutaneous layer, measured 5.4×5.1×2.1 cm (Fig. 1B). The histopathological examination indicated that the lesion was an encapsulated mass, located in subcutaneous tissue with a central cavity. The tumor was well-circumscribed by a fibrous capsule, and no honeycomb pattern was observed in the marginal area. In the tumor periphery, large blood vessels with a dilated, branching appearance were observed (Fig. 2A). The tumor cells were arranged in a palisading and partially

storiform pattern, with focal myxoid change (Fig. 2B). Under high magnification, the tumor cells were spindle shaped with elongated nuclei and an ill-defined cytoplasm; individual cells were infiltrating between collagen bundles and arranged in a palisading pattern, similar to Verocay bodies (Fig. 2C). Cellular atypia and mitotic bodies were not detected. Immunohistochemical analysis revealed that the cells were strongly positive for CD34 (Fig. 2D); focally positive for factor XIIIa; and negative for S-100 (Fig. 2E), desmin, actin, and CD68. A diagnosis of giant, deep, benign fibrous histiocytoma (BFH), with a palisading pattern, was rendered, with no evidence of recurrence at 15 months after excision.

BFH, which is characterized by several histological subtypes, is among the most-common soft tissue skin tumors. Since Fletcher's¹ description in 1990 of 21 cases of deep BFH (DBFH), several more cases have been reported¹⁻³.

Received September 17, 2014, Revised January 5, 2015, Accepted for publication February 4, 2015

Corresponding author: Hyojin Kim, Department of Dermatology, Busan Paik Hospital, Inje University College of Medicine, 75 Bokji-ro, Busanjin-gu, Busan 47392, Korea. Tel: 82-51-890-6135, Fax: 82-51- 897-6391, E-mail: derma09@hanmail.net

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.