http://dx.doi.org/10.5021/ad.2015.27.5.639

Mycobacterium marinum Infection on the Face Diagnosed by Polymerase Chain Reaction Amplification and Direct Sequencing

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Dear Editor:

A 56 years old Korean woman presented with asymptomatic multiple rice-sized erythematous papules on left cheek (Fig. 1A). The scaly papules were noted 2 months ago. She had no history of trauma or exposure to fish. She had been learning to swim for 3 months. Culture examination of the smear for bacteria and fungus revealed no growth. The nested polymerase chain reaction (PCR) for *Mycobacterium tuberculosis* showed negative results. A biopsy specimen taken from the lesion showed granulomatous inflammation with granulation tissue in the deep dermis. The granulomatous infiltration was composed of multiple histiocytes, mononuclear cells, and giant cells (Fig. 2A). Acid-fast bacilli (AFB) staining produced negative findings (Fig. 2B). Hence, the diagnosis was as nonspecific inflammation of the face, and the patient was treated with triamcinolone injection, three times for 6 weeks. After the injection, the lesions improved overall; however, a solitary lesion showed pus formation. Thus, in addition, mycobacterial culture including the pus was performed, which showed growth of yellow pigment-producing *Mycobacterium* at 32°C to 33°C in mycobacterium growth indicator tube (Fig. 2C). Zeihl-Neelsen staining of culture materials showed innumerable AFB (Fig. 2D), and the or-

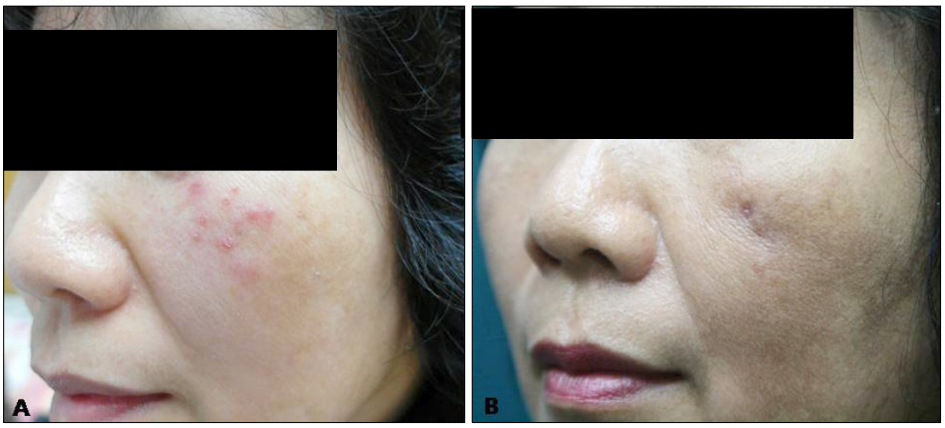

Fig. 1. (A) Multiple, erythematous, various sized, scaly papules on the left cheek. (B) After 4 months, the lesions improved with postinflammatory hyperpigmentation and atrophic scar.

Received November 18, 2014, Revised January 8, 2015, Accepted for publication January 16, 2015

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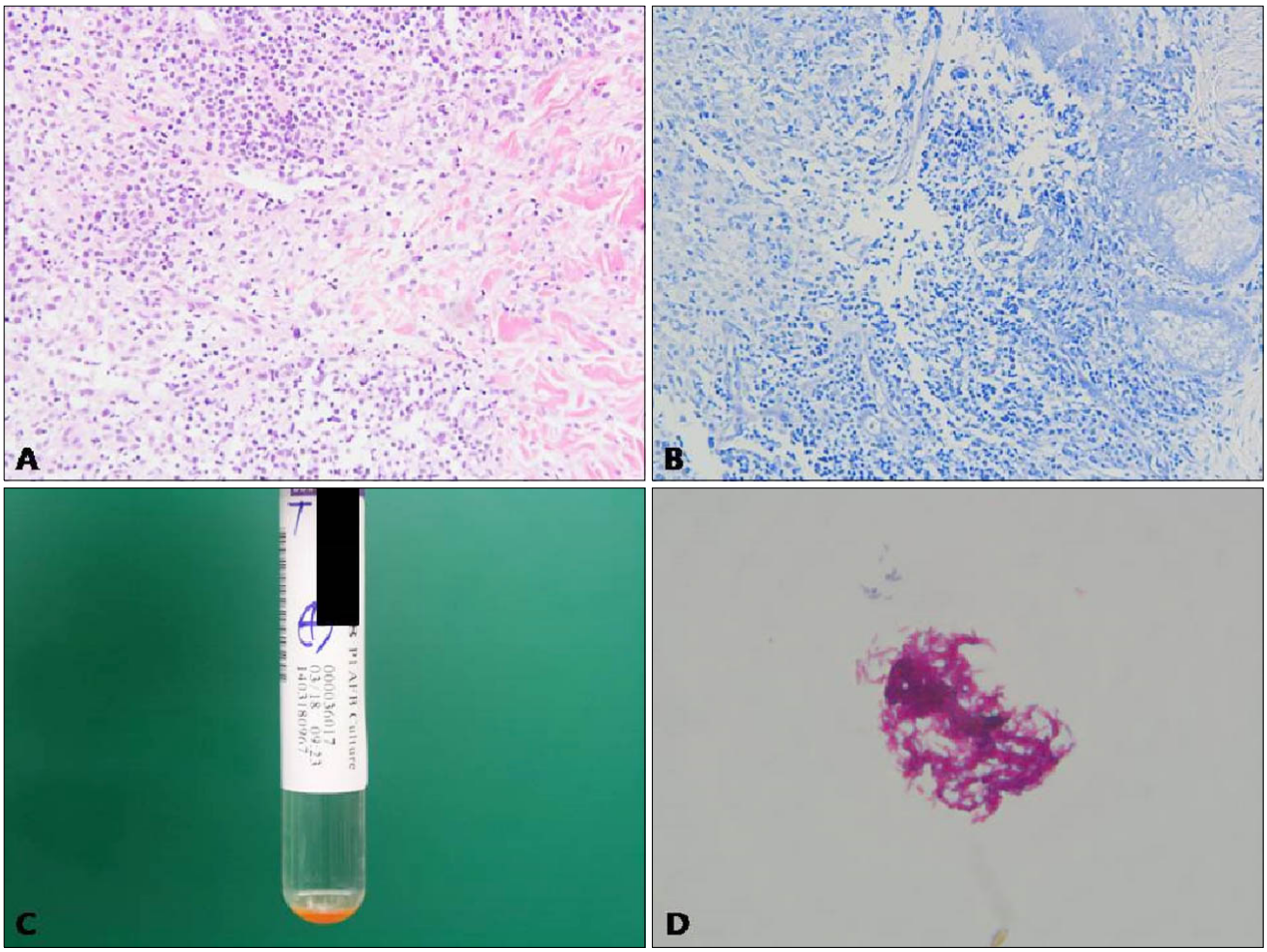

Fig. 2. (A) Granulomatous infiltration in the deep dermis was composed of multiple histiocytes, mononuclear cells, and giant cells (H&E, ×200). (B) Negative findings (acid-fast bacillus, ×100). (C) Growth of yellow pigment producing mycobacterium on 32°C to 33°C mycobacterium growth indicator tube after 45 days. (D) Presence of innumerable acid-fast bacilli (Ziehl-Neelsen, ×1,000).

ganism was identified as *Mycobacterium*. Hence, PCR-reverse blot hybridization assay was performed to identify atypical mycobacterial species, which were confirmed as *M. marinum* or *M. ulcerans*.

Furthermore, PCR amplification and direct sequencing of *16SrRNA*, *tuf*, *rpoB*, *hsp65* genes were performed for the rapid and accurate identification of the organism. In *16SrRNA* and *tuf* gene analysis, both *M. marium* and *M. ulcerans* showed >99% homology; thus, it was not possible to differentiate between the two species. Finally, in the *rpoB* and *hsp65* gene analysis, *M. marium* showed homology of 100% and 86%, respectively, whereas *M. ulcerans* showed homology of 99% and 85%, respectively. On the basis of this result, the organism was identified as *M. marium* and the patient was treated with 200 mg minocycline and 500 mg clarithromycin for 2 months. After the treatment, the lesions improved, showing postinflammatory hyperpigmentation (Fig. 1B).

M. marium is a nontuberculous photochromogenic mycobacterium[1]. The optimal temperature for its growth is 30°C to 32°C, and it rarely grows at 37°C. Thus, *M. marium* infection rarely occurs on the face and most commonly affects the cooler extremities[2]. Thus far, only seven cases of infection on the face have been reported worldwide[3].

The conventional microbiological methods used for *M. marium* diagnosis are slow and solely rely on phenotypic characteristics. As delayed diagnosis is the main cause of adverse effects, rapid and accurate molecular diagnosis methods are necessary. Rapid detection of mycobacteria by using conventional broad-range PCR has previously been described in the literature[4].

Nowadays, *M. marinum* infection due to the use of pools rarely occurs because of chlorination disinfection of pool water. Thus, we report a rare and interesting case of *M. marium* infection showing an unusual location in a patient with a history of exposure to pool water. Hence, clinicians

should consider the possibility of *M. marium* 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.

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

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

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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 reported[1]. 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

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