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CONFLICTS OF INTEREST

The authors have nothing to disclose.

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Type I Lepra Reaction as the Presenting Sign of Histoid Leprosy

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Dear Editor:

A 47-year-old woman presented with a two-week history of multiple asymptomatic erythematous eruptions over the face, trunk and extremities following a transient fever. She was otherwise healthy. Physical examination revealed disseminated erythematous to violaceous plaques and nodules with tumidity and sharp margination over her face, trunk and extremities. The lesions were neither painful nor tender (Fig. 1A, B). Additionally, one asymptomatic skin-colored nodule over her right arm was noted (Fig. 1C). It lasted for 2 years and was previously misdiagnosed as dermatofibroma. Neither anesthesia nor enlarged peripheral nerves was presented. Laboratory tests revealed slightly increased C-reactive protein (11 mg/L; normal range, 0～10 mg/L), marked increased erythrocyte sedimentation rate (42 mm/h; normal range 0～15 mm/h) and elevated serum IgM (3.52 g/L; normal range, 0.63～2.77 g/L).

Skin biopsies were taken from a plaque on the face as well as the persistent nodule on the arm. Histologically, the plaque lesion showed marked dermal edema with loose lymphocyte and histiocyte infiltration (Fig. 2A, B), and the nodular lesion demonstrated dense infiltration of foamy histiocytes (Fig. 2D, E) with abundant acid-fast bacilli (Fig. 2F) which were confirmed as *Mycobacterium*

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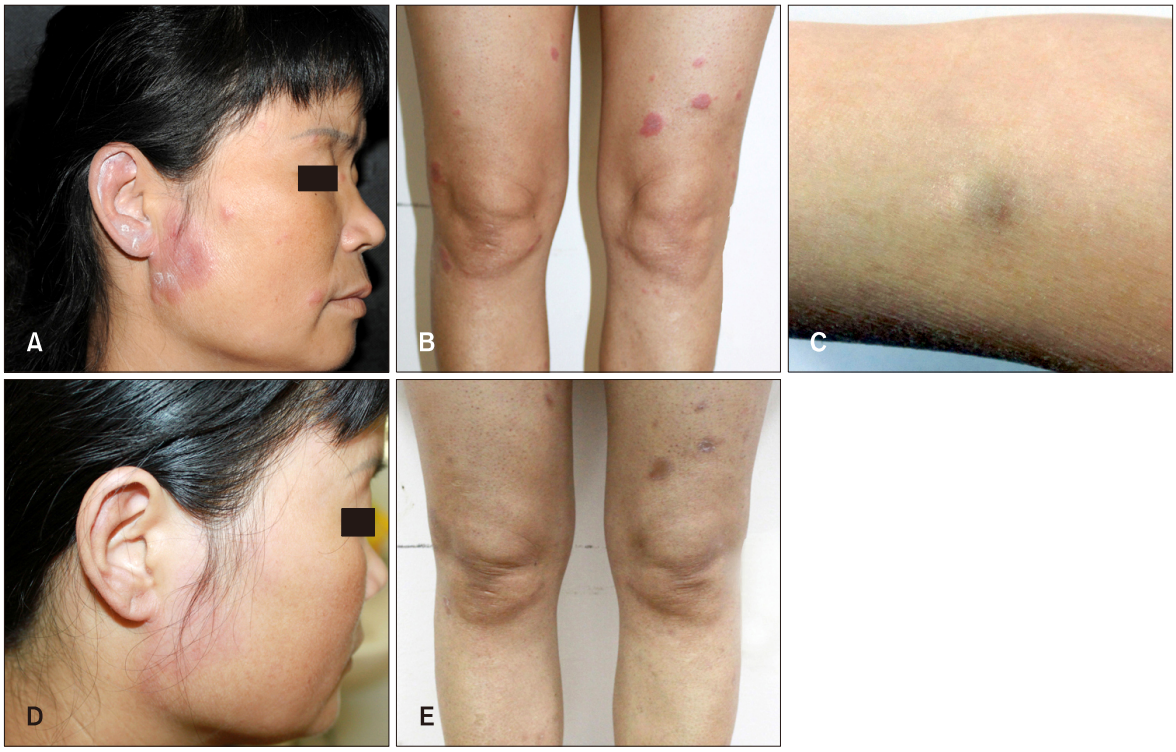

Fig. 1. Clinical appearance of the patient. Erythematous plaques over the face and extremities of the patient (A, B), and a persistent nodule on her right arm (C). The erythematous plaques resolved spontaneously within 4 weeks (D, E).

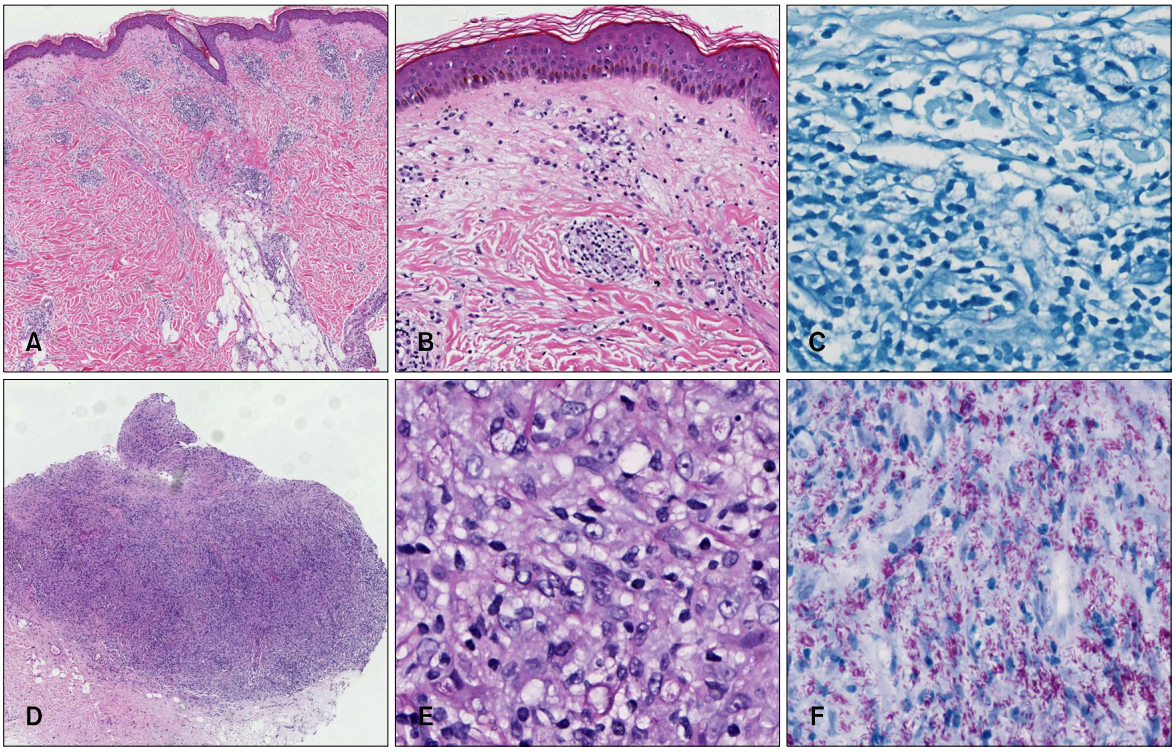

Fig. 2. Histopathology findings of skin lesions. The plaque on the face demonstrates marked dermal edema with focal lymphocyte and histiocyte infiltration (H&E; A: ×40, B, ×100) without acid-fast bacilli (C: acid-fast stain, ×400). The nodule on the arm revealed subcutaneous dense infiltration of foamy histocytes (H&E; D: ×40, E: ×400), containing numerous acid-fast bacilli (F: acid-fast stain, ×400).

leprae by real-time polymerase chain reaction. No acid-fast bacilli was detected in the plaque lesion (Fig. 2C). The patient's plaques resolved spontaneously in four weeks (Fig. 1D, E) while the nodule persisted. She was then diagnosed with type I lepra reaction related to histoid leprosy (HL). The persistent nodule revolved gradually upon multi-drug anti-leprosy treatment.

Leprosy is a chronic infectious granulomatous disease caused by *Mycobacterium leprae*, mainly affecting skin, peripheral nervous system and reticuloendothelial system. Leprosy is classified as different forms according to clinical, histopathological and immunological criteria, encompassing tuberculoid form, lepromatous form and borderline forms[1]. HL, a rare variant of lepromatous leprosy, can occur as a manifestation of drug resistance after inadequate therapy in leprosy patients or appear *de novo*. HL is characterized by skin-colored, soft nodules or plaques on apparently normal skin, especially on thighs, buttocks and arms[2]. It represents a reservoir of infection with high bacillary load but hardly detected due to the inconspicuous skin lesions[3].

Lepra reaction occurs as a result of broken balance between *M. leprae* and cellular immune response in leprosy patients. Type I lepra reaction, driven by delayed hypersensitivity to *M. leprae*, predominantly affects borderline leprosies, but is rarely reported in HL. The reactional states of leprosy are distinctive, tissue destructive, inflammatory processes that are immunologically driven. Patients may upgrade to a more resistant granulomatous posture[4]. Clinically, type I reactions are characterized by abrupt onset of purplish dusky erythematous plaques arising in clinically normal skin with or without neuritis. And these skin lesions commonly demonstrate edema and a mixture of lymphocytes and macrophages in histology. Patients often develop type I reactions in the first year of treatment, but they may occur before treatment is initiated or after it has been completed[5]. When presented, the reactional skin lesions may dominate the clinical picture, especially in HL which often appears insidiously and needs an expert look to diagnose.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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