Sir,

The Köbner phenomenon is the newly development of isomorphic lesions in the mechanically stimulated or injured skin [1,2]. This phenomenon can be seen in various disorders, such as psoriasis, lichen planus, and vitiligo; however, cases of systemic lupus erythematosus (SLE) showing Köbner phenomenon have been rarely reported [3,4].

A 40-year-old woman, suffering from SLE and lupus nephritis for over 5 years, was followed at Tsuchiura Kyodo General Hospital. A biopsy specimen taken from the malar rash showed slight liquefaction of the basal layer of the epidermis, and mild mononuclear cell infiltration in the papillary dermis. Examination by direct immunofluorescence showed linear deposition of IgM, IgG and C3 in the epidermal basement membrane. She was treated with oral prednisolone, and leukopenia and serum hypocomplementemia were improved; however, erythematos lesions on the hands were resistant to therapies. Physical examination revealed infiltrative erythemas on the fingers and dorsa of hands, predominantly involving the previously operated sites (Fig. 1). The second finger showed dactylitis (diffuse swelling). Serum antinuclear antibodies (ANA) (1:320, homogenous), anti-double stranded DNA antibodies (25.3 IU/ml; normal <10), and anti-Sm antibodies (33.1 index; normal <6.9) were detected, whereas anti-SS-A, SS-B, Jo-1, and cardiolipin antibodies were within normal ranges. Because she refused a skin biopsy again, we did not carry out a biopsy from the digital erythema.

The pathogenesis of the Köbner phenomenon is still not fully elucidated as yet. Ueki [2] proposed a second-step theory, a first non-specific inflammatory step and a second disease-specific step, in Köbner phenomenon. In the first step, many environmentally induced factors such as cytokines, stress proteins, adhesion molecules, or autoantigens translocated from intracellular areas are involved in the inflammatory phase. Subsequently, in the second step, there may be disease-specific reactions mediated by T-cells, B-cells, autoantibodies and immune complex deposition under the susceptible backgrounds. Recent studies suggest that upon epidermal injury, alarmins are released from keratinocytes, which subsequently induces activation of the innate immune systems leading to activation of acquired immunity via toll-like receptors [5].

Isomorphic response of Köbner is rarely reported in association with SLE [3,4]. One case is the development of discoid lupus erythematosus (DLE) on the recent herpes zoster scar in a patient with SLE [3], and another case is disseminated linear DLE lesions at the site of trauma [4]. Köbner phenomenon is occasionally observed in DLE [5,6]; however in the present case, cutaneous lupus erythematosus was
observed involving the previously operated sites in a patient with SLE.

Another interesting feature in the present case is the development of dactylitis. Dactylitis is sometimes seen in association with various diseases such as psoriatic arthritis, tuberculosis, injury, gout, and sarcoidosis. Although the pathomechanism of dactylitis is still unclear, dactylitis is speculated to be caused by minor biomechanics, which may be due to deep Köbner phenomenon. Thus, previous physical stress may have induced both cutaneous lupus lesion and dactylitis in this case.

CONSENT

The examination of the patient was conducted according to the Declaration of Helsinki principles.

REFERENCES