White, Fibrous, Papular Lesions Associated with Systemic Lupus Erythematosus: Is this an Ongoing Scar Following Vascular Involvement?

Sir,
We read with interest the article by Hayakawa et al., “White, fibrous, papular lesions associated with systemic lupus erythematosus” (1), because we also experienced a similar case a few years ago. Multiple depigmented papules or small macules developed in a patient with typical active systemic lupus erythematosus (SLE).

CASE REPORT
A 20-year-old Japanese woman visited our hospital complaining of butterfly-like infiltrative erythema on her cheeks. She also complained of arthralgia and persistent slight fever. Physical examination revealed oedematous erythema on her upper back and pea-sized whitish macules and brownish nodules scattered on her upper limbs (Fig. 1a).

Some of the lesions had an atrophic surface. Laboratory findings showed presence of LE cells, positive antinuclear antibody (×320, homogeneous and speckled), anti-DNA antibody (×160), anti-SS-A antibody (×64), anti-cardiolipin IgG antibody (6.0 U/ml, normal; <1.0 U/ml), and decreased complement levels. Systemic investigation, including renal, pulmonary and cardiac function, revealed no involvement of the internal organs. Histological examination of the biopsy from the facial erythema showed liquid degeneration of the basal cells and mild perivascular infiltration of lymphocytes in the mid dermis, with linear deposition of IgG in the basement membrane by direct immunofluorescence test. Histological examination of the atrophic macule showed slightly thickened collagen bundles in the deep dermis and colloidal iron staining showed partially positive mucin findings between collagen bundles, but no evidence of vasculitis (Fig. 1b).

We examined the stimulatory effect of the patient’s serum on the proliferation of 3T3 fibroblasts, using a method described earlier (2). The results showed that stimulation with the patient’s serum (10%) gave a slight increase (980 ± 55 dpm, vs. 220 ± 46 dpm in unstimulated cells) compared with that of 4 SLE patients without such cutaneous features (757 ± 132 dpm) and 4 normal volunteers (607 ± 105 dpm), however, the difference was not significant.

Our case presented mainly with macular lesions rather than nodular lesions, otherwise the clinical and histological features were similar. We believe that the variety of these skin lesions is a sequential event. We speculate that these cutaneous manifestations may be ongoing scars following vascular damage associated with SLE and that the deposition of mucin is secondary.

REFERENCES

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