DISCUSSION

The cutaneous sequelae of burns are usually temporary loss of pigment, hypertrophic scar and keloid. Infrequently skin neoplasia such as squamous cell carcinoma, basal cell carcinoma, malignant melanoma and sarcoma develop within the burn scar. Because ILVEN was developed after a burn in our patient, it is suggested that the possible mechanism for the development of ILVEN may have been that the burn acted as a simple trauma or induced an impairment of the immune function which was responsible for the onset of ILVEN. ILVEN is typically resistant to various therapies including corticosteroids, tar, dithranol and cryotherapy (3, 4). The skin lesions in our patient also responded poorly to various therapies. There are several reports that topical calcipotriol is an effective and safe therapy for the treatment of ILVEN (4, 5). Our patient was treated with topical calcipotriol twice a day for 5 months and showed marked improvement.

REFERENCES


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Anti-centromere Antibodies in 2 Patients with Discoid Lupus Erythematosus and No Signs of Systemic Sclerosis

Sir,

Patients with typical discoid lupus erythematosus (DLE) only rarely have associated systemic sclerosis (SS) (1, 2) and there are no reports of them having anticientromere antibodies (ACA) in the absence of SS signs.

CASE REPORTS

Case 1

A 36-year-old woman presented with a 6-year history of DLE of the scalp, face and ears. She had developed Raynaud’s phenomenon at the age of 20 years. In 1979 she had only speckle-patterned IgG ANA at a ¢nal titre of 1/160. She was treated with chloroquine and the DLE lesions cleared and were then controlled every 2 years. In 1996, she developed centromere-patterned ANA and antimitochondrial antibodies at a ¢nal titre of 1/1280. All other routine and immunological¢ndings were within normal limits. No signs of sclerosis have been observed so far.

Case 2

A 56-year-old woman was seen in 1984 with a 40-year history of DLE of the scalp, face and ears. She had speckle-patterned IgG ANA at a ¢nal titre of 1/40 and low total complementaemia (CH50 $200) without immunocomplexes. Treatment with chloroquine was successful during relapses. The cutaneous lesions were silent for 2 years, but the ANA titre progressively rose, acquiring in 1996 a centromeric pattern at a ¢nal titre of 1/2560. Raynaud’s phenomenon and other signs of SS were absent.

DISCUSSION

ACA are usually present in patients with a limited form of SS and relatively good prognosis. They have also been reported occasionally in patients with systemic lupus erythematosus, rheumatoid arthritis, Raynaud’s disease, Sjögren syndrome, primary biliary cirrhosis, immune haemolytic anaemia and thrombocytopenic purpura (3). They have also been found at low titre in healthy subjects and in patients with non-autoimmune diseases (4).

In our 2 patients, ACA developed long after the onset of DLE and had a high titre. Very rarely have patients been observed with DLE/SS combination and even more rarely with a serology reminiscent of SS. ACA was found in only 1 of the 6 DLE/SS cases described by Sasaki & Nakajima (1). As in all such patients in whom DLE preceded SS, ACA may be regarded as a sign of impending SS development. Whether this is the case in our 2 patients remains to be seen.

REFERENCES


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Sir,

Although erythema multiforme (EM) is known to be associated with a number of infectious and non-infectious diseases, it has rarely been reported to be associated with a cytomegalovirus (CMV) infection. We report here a case of CMV mononucleosis in a healthy adult who presented with EM-like eruptions as the initial symptom.

A healthy 23-year-old woman presented with a 2-day history of erythematous eruptions on the dorsa of the feet. She was otherwise asymptomatic and had taken no drugs for 4 weeks before the onset of the rash. There was no significant family or past medical history and she did not have herpes simplex infection. On examination, positive physical findings were confined to the skin. The dorsal aspect of the feet exhibited oedematous erythema with an annular configuration, which closely resembled EM (Fig. 1). Some lesions coalesced with adjacent lesions that had formed polycyclic patterns. In addition, a few lesions were also seen on her legs. There was no involvement of the mucous membranes and no joint or muscle complaints were observed. She had no fever or lymphadenopathy. Laboratory studies gave the following values: WBC 4200/μl, with a differential cell count of 36% neutrophils, 4% monocytes, 37% lymphocytes and 23% atypical lymphocytes. The platelet count decreased to 10.7 x 10⁴/μl. The biochemical parameters were normal, except for aspartate aminotransferase (AST; GOT) 215 IU/l; alanine aminotransferase (ALT; GPT) 276 IU/l; alkaline phosphatase 521 IU/l; and lactate dehydrogenase 934 IU/l. A high titre of IgM to CMV was detected by the EIA technique (7.24, normal <0.80), thus indicating a recent CMV infection. Hepatitis screening, including tests for hepatitis A, B and C was negative. The titre of VCA IgM to Epstein-Barr Virus (EBV) and EBNA were, respectively, × > 10 and ×10. Auto-antibodies, such as antinuclear antibody, were all negative. A skin biopsy of the lesion revealed focal vacuolar degeneration of the basal layer, slight oedema in the papillary dermis and lymphohistiocytic infiltrate in the upper dermis, which were all consistent with EM. From the clinical and laboratory data, she was diagnosed to have CMV mononucleosis with EM-like eruptions. After symptomatic treatment with topical steroids (0.05% clobetasol propionate), the skin lesions gradually resolved after 2 weeks, leaving only slight residual pigmentation. The haematological and liver function abnormalities returned to normal 4 weeks after the onset of infection, suggesting spontaneous normalization to be the natural course of CMV mononucleosis. A rubelliform rash has been described in CMV mononucleosis which affects the lower legs and lasts up to 2 days. Other skin lesions may also occur, such as urticaria, erythema nodosum, purpura and cutaneous vasculitis, which appear as papules and plaques in a partly annular configuration. EM has only rarely been associated with CMV mononucleosis as far as we could ascertain based on a search of the literature (1, 2).

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