Sir, We describe the case of a man who developed remitting fasciitis without eosinophilia. In 1974, Schulman described a new syndrome characterized by the rapid onset of a woody induration of the skin and subcutaneous tissue with a striking peripheral blood eosinophilia and histological evidence of thickened fascia [1]. This disease was further characterized by Rodnan et al. [2], who proposed the term ‘eosinophilic fasciitis’ (EF). In most cases, the clinical course is progressive and results in joint contractures, so early treatment is necessary. It has been believed that once these cases improve they will not relapse, although in our patient the clinical course was atypical—he experienced recurrent relapses and remissions without any treatment. In addition, throughout his clinical course no abnormality was seen in his laboratory data. No infiltration of eosinophils was detected histopathologically. In this report we demonstrate that some cases of fasciitis may take a clinical course that is different from that of the typical EF.

A 50-yr-old man with a 15-month history of remitting dull pain and swelling of the extremities was admitted to our hospital in March 1999. He claimed that his symptoms continued for a few weeks then remitted without any treatment; such episodes occurred every 2–3 months. He had no known exposure to any drugs and no history of Raynaud’s phenomenon.
Physical examination showed diffuse swelling of the left forearm and lower legs. Sclerotic change of the skin was absent. There was no muscle atrophy, weakness, joint tenderness or contractures. Neurological examinations were normal.

At his first admission, laboratory examination revealed no eosinophilia. Erythrocyte sedimentation rate (ESR) was normal. Antinuclear antibodies and rheumatoid factor were negative. The levels of creatine kinase, serum gamma-globulin and lactate dehydrogenase (LDH) were not elevated. A biopsy specimen obtained from his left calf demonstrated marked thickening of the fascia and lymphocyte infiltration into the fascia and muscle, but no eosinophils were found (Fig. 1). The patient was diagnosed as having fasciitis and was treated with prednisolone 35 mg/day. A few days after administration of prednisolone, swelling of the extremities showed rapid improvement, so the dose of prednisolone was gradually tapered. Throughout this period his symptoms did not relapse.

It has been reported that peripheral eosinophilia or pathological infiltration of eosinophils may be absent at the chronic stage of EF [3], but the laboratory findings for our patient showed no elevation of eosinophils throughout the clinical course. Lakhanpal et al. [4] reported that the occurrence of hypergammaglobulinemia, increased ESR and peripheral eosinophilia was approximately 35, 29 and 63% respectively. In our patient, laboratory findings (ESR, gamma-globulin, LDH) were not elevated even in the active symptomatic phase.

Barnes et al. [5] reported the unique histological findings of full-thickness fascial biopsies in patients with EF. The fascia in EF was found to be 2–15 times thicker than in normal subjects. Moreover, they reported that, surprisingly, only occasional eosinophils were found in specimens obtained during the chronic stage of the disease. In our case, the biopsy specimen was obtained about 15 months after the onset of the first symptoms, so it might have been taken during the chronic stage of EF.

An extraordinary feature of the fasciitis in our case was the atypical remitting clinical course without any treatment. In most cases reported in the literature, patients with EF have excellent responses to steroid therapy with good prognosis and without any relapse. There is no report of relapsing fasciitis during the clinical course, and our case may therefore suggest a different disease. There are some reports of EF cases developing into systemic sclerosis (SSc), so it is important to observe these patients carefully for a long time [6].

Discrimination between EF and SSc has been reported in some papers, and various features serve to distinguish EF from SSc. First, pathologically, the inflammatory cell infiltration is often more pronounced in the dermis or dermossubcutaneous junction in SSc. Secondly, regarding the clinical features, Raynaud’s phenomenon, antinuclear antibodies and visceral organ involvement are uncommon in EF, in contrast to SSc. Thirdly, EF, unlike SSc, often shows an excellent response to prednisolone treatment. In our case, histopathological examination revealed thickening of the fascia and infiltration with lymphocytes, but we found no fibrotic change in the dermis, as occurs in SSc. Additionally, laboratory findings showed no abnormalities of antinuclear antibodies, physical examination showed no visceral organ disease and the patient responded to prednisolone therapy immediately. Therefore, the diagnosis was more likely to be EF than SSc; the atypical clinical course suggests that the patient suffered from some variant form of fasciitis without eosinophilia.

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Fig. 1. Loci of inflammatory cell infiltration in the fascia consisting mainly of small lymphocytes. Fibrosis and capillary proliferation are also seen. Haematoxylin/ eosin, × 50.