Stress fractures of the legs and swelling of the ankles in a patient with lupus: a diagnostic dilemma

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Abstract

A patient with systemic lupus erythematosus (lupus) is described, in whom stress fractures of both legs developed, accompanied by swelling of both ankles. The swelling of the right ankle associated with fracture mimicked lupus synovitis, but resolved with healing of the fracture. On the left side the swelling represented true lupus synovitis coexistent with the tibial stress fracture. This presentation posed a diagnostic dilemma. It is necessary to maintain vigilance in the diagnosis of these fractures in lupus.

Stress fractures of long bones have been reported infrequently in patients with rheumatoid arthritis.¹⁻⁷ Osteoporosis, angular joint deformities, and enhanced activity after reconstructive surgery have been considered important causes in the pathogenesis of these fractures.¹ Such fractures have not been described in patients with lupus, as far as we know. We describe a patient with systemic lupus erythematosus, presenting initially with discoid lesions, who developed stress fractures of both legs. The association of these fractures with swelling in the adjacent ankle joint posed a diagnostic dilemma: true synovitis coexistent with a stress fracture or a reactive (sympathetic) effusion in the joint adjacent to a stress fracture.

Case report

A 51 year old white woman with a 24 year history of discoid skin lesions and a photosensitive malar rash spontaneously developed pain and swelling of the right ankle in May 1987. The pain was aggravated by walking. Radiographs taken at the time were normal. She was referred for rheumatological consultation a month later because of the persistence of her complaints. She had taken prednisone 15 mg every other day and chloroquine phosphate 250 mg a day for the discoid skin lesions. There was some evidence of osteoporosis on old x rays. Skin examination confirmed the presence of discoid skin lesions on her back. On musculoskeletal examination, stress pain was elicited in both the tibiotalar and the subtalar components of the right ankle joint, and an effusion was detected. Straw coloured fluid (2 ml) was aspirated from the right ankle; there were a few cells, no crystals, and cultures were negative. The initial impression was that of a synovitis, and the question of systemic lupus erythematosus was raised. Both rheumatoid factor and antinuclear factor were negative, anti-DNA antibodies were not detected, and serum complement concentrations were normal. The patient was treated with an intra-articular injection

of methylprednisolone, with improvement. A radiograph taken three months later showed an old stress fracture of the distal ends of the right tibia and fibula (fig 1). In October 1987 the patient presented with a sudden onset of pain and swelling of the left ankle, with inability to bear weight. Physical examination showed a large oral mucosal ulcer, 12 actively inflamed joints, including a swollen left ankle, and discoid skin lesions on the scalp and trunk, which were felt to be active. The left ankle was aspirated and injected with methylprednisolone, but failed to improve. Radiographs of both ankles were normal. A bone scan performed in December 1987 because of persistent pain and swelling in the left ankle showed a focal area of intense uptake in the distal left tibia (fig 2). A gallium scan was positive in the same area. Radiographs of the left ankle showed a poorly defined 3.5×1.4 cm geographic lytic lesion in the distal tibia with irregular sclerosis along its medial aspect (fig 3). A bone biopsy was performed to rule out osteomyelitis or a neoplastic process. The biopsy showed a stress fracture in a thin porotic bone with a slightly raised ridge of sclerotic bone. On microscopic examination small fragments of necrotic bone and granulation tissue were seen, but no poly-

Figure 1 Old transverse fractures of the right distal tibia and fibula.

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Figure 2 Bone scan using ^{99m}Tc methylene diphosphonate, showing intense uptake in the left distal tibia.

Figure 3 Radiograph of the lowermost third of the left tibia and fibula, showing a poorly defined 3.5 × 1.4 cm lytic lesion in the distal tibia with irregular sclerosis along its medial aspect.

morphonuclear cells were noted. Evidence for remodelling and reactive bone formation consistent with a healing process was noted. Cultures of the bone specimen were negative. Despite rest, non-weight-bearing, and non-steroidal anti-inflammatory drug treatment the ankle pain and swelling persisted. Radiographs taken in February 1989 showed complete healing of the lesion of left lower tibia. Over the next few months the patient developed a further flare of her discoid lesions as well as a polyarthritis, and her prednisone was increased to 15 mg a day, with subsequent improvement in all her clinical findings, including the left ankle.

Discussion

The presentation of an acutely inflamed right ankle was at first thought to represent synovitis in this patient, particularly as her initial radiographs showed no abnormality. As there was no other evidence for active lupus, the presence of

an old stress fracture could be seen three months later, and the pain and swelling of the right ankle resolved completely with healing of the fracture, however, this suggested that the inflammation might have been a reactive effusion secondary to the adjacent fracture. The swelling of the left ankle was associated with a radiologically apparent lytic lesion on the distal tibia, and not with the transverse line of focal medullary sclerosis in the metaphyseal region of the long bone, typical for a stress fracture.¹ An open bone biopsy confirmed the diagnosis of a stress fracture, however. As the patient had presented with an ankle effusion in conjunction with the right sided stress fracture it was assumed that the same process occurred on the left side. Despite complete healing of the lesion of the left tibia, however, the left ankle continued to be inflamed, and indeed further evidence for synovitis was provided with the development of a polyarthritis during a flare of the discoid lesions. We therefore feel that the left ankle represented true synovitis coexistent with a stress fracture in an adjacent bone.

Stress fractures are well recognised, though uncommon in rheumatoid arthritis.¹⁻⁷ Factors thought to be relevant in the pathogenesis of these fractures include osteoporosis, with or without prior corticosteroid treatment, and abnormal mechanical stresses. The latter may result from angular joint deformities, joint ankylosis, and enhanced activity or unaccustomed exercise after reconstructive joint surgery. A search of published work produced no other cases of stress fractures of the long bones in a patient with lupus. This may reflect the fact that although patients with lupus are often treated with corticosteroids and suffer from osteoporosis, they do not have the other mechanical stresses found in patients with rheumatoid arthritis. On the other hand, as no prospective study was performed to assess the incidence of stress fractures in patients with lupus, the rarity of this disorder may reflect a lack of vigilance and misdiagnosis of such fractures. Serial radiological studies are not usually carried out in patients with lupus as they are in those with rheumatoid arthritis, and possibly these fractures are missed. Our patient showed the same diagnostic difficulty posed by patients with rheumatoid arthritis who develop evidence of synovitis in a joint adjacent to the affected bone. It is necessary to maintain vigilance in the diagnosis of these fractures and in defining the relationship with adjacent joint swelling in patients with lupus.

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