Insufficiency fracture of the tarsal navicular in a patient with rheumatoid arthritis

Sir, Insufficiency fracture is one type of stress fracture that occurs in weakened bone, as described by Pentecost et al. [1], and are sometimes seen in patients with rheumatoid arthritis (RA). Until recently, no report of insufficiency fractures in the tarsal navicular in patients with RA had been found, while several reports of fatigue fractures in the tarsal navicular in athletes have been published [2–4]. We describe a rare case of an insufficiency fracture occurring in the tarsal navicular of a patient with RA.

In December 2002, a 69-yr-old woman with RA, who had been visiting our hospital, complained of left midfoot pain. She had no history of any acute trauma, and the pain increased when she stood and walked. She had suffered from RA for 31 yr, and at the time of the occurrence of the foot pain she was receiving 1000 mg/day of sulphalazine and 5 mg/day of prednisolone. C-reactive protein in serum was 4.5 mg/dl, rheumatoid factor 205 IU/ml, Ca 4.3 mg/dl, and inorganic phosphorus 3.7 mg/dl. Her bone mineral density in lumbar spine 2–4, which we had measured in 2001 by dual-energy X-ray absorptiometry (Lunar DPX-MD densitometer), had shown significant osteopenia (bone mineral density 0.733 mg/cm², T-score: 1.192 ± 3.1SD, Z-score: 0.913 ± SD).

The patient had undergone a forefoot operation for a deformity of metatarsophalangeal (MTP) joint involvement due to RA in 2000. The first MTP joint had been fixed, and MTP joints 2–5 were resected as previously described by Clayton [5]. Upon examination, tenderness of the foot was found over the tarsal navicular body. Ecchymosis, swelling and deformity were not noted. In a standing position, the arch height of the foot was approximately 25° horizontally medial to the talus head. In the frontal plane, it turns and overlaps the talus by 2–4°. The mechanism by which stress fractures occur in a tarsal navicular has been described previously [8, 9]. As the hind foot progresses from eversion to inversion during heel strike to toe-off, the navicular slides 5–10° horizontally medial to the talar head. In the frontal plane, it turns and overlaps the talus by approximately 25°.

Biomechanical analysis of navicular motion during the gait cycle reveals most of the force at the central third of the navicular bone. In our patient, the fracture occurred in a more medial part, but not at the central third of the tarsal navicular. Stress fractures of the tarsal navicular in athletes generally occur due to impingement stress between talus and cuneiform bone. Also, the fractures often occur at the central third of the tarsal navicular; this part of it is an avascular area [10]. Thus, in our case, another mechanism different from that in athletes is considered to be the cause of the onset of the fracture.

Diagnoses of the fractures are made with X-rays and MRI. The sensitivity of plain X-rays for navicular stress fractures is only
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Necrotizing scleritis associated with rheumatoid arthritis: long-term remission with high-dose infliximab therapy

Sir, Scleritis is a well-recognized and severe manifestation of rheumatoid eye disease [1, 2]. Treatment is systemic, usually with steroids and other immunomodulating drugs [1–3]. We report a resistant case of necrotizing scleritis associated with rheumatoid disease, which has been successfully treated for over 2 yr with infliximab.

A 51-yr-old woman presented in 2002 with bilateral painful red eyes associated with photophobia but no visual disturbance, orbital discharge or evidence of infection. She had a 30-yr history of seropositive erosive rheumatoid arthritis (RA) which had not responded over the years to numerous disease-modifying drugs, including penicillamine, methotrexate, sulphasalazine and hydroxychloroquine. Ten years earlier she had had two MCP joint replacements. The past history included hypothyroidism, alopecia areata and hypertension. At presentation with eye disease, she was taking prednisolone 15 mg daily, penicillamine 750 mg daily, hydroxychloroquine 200 mg daily, thyroxine, amlopidine, frusemide and fenbufen.

Ophthalmological examination (Fig. 1) revealed perilimbal injection in the right eye (ciliary flush) and generalized redness of the bulbar conjunctiva of the left eye. There were keratic precipitates in both eyes with vision in right eye 6/6 and left 6/9. She had mild flare, resulting from extra protein in the aqueous. The findings were compatible with bilateral diffuse scleritis with anterior chamber activity.

Systemic examination revealed evidence of active synovitis with five swollen and six tender joints (out of 28), a DAS28 score of 5.84, long-standing rheumatoid nodules, but no signs of cutaneous small vessel vasculitis either around the nails or in association with rheumatoid nodules.

The scleritis was initially treated with continued oral prednisolone and topical prednisolone acetate 1%, one drop every 2 h. This had no effect over several months, so systemic treatment was changed, with an increase in oral prednisolone to 60 mg daily and substitution of sulphasalazine for penicillamine with no change in hydroxychloroquine. After 8 weeks a lack of effect led to the addition of oral cyclosporin 150 mg twice daily (2.6 mg/kg) and prednisolone was tapered to 25 mg daily. A further 6 weeks later no response had occurred. Signs of bilateral scleritis persisted and new areas of necrotizing scleritis and peripheral ulcerative keratitis with blurring of vision developed. Cyclosporin could not be increased because of hypertension, and high-dose methotrexate was avoided because of previous intolerance of this agent at a dose of 2.5 mg per week, in combination with sulphasalazine and n-penicillamine. In view of this, infliximab was commenced at a dose of 3 mg/kg at 0, 2 and 6 weeks followed by an infusion every 8 weeks, with methotrexate 5 mg/week.

The symptoms and signs of scleritis responded dramatically after three infusions of infliximab, and visual acuity improved to 6/6 in both eyes. Methotrexate was tolerated and so the dose was increased to 7.5 mg/week, and prednisolone was reduced to 12.5 mg daily. This led to a relapse in orbital pain and redness 2 weeks before the fourth infusion was due. Methotrexate was increased to 10 mg/week, but despite this the same pattern of relapse in scleritis recurred between the fourth and fifth infusions. Therefore the dose of infliximab was increased to 5 mg/kg at the fifth infusion. Subsequently, this dose every 8 weeks has been completely effective in controlling scleritis. At present she has received a total of 16 infliximab infusions over a period of 2 yr and the scleritis has become totally quiescent, with residual areas of scleral discoloration from previous disease and some inferior corneal scarring from previous keratitis. Prednisolone has been reduced to 6 mg, methotrexate continued at 12.5 mg, and both sulphasalazine and hydroxychloroquine discontinued. The peripheral joint disease has also improved, with resolution of morning stiffness and a fall in the DAS28 score from 5.92 before infliximab to between 3.45 and 3.96 over the follow-up period.

Fig. 1. Post phenylephrine dilatation of left and right eyes showing active inferior scleritis and areas of old scleritis, where thinning of the sclera shows blue-grey.