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Bilateral atraumatic avulsion fracture of the calcaneal tubercle in osteomalacia during fluoride therapy—a case report

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Submitted 98-07-25. Accepted 98-09-17

A 56-year-old woman with insulin-dependent diabetes mellitus, compensated renal failure and osteoporosis treated with fluorides (150 mg natriumfluorophosphate/day) for the last 6 years complained of bilateral painful swelling of the ankles for the past 4 months. She could walk only with elbow crutches.

Radiographics showed that both ankles were normal, apart from low grade arthrosis. A 3-phase radioisotope bone scan demonstrated increased uptake in both os calcis, in the left upper tibia and in the right third, fourth and fifth ribs. A Looser zone was present in the left upper tibia which had not been visible 4 weeks previous. MRI of both ankles revealed arthrosis in both ankle joints and also extensive edema of the marrow mainly in both dorsomedial aspects of the calcanei. On the left side, dorsal and cranial interruptions of the cortex in the insertion area of the Achilles' tendon were evident.

Laboratory tests showed a positive rheumatoid factor and a positive titer for antinuclear factor, however, clinically there was no additional sign of inflammatory joint disease or collagenosis. 25-hydroxyvitamin D was low—i.e., 15 nmoL/L (normal range 43–94 nmoL/L), and 1,25-dihydroxyvitamin D was normal. Calcium, measured in serum and urine, parathyroid hormone and alkaline phosphatase were normal, creatinine clearance, 73 mL/min, and phosphate excretion 0.4 g/24 h (normal range 0.46–1.4 g/24 h) were low borderline. ESR was 29 mm/1h, c-reactive protein and leukocyte count were normal. There were no sensory disturbances. Iliac crest bone marrow biopsy showed moderate osteomalacia, with widened osteoid areas and signs of fluorosis.

During physiotherapy both calcanei became increasingly painful. New radiographs, 4 weeks later, showed bilateral avulsion fractures of the Achilles' tendon (Figure 1). An ultrasound scan had been normal 4 days before. Because of contracture, both Achilles' tendons could be reinserted by screw fixation only after tendon lengthening (Figure 2). Both ankles were immobilized in plaster casts for 2 months after surgery. At the follow-up 1 year postoperatively, standing on tip toes was possible with little aid and the fractures had healed. Therapy for osteomalacia continues with alphacalcidol and calcium carbonate.

Discussion

Avulsion fracture of the Achilles' tendon is rare and often associated with metabolic disturbance (Bierwag 1970, Cooper and Heckman 1989, Biehl et al. 1993, Cole et al. 1995, Itokazu et al. 1996). In our case, the avulsions were not traumatic.

Operative treatment is preferred, since closed treatment does not lead to satisfactory functional results (Brunner 1971). In our case, bilateral Achilles' tendon lengthening was necessary because of contracture and osteoporosis, in order to achieve stable reduction and reduce tractive forces (Itokazu et al. 1996).

Spontaneous fractures in generalized osteoporosis are ascribed to deteriorated microarchitecture of the trabecular structure of cancellous bone (Verlooy et al. 1991). Fluorides stimulate the osteoblasts and formation of new bone which leads to an increase in the bone mineral density (Murray and Ste-Marie 1996, Talbot et al. 1996) and to a diminished fracture rate (Turner et al. 1996a). Thus therapy with fluorides is widely practiced in osteoporosis (Abbott et al. 1996) and in medium-term studies shows positive results, as regards bone mineral density, and a lower fracture

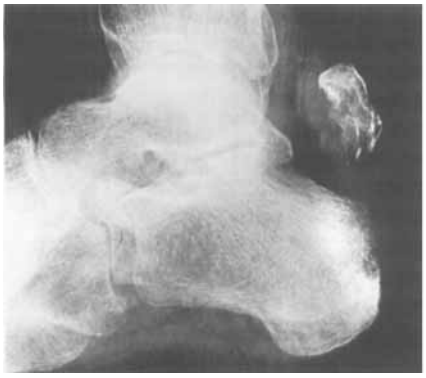

Figure 1. Avulsion fracture of right Achilles' tendon. Identical findings on left side.

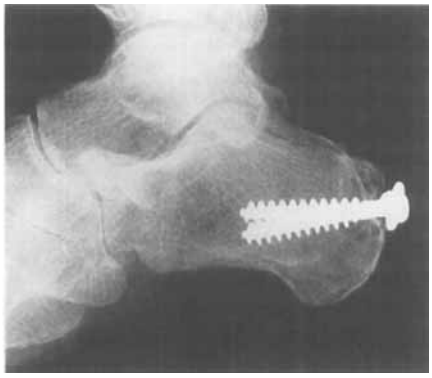

Figure 2. Refixation by screw osteosynthesis.

rate (Geidel 1995, Pak et al. 1996). Slow-release fluoride in combination with high-dose calcium seems especially effective (Deal 1997, Dure-Smith et al. 1996). However, excessively high doses of fluorides can lead to inadequate formation of new bone (Turner et al. 1996b, Ringe 1997) and thus to an increased fracture rate (Schnitzler and Solomon 1986, Orcel et al. 1990). Patients with renal failure may develop osteomalacia (Turner et al. 1997). Osteomalacia causes disturbed mineralization and thus raises the fracture rate. Fluoride therapy causes an additional softening of the bone; therefore osteomalacia is an absolute contraindication for fluoride therapy. Our patient received fluoride therapy for 6 years, which produced brittle bones in histologically proven osteomalacia, with signs of fluorose (Orcel et al. 1990). Consequently, the radiographs showed no clear signs of osteoporosis. Before beginning fluoride therapy, osteomalacia must be excluded. This is possible with an iliac crest punch or measurement of vitamin-D levels.

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