the bone scan is negative and the cause of the pain is still unknown, magnetic resonance imaging will help to rule out pathologies such as spinal cord anomalies, adolescent disc prolapse, and tumours.

Scoliosis develops in osteoid osteoma secondary to paraspinal muscle spasm.[6] The nidus of osteoid osteoma is richly vascularised and innervated. This is thought to account for the relief with salicylates.[2]

Even though the natural history of osteoid osteoma may be one of spontaneous remission, the treatment of spinal osteoid osteoma is surgical removal of the nidus by en bloc excision. This is because the time interval before remission may range from two to eight years and the scoliosis can become structural during this period.[6]

Percutaneous radiofrequency ablation is gaining popularity in the treatment of osteoid osteomas. However, spinal osteoid osteomas should only be treated by radiofrequency ablation if the nidus is located at least 1 cm away from vital structures, in order to prevent neurological complications.[7] If the nidus is removed, the patient is usually relieved of pain at once.[1 2] Scoliosis usually resolves completely if the excision is undertaken within 18 months of the start of symptoms.[4] Complete excision of the lesion results in a cure and recurrence is very unusual.[1]

Final diagnosis

Osteoid osteoma of superior left facet of second lumbar vertebra.

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IMAGES IN MEDICINE

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Charcot's foot: advanced manifestation of diabetic neuropathy

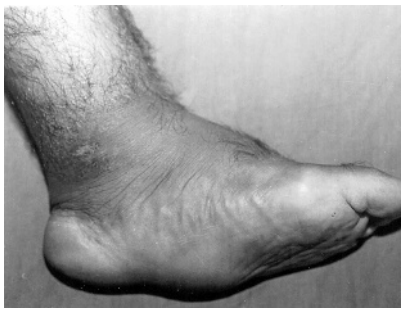

Figure 1 Clinical photograph of the left foot with ''rocker bottom'' deformity.

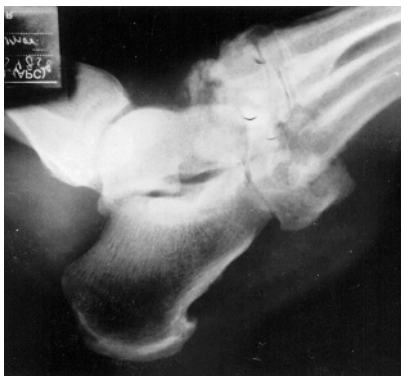

Figure 2 Radiograph of left foot with vertically displaced cuboid and destroyed tarsal and metatarsal bones.

A 48 year old man, a known diabetic for 14 years, who had poor compliance to treatment was admitted with left lower limb pain and deformity of his left foot for two months. He did not have a history of alcohol intake or promiscuity. On evaluation, he had bilateral loss of pain, touch, temperature, and vibration sense below the ankle joint. His vibration perception threshold was increased to 46 mV on the left side and 38 mV on the right side (normal <25 mV). The pressure sense tested with a SG 5.07 monofilament over both feet was impaired and he had ''rocker bottom type deformity'' of the left foot (fig 1). His body mass index was 24 kg/m^2. He was hypertensive and had bilateral advanced non-proliferative diabetic retinopathy. On investigation, random blood glucose was 26 mmol/l, serum creatinine 3.1 µmol/l, and 24 hour urine protein was 6.4 g. His Venereal Disease Research Laboratory test was negative and he had a normal total leucocyte count and erythrocyte sedimentation rate. Radiography of the left foot was suggestive of Charcot's foot (fig 2). ^{99m}Tc methylene diphosphonate (MDP) bone scan showed increased tracer uptake in the region of the left foot suggestive of increased osteoblastic activity (fig 3). Magnetic resonance imaging (MRI) of the left foot revealed diffuse soft tissue oedema with maintained subcutaneous fat and disorganised and malaligned tarsal bones. Subchondrial cyst, marrow oedema, and inferior displacement of cuboid (fig 4) were additional features. The overall picture was of chronic neuroarthropathic joint due to diabetes mellitus. He was advised to have a total contact cast followed by special footwear. Additionally calcium, vitamin D, and alendronate 40 mg per day were prescribed. Bisphosphonates have been used in the treatment of Charcot's foot because increased osteoclastic activity secondary to autonomic dysfunctions has been documented. Calcium and vitamin D were started to prevent bisphosphonate induced osteomalacia. Realignment surgery is planned.

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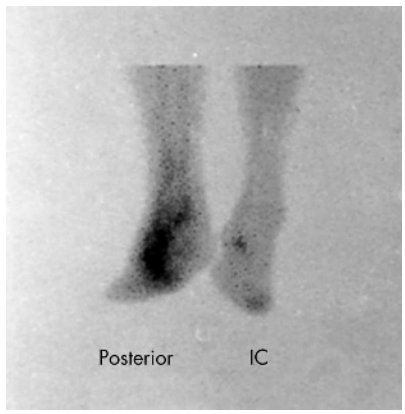

Figure 3 ^{99m}Tc MDP bone scan showing increased tracer uptake in the midtarsal region.

Figure 4 MRI of the left foot showing subcutaneous tissue oedema with malaligned tarsal and metatarsal bones and subchondrial cyst formation.