Case report

Atraumatic avascular necrosis of the head of the talus

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Abstract

Avascular necrosis of the talus is well documented after trauma. This is usually confined to the body and dome of the talus with sparing of the head. A case report of atraumatic avascular necrosis of the head of the talus only is presented.

We discuss the various associations with avascular necrosis and the investigation, differential diagnosis and management in such a case.

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1. Case report

A 21-year-old woman presented with a 12-month-history of pain over the anteromedial aspect of her left foot. The patient had to leave her employment as a racehorse trainer due to the pain upon weightbearing. There was no history of trauma, alcohol abuse, steroid use or any other significant medical history.

Examination of the left foot revealed, localized tenderness over the talonavicular joint. There was neither tenderness along the tibialis anterior and tibialis posterior tendons, nor any lateral joint line tenderness. Movements elicited a restriction of plantarflexion and dorsiflexion, with a mild reduction in inversion and eversion. There was normal mid-tarsal movement of the foot.

Lateral and oblique radiographs showed a cystic lesion in the talar head, and sclerosis and osteophytes at the talonavicular joint (Figs. 1 and 2). An MRI showed at the talar head a well-demarcated area of low signal on a T1 weighted image (Fig. 3). The lesion was also characterized on a MRI STIR sequence as high signal with a surrounding zone of less marked signal change (Fig. 4). The appearances were consistent with avascular necrosis of the head of the talus.

Due to the persistent symptoms and the failure of conservative management, the patient underwent a talonavicular fusion with tricortical iliac crest bone grafting 15 months after the onset of symptoms. Histology of the operative biopsy showed necrotic bone with active remodeling. Postoperatively, the patient was placed in a short leg cast for 3 months and she made a good recovery. The fusion at the talonavicular joint was clinically and radiologically united at 3 months when she began full weightbearing (Fig. 5). She was painfree and was able to return to work, and has been asymptomatic for the last 3 years since her surgery. An X-ray 3yrs after surgery (Fig 6) shows fusion at the talonavicular joint.

2. Discussion

Avascular necrosis of the talus is well documented after trauma. The incidence has been reported to be as high as 50% for Hawkins Type II and between 75 and 100% for Type III talar neck fracture dislocations [1]. The nature of the extraosseous and intraosseous blood supply of the talus has been well described by Mulfinger et al. [2]. The nature of this blood supply is the basis for post traumatic avascular necrosis involving the body of the talus, as compared to the head or neck of the talus.
Atraumatic avascular necrosis has been reported at other locations, such as the femoral head [3], humeral head [4], metatarsal heads and femoral condyles. Avascular necrosis is associated with systemic disease, in particular, diabetes. Minor trauma has been associated with avascular necrosis but this was limited to the body of the talus [5]. Schmidt and Romash [6] have reported on a case of atraumatic avascular necrosis of the head of the talus, but this was associated with alcohol abuse.

More commonly avascular necrosis has been associated with alcohol abuse [7,8], corticosteroid use [9], peripheral vascular disease [10], sickle cell disease [3] and polycythaemia vera [11]. Uncommon associations of avascular necrosis of the body or dome of the talus have been reported with hyperuricaemia [12], pancreatitis [13] and systemic lupus erythematosus [14]. This is the first reported case of atraumatic avascular necrosis of the head of the talus without any predisposing factors.

Investigations for avascular necrosis should include a plain radiograph, which in the talus may show subchondral resorption of bone. In fact, a modified staging of avascular necrosis for the ankle is based on the radiological appearance.
of a plain x-ray (Table 1). However the definitive investigation is an MRI scan which is more sensitive than plain radiography, computed tomography, or radionuclide bone scanning in detecting avascular necrosis [15].

The differential in this case would be osteochondritis dissecans, which has been reported by Powell and Whipple [16] to be present in the head of the talus. Our case differs with there being a lack of trauma and more extensive involvement of the talar head, with degenerative changes involving the talonavicular joint.

The management of symptomatic avascular necrosis of the talus is limited. Conservative management includes analgesia and restricted weight bearing. Operative management initially includes core decompression [7,17], which has been shown to relieve symptoms in Stage II disease (Table 1) of the body of the talus. Finally arthrodesis of the joint may be performed where the necrotic bone is removed and replaced with bone graft. Fusion can be slow.

It is difficult to explain the possible mechanism for this case but consideration must be given to latent diabetic disease which was not present in this case. Talonavicular fusion with bone grafting was a viable treatment option in our case. The presence of localised pain at the talonavicular joint without trauma should alert clinicians to the potential hazards of avascular necrosis of the head of the talus. Early detection would prevent the need for bone grafting and arthrodesis and preservation of the joint.

**References**

