CASE REPORT

Post-traumatic osteonecrosis of distal tibia

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Introduction

Post-traumatic osteonecrosis affecting the femoral head, femoral condyles, humeral head, talus, tibial shaft, and proximal tibia has been commonly reported, but it is very unusual in the distal tibia alone.

We report a case of osteonecrosis of the distal tibia following fracture–dislocation of the ankle. Though the condition has been reported after ankle injury in children,6 we believe this to be the first clinical report in an adult. The patient responded well to conservative management and was reasonably asymptomatic at the time of follow-up.
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A 25-year-old man sustained a fracture dislocation of his left ankle with a high fibular fracture when he was knocked down by a push-bike in June 2002. He was taken to the local hospital where the ankle was manipulated and a plaster backslab was applied. He was seen in our unit 4 days later and the ankle was re-manipulated to correct mild talar shift and immobilised in plaster. Surgery was delayed due to gross swelling around the ankle. He was operated on 5 days later. The medial malleolar fragment was fixed with a single screw and a 2.0 mm K-Wire. The high fibular fracture with talar shift was treated with a single diastasis screw. Ten days later this was revised due to recurrent talar shift and the diastasis was stabilised with two 3.5 mm fully threaded cortical screws. These were subsequently removed at 6 weeks, the patient then started progressive weight-bearing over the next few weeks. Shortly after removal of plaster, the patient complained of increasing pain and swelling of his ankle. His gait was poor in the late stance phase due to pain, there was significant limitation in activities of daily and social living due to pain and swelling. His American Orthopaedic Foot and Ankle Society (AOFAS) score at this stage was 60. Physiotherapy and orthotic support failed to improve his symptoms. Check X-rays were satisfactory. There was no rise in inflammatory markers and there were no systemic signs of infection. An MRI scan was requested due the persistent nature of the problem and this revealed avascular necrosis of the distal tibia laterally involving distal
tibiofibular syndesmosis. There was also a focal defect in the distal tibial articular surface from the osteonecrosis. The fractures were completely healed. There was surrounding bone marrow oedema and oedema of the talar dome.

He was treated with a non-weight-bearing, below-knee plaster for 6 weeks, followed by graduated weight-bearing over the next 6 weeks in a patellar-tendon bearing cast. His symptoms settled and he was left out of the cast and allowed a graduated rehabilitation programme, avoiding undue stress to the joint. Although the stiffness persisted for some time, he was able to undertake activities of daily living reasonably well and had returned to office work. At annual follow-up for 3 years following injury he had a normal gait cycle with a good range of ankle movement (10° of dorsiflexion to full plantarflexion). Apart from this mild stiffness, he had no other symptoms. His American Orthopaedic Foot and Ankle Society score had improved to 77.

Discussion

The precise nature of the initiating event of osteonecrosis still remains unknown. The most likely causes are a primary vascular incident or a subchondral fracture from the traumatic insult with a secondary intracompartmental syndrome and osteonecrosis.²,⁵ There seems to be a disturbance of venous drainage from juxtachondral cancellous bone marrow causing intrasosseous stasis and hyperoedema. Traumatically induced defects in the chondral surface may also allow fluid from the joint to be expressed into the adjacent marrow space, producing an increase in marrow pressure and pain.³

The final common pathway of the bone infarction is irreversible blockade of the microcirculatory bed by cellular aggregates and micro-thrombi leading to alteration of capillary blood flow. The most widespread and consistent infarction occurs in embolisation of the main nutrient artery.³

After a fracture there is usually a variable extent of bone death on either side of the fracture line.⁷ However, necrosis of a large segment of bone after fracture is common to those sites that have a vulnerable blood supply, mostly intra-articular in location and having a limited soft-tissue attachment.¹ It depends on the site and type of fracture, the type of treatment, the timing of surgery, and union of the fracture. Factors increasing the chances of osteonecrosis are open or segmental fractures and fractures associated with dislocations. It is also more common in some bones, e.g. femoral head, talus, humeral head, scaphoid, and other bones.⁴,⁸

There is no account of osteonecrosis in the distal tibia alone.

References