CASE REPORT

Post-traumatic osteonecrosis of distal tibia

D. Chakravarty a,*, A. Khanna a,1, A. Kumar b,2

a Department of Orthopaedics, Peterborough and Stamford Hospitals NHS Trust, Edith Cavell Hospital, Bretton Gate, Peterborough PE3 9BR, United Kingdom
b Department of Orthopaedics and Trauma, Peterborough Hospitals NHS Trust, Edith Cavell Hospital, Bretton Gate, Peterborough PE3 9BR, United Kingdom

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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, 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 grad-
uated 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 dorsi-
flexion 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 osteo-
necrosis still remains unknown. The most likely
causes are a primary vascular incident or a subchon-
dral fracture from the traumatic insult with a sec-
ondary intraosseous compartment syndrome and
osteonecrosis. There seems to be a disturbance
of venous drainage from juxtachondral cancellous
bone marrow causing intraosseous stasis and hyper-
tension. Traumatically induced defects in the chon-
dral surface may also might allow fluid from the joint
to be expressed into the adjacent marrow space,
producing an increase in marrow pressure and pain.

The final common pathway of the bone infarction
is irreversible blockade of the microcirculatory bed by
acellular aggregates and micro-thrombi leading to
alteration of capillary blood flow. The most wide-
spread and consistent infarction occurs in embolisa-
tion of the main nutrient artery.

After a fracture there is usually a variable extent
of bone death on either side of the fracture line. How-
ever, necrosis of a large segment of bone after frac-
ture is common to those sites that have a vul-
nerable blood supply, mostly intra-articular in loca-
tion and having a limited soft-tissue attachment. 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 osteo-
necrosis are open or segmental fractures and frac-
tures associated with dislocations. It is also more
common in some bones, e.g. femoral head, talus,
humeral head, scaphoid, and other bones. There is no
account of osteonecrosis in the distal tibia alone.

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