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Avascular Necrosis of the Talus Following Apparently Minor Ankle injury: A Case Report

Hitoshi Ishikawa¹, Kiyoshi Simooku², Soichiro Hirata¹, Makoto Ishikawa³, and Yoshihiro Andoh³

We report a case of avascular necrosis of the talus which would appear to be related to unrecognized soft tissue damage in association with an apparently minor sprain. The presence of localized pain, with radiographic and MR image findings should alert clinicians to the potential of avascular necrosis of the talus. We emphasized that the surgical procedure may improve clinical signs, and that bone grafting of the aseptic talus should be done early before the body of the talus collapses.

Key Words
Aseptic necrosis, Talus, MR image, Surgical treatment.

INTRODUCTION

Talar osteochondral lesions normally involve the medial and lateral corners of the talar dome (1), and the medial lesions are usually posterior-superior, with a crater-like appearance (2). These lesions involve osteochondritis dissecans, osteochondral fractures, osteochondral defects and osteonecrosis. Avascular necrosis of the talus results most frequently from trauma, with a reported incidence of up to 91% in severe fracture dislocations (3,4). This is caused by interruption of the blood supply to the body of the talus (5,6). Atraumatic aseptic necrosis of the talus is far less common than adult aseptic necrosis of the hip (7). Uniformly, this has been reported in the body of the talus (8).

CASE REPORT

A fifty three-year-old man presented in May of 1993 with 4-year history of pain localized medial side of left ankle joint. There had been some associated pain which was exacerbated by walking. He had had sprain on his left ankle five years ago. His medical record at that time available did not describe any serious injury to his ankle; no ligamentous instability and fracture. Examination of his left foot revealed localized tenderness to palpation at the medial aspect of talus. There are some limitations of range of motion of left ankle joint. Ankle dorsiflexion was 20 degrees and plantar flexion was 35 degrees with pain. Radiographs revealed increased radiodensity of the
supero-medial portion of the talus in the lateral view (Figure 1A) and subchondral lucency and collapse in the antero-posterior view (Figure 1B). He was given a trial on non-steroidal anti-inflammatory agents which resulted in some decreased pain, but due to persistent symptoms, the patient was admitted to the hospital for further investigation. Laboratory findings including CBC, SMAC, erythrocyte sedimentation rate were all within normal limit. The CT scanning revealed the sequestration and fragmentation within the lesion, and T1-weighted sagittal MR image showed decreased signal with no signal in border of the lesion (Figure 2A), and subchondral bone was disrupted superiorly. T2-weighted sagittal MR image showed irregular high signal line and superior subchondral disruption was also seen (Figure 2B). Diagnosis of avascular necrosis was made and due to persistent symptoms he came to surgery in June 1993. The ankle was approached through a medial incision and malleolar osteotomy was performed. At surgery, there was noted to be an osteochondral defect on the supero-medial portion of the body of the talus (Figure 3). Dead bone was removed, and multiple drilling was performed until a bleeding surface was exposed inferiorly. Iliac bone graft was inserted to the defected area under compression. Medial malleolus was refixed with an A-O spongiosa screw. Postoperatively the patient was placed in a short leg cast for two months, and patient was only allowed to ambulate with non weight bearing with crutches. Histologic examination of the removed talar fragments showed that the spongy trabeculae were necrotic and disintegrating and intertrabecular marrow was also necrotic (Figure 4). The patient's postoperative course was uneventful (Figure 5). The external fixator was removed in 2 months. Six months after surgery, the patient was asymptomatic, bearing full weight on the operated extremity. MRI taken in December 1993 revealed the healing
Avascular necrosis of the talus

Figure 2. MR images of the lesion of the talus. T1-weighted image showed decreased signal (A) and T2 weighted image showed irregular high signal line (B).

Figure 3. An osteochondral defect on the supero-medial portion of the talus body was noted during the surgery.

Figure 4. Histologic finding of the removed talar fragments showed the trabeculae and bone marrow were necrotic.
process of the operated area.

DISCUSSION

Avascular necrosis of the talus has been considered to be of traumatic in origin, associated with fracture and dislocation (1-6,12,13). In the present case is not associated with major injury, which is not a dislocation and is not an overt fracture, but it seemed to be minor concussion of the talus itself. Thus the presented case here is likely to be an unusual case in a talus following a minor trauma that produced an avascular necrosis of the talus after injury. Rare instances of osteonecrosis of the body or dome of the talus have been reported in association with hyperuricemia (6), systemic lupus erythematosus (11), chronic pancreatitis (14), chronic alcoholism (7) and corticosteroid excess (9). These report are not associated with a specific episode of trauma. However in both of traumatic and atraumatic osteonecrosis of the talus, local ischemia has been implicated in the pathogenesis of the osteonecrosis.

The extraosseous and intraosseous vascularity of the talus have been well documented (12,16-18). The extraosseous vascularity is through the branches of the three major regional arteries which enter the five nonarticulating surfaces of the bone. The major blood supply to the body is provided by the artery of the tarsal canal (18). The blood supply is very precarious, thus it can be explaining the frequency of avascular necrosis after fractures and dislocations of the body (12). Vascular patterns of the talus probably correlate well with the reported incidence of avascular necrosis of the body of the talus following injury. In our case the osteonecrotic area was postero-medial portion of the body of the talus. The deltoid branches of the posterior tibial artery supply the medial one-third of body. It is likely that avascular necrosis developed in our case would depend upon the extent of soft tissue damage about the

Figure 5. Radiographs taken two months after surgery.
Clinical diagnosis of the osteonecrosis of the talus is difficult, and plain film radiographs of the ankle are important first step in the evaluation (2). In our case it was relatively easy to find the lesion of the disease in plain x-rays.

However, MR of the ankle allowed the status of osteonecrotic area more confidently. The most reliable sign of a lesion is presence of irregular, high signal line on T2-weighted images at the talar interface. The line has been documented by De Smet et al as having of loose granulation tissue (2). At surgery we confirmed this. Thus MR allows accurate determination of the status of the osteonecrosis area of the ankle.

In the present case an avascular necrosis of the talus with partially destruction of the joint cartilage was successfully treated with curettage and bone grafting. The current methods being used to retard or reverse the progression of this disease include drilling or decompression (7), bone grafting (12), talectomy, subtalar arthrodesis (5,16) and vascularized corticocancellous bone grafting from iliac crest (19). Among these procedures we would recommend the procedure involving of excision of the osteochondral fragment and curetting with drilling and bone grafting with medial malleolar osteotomy allows excellent visualization of the medial talus. Finally we emphasize that the presence of localized pain with radiographic MR image findings should alert clinicians to the potential of avascular necrosis of the talus.

REFERENCES