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Case Report

Post-Traumatic Osteonecrosis of the Talar Dome: A Rare Localization

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Abstract

Avascular necrosis of the talus is a debilitating condition that affects the ankle joint and results from a deficiency in bone vascularization. It is defined as the death of bone tissue due to a disruption in blood circulation, which can result from trauma, compression, or vascular occlusion. It presents with ankle pain and decreased mobility. We present a case of a 36-year-old patient with chronic post-traumatic ankle pain underwent an MRI, revealing a signal abnormality in the talus dome characterized by T1 hypointensity, T2 hyperintensity with fat suppression, surrounded by a serpiginous peripheral halo showing T1 and T2 hypointensity. The diagnosis of osteonecrosis was made based on these radiological findings. Treatment can be medical, involving pain relief and offloading, or surgical, depending on the stage of the disease.

Keywords: *Fracture; Talus; Astragalus; Avascular Necrosis; Osteonecrosis*

Introduction

Osteonecrosis is defined as the death of bone tissue secondary to a disruption of blood circulation, which can be caused by trauma, compression, or vascular occlusion [1]. It typically affects the femoral head and, less frequently, the femoral condyles and humeral head. Involvement of the talus or astragalus is rare, often post-traumatic, and carries a risk of subsequent development of tibio-tarsal osteoarthritis [2].

Case Report

A 36-year-old patient with no significant medical history suffered a minor domestic accident two months ago after falling down the stairs. Due to the persistence of pain, an MRI of the ankle was requested. The clinical examination revealed pain on the dorsal aspect of the right foot without any inflammatory signs, along with limited mobility of the limb, particularly in dorsiflexion. Magnetic resonance imaging (MRI) showed a signal anomaly in the dome of the talus, appearing as hypointense on T1-weighted images and hyperintense on T2-weighted FATSAT images, surrounded by a serpiginous peripheral halo that is hypointense on both T1 and T2 (Figure 1 and 2). This was associated with an adjacent area of bone edema and infiltration of the plantar soft tissues (Figure 3). Based on these radiological findings and the clinical presentation resembling an ankle sprain in a traumatic context, the diagnosis of osteonecrosis of the talar dome was established.

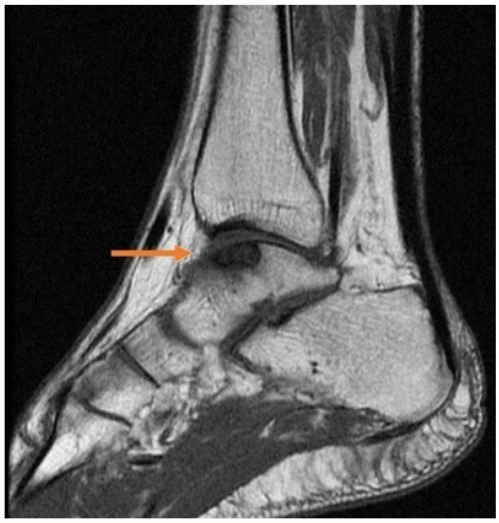

Figure 1: *MRI image in sagittal T1-weighted sequence, showing a signal anomaly in the dome of the talus (orange arrow) as hypointense on T1, surrounded by a serpiginous peripheral halo that is also hypointense on T1.*

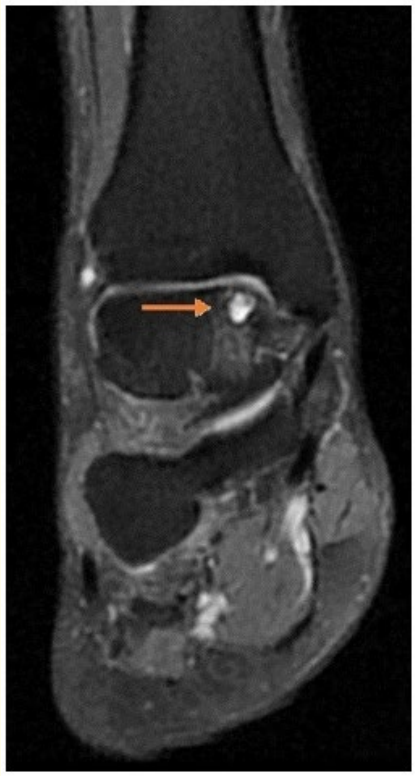

Figure 2: *MRI image in coronal T2 FATSAT sequence, showing a signal anomaly in the dome of the talus (orange arrow) as hyperintense on T2 FATSAT, surrounded by a serpiginous peripheral halo that is hypointense.*

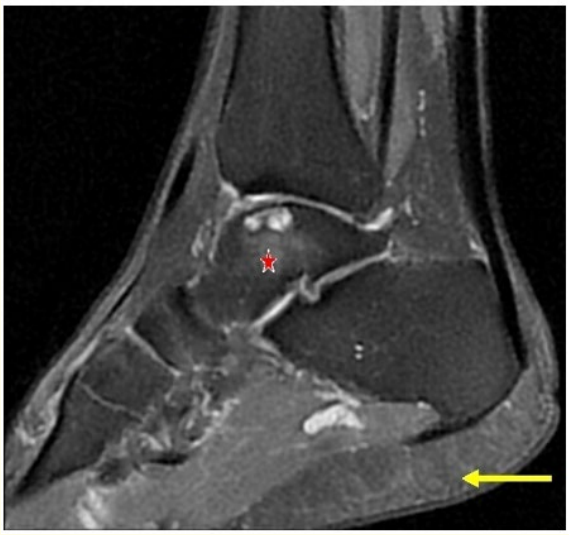

Figure 3: *Area of post-traumatic bone edema (asterisk) and infiltration of the plantar soft tissues (yellow arrow).*

Discussion

The vascularization of the talus is primarily provided by branches of the anterior and posterior tibial arteries and, to a lesser extent, by the fibular artery. This cranio-caudal terminal type of vascularization is precarious, which explains the frequency of necrosis [3].

Apart from trauma, the causes of osteonecrosis include corticosteroid use, alcoholism, systemic lupus erythematosus, kidney transplants, sickle cell disease, hyperlipidemia, radiation therapy, and hereditary thrombophilias [4].

Talus osteonecrosis often presents with mechanical-type ankle pain. Clinically, ankle swelling may be observed, along with the loss of the retro-malleolar grooves and a tender point on the body of the talus when the ankle is in plantar flexion. Mobility limitation begins with the loss of foot dorsiflexion, followed by a loss of plantar flexion at a more advanced stage [2].

X-rays play an important role in post-surgical monitoring but are unable to detect the early stages of the disease [5]. During the initial X-ray, the necrotic bone and the surrounding viable bone have the same opacity, making it difficult to assess the extent of the lesions [4].

Over time, with resulting hyperemia, the healthy bone resorbs and becomes osteopenic. In contrast, the lack of blood supply to the necrotic bone prevents its resorption, giving it a more radiopaque appearance than the surrounding osteopenic bone. It is only at this stage that radiographic signs of osteonecrosis become apparent [4]. The opacity of the necrotic bone continues to increase as reossification occurs and new bone is deposited on the necrotic trabeculae. This process explains the typical sclerotic appearance observed in talus osteonecrosis [4].

Hawkins' sign is characterized by subchondral osteopenia, appearing as a subchondral radiolucent line on anteroposterior or mortise view radiographs. It typically appears 6 - 8 weeks after injury and is highly suggestive of favorable revascularization. This sign is considered a reliable indicator of talus viability; however, its absence does not confirm osteonecrosis, as the sign has much higher sensitivity than specificity [5].

In more advanced cases of the disease, subchondral cystic changes and even collapse of the articular surface may occur. The crescent sign, a curved radiolucent line caused by subchondral collapse, has been described in the proximal femur and proximal humerus but is less commonly observed in the talus [5].

The use of X-rays for staging talus osteonecrosis was described by Mont., *et al*. based on the previous Ficat staging system for the femoral head. Stage I is characterized by normal X-rays, while Stage II is marked by cystic or osteosclerotic lesions, normal talar contours, and the absence of subchondral fracture. In Stage III disease, there is the presence of the crescent sign or subchondral collapse, and finally, Stage IV disease shows arthritic changes [5].

A coronal CT scan of the talus is necessary to visualize the articular surface of the dome to exclude any subtle depression, collapse, or fragmentation, especially during preoperative evaluation [4].

MRI typically reveals an area of osteonecrosis as hypointense on T1-weighted sequences. It can be either homogeneous or heterogeneous with the presence of hyperintense areas within it [2]. On T2-weighted images, osteonecrosis presents a mixed and variable appearance depending on the contents of the avascular region: blood, bone, fluid, or fibrous tissue [5]. A thin band of hypointensity on both T1 and T2 extending between the two edges of the subchondral bone is often seen on MRI; it takes on a serpentine and irregular shape and is manifested by a double line with a proximal hypointense band and a distal hyperintense zone. It is important to note that bone edema may sometimes accompany osteonecrosis [2].

In the absence of acute trauma, the main differential diagnoses include infection, stress fractures, transient migratory osteoporosis, and early neuroarthropathy. The diagnosis is established based on clinical, biological findings, and MRI. If necessary, a percutaneous biopsy may be considered, particularly if there is a suspicion of infection [5].

Treatment of talus osteonecrosis includes two main aspects [2]:

- Medical treatment: Systematic use of analgesics is essential. Weight-bearing relief is necessary during acute pain phases, which may indicate the possibility of a subchondral fracture, although its impact on subsequent progression remains uncertain and its duration is empirical.
- Surgical treatment: Varies depending on the stage. It may include different techniques such as drilling with or without bone grafts, tibiocalcaneal arthrodesis, and ankle prostheses.

Conclusion

In the case of any chronic post-traumatic ankle pain, talus osteonecrosis should be considered, even though it is an atypical and rare location. The final diagnosis would be confirmed based on MRI findings.

Conflict of Interest Statement

The authors declare that they have no conflicts of interest.

Declaration of Interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Informed Consent

Written informed consent was obtained from the patient.

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