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Case Report

A Rare Coexistence: Avascular Necrosis of the Talus with Osteochondritis Dissecans – A Case Report

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Abstract: Avascular necrosis (AVN) of the talus and osteochondritis dissecans (OCD) are uncommon conditions affecting the ankle joint, each with distinct pathophysiologies and clinical implications. The talus's unique vascular anatomy renders it susceptible to AVN, especially in the setting of trauma or compromised vascular supply. OCD, conversely, is typically linked to repetitive microtrauma leading to subchondral bone fragmentation and cartilage instability. Their simultaneous occurrence is exceedingly rare and presents diagnostic as well as therapeutic challenges. We report the case of a 46-year-old female who presented with progressive pain and tenderness over the ventral aspect of the ankle, aggravated by weight-bearing activities. Imaging with radiography, computed tomography (CT), and magnetic resonance imaging (MRI) revealed features consistent with avascular necrosis of the anterior talus and a displaced osteochondral fragment over the medial talar dome, suggestive of OCD (Stage IV – Berndt and Harty classification).

Keywords: OCD – Osteochondritis Dissecans, MRI – Magnetic Resonance Imaging, CT – Computed Tomography, AVN – Avascular Necrosis.

INTRODUCTION

The talar frame, which is made up of the head, neck, and body, articulates with the navicular bone distally, the tibia and fibula superolaterally (proximally), and the calcaneus inferiorly. This bone has no muscle or tendinous attachments, and articular cartilage covers around 60% of its surface. As a result, the amount of penetrable bone that can be used for vascular perforation is restricted. When the talus's vascular supply is disrupted, this characteristic, together with tiny nutrition vessels, differences in intraosseous anastomoses, and a lack of collateral circulation, makes it more susceptible to osteonecrosis [1]. One of the most dreaded outcomes of talar fractures is avascular necrosis (AVN) of the talus, which is more likely to occur the more severe the trauma and the resulting harm to the already fragile blood supply. It can also have non-traumatic causes in addition to posttraumatic ones, such as idiopathic reasons, alcoholism, steroid usage, or dyslipidaemia [2]. The talar dome collapsing causes talar impingement and increasing loss of range of motion, which are the initial symptoms of avascular necrosis of the talus that appear 6–8 weeks after fracture. Six to eight weeks after the fracture, X-ray imaging should be done as the initial diagnostic procedure. The first warning indication at this stage is the Hawkins sign's absence. The talus's subchondral radiolucent band is known as the Hawkins sign [1-3]. The most sensitive method for identifying talar osteonecrosis, particularly in its early stages, is magnetic resonance imaging (MR imaging). Furthermore, in the presence of normal radiography findings and a high clinical suspicion for AVN, MR imaging may be utilised. CT scans can be utilised to validate radiography results and also show distinctive talar AVN patterns [1]. OCD is a rare, localised condition that affects the subchondral bone and can cause the articular cartilage that covers it to delaminate and become unstable [4]. Although the exact cause of OCD is still up for question, the most widely accepted theory is that repetitive microtraumas linked to vascular impairment induce progressive ankle discomfort and dysfunction in individuals who are young adults and skeletally immature. Although lateral and posterior involvement are less common, ankle OCD is traditionally found in the medial portion of the talus [5]. Chondral (cartilage only), chondral-subchondral

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(cartilage plus bone), subchondral (intact overlaying cartilage), and cystic are the terms used to characterise an OCD. Then, lesions can be classified as either non-displaced or displaced, stable or unstable. According to certain writers, a lesion can also be classified as medial, lateral, or central based on where it is located on the talus' articular surface, with additional subdivisions into anterior, central, or posterior [6]. The first line of treatment for symptomatic lesions is immobilisation and activity restriction for 6–12 weeks. Otherwise, for intact lesions with immaculate articular cartilage, retroarticular drilling with or without bone grafting is recommended. Compression screw repair may work effectively for nondisplaced lesions with an articular fissure or demarcation, particularly if the cartilage is connected to subchondral bone [7].

Case Report

A 46-year-old female presented with complaints of pain and tenderness over the ventral aspect of the ankle joint. The pain was insidious in onset and progressively worsening, particularly aggravated by standing and walking. On clinical examination, range of motion was preserved. There was no history of trauma, fever, neurological deficits, or prior surgical intervention. The patient denied any known comorbidities, relevant past medical history or use of any drugs.

A radiograph of the ankle joint was performed. AP view of ankle joint (Figure 1A) showed osteochondral fragment at the medial aspect of the talar dome with a corticated outline and minimal separation. Lateral view of Ankle joint (Figure 1B) showed cortical irregularity in the anterior aspect of talus.

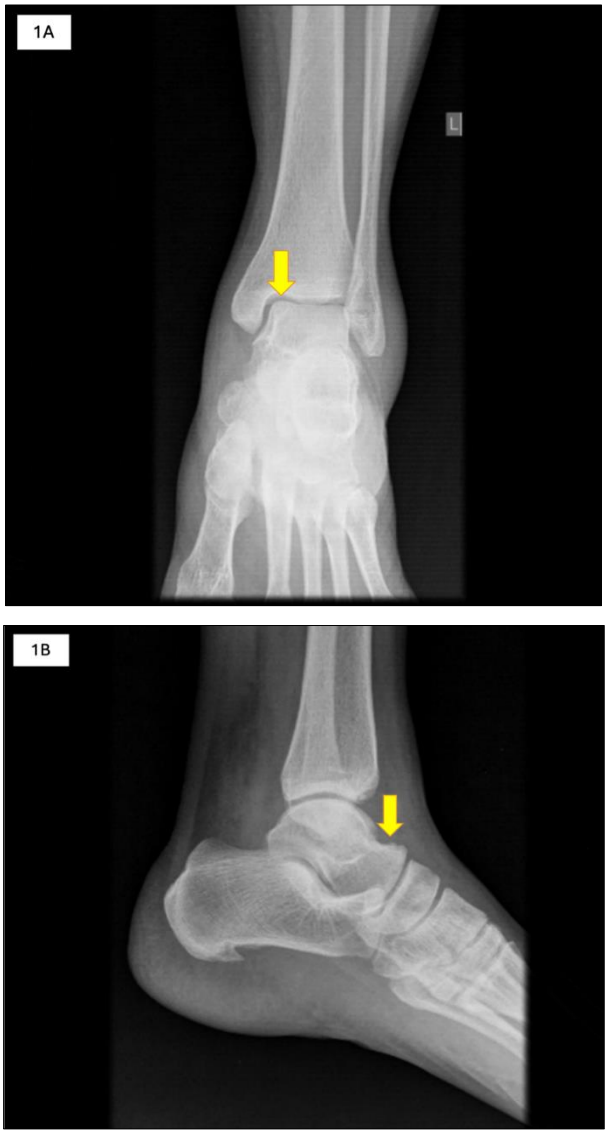


Figure 1: A 46-year-old female presented with complaints of pain and tenderness over the ventral aspect of the ankle joint. Radiograph of ankle joint AP View (A) showed osteochondral fragment at the medial aspect of the talar dome with a corticated outline and minimal separation. Lateral view (B) showed cortical irregularity in the anterior aspect of talus

Plain Computed Tomography of the Ankle joint lateral view (Figure 2A) shows irregularity of the articular surface of the anterior aspect of talus (arrow), with underlying mixed hypoattenuating sclerotic regions of necrotic subchondral bone. Coronal view (Figure 2B) of ankle joint shows osteochondritis dissecans seen involving medial aspect of talar dome (Arrow) with mild displacement of fragment.

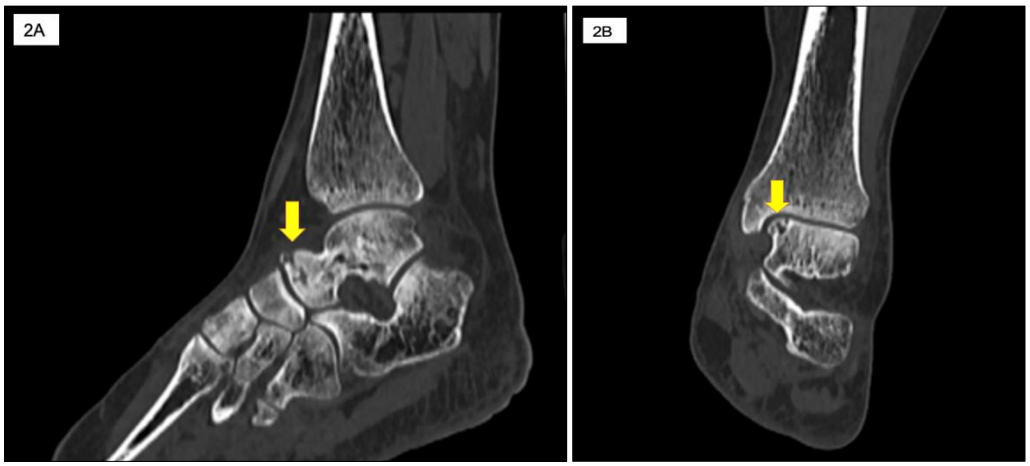
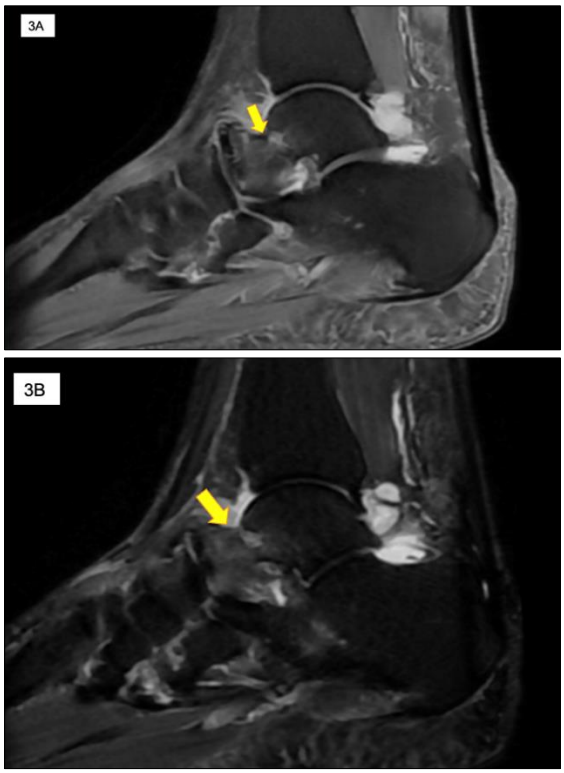


Figure 2: A 46-year-old female presented with complaints of pain and tenderness over the ventral aspect of the ankle joint. Computed Tomography of ankle joint Lateral view (A) shows irregularity of the articular surface of the anterior aspect of talus (arrow), with underlying mixed hypoattenuating sclerotic regions of necrotic subchondral bone. Coronal view (B) shows osteochondritis dissecans seen involving medial aspect of talar dome (Arrow) with mild displacement of fragment

MRI of the Ankle joint was performed on 3.0 T MRI scanner (GE Signa pioneer). Sag PDFS (3A) and Sag T2 STIR (3B) of ankle joint demonstrates flattening, subchondral cystic change and sclerosis seen in the anterior aspect of talus with minimal associated marrow edema involving anterior half. Coronal PDFS (3C) of ankle joint reveals Changes consistent with osteochondritis dissecans seen involving medial aspect of talar dome with mild displacement of fragment (Stage IV Berndt and Harty classification). The fragment measures ~ 6.4 x 4.3 x 4.4 mm. The above findings were consistent with Avascular necrosis of talar head with osteochondritis dissecans.



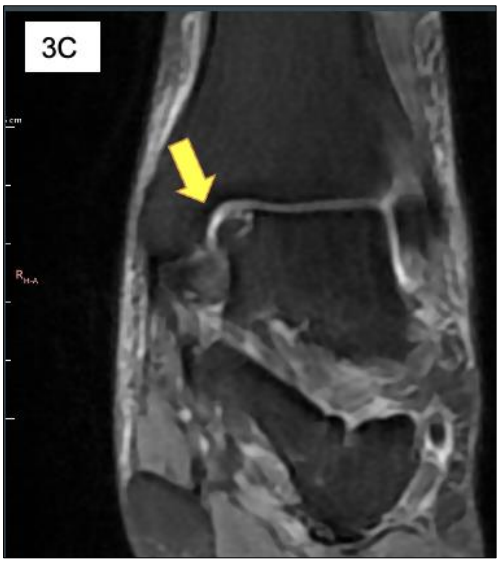


Figure 3: A 46-year-old female presented with complaints of pain and tenderness over the ventral aspect of the ankle joint. Magnetic Resonance Imaging of ankle joint Sag PDFS view (A) and Sag T2 STIR (B) of ankle joint demonstrates flattening, subchondral cystic change and sclerosis seen in the anterior aspect of talus with minimal associated marrow edema involving anterior half. Coronal PDFS (C) Changes consistent with osteochondritis dissecans seen involving medial aspect of talar dome with mild displacement of fragment (Stage IV Berndt and Harty classification)

DISCUSSION

Osteonecrosis, or AVN, of the talus happens when any segment of the vascular network—including the veins, sinusoids, capillaries, and arteries—is disrupted. This disturbance might be categorised as vascular obstruction, compression, or physical disruption (trauma). The body's response to AVN is an attempt at repair by means of reossification, revascularization, and resorption of necrotic bone. It is when these processes occur that AVN becomes apparent radiographically [1]. Although its aetiology includes a broad range of systemic illnesses, including type 2 diabetes (DM II), systemic lupus erythematosus (SLE), and steroid medication, it usually arises as a consequence of a talar neck fracture [2, 3]. Talar AVN can be diagnosed and assessed with the aid of articular collapse and the "Hawkins sign." Anteroposterior radiographs show the Hawkins sign the best, while lateral radiographs may not show it. This symptom, if it exists, shows that the relevant area of the talar body is receiving enough blood. A partial Hawkins sign may also be a symptom of partial or incomplete AVN [3]. A nondisplaced talar neck fracture linked to a 0%–15% prevalence of AVN is known as a Hawkins type I fracture. The risk of AVN is 20% to 50% for type II fractures, which are displaced fractures with subtalar joint dislocation or subluxation. Hawkins type III fractures are displaced fractures that cause the ankle and subtalar joints to dislocate or subluxate. In Hawkins type III fractures, the chance of AVN is almost 100%. Lastly, type IV fractures are displaced fractures that cause the talonavicular, tibiotalar, and subtalar joints to dislocate or subluxate. The related risk of AVN for type IV fractures is 100%.In the case of persisting absence of the Hawkins sign and the presence of talar AVN symptoms after 12 weeks, MRI should be performed [1-3]. An MRI should be done on high-risk patients who have a negative Hawkins sign and a clinical suspicion of AVN and negative radiography. When paired with a clinical examination, any high-signal-intensity line in the talar dome fat-saturated in T2-weighted MR or a serpiginous low-signal-intensity line in the talar dome T1-weighted MR may suggest AVN [3]. Osteochondritis dissecans is a condition in which a piece of bone and cartilage separates from the joint surface. Since OCD presents in a vague manner, imaging is essential to diagnosing OCD. Imaging modalities used for assessment of OCD include conventional radiography, nuclear medicine, computed tomography (CT), CT arthrography, magnetic resonance imaging (MRI), and magnetic resonance (MR) arthrography [4]. The knee and elbow joints are the two most commonly impacted anatomical sites, followed by the talus. Ankle OCD is classically located in the medial part of the talus, while lateral and posterior involvement is less frequent. There are various etiological theories for OCD: traumatic, ischemic and micro-traumatic. Most OCD patients do not have a history of previous traumatic events, and it has been suggested that repetitive micro-traumas could instead contribute to the development of the lesion. Even though several categories have been proposed over time utilising arthroscopy, CT, or MRI, the Berndt and Hartys classification is still the most commonly used: Stage 1. Small area of compression of the subchondral bone, Stage 2. Partially detached osteochondral fragment (flap), Stage 3. Completely detached osteochondral fragment but undisplaced, Stage 4. Free osteochondral fragment [5]. MRI is the modality of choice for diagnosis of OCD. Accurate evaluation of cartilage thickness, signal alterations inside cartilage, the interface between cartilage and bone, and the subchondral bone are all necessary components of an optimal MRI strategy for OCD lesions and OCD treatment. Additionally, it ought to offer important

details regarding the tissue that repairs articular cartilage following surgery. Numerous MRI pulse sequences, both established and in development, are useful for evaluating osteochondral lesions. In this context, the two most popular types of pulse sequence acquisition are three-dimensional (3D) spoilt gradient echo (SPGR) or fast low-angle shot (FLASH) sequences, as well as intermediate and T2-weighted rapid spin echo (FSE) approaches [4]. For most symptomatic talar OCD, marrow stimulation of the OCD, typically through arthroscopic debridement and microfracture, has been found to be an effective treatment strategy. But instead of hyaline cartilage, fibrocartilage is the reparative tissue that develops in response to the marrow stimulation. The clinical success rate of osteochondral autografting seems to be comparable to that of bone marrow stimulation [7].

CONCLUSION

Avascular necrosis and osteochondritis dissecans of the talus are individually uncommon entities, and their concurrent manifestation is exceptionally rare. This case underscores the significance of considering a dual pathology in patients with persistent ankle pain despite the absence of trauma. Radiological evaluation, especially with MRI and CT, plays a pivotal role in the timely and accurate diagnosis of such overlapping conditions. Early diagnosis is key to preventing further articular damage and optimizing clinical outcomes through tailored treatment strategies.

Conflict of Interest: None

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