The case:

A 20-year-old man presented with an ankle deformity following a motor vehicle collision.

Figure 1: Modified lateral radiograph of the right ankle.

Your diagnosis?

For answer see page 246
Diagnosis:

Talar Neck Fracture With Tibiotalar and Posterior Subtalar Dislocation

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The patient presented with a complete fracture of the talar neck (Figure 1). Dislocation of the dominant talar dome fragment can be seen at the tibiotalar and subtalar joints. This fracture-dislocation pattern corresponds with a Hawkins III fracture, which has a moderate risk of fracture nonunion and an 80% to 100% risk of subsequent talar dome avascular necrosis.

Talar fractures are serious injuries of the hindfoot that can result in long-term ankle and hindfoot complications.

**DISCUSSION**

Talar neck fractures comprise approximately half of all talar fractures and are associated with several significant complications. The severity of injury is prognostic of outcome and is graded by the Hawkins classification. Avascular necrosis (AVN) of the talar body is a complication that approaches a rate of 100% in Hawkins IV talar neck fracture dislocations. Sequela of these injuries can lead to chronic disability and pain due to delayed union, collapse of the talar dome, or posttraumatic osteoarthritis.

The fractures are caused by high-energy injuries, such as falls from high heights and motor vehicle collisions. Some studies have reported that men experience these fractures more often than women. No predilection for age exists.

**ANATOMY**

The anatomy of the talus renders it susceptible to bone and vascular injury. The talus can be described in 3 parts: a head, neck, and body. The head of the talus bears the rounded articulation for the navicular and allows rotation in the medial-lateral and dorsal-plantar planes, giving the midfoot flexibility.

The body articulates with the calcaneus inferiorly and with the fibula and tibia superriorly. The body is wider anteriorly than posteriorly and contains the talar dome, which forms the talocrural joint with the tibia. This joint bears more weight per unit of area than any other joint in the body, contributing to the significance of the complications related to fracture.

The inferior talar surface contains the posterior, middle, and anterior facets, which articulate with corresponding calcaneal facets to form the subtalar or talocalcaneal joints. Along the inferior surface of the talus is the sulcus tali, a groove that extends anterolaterally from the inferior posterior medial malleolus to the sinus tarsi, between the middle and posterior facet talocalcaneal joints.

The talar neck sits between the middle and posterior facets; its inferior surface forms the roof of the tarsal canal. It has a relatively narrow di-
ameter and, due to a lack of constraints imposed by the articulations of the marginal portions of talus, it is the weakest part of the talus.\textsuperscript{2,3}

**Bone Injury**

Several mechanisms of injury have been described.\textsuperscript{1,3,4} Talar neck fractures are often due to high-energy trauma from an axial load to the foot. Common mechanisms include falls from heights and landing feet first or front-end motor vehicle collisions while the foot is on the accelerator or brake.

Inversion of the ankle causes impingement of the neck against the medial malleolus, leading to fractures of the medial neck and associated medial malleolus fractures. Dorsiflexion of the ankle leads to impingement of the talar neck against the anterior plafond of the tibia. With continued force, the talus displaces posteriorly, and subtalar dislocation occurs. Axial loading may also result in associated calcaneal and spine injuries.

**Vascular Injury**

Two-thirds of the talar surface is covered in articular cartilage, resulting in a limited area for vascularization. Vascular supply to the talar dome is an end-artery system, with blood vessels entering from the talus neck and plantar talar body. Circulation to the talus is supplied by the posterior tibial artery, anterior tibial artery, and peroneal artery, whose branches form a vascular sling around the talar neck and sinus tarsi.

The posterior tibial artery gives rise to the tarsal canal artery, which runs along the tarsal canal anterolaterally to the sinus tarsi. There, it anastomoses with the tarsal sinus artery, which is derived from branches of the peroneal and anterior tibial arteries. The tarsal canal artery and its deltoid branch enter the talar neck inferiorly and the talar body medially to provide the majority of the blood supply to the talar body.\textsuperscript{5,6} When the superior aspect of the talar dome is considered from an axial perspective, the dome has 3 vascular zones from the 3 arterial contributors, with dominance of the medial vascular supply for >50% of the dome.

**Avascular Necrosis**

Avascular necrosis occurs when the blood supply to the talus is compromised and leads to ischemic bone death. Clinically, patients present with an inability to bear weight, pain, joint effusion, and decreased range of motion. The talar neck contains a major portion of the vascular supply for the talar body. Disruption of this blood supply by fracture can contribute to delayed union or nonunion at the fracture site. Avascular necrosis of the talar body or dome can result in the collapse of the articular surface, leading to arthrosis.

Poor prognostic indicators for talar neck fractures include comminution, dislocation of the talar body, and open fractures. Long-term outcomes include joint incongruity, osteoarthritis, decreased mobility, and chronic pain.\textsuperscript{3}

The Hawkins classification predicts the incidence of AVN of the talar dome based on the pattern of fracture and dislocation.\textsuperscript{7} A Hawkins I fracture is a nondisplaced fracture typically extending vertically from the dorsal neck to the tarsal canal, with an AVN risk of 0% to 13%. The blood supply entering the neck is disrupted.

Hawkins II fractures are seen with continued dorsiflexion, leading to posterior displacement of the body and subluxation or dislocation of the subtalar joint. The blood supply through the neck and the tarsal canal is affected. The AVN risk is 20% to 50%.

Hawkins III fractures are classified as displaced fractures with subtalar and tibio-talar dislocation, with an AVN risk of 80% to 100%. Hawkins IV fractures are seen when talonavicular joint dislocation is superimposed on class III injuries.\textsuperscript{8} The AVN rate of these injuries historically approaches 100%, although recent data may indicate a lower incidence of AVN than previously thought.\textsuperscript{9} In class III and IV fractures, all 3 arterial supplies are disrupted.\textsuperscript{10}

**Imaging**

Anteroposterior (AP), lateral, and mortise radiograph views of the ankle are used for initial evaluation of suspected fracture. The AP view shows the alignment of the talar body, whereas the lateral view best demonstrates the talar neck. A visualized fracture line is the most reliable diagnostic finding. All views must be carefully scrutinized because talar fractures are often missed or not visible due to overlapping bones (Figure 2).

A modified AP view was described by Canale and Kelly\textsuperscript{8} for optimal visualization of the talar neck. The ankle is held in maximal equinus and pronated 15° against the cassette. A cephalad beam is then directed 75° from horizontal. However, this view can be technically difficult to execute, and patients often have pain or additional injuries that prevent optimal positioning for plain radiographs (Figure 3A). Many studies have shown the value of obtaining a computed tomography (CT) scan for superior detection of fractures in cases where there is high clinical suspicion of fracture but radiographs are negative.\textsuperscript{11,12} Computed tomography with multiplanar reconstruction can demonstrate occult fractures, comminution, degree of displacement or malignment, and extension to articular surfaces (Figure 3B).\textsuperscript{11,12} Follow-up radiographs are routinely obtained to evaluate fracture healing and monitor the development of AVN.
Initially, radiographs cannot distinguish devascularized bone from vascularized bone because they have similar bone densities at this point. Approximately 6 to 8 weeks after injury and immobilization, subtle radiographic differences are appreciated.

If the fracture fragment has an intact vascular supply, disuse osteoporosis will be evident equally throughout the foot and ankle from an intact remodeling response. In the case of devascularization, osteoclasts are unable to migrate to the devascularized fragment or to initiate the physiologic resorption of bone that occurs in disuse osteoporosis and early stages of healing. Thus, the bone density of the talar body is unchanged relative to decreased density in the surrounding bone that has an intact blood supply. This creates the appearance of increased bone density in devascularized bone, although it is the healthy bone around it that has actually changed in density.

The Hawkins sign is an indicator of intact vascularity to the talar dome that has a high sensitivity. It can be valuable in the assessment of AVN in talar neck fractures; however, the absence of a Hawkins sign has low specificity for avascular necrosis. This sign is determined on an AP view of the ankle. If a Hawkins sign is present, thin subchondral lucency is seen across the talar dome, representing osteoclastic migration and resorption (Figure 4).

In contrast, an absent Hawkins sign appears as a homogeneously dense talar dome (Figures 5, 6). A partial Hawkins sign indicates partial vascularization of the dome, which is usually present medi- ally due to a more robust blood supply to this portion (Figure 3C).

Computed tomography is a valuable tool for follow-up of talar fractures and has a greater sensitivity than radiographs in detecting failure to heal and malalignment. Avascular necrosis can also be detected by identifying a difference in the bone density of the talus in the setting of surrounding disuse osteoporosis. Coronal reformat images provide a talar dome profile similar to AP ankle radiographs, depicting sequelae such as articular collapse and secondary osteoarthritis, although axial images profile the dome well (Figures 7, 8).

Magnetic resonance imaging (MRI) is sensitive for the detection of occult fractures but is used as a problem-solving tool for this diagnosis. However, MRIs are more sensitive than radiography and CT for detecting early manifestations of AVN. Peritraumatic imaging can be problematic because ischemic changes parallel those of devascularization, with non-specific ill-defined low T1 and high T2 signal changes termed bone marrow edema pattern. The volume of marrow at risk for long-term sequelae of devascularization can be overestimated. Although ischemic changes subside with
improved vascular status, infarction progresses to increasing demarcation of a zone of low T1 signal, with a marginal serpiginous signal void in an expected vascular distribution (Figure 9).

On T2-weighted images, the necrotic area shows a serpiginous high T2 signal line on the vascularized margin, paralleling the low signal demarcation of the infarcted region. Although generally not needed, contrast enhancement is seen at the vascularized margin of the necrotic focus following intravenous infusion of gadolinium. In chronic AVN, resolution of the T2 hyperintense marginal marrow with residual hypointense necrotic focus occurs (Figure 10).

Unfortunately, MRI may be limited for the assessment of fracture healing and AVN in the presence of metal implants for fracture stabilization due to artifact.

TREATMENT

Nondisplaced Hawkins I fractures can be treated conservatively with cautious monitoring for reduction maintenance in a nonweight-bearing cast for 6 to 12 weeks. Open reduction and internal fixation or percutaneous fixation in Hawkins I fractures has the benefit of allowing for early range of motion to decrease the likelihood of joint stiffness.

Hawkins II to IV fractures are displaced and benefit from open reduction and internal fixation. Severe injuries may be treated with primary arthrodesis if anatomic reconstruction is not possible. For closed fractures, the dislocated talar body may exert pressure on the medial skin, leading to skin necrosis. In all cases, progression to weight bearing is not permitted until bone trabeculation is seen at the fracture site.

Patients are monitored with radiographs to assess for the development of AVN during the recovery period. The Hawkins sign, if present, is usually seen at 6 weeks. If AVN is suspected or radiographically evident, weight bearing may be protected to decrease the likelihood of collapse depending on the degree of AVN and stage of fracture healing. Avascular necrosis of the talar body is not necessarily prognostic of functional outcome, and its treatment depends on the degree of pain and disability.

Core decompression and vascularized bone grafting in early AVN prior to collapse have been performed with some success in improving pain; however, the outcome data are limited. In cases of significant clinical disability due to posttraumatic arthropathy, subtalar tibiotalar or tibiocalcaneal arthrodesis may be indicated.

CONCLUSION

Talar neck fractures are the result of high-energy injuries...
and require vigilant treatment and monitoring. The tenuous blood supply to the talus and its multiple articulations make it vulnerable to multiple significant complications. Early recognition of displacement with anatomic reduction and stabilization is necessary to offer the best chance of a good outcome. Careful radiographic assessment and monitoring with plain radiographs, CTs, and MRIs can guide treatment from injury through recovery.

REFERENCES