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Management of Talar Neck Fractures

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Abstract: Talar neck fractures are usually the result of highenergy trauma. It remains controversial whether talar neck fractures require emergent treatment. Most surgeons recommend the use of dual surgical approaches, anteromedial and anterolateral, to allow accurate visualization and anatomic reduction. It is important to carefully preserve any remaining talar blood supply. Obtaining satisfactory clinical results, while avoiding complications, presents a unique challenge in the treatment of talar neck fractures. Common complications include posttraumatic arthritis, avascular necrosis, malunion, and nonunion.

The talus has a unique anatomic shape and function that predisposes it to uncommon but often serious injuries. It has 7 articular surfaces. The calcaneal articular facets form the subtalar joint. The anteromedial trochlear surface, central trochlear surface, and lateral process form the talar portion of the ankle joint. The talus is held in the ankle by bony constraints, the medial and lateral malleolus, and constraining ligaments. Talar fractures are uncommon, constituting <1% of all reported fractures, and only 3% to 6% of all foot fractures.¹ Most talar fractures result from high-energy trauma and have a high rate of associated injuries. Approximately 50% of all talar fractures occur through the talar neck, which is the portion of the talus that has the smallest cross-sectional area and is covered with a relatively weak cortex. High-energy trauma that produces displaced talar neck fractures interrupts the blood supply of the talus and often causes varying degrees of articular cartilage damage. Obtaining satisfactory clinical results while avoiding complications presents a unique challenge in the treatment of talar neck fractures.

MECHANISM OF INJURY

Talar neck fractures are usually the result of high-energy trauma because the thick subchondral bone requires high forces to produce a fracture. The accepted mechanism of talar neck fractures is a hyperdorsiflexion force.² With dorsiflexion, the posterior ligaments of the subtalar joint initially rupture, and the neck of the talus impacts against the leading anterior edge of the distal tibia. A fracture line develops at this point and enters the nonarticular portion of the subtalar joint between the middle and posterior facets. With the continuation of force, the calcaneus and the rest of the foot, including the head of the talus, sublux anteriorly. If there is a concomitant inversion component to the force, the foot may sublux or

dislocate medially. Similarly, if there is concomitant eversion force, the foot dislocates laterally. If the force subsides at this moment, the foot recoils, the body of the talus tips into equines, and the fracture surface of the neck comes to ride on the upper surface of the calcaneus. However, if the dorsiflexion force continues, further rupture of the posterior ankle capsular ligament, the strong posterior talofibular ligament, and the superficial and posterior aspects of the deltoid ligaments occurs. The body of the talus is then wedged posteriorly and medially out of the mortise and rotates around a horizontal and transverse axis so that the fracture surface faces upwards and laterally.3

FRACTURE CLASSIFICATION

The most widely accepted classification system of talar neck fractures is that by Hawkins⁴ (Figure 1), which is based on displacement and dislocation, and therefore, presumed damage to the blood supply of the talus. This classification was further expand-

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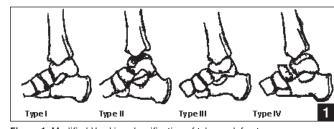


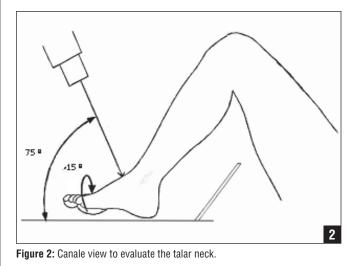
Figure 1: Modified Hawkins classification of talar neck fractures.

ed by Canale and Kelly,⁵ who added the type IV category. A Hawkins type I fracture is a nondisplaced fracture, without subluxation or dislocation. A Hawkins type II fracture is a displaced vertical talar neck fracture with a subluxation or dislocation of the subtalar joint. A Hawkins type III fracture is a displaced fracture extending through the talar neck with dislocation at both the subtalar and tibiotalar joints. The degree of displacement and dislocation is thought to be the primary measure of the interruption of blood supply, and therefore, the risk for the development of avascular necrosis. A type IV fracture is associated with a dislocation of the ankle and subtalar joint, and with an additional dislocation or subluxation of the head

of the talus at the talonavicular joint. Several authors^{5,6} have found that the Hawkins classification correlates with the final outcome and the incidence of avascular necrosis. However, there has been an inconsistency in results between other studies that have looked at the relationship between the Hawkins classification and avascular necrosis.7 Despite these concerns, the Hawkins classification system remains the most commonly used system for talar neck fractures.

CLINICAL AND RADIOLOGIC EVALUATION

Talar neck fractures are usually due to high-energy trauma. The most common mechanism of injury is either a high-speed motor vehicle accident or fall from height. Talar neck frac-



total spectrum of the patient's injuries, and a general trauma survey should be included in each patient's evaluation. Particular attention should also be directed to the thoracolumbar spine, because spine fractures have been found in association with talar neck and body fractures.8 Awake patients will report severe foot and ankle pain. Physical examination will reveal significant swelling and tenderness over the hindfoot and midfoot. Gross deformity may be present depending on the displacement of the fracture and any associated subluxation or dislocation of the subtalar or ankle joint. Because of potential damage to the neurovascular structures, it is important to perform a thorough assessment and examination of the affected foot.

tures may be only part of the

Routine radiographs of the ankle, consisting of anteroposterior, mortise, and lateral plain radiographs, are used to identify fractures and displacement of the talar neck. The special oblique view of the talar neck described by Canale and Kelly⁵ provides the best evaluation of talar neck angulation and shortening, which is not easily appreciated on routine radiographs. This radiograph is made by placing the ankle into maximum equinus and pronating the foot 15° while the x-ray tube is angled 75° from the horizontal plane (Figure 2). If plain radiographs do not clearly identify a fracture in a patient with a high suspicion for a nondisplaced talar neck fracture, computed tomography (CT) may be recommended to avoid the possibility of a missed talar neck fracture.⁹ Preoperatively, CT scans are useful for assessing comminution and displacement of the fractures, as well as providing accurate images of the ankle, subtalar, and transverse tarsal joints.

EMERGENCY TREATMENT

Fractures of the talar neck that are completely nondisplaced on a CT scan can be treated in a short-leg nonweight-bearing cast in neutral position. The patient should be carefully followed with serial radiographs to ensure that the fracture does not displace during treatment. Treating physicians should bear in mind that failure to address subsequent fracture deformities leads articular incongruency. to Therefore, some authors recommend internal fixation for even nondisplaced talar neck fractures.10 An additional benefit of internal fixation is that it permits early motion.

Because of the high-energy mechanism and limited soft tissue envelope, 21% of talar neck fractures are open fractures,⁴ requiring emergent operative debridement and irrigation to reduce the risk of infection. If primary anatomic reduction and fixation is not possible, (eg, seriously ill multiply-injured patients or patients with complex foot trauma) provisional fixation may be performed by either K-wires or a spanning external fixator.10 Some authors have advocated the use of an external fixator that provides distraction to the ankle joint to unload the talus, with hopes of reducing the morbidity of avascular necrosis.^{11,12} However, Besch et al¹³ concluded that the external fixation has no effect in the prevention of avascular necrosis following talar neck fractures.

It remains controversial whether talar neck fractures require emergent treatment. The time of definitive fixation always depends on multiple factors, including fracture comminution, soft tissue conditions, available resources, surgeon experience and comfort level, and medical status of the patient.14 In several clinical studies, the timing of internal fixation did not have a significant effect on the rate of avascular necrosis or the functional outcome.^{15,16} Hence, the investigation done by Patel et al¹⁷ indicates that most expert orthopedic trauma surgeons do not believe that immediate operative treatment is necessary for displaced talar neck fractures. Most reported that the operation can wait more than 8 hours, with a significant proportion reporting that treatment in more than 24 hours is acceptable.

Although delayed fixation may be suitable for talar neck fractures, a provisional closed reduction under local anesthesia to relieve the increased skin and neurovascular bundle tension caused by displaced fracture fragments should be considered. Once reduced, the dislocated joint typically stabilizes because of the shape and fit of the articular surfaces and surrounding structures. Repeated forceful reduction attempts should be avoided.

Surgical intervention is indicated for type II, III, and

IV fractures if an acceptable closed reduction cannot be obtained. Adelaar¹⁸ recommended open reduction and internal fixation of any fracture with more than 3 to 5 mm dorsal displacement or any rotational deformity. Most authors have stressed that type II, III, and IV fractures should be treated by open anatomic reduction and stable internal fixation to restore articular congruity and permit early motion.

OPERATIVE TREATMENT

Most surgeons recommend the use of dual surgical approaches, anteromedial and anterolateral, to allow accurate visualization and anatomic reduction of talar neck fractures.^{16,19,20} The anteromedial approach begins at the anterior border of the medial malleolus and extends toward the navicular tuberosity, just between the anterior tibial and posterior tibial tendons. Laterally, the incision begins at the Chaput tubercle on the tibia and extends toward the bases of the third and fourth metatarsals.²¹ However, the Ollier approach, oblique from the tip of the lateral malleolus to the neck of the talus, is also effective, and allows better control of the lateral process and the anterior part of the posterior subtalar joint.22 If the fracture progresses posteriorly into the body of the talus, a medial malleolar osteotomy is recommended,^{19,23} although this is more frequently suggested for talar body fractures.

While dual approaches are commonly used, Ohl et al²⁴ cautioned that aggressive surgical dissection with dual approaches might be harmful to the talar blood supply, increasing the risks of skin necrosis and avascular necrosis. It is important to carefully preserve any remaining talar blood supply, regardless of the approach or approaches.

The goal of talar neck fracture treatment is anatomic reduction of both the neck and subtalar joint, because even minimal residual displacement can adversely affect subtalar joint mechanics.8,25 It is important to avoid reducing the talar neck fragment in supination, pronation, or axial malalignment. Because rotational alignment is very difficult to judge, dual approaches are usually required. Provisional K-wires may be placed in the talar body and talar head fragment to serve as a joystick to correct the displacement and deformity. This technique avoids the use of a pointed reduction clamp that may require a larger exposure and cause more vascular compromise.22

To achieve stable internal fixation and decrease the rate of malunion, at least 2 screws are required. Numerous types of screws have been described for talar neck fracture fixation, but titanium screws have the advantage of compatibility with MRI, allowing early detection of osteonecrosis. Bioabsorbable screws have some theoretical advantages, in that they can be placed through the articular surface and resorb over time.²⁶

Most authors^{10,27} prefer to place screws from anterior to posterior because the fracture site is routinely exposed from an anterior approach (Figure 3). However, Swanson et al²⁸ compared the biomechanical strengths of various fixation methods in a transverse, noncomminuted talar neck fracture model, and concluded that posterior-to-anterior screw fixation was stronger. Posterior-to-anterior screw fixation has potential disadvantages, including requiring an additional posterior approach with potential injury to the peroneal artery and its branches and screw head prominence that can limit ankle plantarflexion. Furthermore, if a posteroanterior screw is situated in the lower half of the head, the shaft of the screw protrudes into the roof of the sinus or canal tarsi, and can injure the canal tarsi artery.29 Attiah et al27 studied different screw configurations in a comminuted talar neck fracture model. They compared 3 anteroposterior screws, 2 cannulated posteroanterior screws, 1 screw from anterior to posterior, and a medially applied blade plate. They concluded that the anteroposterior screws had approximately 20% lower yield point and stiffness compared to the posteroanterior screws or blade plate techniques, but this difference was not statistically significant.

Lag screws are typically used to compress talar neck fractures to withstand early motion which, is beneficial for ankle and subtalar joint function. However, when there is comminution of the talar neck, especially the medial column, the use of a lag screw may be contraindicated, as it will

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Figure 3: A 37-year-old woman sustained multiple injuries in an automobile vs train accident, including a displaced talar neck fracture with subtalar joint dislocation (A) and subluxation of the tibiotalar joint (B). Due to her multiple associated injuries, her talus fracture was initially treated by closed reduction (C) and application of a spanning external fixator (D). When her condition improved, she underwent open reduction and internal fixation using dual surgical approaches and 2 anterior to posterior lag screws (E-G). The patient has a good clinical and radiographic result 18 months after reconstruction (H, I).

cause deformity and malunion. Transfixion screws are used to avoid compression and maintain the correct length of the talus.^{8,20} Bone grafting is occasionally needed to replace areas of impaction defects to restore the neck length.

For comminuted talar neck fractures, many authors have advocated plate fixation with or without neutralization screw fixation (Figure 4).^{10,19,20,30} By providing a solid buttress as a bridging strut, plates can be placed on the most comminuted column of the talus,

either medial, lateral, or bilateral columns. Plate sizes used range from 2 to 2.7 mm. Plates not only provide longitudinal structural support, but also prevent supination or pronation of the distal fragment. Charlson et al³¹ compared posteroanterior screw fixation and plate fixation in comminuted talar neck fractures, and found that while plate fixation may offer substantial advantages in the ability to control the anatomic alignment, it does not provide any biomechanical advantage compared with screw fixation.

Intraoperative fluoroscopy is a valuable tool to assess the reduction accuracy and implant position. Arthroscopic techniques under fluoroscopy may be helpful to provide better visualization of the articular surface, which may enhance reduction accuracy and allow debridement of loose fragments.

AFTER TREATMENT

Patients treated conservatively in a below-the-knee cast are kept nonweight bearing for at least 6 weeks. Partial weight bearing is generally allowed after 6 to 8 weeks, and total weight bearing is permitted when there is convincing evidence of healing.

Open reduction and internal fixation aims at stable fixation, which permits early mobilization, decreasing the likelihood of stiffness. Once the wounds are healed, early postoperative active motion begins, depending on the degree of stable fixation and fracture comminution. Motion of the joints improves cartilage healing.22 Partial weight bearing is generally restricted for 6 to 12 weeks, and full weight bearing is delayed until radiographs show the fracture healing.

AVASCULAR NECROSIS

Avascular necrosis of the talar body, resulting from interruption of the precarious vascular supply to the talus, is the most dreaded late complication after talar neck fractures. The risk of developing avascular necrosis in a Hawkins type I fracture is only 0% to 15%, since only the blood supply entering through the neck is disrupted. Hawkins type II fractures have a 20% to 50% risk of avascular necrosis, with the artery of the tarsal canal and the dorsal blood supply from the neck being disrupted. Type III and IV

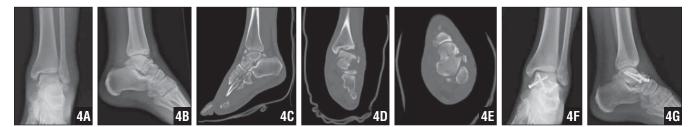


Figure 4: A 24-year-old man sustained a Hawkins type II talar neck fracture while riding his bicycle with a fixed cleat pedal (A, B). CT scans show the comminuted fracture of the talus, which extends through the neck neck to the middle articular facet (C-E). Radiographs 7 months after open reduction and internal fixation. Dual approaches were used, and that fracture fixed with 2 AP screws and a 2-mm lateral plate to maintain the talar neck length (F, G).

fractures have a 69% to 100% risk of avascular necrosis, with all 3 main sources of blood supply damaged.4,5,7,32 Greater displacement, comminution, and open fractures could increase the likelihood of developing avascular necrosis. Avascular necrosis of the body weakens the talar trochlea, subjecting it to collapse if full weight bearing is allowed in the presence of avascular necrosis.1 Whether collapse of the talar dome is partial or full, the subsequent degenerative changes lead to pain and disability in both the ankle and subtalar joints, along with shortening of the affected leg.4,5,33

The Hawkins sign, which is described as a prognostic indicator of revascularization to the talar body, appears between 6 and 8 weeks after talar neck fractures, and can be radiographically visualized on the anteroposterior or mortise view. The preserved blood supply resorbs the subchondral bone of the talar dome, creating a disuse osteopenia, which appears as a radiolucency of the talar dome and indicates preserved vascularity of the talus.⁴ In clinical practice, the Hawkins sign is strongly predictive of the absence of avascular necrosis. However, the sign is highly sensitive but less specific, such that its absence does not universally predict avascular necrosis.^{5,32,34} Magnetic resonance imaging evaluation can also be useful to assess the percentage of avascular necrosis involvement and help guide appropriate treatment.35

Imaging should begin with plain radiography, which

shows avascular necrosis as a relative sclerosis of the talar body when compared with surrounding bone. The presence of this relative sclerosis may not become apparent until as late as 4 to 6 months after injury. Magnetic resonance imaging is the most sensitive test for determining the presence of avascular necrosis and estimating the amount of talar dome involvement. Adipocyte viability produces strong T1weighted images. Magnetic resonance imaging is sensitive to the change signals of death of marrow adipocytes resulting from avascularity. Some authors, however, point out that MRI is not helpful in assessing osteonecrosis until at least 3 weeks after the time of injury.36,37

Although the chance of developing avascular necrosis is almost completely determined at the time of injury, surgical management techniques, including prompt and accurate reduction of the dislocation, may decrease this likelihood. A thorough understanding of the anatomy and meticulous surgical dissection are essential to prevent further injury to the remaining vessels.³⁸ Tang et al¹² reported on the use of a vascularized cuboid pedicle bone graft, combined with internal and external fixation, and showed that this method effectively could prevent avascular necrosis in their preliminary study. Mei-Dan et al³⁹ also suggested that the addition of hyperbaric oxygen therapy to both operative and rehabilitative therapy may be associated with significantly

improved outcomes. While these techniques aim to decrease the risk for posttraumatic talar avascular necrosis, there are no critical reviews of their effectiveness.

Before articular collapse, the patient may be asymptomatic and function satisfactorily without discomfort. The talus will often revascularize spontaneously if given enough time. This occurs from medial to lateral through creeping substitution and takes several years.4 Once the diagnosis of avascular necrosis has been established, nonweight bearing, or partial weight bearing, should be recommended to prevent talar collapse. Canale and Kelley⁵ found that those who were kept nonweight bearing on crutches for an average of 8 months had fair-to-excellent results, and those who were partial weight bearing in a patellar tendon brace or short leg brace with limited ankle motion had poorto-good results. However, those receiving no treatment, defined as nonweight bearing for <3months, had mostly poor results. No consensus exists on either the duration or degree of restricted weight bearing, or on the utility of bracing or immobilization in minimizing the sequelae of osteonecrosis.40 Other authors believe that nonweight bearing is of questionable value in preventing collapse if avascular necrosis develops.3,4

When nonsurgical management fails to prevent avascular necrosis and collapse of the talar dome, surgical interventions should be considered. Secondary or salvage treatments include talectomy, bone grafting, tibiocalcaneal fusion, Blair fusion, and pantalar fusion. Talectomies yield poor outcomes, resulting in frequent pain, a short limb, and significant loss of ankle and subtalar motion.^{4,5} Arthrodesis has been suggested for use during primary treatment of severe talar neck fractures, with the aim of eliminating pain and the limitations of subtalar arthritis.41 However, we generally reserve arthrodesis as a salvage treatment following failure of internal fixation. A stainless steel talar body prosthesis introduced by Harnroongroj42 may be useful in treating avascular necrosis or severe crush injuries of the talus.

MALUNION AND NONUNION

Talar neck fractures frequently develop malunion and nonunion, leading to decreased range of motion. The incidence of malunion has been reported to be approximately 30%,^{5,43} and the incidence of nonunion is approximately 2.5%.8,16 Typical findings of malunion are varus malalignment of the talar neck and deformity of the medial column. Sangeorzan et al²⁵ stressed that malalignment of only 2 mm results in significant changes in the subtalar contact characteristics that could lead to the progressive development of posttraumatic arthritis. It is difficult to accurately evaluate residual step-offs and alignment on plain radiographs. Chan et al⁴⁴ compared the ability of plain radiographs, computed tomography (CT), and radiostereometric analysis to detect changes in talus fracture fragment position and alignment using an in vitro model. The most accurate imaging method to measure malunion was CT scan. The 3D CT scan reconstructions allow the clinician to better appreciate the talar neck malunion.

Arthrodesis is the primary salvage procedure for talar neck malunion or nonunion, but does not restore normal foot function.5,43,45 Some authors46-48 have recommended surgical restoration of the anatomical shape of the talus for the treatment of malunions. Secondary reconstruction is dependent on the status of the soft tissues, the joint cartilage, and the presence of avascular necrosis. This salvage procedure corrects the foot malposition by an osteotomy through the malunited fracture or removal of the pseudarthrosis, and restoring the medial neck length using additional bone grafting if necessary. If talar neck malunion or nonunion occurs in the presence of complete avascular necrosis or septic necrosis of the talus, removal of all necrotic and infected bone combined with bone grafting or shortening and arthrodesis of the affected joints are recommended.48

POSTTRAUMATIC ARTHRITIS

Long-term follow-up studies have shown high rates of posttraumatic arthritis after talar neck fractures. Causes of posttraumatic arthritis may be multifactorial, and may include damage to articular cartilage at the time of injury, progressive cartilage degeneration from fracture malunion, nonunion causing malalignment and incongruence, or osteonecrosis. The incidence of posttraumatic arthritis after talar neck fractures ranges from 50% to 100%,43,49 and is a more common finding than osteonecrosis.49 The arthritis primarily involves the subtalar joint, but may also affect the ankle and talonavicular joints. The subtalar joint is prone to arthritis because the calcaneus slides past the talus during dislocation, causing compressive and shearing forces that may result in injury to the cartilage.50 Not all cases of posttraumatic arthritis become symptomatic. The development of severe arthrosis causing chronic pain and stiffness may necessitate arthrodesis if conservative treatment is ineffective.

CONCLUSION

Talar neck fractures have been associated with a high incidence of complications, including osteonecrosis, infection, skin necrosis, malunion, nonunion, and posttraumatic arthritis. The high-energy nature of the injury required to produce a displaced talar neck fracture also causes severe associated soft tissue damage, including damage to the precarious blood supply. Anatomic reduction and internal fixation of displaced neck fractures to restore and to maintain alignment has a key role in minimizing the complications rate. However, the sequelae of posttraumatic complications may be inevitable. Even in the absence of osteonecrosis following anatomic fracture reduction and fixation, patients frequently experience chronic pain and stiffness due to posttraumatic arthritis. 0

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