CHAPTER 8

FRACTURES OF THE LATERAL TALAR PROCESS: A Case Presentation and Mechanical Proposal

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Fractures of the lateral talar process are considered to be a relatively uncommon injury. These fractures account for less than one percent of all ankle injuries. It is interesting to note, however, that this particular injury is the second most common fracture according to the Hawkins series from the University of Iowa. This injury is frequently missed in general emergency room settings, as illustrated by the Hawkins study, which reported six of thirteen cases being missed upon initial evaluation. To appreciate the morbidity of this injury, it is important to recall that in many cases, this fracture is intra-articular to the ankle as well as the subtalar joint.

ANATOMY

The lateral talar process articulates with the fibula superiorly and laterally, and the posterior facet of the calcaneus inferiorly. Soft tissue attachments are limited to the lateral talocalcaneal ligament, the anterior talofibular ligament, and the posterior talofibular ligament. The anterior and posterior talofibular ligaments originate, to a great extent, superior to the apex of the lateral talar process, while the lateral talocalcaneal ligament originates from the apex. The lateral talocalcaneal ligament is thought by Sarrafian to be intimately associated with the calcaneofibular ligament, both structurally and functionally. Some authors have described the lateral talocalcaneal ligament as "a mere thickening of the capsule of the subtalar joint."

MECHANISM OF INJURY

Previous accounts in the literature have credited several different mechanisms with generating the lateral talar process (LTP) fracture. The most popular theory has attributed the injury to a dorsiflexion and inversion mechanism. Certainly, some cases may be attributed to this mechanism, such as those described by Hawkins, with associated vertical fractures of the medial malleolus, lateral ligament disruption, and avulsion fractures of the distal fibula.

Other suggested mechanisms have included lateral talar process fracture as an avulsion by the lateral talocalcaneal ligament with inversion of the rearfoot, and direct compression or trauma to the lateral talar process. Although these mechanisms may hold some credence, it is unclear to the authors the mechanical forces involved to generate the lateral talar process injury by their proposed mechanism.

Several other authors, in their descriptions of this injury, have given cursory mention to associated injuries, but have failed to correlate them with the lateral talar process injury itself. One example of this can be seen by Kettumen et al. where an arthrogram was performed, and extravasation of the contrast media was noted into the tibialis posterior tendon sheath and the area of the anterior talofibular ligament. Lateral leakage was attributed to attenuation, without disruption of the anterior talofibular, and calcaneofibular ligaments, while the medial leakage was suggestive of a medial lesion. No correlation was suggested between the deltoid lesion and the lateral talar process fracture.

Of particular interest when discussing the mechanisms of the lateral talar process fracture are Dimon's hypothesis for this injury. Dimon postulated three possible mechanisms: 1. The lateral talar process fragment is sheared-off by the fibula when the foot is forced into eversion (Fig. 1). 2. The fragment is avulsed by the anterior talofibular ligament when the foot goes into inversion (Fig. 2). 3. The fragment is sheared-off of the posterior facet of the talus by the corresponding area of the calcaneus during forced dorsiflexion and external rotation (Fig. 3).

Dimon discounted the forced eversion mechanism due to the fact that he had not seen any cases of lateral talar process fracture with associated deltoid sprain/disruption or medial malleolar

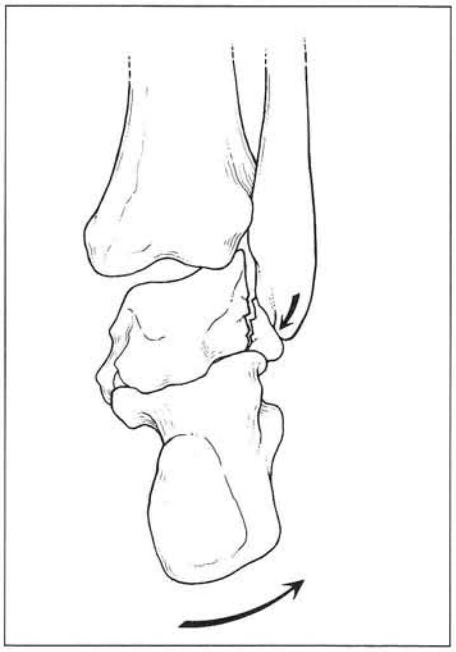

Figure 1. The lateral talar process fragment is sheared-off by the fibula when the foot is forced into eversion

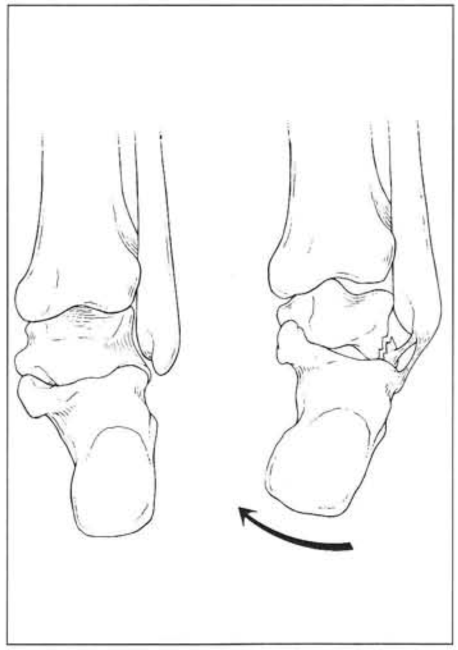

Figure 2. The fragment is avulsed by the anterior talofibular ligament when the foot goes into inversion.

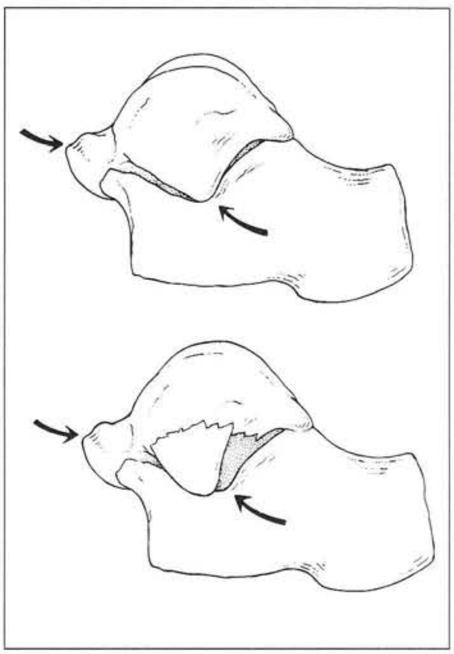

Figure 3. The fragment is sheared-off of the posterior facet of the talus by the corresponding area of the calcaneus during forced dorsiflexion and external rotation.

fracture. The second mechanism, he also doubted, since the anterior talofibular ligament at surgical evaluation in several injuries was found to originate for the great part superior to the fracture line. Finally, Dimon stated that he felt that the injury was caused by dorsiflexion and slight external rotation.

PROPOSED MECHANISM

A combination of Dimon's dorsiflexion-external rotation and forced eversion mechanisms has been proposed by the principle author. With the foot in a dorsiflexed position, eversion and external rotation forces would impact the lateral aspect of the posterior facet against the lateral talar process and potentially the lateral talar process against the fibula (Fig. 4). The velocity, and therefore the fragmentation, would be increased if the restraining device (the deltoid ligament) were first disrupted. A mental comparison of this mechanism to that involved in the pronation-eversion injury in the Lauge-Hansen scheme makes this an even more plausible proposal, with the position of the calcaneus at initiation of the injury perhaps being the determining factor as to which injury pattern will occur. One should recall that stage one of the pronation-eversion fracture pattern is disruption of the deltoid ligament or fracture of the medial malleolus. The hypothesis being that the more everted the foot, the more likely the lateral talar process injury and the less likely the pronation eversion injury. This is based on clinical observation and evaluation of LTP fractures, such as the following case.

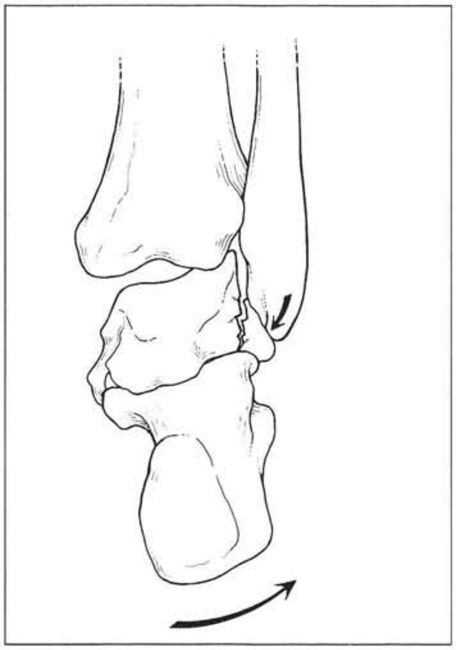

Figure 4. A combination of Dimon's dorsiflexion-external rotation, and forced eversion mechanisms proposed by the principle author.

Figure 5. AP view of the ankle showing fragmentation of the lateral process of the talus.

CASE PRESENTATION

K.H., a 27-year-old male presented to the emergency room following a Sunday-afternoon softball game. The patient related that he hurt his right ankle while sliding into home plate. The patient was unsure as to the mechanism of injury.

Clinical examination showed marked edema, both medially and laterally. Physical examination revealed pain on palpation of the lateral inframalleolar area, not any different than would be expected with a classic grade two ankle sprain. In addition, however, there was also pain on palpation of the deltoid ligament, medially.

Plain film x-rays showed a comminuted fracture of the lateral talar process (Fig. 5). Prior to re-evaluation, a CT scan was performed to further inspect the degree of intra-articular involvement (Fig. 6). Significant involvement of both the ankle and subtalar joints, combined with the principal author's previous experience with this injury, led to the decision to undertake surgical treatment of the pathology.

Following four days of compression, elevation, and cryotherapy, the patient was scheduled for excision of the fracture fragments and primary repair of the deltoid ligament. Intraoperatively, a midbody tear was identified in the deltoid ligament, with

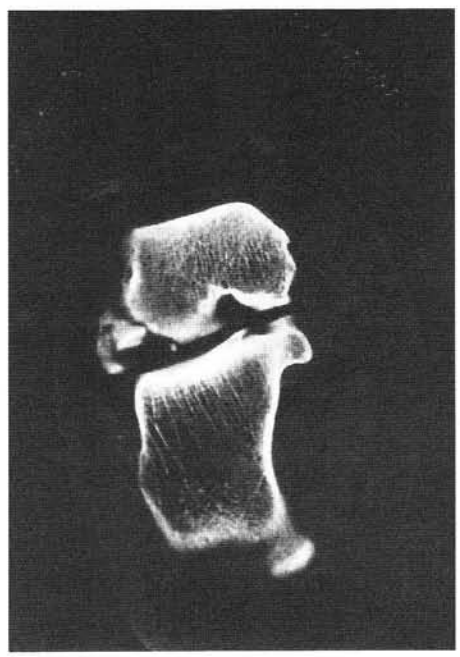

Figure 6. Coronal section CT scan showing the degree of comminution, and intra-articular nature of the fracture.

organized hematoma formation. The hematoma was removed with copious amounts of irrigation, and the ligament was reapproximated using 0 and 2-0 non-absorbable sutures. Reinforcement was performed with 2-0 absorbable suture. Subcutaneous and skin closure was then performed in the usual fashion.

Laterally, the fragments were accessed anterior and inferior to the fibula. At the level of the deep fascia, palpation of the lateral process showed crepitation. After opening the ankle joint capsule, careful evaluation confirmed that primary repair would be impossible due to the degree of comminution, therefore removal of the fragments was undertaken. Following the removal of the fragments, remodeling of the lateral aspect of the talus was performed using power and hand instrumentation (Fig. 7).

Figure 7. Postoperative AP radiograph showing removal of the fragments.

Postoperatively, the patient was maintained non-weight bearing in a short leg cast for four weeks. At four weeks, the patient was placed in a short leg, removable splint, and range of motion exercises were instituted. Short-term follow-up of the patient revealed a good range of motion with minimal discomfort.

DISCUSSION

The literature is filled with descriptions of below average to poor result in patients with lateral talar process fractures. The morbidity is considered by many authors to be out of proportion to the magnitude of the injury, but in the authors' opinion is quite reasonable considering the dual intra-articular nature of many of these injuries.

Conservative treatment has previously been advocated, but more recent reports in the literature have related good result with primary repair of these injuries, restoring anatomic alignment to the joint. Understanding the mechanism of injury is paramount to treating it successfully. A careful clinical examination, either shortly after the injury (less than 1 hour) or after several days of compression and cryotherapy, should aid in determining the mechanism. The authors believe that many of these patients will exhibit some degree of deltoid tenderness, indicating partial or complete disruption of this structure. When medial tenderness is noted, it should lead the practitioner toward the eversion/external rotation mechanism discussed.

In the case presented, if one imagines an athlete sliding into home plate on his right side, it is easy to identify the forces involved as strong eversion, from the lateral border of the foot against the ground, and an external rotation force from the foot hitting the base and the leg rotating upwards. The description of eversion and external rotation (with the foot in a dorsiflexed attitude) is open-kinetic chain, while the injury itself is obviously occurring with closed-kinetic chain events and forces.

Although further experimental investigation needs to be done, with the imaging studies currently available, it becomes a fairly elementary exercise to evaluate the soft tissue damage, the degree of intra-articular involvement, and the potential for repair of this injury. Treatment should involve primary repair of involved soft tissues, as well as the anatomic reduction and fixation of fracture fragments when possible. When marked comminution is encountered, excision of fragments with remodeling of the remaining architecture should strongly be considered.

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