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ORIGINAL RESEARCH

Incidence of Lateral Talar Dome Lesions in SER IV Ankle Fractures

Dean L. Sorrento, DPM,[1] and Alan Mlodzienski, DPM, FACFAS[2]

Osteochondral lesions of the talar dome are commonly the result of ankle trauma. While the technique of surgical repair of ankle fractures has been well reported, there are no studies that correlate the presence or absence of talar dome lesions. A possible explanation for this may be lack of intraoperative inspection of the talar articular surface. This retrospective study evaluates the incidence of lateral talar dome lesions in 50 supination–external rotation stage IV ankle fractures. Specifically, operative reports were reviewed for the presence of lateral talar dome lesions documented through intraoperatve inspection. Overall, 19 of 50 fractures, or 38%, were found to have a lateral talar dome lesion. While the bimalleolar and deltoid ligament tear type fractures exhibited more talar dome lesions, there was no significant difference between these two fracture types ($p = .1111$). There was no statistically significant difference among the three types (unimalleolar, bimalleolar, and trimalleolar) of supination–external rotation ankle fractures ($p = .0804$). The authors conclude that intraoperative inspection of the lateral talar dome should be a routine part of ankle fracture repair. (The Journal of Foot & Ankle Surgery 39(6):354–358, 2000)

Key words: ankle, fracture, talar dome

A talar dome lesion (TDL) is a localized injury to the articular surface of the talus. This injury involves damage or separation of the cartilage and underlying subchondral bone, hence the term *oseochondral fracture*. The fracture damages the vascular supply to the subchondral bone. The lesions may heal with nonweightbearing immobilization if treated early, as capillaries can restore blood flow to the fracture site via the process of creeping substitution. The diagnosis frequently, however, is delayed or missed and with prolonged weightbearing, fibrous tissue accumulates, blocking capillary ingrowth and leading to avascular necrosis. Ultimately, subsequent pain ensues and the TDL progresses to degenerative joint disease (1).

A paucity of literature exists that directly correlates TDL with ankle fractures. Roden and Tillegard were the first to propose two etiologies based on the presence or absence of trauma (1). Lateral osteochondral lesions had a history of trauma and medial lesions had no known traumatic episode. Because lateral lesions are the focus of this study and are more likely to be secondary to trauma, their discussion will be emphasized. The work of Berndt and Hardy popularized a traumatic etiology of lateral TDL and provided a staged mechanism of injury and classification system (2). Using cadaveric specimens, they demonstrated that the lateral lesion was the result of inversion of the dorsiflexed ankle. A strong inversion force rotates the talus laterally in the ankle mortise, resulting in impaction of the superolateral talar margin against the articular surface of the fibula. With a progressive inversion force, the severity of the lesion, increases. Medial lesions, conversely, resulted from inversion of the plantarflexed ankle allowing the inferior lip of the tibia to come in contact with the superomedial ridge of the talus. Additionally, they developed a classification system based on the radiographic appearance of the lesion. Others have also found trauma to be the cause of lateral lesions, while 80% of medial lesions were not associated with a traumatic event (3, 4). The majority of traumatic lesions result from an acute ankle sprain (5). Approximately 2–6% of acute ankle sprains have concomitant osteochondral fractures

From University of Pennsylvania Health System/Presbyterian Medical Center, Philadelphia, PA. Address correspondence to: Dean L. Sorrento, DPM, University of Pennsylvania Health System/Presbyterian Medical Center, Medical Arts Building, Suite III, 39th & Market Streets, Philadelphia, PA 19104-9563.

[1] Chief Resident, Podiatric Surgery.

[2] Direct of Residencies, Podiatric Medicine and Surgery.

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of the talar dome (6). Hutchinson and Wardle reported the incidence of osteochondral fractures to be 77% when the inversion talar tilt was 18° or greater utilizing a Telos stress device (7). These lesions may not be readily detected by standard radiography. Initial anteroposterior and lateral radiographs have been reported to be negative for osteochondral TDL in one third of patients with a lateral ankle sprain (8, 9). It has been well established that the lateral ligament complex must be injured for the stage of the lesion to advance (2). Flick and Gould found that a concurrent ligament injury occurs in 28–45% of talar dome fractures (10).

Many authors site trauma as the etiology of TDL, but few have addressed their concurrent presence with ankle fractures. Berndt and Hardy, in their series of 24 patients with TDL, reported that five patients had a history of a lateral malleolar fracture and four had a medial malleolar fracture (2). In another study, Scharling studied 19 patients with a talar dome lesion, only one of which had a lateral malleolar fracture with dislocation (11). In both studies, the diagnosis was delayed. In the latter study, no intraoperative inspection of the talar dome was mentioned.

Based on the aforementioned reports, one would suspect a higher correlation between ankle fractures and TDL due to the significant force and resultant instability of the injury. Supination–external rotation stage IV (SER IV) ankle injuries are one of the more common types of ankle fractures second only to SER II (11–13). It has been well documented that these injuries often require open reduction and internal fixation to restore stability to the ankle joint (11–16). No studies, however, mention inspection of talar dome as part of the operative technique.

Open reduction of these injuries does afford better anatomic alignment, but the results are not without complication. Bauer and Bergstrom, in a series of 92 ankle fractures, found that 30% of their patients had residual subjective discomfort more than 5 years after injury (17). This is consistent with earlier studies by Cedell (18) and Klosser (19), where each had 27% and 28% of patients, respectively, with residual complaints of stiffening swelling and pain with activity. Similarly, Hughes and Weber. (20) found poor results in 17–24% of their patients after operative treatment, while Wilson and Skilbred (21) had 65% of their patients complain of discomfort.

In addition to subjective complaints, the incidence of arthritis has been a documented concern. Bauer and Bergstrom's study revealed as many as 60% who still had arthritis even after "exact anatomic reduction"(17). Wilson and Skilbred concluded that operative reduction more often caused arthritis than nonoperative reduction (21). As Bauer and Bergstrom stated, "other factors predisposing to late discomfort and arthritis may be primary injury to cartilage and/or soft tissues as suggested by Felsereich"(17). Hence, a possible explanation for persistent intra-articular complaints may be the presence of concomitant TDL. The purpose of this study is to document the incidence of TDL in SER IV ankle fractures.

FIGURE 1 Intraoperative technique of viewing the lateral shoulder of the talar dome. Note bone hook displacing fibular fragment exposing the talar dome.

Materials and Methods

This study consisted of a retrospective review of operative reports and medical records from 50 randomly selected patients who underwent open reduction of SER IV ankle fractures. All cases were performed at the authors' institution from 1996 to 1998 by multiple surgeons. Each fracture was repaired utilizing internal fixation according to standard AO technique. Intraoperative inspection of the lateral articular surface of the talus, if performed, was through a standard approach demonstrated in Figure 1. In cases where the medial side was opened, such as a medial malleolar fracture or failure to reduce a medial clear space (deltoid tear), the medial articular surface was inspected.

Recorded parameters included patient age, fracture type (deltoid ligament tear, bimalleolar, or trimalleolar), and presence or absence of a lateral TDL. Only those reports with specific reference to the absence or presence of a lateral TDL requiring treatment (excision of cartilage and subchondral drilling) were included in the study. Those fractures in which no mention of the presence or absence of a TDL was made in the operative report were omitted, as it was unknown whether or not the surgeon or resident dictating the report failed to include it or it was not present. Fifty fractures and operative reports were available for analysis, which documented either the presence or

TABLE 1 Classification of osteochondral lesions according to Berndt and Hardy

Stage	Description
I	Compression of articular cartilage and subchondral bone
II	Partially detached fragment of subchondral bone and overlying cartilage
III	Complete detachment of the fragment without displacement
IV	Complete detachment of the fragment with displacement

absence of a lesion. The lesion was determined to be acute and traumatically induced based on intraoperative appearance, which consisted of an exposed and hemorrhagic subchondral base, irregular border, absence of fibrous tissue, and fresh cartilage detachment. No attempt was made to stage the lesion. However, all osteochondral fragments were excised and the subchondral bone drilled with a 0.45-inch Kirshner wire, which presumes that they were at least a stage III injury (Table 1). Categorical descriptive statistics were performed using Statgraphics software, STSC, Inc., Rockville MD. Specifically, a chi-square test was performed to determine if there was a significant relationship between fracture types and presence of lesions. Values less than .05 indicate a significant relationship.

Results

Average patient age was 44 with a range of 19–69. Table 2 lists the incidence of lesions according to fracture type and presence or absence of talar dome lesions. Of the fractures reviewed, only the trimalleolar type occurred with dislocation. Nineteen of 50 fractures (38%) had a lateral lesion present. When the medial side was opened in the bimalleolar and trimalleolar fractures, no talar dome lesions were present. Figure 2 graphically depicts the distribution of lesions according to fracture type. Nine of the 19 fractures (47%) with lesions consisted of the bimalleolar type, while six of the 19 (32%) occurred in those with a deltoid ligament tear. Statistical analysis using chi-square test did not reveal a significant difference between the bimalleolar and deltoid ligament tear fracture types ($p = .1111$). Additionally, there was no significant relationship between all three fracture types and lesions ($p = .0804$).

Discussion

Ankle trauma has been described as an etiology of lateral TDL (2–4). According to Berndt and Hardy, the severe inversion force results in the talus externally rotating in the ankle mortise, causing an abutment of the talar dome against the articular surface of the fibula (2).

The correlation between TDL and ankle fractures has not been previously reported. Some studies mention a history of an ankle fracture as a possible etiology, but none link the acute presence of a lesion at the time of surgery (2, 10). This could be due to lack of inspection of the talar dome.

BM=bimalleolar
DLT=deltoid ligament tear
TMD=trimalleolar/dislocation
TM=trimalleolar

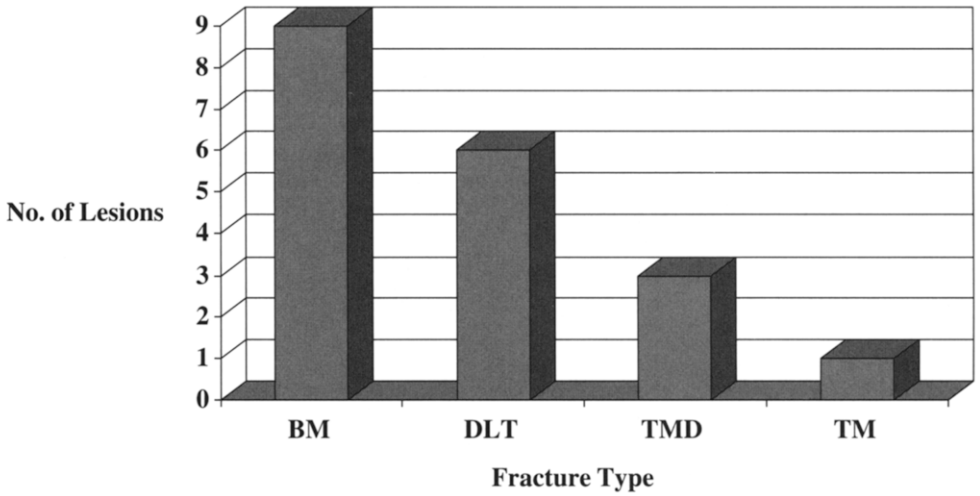

FIGURE 2 Graph demonstrating the distribution of talar dome lesions according to fracture type.

TABLE 2 Incidence of lesions according to fracture type

Fracture type	Lesion	No lesion
Bimalleolar	9 (18%)	8 (16%)
Deltoid ligament tear	6 (12%)	19 (38%)
Trimalleolar	1 (2%)	2 (4%)
Trimalleolar/dislocation	3 (6%)	2 (4%)

Table 2 demonstrates that most of the lesions, 15 of 19 (79%), were found with the bimalleolar and deltoid ligament tear type fractures. A reason for this is that these comprised the majority of fractures reviewed: 17 of the 50 (34%) were bimalleolar and 25 of 50 (50%) had a deltoid ligament tear. One would think a more unstable fracture would increase the chance of having a TDL. A dislocated type of fracture completely disrupts the osseous and soft-tissue constraints of the ankle mortise that may increase the vulnerability of the talar dome. A larger series with more of these fractures is needed to accurately support this notion. Nineteen of 31 (61%) of the patients without a lesion had a deltoid ligament tear type fracture. While these fractures seem less likely to have a TDL, the series contained more of this type compared to others. Clearly, a larger sample size with even distribution of fracture types is needed to accurately determine if SER IV with deltoid tear fractures have fewer TDLs. The bimalleolar fractures, conversely, demonstrated a near-even distribution of lesions and no lesions, 18% and 16%, respectively. We should note that the bimalleolar fractures reviewed did not include ankle dislocation. In the "lesion " group, most lesions occurred with the bimalleolar (47%) and deltoid tear (32%) type fractures. There was no significant difference between these two fracture types (p value = .1111). Conclusions regarding the trimalleolar and trimalleolar with dislocation type fractures are difficult since they comprised a small portion of the series, 6% and 10%, respectively. Because of the small sample size, the data were pooled and demonstrated no significant difference between fracture types and lesions (p value = .0804).

There is clear evidence that despite anatomic reduction, postoperative results of ankle fracture repair are not free of complications (17–21). These studies fail to mention inspection of the talar dome as part of their operative procedure. The authors believe that close inspection of the talar dome should be routinely considered in the surgical repair of ankle fractures. Proper identification and treatment of these lesions could possibly lead to improved long-term results with decreased postoperative sequelae.

Several limitations exist in this study. First, the study consisted of a retrospective rather than prospective design, based solely on medical records and operative reports. An inherent limitation with this design included multiple contributors to the data (residents dictating operative reports and multiple attendings) and different surgical techniques. For example, it is unknown if the failure to mention talar dome inspection in the operative report is an accurate account of the failure to inspect (as some surgeons may fail to inspect the talar dome or do not think it is necessary), an unconscious bias as to the presence or absence of a lesion, or simply an omission. Second, only SER type ankle fractures were included in the study because these were the more frequently encountered at our institution. Obviously, lesions may occur with other ankle fracture mechanisms and should be included in future studies. Bimalleolar fractures with dislocation were not observed and may have altered the results. The study also focused only on lateral lesions because they are more commonly associated with trauma. Lastly, this study did not prove or disprove a hypothesis but rather documents a previously undocumented correlation between ankle fractures and talar dome lesions. We believe this correlation is significant to justify routine inspection of the talar dome in the surgical treatment of ankle fractures.

In conclusion, the incidence of lateral TDL in SER IV ankle fractures was found to be 38%. This study lays the foundation for future research. Long-term studies with larger sample sizes comparing treatment versus nontreatment of talar dome lesions can lead to more predictable outcomes.

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