Evaluation of Posterior Malleolar Fractures

by Andrew Franklin DPM, PhD

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Posterior malleolar fractures are often observed within the collective environment of medial and lateral malleolar fractures. Its occurrence is such that there is considerable literature regarding their role and treatment protocols. Despite significant publications however, a clear unifying stance regarding the biomechanical and structural significance and influence on soft tissue attachments remains elusive. The objective of this article is to explore the current observations, attitudes and limitations of our understandings towards the significance of posterior malleolar fractures.

Key words: Posterior malleolus, PITFL, Syndesmosis, Ankle joint congruency

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The incidence of posterior malleolar (PM) fractures have been quoted to occur within 7% -44% of rotational ankle fractures [1]. Its respectable propensity has such dictated the customary classifications, treatment and fixation modalities within the literature. The literature however remains vague with conversing views and statements as to the effect that such fractures have on the stability and congruency of the ankle joint [2]. This is further complicated when one is obligated to address the ligamentous attachments to the posterior malleolus; specifically the posterior inferior tibial fibular ligament (PITFL) and whether one can consider them within certain scenarios as a collective unit. Such apparent complexity has culminated in a conjectured disposition or at least significant disparity as to the role, protocol and necessity of treatment of the posterior malleolar fracture. It is therefore prudent to re-assess the current views and past literature as to the role of the posterior malleolus in an attempt to gain clarity as to the significance or indeed, insignificance of these fractures. This assessment must begin with understanding the anatomy of the PM and its ligamentous associations. From there, one can speculate and review the structural and indeed syndesmotic implications of these fractures in its relationship to the ankle joint.

Anatomy

It would appear difficult to address the fracture of the posterior malleolus without considering the PITFL. Indeed the necessity to fixate a posterior malleolar fracture has frequently been associated with the role of the PITFL in its significant contribution to the strength of the syndesmosis. As such, the early work by Ogilvie-Harris is often quoted as the fundamental argument for addressing a posterior malleolar fracture [3]. To address this argument and before analysis of any other work can be implemented it is consequently imperative to elucidate whether one can assume a posterior malleolar fracture is indicative of an intact but removed PITFL. One must therefore fully understand the anatomy, attachment and propensity to remain intact (after PM fractures) of this ligament.
The syndesmotic joint as a whole appears somewhat neglected with little devotion to its structure and attachments within the current anatomical textbooks. Bartonicek however attempts to address this in his dissection and evaluation of the tibiofibular syndesmosis in 30 cadaveric specimens [4]. His work describes the PITFL as a strong compact ligament that runs horizontally and often continuous with the interosseous ligament. In addition he states that its lower margin essentially forms the articular labrum for the lateral ridge of the trochlea of the talus. In a separate study, Hermans et al used a combination of plain film, CT, MRI and cadaveric studies to assess the anatomy of the distal tibiofibular syndesmosis [5]. His observational study states that the PITFL forms a triangular shape with a broad base at the tibial insertion. The mean width of the ligament is 17.4 ± 3.5 mm with a tibial insertion thickness of 6.4 ± 1.9 mm and a fibular insertion thickness of 9.7 ± 1.7 mm (8.0–11.4 mm) [6]. Such differentiation in thickness would support the findings of Warner et al that found that 97% of all PITFL injuries in the absence of a PM fracture resulted from delamination from the tibia (with the remaining 3% from intrasubstance ruptures) [7]. In Ebraheim's study, the PITFL’s most distal fibers are found to be in close contact with the transverse ligament and often appear continuous with the fibular side of the PITFL [8]. The transverse ligament is observed to course horizontally between the proximal margin of the fibular malleolar fossa and the dorso-distal rim of the tibia. Whether the PITFL and the transverse ligament should be considered as one anatomic unit (as is the case of Bartonicek's work) or two distinct structures remains an area of debate, regardless; due to the terminal positioning of the transverse ligament and its intimate anatomical relationship with the PITFL the author speculates that both would be involved with all extents of PM fractures (excluding the most minimal). Indeed, the propensity of PM fracture resulting in the avulsion of an intact PITFL has been observed in numerous publications. Before Warner excluded all PM fractures in his study of isolated PITFL injuries, he noted that of the 34% of SER III and IV injuries that involved PM fractures, all had an attached and intact PITFL (elucidated via plain film and MRI) [7]. Furthermore, Gardner observed that of the 15 patients who sustained a closed PER IV fracture no complete ruptures of the PITFL were seen via MRI with only one exhibiting a partial rupture [9].

**Syndesmosis**

If we are therefore allowed to conclude that all PM fractures results in collective insult to the PITFL, one can then assume that all PM fractures will result in some form of syndesmotic destabilization. Subsequently the extent of that destabilization and its effect on the ankle joint requires examination.

The early work of Olgivie-Harris concluded that approximately 42% of the strength of the syndesmosis is made up of the PITFL [3]. This work is based on the difference of average force required to cause 2mm of lateral diastasis within the inferior tibiofibular joint when ligaments of the syndesmosis are resected. It should be noted however that such values are based on a limited number of specimens (8) with a high level of standard deviation (41.4% ± 12.1). Furthermore such work does not address whether such forces necessary for diastasis are experienced within the ambulating foot or whether other peri-ankle joint structures are contributory to the event of diastasis. Nevertheless, such critique may appear academic when one considers the literature support for fixation of the PM in the endeavor of recovering syndesmotic integrity. Indeed this has perhaps most notably been seen in Gardner's work on the assessment of syndesmotic stabilization [9]. Gardner assessed the resistance to rotation of the foot under 4N of torque to interpret ankle stability following PM fixation compared to syndesmotic screws (without PM fixation). Reduction of the PM with an intact PITFL restored stiffness of the distal tibiofibular articulation to 70% compared to 40% by syndesmotic screws further suggesting the important role the PITFL plays in syndesmotic stabilization. Miller also supports the necessity for fixation of the posterior malleolus in her study towards the stabilization of syndesmotic injuries [10]. She speculates that fixation of the posterior malleolus fracture restores more stability to the syndesmosis than transsyndesmotic fixation alone. The study essentially compares one group of PM fractures (with intact PITFL) treated with PM fixation to another group of PITFL ruptures without PM fracture treated with syndesmotic screws. Based on sequential post-operative FAOS scores and plain film radiographs it was concluded that PM fixation is at least equivalent to syndesmotic screws. We are obligated however to acknowledge that conclusions remain limited when one attempts to compare different
treatment modalities directly with different subject groups; that is unless the reader is absolute in the notion that the PM fracture and loss of the PITFL can be considered the same in this instance. The fact that Miller’s paper actively separates PM fracture from isolated PITFL would however suggest otherwise.

**Ankle Joint stabilization**

If the ability of PM fixation to re-acquire a level of syndesmotic strength is not of debate then the assertion that such action is mandated for ankle stability clearly is. Weening and Bandhari showed improved quality of life and functional outcomes (SMFA and Olerud & Molander scale) after 18 months follow up with accurate reduction of the syndesmosis (through transsyndesmotic screw placement) compared to their non-reduced counterparts [11]. Additionally, Leeds and Ehrlich showed decreased clinical outcomes and propensity for ankle arthritis in subjects with inadequate syndesmotic reduction [12].

To clarify, it would therefore appear a necessity to reacquire syndesmotic stability and integrity in the endeavor of improved long-term functional outcome of the ankle joint. Acquisition of this stability can be achieved through fixation of the PM fractures due to its intimate relationship with the PITFL. Such claims are however contrary to the findings of Boden in his study of the mechanical necessity of syndesmotic repair [13]. Boden et al has suggested that ankle joint stability is not necessarily based on the integrity of the distal tibiofibular syndesmosis but rather on the integrity of the deltoid ligament. This was postulated in his cadaveric study comparing the extent of diastasis (radiographically) in simulated PER injuries with sectioned syndesmosis and deltoid ligament (group I) with those with a sectioned syndesmosis with an intact deltoid ligament (group II). Boden found that syndesmotic widening occurred in Group I from 0.5 to 4.5mm under application of a 440N load with a maximum change in diastasis seen with a fibular fracture at 3.0cm to 4.5cm proximal to the ankle joint (referred to as the critical transition zone). Such widening is significant when compared to only 1.4mm increase when the deltoid remained intact; this observation is presumably due the restriction in translocation of the talus when still tethered to the medial malleolus. Thus Boden advised that rigid fixation of the fibular and medial malleolus should restore adequate stability of the syndesmosis without the need for transsyndesmotic screws when the deltoid ligament remains intact. It needs to be noted however that it is unclear in the paper whether the PITFL is involved in their resectioning of the syndesmosis. Despite this lack of clarification it would appear insignificant when addressing the suggested role of the deltoid towards maintaining tibiofibular cohesion. This concept gained support from Burns et al in 1993 in his study towards the indications of syndesmotic screws [14]. His cadaveric evaluation looked at the change in diastasis with application of a 445N axial load to the ankle joint in PER simulated fractures. He established that complete disruption of the distal tibiofibular syndesmosis resulted in a diastasis of 0.24mm. This was compared to a diastasis of 0.73mm with the disruption on the deltoid ligament, resulting in a 0.49mm increase in diastasis. The correlation to ankle instability was suggested with the addition of an observed 39% reduction in tibiotalar contact area and a 42% increase in tibiotalar peak contact pressure with the loss of the deltoid ligament (not observed when deltoid remained intact). Further more, Michelson and Waldman studied the effect of all simulated stages of PER type fractures on the cadaveric ankle joint [15]. He noted that neither fracture of the fibula (4 cm above the plafond) nor disruption of the syndesmotic apparatus to 6.0 cm above the plafond resulted in a significant change in coupled motion of the talus. In fact, only when the superficial deltoid was sectioned did the ankle exhibit increased external rotation in plantar flexion. Additionally when the deep deltoid was sectioned, the ankle dislocated in plantar flexion unless the fibula was stabilized. Michelson and Waldman thus concluded that there is no biomechanical support for placement of a syndesmotic screw unless the medial side cannot be stabilized anatomically.

From such evidence it can be considered that ankle syndesmotic injury may not necessarily lead to diastases; however, the coexistence of a deltoid ligament rupture will equate to talus instability thus directly destabilizing the ankle joint if not also potentiating the syndesmotic unit to future diastasis. This is perhaps contrary to Kennedy’s work that found that a deltoid ligament tear was not a good predictor of poor functional outcome albeit in a limited study of 4 patients [16]. Additionally, Hartford’s work showed that transection of the deltoid ligament
produced no statistically significant further change in contact area with simulated PM fractures. The relationship is further complicated when Mont et al explored the ability of post-operative radiographs to predict clinical outcomes of unstable ankle fractures after fixation [17]. His work reviewed 80 surgically treated ankle fractures and found that the only statistically significant radiographic predictors for poor outcomes were an abnormal medial clear space and PM fractures (>20%).

Interestingly, although syndesmotic widening was also suggestive of poor clinical outcome it was not statistically significant suggesting that the PM role in ankle stability may be independent of its contribution to syndesmotic stability. The author however speculates that it would appear that there is a respectable indication that the surgeon is at a minimum obligated to address either the deltoid insufficiency or insufficiency of the syndesmosis to acquire acceptable functional outcomes of the ankle joint.

**Talus Subluxation**

The assessment for the need for fixation is complicated with the perpetual challenge of distinguishing but also unifying the role the PM with the PITFL. If we are given the liberty to assume that the requirement of syndesmotic integrity is at least secondary in maintaining ankle stability; one must still consider the structural role the PM has towards the containment of the talus and thus ankle joint congruency.

The necessity for a stable and congruous ankle joint was highlighted by the fundamental work of Ramsey and Hamilton that reported a 42% reduction in contact area with 1mm of lateral displacement of the talus [18]. It is thus suggested that the maintaining the position of the talus within the ankle mortise is paramount to ankle stability. As such, many have speculated as to the role of PM fractures towards possible resultant joint incongruities. This has been explored in several clinical studies that have showed significant osteoarthritic changes and poor functional outcomes in ankle fractures that have involved the PM [19]. As such the necessity to fixate PM fractures fragments >25% has traditionally (but recently questioned) been advocated [20]. Macko et al explored this concept in 1991 when he compared the sequential removal of the PM fragments of increased size on the surface area of joint contact [21]. He noted that progressive loss in contact area was associated with increase fragment size of the PM (with one-half size fractures resulting in 35% loss of contact area with the static foot in the neutral position). In a similar study Hartford showed posterior malleolar fracture fragments of 25%, 33%, and 50% as visualized on lateral radiographs resulted in a decrease of 4%, 13%, and 22% in tibiotalar contact area respectively [22]. The assertion is made (but not calculated) that such decrease in contact area will result in increase peak pressures resulting in loss of stability, injury to cartilage and contribute to the development of early degenerative changes.

These studies are however contrary to the findings of Fitzpatrick, he used real time data acquisition methods to evaluate contact pressure analysis during ankle motion on cadaveric subjects with a 50% PM osteotomy [23]. Interestingly, during ankle range of motion, Fitzpatrick detected no change in ankle stability or ankle motion patterns when compared to the intact foot (with intact medial and lateral constraints). There was also no increase in peak or mean contact stresses for the simulated PM fracture relative to the intact foot. Such lack in loss of contact area and thus pressure is attributed to a redistribution of load in the fractured specimens to a more anterior and medial position without talar subluxation. This would perhaps indirectly support the findings of Harper that found no posterior subluxation of the talus was observed when as much as 50% of the PM was removed [24]. Additionally, Raasch in 1992 looked at the extent of posterior translation of the talus after removal of as much as 40% of PM [25]. He compared isolated PM fractures with PM fractures combined with transection of the AITFL and fibula and noted that significant posterior translation of the talus was only seen with that of the latter. He therefore concluded that the fibula and AITFL are the primary restraint to posterior instability of the ankle. Fitzpatrick argues that this observed medial and anterior loading pattern creates a new force on previously underutilized cartilage following this redistribution of load that is responsible for the reported post-traumatic arthrosis in PM fracture patients. Whether such application of weight-bearing forces on naive cartilage would result in such an inflammatory process is however beyond the scope of this paper. Despite differing views on the structural etiology, all parties collectively agree that reduction on the PM fragments is necessary to prevent...
long-term detrimental effects to the ankle joint. De Vries work on the long-term (13 year f/u) results of 45 patients with ankle fractures with PM fragments showed that there was no statically significant correlation between fragment size and long-term outcome (based on AFSS, numeric pain score and Osteoarthritis score) [26]. Statistically significant worse outcomes were seen however with PM fracture dislocations compared to their non-dislocated counterparts. This perhaps further questions the speculations of Macko and Hartford but equally does not directly support the findings of Fitzpatrick.

Conclusion

It is impossible to assess the significance of any fracture without considering how such osseous destabilizations influence or is influenced by their surrounding soft tissue attachments. Much of the role of the PM fracture is therefore rooted not solely in its structural composition but also on its ligamentous effect. If one assumes, as discussed that PM fractures (beyond the most minimal) result in some form of PITFL insult, then in order to review PM fractures we are obligated to review the PITFL. The significance of the PITFL towards the preservation of syndesmotic integrity is well established, however this fact alone should not be seen as the accepted stance on the need for PM fragment fixation. Further exploration is demanded in order to derive the significance of the syndesmosis towards the functional outcome of the ankle joint. As discussed such correlations are less than clear, the work of Boden and Burns would suggest there is not a direct correlation between loss of syndesmotic integrity and diastasis when the deltoid ligament is intact. [13,14] In fact, it has been touted that sufficient syndesmosis insult to produce diastasis cannot even be realized without initial injury to the deltoid ligament [27]. Such opposition to Boden’s and Burn’s findings are often based on the argument that these cadaveric studies are not indicative of the true functional limb. Stark has actively opposed the work of Boden stating that the criteria for syndesmotic instability based on cadaveric studies are not representative of the clinical situation [28]. However much of Stark’s conflict with Boden is based on the perceived greater necessity of syndesmotic fixation with deltoid ligament incompetency (not just at the critical transition zone as refereed in Boden’s paper) and does no compare that with an intact deltoid ligament. Greater opposition is perhaps seen in Heim’s paper that found that 17/90 (19%) patients with a weber B type fracture continued to exhibit diastasis even after lateral and medial fixation [29]. Furthermore, greatest residual diastasis was seen with trimalleolar type fractures. Heim speculated that such instability is associated with an interosseous membrane rupture well above the level of the fibular fracture, a notion not entertained within Boden’s work. However one must also consider the role of the deep deltoid ligament and whether its competency in restored with such fixation of the medial malleolus [30]. Residual lack of deep deltoid integrity in this study may still dictate that Boden’s and Burn’s finding can not be negated.

The fact that limitations of diastasis through fixation have shown to be directly linked to improved functional outcome in patients following ankle fractures is not of debate. [11,31] However the current literature limits us in directly associating PM fractures with an unstable ankle joint when the deep deltoid ligament is intact or reattached. Clarity is perhaps obtained when we transition and consider the structural implications the PM fragment. There is general agreement that anatomical restoration of a normal ankle mortise is essential for the long-term function of the ankle. Thus the structural role the PM in maintaining tibiotalar congruency has also been explored. Although disparity exists between what influences such fractures have on the positioning and pressures on the talus, this is not followed within the perceived consequences of such instability. The need for anatomical reduction of the PM fracture within the realms of ankle congruency despite the lack of allocation of direct etiology is thus validated.

References


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