Is Ankle Post-traumatic Osteoarthritis Inevitable after Malleolar Fractures?

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ABSTRACT

The tibiotalar joint is one of the most prevalent intra-articular fracture sites in the human body, resulting in high rates of post-traumatic ankle osteoarthritis (PTOA). Studies have shown multifactorial causes for PTOA and highlight the importance of three determining factors for clinical outcomes after malleolar fracture: quality of joint reduction and fracture fixation, residual ligament instability, and initial damage to joint tissues—including chondral tissue, synovial tissue, and synovial fluid. This special article summarizes recent evidence of malleolar fractures treatment, with a main focus on important factors related to improve clinical outcomes in order to avoid post-traumatic ankle osteoarthritis (OA).

Keywords: Ankle joint, Arthritis, Malleolar fracture.

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INTRODUCTION

Tibiotalar injuries are frequently evaluated by orthopedic trauma surgeons. In the United States, ankle sprains and fractures are highly prevalent,¹ malleolar fractures represent 9–18% of all fractures in emergency services.²–⁴ The most prevalent etiological factor of ankle osteoarthritis (OA) is tibiotalar joint fracture, added to other lesions around the foot and ankle, post-traumatic etiology represents about 78% of causes of ankle OA (Fig. 1).⁵–⁷

Secondary ankle OA due to other causes, such as rheumatoid disease, represents 13% of etiology, it was also more common than the primary OA (9%).⁴,⁵

Ankle OA affects approximately 1% of the population, some authors estimate an incidence of 30 cases per 100,000 population and this represents a share of 2–4% of all patients with OA.⁵–⁹

Historically, the treatment for ankle fracture is anatomical reduction of the joint, initially with plaster and in 1984 Lane started to recommend surgery.¹⁰,¹¹ Even with the evolution in the principles of management of ankle fractures, the estimated risk of clinical outcome with post-traumatic ankle osteoarthritis (PTOA) after 20 years is around 40%, which is more frequent in Weber types B and C and fractures involving the posterior malleolus (PM).

The therapeutic options are limited for PTOA. Furthermore, the low comprehension of the molecular events involved in chondrocyte cell death limits the progress in this area of knowledge.⁶–⁹

This article show recent evidence of malleolar fractures treatment.

DETERMINING FACTORS

The three determining factors for clinical outcomes are:¹²,¹³

• Quality of joint reduction and fracture fixation.
• Residual ligament instability.
• Initial damage to joint tissues.

QUALITY OF JOINT REDUCTION AND FRACTURE FIXATION

Lateral Malleolar Fracture

The level of fracture by the Weber classification interferes with clinical follow-up outcome: 82% type Weber A, 83% type Weber B, 70% type Weber C.¹³

Open anatomic reduction and rigid internal fixation of unstable lateral malleolar fractures are indicated to avoid shortening, lateral displacement, and external rotation of the fibula,¹⁴ to maintain joint contact pressures at normal levels.
The fixation options include tubular lateral or posterolateral plates, non-locked or locked plates, lateral anatomic distal fibular plate, and distal fibula intramedullary nail (Fig. 2).13,15

**Medial Malleolar Fracture**

The medial malleolar fractures can be classified as horizontal (A—avulsion; B—between the apex and the plafond, and C—at the plafond) or vertical (type D).16,17

The role of clinical and biomechanical aspects of the medial malleolus and ligamentous structures has been well studied. Some authors have shown in isolated medial malleolar osteotomy a decrease in medial articular contact pressure of 27.8% following fixation when compared with non-fixation.18,19

Open anatomic reduction and lag screw fixation is indicated for horizontal medial malleolar fracture, on the other hand, the lag screw through buttress plate technique is recommended for vertical medial malleolar fracture. The new implant’s design minimizes pain related to plate and screws prominence (Fig. 3).16–19

**Posterior Malleolar Fracture**

Studies show significantly worst clinical outcomes after a surgical of ankle fractures involving PM comparing with patients without this lesion.20,21
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There is already a consensus in the literature to recommend MP reduction and fixation whenever it represents a joint instability factor (sagittal and coronal plane) and interferes with the anatomical reduction of the syndesmosis and its stability, regardless of the size of the bone fragment.

In the 1920s, Lounsbury, Metz, and Leveuf published the first PM fixation reports.

Currently, the Bartonícek et al.’s Classification System based on 3D CT scanning represents a useful tool in decision making.

Open reduction and internal fixation (ORIF) of PM fragment results in a decrease of up to 60% in trans-syndesmotic fixation needs as well as better quality of distal tibiofibular joint congruency compared with closed reduction and anteroposterior screw or fractures in untreated PM.

Posterior malleolar fracture reduction and fixation could be performed:
- Direct through posterolateral or posteromedial approaches and lag screws—small fragment or with plate butresses and lag screw (lag screw through the plate or outside the plate)—large fragment.
- Indirect approach is more difficult to evaluate the reduction and soft tissue interposition could be an issue, usually, the sagittal lag screws are inserted through separate incisions.

In the study of a specimen, PM fixation presented superior biomechanical joint stability (70%) compared with trans-syndesmotic fixation (40%).

Direct reduction and posterior to anterior fixation with screws or a plate + screw allow greater biomechanical joint stability when compared with indirect reduction and anterior to posterior fixation (Fig. 4).

**Residual Ligament Instability**

**Syndesmosis**

Syndesmosis injury can occur with almost any fracture pattern, approximately up to 23% of all ankle fractures.

All four ligaments that compound syndesmosis complex are:
- Anterior inferior tibiofibular ligament (AITFL).
- Interosseous membrane ligament (IOM).
- Transverse tibiofibular ligament (TTFL).
- Posterior inferior tibiofibular ligament (PITFL).

After bone fixation, the first step is to check for residual syndesmotic instability in the absence of evident instability.

Undiagnosed and not well reduced syndesmotic instability represent determining factors for a poor clinical outcome.

There are basically three intraoperative reliable dynamic intraoperative tests described in the literature:
- Worldwide used as a clinical test for deltoid ligament lesion and medial ankle instability, the external rotation stress test, remains not standardized in the diagnosis of isolated distal tibiofibular.
- Some authors have shown that measurement of sagittal instability allowed by the Cotton test or the Hook test is more sensitive than for coronal instability. This test has high specificity but low sensitivity.

The simple Tap Test shows good sensitivity, specificity, and accuracy in the diagnosis of coronal syndesmotic instability.

**Radiographic Findings**

There are three (Fig. 5) consensus radiographic measures used to evaluate residual syndesmosis instability intraoperatively after bone fixation using conventional fluoroscopy anteroposterior and mortise view:
- The normal tibiofibular clear space (TFCS) is <6 mm.
- The normal tibiofibular overlap (TFO) is <2 mm.
- The medial clear space (MCS) is <6 mm.

These radiographic parameters show 47% sensitivity and 100% specificity compared with magnetic resonance image.

It is recommended that, after bone fixation, a syndesmosis diastasis of >2 mm be reduced and fixed.

**Rigid Fixation**

There is no clinically significant difference for the treatment of syndesmosis instability comparing:
- One vs two syndesmotic screws.
- Tricortical vs quadricortical screws.
- Trans-syndesmotic vs supra-syndesmotic screws.
- Metal screws vs bioabsorbable screws.

Biomechanical studies comparing 4.5 mm screw vs 3.5 mm screw show divergent results.

A small plate connecting two syndesmotic screws increases mechanical strength in poor-quality bone.

The lowest tension for screws positioned between 3 cm and 40 cm above the tibiotalar joint line was advocated by McBryde et al.
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However, Seitz et al. showed that a screw positioned 2 cm from this joint line resulted in less enlargement of the syndesmosis.  

Flexible Fixation
The suture button fixation provides stability and allows distal fibula-tibial joint movement close to the physiological one. This option is equivalent to a 3.5 mm tricortical screw or a 4.5 mm quadrincortical screw. However, the functional results in the short-term do not show relevant differences.

Flexible fixation shows less indication for implant removal. While comparing rigid and flexible fixation the only variable that had a significant influence on the functional outcome was the anatomical reduction of the distal fibula in the tibial notch.

Deltoid Ligament
The treatment of deltoid injuries in malleolar fractures remains without consensus.

The role of the deltoid ligament is fundamental in normal ankle biomechanics, its superficial component participates in the hindfoot eversion restriction, and its deep component participates in the talus external rotation restriction.

How to Determine when Surgical Treatment is Required for Deltoid Ligament Injury Related to Medial Malleolar Fracture?

Physical Exam
Some authors observed that the classic findings on clinical examination showed a poor correlation with deltoid complex insufficiency.

Simple Radiography
The measurement of the MCS outside the normal range on static radiography (without stress) mortise view is a method used to identify residual medial instability. However, the normal MCS does not exclude residual medial instability.

Stress or Gravity Stress Radiography
Park et al. and Michelson et al. performed similar studies regarding the radiographic findings of gravitational stress, the authors observed that the medial free space of 5 mm is a good parameter for damage to the deep component of the deltoid complex.

Magnetic Resonance Image
In the treatment of malleolar fractures, magnetic resonance still does not show a standardized role in therapeutic decision-making regarding the deltoid complex.

Nortunen et al. observed that all volunteers with isolated fractures of the fibula by SER mechanisms showed positive magnetic resonance imaging (MRI) findings for ligament involvement of the deltoid. On the other hand, they observed high variation in MCS in volunteers with similar MRI findings, as well as low interobserver reliability for MRI compared with the stress test. Thus, they do not recommend MRI for therapeutic decision-making.

Warner et al. showed that magnetic resonance image allows correct diagnosis of ligament injuries associated with ankle fracture in 94% of patients.

Treatment
Salamah et al. and Dabash et al. concluded that deltoid ligament repair in Weber type B and C malleolar fractures confers:

• Better joint reduction.
• Lower pain score.
• Similar complication rates.
• No difference in functional outcome.

Initial Damage to Joint Tissues
Immediately after trauma, articular chondrocytes are influenced by various inflammatory chemokines, including TNF-α, IL-1β, and metalloproteinases (Fig. 6).

In these contexts, minimizing the acute injury and its progression at the tissue and molecular level should be targeted along with the macroscopic alterations induced by the trauma to prevent the evolution to post-traumatic OA.

Dingle et al. first studied in vivo the detrimental effects of cytokines on articular tissues. In an earlier study, the same author also stated that synovium affected the cartilage in two ways:

• Direct action on the matrix, possibly by secreted proteinases, like collagenase.
Indirect action mediated through the living chondrocytes which produce acidic proteinases of the lysosomal system.

Adams et al. observed an elevation of pro-inflammatory cytokines in synovial fluid in a fractured ankle compared to an uninjured ankle at different post-injury times.80–82 Godoy-Santos et al. analyzing the cellularity and synovial profile in patients with acute ankle fractures, the authors also observed high concentrations of cytokines in the synovial fluid, in addition to the accumulation of collagen and proteoglycans in the synovial tissue.83

A recent analysis by Leimer et al. revealed a significant elevation of 19 amino acids metabolites in ankle fracture in comparison to the contralateral sides.84

These results show that pathological changes happen simultaneously to different articular tissues following ankle fractures.

Traumatic aggression to synovial tissue results in the release of pro-inflammatory substances, which reach the chondral tissue through the synovial fluid, causing high cell death of chondrocytes.85

Another pathway leading to damage to synovial tissue and cartilage involves complement activation.86

Finally, activation of both pathways in joint tissues and fluids leads to increased genetic modulation for the production of pro-inflammatory substances.87

Detecting Early Osteoarthritic Changes in the Ankle Joint

Biomarkers

Inflammation biomarkers may be the first identifier of PTOA. Although studies show that these substances can be identified in blood, urine, and synovial fluid, there is no consensus on the ideal marker.88

IL-2, IL-6, IL-8, IL-10, IL-17, MMP-1, MMP-2, MMP-3, and TNF-α are biomarkers under study.80–84 However, it is not a routine yet.

Imaging

Magnetic resonance imaging is an adequate option for the early detection of OA. In this sense, the new cartilage mapping acquisition
allows us to visualize alterations in the microstructure, in the composition of the extracellular matrix, and in the chondrocytes. T1p allows proteoglycan content assessment and T2 relaxation allows collagen organization assessment.\textsuperscript{89,90}

T2 mapping shows low sensitivity for deep chondral tissue layers.\textsuperscript{91,92}

For the analysis of chondral damage extension and its biological activity, single-photon emission computed tomography (SPECT-CT) shows high interobserver and intraobserver reliability compared with a conventional CT.\textsuperscript{93,94}

**EARLY TREATMENT**

Joint washing at the time of surgery to remove a hematoma, small bone fragments, and inflammatory substances involved in ankle joint degeneration seems to contribute to PTOA prevention.\textsuperscript{80 – 84}

Some medications can modulate chondrocyte metabolism; on the one hand, promoting its proliferation and on the other, inhibiting cell death, e.g., lovastatin-induced proliferation and reduced apoptosis in an experimental study in rabbits;\textsuperscript{95} celecoxib and indomethacin reduce trauma-induced cell death in a study with impacted human cartilage.\textsuperscript{96,97}

Although hyaluronan (HA) has a proven role in chondrocyte apoptosis in already established OA,\textsuperscript{98} there is no evidence of this action in acute fractures. In an in vitro study, the platelet-rich plasma (PRP) promoted cell proliferation and decreased cell death.\textsuperscript{99,100}

Inflammatory modulators are potential therapeutic tools against intra-articular tissue degeneration. However, we have not yet identified solid evidence in the literature.

Randomized clinical trials and experimental studies of PTOA are needed to advance this area of knowledge.\textsuperscript{101–103}

**CONCLUSION**

Post-traumatic ankle OA is avoidable and minimizable after malleolar fractures.

The key factors to prevent PTOA are:

- Anatomically reducing all three joints—tibiotalar, tibiofibular, and fibulotalar.
- Properly fixing fractures.
- Correctly restoring ligament stability.

The strategies to modulate initial damage to joint tissues are still under investigation.

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