Posttraumatic ankle osteoarthritis: How initial cartilage lesions, the deltoid ligament and hindfoot alignment affect the outcome of operatively treated ankle fractures

Stufkens, Sjoerd

Citation for published version (APA):
Stufkens, S. A. S. (2014). Posttraumatic ankle osteoarthritis: How initial cartilage lesions, the deltoid ligament and hindfoot alignment affect the outcome of operatively treated ankle fractures
CHAPTER 3

THE ROLE OF ACCOMPANYING INITIAL CARTILAGE LESIONS AFTER ANKLE FRACTURES

Published as:

CHAPTER 3 - CARTILAGE LESIONS

Introduction

The ankle is a very congruent joint and highly resistant to primary degenerative disease. Unlike osteoarthritis in the hip or knee, osteoarthritis in the ankle joint is predominantly the result of a traumatic event. Posttraumatic osteoarthritis accounts for at least 12% of the combined prevalence of hip, knee, and ankle arthritis1, whereas between 70% and 80% of the cases of ankle arthritis alone are of posttraumatic origin2,3.

Malunion is probably the most important cause of osteoarthritis after an ankle fracture4,5. Other factors that are considered to play a role are suboptimal alignment in the coronal6-10 or sagittal11 plane, ligament damage leading to ligamentous instability12,13, and the development of arthrofibrosis14,15. Relevant factors affecting the early outcome after ankle fracture are sex16, age17, body mass index18, and diabetes19,20, but the roles of these variables in the long-term outcome are unclear. Surprisingly little is known about the relationship between initial cartilage damage and the development of osteoarthritis of the ankle. Nevertheless, some authors consider these lesions to influence the development of posttraumatic osteoarthritis21,22. Arthroscopy performed directly after an ankle fracture often shows more damage to the cartilage than was expected on the basis of plain radiographs. Cartilage lesions have been found in 79% to 90% of ankles examined arthroscopically after an ankle fracture14,23. Although the role of arthroscopy in the acute treatment of ankle fractures has not been established24, the modality has been shown to be valuable for obtaining detailed knowledge about the fractured ankle23,25 and in the treatment of symptomatic osteophytes after ankle fracture26.

We hypothesized that the more extensive the initial cartilage damage, the higher the chance of osteoarthritis developing later. Although we know that the talus is the most common location of cartilage damage caused by an ankle fracture14,21,23,25, we do not know whether the location of the cartilage injury influences the development of posttraumatic osteoarthritis. To the best of our knowledge, there have been no previous studies correlating the location and extent of cartilage lesions following ankle fractures with the long-term outcome. In this follow-up study of a consecutive series of patients, we examined the correlation between the initial cartilage damage seen at arthroscopy performed directly after a displaced ankle fracture23 and the clinical and radiographic long-term results associated with that fracture.
Materials and Methods

Study Design and Patient Demographics

We performed a long-term follow-up study of 109 patients in whom an ankle fracture had been treated operatively according to the AO principles between June 1993 and November 1997 at the Kantonsspital Liestal and the Kantonsspital St. Gallen, both in Switzerland. These patients were recruited from a consecutive cohort of 288 patients for whom arthroscopy had been performed prior to the surgical management of the ankle fracture. Fifty-seven (19.8%) of these patients had died, and an attempt was made to contact the remaining 231 patients. Thirty-seven (16.0%) of the 231 patients had emigrated, seventy-one (30.7%) refused to participate in the study, fourteen (6.1%) could not be located, and 109 (47.2%) were recruited into the study. The two main reasons why patients declined our invitation to participate in the follow-up study were the requirement for additional radiographic evaluation and health and age-related problems that limited the patients’ ability to travel to our hospital. We identified no differences in terms of the demographic data or the original arthroscopic findings between the study subjects and those who did not participate.

The mean duration of follow-up of the 109 patients was 12.9 years (range, 11.3 to 14.8 years). Sixty-one patients (56%) were male, and forty-eight (44%) were female. The mean age at the time of injury was 37.4 years (range, sixteen to seventy-one years) for the male patients and 50.2 years (range, twenty to seventy-six years) for the female patients. The right ankle was involved in fifty-three (49%) of the cases. Most (40%) of the injuries had been the result of a sports accident. The other fractures were sustained in a traffic collision (18%), at home (17%), at work (8%), or for another reason (16%). Approval for this long-term outcome evaluation was provided by our institutional review board and the cantonal ethical committee. We included all patients who were available for follow-up and provided written informed consent. None of the patients who were available had a systemic inflammatory disease or were unable to complete questionnaires, both of which were exclusion criteria. We did exclude one patient who had had a substantial ankle injury before the index ankle fracture and two patients who had had a subop-
timal reduction of the fracture, defined as shortening of the fibula or lateral
displacement of the fibula identified on reevaluation of the postoperative
radiographs.

Main Variables

Arthroscopy had been carried out with the patient under general or regional
anesthesia and had been followed by open reduction and internal fixation. The
patients were placed supine on the table with the knee flexed in a knee-holder,
which allowed the experienced arthroscopists to perform the arthroscopy
without a distraction device. Before creation of the portals by blunt dissec-
tion, the joint was inflated with saline solution. A 4.5-mm, 30° arthroscope
was used in a standard central anterior portal. After aspiration of the saline
solution, the examination was performed in a CO2-filled joint. When neces-
sary, additional anteromedial and anterolateral portals were created for the
insertion of instruments. All intra-articular lesions were extensively docu-
dmented. Inspection and probing were used to grade the cartilage lesions.
Grade I represented intact cartilage; grade II represented superficial bruises,
fissuring, or degeneration of <50% of the thickness of the cartilage; grade-III
lesions involved deeper changes, involving >50% of the cartilage thickness;
and grade IV indicated that subchondral bone was visible (Fig. 1). There were
ten possible locations of the cartilage lesions: the medial malleolus; the lateral
malleolus; the anterior, medial, lateral, and posterior aspects of the tibia; and
the anterior, medial, lateral, and posterior aspects of the talus.

Outcome Parameters

The clinical results were evaluated by an independent orthopaedic foot and
ankle surgeon without knowledge of the injury or the intra-articular lesions
that had been identified arthroscopically. The American Orthopaedic Foot
and Ankle Society (AOFAS) hindfoot score was used to quantify the long-
term clinical outcome. Anteroposterior and lateral radiographs of the ankle
were evaluated by an experienced radiologist, without knowledge of the clini-
cal results, who used a modification of the Kannus arthritis score to determine
the severity of the osteoarthritis on a 100-point scale. This score is based on
several aspects of arthritis, such as the amount of sclerosis visible, formation of osteophytes, calcification of the ligaments, joint space narrowing, and cyst formation\textsuperscript{28,29}.

\textit{Statistical Methods}

The main analysis focused on the correlation between the depth and location of cartilage damage and the development of clinical or radiographic signs of osteoarthritis. To detect the influence of various variables on the two main clinical and radiographic outcome parameters (the AOFAS hindfoot score and the modified Kannus arthritis score), variables were dichotomized at $\leq$90 points (indicating signs of joint degeneration) and $>$90 points (indicating an optimal long-term outcome). First, patients were divided into two groups: those with an initial cartilage lesion and those without such a lesion. Second, the damage levels were reduced to two categories in order to investigate the role of the depth of the lesions in several locations within the joint. The first category included intact cartilage and lesions up to 50\% in depth. The second category consisted of lesions exceeding 50\% in depth, up to the subchondral bone. Multiple logistic regression analysis was performed to detect the in-
fluence of each damage variable separately on the dichotomized AOFAS and Kannus scores. Results are presented as odds ratios with corresponding 95% confidence intervals and p values. An odds ratio of greater than one indicates that the odds of disease developing (clinical and radiographic scores of $\geq 90$ points) is greater in the exposed group (patients with an intra-articular lesion) than in the unexposed group (patients who do not have such a lesion). To adjust for sex, age, and body mass index, these variables were also included in the regression model. A p value of $<0.05$ was considered significant. Because this study was exploratory, there was no adjustment for multiple comparisons.

Table 1: Frequency of initial cartilage lesions seen in the ankle joint

<table>
<thead>
<tr>
<th></th>
<th>Lesions on the Talus</th>
<th>Lesions on the Tibia</th>
<th>Lesions on the Malleoli</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Anterior</td>
<td>Medial</td>
<td>Lateral</td>
</tr>
<tr>
<td>Intact</td>
<td>76.1%</td>
<td>66.1%</td>
<td>78.9%</td>
</tr>
<tr>
<td>&lt;50% depth</td>
<td>17.0%</td>
<td>20.2%</td>
<td>11.9%</td>
</tr>
<tr>
<td>&gt;50% depth</td>
<td>5.0%</td>
<td>11.9%</td>
<td>7.3%</td>
</tr>
<tr>
<td>Subchondral bone</td>
<td>3.0%</td>
<td>1.8%</td>
<td>1.8%</td>
</tr>
<tr>
<td></td>
<td>100.0%</td>
<td>100.0%</td>
<td>100.0%</td>
</tr>
</tbody>
</table>
Results

Descriptive Statistics

According to the Weber classification system\textsuperscript{30}, 15% of the fractures were type A, 68% were type B, and 17% were type C. The AO 44-B1 fracture\textsuperscript{31} was the most common, accounting for 27% of all fractures, followed by the 44-B3 type, accounting for 21%. At arthroscopy, a cartilage lesion was found on the talus in 65% of the patients, on the tibia in 50%, and on the fibula in 39%. No cartilage damage was seen in 19% of the patients. Only the talus was involved in 17% of the patients; only the tibia, in 8%; and only the fibula, in 6%. Both the talus and the tibia had damaged cartilage in 17% of the patients; both the talus and the fibula, in 7%; and both the tibia and the fibula, in 5%. All three surfaces were affected in 21% of the patients. In total, 81% of the patients had some form of initial cartilage damage in the ankle joint directly after the ankle fracture (Table I, Figs. 2 and 3). At the time of long-term follow-up, clinical signs of osteoarthritis as defined by an AOFAS score of =90 points were seen in 39% (forty-three) of the 109 patients, whereas radiographic signs of osteoarthritis as defined by a modified Kannus score of =90 points were seen in
43% (forty-seven). The mean AOFAS score was 88.9 points (range, 18 to 100 points), and the mean radiographic score was 89.8 points (range, 54 to 100 points). The data for both main outcome parameters were skewed toward the maximum score.

**Statistical Analysis**

As mentioned, we first divided the patients into two groups: those with an initial cartilage lesion and those without such a lesion. Cartilage damage in the ankle joint was associated with an AOFAS score of ≥90 points (odds ratio = 5.0 [95% confidence interval = 1.3 to 20.1]; p = 0.02) and with a radiographic score of ≥90 points (odds ratio = 3.4 [95% confidence interval = 1.0 to 11.2]; p = 0.04). Then we assessed the cartilage damage at each separate surface.

<table>
<thead>
<tr>
<th>Cartilage damage predictor of AOFAS hindfoot score of &lt;90 points when:</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present in ankle joint</td>
<td>5.0</td>
<td>1.25-20.12</td>
<td>0.02*</td>
</tr>
<tr>
<td>Present on tibia</td>
<td>2.7</td>
<td>1.14-6.44</td>
<td>0.02*</td>
</tr>
<tr>
<td>Present on fibula</td>
<td>1.8</td>
<td>0.78-4.14</td>
<td>0.17</td>
</tr>
<tr>
<td>Present on talus</td>
<td>3.7</td>
<td>1.39-9.97</td>
<td>&lt;0.01†</td>
</tr>
<tr>
<td>&gt;50% of depth on posterior aspect of tibial plafond</td>
<td>0.6</td>
<td>0.15-2.03</td>
<td>0.38</td>
</tr>
<tr>
<td>&gt;50% of depth on medial malleolus</td>
<td>5.2</td>
<td>1.85-14.57</td>
<td>&lt;0.01†</td>
</tr>
<tr>
<td>&gt;50% of depth on anterior aspect of talus</td>
<td>12.3</td>
<td>1.41-108.0</td>
<td>0.02*</td>
</tr>
<tr>
<td>&gt;50% of depth on lateral aspect of talus</td>
<td>5.4</td>
<td>1.24-23.48</td>
<td>0.02*</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cartilage damage predictor of Kannus radiographic score of &lt;90 points when:</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present in ankle joint</td>
<td>3.4</td>
<td>1.04-11.17</td>
<td>0.04*</td>
</tr>
<tr>
<td>Present on tibia</td>
<td>3.3</td>
<td>1.42-7.61</td>
<td>&lt;0.01†</td>
</tr>
<tr>
<td>Present on fibula</td>
<td>0.8</td>
<td>0.34-1.76</td>
<td>0.54</td>
</tr>
<tr>
<td>Present on talus</td>
<td>2.4</td>
<td>0.99-5.78</td>
<td>0.05*</td>
</tr>
<tr>
<td>&gt;50% of depth on posterior aspect of tibial plafond</td>
<td>4.7</td>
<td>1.18-18.79</td>
<td>0.03*</td>
</tr>
<tr>
<td>&gt;50% of depth on medial malleolus</td>
<td>2.9</td>
<td>1.08-7.90</td>
<td>0.03*</td>
</tr>
<tr>
<td>&gt;50% of depth on anterior aspect of talus</td>
<td>4.7</td>
<td>0.88-25.63</td>
<td>0.07</td>
</tr>
<tr>
<td>&gt;50% of depth on lateral aspect of talus</td>
<td>1.6</td>
<td>0.42-6.30</td>
<td>0.48</td>
</tr>
</tbody>
</table>

*Significant at p < 0.05.
†Highly significant at p < 0.01.
Cartilage damage on the tibia, including the medial malleolus, was associated with an AOFAS score of ≥90 points (odds ratio = 2.7 [95% confidence interval = 1.1 to 6.4]; p = 0.02) as well as with a radiographic score of ≥90 points (odds ratio = 3.3 [95% confidence interval = 1.4 to 7.6]; p < 0.01). Cartilage damage on the talus was associated with an AOFAS score of ≥90 points (odds ratio = 3.7 [95% confidence interval = 1.4 to 10.0]; p < 0.01) as well as with a radiographic score of ≥90 points (odds ratio = 2.4 [95% confidence interval = 1.0 to 5.8]; p = 0.05). Lesions of the fibula were not found to be associated with a significant increase in the risk of the development of posttraumatic osteoarthritis.

When we assessed the depth of the lesions at different locations, we found significant relationships between the long-term clinical outcome and a lesion exceeding 50% of the cartilage depth on the anterior aspect of the talus (odds ratio = 12.3 [95% confidence interval = 1.4 to 108.0]; p = 0.02) and such a lesion on the lateral aspect of the talus (odds ratio = 5.4 [95% confidence...
interval = 1.2 to 23.5]; p = 0.02). Damage exceeding 50% of the cartilage depth on the medial malleolus alone was associated with both clinical signs of osteoarthritis (odds ratio = 5.2 [95% confidence interval = 1.9 to 14.6]; p < 0.01) and with radiographic signs of osteoarthritis (odds ratio = 2.9 [95% confidence interval = 1.1 to 7.9]; p = 0.03). The deep lesions on the tibial plafond had little predictive value, with only the lesions on the posterior aspect of the tibial plafond having an association with a radiographic score of =90 points (odds ratio = 4.7 [95% confidence interval = 1.2 to 18.8]; p = 0.03) (Table II, Figs. 4 and 5).

Discussion

It is generally accepted that open reduction and internal fixation is the best treatment for unstable or displaced ankle fractures. This is reflected by our results, which showed high mean overall clinical and radiographic outcome scores at a mean of thirteen years after ankle fracture. In general, posttraumatic arthritis is reported to occur in 14% to 50% of all fractured ankles. Few authors have suggested that the need for a perfect reduction was not supported by their data. Bauer et al. studied 143 patients thirty years after the treatment of an ankle fracture with closed methods; 82% were free of arthritis, and 83% were free of symptoms. This finding is in contrast with that in the series reported by Beris et al., in which osteoarthritis developed in 78% of twenty-three patients who had had a poor reduction. Most authors have agreed that accurate reduction leads to the best results. While malunion is probably the most important cause of osteoarthritis after ankle fracture, other factors that may play a role are varus or valgus alignment of the distal part of the tibia, ligament damage leading to ligamentous instability, and the development of arthrofibrosis.

Recent studies have suggested that abnormal loading alone does not explain the increased prevalence of osteoarthritis in fractured ankles. In a large series of 345 ankle fractures treated operatively, Lindsjö found that the rate of excellent or good results was 81% for the displaced fractures but only 38% for the impacted fractures. He suggested that articular cartilage damage at the time of the fracture could have an influence on the overall outcome.
opinion was shared by Marsh et al., who considered the extent of the initial cartilage injury to be the primary determinant of joint degeneration after trauma\(^{22}\). Also, Lantz et al. mentioned that unrecognized injuries to the cartilaginous surfaces of the tibiotalar joint could be the origin of osteoarthritis after anatomical reduction and stabilization of ankle fractures\(^{21}\). In their series of sixty-three surgically treated patients, those authors found many talar dome chondral injuries, and the overall results were worse in patients with such lesions. Some studies do not support this hypothesis. Ono et al. reported cartilage damage in only 20% of 105 patients who had undergone open reduction and internal fixation of a malleolar fracture with arthroscopic confirmation of an anatomical reduction\(^{42}\). Those authors concluded that cartilage injury could not be of much importance. However, we found no studies in the literature in which a long-term evaluation of clinical and radiographic outcome parameters was performed in a group of patients who had had arthroscopy before fracture treatment. In our series, we correlated the outcome with the articular cartilage lesions that had been seen initially in order to determine the role of those lesions in the development of posttraumatic osteoarthritis.

Our hypothesis was that the more extensive the initial cartilage damage, the higher the chance that osteoarthritis would develop later. Regarding the location of the cartilage damage, we expected worse outcomes when lesions had been seen on the talus, as other authors had reported concern about these lesions\(^{21,43}\). Our results showed that cartilage damage at the time of an ankle fracture does play an important role in the development of posttraumatic osteoarthritis. When cartilage damage is present in the joint, the odds of the patient having a suboptimal long-term clinical outcome (an AOFAS score of <90 points) is five to one, whereas the chance of showing radiographic signs of joint degeneration (a Kannus arthritis score of <90 points) is three and a half to one. We found no correlation between the number of lesions and the long-term outcome; however, we found specific locations in the joint to be important factors. The lesions on the anterior and lateral aspects of the talus (Fig. 2) and those on the medial malleolus (Fig. 3) were shown to significantly increase the risk of posttraumatic osteoarthritis. Lesions on the posterior aspect of the tibial plafond also led to an unfavorable radiographic score. In addition, there was a correlation between the depth of the lesions and the long-term outcome. Deep lesions extending into the subchondral bone are
thought to heal better than lesions that do not extend into the subchondral bone\textsuperscript{44,45}. The deep lesions cause hemorrhage and fibrin-clot formation and activate an inflammatory response. In this study, however, the deeper lesions correlated with worse long-term outcomes. This could be the result of the location of the lesions. The deepest lesions were found in the medial and lateral regions of the talus, where the cartilage is, on the average, thicker. Mean cartilage thickness ranges from 0.91 mm in the fibula, to 1.21 mm in the tibia, to 1.34 mm in the talus\textsuperscript{46}, and the thickest cartilage is found over the talar shoulders, where osteochondritis dissecans lesions commonly occur\textsuperscript{47-49}. Over time, the fibrocartilage that fills a deep defect often begins to show evidence of depletion of matrix proteoglycans, fragmentation, and fibrillation. The fibrocartilage then fragments and disintegrates\textsuperscript{22}. The defects that result from ankle fractures in locations where the cartilage is thickest may therefore be too deep to heal adequately and remain filled with fibrocartilage. This would mean that the largest and deepest defects would recur over a long period of time. This could also explain why it sometimes takes several decades for posttraumatic osteoarthritis to develop in the ankle joint. We did not perform arthroscopy at the time of follow-up, and hence we cannot confirm whether lesions recurred.

Other limitations of our study include the loss to follow-up. The data were gathered prospectively, but no protocol was installed to track patients at specific intervals. Therefore, the >80% recruitment criterion for a well-documented follow-up study\textsuperscript{50} was not met. The initial population in which arthroscopy was performed consisted of 288 consecutive patients with an ankle fracture. We were able to contact and recruit only 109 of them. The two main reasons for patients not wanting to participate in the follow-up study were the requirement for additional radiographic studies and health-related problems that limited their ability to travel. No one mentioned an ankle-related problem as the reason for refusing to return for our long-term evaluation, and the demographics and arthroscopic findings of the study group were similar to those of the patients who were lost to follow-up. The mean follow-up time of nearly thirteen years could also be an explanation for the reduced number of patients who were available to us.

Our findings show that initial cartilage damage seen arthroscopically after an ankle fracture is an independent predictor of posttraumatic osteoarthritis. Lesions on the talus and tibia are associated with negative long-
term results, whereas lesions on the fibula do not correlate with a worse long-term outcome. Specifically, deep lesions on the anterior and lateral aspects of the talus and on the medial malleolus correlated with an unfavorable clinical outcome. Newer imaging techniques, such as diffraction-enhanced x-ray imaging\textsuperscript{51}, may make it possible to identify these articular cartilage lesions without resorting to arthroscopy. The location and severity of osteochondral lesions are important factors in the prognosis of ankle fractures, and it is important to identify these lesions. Additional biochemical and clinical research is needed to develop an effective treatment plan for these lesions in order to improve the long-term outcome after ankle fractures.

References


9. Stamatis ED; Cooper PS; Myerson MS. Supramalleolar osteotomy for the treatment of distal tibial angular deformities and arthritis of the ankle joint. Foot Ankle Int. 2003;24:754-64.


11. Tarr RR; Resnick CT; Wagner KS; Sarmiento A. Changes in tibiotalar joint contact areas following experimentally induced tibial angular deformities. Clin Orthop Relat Res. 1985;199:72-80.


15. Utsugi K; Sakai H; Hiraoka H; Yashiki M; Mogi H. Intra-articular fibrous tissue formation following ankle fracture: the significance of arthroscopic debridement of fibrous tissue. Arthroscopy. 2007;23:89-93.


22. Marsh JL; Buckwalter J; Gelberman R; Dirschl D; Olson S; Brown T; Llinias A. Articular fractures: does an anatomic reduction really change the result? J Bone Joint Surg Am. 2002;84:1259-71.


27. Kitaoka HB; Alexander IJ; Adelaar RS; Nunley JA; Myerson MS; Sanders M. Clinical rating systems for the ankle-hindfoot, midfoot, hallux, and lesser toes. Foot Ankle Int. 1994;15:349-53.


CHAPTER 3 - CARTILAGE LESIONS


41. Bauer M; Bengner U; Johnell O; Redlund-Johnell I. Supination-eversion fractures of the ankle joint: changes in incidence over 30 years. Foot Ankle. 1987;8:26-8.


46. Millington SA; Li B; Tang J; Trattnig S; Crandall JR; Hurwitz SR; Acton ST. Quantitative and topographical evaluation of ankle articular cartilage using high resolution MRI. J Orthop Res. 2007;25:143-51.


