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Lateral ligament injury of the ankle and associated articular cartilage degeneration in the talocrural joint: anatomic study using elderly cadavers

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Abstract We investigated degenerative changes (DCs) of the articular cartilage using 33 cadaveric talocrural joints with anterior talofibular ligament injuries. The control group (93 normal ankles) were age- and sex-matched with the injury group. The injured ligament carried unusual wavy collagen bundles with hyalinization (or elongation) or rupture with scar. In both groups, we found talar-side dominance of DCs, multifocus occurrence, and a high incidence of mirror-image DCs in the medial parts of the joint. However, the mirror-image lesion was multiple or large (or both) in the injury group in contrast to its restricted nature in the control. Thus, the averaged numbers of affected areas or sectors in the injury group were almost twice as large as in the controls. A significantly high incidence of DCs, especially of the mirror-image lesion, was found in the lateral malleolar facet in the injury group. Although initial DC lesions might be at a medial site in the joint, as hypothesized in the control group, the lateral ligament injury seemed to increase the dominance of DCs on the lateral site. Consequently, early evaluation and treatment for lateral ligament insufficiency is more necessary in elderly patients than in younger patients to avoid widespread development of osteoarthritis.

Key words Lateral ligament injury · Anterior talofibular ligament · Osteoarthritis · Cartilage degeneration · Ankle joint

Introduction

Rupture or sprain of the lateral ligaments of the ankle is a common injury. According to Broström[1] and Taga et al.,[11] anterior talofibular ligament (ATFL) rupture is most frequently followed by a combined injury of the ATFL and the calcaneofibular ligament (CFL); it is rarely followed by an isolated CFL or a posterior talofibular ligament injury. Although adequate treatments were applied, 10%–30% of those injuries developed into a chronic problem, causing instability of the talocrural joint.[2–4] Most surgeons seem to believe that joint looseness and abnormal kinematics with ligament insufficiency are strongly connected to the occurrence of degenerative changes (DCs) of the articular cartilage, ultimately resulting in severe osteoarthritis.[4,11] Harrington[4] reported that 10 years after lateral ligament insufficiency 77% of the patients investigated had osteoarthritis. His study had only a limited sample for exploring whether DC progress was secondary to lateral ligament insufficiency. He described in detail, however, the long natural course before the onset of pain.

Previously, we described in detail DCs of the articular cartilage with aging in the talocrural and talocalcaneal joints.[5] In that study, we revealed that several types of DCs occurred in the talocalcaneal joint: (1) talar-side dominance; (2) medial malleolar facet dominance; (3) multifocus occurrence; (4) lower-than-expected incidence of mirror-image lesions except for those in the medial malleolar facet; and (5) the medial malleolar facet as the most frequent site of DCs. Although it may be an unfamiliar term, the mirror-image lesion seems to correspond to relatively advanced DCs identifiable on routine radiological examinations.[9,12]

Are these rules changed or modified when a lateral ligament injury is associated? Previously, Tsukahara[13] incidentally found 2 ATFL injury cases (a thin ligament with a scar) among 82 cadaveric ankles in his study of DCs. Notably, during our previous study,[5] we found the ligament injuries to be more frequent than did Tsukahara. Consequently, in the present study, we aimed to find lateral ligament injuries in donated cadavers and characterize DCs in the talocrural joint when it was associated with these injuries to provide better understanding of a suggested causal relation between the ligament insufficiency and DCs.

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Materials and methods

Materials

Altogether, 128 ankles (69 right, 59 left) were obtained from 108 postmortem-treated Japanese cadavers (64 men, 44 women) that had been donated for medical education and research at Sapporo Medical University. The age of the 108 cadavers at death ranged from 43 to 97 years (mean 78.8 years). These cadavers had been fixed postmortem with an arterial injection of 10 L of formalin solution (10% v/v in water). The past history of trauma and the clinical symptoms of the ankle joints were unknown. Among these 128 ankles from 108 cadavers, we found 33 ankles (20 right, 13 left) from 27 cadavers (13 men, 14 women) with ATFL injuries (Table 1). The incidence was 25.8% among the 128 ankles examined. In short, according to macroscopic viewpoints during dissection the injured ankle was discriminated from the normal ankle. For a comparison, we prepared a control group (93 ankles from 63 cadavers) whose lateral ligaments were not injured and in which the distributions of age and sex corresponded to those of the injury group. These 93 ankles were chosen from another population used for a previous study[5] as well as from the residual 95 ankles after excluding the 33 injured ankles from the original population.

Methods

The ATFL and CFL were dissected carefully from the lateral aspect of the ankle. The morphology of the ligaments, including the injured area, was classified into three groups; normal, elongation, and rupture. At the beginning of this study, all candidates with the ligament injury diagnosed based on macroscopic observations were, after decalcification with Plank-Rychlo solution ($AlCl_3 \cdot 6H_2O$ 7.0%, w/v; HCl 3.6%, v/v; and HCOOH 4.6%, v/v; in water) (Wako, Tokyo, Japan), processed for paraffin-embedded histology with hematoxylin and eosin (H&E) staining. Thus, we tried to define histologically the criteria of the three groups (i.e., normal, elongation, rupture) (Fig. 1). Although we could not always clearly identify the category "elongation" histologically, after several attempts it became easy to identify specific morphologies corresponding to the injuries macroscopically, rather than histologically. A normal ligament was clearly separated from surrounding loose connective tissues, and it was relatively thick and tough with regularly arranged fiber bundles, whereas the injured ligament was usually thin and soft, with unclear margins. Clear distinction was difficult because connective tissue around the ligament tended to display "swelling." The rupture that made a mass of the scar tissue or ligamentous fiber bundles was completely absent. With the help of the associated histological findings in these cases that were difficult to distinguish, we came to trust the macroscopic identification. Therefore, in the present study the ligament injury was usually not identified by the ligament itself but by the surrounding tissue. This might be one of the study's limitations.

We hypothesize the "typical" histology of ATFL *elongation* to be as follows. The ligament was composed of dispersed collagen fiber bundles in association with hyalinization, in contrast to the well-aligned collagen fiber bundles found in the control group (Fig. 1). Hyalinization could be identified even macroscopically at a cut surface of the ligament. The unusual crimp pattern of the collagen bundles was much more evident in the elongated ligament than was usual. Thus, the elongated bundle appeared extremely wavy, as is seen in joint capsule-composite bundles (Fig. 1). In the *ruptured* cases, well-aligned collagen bundles were suddenly interrupted. The scar contained abundant thin vessels and sometimes fatty tissue. However, inflammatory cells were not observed in any of the injury group. Notably,

Table 1. Specimens with lateral ligament injuries in the ankle

Parameters	ATFL injury	CFL injury
No. of cadavers (injured/examined)	27/108 (25.0%)	5/108 (4.6%)
No. of ankles (injured/examined)	33/128 (25.8%)	7/128 (5.5%)
Cadavers with injured ankles (male/female)[a]	13/14	5/0
Right/left injured ankles[b]	20/13	3/4
Types of injury: elongation/rupture[b]	20/13	5/2
Age distribution of injured cases		
<70 Years	8 (ankles)	3 (ankles)
70–79 Years	10	3
80–89 Years	11	1
>90 Years	4	0

ATFL, anterior talofibular ligament; CFL, calcanofibular ligament
[a] All 27 (ATFL injury) or 5 (CFL injury) cadavers, respectively. All 5 CFL injuries were seen accompanying an ATFL injury. Thus, all cadavers with a ligament injury accounted for the 27
[b] All 33 (ATFL injury) or 7 (CFL injury) ankles

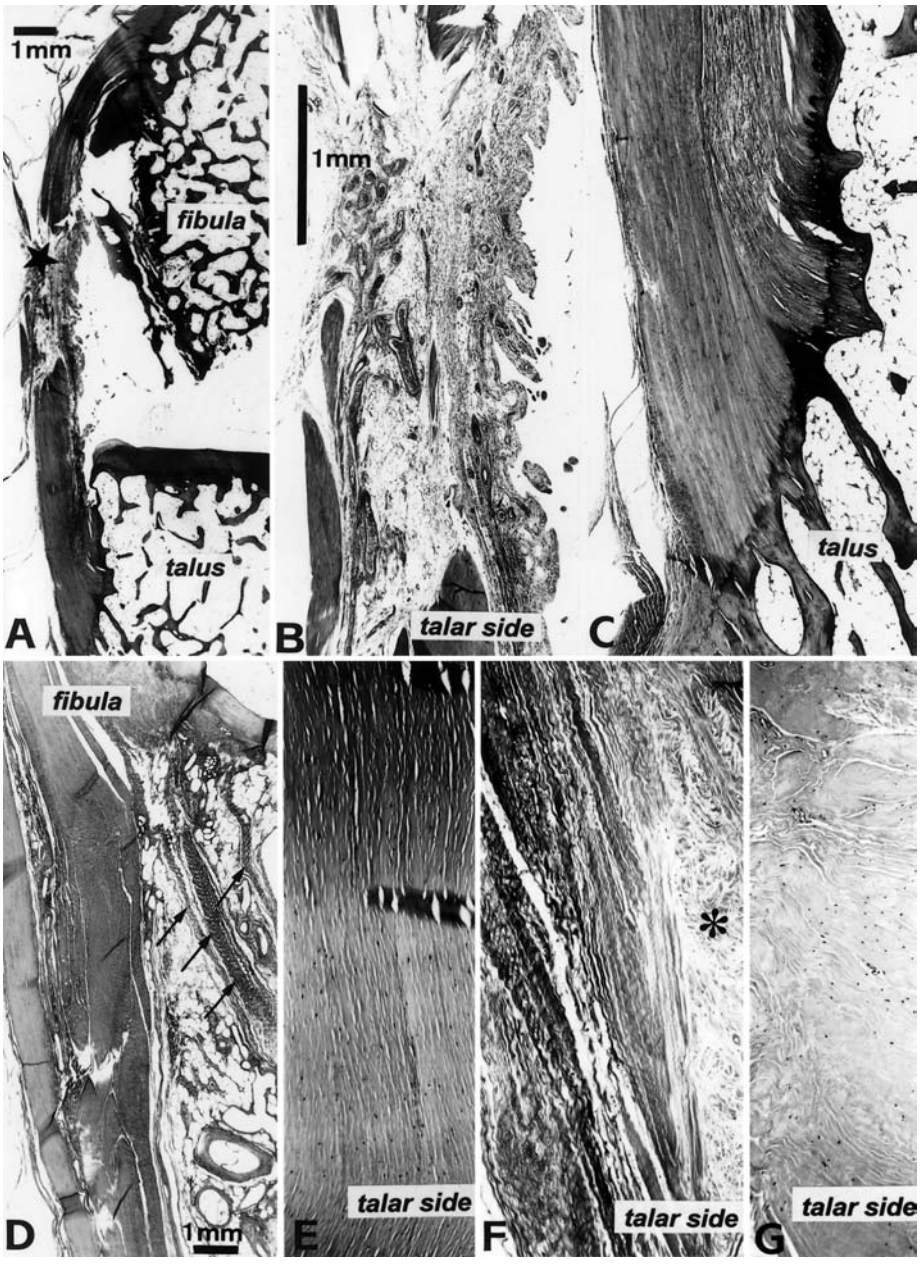

Fig. 1. Histology of anterior talofibular ligament (ATFL) injury. **A–F** Longitudinal sections of the ATFL. **G** Oblique section. **B,C,E–G** are the same magnification (scale bar is inserted only in **B**). **A** Rupture of the ligament (*black star*), **B** (scar at the injury part) and **C** (ligament–bone interface) are higher magnification views of the same specimen. In rupture cases, collagen bundles are arrayed almost regularly (**C**), and the morphology of the enthesis appears normal. The fibrocartilage tissue is not evident, but it is present. **D,E** Normal ATFLs comprising well-arrayed collagen bundles (**E**). **F,G** Specimens identified macroscopically as "elongation" of the ligament. In the normal specimen, wavy collagen bundles tended to be restricted to those contained in the joint capsule (*arrows* in **D**). In an elongation case (**F**), the ligament is occupied by the wavy bundles (at *left*) and slight hyalinization (*asterisk*). In another case (**G**), almost the entire ligament is replaced by hyalinization

with both injury patterns the bone–ligament interface maintained its basic morphology; that is, fibrocartilage mediated between the collagen bundles and the bone (Fig. 1).

After recording our observations of the lateral ligaments, the ankle joints were dissected from the medial side to expose the entire joint surface. According to Hirose et al.,[5] DCs can be classified into two groups: (1) a normal group that includes slight degeneration (e.g., swelling or fibrillation); and (2) a group with distinct degeneration including wide erosion, deep fissures, and cartilage defects (not illustrated). When reporting the results, the latter group is simply called "cartilage degeneration." In our previous study,[5] we had used a term "sector" to avoid confusion with the words "parts" and "portion" because these two words seemed to provide images other than our artificial division and because the word "sector" is familiar in fields such as liver anatomy and surgery. Therefore, to depict DCs in the present study, eight portions, or "sectors," were defined (i.e., two pairs of the medial and lateral halves of the plafond and dome and four malleolar facet surfaces). The area of the DC was not evaluated with an absolute value (e.g., square millimeters) but with an approximate value according to how many sectors were affected. Those results were analyzed using a two-tailed *t*-test.

Terminology

For our purposes, we often use such specific terms as the (talar) "dome" and (tibial) "plafond" for the major articular surfaces to distinguish them from the medial and lateral maleollar facet surfaces. The dome corresponds to the convex surface of the talus for articulation except for the malleolar facets, and the plafond corresponds to the complementary, concave surface of the distal end of the tibia.[3,11]

Results

Lateral ligament injury

We obtained 33 ankles with ATFL injuries from 27 cadavers (Table 1). Of the 33 ankles, bilateral injury existed in 12 ankles (4 men, 2 women). These lesions were classified into 20 elongation and 13 rupture cases. The two types of ATFL injury were almost equally common among the age groups (Table 1). All seven CFL injury cases, found in five male cadavers (three right, four left; 5.5% of the 128 ankles), were combined with the ATFL injuries. The CFL injury included five elongation and two rupture cases. Notably, six of the seven ankles with the CFL injury were combined with an ATFL rupture. The histological findings in the injury group were described in the Materials and Methods section because they were closely connected to the definitions of the injuries.

In addition, we found two avulsion fractures at the fibular connection of the ATFL with no pathological findings in the ligaments. These two avulsion fractures were found in a male cadaver aged 84 and a female cadaver aged 97 years.

Cartilage degeneration of the articular surface

Degenerative changes were found more frequently in the ligament injury group than in the control group: 78.8% in the injury group in contrast to 49.5% in the control group ($P < 0.01$). This difference was more significant on the talar side than on the tibial side of the joint and in the lateral sites than in the medial sites of the joint (Table 2). The numbers of affected areas, or sectors, were larger in the injury group than in the control group. The talar-side dominance of DCs was evident in both groups. However, the medial facet dominance was unclear in the ligament injury group because the lateral facet and the lateral halves of the plafond and dome were also frequently affected (Table 2). Especially, the lateral malleolar facet encountered a significantly higher incidence of DCs in the injury group than the control group (45.2% vs. 18.3% of ankles examined; $P < 0.01$).

The injury group had DCs in 2.61 sectors, on average, in contrast to 1.39 sectors in the control group. We defined "multifocus" DCs as lesions seen in more than two of the eight sectors. In both groups, multifocus DCs were found much more frequently than solitary lesions, which occupied only one sector (Table 1). Solitary lesions tended to occur more frequently on the talar side than the tibial side. On the tibial side of the injury group, we found no solitary lesions restricted to one

Table 2. Incidence, site, and solitary/multiple occurrence of cartilage degeneration in the tibial and talar sides of the talocrural joint: control and ligament injury groups

Site	Control group	Ligament injury group
Tibial side		
Medial half of the plafond	15/93 Ankles (16.1%); solitary 1, multiple 14	8/33 Ankles (24.2%); solitary 0, multiple 8
Lateral half of the plafond	5/93 (5.4%); solitary 0, multiple 5	3/33 (9.1%); solitary 0, multiple 3
Medial malleolar facet	19/93 (20.4%); solitary 2, multiple 17	13/33 (39.4%)*; solitary 0, multiple 13
Lateral malleolar facet	4/93 (4.3%); solitary 0, multiple 4	5/33 (15.2%)*; solitary 0, multiple 5
All four sites on the tibial side	26/93 (28.0%)	13/33 (39.4%)
Talar side		
Medial half of the dome	33/93 ankles (33.5%); solitary 5, multiple 28	21/33 (63.6%)**; solitary 6, multiple 15
Lateral half of the dome	13/93 (14.0%); solitary 1, multiple 12	15/33 (45.5%)**; solitary 1, multiple 14
Medial malleolar facet	23/93 (24.7%); solitary 1, multiple 22	12/33 (36.4%); solitary 0, multiple 12
Lateral malleolar facet	17/93 (18.3%)*; solitary 4, multiple 13	14/33 (42.4%)**; solitary 3, multiple 11
All four sites on the talar side	42/93 (45.2%)**	25/33 (75.8%)**
Both sides		
One or more of four medial [lateral] sites[a] affected	40/93 (43.0%); [25/93 (26.9%)]	22/33 (66.7%)*; [18/33 (54.5%)]**
At least one site affected	46/93 (49.5%)	26/33 (78.8%)**

[a] The four medial [lateral] sites: medial [lateral] halves of the dome and plafond and the talar and tibial sides of the medial [lateral] malleolar facet
* $P < 0.05$; ** $P < 0.01$

Table 3. Incidence of mirror-image degeneration in the talocrural joint: control and ligament injury groups

Site	Control group	Ligament injury group
Medial half of the plafond and dome	12/93 Ankles (12.9%)	6/33 Ankles (18.2%)
Lateral half of the plafond and dome	4/93 (4.3%)	3/33 (9.1%)
Medial malleolar facet surfaces	12/93 (12.9%)	9/33 (27.3%)
Lateral malleolar facet surfaces	4/93 (4.3%)	4/33 (12.1%)
Both medial sites	19/93 (20.4%)	10/33 (30.3%)
Both lateral sites	7/93 (7.5%)*	7/33 (21.2%)*
In the ankle[a]	21/93 (22.6%)[b]	11/33 (33.3%)[b]

* $P < 0.05$

[a] One or more of the four sites were affected with mirror-image lesions

[b] Specimens in which degenerative changes were restricted to the medial malleolar facet: seven ankles in the control group and three in the injury group. They were restricted to the medial malleolar facet and other site(s) in five controls and six in the injury group. The medial malleolar facet was not involved in nine controls and two in the injury group

sector. Thus, a solitary/multiple ratio, if considered, was usually smaller in the injury group than in the control group.

A site in a joint surface and its "complementary site" face each other and articulate together. At every four sites on the tibial side of both groups, more than half or even all lesions on the tibial side had their mirror image lesion in the complementary site of the talus (Tables 2, 3). Conversely, complementary lesions were formed in fewer than half of the talar DC areas. Thus, the talar lesion often did not accompany the complementary tibial lesion. The mirror-image lesion was found more frequently in the ligament injury group than in the control group at every site (Table 3). Especially in the lateral sites (lateral malleollar facet and lateral halves of the plafond and dome), the difference was evident between the two groups: 21.2% (7/33) in the injury group and 7.5% (7/93) in the controls. The mirror-image lesion was often found at multiple sites in a joint. Conversely, the solitary mirror-image lesion, restricted to one portion of a joint, was found in 36.4% of the 33 injured ankles, whereas 61.9% of control specimens carried the solitary mirror-image lesion. In the control group, the medial malleolar facet was affected in 12 of the 21 ankles with mirror-image lesions; 9 of them had no DCs (Table 3). Notably, in the ligament injury group, medial malleolar lesions were found in 9 of the 11 ankles with mirror-image lesions, despite the fact that medial facet dominance was unclear in the ligament injury group (see above).

Although data are not shown in detail, a cartilage defect was more frequently observed in the injury group than in the control group, except in the lateral halves of the plafond and dome. In the control group, a cartilage defect was not found in the younger (<70 years old, 11 ankles) or older (>90 years, 5 ankles) populations. We found no clear difference in localization or incidence of DCs between bilateral and unilateral injuries of the ATFL or between elongation and rupture cases. Although most specimens were obtained from the elderly, DCs tended to occur more frequently in the more aged population.

In addition, in two avulsion cases, DCs were found in the medial halves of the dome. One (male) case carried the mirror-image lesion in the medial halves of the dome and plafond in combination with the maleollar facet DCs, whereas in another (female) case the lesion was restricted to the medial half of the dome.

Discussion

Evaluation of the lateral ligament injury

All of the CFL injuries were seen in combination with the ATFL injuries. This is consistent with a clinical report,[1,11] although their subjects were younger than those in the present study. Notably, in our study all CFL injuries were seen in male cadavers. Although to the best of our knowledge the morphology of lateral ligament injuries of the ankle has not been described in detail, the ligament with abnormal macroscopic findings, such as unclear separation from the surrounding tissue, could be simply divided into (1) rupture with scar and (2) elongation according to the present histological definition. The former status was clearly confirmed by histology, whereas in the latter category ligaments carried unusual wavy collagen bundles with hyalinization. It was unlikely that elongation was the final stage of recovery after a rupture because in rupture cases collagen bundles were well arrayed and displayed a slight crimp pattern.

We have no information about the time of onset of the injury. That no inflammatory cells were associated with the ligament injury suggest that a long time had passed. This seemed to be a critical limitation of the

present study. Surprisingly, however, the histological configuration of the ligament–bone interface was well preserved (Fig. 1). The morphologies of the fibrocartilage-mediated entheses were essentially consistent with a recent excellent description by Kumai et al.,[7] although our staining method was limited. Laros et al.[8] experimentally demonstrated that the time duration varied significantly among individual specimens (dogs) until the interface was reconstructed after the ligament was cut. Because we were using preserved cadavers, we could not check for abnormal movement of the joint with lateral ligament insufficiency (see below).

Which DC morphologies characterized the injury group?

In both groups, we found talar-side dominance of DCs, multifocus occurrence, and a high incidence of mirror-image DCs in the medial sites of the joint, as reported in our previous report.[5] The talar dominance was consistent with the articular cartilage being thinner than on the tibial side.[14] However, in young patients around 20 years old, chronic injury of the ATFL is liable to cause DCs on the anteromedial edge of the tibial plafond and lateral side of the talar dome.[11] As a result of lateral ligament insufficiency, the talus seems to be internally rotated, inverted, and pulled out of the ankle mortise; and in this subluxated and supinated position,[3,6] the body weight is likely to exert axial compression stress on the medial malleolar facet. The shearing and impaction forces seem to generate DCs easily. Moreover, in an in vitro study, Noguchi[10] noted that the stress distribution is shifted medially after sectioning the lateral ankle ligaments. Furthermore, the mirror-image lesion was multiple or large (or both) in the injury group, in contrast to the relatively restricted manner in the control group. The averaged numbers of affected areas, or sectors, in the injury group were almost twice as large as in the control group. Consequently, lateral ligament insufficiency is more likely to accelerate the progress and advancement of DCs in the lateral site than in the medial site.

The difference between the two groups was most evident in the lateral sites of the joint; that is, a significantly high incidence of DCs ($P < 0.01$), including mirror-image lesions ($P < 0.05$), was found in the lateral facet of the injury group (Tables 2, 3). Chondral lesions are known to occur secondary to supination trauma of the ankle. Among those with an acute ATFL injury the chondral lesion is encountered in 89% of patients.[11] The lateral site dominance found in the present elderly population might not argue against the usual biomechanical discussion about medial site dominance, but it suggests another specific manner of progression and advancement that is secondary to the lateral ligament injury. We speculated that it was direct damage due to trauma because of flap-like lesions observed by arthroscopy in acute cases.[11]

Because of the high incidence of the lateral sites in the injury group, the medial malleolar facet dominance of DCs[5] became invisible. In the ligament injury group, however, there were no medial malleolar lesions in 2 of the 11 ankles with mirror-image lesions, in contrast to 9 of the 21 in the control group. Previously, according to a detailed analysis of DC localization, we hypothesized that the medial malleolar facet often carries the initial DC lesion.[5] Conversely, the medial facet seemed not to be the initial site of DC in 42.9% (9/21; see just above) of the control group, whereas in the injury group such "exceptions" were limited to 18.2% (2/11). Thus, the medial facet generator hypothesis seemed to be consistent with findings in the injury group rather than those in the normal controls. Because the population examined was elderly, we hypothesized that DCs in the ankle had already occurred and had advanced to a greater or lesser degree. Therefore, it was likely that a progression pattern of DC was similar to that in the control group, but it was strongly accelerated and modified by suggested direct chondral damage that occurred in combination with the lateral ligament injury. Consequently, in elderly patients, early evaluation and treatment for lateral ligament insufficiency of the ankle is necessary to avoid osteoarthritis, which seems to spread more widely and perhaps more rapidly than in younger patients.

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