Surgical Treatment of Osteochondral Lesions of the Talus in Young Active Patients

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Introduction

Cartilage is a smooth, highly specialized tissue that coats the surface of the joint. Although it is only a few millimeters thick, it has exceptional stiffness to compression and resilience and is able to distribute loads[1]. It is susceptible to injury and is limited in regenerative capability[2]. The biological function of cartilage is to permit articular movement while minimizing surface friction, to absorb loads in the weight-bearing joints, and to reduce the stress on the subchondral bone.

Hyaline (articular) cartilage consists of 70% water; 15% collagens (primarily type II); and 15% proteoglycans (in particular, aggrecan), noncollagen proteins, lipids, and inorganic material. Chondrocytes, the only cell type in this tissue, sit within the matrix of proteoglycans and collagen, which give the cartilage its compressive and tensile properties[3] (Figs. 1-A and 1-B).

Osteochondral lesions of the talus are defects of the cartilaginous surface and underlying subchondral bone of the talar dome[4]. The etiology of osteochondral lesions of the talus can be divided into primary and secondary. Primary osteo-

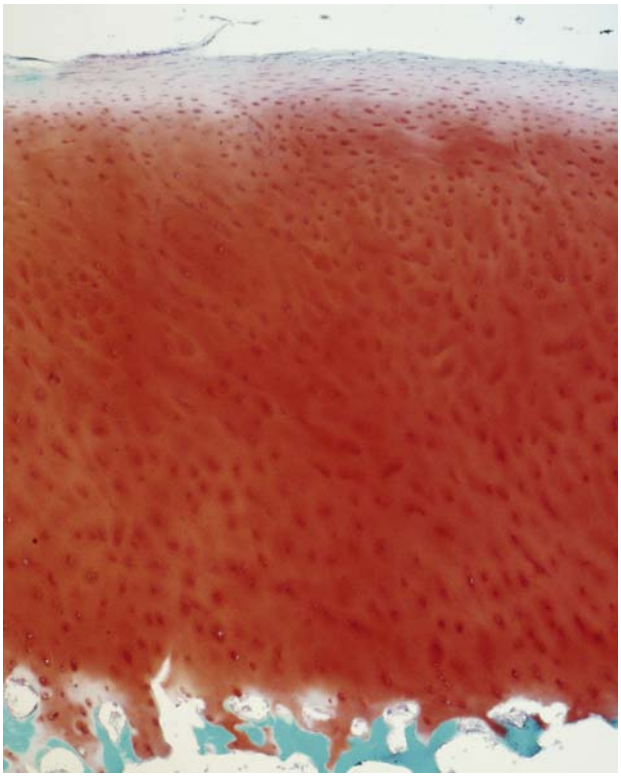

Fig. 1-A

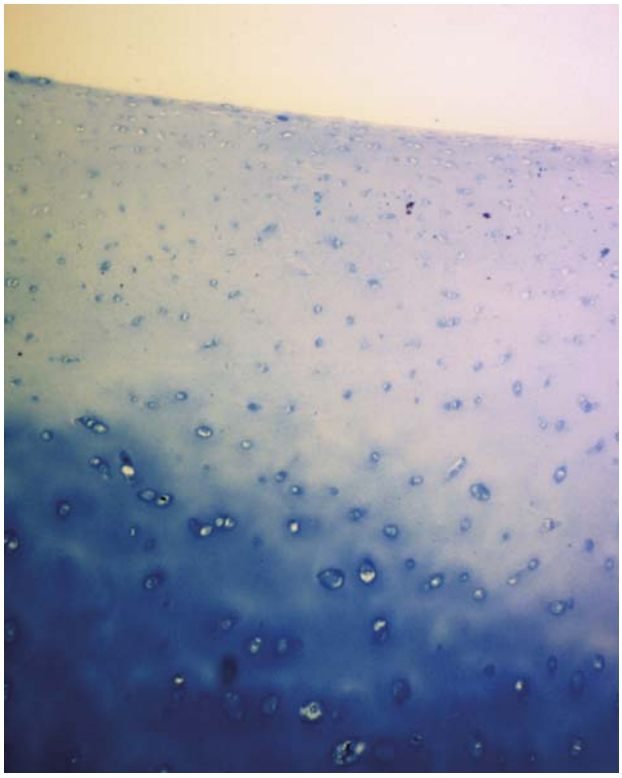

Fig. 1-B

Figs. 1-A and 1-B Histological appearance of hyaline cartilage. **Fig. 1-A** After staining with safranin O (×30). **Fig. 1-B** After staining with alcian blue (×100).

TABLE I Classification System for Osteochondral Lesions of the Talus

Type of Lesion	Surface	Extension	Treatment
Acute			
I	Damaged	<1 cm^2	Débridement
II	Damaged	≥1 cm^2	Fixation
Chronic			
0	Intact	Any	Drilling
I	Damaged	<1.5 cm^2	Microfractures
II	Damaged	≥1.5 cm^2	Cartilage replacement
IIA	Damaged	≥1.5 cm^2, >5 mm deep	Cartilage replacement and bone graft
III	Damaged		Osteochondral massive graft

TABLE II Etiology of the Lesions in the Eighty Patients

Etiology	No. of Patients	Description
Primary osteochondral lesion	5	No trauma or biomechanical causes were definable at clinical interview and examination
Secondary osteochondral lesion		
Chronic instability	7	Chronic instability of the ankle (five patients) and a mild-severe associated cavus foot (two patients)
Lower limb malalignment	1	Valgus deviation of the lower part of the tibia following a lower limb fracture
Trauma	67	One or recurrent ankle sprains reported at the clinical interview (sixty-one patients) and fracture of the talus (six patients)

chondral lesions of the talus represent chronic diseases of the subchondral bone, most likely due to a deficiency of the vascular supply. More rarely, they are described as associated with endocrine disorders and genetic abnormalities[4]. Primary osteochondral lesions were formerly described as osteochondritis dissecans[5]. Secondary osteochondral lesions of the talus most likely occur as a result of ankle injuries (ankle sprain or fracture), chronic instability, axial defects of the lower leg, or dysbaric osteonecrosis[6,7].

Because joint cartilage has a poor reparative capability, osteochondral lesions of the talus rarely heal spontaneously. More frequently, patients with osteochondral lesions of the talus have chronic pain develop at the ankle joint, which seriously limits daily life and sports activities[3,8]. If osteochondral lesions of the talus are not diagnosed early and treated, they may evolve into arthritis of the ankle joint.

The surgical strategy we use is planned according to the classification, diameter, stage, and depth of the lesion (Table I). Other factors to be considered are patient age (autologous chondrocyte implantation should not be used in patients who are more than fifty years old), the degree of functional impairment, the level of patient activity, the absence of kissing lesions, and lower limb malalignment or associated ankle instability or deformity of the foot (correction of these defects, if present, must be achieved at the same time or in an earlier operation).

The purpose of this study was to present our treatment guidelines for osteochondral lesions of the talus based on the results of a series of eighty consecutive patients.

Materials and Methods

Eighty patients (mean age [and standard deviation], 27 ± 8 years) were treated between 1996 and 2001 (Table II). According to the arthroscopic and magnetic resonance imaging classification described by DiPaola et al.[9], all lesions were stage III or IV. All patients had a clinical examination preoperatively and at twelve months and four years postoperatively to assess pain, range of motion, and alignment with use of the American Orthopaedic Foot and Ankle Society score[10]. In addition, radiographs were made and magnetic resonance imaging scans were acquired.

Surgical Technique

Surgical treatment was performed with the patient under general, spinal, or block anesthesia. A tourniquet was applied at the thigh level. The approach to the lesion can be open or arthroscopic, depending on the type of the lesion, the location, and the surgeon's preferences or experience. In patients who had arthrotomy, the skin incision was longitudinal on the side of the lesion. If the lesion was covered by the medial or lateral malleolus, an osteotomy was performed in order to obtain better exposure. In patients who had arthroscopy, anteromedial and anterolateral approaches were used. The joint was stressed with manual traction.

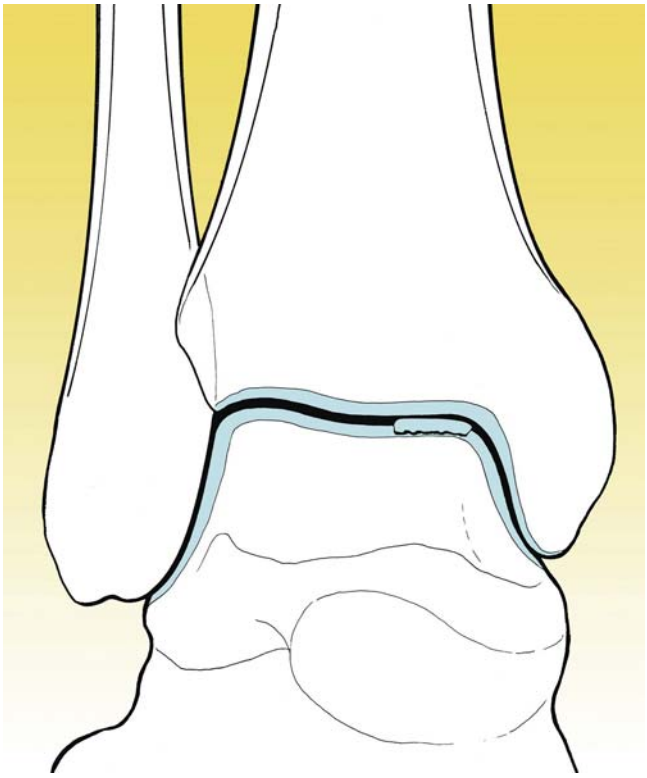

Fig. 2-A

Figs. 2-A, 2-B, and 2-C In an acute type-I lesion, only the superficial layer of the cartilage is involved. **Fig. 2-A** Diagram of the lesion.

In three patients with an acute type-I osteochondral lesion of the talus, arthroscopic débridement of the lesion was performed (Figs. 2-A, 2-B, and 2-C). In one patient, this occurred as a consequence of an ankle sprain with lateral ligamentous disruption. A direct suture repair of the ligamentous lesion was also performed.

In four patients with an acute type-II osteochondral lesion of the talus, open reduction and internal fixation was performed. In one patient, a lateral ligamentous lesion repair was also performed (Figs. 3-A, 3-B, and 3-C).

In two patients with a chronic type-0 osteochondral lesion of the talus, retrograde drilling, according to the technique described by Conti and Taranow[11], was performed in order to preserve the intact cartilage and repair the underlying subchondral bone (Figs. 4-A, 4-B, and 4-C). Softening of the cartilaginous surface was detected arthroscopically, but the cartilage surface was intact. In one patient with a mild cavus foot, predisposing to recurrent ankle sprains, the cavus foot was corrected with an osteotomy of the first metatarsal and a percutaneous plantar fasciotomy was performed as an associated procedure.

In thirty-one patients with a chronic type-I osteochondral lesion of the talus, arthroscopic microfractures were performed (Figs. 5-A through 5-D). The lesion was detected, the degenerated cartilage was excised, and the subchondral bone was exposed. The lesion was curetted down to the bleeding bone. An arthroscopic awl gently pushed with a mallet was used to make multiple microfractures approximately 3 to 4 mm apart in the exposed subchondral bone plate. In one patient with a mild cavus foot, predisposing to recurrent ankle sprains, correction of the cavus foot by osteotomy of the first metatarsal and percutaneous fasciotomy was performed. Furthermore, in three patients with ankle chronic instability, a lateral ligament repair according to the technique described by Brostrom[12] was also performed.

In thirty-four patients with a chronic type-II osteochondral lesion of the talus, cartilage replacement was performed with use of autologous chondrocyte implantation (Figs. 6-A, 6-B, and 6-C). In one patient with lower limb malalignment following a fracture, a tibial osteotomy was performed. Furthermore, in one patient with chronic ankle instability, an external ligament repair according to the technique

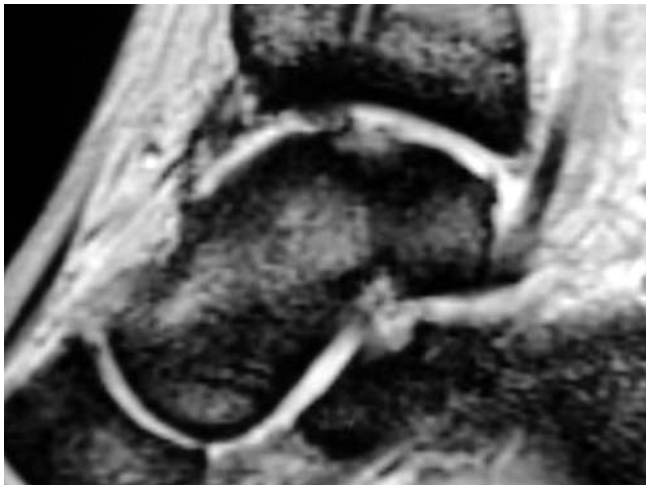

Fig. 2-B

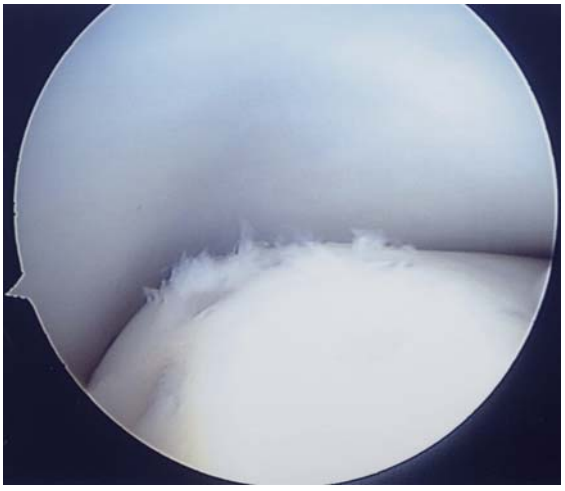

Fig. 2-C

Fig. 2-B The lesion was detected on T2-weighted magnetic resonance imaging. **Fig. 2-C** The fragment was arthroscopically excised, and the lesion site was carefully shaved.

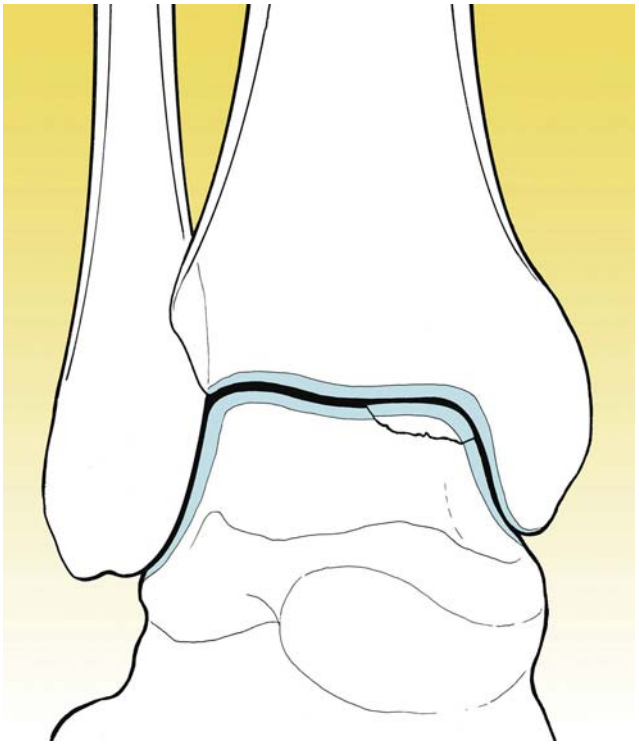

Fig. 3-A

Figs. 3-A, 3-B, and 3-C In an acute type-II lesion, open reduction and internal fixation of the fragment is indicated. **Fig. 3-A** Diagram of the lesion.

of Brostrom[12] was also performed as an associated procedure.

In five patients with a chronic type-IIA osteochondral lesion of the talus (the defect was >5 mm deep), a cancellous bone graft combined with cartilage replacement (autologous chondrocyte implantation) was performed (Figs. 7-A through 7-D). One patient with chronic ankle instability also had a lateral ligament repair according to the technique of Brostrom[12].

Two-Step Surgical Technique for Autologous Chondrocyte Implantation in Chronic Type-II and Type-IIA Osteochondral Lesions of the Talus

First Step

A first-step arthroscopy that allowed direct evaluation and accurate measurement of the lesion was performed in all patients. In the first fourteen patients, cartilage was harvested arthroscopically from the ipsilateral knee. In the last twenty-five patients, the cartilage was harvested directly from the affected ankle in the first-step arthroscopy, with the chondrocytes of the detached osteochondral fragment used as a source of cells for culture. In the five patients with a defect that was >5 mm deep, bone-grafting was performed at this step. Cartilage was sent for cell expansion and was available for implantation four weeks later.

Second Step

In the first nine patients, the surgical approach to the lesion was transmalleolar, medial, or lateral. Once the lesion was adequately exposed, the damaged cartilage was removed up to a

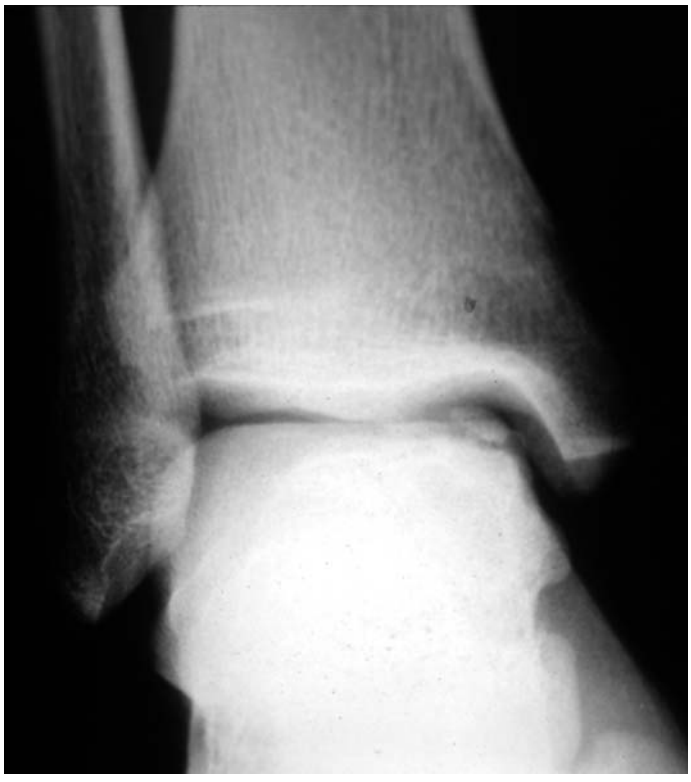

Fig. 3-B

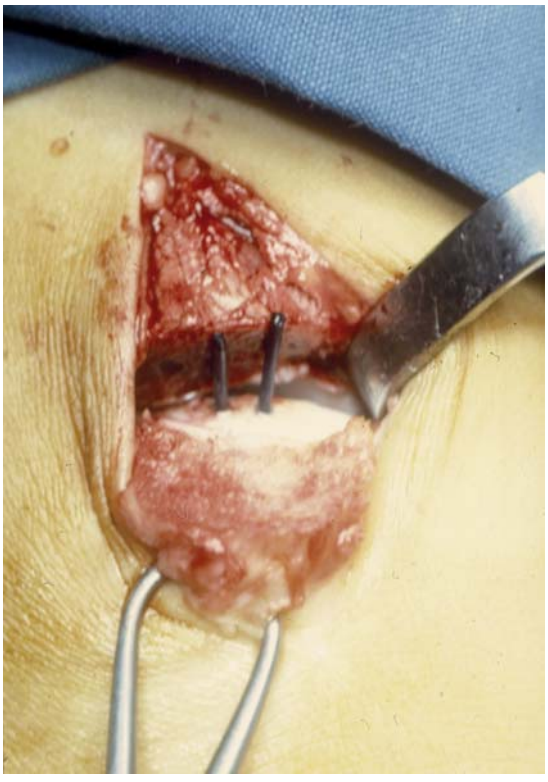

Fig. 3-C

Fig. 3-B The lesion was detected on radiographs. **Fig. 3-C** Reduction was achieved, and fixation was performed with use of bioabsorbable pins.

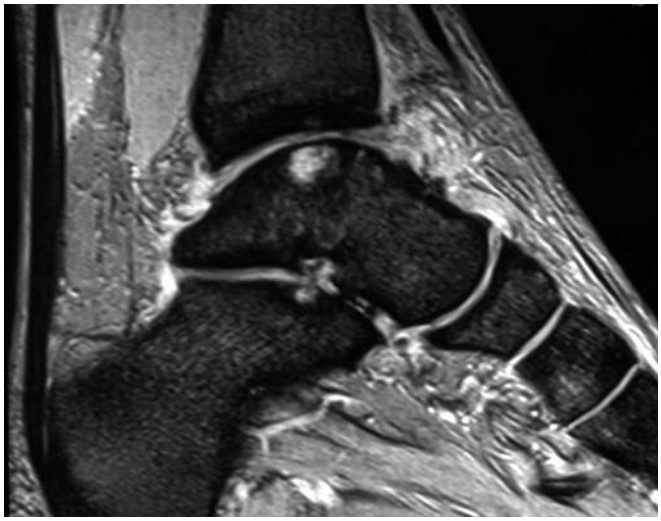

Fig. 4-A

Figs. 4-A, 4-B, and 4-C In a chronic type-0 lesion, a defect of the subchondral bone is covered by an intact cartilage layer. **Fig. 4-A** A T2-weighted magnetic resonance imaging scan showing the lesion.

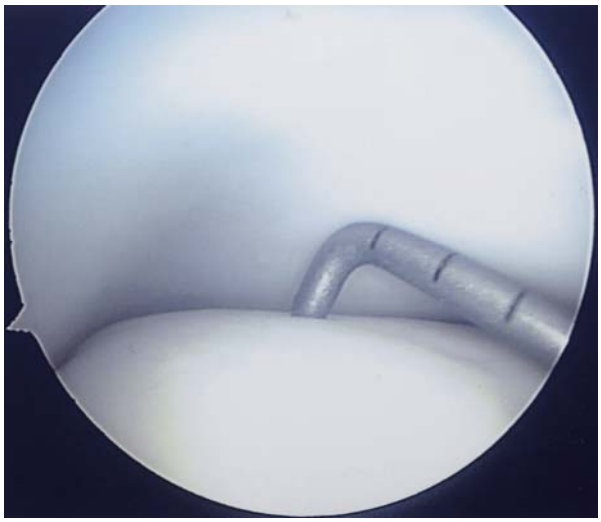

Fig. 4-B

The cartilage sheet appeared intact at arthroscopy.

sharply defined rim of healthy cartilage. The lesion was measured. A periosteal flap of adequate size, harvested either from the proximal or distal aspect of the tibia, was then fixed over the cartilaginous gap with reabsorbable 6-0 Vicryl suture (polyglactin 910; Ethicon, Somerville, New Jersey) and sealed with Tissucol (Baxter International, Vienna, Austria) fibrin glue (Figs. 8-A through 8-D). The chondrocytes in a liquid medium were then implanted under the periosteal flap, and the malleolar osteotomy was repaired.

Arthroscopic approaches were used in the subsequent thirty patients (Fig. 9). The lesion site was trimmed to safeguard the integrity of the subchondral bone, and a sharp rim of healthy cartilage was defined. The lesion was measured with use of a probe graduated in millimeters. The chondrocytes were supported directly on Hyalograft-C scaffolds (Fidia Advanced Biopolymers, Abano Terme, Italy), which were positioned arthroscopically on the lesions with a custom-made cannula.

In one patient with a chronic type-III osteochondral lesion of the talus, an osteocartilaginous graft was performed (Figs. 10-A through 10-D). The diseased cartilage and subchondral bone were removed until healthy tissue was reached. An accurate measurement was made of the size of the osteochondral fragment to be replaced. The autologous graft was then obtained from a non-weight-bearing area of the ipsilateral knee through a miniarthrotomy incision. This graft may also be obtained from a fresh or frozen talus as an allograft. At the recipient site level, the graft was placed appropriately and fixed with screws and the malleolar osteotomy was repaired.

Postoperative Treatment

All patients were discharged the day after surgery. They were managed with antibiotic therapy for three days and with antithromboembolic therapy (low-molecular-weight heparin) until the time of complete weight-bearing.

In all of the patients treated with only cartilage repair procedures, a soft elastic bandage was applied and a rehabilitation program was begun. Continuous passive motion was started immediately the day after surgery and was gradually increased as tolerated. No weight-bearing on the affected ankle was advised in this phase. At three weeks, progressive weight-

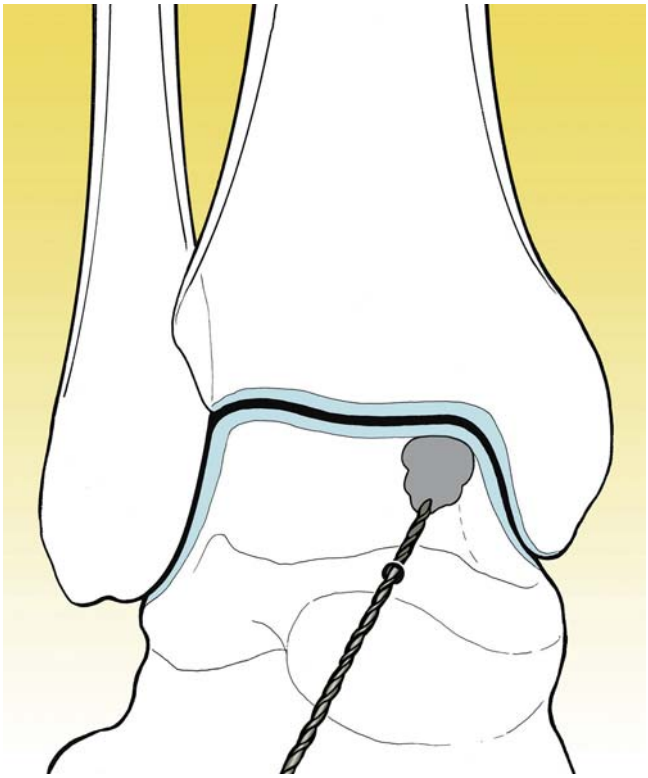

Fig. 4-C

Retrograde drilling is indicated to preserve the integrity of the cartilage tissue.

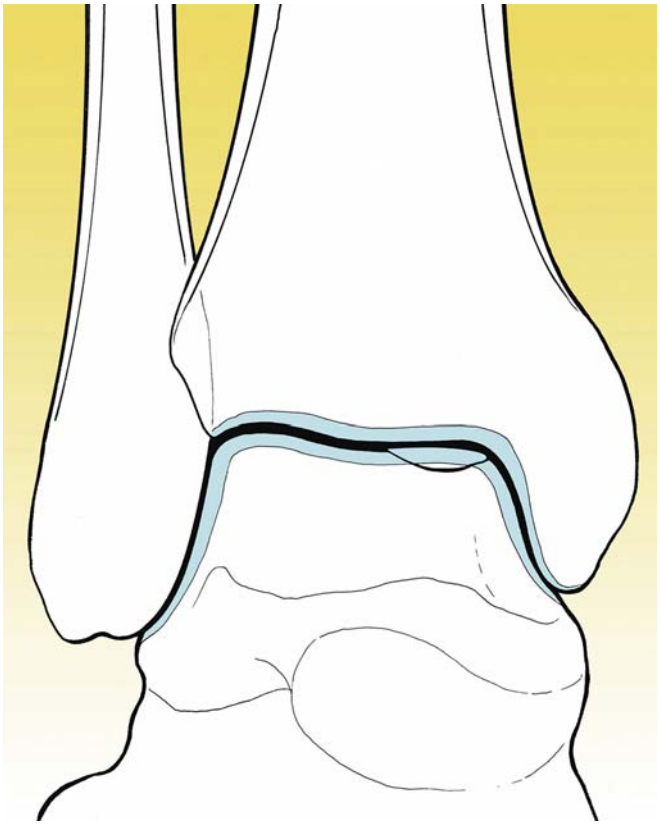

Fig. 5-A

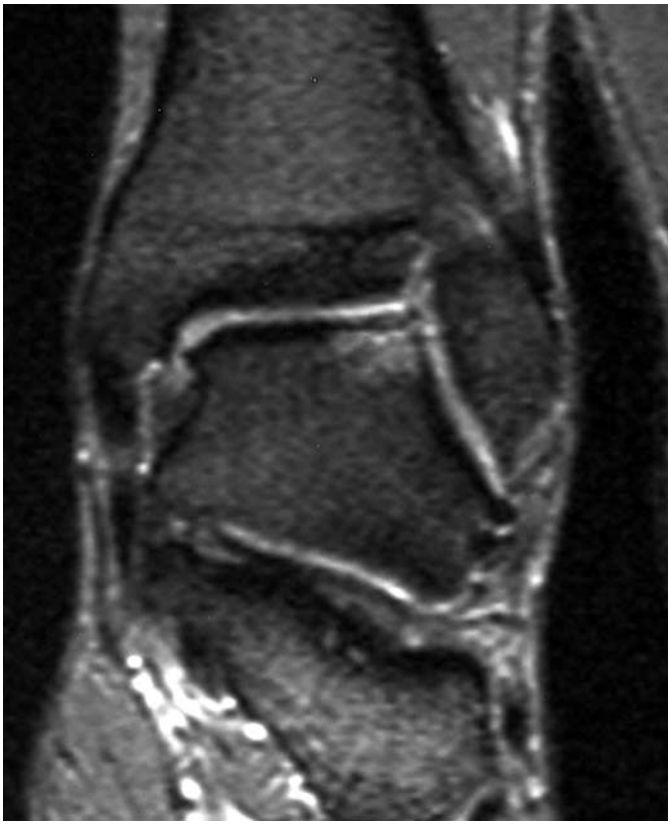

Fig. 5-B

Figs. 5-A through 5-D A chronic type-I lesion. **Fig. 5-A** Diagram of the lesion. **Fig. 5-B** The lesion was detected by T2-weighted magnetic resonance imaging.

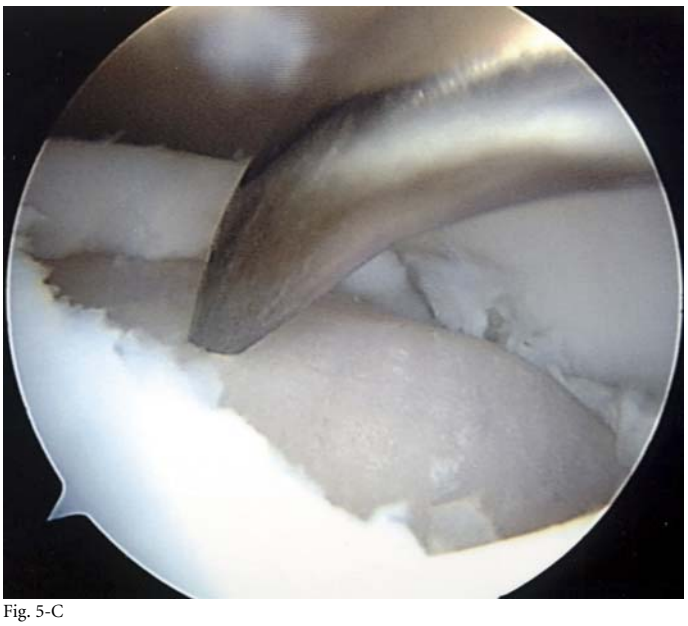

Fig. 5-C

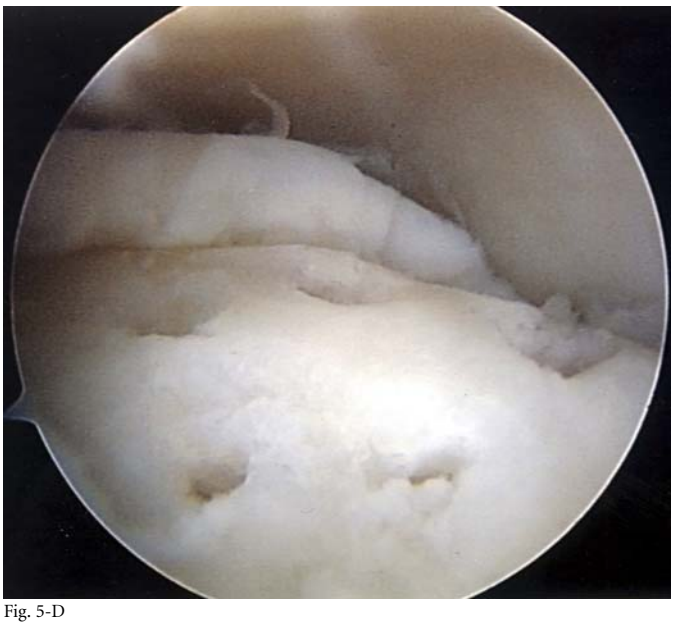

Fig. 5-D

Figs. 5-C and 5-D The lesion was treated arthroscopically by performing multiple microfractures 3 to 4 mm apart.

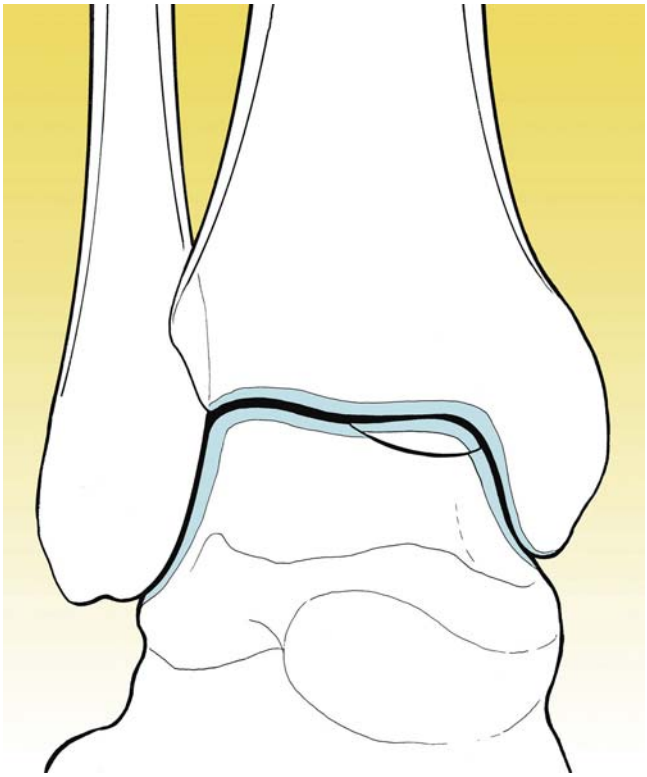

Fig. 6-A

Figs. 6-A, 6-B, and 6-C A chronic type-II lesion. **Fig. 6-A** Diagram of the lesion.

bearing was allowed for patients who received débridement, drilling, and microfractures. Cycling with low resistance and swimming were allowed. Partial weight-bearing at six weeks, increasing to complete weight-bearing at eight weeks, was allowed for the patients who received autologous chondrocyte implantation, fixation of the fragment, or an autologous graft. At four months after surgery, all of the patients were allowed to resume low-impact sports activities and the patients who had débridement, drilling, and microfracture were also allowed to resume running and high-impact activities such as tennis and soccer. At eight to ten months after surgery, those with autologous chondrocyte implantation, fixation of the fragment, or autologous grafts also commenced progressive training for high-impact activities.

Follow-up Evaluation

All patients were examined clinically and radiographically at monthly intervals during the first six months after surgery and then yearly up to the most recent follow-up examination.

After one year, in the first nine patients managed with autologous chondrocyte implantation who had a malleolar osteotomy, the hardware was removed and, at the same time, an arthroscopy with biopsy was performed in order to assess the transplant condition. Arthroscopy was also performed in three patients in the series that had autologous chondrocyte implantation performed arthroscopically. A 2-mm cylinder of cartilage was removed from the transplant area and was subjected to histological and immunohistochemical analyses.

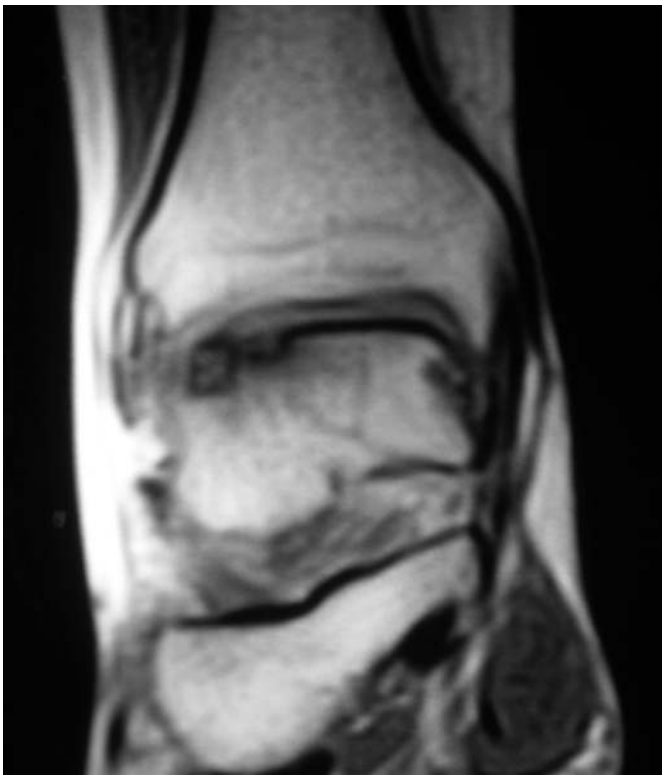

Fig. 6-B

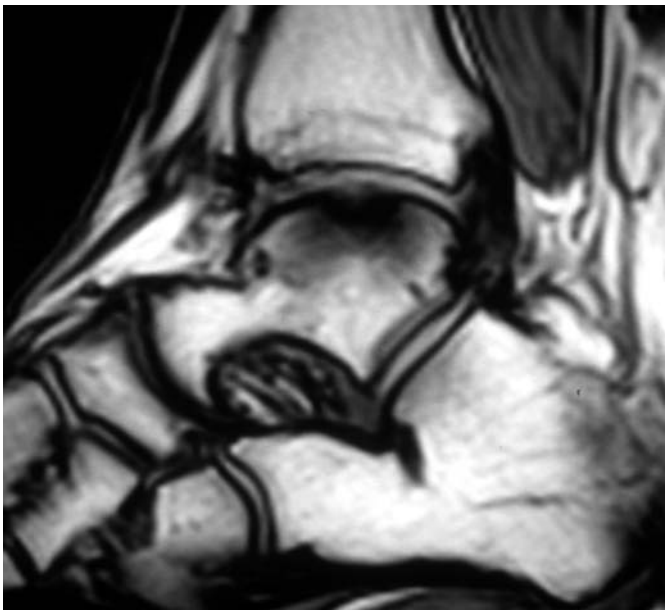

Fig. 6-C

Figs. 6-B and 6-C The lesion was detected by coronal and sagittal T1-weighted magnetic resonance imaging.

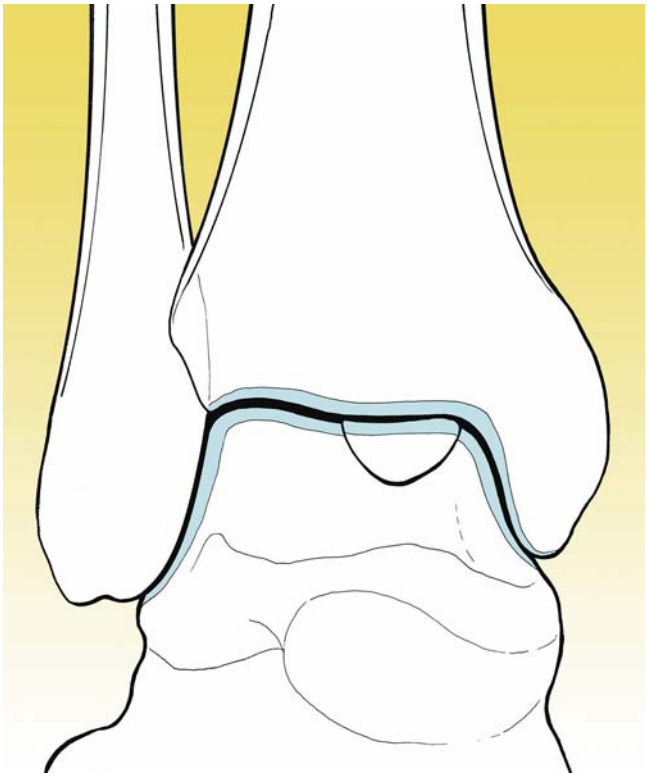

Fig. 7-A

Figs. 7-A through 7-D A chronic type-IIA lesion with an osteochondral defect that was >5 mm deep. **Fig. 7-A** Diagram of the lesion.

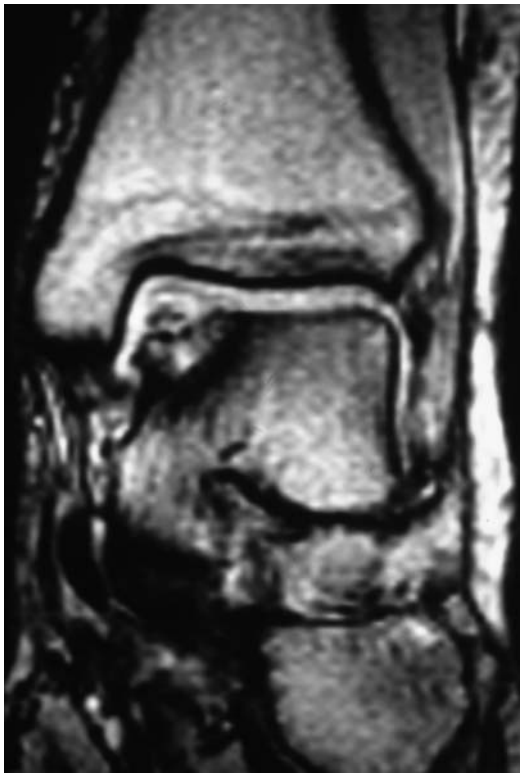

Fig. 7-B

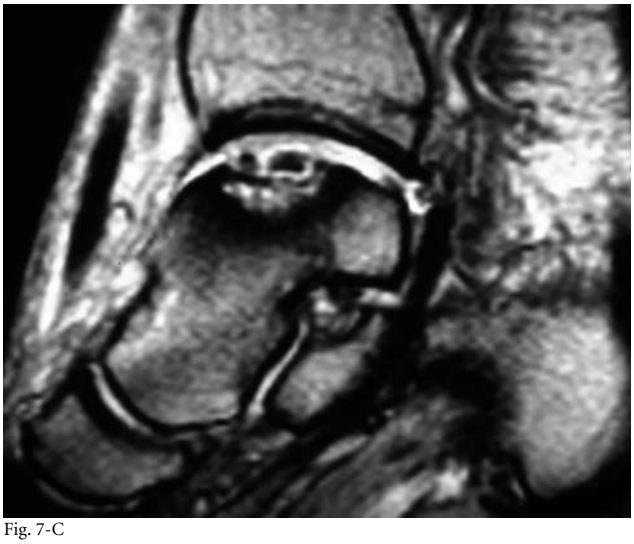

Fig. 7-C

Figs. 7-B and 7-C The lesion was detected by coronal and sagittal T1-weighted magnetic resonance imaging.

Histological and Immunohistological Analyses

Samples for histological analysis were fixed in 10% buffered formalin, washed, and decalcified. The samples were then dehydrated through a graded series of alcohols and were embedded in paraffin. Four-micrometer-thick sections were obtained from the cartilage specimens, and the slides were stored at room temperature until analysis. Slides were stained with 0.001% fast green in distilled water for three minutes at room temperature. The slides were then quickly dipped in 1% acetic acid and were stained with 0.1% safranin O for five minutes at room temperature. Finally, the samples were counterstained with hematoxylin.

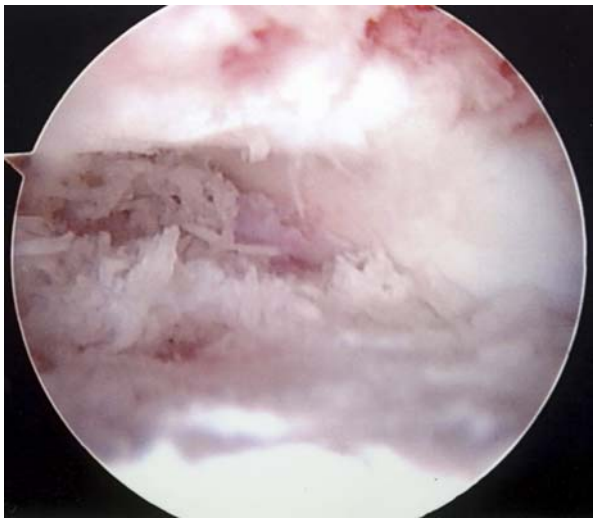

Fig. 7-D

A graft of cancellous bone harvested from the distal aspect of the tibia was placed before the autologous chondrocyte implantation procedure.

The slides were stained in 1% alcian blue 8GX in 3% glacial acetic acid (pH 2.5) for thirty minutes at room temperature, briefly washed in distilled water, allowed to "blue" for twenty minutes in running tap water, and then were dehydrated through a series of alcohols and cleared in xylene.

For immunohistochemical staining of type-I and II collagens, paraffin sections were deparaffinized and rehydrated. For epitope unmasking, the samples were treated with 0.1% hyaluronidase in phosphate-buffered solution at 37°C for five minutes and then were incubated at room temperature for

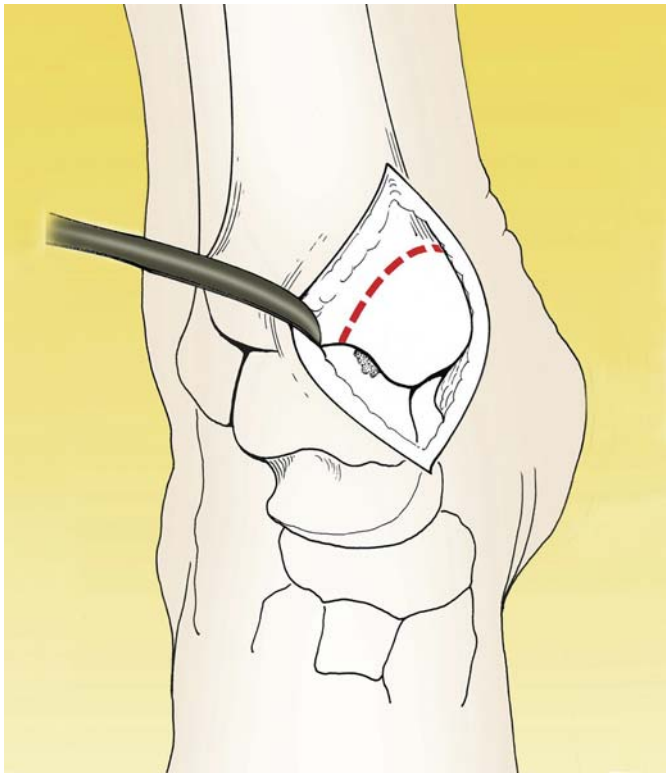

Fig. 8-A

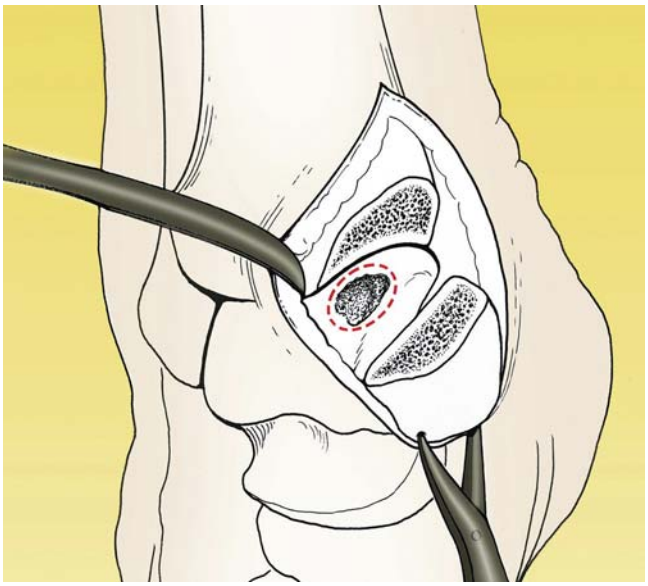

Fig. 8-B

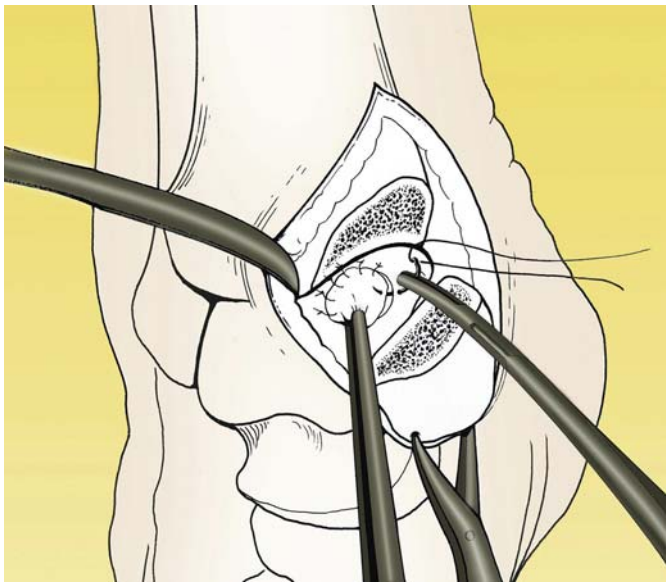

Fig. 8-C

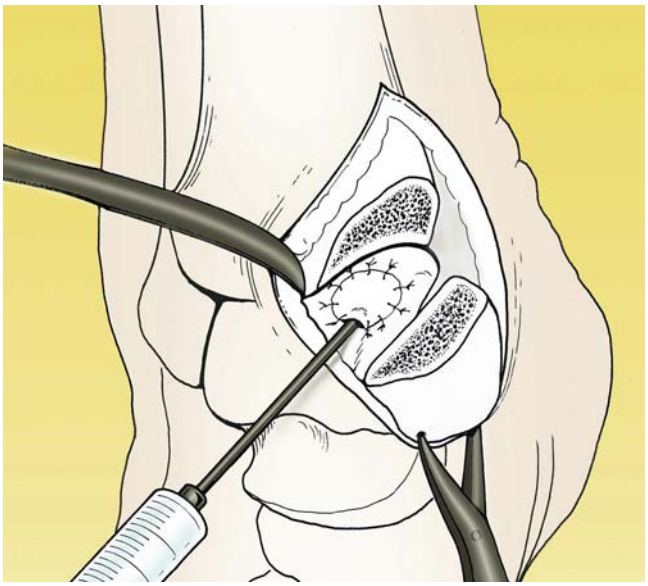

Fig. 8-D

Figs. 8-A through 8-D Open field surgical technique for repair of chronic type-II and type-IIA lesions. **Figs. 8-A and 8-B** A transmalleolar, medial, or lateral approach was used to expose the lesion. **Fig. 8-C** A periosteal flap was sutured over the cartilaginous gap. **Fig. 8-D** The chondrocytes in a liquid medium were then implanted under the periosteal flap.

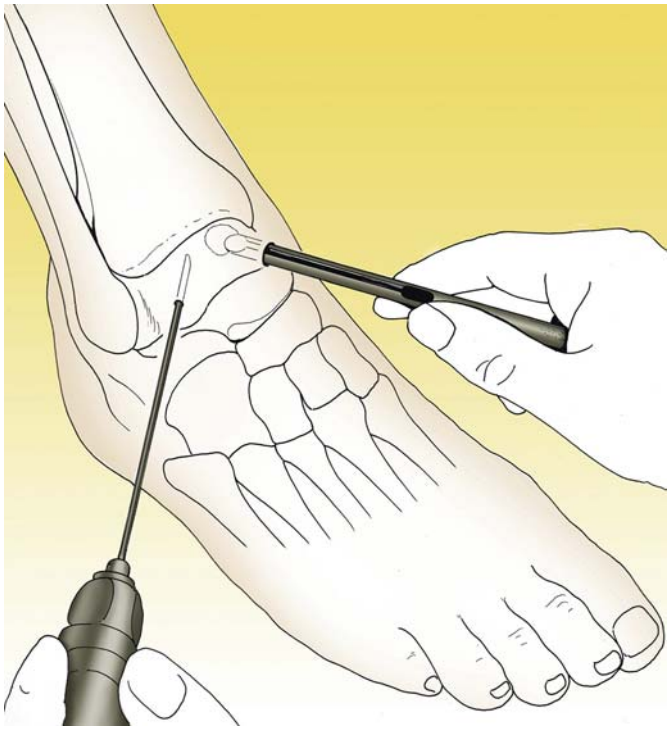

Fig. 9
Arthroscopic treatment of a chronic type-II osteochondral lesion of the talus with use of autologous chondrocyte implantation.

thirty minutes in phosphate-buffered solution containing 5% normal rabbit serum to prevent nonspecific bindings. The slides were then incubated with the primary antibodies to type-I or type-II collagens diluted 1:20 in 0.04 M Tris-buffered saline solution (pH 7.6) containing 0.1% Triton X-100 for one hour at room temperature. The slides were washed three times with 0.04 M Tris-buffered saline solution and then were incubated with goat anti-mouse and anti-rabbit immunoglobulins labeled with dextran molecules-alkaline phosphatase at room temperature for thirty minutes. After three washes with Tris-buffered saline solution, the reaction was developed with use of the new fuchsin kit. Negative controls were performed by omitting the primary antibody.

Results

Clinical Outcome

No intraoperative or postoperative complications were observed. The American Orthopaedic Foot and Ankle Society clinical score was a mean (and standard deviation) of 41 ± 9 points preoperatively, 90.5 ± 12 points at twelve months, and 93.2 ± 9 points at a mean of four years (range, three to eight years) ($p < 0.0005$) (Fig. 11).

Radiographic Outcome

Radiographic results at the time of follow-up demonstrated no increase in arthritis and a well-restored talar surface in nearly all patients. In all of the patients who had autologous chondrocyte implantation and microfracture, the magnetic

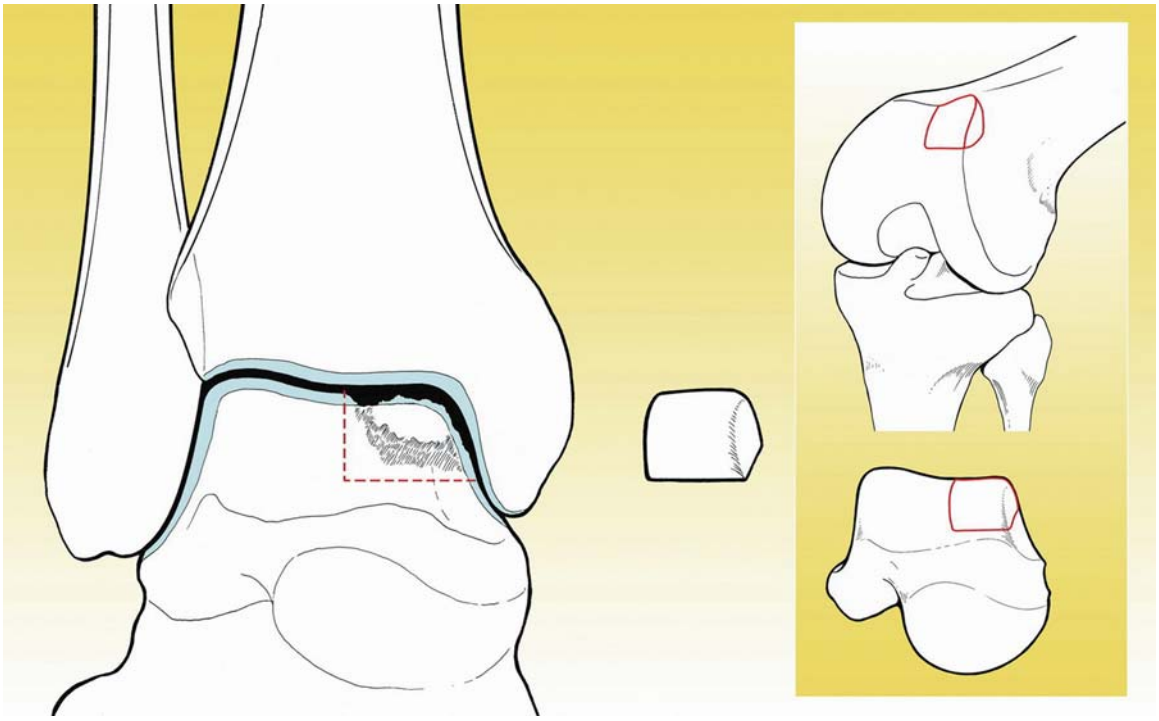

Fig. 10-A
Figs. 10-A through 10-D A chronic type-III lesion. **Fig. 10-A** The osteochondral fragment to be replaced may be obtained either from a non-weight-bearing area of the ipsilateral knee or from a fresh-frozen allograft.

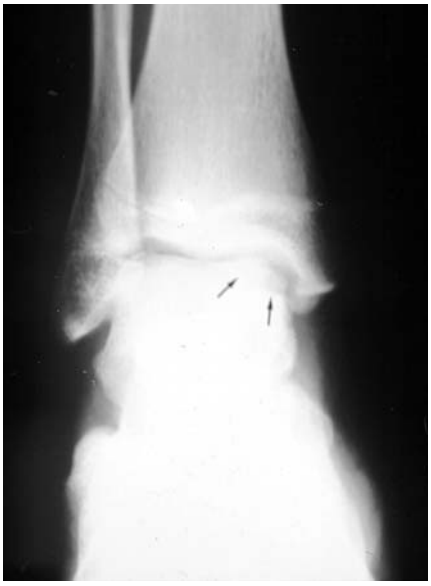

Fig. 10-B

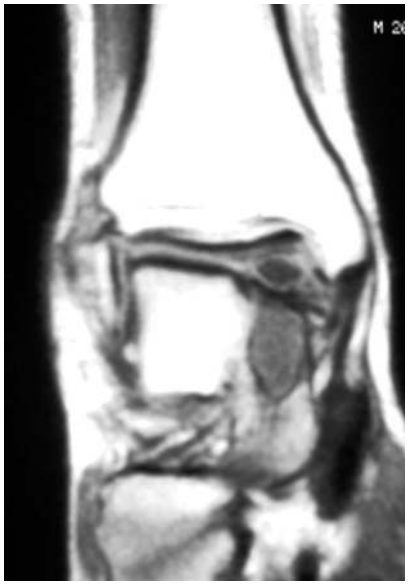

Fig. 10-C

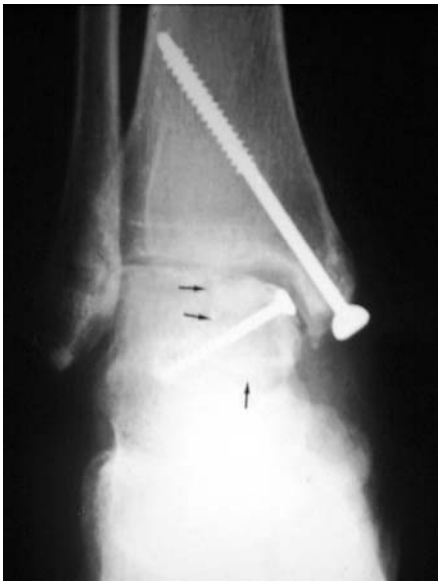

Fig. 10-D

Fig. 10-B The lesion (arrows) was visible on radiographs. **Fig. 10-C** The lesion was also visible on T1-weighted magnetic resonance imaging. **Fig. 10-D** The graft was fixed with a screw (arrows), and the malleolar osteotomy was repaired.

resonance imaging results demonstrated >75% repair of the defect depth, although an edema-like signal in the marrow beneath the cartilage-repair site was often still present even at three to four years.

Histological Findings

The morphological appearance of the newly reconstructed tissues in all of the samples examined showed a "primitive" organization of the structures. In fact, even if all of the components of the hyaline cartilage were present, various degrees of tissue-remodeling were observed. The normal content of glycosaminoglycans was highlighted by safranin-O and alcian-blue staining, which also showed the presence of collagen fibers mainly localized in the superficial zone. Type-II collagen was evident in all of the samples evaluated, and the positive stain was intracellular and mainly confined to the deep zone. An initial attempt at columnarization of the chondrocytes was observed in the deep layers.

Discussion

The appropriate surgical treatment for osteochondral lesions of the talus in young active patients remains a con-

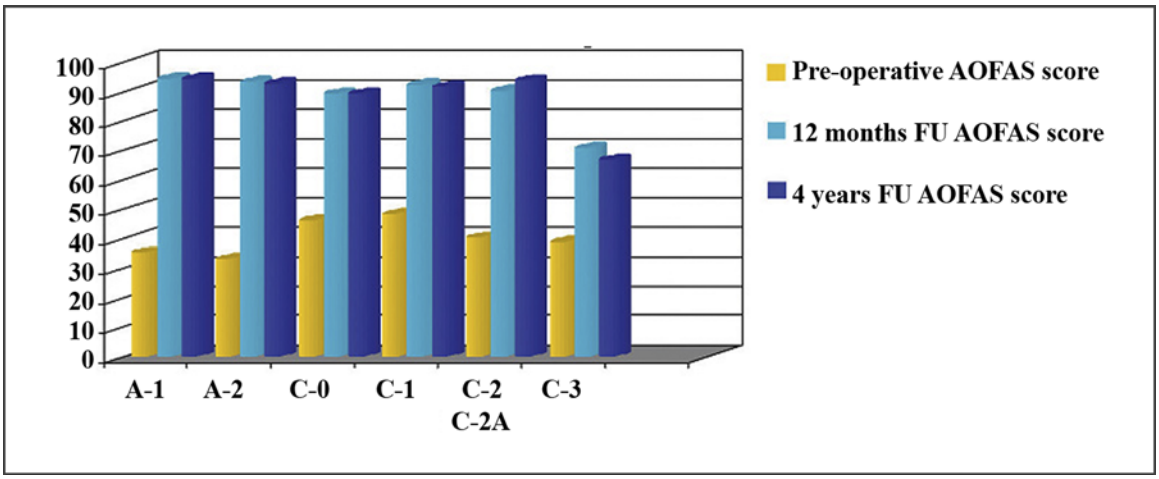

Fig. 11

Graph showing the clinical results. The mean clinical score (and standard deviation) was 41 ± 9 points preoperatively, 90.5 ± 12 points at twelve months, and 93.2 ± 9 points at a mean of four years (range, three to eight years) postoperatively ($p < 0.0005$). A-1 = acute type I, A-2 = acute type II, C-0 = chronic type 0, C-1 = chronic type I, C-2 = chronic type II, and C-3 = chronic type III.

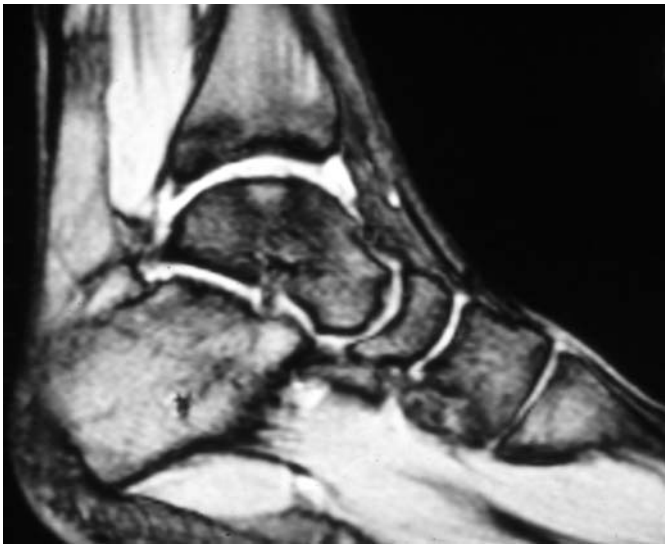

Fig. 12-A

Figs. 12-A through 12-F A twenty-five-year-old woman with a chronic type-II osteochondral lesion of the talus and lower limb malaligment.
Fig. 12-A T1-weighted magnetic resonance imaging scan showing the lesion.

troversial topic. Acute osteochondral lesions of the talus are frequently misdiagnosed. When they are detected, early treatment with débridement of small lesions or fixation of large fragments provides satisfactory and stable results.

Curettage and microfractures in chronic osteochondral lesions of the talus are reported to have good results. However, the newly formed tissue is in fibrocartilage[13-15]. Because of the poor mechanical quality of fibrocartilage, if the lesion is large, the results are prone to deteriorate over time. Therefore, in this series, only small defects were considered for microfracture. The results were satisfactory in >90% of the patients, and only a slight decrease in the clinical score was observed at the four-year follow-up evaluation.

Autologous chondrocyte implantation resulted in effective treatment of large osteochondral lesions of the talus and proved capable of producing new hyaline cartilage[16-19] (Figs. 12-A through 12-F). Arthroscopy and, in particular, histological analysis showed that the area treated with autologous chondrocyte implantation appeared to be covered by hyaline cartilage. The clinical validity of the technique is confirmed by the improvement in the clinical scores over time. At this time, we can state that the evolution in the surgical technique has enabled autologous chondrocyte implantation to be carried out completely arthroscopically. This treatment is fast and effective, it is associated with a very low morbidity rate, and no complications have occurred thus far.

Mosaicplasty has also been described as a reliable technique for cartilage replacement[20,21]. However, the filling of osteochondral defects represents a technical challenge; the dead spaces between the circular grafts and the portion of unfilled le-

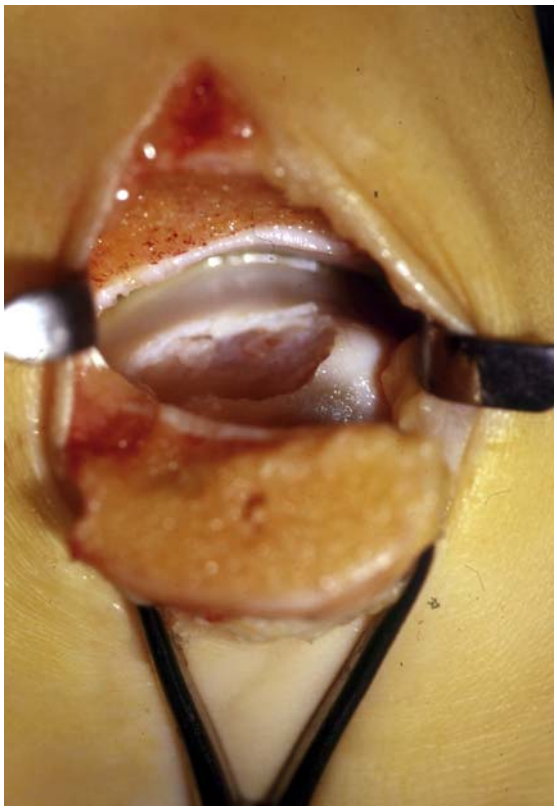

Fig. 12-B

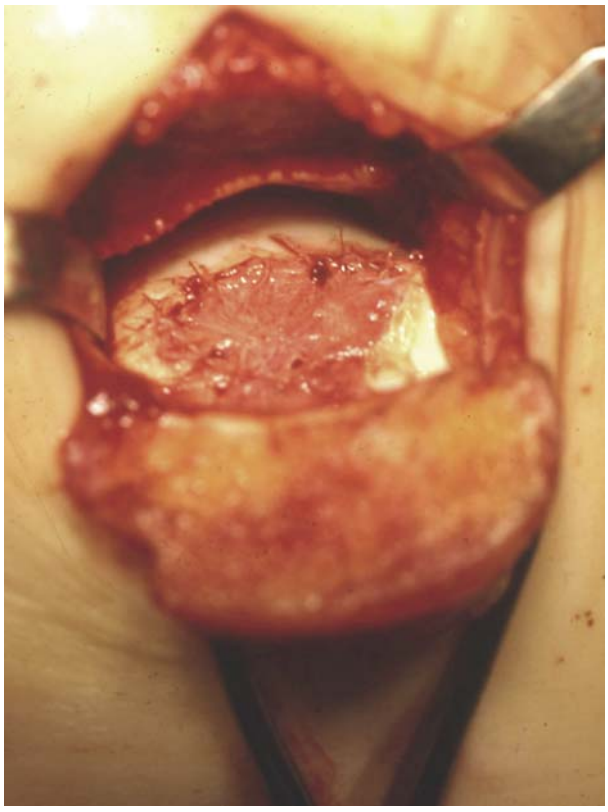

Fig. 12-C

Figs. 12-B and 12-C Autologous chondrocyte implantation (open technique) and a distal tibial osteotomy was performed.

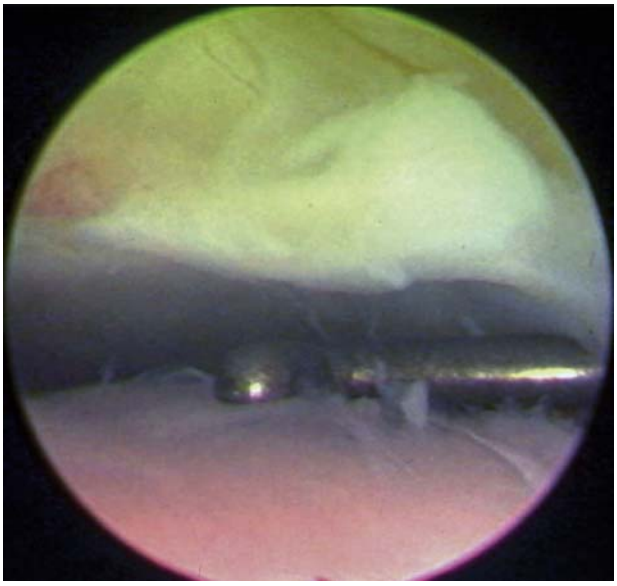

Fig. 12-D

At one year, arthroscopy revealed a good macroscopic appearance of the cartilage surface with good integration of the repaired tissue into the surrounding cartilage.

sions are left to heal as fibrocartilage. Furthermore, complications at the harvest sites can be a drawback of this technique.

Autologous grafts are able to fill some defects, but, in large, extensive shoulder lesions, fresh allograft tissue should be considered the solution when duplication of the anatomy is difficult to achieve with autologous tissue[22].

Osteochondral lesions of the talus were reviewed first, as far as we know, by Berndt and Harty[23], in 1959, and, since then, many authors have proposed modifications of their classification system or new classification systems using more advanced imaging techniques[9,24,25]. However, no system that we know of is able to provide clear indications for surgery considering all of the factors that define these lesions. We believe that the extension and depth of the lesions should be considered the most important parameters in choosing the appropriate surgical option.

The above guidelines have proved to be effective in al-

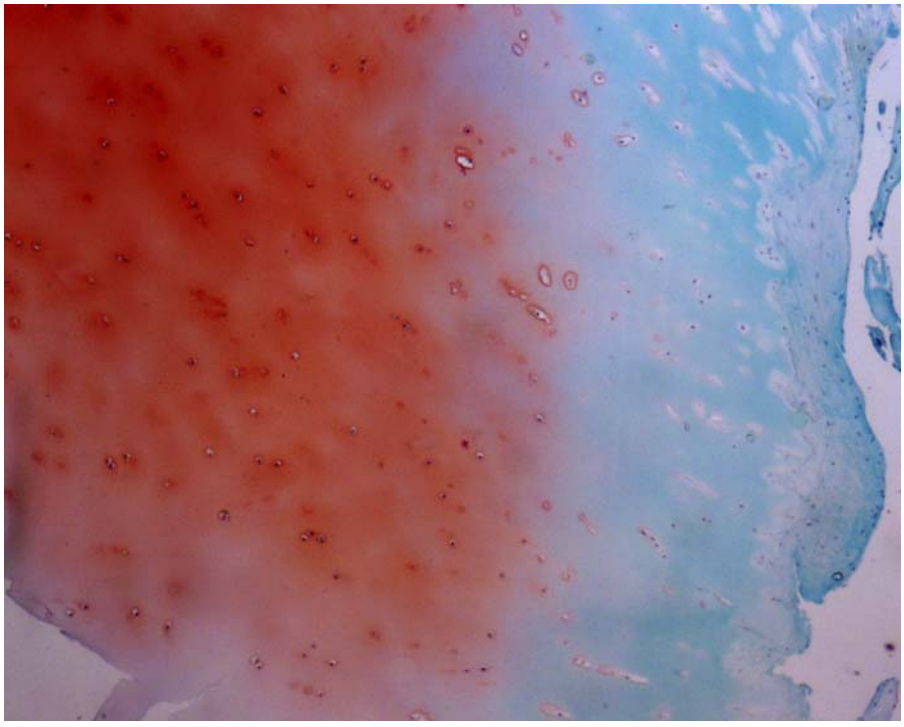

Fig. 12-E

Safranin-O staining demonstrated a typical hyaline-like appearance (×50).

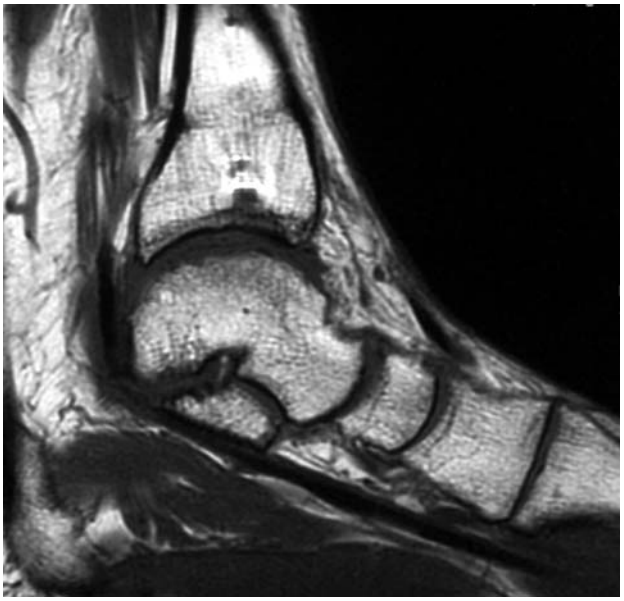

Fig. 12-F

At eight years, excellent results were seen on magnetic resonance imaging and at the clinical evaluation.

lowing the surgeon to choose the proper surgical technique for the treatment of osteochondral lesions of the talus. It is important to appreciate that accurate preoperative evaluation of the etiology and predisposing factors of the lesions were taken into account and treated as well. If left untreated, these factors are likely to cause a recurrence of the osteochondral lesions of the talus.

In conclusion, osteochondral lesions of the talus are a challenging problem. Only with a careful choice of the surgical technique, while considering the pathoanatomy and causative factors, can optimal results be achieved. ■

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