Osteochondral lesions of the talus: Current concept

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Accepted: 20 May 2010

Summary  Osteochondral lesions of the talus (OTL) are among those injuries that we should not fail to recognize, especially following any type of hindfoot injury. They were thoroughly described 15 years ago in a round table session organized by Doré and Rosset for the Société orthopédique de l'Ouest. Their physiopathology has not yet been definitely determined, even though some of the pathogenic mechanisms are known. They are best characterized using the fractures, osteonecroses, geodes (FOG) radiological classification. Both their diagnosis and their surgical treatment remain a challenge to the orthopaedic surgeon: some basic surgical principles apply to all of the lesions, such as cartilage debridement and shaving of necrotic tissues, while others will be used depending on the location and size of the lesions as well as the surgeon's experience. Finally, no specific technique appears to be superior to the others. Arthroscopy appears to be the most effective procedure for lesions smaller than 1 cm², whereas larger lesions should be filled, either with cancellous bone or with an osteochondral graft or using autogenous chondrocyte implantation. The data available in the literature should also incite orthopaedists to consider the results of surgical management with some modesty, and conservative management should remain among the therapeutic options.

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Introduction

The talus, located within the ankle joint complex, faces the tibia above, the calcaneus below, and the navicular bone in front. In this strategic location half way between the leg and the foot, where its essential biomechanical role is to distribute forces, it remains highly exposed to injury because it is subjected to substantial stresses during the standing position, locomotion and physical activities. Among the relatively rare fractures that have been described, osteochondral fractures hold a very particular place in terms of both their diagnosis and their therapeutic management.

Nosology

The breakdown of talus fractures is quite complex because although authors are in agreement on the classical separation between fragmentary and total fractures, a certain number of more or less complex classifications have
lesions of the talus (OLT).

Consequently, fractures described as osteochondral must be sought within the fragmentary fractures. There is a certain nosological confusion because many names have been given to these lesions involving both the cartilage and the subchondral bone: osteochondritis, osteonecrosis, osteochondral lesion or fracture, etc.

For reasons of convention, fragmentary fractures of the talus other than those called subchondral fractures of the dome and therefore fractures of the lateral process (also broken down by Hawkins), the posterior processes, and the head will not be covered in this article.

The updated terminology designated by the Round Table of the Société orthopédique de l’Ouest (SOO) is today’s reference: the term “osteochondritis” has been abandoned; there is no objective inflammation in these lesions, as the ending “-itis” would imply. The term “osteonecrosis” prevails instead. We adopt the convention of osteochondral lesions of the talus (OLT).

Vascularization

This area’s vascularization should be well known to understand the risks of necrosis related to fracture sites, but particularly to guide the surgeon in the choice of surgical approaches. The main, terminal, part of this blood supply penetrates the bone via the dorsal, lateral, and inferior aspects of the neck. However, many anatomic vascular variations exist and are relatively frequent. Three vascular trunks irrigate the talus (Fig. 2):

- posterior tibial artery, medial retromalleolar. It supplies a few posterior branches penetrating the bone in its capsular insertions, anastomosing into a posterior network with the collaterals of the fibular artery. In particular, under the malleolus, it gives rise to the artery of the sinus tarsi, and the main blood supply of the talar body;
- anterior tibial artery, at this level the dorsalis pedis artery. It provides several branches penetrating the neck dorsally and laterally, and more particularly a lateral branch, which, after giving rise to a few branches supplying the head, penetrates the sinus tarsi to anastomose with the artery of the sinus tarsi;
- fibular artery, which gives only a few branches running posteriorly and laterally.

History

In 1738, Monroe [4] was undoubtedly the first to have described the presence of a foreign body in the talocrural joint following an injury. As Doré and Rosset [5] reported, four distinct periods cover the history of these lesions, to which a fifth one must be added.

It was in 1888 that the pathology was discovered, after König [6] had first used the term “osteochondritis disseca ns” to define a lesion involving the cartilage and the subchondral bone to the medial femoral acetabulum. Kapps [7] seems to have been the first to have described the same pathology at the ankle while working with intra-articular foreign bodies. In 1932, in France, Rendu [8] reported a fragmentary intra-articular fracture, then, in 1951, Delahaye [9] defended his thesis on osteochondritis dissecans based on three new observations. At the time, he had found only 58 observations in the world literature.

In 1959, Berndt and Harty published [10] 220 observations and several cadaver experiments and proposed a definition of the disease, including a classification that would be the reference until the SOO Round Table in 1994. For years to come, many authors reported scattered observations referring to Berndt and Harty despite their quite mechanistic and simplistic analysis.

Beginning in 1960, the disease became known and recognized through the publication of large series of patients. Besson and Weelinger were the first to report 12 cases in 1967 [11]. Kouvalchouk et al., and Kouvalchouk and Watin-Auguard must be recognized for having published major research beginning in 1984, emphasizing the improper term “osteochondritis dissecans,” to which they preferred to substitute the term “osteochondral lesions of the talus dome.” They also proposed a specific treatment of curettage and filling with bone material [12,13]. In 1990, Gérard...
et al., published the results of a series of 102 observations [14]. In 1986, Parisien [15] and then Pritsch et al. [16] were the first to describe arthroscopic treatment, followed, in 1988, in France, by Frank et al. [17].

In the 1990s, imaging studies began to explode. The use of standard radiography, computerized tomography, arthrography, and bone scintigraphy did not progress knowledge of the disease, but the development of CT, then rapidly the arthro-CT made it possible to analyze the extent of the subchondral lesions with very high precision, advancing surgical treatment in turn. A little later, MRI also showed itself to be valuable, despite a certain tendency to magnify the lesions, without necessarily providing a fine analysis of the cartilage.

Doré and Rosset, who directed the SOO Round Table on the subject, must be recognized for having provided the description and classification that continues to be the reference today [5].

Classifications

The pathogenesis, physiopathology, and course of OLTs are still being debated. Based on a biomechanical experiment conducted on amputated limbs submitted to forced movements, Berndt and Harty [10] considered the different lesional aspects observed as being only anatomic and evolving variations of an injury lesion (Fig. 3).

By reproducing four types of fragmentary fractures, they concluded that traumatic injury was the cause of all talar dome lesions and described the different lesional stages, which, in their princeps description, only concerns lateral facet locations. This classification was challenged because many radiographic images could not be classified. Certain authors pointed out radiographic differences between traumatic lesions and those cases in which injury was not found in the patient’s history [14,18]. In 1993, a fifth stage was
lesions, which accounted for 73% of cases [19]. The existence of family forms or non-injury forms should bring out other pathogenic hypotheses.

In 1995, Doré and Rosset, studying the SOO series of 169 cases, proposed a new radiological classification called FOG, based on the lesional aspect (fracture [F], osteonecrotic sequestrum [O], geode [G]) and the relation of the lesion with the talus body (situation in relation to the surface, condensation around the fragment) [5]. Three forms can be distinguished:

- the F form (fracture). This isolated fragment is associated with no modification of the bone matrix, condensation, or cyst (Fig. 4). This fracture can be recent or old, with a slightly lytic aspect in the latter case. In the SOO series, these F forms accounted for 18% of the cases and were found on the anterolateral side with a history of injury in 87.5%;
- the O form (osteonecrosis). The aspect here is necrotic and includes a sequestrum (Fig. 5). The underlying bone matrix has been modified with a radiolucent line of condensation associated with microcysts. In the series, this accounted for 75% of the cases. There was no history of injury in 66% of the cases and in seven out of 10, the lesion was located on the medial side of the talus;
- the G form (geode, bone cyst). This form is characterized by the absence of a free fragment or a sequestrum. However, a radiotransparent intraosseous cystic aspect can be noted (Fig. 6) in the body of the bone. In the series, this accounted for only 7% of the cases, which is much rarer than in other series [19]. In these bone cyst forms, the authors saw a particular entity similar to the cystic lesions described by Kouvalchouk et al., and Kouvalchouk and Watin-Augouard. [12,13].

These radiological descriptions do not take into account the condition of the cartilage, yet this condition the therapeutic and prognostic aspects. Therefore, a certain number of additional imaging classifications can be useful. Arthro-CT provides a quite detailed view of the cartilaginous covering as well as the lesional extension of the subchondral bone. Ferkel et al. [20] proposed a four-stage classification:

1. stage I: subchondral cyst but intact joint surface;
2. stage IIa: subchondral cyst with open cartilage;
3. stage IIb: open cartilage, nondisplaced fragment;
4. stage III: subchondral cyst, nondisplaced fragment;
5. stage IV: displaced fragment.

MRI seems less precise in the analysis of the cartilage and the developments of arthro-MRI have not confirmed the hopes placed in this technique at the end of the 1990s. A relatively similar classification to Ferkel’s, based on MRI, was proposed by Anderson and Crichton [21]:

1. stage 1: trabecular compression;
2. stage 2a: subchondral cysts;
3. stage 2b: nondetached fragment;
4. stage 3: nondisplaced fragment;
5. stage 4: displaced fragment.

Finally, in 2003, Mintz and Tashjian [23] published their MRI-arthroscopic classification based on a retrospective study of 54 patients with excellent sensitivity (95%), specificity (100%), NPV (88%) and PPV (100%) for this exam:

1. stage 0: normal cartilage;
2. stage 1: intact cartilage but abnormal MRI signal;
3. stage 2: fibrous cartilaginous fissures or lesions but bone intact;
4. stage 3: cartilaginous flap or subchondral bone exposed;
5. stage 4: free but nondisplaced cartilaginous fragment;
6. stage 5: free displaced fragment.

Etiopathogenesis

Reading the voluminous literature on the subject suffices to understand that a large number of hypotheses have been advanced on the origin of these osteochondral lesions: simply the abundance of names used reflects this misunderstanding.

There are indeed traumatic forms with the fractures attributable to true shearing, as Bernt and Harty [10] showed, but the missteps that followed should not be a reason to throw out the other physiopathological mechanisms that others had formulated before them, i.e., the possibil-

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Figure 3 Bernt and Harty classification, 1959.
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ity of a second type of lesion without the notion of a history of injury, preferentially located on the medial side (Fig. 7) [24]. These necrotic forms suggest other etiologies: vascular or synovial, with or without a microtraumatic context, localized hyperpressure, etc.

A shearing mechanism of osteochondral fracture is recognized in the F forms, the fractures being either new or old depending on the time of diagnosis.

What is the mechanism at play in the other forms? In the type O forms, a cartilaginous fragment with a variable thickness of the avascular necrosed subchondral bone is isolated from the rest of the bone. The cartilage in contact with the synovial fluid remains normal. It can be continuous (no communication between the joint cavity and the groove surrounding the sequestrum), or discontinuous, with all the intermediate possibilities between fissure and a free fragment in the joint.

Repeated injury or microinjuries can result in the individualization of a nondisplaced osteochondral fragment. Isolated from its bony base, it could evolve either toward

Please cite this article in press as: Laffenêtre O. Osteochondral lesions of the talus: Current concept. Orthopaedics & Traumatology: Surgery & Research (2010), doi:10.1016/j.otsr.2010.06.001
pseudarthrosis with the appearance of a sequestrum corresponding to its necrosis or toward fracture union [25]. Transitions from the F-type to the O-type forms argue in favor of this O form injury etiology, in particular in lateral injuries. However, during an inversion injury, the contusion of the superomedial angle of the talus under the tibia can secondarily become the bed of an O lesion, which may not be diagnosed until much later, while the initial injury will have been forgotten.

This hypothesis, like the medial hyperpressure hypothesis, since it does not involve injury causing fracture, seems useful in explaining these locations. Some authors have demonstrated that the center of pressure distribution in the talocrural joint is slightly medialized. These stresses could be the cause of metabolic modifications associated with microfractures, which could end in localized necrosis and the appearance of a type-O fragment or a bone cyst. This theory could explain the medial lesions in the absence of a history of injury and bilateral lesions that may occur on the varus hindfoot. Type-G lesions could also stem from excessive stresses on the medial side of the talar dome, associated with vascular or metabolic modifications, leading to deep necrosis farther from the subchondral bone than in type-O lesions. MRI images after injury with no fracture, where the lesion zone clearly extends very far into the subchondral cancellous bone, are another argument supporting this hypothesis.

The F forms, for the most part lateral, stem from injury, whereas the O and G forms, more often medial, most likely result from another cause. It is probable that the F and O forms are related.

Diagnosis

The diagnosis of an OL T generates few problems. Clinically, the ankle is painful with a succession of totally aspecific signs (pain without the location necessarily related to the lesion location, blocking, click, instability, joint swelling) with most often moderate limitation in joint range of motion.

The notion of injury when questioning the patient is a major part of diagnostic orientation, since these lesions are most often found in young patients engaged in sports. Therefore, with a lateral sprain, this diagnosis will be suggested while taking care not to relate this pain to simple ligament sequelae and to frequent lesional associations.

The major part of the diagnosis is based on complementary imaging examinations.

Plain radiographs remain indispensable, AP images with 20° medial rotation to clearly show the talar dome, and lateral images (Fig. 8). The classical images described are thus often identified, but in case they are negative ones must not hesitate to request further images and add a three-quarter image of the hindfoot.

Although it does not establish the diagnosis, a bone scintigraphy remains a sometimes useful examination in the exploration of unexplained pain in the hindfoot or midfoot.
CT, but particularly arthro-CT, seems to be a choice exam for very precise analysis of the bone matrix and the cartilaginous cover provided by arthrography (Figs. 9, 10, 11). It must be undertaken in millimeter slices with reconstructions and confirm or specify a diagnosis such as a fracture that is difficult to visualize on the x-ray, localize a foreign body, demonstrate a sequestrum or a cyst and evaluate the extension and the depth of necrosis. The existence of a fissure, its depth, and its constancy on the different views are also indirect signs of the fragment’s fixity, an important part of the pretherapy workup [26,27]. However, the arthro-CT, despite its potential superiority, is a sort of French exception: authors elsewhere, except Ferkel, have little experience with it.

MRI is also used for diagnosis, but bone analysis is less precise than with CT (Fig. 12a, b). Both exams provide similar results [28], although MRI can be superior for diagnosis [21] and one can observe a greater surface and depth in medial lesions compared to lateral lesions [29]. Even though its interpretation can be delicate, it has its own advantages in providing information on both the articular and extra-articular components. It can also detect vascular modifications with no underlying fracture and in certain cases distinguish between a recent and an old lesion.

Finally, ultrasound has even been proposed [30] during arthroscopy to assist in the diagnosis of post-traumatic osteochondral lesions or lesions stemming from other etiologies.

In practice, the procedure is a bit different depending on the context. In traumatology, the basic examination is plain x-ray [31], completed if need be with complementary images or a CT scan. In the chronic context of a painful ankle, if the x-ray provides diagnosis and if discussion of surgery seems necessary, the arthro-CT remains the exam that can best analyze the location and extension of a lesion as well as its cartilaginous cover. It can also be useful for intermediate- and long-term follow-up. If on the contrary, the initial x-ray is normal, MRI seems preferable for diagnosis. It can also assess both the articular and extra-articular structures. It can be completed by an arthro-CT if, despite the diagnosis, it remains insufficient in the planning of any surgery that may be necessary.

Progression is totally unpredictable and often no relation exists between the size of a lesion and the intensity of the functional signs. However, general progression can be noted because these lesions, however severe they may be, become the bed of overall ankle osteoarthritis, with the two conditions often coexisting.

**Treatment**

As always, in orthopaedics, treatment includes several possibilities: conservative management, orthopaedic treatment, and surgery in all its forms. Even if surgical treatment of these lesions is relatively specialized and often technically delicate, these are indeed surgical indications that are difficult to determine. Two recent publications invite the surgeons that we are to be extremely cautious in deciding on surgery, with less than 50% good and very good results.
no matter which method is retained. The psychological component of pain, which is the main motive for consultation, should not be neglected. Often, the patient has been suffering for several months, even several years, perhaps having consulted several physicians or specialists, without the underlying problem being identified. Once the diagnosis has been made, the patient must be reassured that this pathology is normally benign, explaining the origin, progression and therapeutic possibilities. The objective of treatment will first be to act on the pain and thereby improve function. Everything that can be done to interrupt the progression of lesions should be undertaken provided that the means implemented do not worsen the local conditions.

Before any decisions are made, initiating treatment requires three essential questions. First of all, what is the patient’s motivation? Then, how severe is the problem? Finally, does it warrant medical or surgical treatment?

The first step is therefore the analysis of the functional repercussions. As Versier et al. [34] proposed, it is entirely possible to use the International Cartilage Repair Society (ICRS) knee classification, modified for the ankle, which integrates the subjective state of the joint evaluated by both the patient and the clinician with information such as physical activity, function, comparison of the two ankles, and the handicap stemming from the different symptoms. Pain is evaluated on a visual analog scale (0 to 10). An overall patient score (I—IV) is given by the surgeon corresponding to the highest score obtained for the items studied.

Conservative management does not mean doing nothing but rather delaying treatment, considering, after rigorous analysis, that the lesion is above any reasonable therapeutic proposal with a success rate that is deemed low. In these cases, a strictly medical treatment is appropriate, possibly combined with other treatments if there is more extensive talocrural osteoarthritis: joint hygiene, weight loss, antalgic and/or anti-inflammatory treatment, and orthotics.

Orthopaedic treatment consists in strict unloading, with or without joint immobilization, of variable lengths of time but less than 4 weeks. The goal is osteocartilaginous healing and is only appropriate in fresh injuries, nondisplaced or only very little, with a potential for true healing depending on their size, location, and patient parameters (age, socioprofessional context, defects, smoking).

Surgical treatment is more complex, with a multitude of options: using different means, the objective of surgery will always be to restore a painless cartilage cover, either by simple excision of the unstable fragments, stimulation of a fibrocartilaginous healing process, or bone, osteochondral, or autogenous chondrocyte implantation. It is not within the scope of this article to present the principles and approaches of the classical and arthroscopic procedures used for this treatment. As for the latter, let us simply note the undeniable contribution of the posterolateral and posteromedial approaches described by van Dijk, making very posterior lesions accessible to treatment [35]. As for the classical procedures, a few comments are necessary. Most lateral lesions are accessible via an anterior, more or less lateralized approach. It is exceptional to have to turn to fibular osteotomy for very posterior lesions. Medial lesions, generally posterior, are only directly accessible after malleolar osteotomy, or tibial osteotomy for certain authors [36,37], which must be undertaken with extreme caution because of their uncertainties (union delay, pseudarthrosis, irreversible arthrogenic cartilage involvement, septic osteoarthritis) [38].

Associated treatments

It is clear that one must look beyond this anatomically-limited lesion and attempt to correct any factors that could influence the joint’s prognosis, in particular axial deviation or laxity in the hindfoot, by whatever medical or surgical measures necessary.
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With this in mind, and before specifying the lesion type (F, O, or G) and what the therapeutic indications may be, the clinician should reply to three questions.

Should weightbearing be suspended? This is certainly true in acute lesions, but not if the signs are truly chronic; immobilization will be recommended even more in cases of fracture than necrosis.

Should the joint be immobilized? Here again, this is certainly true for cases of nondisplaced fracture, but not for a chronic necrotic lesion.

Finally, and most importantly, should the joint be operated? The answer to this question is much less clear, given the quality of the results reported, no matter what techniques are used [32,33]. Although in the 1990s arthroscopic techniques were successfully developed, they seem rather inadequate for lesions exceeding 1 cm². This explains the new therapeutic approaches of auto- and even allograft and cell therapy techniques developed over the last decade to assist reconstruction.

Fractures (F forms) are approached differently depending on the time lapse before diagnosis. In the acute forms, the question of conservative or orthopaedic treatment with immobilization is raised, in relation to fragment size and location, and of course whether or not it is displaced. The alternative is surgical treatment. Sufficiently large fragments can be repositioned and fixed using osteosynthesis (Fig. 13), or even glued [39,40,41]. This treatment can be provided arthroscopically depending on the operator’s experience. Fragments that are too small are simply extracted.

Nondisplaced fractures are immobilized with no weightbearing for at least 4 weeks [5]. The chronic forms can be a challenge because they evolve toward free arthrogenic foreign bodies, most particularly toward authentic O forms via pseudarthrosis and then necrosis. The arthrogram is the choice exam here, guiding treatment depending on the situation and the fragment size. Free fragments are excised arthroscopically as best possible. For those fragments that remain in place, treatment is the same as that described in the next section for osteonecrotic lesions.

Osteonecrosis (O forms) are the most challenging, as already demonstrated by Kellgren and Frank, with only 13% excellent results versus 56% for fractures in a series of 48 cases [39]. Opinions diverge as much on the indications for surgery as on the method and the means to use. Depending on the above-cited patient characteristics, or even the operator’s “arthroscopic dependence” or experience, the situation as well as the size and depth of the lesion will guide the surgeon’s decision. It should be repeated, however, that one must not lose sight of the fact that overall, only half of these patients will have a satisfactory result whatever technique is used, explaining the poor reputation of these osteochondral lesions. One must know how to prescribe conservative treatment, above all if the lesion is located at the top of the dome, a region that is particularly difficult to access (especially in the medial position), if it is very deep or very extensive (well beyond 1 cm²).

If the indication for surgery is retained, a certain number of procedures are proposed, certain systematic such as excision of free fragments, chondroplasty with lesion curettage or wire-guided drilling after excision of the necrotic or unstable tissues, [42] until a “healthy” base can be found (at best, bleeding; Fig. 14 a, b). Others are optional, depending on the situation and the technical choices available: microfracturing, more than Pridie-type perforations, and bone graft filling. Publications before 1998 have been rather favorable for arthroscopy and validated it for lesions smaller than 1 cm² [5,15,16,19,39,43]. After 2000, several authors compared the effectiveness of the different procedures. Although it remains entirely current, microfracturing must be executed very rigorously so that no bone debris is left in the joint [44]. For some, it has provided 93% good and excellent results at 2 years [45], 87% at 5 years [46], but has not been more effective, however, than autogenous bone grafting [47], osteochondral grafting, or chondroplasty [48]. Intra-articular wire-guided drilling, although patients are improved, is followed by deterioration on postoperative MRIs (particularly when the procedure is transmalleolar) and the authors are apprehensive about what the results will be at 10 years [31]. It seems preferable to undertake this procedure via the transtalar extra-articular approach, but this requires intact cartilage, because when it is not intact, not removing unstable fragments gives poor results [49].

Beyond 1 cm², most authors recommend grafting using various methods depending on the type (autografting, allografting, or cellular therapy) or the technique (simple cancellous filling, mosaicplasty-type osteochondral grafting). Some use osteochondral allograft implantation, which seems reserved for rare indications; an adequate infrastructure must be available for this procedure [50,51]. The published results [36,37,52], including those from vascularized bone grafts [53], are listed in Table 1. They are good despite the occasional second-line intervention. The mosaic graft (Fig. 15), more classical, as described by Hangody and Füles, is not straightforward. Despite the technical challenges in applying it to the ankle (the need for osteotomy to expose the medial malleolus or the anterior tibia to implant grafts perpendicularly, harvesting on another joint site), the results of their series of 36 patients are favorable at 2–7 years of follow-up, with 94% good or excellent results with...
Figure 14  A, B. Aspects of a curetted lesion in the healthy zone (bleeding) evaluated using a probe.

Table 1  Results of autograft series.

<table>
<thead>
<tr>
<th>Year</th>
<th>Publication</th>
<th>Number of patients</th>
<th>Approach</th>
<th>Graft</th>
<th>Follow-up (months)</th>
<th>Evaluation</th>
<th>RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Kreuz</td>
<td>2007</td>
<td>Anterior tibial osteotomy</td>
<td>Tibia</td>
<td>60</td>
<td>AOFAS</td>
<td>$p &lt; 0.01$</td>
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<tr>
<td></td>
<td>Tanaka</td>
<td>2006</td>
<td>Medial transmalleolar</td>
<td>Vascularized medial calcaneal</td>
<td>34</td>
<td>AOFAS</td>
<td>$p &lt; 0.001$</td>
</tr>
<tr>
<td></td>
<td>Sammarco</td>
<td>2002</td>
<td>Anterior tibial osteotomy</td>
<td>Lateral or medial talar Face</td>
<td>25</td>
<td>AOFAS</td>
<td>$p &lt; 0.001$</td>
</tr>
<tr>
<td></td>
<td>Al Shaik</td>
<td>2002</td>
<td>Age = 41</td>
<td>Age = 32</td>
<td>19</td>
<td>AOFAS</td>
<td></td>
</tr>
</tbody>
</table>

Several recent publications testify to the current development of cellular therapy and cartilage transplantation [57–61]; the results are compared in Table 2. Like Aurich and Venbrocks [62], we believe that this is undoubtedly a promising alternative for lesions larger than 1 cm² despite the technological requirements and the cost. It is certain that combining this with a purely arthroscopic approach reducing surgical aggression is highly seductive, but it is technically very demanding.

Bone cysts (G forms) are ideally curetted and filled via the transtalar extra-articular approach if they are symptomatic. Once the diagnosis has been made with modern imaging techniques, the challenge remains the evaluation of the true quality of the cartilage. Only arthroscopy can provide this information today. If the cartilaginous cover is indeed intact, arthroscopically guided extra-articular packing-grafting remains the choice solution [63]. Some authors ream the cavity without filling it, but this depends on its size [64]. More or less sophisticated aiming systems have been described for this use. The anterior or posterior approach depends on the site of the lesion and the operator’s habits.

Finally, it is difficult to guide the choice of therapy because of the multitude of possible procedures. Surgeons, even specialized, should not be tempted by the most seductive techniques and should remain modest in their

Figure 15  Intraoperative aspect of an osteocartilaginous graft after raising medial malleolus.

no knee morbidity [54]. Versier et al. presented comparable preliminary results at 18 months, with 86% of the ankles considered normal or nearly so [34]. However, unequal results are found in the literature on this subject [55,56].
Table 2. Results of series of chondrocyte grafts.

<table>
<thead>
<tr>
<th>Year</th>
<th>Publication</th>
<th>Number of patients</th>
<th>Material</th>
<th>1st stage</th>
<th>2nd stage</th>
<th>Follow-up (months)</th>
<th>Evaluation</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year</td>
<td>Publication</td>
<td>Number of patients</td>
<td>Material</td>
<td>1st stage</td>
<td>2nd stage</td>
<td>Follow-up (months)</td>
<td>Evaluation</td>
<td>Results</td>
</tr>
<tr>
<td>2008</td>
<td>Orthopade</td>
<td>9</td>
<td>MACT (hyalograft C)</td>
<td>Ankle arthroscopy</td>
<td>Ankle arthroscopy</td>
<td>48</td>
<td>Hannover Arthroscopy (ICRS score), MRI</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>2008</td>
<td>Am J Sports Med</td>
<td>46</td>
<td>MACT (Hyalograft C)</td>
<td>Ankle arthroscopy</td>
<td>Open (periosteal flap)</td>
<td>36</td>
<td>AOFAS Arthroscopy only first 3 patients (ICRS score - biopsies)</td>
<td>p &lt; 0.0005</td>
</tr>
<tr>
<td>2007</td>
<td>JBJS Am</td>
<td>12</td>
<td>Lab culture</td>
<td>Ankle arthroscopy</td>
<td>Open (± autograft/periosteal flap)</td>
<td>63</td>
<td>Hannover AOFAS/EVA/IRM</td>
<td>p &lt; 0.05</td>
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<td>2005</td>
<td>JBJS Br</td>
<td>10</td>
<td>Lab culture</td>
<td>Knee arthroscopy Ipsilateral</td>
<td>Open (± autograft/periosteal flap)</td>
<td>48</td>
<td>Masur score Arthroscopy 9 patients (biopsies)</td>
<td>9 excellent or good</td>
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<td>2002</td>
<td>Cl Orthop Relat Res</td>
<td>8</td>
<td>Lab culture</td>
<td>Ankle arthroscopy</td>
<td>Open (± autograft/periosteal flap)</td>
<td>17</td>
<td>Score de Finnsen/arthroscopies 3 patients (1 biopsy)/IRM</td>
<td>5 excellent</td>
</tr>
</tbody>
</table>

ambitions. Conservative management very often remains the wisest solution. It can be followed by a decision for surgery. Depending on the type of lesion, the situation, and the patient, if a surgical approach is the treatment retained, two parameters should be considered: the size of the lesion and its stability. The first guides the choice of the appropriate surgical procedure, the second requires debridement and resection of the entire unstable lesion and the use of one of the above-cited techniques (simple shaving, drilling, microfracturing) to stimulate the bone marrow toward a process of fibrocartilaginous healing. On the other hand, if the lesion leaves the cartilage covering intact (which can only be confirmed by arthroscopic palpation), it allows extra-articular filling, including with arthroscopic guidance and
even if the lesion is voluminous as in G forms. Easily reproduced with arthroscopic guidance for lesions that are small in size and depth, these techniques give results that all seem good. They are less so when the lesions are more extensive in surface area and depth. In these cases, the authors recommend bone, osteocartilage, and even autologous chondrocyte grafts. The place of the latter procedure, certainly very promising, is nevertheless difficult to appreciate, since it is only performed by a few highly specialized teams. Figure 16 summarizes the course to follow once the diagnosis has been made for this type of lesion.

Conclusion

Treatment of OLTs begins by early screening of the post-traumatic forms that are particularly numerous in hindfoot traumatology. Knowing how to search for them often means finding them. Doré and Rosset’s FOG classification should be used by everyone. We remain faithful to the arthro-CT more than to MRI, which tends to overestimate lesions in their pretherapy workup. The F and G forms are easier to treat, making early diagnosis all the more important. Managing the O forms continues to be debated today. However that may be, surgeons aspiring to treat these lesions should be skilled in talocural arthroscopy, including the most recent advances. However, despite the hopes raised, this procedure cannot claim to treat every situation, particularly lesions extending over more than 1 cm². In these cases, filling is necessary: cancellous bone for the smallest lesions in favor-able situations (bone loss perfectly surrounded by healthy tissue that only lacks a “roof”), osteocartilaginous material, with awareness of the technical challenges and notably the need to implant grafts perpendicular to the talar dome, and chondrocytes in the most modern form, but whose cost remains prohibitive, thus ruling out its generalization.

Conflict of interest statement

None.

References

Osteochondral lesions of the talus or OLT


