Chapter

Reconstruction with Joint Preservation

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Abstract

The joint injury is a common disorder. Some techniques have been employed to repair the joint or regenerate the cartilage defects with different degrees of success. Four commonly performed techniques to preserve the joint included osteotomies, bone marrow stimulation, cartilage repair, and cartilage regeneration.

Keywords: cartilage, articular/injuries, cartilage, articular/surgery, chondrocytes/transplantation, periosteum/transplantation, treatment outcome

1. Introduction

Musculoskeletal injuries that disrupt the structure and function of diarthrodial joints can cause permanent biomechanical alterations and lead to a more severe, chronic condition. Despite advancements that have been made of restore tissue function and delay the need for joint replacement, there are currently no disease-modifying therapies for osteoarthritis (OA). To reduce the risk of OA, innovative preventive medicine approaches have been developed over the last decade to treat the underlying pathology.

The lesions of the articular cartilage are a common disorder that with the aging of the population its prevalence is increasing. More than 500,000 procedures are performed for the treatment of articular cartilage-related injuries, and many of these procedures are repeated in the same patients. This demonstrates the ineffectiveness of this isolated procedure [1].

Hunter [2] noted that the cartilage, "once destroyed, is not repaired." Fact that keeps current. Some studies have shown an incidence of cartilage lesions greater than 65% in routine arthroscopy [3–6]. No procedure nowadays is reliable for the regeneration of articular cartilage. This is due to the complexity of its structure and functional properties, such as minimizing friction and increasing the contact surface area to decrease wear under load.

Cartilage lesions (9 mm or greater) have been reported to be biomechanically unstable with a high propensity of progression to degenerative osteoarthritis [7, 8]. The main characteristics of the clinical presentation are pain, loss of movement, and alteration of function. Various surgical procedure options can be used for treatment; this will depend on the location, size, and stage [9, 10].

Articular cartilage is composed of chondrocytes (5–10%), water (65–80%), collagen, smaller glycoproteins such as fibronectin and oligomeric cartilage proteins, and large negatively charged hydrophilic proteoglycans (aggrecan, hyaluronan). Four distinct zones are described microscopically.

The superficial zone protects the deeper layers of shear stresses and composes approximately 10–20% of the thickness of the articular cartilage. The main collagen fibers found are type II and IX with a high number of flat chondrocytes. It is the layer that protects and maintains the integrity of the deeper layers, is in direct contact with the synovial fluid, and is responsible for most of the traction properties of the cartilage, which allows it to resist the pure, elastic, and compressive forces imposed by the joint.

The intermediate (transitional) zone provides an anatomic and functional bridge between the superficial and deep zones, accounts for 40–60% of the total cartilage volume, and contains thicker collagen proteoglycans and fibrils. Collagen is organized obliquely, and the chondrocytes are spherical and low density. The compressive forces mainly exhibit resistance.

The deep zone is responsible for providing greater resistance to the compressive forces, since the collagen fibrils are arranged perpendicular to the articular surface. The deep zone contains collagen fibrils of larger diameter in radial arrangement, higher content of proteoglycans, and lower concentration of water. Chondrocytes are typically arranged in columnar orientation, parallel to collagen fibers, and perpendicular to the joint line. It represents approximately 30% of the articular cartilage volume.

The calcified layer plays an integral role in the attachment of the cartilage to the bone, anchoring the collagen fibers from the deep zone to the subchondral bone. There are few cells and the chondrocytes are hypertrophic [9, 11–13].

Several factors are part of the etiology of the chondral or osteochondral lesion; among them are metabolic, such as obesity, alcohol abuse, and diabetes, as well as mechanical factors such as trauma, joint misalignment, and instability [12, 13].

Osteochondral lesions heal by formation of fibrocartilage secondary to the initial inflammatory response. Although mesenchymal cells produce collagen type I and II, the repair is mostly fibrocartilaginous in nature. The orderly structural organization of normal hyaline cartilage is lacking and results in early degradation and fragmentation. However, pure chondral lesions are painless and poorly repaired due to lack of vascularity [9, 14].

Surgical and nonoperative procedures are employed in the treatment of cartilage lesions. The main objective goal is to reduce pain and restore function. Nonsurgical treatments include physical therapy, activity modification, braces and orthoses, weight loss, steroid injections, chondroitin sulfate, and viscosupplementation with hyaluronic preparations [15–20]. The operative treatment aims to improve joint function and congruence as well as prevent osteoarthritic damage in intact areas of cartilage. It may be divided into three techniques commonly performed to preserve the joint including bone marrow stimulation, cartilage repair, and cartilage regeneration.

2. Bone marrow stimulation (BMS) techniques

2.1 Drilling/microfracture/abrasion techniques

Burmann in 1931, Haggart in 1940, and Magnuson in 1941 described joint debridement techniques for the treatment of osteoarthritis. Pridie in 1958 introduced the technique of perforation of the subchondral tissue exposing the vascularization of bone marrow, and later Ficat in 1979 described the spongialization, a resection of the entire subchondral bone plate chondromalacia patellae, with good to excellent results. Steadman suggested that specially designed awls are used to make multiple perforations or "microfractures," into the subchondral bone plate [21–30]. The perforations are made as close together as necessary, but not so close that one breaks into another. Consequently, the microfracture holes are approximately 3–4 mm apart (or three to four holes per square centimeter) [31, 32].

Chondroplasty by abrasion depends on the mechanical stimulation, like burrs, of the joint defect, without penetration of the subchondral bone. Exposure of small blood vessels generates formation in a clot attached to the surface. Fibrous tissue metaplasia occurs for fibrocartilage.

Multiple perforations have the benefit of causing less thermal damage than chondroplasty by abrasion and also leave the subchondral surface more rugged, allowing better adhesion of the blood clots. The penetration of the subchondral bone stimulates the local release of growth factors from the underlying bone. These factors attract and aid the differentiation of mesenchymal stem cells from the bone marrow in chondrocyte-like cells [33, 34].

Patients require a period of 6–8 weeks of non-weight-bearing to allow maturation of the fibrocartilage. Also, according to some authors, continuous passive motion for pain control and better function may be necessary [35].

For better results, some important factors include a body mass index below 30 kg/m^2, age under 40 years, defect less than 4 cm^2, volume of repaired cartilage (defective filling) greater than 66%, and symptoms less than 12 months [35].

The repair tissue may be able to fill the defect, but it lacks the normal histological or biomechanical properties of hyaline cartilage. Therefore, it has a stability inferior to the compressive and shear forces and tends to deteriorate with the time [35–39].

However, in their 2017 study, Frehner et al. concluded that treatment of osteochondral lesion by microfracture cannot be seen as an evidence-based procedure [39].

3. Cartilage replacement techniques

3.1 Chondrocyte autograft transfer and mosaicplasty

The description of the technique using osteochondral autografts for the treatment of joint defects was firstly studied by Pap and Krompecher [40]. Later, Wagner and Muller in Germany used the posterior part of the femoral condyle as an osteochondral autograft [41, 42]. Motions came in the 1990s by Matsusue in Japan and Hangody and colleagues in Hungary [43, 44].

The osteochondral plugs are harvested from non-weight-bearing areas and are transplanted into a small osteochondral defect. A larger lesion is filled in with multiple cylinders; it is also possible to transfer the posterior femoral condyle. Due to multiple cylinders, the gaps between the plugs produce an irregular articular surface.

The main indications for mosaicplasty include the chondral or focal osteochondral lesion in a stable knee, with lesions smaller than 22 mm in diameter and no more than 10 mm in depth.

The main benefits of this technique are that it is a single-stage procedure and there is rapid subchondral bone healing with restoration of native type II hyaline cartilage at the articular surface.

In a series by Hangody et al. with 57 patients and follow-up of more than 3 years, reported 91% good to excellent results with a mosaicplasty [45]. Gudas et al. in a prospective randomized study showed better clinical-functional and MRI results after 3 years for osteochondral transplants than for microfracture surgery [46].

Most of the studies showed good to excellent results in the short and long term, with a greater return to athletic activity when compared to microfracture [47–54].

Major complications of the osteochondral graft include donor site morbidity such as patellofemoral arthritis, fibrocartilage hypertrophy of the donor area, and unsatisfactory filling of the cartilage defect (especially with grafts > 8 mm in diameter) [49, 52, 54–56].

3.2 Osteoperiosteal graft

Another option we have for the treatment of osteochondral lesions is the mosaicplasty technique with bone-periosteum graft of the iliac crest. Its two advantages compared to the conventional technique include the absence of joint morbidity from the donor site defect [54] and that the periosteum (with its pluripotent stem cells) has the potential to differentiate into fibrocartilage [56–63].

3.2.1 Reconstruction with periosteal-cortical graft of the tibial lateral plateau: clinical outcome with an 18-month follow-up case

A 63-year-old woman presented with a bicondylar fracture of the right tibial plateau with extension to the diaphysis. She underwent surgical treatment 40 days after the fracture. It presented great destruction of the lateral articular surface, being reconstructed with the use of periosteal cortical graft of the external iliac board, suture of lateral meniscus, and reinsertion. Fixation of the graft with Kirschner wire and cortical screw is associated with lateral support plate and medial locked plate (**Figures 1–4**).

3.2.2 Cartilage regeneration techniques

3.2.2.1 Osteochondral allograft case 1

A 45-year-old male presented to us with 1 year of posteromedial right ankle pain. He reported pain related to physical activities, with a history of previous trauma, swelling of the joint, no feeling of instability, or joint blockage. Physical examination showed diffuse tenderness of the joint during maximal flexion and areas sensitive to touch in the medial tibiotalar joint line with negative ankle stability test (**Figures 5** and **6**).

The patient did not show good evolution with nonoperative treatment; a mosaicplasty with medial malleolus osteotomy was indicated (**Figures 7–11**).

The patient progressed well and returned the physical activities, including running without pain in the 6 month postoperatively.

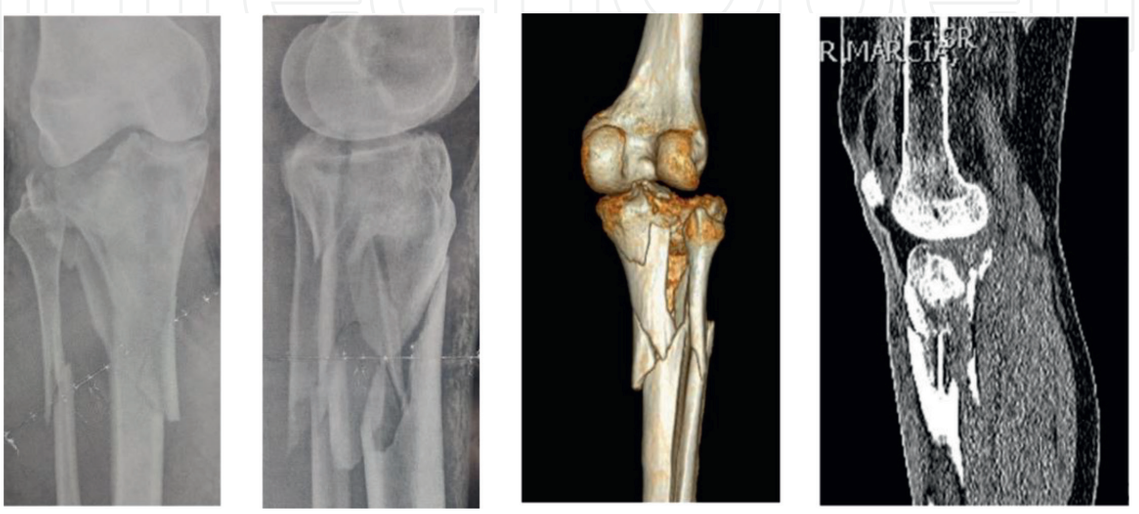

Figure 1.
Image arrival to the service.

DOI: http://dx.doi.org/10.5772/intechopen.84354

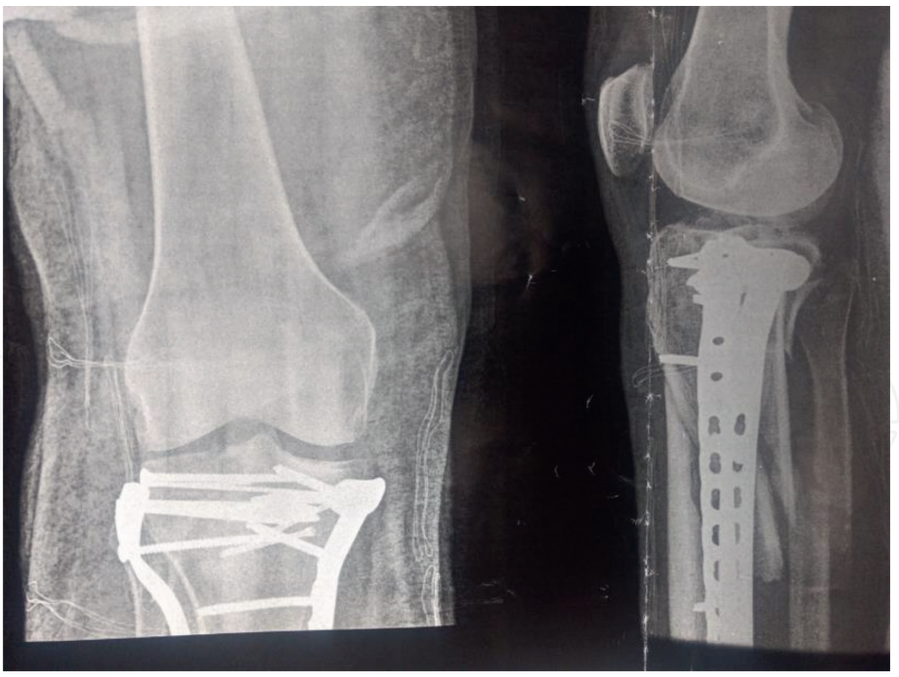

Figure 2.
Immediate postoperative.

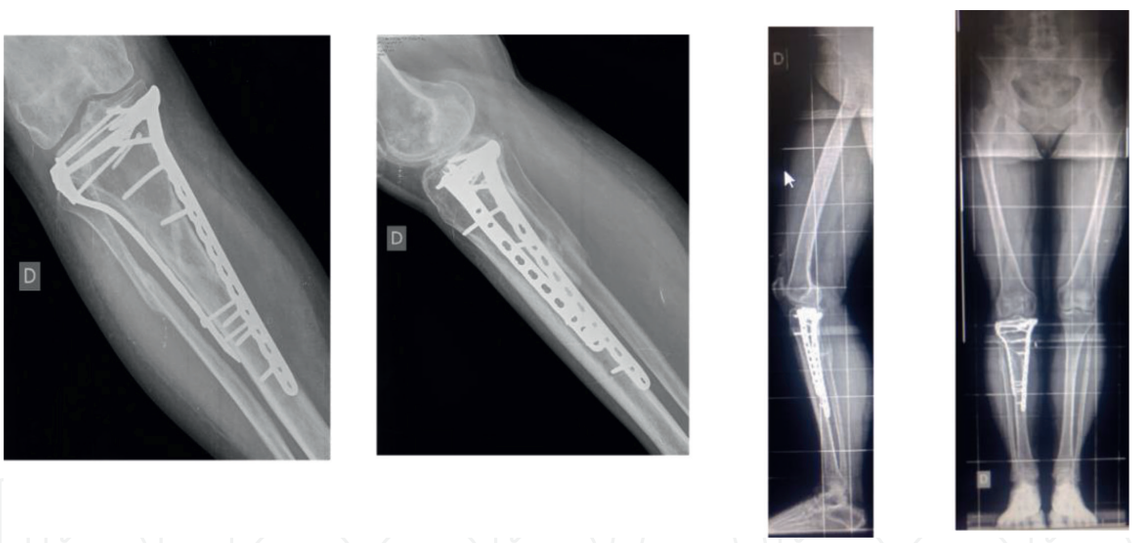

Figure 3.
An 18th month of evolution.

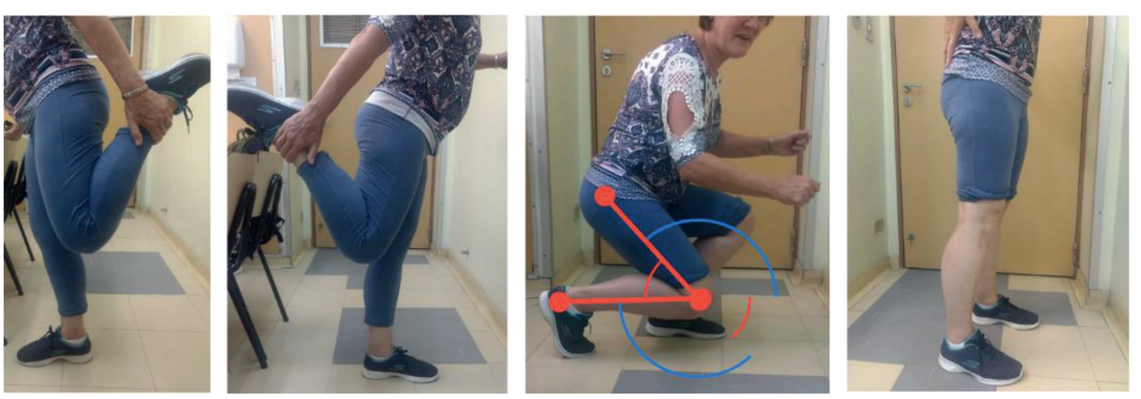

Figure 4.
Final result.

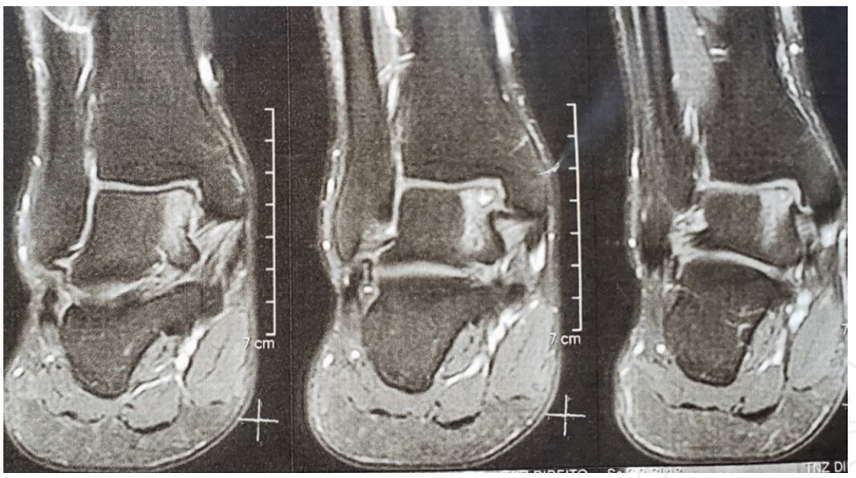

Figure 5.
Osteochondral lesion with cystic formation in the domus talar medialis measuring 1.0 × 0.7 × 0.7 cm, surrounded by area of bone edema. Stage V by Berndt & Harty.

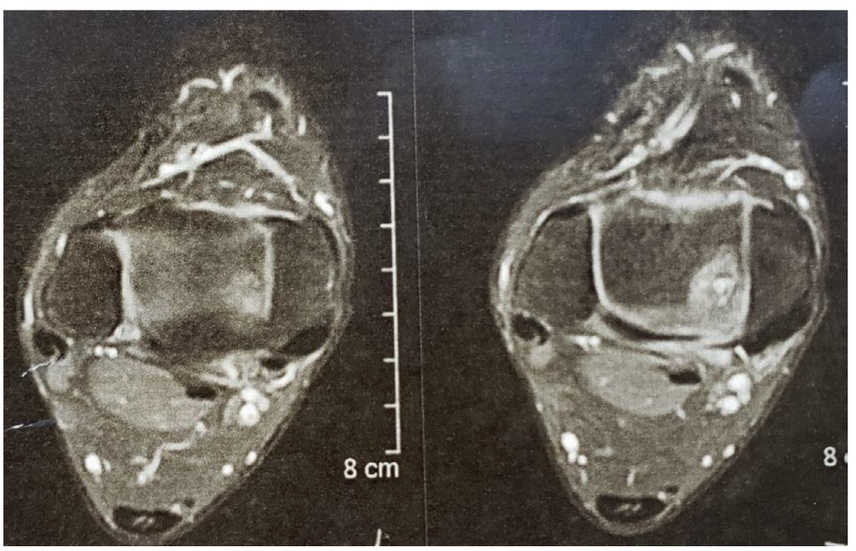

Figure 6.
Osteochondral lesion contained in zone 7 of Raikin.

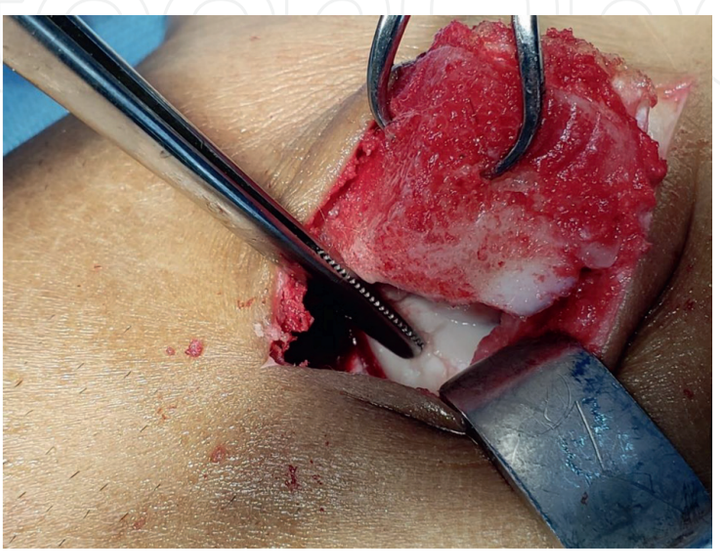

Figure 7.
A osteochondral lesion on the talar medial shoulder after osteotomy of the medial malleolus.

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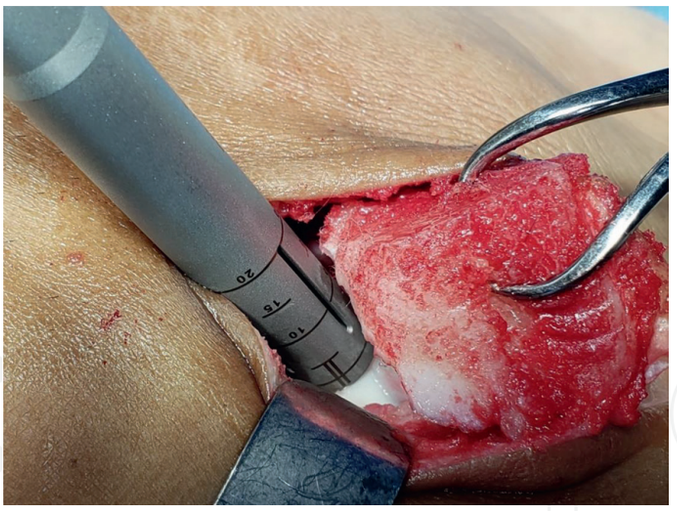

Figure 8.
Intraoperative image of cartilage defect removal.

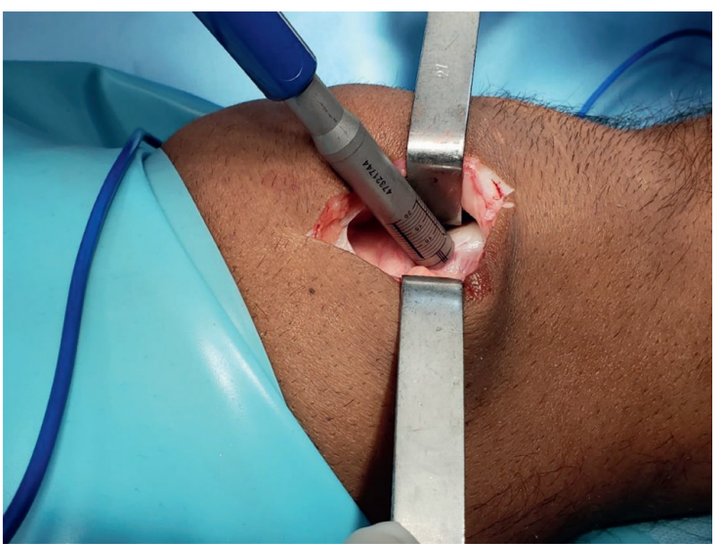

Figure 9.
Removal of the cylinder from the lateral superior region of the femoral trochlea (donor area).

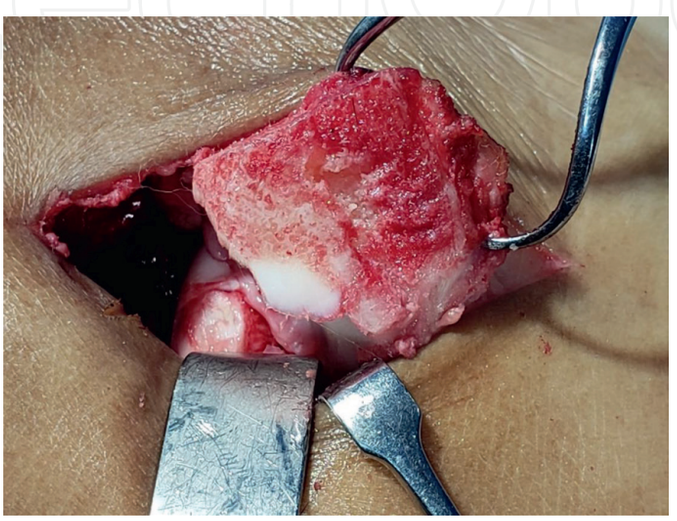

Figure 10.
Osteochondral cylinders inserted perpendicularly to the receiving surface.

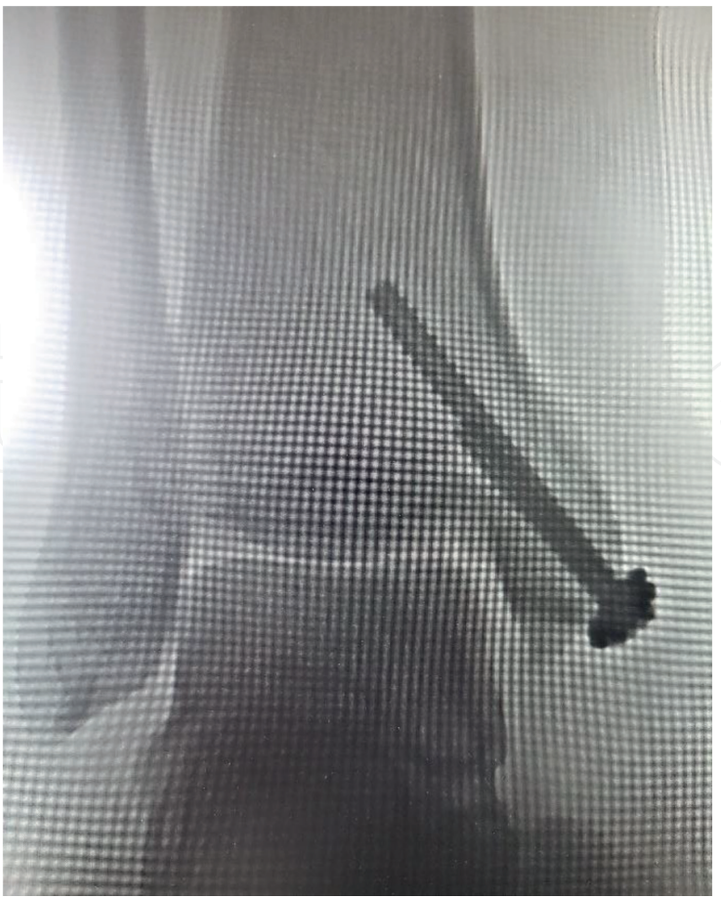

Figure 11.
Postoperative radiography.

4. Cartilage regeneration techniques

4.1 Autologous chondrocyte implantation (ACI)

The technique initially described by Brittberg in 1994 nowadays is the most used for cartilage regeneration [60]. ACI is a two-stage procedure; arthroscopy is initially performed to evaluate the lesion, and three to four CA chondral biopsies are taken from non-weight-bearing surfaces of the joint (intercondylar notch, peripheral edges of the femoral condyles). The sample is then sent to the laboratory, where chondrocytes are isolated with an enzymatic process. The chondrocytes are then cultured for 3–4 weeks until the volume increases by 30 times for implantation (12 million chondrocytes approx.). Usually, after 6 weeks of the initial surgery, the second procedure is performed [59–66].

4.1.1 First generation

Access with medial or lateral patellar arthrotomy is performed in association with defect debridement. A periosteal flap is removed from the proximal region of the tibia or medial femoral condyle. The flap is then attached to the defect (with its cambium layer facing the bone) on all sides, except at the top. The cultured chondrocytes are then injected under the flap, and, finally, the flap is then attached superiorly as well. The fibrin glue can be used to seal the edges of the flap [60].

4.1.2 Second generation

Due to complications arising from calcifications, the periosteum was replaced by a reabsorbable collagen membrane [66].

4.1.3 Third generation

The modification is the cultivation of the articular cells directly on a surface of a membrane-like MACI or cells grown within a scaffold [67].

4.1.4 Surgical technique

This procedure is a two-stage technique in which an arthroscopic approach is performed to evaluate the lesion and second used to harvest a sample of normal articular cartilage from a non-weight-bearing region of the knee. Chondrocytes are then isolated, cultured, and seeded onto a hyaluronan-based scaffold or collagen. The chondrocytes are then cultured for 3–4 weeks until the volume increases by 30 times for implantation, the second stage of the procedure arthrotomy to implant the scaffold in the lesion site. The chondral defect is prepared and is then used to shape the scaffold, which is pressed into the lesion site and secured with a thin layer of fibrin glue. The graft is assessed for stability before the wound is closed.

The best postoperative rehabilitation protocols are those of 6 weeks, starting the first 2 weeks with a partial load of 20% of body weight, followed by a progressive increase to a full load at 6 weeks postoperatively [67–73].

5. Conclusion

The articulations in their particularities refine the movement and enable a series of domains and skills of great importance for the development of human activities.

Thus, there is a growing interest in achieving more promising techniques in joint maintenance through cartilage repair. Some more modern techniques, involving the development and application of stem cells or the use of vectors to carry chondrocytes to the target lesion, still lack more consistent evidence in the long term that can justify their costs. Techniques that employ older, established concepts, such as microfracture and abrasion arthroplasties, are more accessible but fail to maintain their initial results over the years.

In this way, the development of a less invasive technique, aimed at preserving joint functions and minimizing symptoms, with sustainable durability and feasible cost, continues to guide the search for innovations in the arid terrain of joint preservation.

Conflict of interest

The author declares that have no competing interests.

Acronyms and abbreviations

OA	osteoarthritis
ACI	autologous chondrocyte implantation

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