Articular cartilage is prone to damage from acute high-energy trauma and from repetitive shear and torsional forces applied to the superficial surface [1]. Therefore, chondral lesions are a common pathology of the knee joint; they have been found in 60% of knees undergoing arthroscopy [2]. Untreated full-thickness cartilage lesions (grade III or IV according to Outerbridge [3]) are usually associated with significant pain and swelling; furthermore they provoke an increased risk of subsequent osteoarthritis [4-8]. Due to the fact that individuals tend to remain active for longer the need for durable alternatives to arthroplasty increases. Reparative and restorative techniques are available to treat cartilage defects surgically; unfortunately, no treatment option has been identified as the gold standard yet [9,10].

In 2007, 2.7 million knee arthroscopies were performed in the United States and in Europe; they included 1.8 million cartilage procedures, among them 450,000 were Micro Fractures (MF) and 50,000 autologous chondrocyte implantations (ACI) [11]. Reparative Micro Fracture (MF), a single-stage arthroscopic technique developed by Steadman in 1980 [12], results in an intrinsic repair of the chondral lesion. Penetrating the sub-chondral bone plate enables bone marrow cells to migrate into the defect and to create a “super clot” that eventually matures into firm repair tissue. Due to its minimally invasive approach, technical simplicity, limited surgical morbidity and low costs [4] and because it does not prevent the application of other cartilage repair procedures that may be needed in the future [13], it is now a common first-line treatment [14,15] for full-thickness chondral defects of the knee. In 1987, the first ACI was performed by Brittberg [16]. This technique is a two-stage biological approach. In an initial arthroscopy a cartilage biopsy is harvested from a low-load-bearing area on the proximal part of the medial femoral condyle of the affected knee and sent for chondrocyte culturing. During an arthroscopy three to six weeks later the cartilage lesion is debrided. At present, many modifications exist. According to the original technique a watertight chamber is formed into which a suspension [17] with the expanded (characterized [18] or non-characterized) chondrocytes is injected; they start to fill out the lesion by producing a matrix. Finally, autologous periosteum [16-19] or a resorbable bi-layer collagen membrane is used to cover the watertight chamber [20]. To prevent uneven cell distribution and leakage of chondrocytes from the defect [17], the matrix-associated ACI was developed. The chondrocytes are immediately cultured in a three-dimensional biocompatible scaffold (collagen membrane, hyaluronic polymer, collagen gel [21-24]) which contains and stabilizes the chondrocytes; it is trimmed to exactly match the defect before it is implanted. [19,21,25].

However, up to now neither MF nor ACI has proven to be superior to the other in terms of efficacy and safety [9]. Both have shown to relieve symptoms and improve function significantly [26] and both provide better results in the treatment of defects in the femoral condyles rather than in the patellofemoral compartment [27]. Furthermore, younger and more active patients, with a shorter duration of preoperative symptoms, fewer surgical procedures prior to cartilage repair or restoration, and without concomitant ligamentous instability, meniscal deficiency, or femorotibial or patellofemoral malalignment can expect the best outcome regardless of the used technique [28]. Of interest, the situation looks quite different for the defect size. It does not have an impact on ACI [25,29,30], but it significantly influences the outcome after MF. Due to the fact that this technique is based on a sufficient pool formation in the bed of the lesion, the super clot has to be protected by a stable cartilage shoulder [31] which is normally provided by defects smaller than 200 mm² [32]. Whereas defects up to 500 mm² are surrounded by healthy tissue which does not break up under load, defects larger than 500 mm² do not have a mechanical environment that reduces shear and compressive forces [32]. Therefore, Microfracture is not effective for the treatment of large lesions [25,31,33,34]; significantly less improvement was achieved for lesions larger than 400 mm² [35] and 200 mm² [36-38], respectively as compared to smaller lesions.

Furthermore, the quality of the repair tissue plays an important role because good or excellent clinical outcomes are directly correlated with hyaline-like defect fill [39]. Microfracture induces the growth of a repair tissue that consists of type-I, type-II, and type-III collagen [40] and varies from fibrocartilage and hyaline-like cartilage [29,41-43]. Some researchers assume that the original defect fill will become stable and can take over the function of hyaline cartilage [33,35,42], others express the opinion that the hybrid tissue does not support weight bearing in the long-term [38,43] and therefore deteriorates in its function over time; a decrease in score values was observed after 18 [44,45] and 24 [29,34,41,46] months, respectively. In contrast, ACI can restore the integrity of damaged articular areas with hyaline-like cartilage, a hybrid of fibrocartilage and hyaline-like tissue (with chondrocytes organized in isogenic groups and with proteoglycans and glycosaminoglycans in the extracellular matrix) or with fibrocartilaginous material containing type-I and type-II collagen [16,20,39,47-49]. Several studies evaluated that ACI may offer good and stable clinical results up to 11 years [30,39,47,49-51]. Due to the fact that the final matrix remodeling phase starts approximately six months [1] after surgery and continues for two [20,39,52] to three years [1] the regenerated tissue progressively hardens resulting in further functional improvement over time. [30,39].

Undoubtedly, ACI is superior to MF with regard to the quality of the defect fill. Nevertheless, the available literature does not address the necessity for all chondral lesions to be filled with normal stratified hyaline cartilage; the concept of “Demand Matching” [53] (patient goals, compliance, expectations and perceptions as well as physical demands on the knee due to daily routine and recreational activities) should be applied when deciding on the appropriate technique for a particular lesion. Therefore the disadvantages of ACI must not be neglected. ACI is a technically demanding surgical procedure which...
requires an extensive and strenuous rehabilitation; achieving the ultimate clinical benefits may be delayed for at least six to 12 months in some patients [54]. On the contrary, MF involves less surgery, thus the rehabilitation is easier, allowing a faster recovery and enabling athletes a faster return to competition [55,56]. Finally, the cost factor has to be taken into consideration. The costs for ACI are approximately ten times as high as for MF, mainly caused by the chondrocyte cell cultivation. [57].

In conclusion, ACI and MF are complementary procedures. Decision-making has to take individual circumstances and personal preferences as well as patient- and defect-specific factors into consideration. For cost reasons and due to the fact that ACI is suggested to provoke a better outcome than MF only when treating defects larger than 400 mm² [28], MF has to be considered indispensable for smaller lesions. Although, it may be appropriate for larger lesions too, it should not be applied routinely to them because ACI results in a less favorable and less predictable outcome when it is performed as second-line procedure after MF [58]. In larger defects and in high-demand patients ACI should be the treatment of choice because its hyaline-like defect fill will most likely enable potential long-term benefits whereas MF may not adequately relieve symptoms and restore function.

References


