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Cartilage Restoration of the Hip

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The management of articular cartilage defects within the hip represents a challenging problem. Surgical options currently available for the treatment of these injuries remain limited. Although restoration procedures for chondral and osteochondral lesions in the knee have been well published previously, there is a paucity of data available to guide surgeons in the selection, technical performance, and anticipated outcomes of these procedures within the hip. The purpose of this review is to outline the indications, surgical techniques, and outcomes for available cartilage restoration procedures of the hip, including microfracture, osteochondral autograft transfer, and osteochondral allograft transplantation.
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Introduction

Isolated chondral and osteochondral defects within the hip represent a challenge for orthopaedic surgeons. Advancements in both imaging modalities and hip arthroscopy have led to increased recognition of isolated chondral lesions.[1,2] These lesions may arise as sequelae secondary to atraumatic hip pathology such as developmental hip dysplasia, Legg-Calve-Perthes disease, slipped capital femoral epiphysis, avascular necrosis, or osteochondritis dissecans,[1-12] or less commonly secondary to traumatic injury. Osteochondral defects are less common, and are typically seen in association with a traumatic insult to the hip, including acetabular fracture, hip fracture-dislocation, or isolated femoral head fracture.[3,13]

Femoroacetabular impingement (FAI) has increasingly gained recognition as a potential cause of chondral defects and subsequent development of arthritis within the hip.[6] It is reported within the literature that up to 79% of hips with cam impingement have articular cartilage pathology at the time of surgery. The size or extent of these cartilage defects often correlates with the magnitude of associated labral tearing.[14] Furthermore, delamination at the chondrolabral junction is reported to occur in up to 44% of patients undergoing an open surgical hip dislocation, with a large cam lesion being the most common risk factor.[6] In the spectrum of FAI, the pattern of chondral damage is determined by the pathologic shape of the hip and resultant impingement that occurs.[5] At the limits of hip motion, the resulting aspherical femoral head impinges with the anterosuperior acetabulum and results in delamination of the chondrolabral junction.[5]

Articular surface defects can be thought of as occurring along a spectrum of severity, progressing from superficial or partial-thickness disruptions to more complex and full-thickness defects with underlying subchondral bone loss. In general if left untreated, superficial cartilage lesions have a limited capacity to spontaneously heal secondary to the avascularity, lack of progenitor cells, and decreased mitotic activity within mature articular cartilage.[17,18,37] However, full-thickness lesions, which violate the subchondral bone, have been shown to demonstrate healing secondary to subsequent migration of marrow-derived mesenchymal stem cells, formation of an inflammatory "super-clot," and eventual production of reparative cartilage tissue. The resultant cartilage produced is a hybrid mixture consisting mostly of fibrocartilage (type I collagen), with only minute amounts of hyaline cartilage (type II collagen) appreciated microscopically.[15,16] Although both the structure and mechanical properties of this reparative cartilage have been shown to differ from those of

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native hyaline cartilage, its production forms the basis of the microfracture (MF) technique.[18-20]

The long-term outcome of cartilage defects within the hip has been previously shown to result in pain, early secondary degenerative changes, and subsequent development of arthritis.[5,20,21] Although a number of procedures to manage similar lesions in other large joints have been well-described, there currently remains little information available to appropriately guide management in the hip.[22,23] Additionally, the anatomy of the hip creates certain challenges specific to the hip joint itself. Not all cases of chondral or osteochondral defects are amendable to arthroscopy, and in fact most large defects would need to be addressed by surgical dislocation of the hip. Current surgical treatment options available include chondroplasty, microfracture (MF), osteochondral autograft transfer (OAT), osteochondral allograft transplantation (OCA), autologous chondrocyte implantation (ACI), Matrix-induced autologous chondrocyte implantation (MACI), and autologous matrix-induced chondrogenesis (AMIC).[16,24-28]

Indications

The choice of cartilage restoration procedure should be individualized on a case-by-case basis. Selection should be based on lesion-specific factors including defect size, containment, location, and associated subchondral bone loss, as well as consideration of patient-specific factors including age, activity level, and ability to participate fully in postoperative rehabilitation.[1] Most of the surgical indications currently utilized in the selective use of hip cartilage restoration procedures have been extrapolated from data published from similar procedures in the knee.[1] Limitations remain with what can be addressed arthroscopically in the hip, and chondral debridement remains the most common treatment option for partial-thickness chondromalacia.

In the knee, MF is considered for smaller, well-contained lesions that are unipolar and cartilage only. OAT or mosaicplasty is indicated for full-thickness lesions, measuring 4 cm^2 or less that occur in a young, active patient population. Indications for this technique may be extended to similar-size lesions with bone loss due to the presence of subchondral bone within the harvested osteochondral plugs. ACI may be indicated in larger lesions measuring 4 cm^2 or greater, with either intact subchondral bone or minimal bone loss. OCA is indicated in isolated chondral lesions measuring greater than 4 cm^2 or osteochondral lesions with substantial bone loss[1] (Table). In addition to size, another important consideration is the location of the lesion, specifically whether it is acetabular or femoral based. In general, acetabular-sided defects tend to be better tolerated than those on the femoral head.

Patient Evaluation

Every patient evaluation should begin with a detailed history and a thorough physical examination.[1,26] A history of anterior groin pain typically represents true intra-articular hip pathology, but less commonly intra-articular pain may radiate posteriorly over the buttock or laterally over the trochanter. Pain in these later locations must be differentiated from pain secondary to an extra-articular origin, including the lumbar spine or trochanteric bursa. Mechanical symptoms such as clicking or catching are commonly reported in the presence of a labral tear.[26] Standard plain-film radiographs, including an AP pelvis, frog-leg lateral, and cross-table lateral, should be inspected for structural deformities, loose bodies, or signs of impingement, which may be addressed at the time of surgery. Images should be reviewed for the presence of diffuse degenerative change within the hip, which represents a contraindication to performing a chondral restoration procedure.[1,26] Advanced imaging modalities are useful both to confirm the presence of and better delineate the extent of an articular defect. A computed tomography scan of the hip is useful to detail bone loss, but it offers very little in the way of soft tissue detail. Magnetic resonance imaging (MRI) offers improved visualization of soft tissues, early detection of degenerative changes not appreciated on x-ray imaging, and identification of osteonecrosis. However, MRI was previously shown not to accurately and reliably identify chondral or labral lesions.[29] Although addition of gadolinium contrast (MR angiography [MRA]) offers improved visualization of labral tears and isolated chondral lesions compared with conventional MRI, the overall sensitivity and negative predictive value of MRA remain low at only 47% and 59%, respectively (Fig. 1). Keeney et al[30] demonstrated that a negative MRA does not effectively rule out the presence of a chondral lesion.[1,29] However, biochemical-based MRI adjuncts, such as T2 mapping, T1 rho, sodium MRI, and delayed gadolinium-enhanced MRI of cartilage, have been shown to take advantage of changes in the biochemical composition of articular cartilage, and they may prove useful to detect damage in articular cartilage earlier and more reliably than by conventional MRI.[45] Following diagnosis of a chondral lesion, all patients should be further assessed for their ability or willingness to follow the postoperative rehabilitation protocol.[26]

Table Treatment Options

	Depth	Size	Subchondral bone	Bone loss
Chondroplasty	Partial thickness	Variable	Intact, unexposed	None
Microfracture	Full thickness	$\leq$2-3 cm^2	Intact	None
OAT/mosaicplasty	Full thickness	$\leq$4 cm^2	Intact or disrupted	Mild-moderate
OCA	Full thickness	$\geq$4 cm^2	Intact or disrupted	Severe
ACI	Full thickness	$\geq$4 cm^2	Intact	None or minimal
MACI	Full thickness	$\geq$4 cm^2	Intact	None or minimal