Journal of Hip Preservation Surgery Vol. 6, No. 1, pp. 3–15
doi: 10.1093/jhps/hnz002
Advance Access Publication 16 April 2019
Review article

Chondral lesions in the hip: a review of relevant anatomy, imaging and treatment modalities

Alison A. Dallich[1], Ehud Rath[1]*, Ran Atzmon[2], Joshua R. Radparvar[1], Andrea Fontana[3], Zachary Sharfman[4] and Eyal Amar[1]

[1]Division of Orthopaedic Surgery, Tel Aviv Sourasky Medical Center, Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel,
[2]Department of Orthopedics, Assuta Medical Center, Ashdod, Israel,
[3]Orthopaedics Department, C.O.F. Lanzo Hospital, Como, Italy in association with the Orthopaedics Department, University of Pavia, Pavia, Italy and
[4]Department of Orthopedic Surgery, Montefiore Medical Center, The University Hospital for Albert Einstein College of Medicine, USA.
*Correspondence to: E. Rath, Division of Orthopaedic Surgery, Tel Aviv Sourasky Medical Center, Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel. E-mail: udirath@gmail.com
Submitted 24 April 2018; Revised 13 December 2018; revised version accepted 3 February 2019

ABSTRACT

The diagnosis and treatment of chondral lesions in the hip is an ongoing challenge in orthopedics. Chondral lesions are common and several classification systems exist to classify them based on severity, location, radiographic parameters, and potential treatment options. When working up a patient with a potential hip chondral lesion, a complete history, thorough physical exam, and ancillary imaging are necessary. The physical exam is performed with the patient in standing, supine, prone, and lateral positions. Plain film radiographs are indicated as the first line of imaging; however, magnetic resonance arthrogram is currently the gold standard modality for the diagnosis of chondral lesions outside of diagnostic arthroscopy. Multiple treatment modalities to address chondral lesions in the hip exist and new treatment modalities continue to be developed. Currently, chondroplasty, microfracture, cartilage transplants (osteochondral autograft transfer, mosaicplasty, Osteochondral allograft transplantation) and incorporation of orthobiologics (Autologous chondrocyte implantation, Autologous matrix-induced chondrogenesis, Mononuclear concentrate in platelet-rich plasma) are some techniques that have been successfully applied to address chondral pathology in the hip. Further refinement of these modalities and research in novel techniques continues to advance a surgeon's ability to address chondral lesions in the hip joint.

INTRODUCTION

The diagnosis and treatment of chondral lesions of the hip remain a challenge for orthopedic surgeons. Advances in imaging technology, arthroscopic instrumentation, increased fellowship opportunities, as well as insights from basic science and clinical research have resulted in a significant increase in hip arthroscopy procedures in the last decade. These factors have resulted in an increase in the diagnosis and treatment of chondral lesions in the hip [1–3]. The acetabulum is the most common location of chondral lesions in the hip with one study reporting that 88% of chondral defects are found in the anteriorsuperior acetabulum [4]. McCarthy and Lee [5] found that 59% of chondral injuries occurred in the anterior acetabulum and 24% occurred in the superior acetabulum. Chondral lesions are often associated with a labral tear [6], so it is logical that the most common location for labral tears is the anteriorsuperior labrum [7] and correlates well with data regarding chondral lesions.

Chondral lesions generally do not have self-healing properties. Full thickness lesions penetrate into the subchondral bone and do allow for some degree of healing, although the new cartilage that is produced from the migration of bone marrow mesenchymal cells and formation of an inflammatory 'super clot' differs in structure compared with the original cartilage [8]. This cartilaginous tissue is termed fibrocartilage

which contains Type I collagen in addition to the Type II collagen which is ubiquitous in hyaline cartilage [9].

Buckwalter [10] identified three types of chondral and osteochondral injuries based on the severity of tissue damage and repair response. Type I injury is defined as damage to the cartilage matrix not apparent by visual inspection or by clinical imaging methods and possible injury to the subchondral bone which may be visualized by scintigraphy or MRI. Type I injury represents elastic deformation of the cartilage and possible bone marrow edema which may cause pain; however, the basic matrix structure remains intact. The repair response is synthesis of new matrix macromolecules. Type II injury is defined as cartilage disruption limited to the articular cartilage. This represents plastic deformation of cartilage such as chondral fractures or ruptures which may cause mechanical symptoms, joint effusions, synovitis and pain. The repair response also results in synthesis of new matrix macromolecules and cell proliferation; however, there is no fibrin clot formation, no inflammation and no cartilage defect filling by new tissue. The lesion may progress to cartilage degeneration. Type III injury is defined as mechanical disruption of both the articular cartilage and subchondral bone. These injuries may cause mechanical symptoms, joint effusions, synovitis and pain. The body's repair response to cartilage and bone disruption results in fibrin clot formation, inflammation, new cells invading the lesion, and production of new fibrocartilage and osseous tissue. The lesion may progress to cartilage degeneration, which is dependent on the stability and alignment of the joint as well as the location and size of the lesion.

It is thought that chondrocytes possess some ability to repair chondral damage when the tissue is not visibly disrupted, further injury is avoided, and the macromolecular collagen matrix framework is intact. However, chondrocytes cannot produce hyaline cartilage in response to osteochondral fractures nor can they repair chondral fractures or flaps in adults [10]. Chondral lesions may progress secondary to mechanical stresses and poor healing potential which often results in pain and reduced function. This article reviews the literature pertaining to chondral lesion diagnosis and treatment modalities.

ETIOLOGY AND CLASSIFICATION OF CHONDRAL LESIONS IN THE HIP

Chondral lesions in the hip may arise secondary to a variety of pathologies including femoracetabular impingement (FAI), avascular necrosis (AVN), developmental dysplasia (DDH), acetabular osteochondritis dissecans and others [11–13]. These lesions may be associated with traumatic injury such as dislocation of the hip joint, acetabular fracture, femoral head fracture or osteoarthritis (OA) [14, 15].

Several classification systems have been developed to classify chondral pathology. Outerbridge [16], Beck *et al.* [11], and the International Cartilage Repair Society (ICRS) [17] have developed classification systems based on the severity of cartilage disruption, while Konan *et al.* [18] uses an expanded classification system particularly for FAI pathology to include the location of the lesions as defined by the six acetabular zones proposed by Ilizaliturri *et al.* [19] (Fig. 1) and the size of the lesion (Table I). Sampson [20] established a classification system specific to cartilage lesions of the femoral head and one specific to acetabular lesions cartilage lesions, with recommended treatment protocols based on these classifications (Table II).

CLINICAL ASSESSMENT

The evaluation of chondral lesions in the hip includes a complete history, a thorough physical examination, and ancillary imaging. The role of ancillary laboratory tests may also play a role in the work up of chondral lesions depending on history and physical examination.

Chondral lesions in the hip rarely exist as isolated pathology and most often present secondary to conditions such as labral tears, FAI, DDH, OA, loose bodies, traumatic injury osteonecrosis, osteochondritis, or others. It is important to assess athletic participation, previous trauma, childhood hip pathology, past surgical interventions and various other factors which may influence possible treatments.

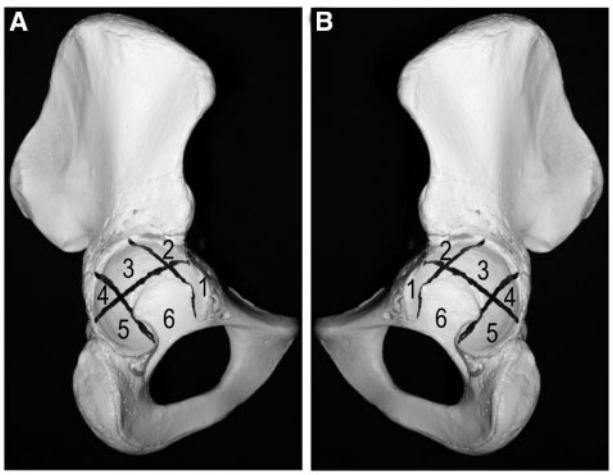

Fig. 1. (A, B) Ilizaliturri's [19] six acetabular zones (Zone 1: anterior-inferior acetabulum, Zone 2: anterior-superior, Zone 3: central superior, Zone 4: posterior-superior, Zone 5: posterior-inferior, Zone 6: acetabular notch) for the right (A) and left (B) hip. Reproduced with permission from Ilizaliturri et al. [19].

Table I. Chondral lesion classification systems [11, 16–19]

Classification	*Grade*	*Description*
Outerbridge	0	Normal
	1	Softening and swelling of the cartilage
	2	Partial-thickness, diameter <0.5 inch
	3	Partial-thickness, diameter >0.5 inch
	4	Full thickness lesion down to subchondral bone
Beck	0	Normal
	1	Malacia
	2	Debonding
	3	Cleavage
	4	Full thickness lesion
ICRS	0	Normal
	1	Nearly normal: superficial lesion
	2	Abnormal: lesions <50% of cartilage depth
	3	Severely abnormal: lesions >50% of cartilage depth
	4	Severely abnormal: lesions through subchondral bone
Konan	0	Normal
	1	Wave sign
	2	Cleavage tear
	3	Delamination
	4	Exposed bone in acetabulum
		Acetabular Zones (Ilizaliturri *et al.*)
		1 (anterior inferior)
		2 (anterior superior)
		3 (middle superior)
		4 (posterior superior)
		5 (posterior inferior)
		6 (middle inferior, cotyloid fossa)
		Size
		A (<1/3 the distance from the acetabular rim to the cotyloid fossa)
		B (1/3 to 2/3 this same distance)
		C (>2/3 this same distance)
Konan Final Classifications		
		Zone-(1–6) Grade-1 (A, B or C)
		Zone-(1–6) Grade-2
		Zone-(1–6) Grade-3 (A, B or C)
		Zone-(1–6) Grade-4 (A, B or C)

A thorough physical examination includes tests in the standing, supine, prone and lateral positions. Observation of passive and active range of motion, positions which illicit pain, and tenderness to palpation are often very informative. Mechanical symptoms such as clicking, catching or popping may be noted throughout the exam. Gait analysis and limb length discrepancies are also assessed. Specific tests should be guided by history. Tests for cartilage lesions or for hip pain damage may include the log roll test, axial loading of the hip joint, dial test, apprehension testing, impingement testing, the hip flexor contraction test (Thomas), the Stinchfield test (straight leg raising against resistance), the flexion, abduction, external rotation test, the flexion, adduction, internal rotation test, and the dynamic internal rotatory test [20]. The relevant history and physical examination of the hip is well outlined by Dr Martin and Dr Palmer in their valuable paper [21]. Examination of the spine to rule out referred pain may also be indicated.

Ancillary imaging studies most often begin with plain radiographs because they are cheap, available, and valuable to evaluate bony morphology. Attention is paid to joint

Table II. Sampson [20] classification system with treatment guidelines

		Description	*Recommended Treatment*
Femoral head	HC 0	No damage	Little to no treatment
	HC 0T	Uniform thinning (T)	Little to no treatment
	HC 1	Softening	Little to no treatment
	HC 2	Fibrillation	Debridement
	HC 3	Exposed bone in acetabulum	
	HC 4	Any delamination	Debridement and microfracture
	HTD	Traumatic defect (size in mm)	Excision of loose fragment
	HDZ	Demarcation zone from FAI	Treat the Cam bump
Acetabulum	AC 0	No damage	Little to no treatment
	AC 1	Softening no wave sign	Little to no treatment
	AC 1w	Softening with wave sign intact labrocartilage junction	Microfracture and suture
	AC 1wTj	Softening with wave sign and torn labrocartilage junction	Microfracture and suture
	AC 1wD	Softening with wave sign and intact labrocartilage junction with delamination	Elevation of cartilage defect, microfracture, trimmed if necessary
	AC 2	Fibrillation	Debrided or excised to the bone (add microfracture)
	AC 2Tj	Fibrillation with torn labrocartilage junction	Rim trimming, labral refixation, debridement
	AC 3	Exposed bone small area <1 cm^2	<1 cm^2 of exposed bone: debridement
	AC 4	Exposed bone larger area >1 cm^2	>1 cm^2 of exposed bone: microfracture

A, acetabulum; C, cartilage defects; D, with delamination; DZ, demarcation zone from FAI; HC, femoral head cartilage; T, thinning; TD, traumatic defect; Tj, Torn labrocartilage junction, w, with wave sign.

space narrowing and the presence of subchondral sclerosis, subchondral cysts, and marginal osteophytes or other stigmata of OA. Direct imaging of cartilage has proven difficult with CT or MRI. Nishii *et al.* [22] found a sensitivity and specificity of 67–82% in CT arthrography for diagnosing chondral lesions. MRI is considered to be more sensitive for soft tissue imaging, although poor visualization of chondral and labral lesions has been demonstrated [23]. Smith *et al.* [24] found a pooled sensitivity of 0.59 (95% CI: 0.49–0.07) and specificity of 0.94 (95% CI: 0.90–0.97) for detection of chondral injuries by MRI. MRI does however, allow for early detection of osteonecrosis and degenerative changes [25].

MR arthrography, a process in which a contrast medium is injected by use of fluoroscopic guidance before obtaining the MR image, is another method of imaging hip joint pathologies. In a meta-analysis conducted by Naraghi and White [26], 12 studies, with a range in sensitivity from 22 to 92% and a range in specificity from 25 to 100%, examined the accuracy of accessing chondral lesions of the hip by MR arthrography. In comparing MRI and MR arthrography, Byrd and Jones [27] reported that MR arthrography had higher sensitivity but lower sensitivity, while Sutter *et al.* [28] reported that MR arthrography had higher sensitivity with two evaluators, but specificity was higher and lower with the respective evaluators. MRI was found to be

superior to MR arthrography in the diagnostic accuracy of chondral lesions based on the conclusions of Smith *et al.* [24].

The modality of choice for imaging and diagnosing chondral lesions is magnetic resonance arthrogram (MRA) [29]. Gadolinium contrast MRA offers improved visualization of chondral lesions compared with conventional MRI. Studies comparing MRA to either arthroscopic findings [30], open surgery [4] or combined approaches [24] found a sensitivity of 47–79% and specificity from 77 to 89%. However, with a negative predictive value of 59%, MRA cannot rule out chondral lesions [30].

Delayed Gadolinium Enhanced Magnetic Resonance Imaging of Cartilage (dGEMRIC) consists of an intravenous injection of double negatively charged contrast agent or single negatively charged contrast and intra-articular contrast to penetrate the cartilage [31, 32]. A review by Zilkens *et al.* [33] of dGEMRIC in different pathologies found that this imaging modality was sensitive to cartilage defects occurring in early OA. Bulat *et al.* [34] suggests that dGEMRIC has potential for diagnosis of FAI, and found that planar dGEMRIC maps improved the correlation of dGEMRIC and MRI-based Outerbridge and Beck grading in the anterior-central sub-region. Lattanzi *et al.* [35] retrospectively reviewed 20 hips and found that dGEMRIC maps were accurate in detecting cartilage damage in FAI with 52, 67 and 58% sensitivity, specificity and accuracy respectively, and the acetabular cartilage which is compared with morphologic assessment of 37, 90 and 56% for sensitivity, specificity, and accuracy respectively. Hesper *et al.* [36] suggests that T2 mapping may work as well as dGEMRIC when diagnosing defects at the chondrolabral junction based on their study of 31 patients with FAI. Fernquest *et al.* [37] conducted scans of 24 healthy hips and 10 hips at high risk of developing OA with both T2 and dGEMRIC imaging, and found these methods are similar in sensitivity to detect early cartilage disease, although T2 mapping does not require the use of a nephrotoxic contrast agent and has shorter scan times than dGEMRIC scanning.

Biomarkers are another potential tool for clinical assessment of hip pathologies related to cartilage defects. A study by Bedi *et al.* [38] compared the Cartilage Oligomeric Matrix Protein (COMP) and C-reactive protein (CRP) levels in plasma of 29 male athletes, 10 with radiographic evidence of FAI and 19 without and found that the cartilage degradation marker and inflammation marker were increased in the FAI males by 24 and 276%, respectively. In addition, the study found that those with FAI had corresponding deceased scores in the Short Form-12 physical/mental component score (22%) and all subscale scores in the Hip Disability and Osteoarthritis Outcome Score (HOOS) survey. Fibronectin-aggrecan complex (FAC), a cytokine and cartilage breakdown product measured in the synovial fluid, had higher concentration in those undergoing microfracture without radiographic evidence of OA ($P > 0.05$) [39]. These early studies suggest that increased COMP, CRP, and FAC levels may suggest cartilage breakdown and assist in identifying cartilage lesions.

TREATMENT MODALITIES

Treatment modalities for chondral lesions in the hip range from local measures such as debridement (chondroplasty) or microfracture, to cartilage transplants (Osteochondral autograft transfer [OAT], mosaicplasty, Osteochondral allograft [OCA] transplant) and the incorporation of orthobiologics (Autologous chondrocyte implantation [ACI], Autologous matrix-induced chondrogenesis [AMIC], Mononuclear concentrate [MCC] in platelet-rich plasma [PRP] matrix, expanded mesenchymal stem cells [MSCs]). The pros and cons of each of these treatment modalities are summarized in Table III. Oliver-Welsh *et al.* [40] created a general treatment algorithm for articular cartilage defects that first takes into consideration criteria for surgery such as pain, dysfunction, concomitant pathologies, as well as the lesion size and patient activity. The algorithm then suggests second line therapies, and is summarized in Table IV. El Bitar *et al.* [41] created separate algorithms to guide treatment for femoral and acetabular cartilage defects respectively, which are combined and summarized in Table V. By following these algorithms and weighing the risks, benefits and alternatives of operative intervention to potential risk factors, and individual patient factors that may affect outcomes, an informed treatment can be tailored to each individual patient.

Chondroplasty

Chondroplasty, or debridement, is a procedure in which areas with partial-thickness lesions or a loose flap are smoothed [29]. Chondroplasty is targeted at reducing unstable chondral flaps to clean edges, preventing the development of loose bodies and removing potential mechanical blocks in the joint [29, 42, 43].

A retrospective insurance database study of 1728 hip arthroscopy procedures demonstrated that chondroplasty was the most common procedure performed and was utilized in ~49.3% of cases [1]. If chondroplasty is performed during hip arthroscopy there is increased odds of conversion to total hip replacement in patients of all ages (<50 years, OR: 2.7, $P = 0.01$; >50 years, OR: 3.6, $P < .001$) [23]; however chondroplasty is preferentially performed in patients with preexisting arthritis and is not the cause of

Table III. Summary of treatment procedures, indications, contraindications and notes for chondral lesions in the hip

Procedure	*Indications*	*Contraindications*	*Comments*
Chondroplasty (Debridement)	Low-grade, partial-thickness lesions		Radiofrequency ablation should not be done
Microfracture	Lesions < 2–4 cm^2	Partial-thickness chondral defects or underlying bony pathology	Take into account the patient's age, activity level, and adherence to post-operative rehabilitation plan
ACI	Lesions too large for microfracture alone		Potential for serious complications if hip dislocation necessary
AMIC	Grades 3 and 4 acetabular chondral defects, 2–4 cm^2, patients ages 18–55		
MCC in a PRP Matrix	Used in conjunction with microfracture		
Intra-articular injections of expanded MSCs	Diffuse chondral damage, mild OA, patients seeking a non-arthroplasty treatment		
OAT	Lesions too large for microfracture, subchondral damage, microfracture or abrasion chondroplasty have failed	Patients older than 50 years of age, signs of OA	Potential for serious complications if hip dislocation necessary
Mosaicplasty	Multiple smaller lesions on the femoral head		Hip dislocation complications possible
OCA transplant	Young patients with AVN and segmental collapse of the femoral head		Systemic steroids risk for failure in the procedure, Hip dislocation complications possible.
Fibrin adhesive	Delaminated, viable cartilage (wave or carpet sign).		Suture repair and scaffold implantation lasts longer than fibrin glue alone.

ACI, autologous chondrocyte implantation; AMIC, autologous matrix-induced chondrogenesis; MCC in PRP, mononuclear concentrate in a platelet-rich plasma matrix; MSCs, matrix expanded mesenchymal stem cells; OA, osteoarthritis; OAT, osteochondral autograft transfer; OCA, osteochondral allograft transplant; AVN, avascular necrosis.

advanced arthritis warranting total hip arthroscopy (THA). Furthermore, Yen and Kocher [23] have shown chondroplasty to be successful in low-grade, partial-thickness lesions. The use of radiofrequency ablation devices around chondral tissue should be avoided as they have been shown to damage chondrocytes [44].

Microfracture

Microfracture utilizes the body's healing potential and stem cells found in bone marrow to initiate cartilage growth. Cartilage is first debrided and the calcified layer of bone is removed. Then, holes are drilled or tapped in subchondral bone ~3 mm in width and spread 3 mm apart at

Table IV. Summary of treatment algorithm created by Oliver-Welsh [40] for articular cartilage defects

	Lesion Size	
	<2–3 cm²	*≥2–3 cm²*
First line treatment	Low physical demand: Chondroplasty Microfracture (with or without orthobiologics, e.g. MCC in PRP) High physical demand: Chondroplasty Microfracture (with or without orthobiologics, e.g. MCC in PRP) Allograft surface treatment OAT	Low physical demand: Chondroplasty Microfracture (with or without orthobiologics, e.g. MCC in PRP) Allograft surface treatment OCA ACI High physical demand: Allograft surface treatment OCA ACI
Second line treatment	Allograft surface treatment OAT or OCA ACI	Allograft surface treatment OCA ACI

ACI, autologous chondrocyte implantation; MCC in PRP, mononuclear concentrate in a platelet-rich plasma matrix; OAT, osteochondral autograft transfer; OCA, osteochondral allograft transplant.

Table V. Treatment algorithms created by El Bitar et al. [41] based on the size of full thickness femoral head lesions and acetabular lesions in patients presenting with symptoms

Lesion size	*<2 cm²*	*2–6 cm²*	*6–8 cm²*	*>8 cm²*
Treatment	First line: Microfracture (FH, A) Suture repair (FH) Second line: Mosaicplasty (FH) OCA transplantation (FH)	Microfracture (FH, A) Osteochondral allograft transplantation (FH)	Total hip arthroplasty (FH, A) Osteochondral allograft transplantation (FH)	Total hip arthroplasty (FH, A)

A, acetabulum; FH, femoral head.

a depth that allows the for excursion of marrow and for clot formation, containing multipotent MSCs and growth factors which promote the growth of fibrocartilage [45].

A systematic review by MacDonald *et al.* [46] looked at the indications for microfracture in the hip in which 10 studies listed lesions with Outerbridge Grades II, III and IV and 3 studies designated the size of the defect as <4 cm. Some investigators performed microfracture only when the defect was at the chondrolabral junction, and in another study, microfracture was only performed in weight-bearing areas. Lesions that respond best are smaller than 2–4 cm^2 in size [47]. Patient with partial-thickness chondral defects or underlying bony defects are not good candidates for microfracture [48]. Other factors such as age, activity level, and adherence to the post-operative rehabilitation protocol are also taken into consideration when deciding if a patient is a good candidate for this procedure [45].

In a study of 20 patients undergoing microfracture, 19 had a mean fill of 96 ± 7% of primarily fibrocartilage with some Type II collagen near the bone, and one patient had 25% fill with poor quality repair tissue at an average follow up of 17 months [49]. Fill percentage was measured by a graduated arthroscopic probe, and the quality of cartilage was assessed visually. The follow-up hip arthroscopy was indicated in these patients because of persistent or further symptoms after initial improvement following microfracture. Bedard et al. [1] found that 5% of 1577 hip arthroscopy patients converted to total hip replacement within 4 years, which suggests that some patients may not have been ideal candidates for hip arthroscopy and that when hip arthroscopy fails; it does so in a short time frame.

Domb *et al.* [50] compared 54 patients with labral tears, FAI, or both who completed hip arthroscopy with microfracture with those who did not undergo microfracture and found that there was no statistically significant difference in clinical outcomes after two years in the two populations. This study found that patient-report outcome measures were improved in both groups.

In a study of 10-year outcomes following hip arthroscopy for FAI, Menge *et al.* [51] found acetabular microfracture to be independently associated with an increased hazard rate for THA. The authors explained that requiring microfracture treatment is indicative of greater injury at the chondrolabral junction.

Advantages of microfracture include that it is low cost and it is not especially technically challenging [29]. Risks of microfracture include ossification, fragility, imperfection of the new tissue [52, 53], failure to fill the lesion, and new cartilage is prone to breakdown. Microfracture has not been as successful in patients who have advanced Tonnis Grade III arthritis [54]. Chen *et al.* [55] compared osteochondral characteristics in a mature rabbit model 24 h after either microfracture or microdrilling and confirmed that microfracture produced fractured and compacted bone around holes, while micro drilling cleanly removed bone from holes. Micro drilling to a depth of 6 mm allowed for open channels to marrow stroma, while microfracture to a depth of 2 mm sealed off the holes from bone marrow and potentially impeded repair.

Autologous chondrocyte implantation

ACI is a modality which was designed to improve the results of microfracture, especially for chondral lesions that are too large for microfracture [29]. ACI is a two-stage process in which first, the chondral defect is harvested and the damaged cartilage is cleared in a manner similar to microfracture; and second, the previously harvested chondrocytes which have been mixed with a bioabsorbable matrix are implanted back into the cleared defect [29]. Biodegradable scaffolds such as matrix-induced chondrocyte implantation allow for ACI through arthroscopy; however, inserting a Type 1/3 bilayer collagen patch, which is a patch seeded with MSCs, requires hip dislocation [29]. Hip disarticulation carries with it serious risks such as AVN and collapse of the femoral head [14].

Korsmeier *et al.* [56] conducted a study of 16 hips with acetabular chondral defects secondary to FAI that were treated by ACI. The authors reported excellent results as measured by improved mobility, reduced pain and sports performance in eight patients, very good results in four patients, good results in three patients, and one patient reported fair results.

Al-Qarni *et al.* [57] used a product called BST-CarGel for ACI with the theory that the graft would support and stabilize the clot while restoring the 3D architecture of the femoral head. In a prospective study of 13 hips treated with ACI using a BST-CarGel graft, the authors found that mean hip outcome scores increased in a statistically significant manor from 64.4 to 87.4 (daily life activities subscore) and from 35.2 to 75.2 (sports subscore). Furthermore, filling of chondral defects was reported at >90% [58].

Al-Qarni *et al.* [57] lists several advantages of ACI for chondral defects. The technique is minimally invasive and allows for stabilization of the microfracture clot in a joint that is difficult to immobilize or to avoid bearing weight. Other proposed advantages of this technique include prolonging the effect of tissue factors derived from marrow and increasing hyaline cartilage percentage as compared with microfracture alone. Risks of ACI include nerve injury from traction during the procedure, resulting cartilage may be fragile and hip instability, subluxation or dislocation which may warrant hip abduction bracing postoperatively depending on the operative approach to the hip [57].

Autologous matrix-induced chondrogenesis

AMIC is a single step procedure for the treatment of Grade 3 and 4 acetabular chondral defects, 2–4 cm^2 wide, in patients 18–55 years old. This technique combines microfracture to the application of a Type I/III collagen matrix (Chondro-Giade: Geistlich Pharma AG, Wolhusen, Switzerland). The matrix is arthroscopically inserted into the joint to cover the defect and to stabilize the blood clot arising from microfracture, therefore providing infrastructure for repair tissue formation. An accurate chondrectomy creating very sharp edges, the concavity of the acetabulum and pressure exerted by the femoral head, are proposed to give the implant sufficient stability.

A recent publication demonstrated that AMIC leads to improved outcomes at 5 years follow-up as compared with microfracture [59]. In a comparative study, Mancini and Fontana [60] demonstrated that ACI and AMIC both provided marked clinical improvement in patients with acetabular chondral defects secondary to FAI, without significant differences between the two groups at 5 years follow up. Both studies established the therapeutic effectiveness of the AMIC technique in terms of clinical results, minimal invasiveness and time and costs abatements.

MCC in a PRP matrix

MCC in a PRP matrix is a treatment modality used in conjunction with microfracture. In this procedure, as described by Mardones and Larrain [47], autologous bone marrow stem cells are harvested and centrifuged to form a concentrate. Simultaneously, peripheral blood is taken, centrifuged, and activated with autologous thrombin. The chondral surface is prepared with debridement of unstable cartilage and microfracture holes are drilled. The PRP clot is then placed over the microfractured area.

Despite varying results from an assortment of studies addressing PRP therapies in a variety of pathologies, a common finding was that PRP has an anti-inflammatory and procoagulant effect [47]. The PRP clot has shown to result in complete filling of chondral lesions with nearly normal hyaline cartilage in an ovine model [61]. Preliminary results in 13 patients demonstrated that symptoms and scores (Average hip outcome, Vail Hip and Modified Harris Hip Scores) improved at 3 and 6 months, and dGEMRIC imaging at 6 months demonstrated complete defect fill in four patients [47]. Larger numbers and longer follow-up times are required to thoroughly examine the efficacy of this treatment in chondral lesions of the hip.

Intra-articular injections of expanded MSCs

Hernigou *et al.* [62] described the intra-articular injection of expanded MSCs for the treatment of AVN of the femoral head. Bone marrow aspirate is collected from the anterior iliac crest, and the mononuclear cell fraction is isolated. Following core decompression, the cell suspension is injected into the defect under fluoroscopy. It is thought that the MSCs act as a 'homing signal' at the injury site [63] and respond to signals which stimulate differentiation into chondral tissue [64].

As of 2015, seven patients had inter-articular injections of MSC for mild OA, and all patients showed improved symptoms over an average of 10 months, without conversion to total hip replacement at the time of reporting [47]. Mardones *et al.* [65] found statistically significant (P = 0.0001–0.15) evidence that infusing the cell product in a cohort of 10 patients in three consecutive weekly doses led to further improvement in hip function, pain and range of motion, and radiographic scores (Tonnis Classification of Osteoarthritis) remained constant in all but one patient. Mardones *et al.* [65] reported no major complications or side effects in their study. The authors advocate for this treatment in patients who either have diffuse chondral damage or mild OA, and are currently treating symptoms with painkillers but are seeking a non-arthroplasty treatment [47]. Risk and pitfalls of the procedure include long prep time for stem cells, the complex nature of the process and donor site morbidity.

OAT and mosaicplasty

Osteochondral autograft transplantation (OAT) and mosaicplasty are procedures in which osteochondral plugs are harvested from a non-weight bearing surface in the body and are inserted into a chondral lesion which was prepared by drilling a hole similar to the size of the lesion [29]. The difference between OAT and mosaicplasty has to do with the size and number of lesions being treated. Mosaicplasty treats multiple smaller lesions and is typically performed with the hip dislocated. OAT is typically utilized for lesions that are too large for microfracture, with subchondral deficiency or damage, or when microfracture or abrasion chondroplasty have failed [29]. OAT may be performed via an arthroscopic approach depending on lesion location.

In mosaicplasty, the osteochondral plugs must be the correct size and number to match the lesion and are preferentially taken from peripheral, anterior non-weight bearing zones of the femoral head or the ipsilateral knee [66]. A review by Logan *et al.* [42] found that mosaicplasty is utilized to treat defects of the femoral head and has not been routinely performed on acetabular cartilage. Girard *et al.* [67] treated femoral head cartilage damage with mosaicplasty and found increased range of motion and increased Postel Merle d'Aubigné score from 10.5 to 15.5 points after a mean follow up of 29.2 months in their cohort of 10 patients.

OAT can be performed arthroscopically if the defect is located in the anterior aspect of the femoral head, but an open arthroscopic retrograde approach is used if the defect is located in the central or posterior area [68]. Patients older than 50 years of age or with signs of OA are not good candidates for OAT [42]. Gagala *et al.* [15] conducted a study in which 20 patients (21 hips) with osteonecrosis of the femoral head were treated with OAT and 13 patients were treated with OAT and morcellized bone allografts. HSS scores improved from 42 to 87.5 over an average of 46.14 months in the OAT only group and HSS

scores improved from 35.2 to 65.7 after 32.7 months in the OAT/allograft group.

Mosaicplasty and OAT are low cost, single-step surgeries, which use a patient's native hyaline cartilage [29]. The disadvantages of these procedures include potential problems with the harvest site [29]. Furthermore, the contour of the cartilage in the harvested area may not match that of the lesion. Complications associated with hip dislocation also apply to this procedure. Hangody *et al.* [69] found that 3% of patients experienced donor site morbidity after these procedures.

OCA transplant

OCA transplantation is a surgical procedure where allograft bone plugs are harvested and transferred from a cadaveric donor to the recipient's chondral defect once it has been inspected and debrided to uniform edges [70]. The size of the defect must be measured, and a guide pin is used to determine the depth of the defect. Once the graft is placed, press-fit placement alone or various fixation methods can be used.

Krych *et al.* [71] found that after OCA the graft fully incorporated into the cartilage at 18 months without progression of arthritic changes on MRI at 24 months. A study by Meyers consisting of 21 patients receiving 25 osteochondral allografts reported an 80% success rate in patients not using systemic steroids. This cohort was compared with patients on systemic steroids which were found to be a risk factor for OCA failure [72]. The study found OCA to be specifically successful in young patients with AVN and segmental collapse of the femoral head. Oladeji *et al.* [73] followed 10 patients who underwent OCA in the femoral head and/or acetabulum for an average of 1.4 years. Seven patients had successful outcomes as measured by HOOS outcomes. Remaining 3 patients converted to THA 5–29 months after OCA. The authors concluded that OCA can be effective in young, healthy individuals, but risk factors for unsuccessful outcomes include smoking, acetabular involvement, AVN and concomitant procedures.

The benefits of OCA include the use of hyaline cartilage and the lack of donor-site morbidity [70]. Using CT to guide donor tissue matching may assist in finding donor tissues which match recipient anatomy. Timing donation and implantation remains a challenge in OCA, as the time from harvest to transplant must be <28 days. The supply of donor tissue is limited and finding appropriate matches remain challenges of OCA in addition to the risk of disease transmission [70]. Other disadvantages include the cost, potential non-union of the transplant and failure of transplant transformation to live tissue.

Fibrin adhesive

Fibrin adhesive products are used to seal delaminated cartilage, allowing hyaline cartilage to be reattached to subchondral bone [74]. The indication for fibrin adhesive is usually a wave sign or carpet sign [74], and 1 cm of delamination minimum from the chondrolabral junction is recommended in order to complete the procedure [29]. The fibrin adhesive is also thought to act as a scaffold for cartilage regrowth while recruiting MSCs, helping the release of growth factors, and stimulating fibroblast differentiation [75]. The fibrin adhesive is often used in conjunction with microfracture where indicated [29].

Tzaveas and Villar [75] used fibrin adhesives in 19 consecutive patients who had chondral delamination and demonstrated improved pain and function scores measured at 6 months and 1 year. However, ten patients in this cohort required further interventions, including steroids and local anesthetic injections, revision hip arthroscopy, resurfacing arthroplasty and excised cartilage, though the authors reported that the adhesives were still in good condition when examined during these procedures.

A study by Stafford *et al.* [74] followed 43 patients with FAI who underwent treatment with fibrin adhesives and found a statistically significant improvement in the modified Harris hip score at a mean of 28 months post-surgically (21.8 pre-operatively to 35.8 post-operatively, $P < 0.0001$) and a smaller improvement in function (40.0 pre-operatively to 43.6 post-operatively, $P = 0.0006$).

Cassar-Gheiti *et al.* [76] compared four chondral repair techniques in a cadaver model. The authors created chondral flaps in 24 cadavers, treating six hips each with either fibrin glue, cyanoacrylate, a suture technique or an agarose hydrogel scaffold sealed with fibrin glue. They found that fibrin glue on its own was the easiest technique to perform but that fixation failed at 50 gait cycles in a validated jig, whereas suture repair and scaffold implantation lasted 1500 cycles.

SUMMARY

Location, severity and comorbidity or causation of the lesion, as well as patient history and examination, ancillary imaging, and biomarkers are all taken into consideration when discussing treatment modalities for chondral lesions in the hip. There are number of overlapping classification systems to grade lesions, while MRA is the gold-standard for imaging lesions. More research into biomarkers such as COMP, CRP and FAC may help identify lesions earlier, and improved diagnostics assist physicians choose between instrumental modification of the lesion (chondroplasty and microfracture), cartilage transplants (OAT, mosaicplasty

and OCA), or incorporation of orthobiologics (AIC, AMIC, MCC in PRP, MSC). Further research is needed to better diagnose and treat chondral lesions with the end goal of hip preservation.

CONFLICT OF INTEREST STATEMENT

None declared.

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