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Adhesions in the setting of hip arthroscopy

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- With the growing number of primary arthroscopies performed, patients requiring revision hip arthroscopies for various issues is high including postoperative adhesion formation, a source of pain, mechanical symptoms, range of motion limitation, stiffness, and microinstability.
- Adhesions are a consequence of biological pathways that have been stimulated by injury or surgical interventions leading to an increased healing response.
- Preventative efforts have included surgical adjuncts during/after primary hip arthroscopy, biologic augmentation, and postoperative rehabilitation.
- Treatment options for adhesion formation includes surgical lysis of adhesions with or without placement of biologic membranes aimed at inhibiting adhesion reformation as well as systemic medications to further reduce the risk.
- Postoperative rehabilitation exercises have also been demonstrated to prevent adhesions as a result of hip arthroscopy. Ongoing clinical trials are further investigating pathways and prevention of adhesion formation.

Keywords

- hip pain
- arthrofibrosis
- adhesions
- stiffness
- hip arthroscopy

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Introduction

Hip arthroscopy, as a surgical technique, has advanced greatly over the years with the treatment of intraarticular pathology such as femoroacetabular impingement (FAI), chondral lesions, loose bodies, synovial abnormalities, labral tears, and extra-articular tendon pathologies. FAI results from bony incongruity between the femoral head (cam impingement), the acetabulum (pincer impingement), or both (mixed impingement). When conservative treatments fail, hip arthroscopy has become a popular treatment modality used to relieve symptoms such as pain, range of motion limitation, and/or microinstability. Unfortunately, revision rates after hip arthroscopy are reported as high as 13.2% and occur for a variety of reasons (1). One common reason for revision is postoperative scarring between the capsule and the labrum or a postoperative adhesion (2, 3). Histologically, adhesions are characterized as an excessive accumulation of fibrous connective tissue, which in itself consists of the extracellular matrix components of collagen and fibronectin. This tissue is typically deposited in and around damaged or inflamed tissue which in a joint space results primarily in stiffness and pain (4, 5).

Fibrosis may not be the only culprit contributing to the previously stated symptoms after hip arthroscopy. It is also important to consider other diagnosis such as untreated cartilage lesion, capsular defects, severely deficient labrum, and persistent or unaddressed ligamentum teres abnormality (6). If a patient presents with any of these abnormalities, then conservative efforts may be used to restore range of motion for the joint (4). These therapies may include physical therapy, anti-inflammatories, or intraarticular injections (4). If pain persist despite conservative modalities, then a revision hip arthroscopy may be warranted.

Locks *et al.* (7) reported on revision hip arthroscopy after labral reconstruction using an iliotibial band autograft. Patients who underwent revision hip arthroscopy after a previous labral augmentation using iliotibial band autograft from 2006 to 2014 were included in the study. The authors report that out of 347 patients, 28 hips (26 patients – 18 females and 8 males) had revision hip arthroscopy after a previous labral reconstruction. The mean age and follow-up time were 32 and 3.6 years, respectively. After revision arthroscopy, it was seen that four patients had undergone total hip arthroplasty and two required subsequent revision arthroscopy. The authors concluded that patients who

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underwent revision surgeries after labral reconstruction were mostly female, with adhesions and residual FAI as the most common findings during revision hip arthroscopy.

Evaluation

A definitive diagnosis and etiology of pain, stiffness, or discomfort after a failed hip arthroscopy is not always clear and obvious. Evaluation starts with a thorough history and physical exam. Unfortunately, most provocative maneuvers on the physical exam such as the flexion, adduction, and internal rotation test (FADIR) or the flexion, abduction, and external rotation test (FABER) can indicate an intraarticular source but not definitely indicate the specific pathological cause such as recurrent impingement, microinstability, labral retear, or adhesions. Imaging in the form of a computerized tomography (CT) scan may therefore be useful for diagnostic and therapeutic planning especially when open surgery or revision arthroscopy is being considered (1). This is mostly to determine the appropriateness of bony resection, any residual impingement, or over-resection. Magnetic resonance imaging (MRI) is especially helpful in the potential revision arthroscopy setting because of its ability to evaluate cartilage and soft tissue. MRI may be useful in assessing the labrum, capsule, the presence or absence of effusion, avascular necrosis, and the status of the articular cartilage. Furthermore, the use of intraarticular contrast improves the sensitivity and specificity for evaluating capsular volume/defects, labral tears, and postoperative adhesions (1). When comparing MRI to magnetic resonance arthrogram (MRA), the latter was found to have improved sensitivity at detecting various lesions and a rate of false-negative results at 8% as compared to 42% in MRI (8).

Another critical factor to consider when evaluating a patient with persistent pain following hip arthroscopy is the type of capsulotomy performed. Because a T-capsulotomy creates a more extensive disruption to the capsular tissue than a periportal or interportal capsulotomy, there is a theoretically increased risk of additional postoperative bleeding and potential adhesions, but this has not been born out in clinical studies. Furthermore, it is important to know if the capsule was excised or closed which can also play a role in the formation of adhesions but a capsular defect can also be the origin of the patient's persistent pain and dysfunction. The dial test, where the examiner allows the supine patient's feet to fall to a resting position and assesses for a difference in resting external rotation, can be used, in addition to the MRA, to evaluate for symptomatic capsular deficiency.

Clinical data

Despite a significant contributor to postoperative morbidity and reoperation, there is a paucity of literature reporting on the incidence, prevention, pathophysiology, and treatment options relating to adhesions. Table 1 summarizes the current literature on the prevention and treatment of adhesions (9, 10, 11, 12, 13, 14, 15, 16, 17).

Adhesions after a hip arthroscopy has been performed for FAI can form in two separate locations and may produce different clinical symptoms. The first location is between the capsule and the labrum which can lead to eversion of the labrum (Fig. 1). This eversion is thought to compromise the suction seal and potentially lead to microinstability and pain. A second location is between the femoral osteoplasty and the capsule. Adhesions in this location are thought to restrict motion and cause pain through capsular tethering. In the largest study in the published literature focusing on adhesions, Willimon *et al.* (10) report on only primary hip arthroscopies performed between 2005 and 2009. A total of 1264 hips over the age of 18 underwent a primary hip arthroscopy during this period. The authors reported that patients under the age of 30 were 5.9 times more likely to be in the adhesion cohort. Interestingly, patients who underwent a concomitant microfracture procedure at the time of primary arthroscopy were 3.1 times less likely to have adhesions as compared to those who did not undergo this procedure. The reasons are unclear, but this could potentially parallel the differing synovial milieus among healthy hips with FAI and an isolated labral tear and those with increased degeneration. In the same study, postoperative circumduction therapy, or continuous passive circular rotation of the hip joint during the immediate perioperative period, was also analyzed as an intervention. Patients who did not receive this therapy were 4.1 times more likely to have adhesions than those who did not perform these therapeutic exercises. Interestingly, the authors concluded that risk factors for adhesion formation included poor preoperative function (modified Harris Hip scores less than 50), age (less than 30 years), rehabilitation without circumduction, and the absence of concomitant microfracture.

Prevention of arthrofibrosis and treatment options

Adhesion formation is a risk after primary and subsequent revision hip arthroscopy, but there have been advances over the years in preventing this surgical complication. Recently, systemic pharmacological interventions have been used to block molecular pathways and key molecules that are thought to lead to the generation of

Table 1 Summary of strategies for prevention and treatment of adhesion formation following hip arthroscopy.

Intervention/Study	Findings	LOE
Prevention		
Preservation of chondrolabral junction		
Webb *et al.* (9)	Labral detachment and refixation (546 hips) compared to chondrolabral junction preservation (464 hips) during arthroscopic treatment of pincer-type FAI resulted in higher revision rates (9.9% vs 7.8%) and the presence of capsulolabral adhesions (46% vs 17%) upon revision surgery.	III
Early postoperative passive ROM		
Willimon *et al.* (10)	In a cohort of 1264 hips that underwent primary hip arthroscopy, patients who did not receive circumduction therapy were 4.1 times more likely to have adhesions compared to those who performed circumduction exercises (95% CI: 1.25–11.0)	IV
Sauber *et al.* (11)	Description of passive circumduction exercises that can be performed postoperatively at home by a caregiver to lower the risk of adhesion formation	NA
Biologics		
Utsunomiya *et al.* (12)	In a rabbit knee model, biologically regulated marrow stimulation by blocking TGF-β1 (oral intake of losartan) provided superior repair via decreasing fibrocartilage formation and resulting in hyaline-like cartilage as compared with outcomes from bone marrow stimulation only	NA
Kobayashi *et al.* (13)	In a murine model of muscle contusion, treatment with muscle-derived stem cells (MDSCs) and losartan decreased scar formation compared to treatment with MDSC alone	NA
PRP		
Li *et al.* (14)	In a rat model, neutralization of TGF-β1 with TGF-β1 antibodies within PRP significantly promotes muscle regeneration while significantly reducing fibrosis after cardiotoxin-induced muscle injury	NA
Treatment		
Revision hip arthroscopy		
Philippon *et al.* (15)	Lysis of adhesions and preservation of labral tissue with implementation of an iliotibial band allograft in the capsular recess to prevent recurrence of adhesions	NA
Ruhmann *et al.* (16)	Revision hip arthroscopy with lysis of adhesions resulted in improved flexion, abduction, internal rotation, and external rotation in a series of 49 hips presenting with capsulolabral adhesions	IV
Ultrasound-guided release of adhesions		
Reddy *et al.* (17)	After ultrasound-guided pressure injection, releasing postoperative extra-articular adhesions between the joint capsule and flexor tendons, 12/21 patients showed response to injection and 9/11 showed improvement in Hip Outcome Scores at 6 weeks and 6 months	IV

ROM, range of motion.

fibrotic tissue after surgery or injury. Several key regulatory molecules in fibrogenesis have been recognized including transforming growth factor beta 1 (TGF-β1) and its related pathway (4). Other regulatory molecules implicated in tissue remodeling include platelet-derived growth factor (PDGF) which affects cell migration and proliferation as well as fibroblast growth factor (FGF) and vascular endothelial growth factor (VEGF) promoting angiogenesis (4). TGF-β1 is produced from a wide range of molecules including platelets. At the site of injury, an inactive form of TGF-β1 is bound to the extracellular matrix and is activated thereby increasing TGF-β1 levels during injury. This phenomenon then leads to immune cells chemotaxis with increased concentrations of T-cells, neutrophils, monocytes, and fibroblasts, which initiate the initial robust inflammatory response. TGF-β1 also acts as a regulatory molecule by not only providing the deposition of extracellular matrix during cellular repair but stimulating or blocking the actions of other molecules (4, 17). TGF-β1 enhances the production and expression of matrix proteins and integrin molecules that increase cell adhesion to the matrix (4, 18). Furthermore, TGF-β1 decreases protease formation that can degrade the matrix and can also increase inhibitory molecules that regulate the aforementioned proteases (4). This inflammatory cascade ultimately results in tissue fibrosis and scarring.

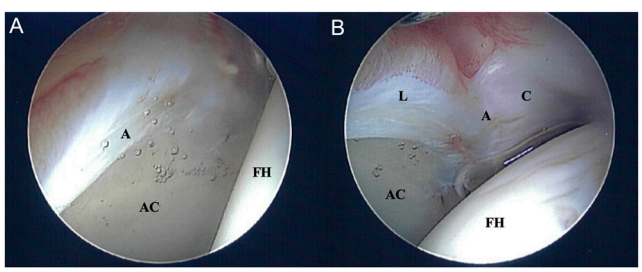

Figure 1
Arthroscopic view of right hip demonstrating capsulolabral adhesions. A, adhesions; AC, acetabular cartilage; C, capsule; FH, femoral head; L, labrum.

An improved understanding of the aforementioned signaling pathways has increased interest in testing pharmacologic agents to interfere with and ultimately block these pathways. It was first hypothesized and confirmed through studies that blocking the actions of TGF-β1 using agents such as decorin, relaxin, and suramin can result in a decrease in scar tissue formation (4, 19, 20, 21, 22). Interestingly, blocking the renin-angiotensin-aldosterone pathway, specifically, angiotensin II receptor blockade has been shown to have action on decreasing TGF-β1 at the site of tissue repair. Specifically, a study conducted by Cohn *et al.* (23) found that blockade of angiotensin receptors with the use of the pharmacologic agent losartan can alter the TGF-β1 signaling pathway

and inhibit the effect of this cascade. Losartan is an FDA-approved commonly prescribed antihypertensive drug that belongs to the angiotensin receptor-blocking class. Preclinical, animal, and human clinical studies have studied the use of losartan and its effect on the inhibition of the TGF-β1 signaling pathway and its effect on decreasing fibrosis in various tissues (23, 24, 25).

First, Li *et al.* (26) showed that inoculation *in vivo* of TGF-β1 could induce fibrosis of skeletal muscle. In a follow-up study in another murine model, losartan administration was shown to improve muscle regeneration while reducing fibrosis in a skeletal muscle injury model (25). Utsunomiya *et al.* (12) performed a murine study that looked into improving cartilage repair with regulated bone marrow stimulation while also blocking the effects of TGF-β1. Forty-eight New Zealand Rabbits were divided into three groups which included a control group with just an osteochondral defect, a BMS group with osteochondral defect and bone marrow stimulation, and a losartan-treated group with osteochondral defect, bone marrow stimulation, and losartan. In the study, the authors showed through histological assessment, macroscopic appearance, microcomputed tomography, and gene expression that the losartan-treated group scored significantly improved scores as compared to the BMS and control group. There are currently several registered clinical trials focusing on the effects of losartan on fibrosis prevention in humans (27).

Biologics are an alternative source that is also being explored. Biologics including platelet-rich plasma (PRP) or bone marrow aspirate concentrate (BMAC) being investigated throughout medicine and orthopedics as a source of healing (28, 29, 30, 31). PRP is produced from peripheral blood by centrifugation to separate and concentrate platelets within autologous plasma (19). Platelets themselves contain alpha and dense granules with the former secreting various growth factors, chemokines, cytokines, and proteins like insulin growth factor 1 (28). PRP can further be prepared as a fibrinous product with adhesive hemostatic properties through an exogenous or endogenous activation. This PRP-fibrin preparation can enhance angiogenesis, improve collagen synthesis, epidermal, epithelial, and endothelial regeneration and has been theorized to decrease scar tissue formation (32). Li *et al.* (14) studied in a murine model whether neutralizing TGF-β1's action with neutralizing antibodies could improve PRP's benefits in skeletal muscle repair. A TGF-β1 neutralizing antibody was used in order to potentially block the effects of TGF-β1 and ultimately reduce fibrosis. The results showed that there was a significant increase in regenerative myofibers in the PRP groups as compared to those rats who were not treated with PRP. The group tested with neutralizing antibody to TGF-β1 decreased fibrosis formation, while promoting muscle regeneration. Although this study provides evidence for fibrosis prevention in skeletal muscle, there is no clinical or preclinical evidence that it leads to decreased formation of intraarticular adhesions.

In addition to both pharmacologic and local interventions aimed at decreasing intraarticular adhesions following hip arthroscopy, surgical techniques have also been described (Fig. 2). Philippon *et al.* (15) present a surgical technique for the treatment of severe capsulolabral adhesions after hip arthroscopy. The treatment is recommended in instances of revision hip arthroscopy when capsulolabral adhesions are present. First, a complete lysis of adhesions is performed. Following the lysis of adhesions, if labral tissue is hypotrophic, it is better to remove the adhesions/scar tissue and proceed with a full labral augmentation, preferably using iliotibial band autograft. Conversely, if the labral tissue is determined to have good thickness (height >6 mm) and integrity, then it should be preserved (33). If determined that the labrum will be preserved after lysis of adhesions, an allograft capsular spacer placement can be inserted in the capsulolabral recess in an attempt to decrease the formation of recurrent adhesions as well as to inhibit any potential labral eversion to preserve the fluid seal. The graft (senior authors' preference is iliotibial band allograft) is placed between the capsule and the labrum and is fixed with suture anchors. Two-year follow-up data

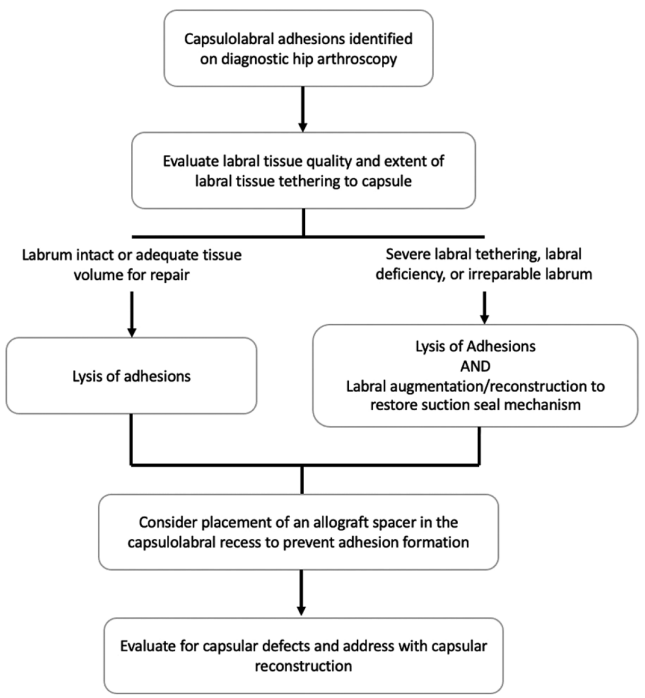

Figure 2
Surgical treatment algorithm for capsulolabral adhesions during revision hip arthroscopy.

using this technique reveals improved patient-reported outcome measures and good survivorship for this salvage circumstance (34).

Finally, building upon the finding that patients undergoing hip arthroscopy with microfracture were found to have decreased rates of adhesion formation, attention was drawn towards postoperative rehabilitation as in addition to the underlying differences in joint health, those patients undergoing microfracture also had a distinct postoperative rehabilitation protocol (10). In their cohort the differences in rehabilitation protocol in patients undergoing microfracture included the use of a continuous passive motion (CPM) machines 6 h per day for a total of 6 weeks postoperatively instead of 2 weeks for the patients that did not undergo the procedure. Furthermore, the patients who underwent microfracture were placed on crutches for a total of 7–8 weeks vs 3–4 weeks (9.1 kg flat foot for both) for those who did not. As a result, the authors concluded that this increased exposure to passive joint exercises and joint loading extended protection may decrease adhesion formation (10).

Conclusion

Intraarticular adhesions following hip arthroscopy are an important cause of surgical failures and a source of stiffness, pain, and decreased range of motion. Research has been conducted to better understand this phenomenon and to establish points of prevention and treatment. Systemic pharmacologic interventions such as losartan, local agents such as PRP, surgical techniques such as capsulolabral allograft spacers, and postoperative rehabilitation techniques have all been studied to different levels and it is likely that the ultimate prevention may be a combination of the above.

ICMJE Conflict of Interest Statement

MJP receives royalties from Smith+Nephew, Inc., Arthrosurface, Bledsoe, ConMed Linvatec, DJO, SLACK Inc., and Elsevier; owner/shareholder in Arthrosurface, MJP Innovations LLC, Vail Valley Surgery Center, Vail MSO Holdings LLC; Shareholder in MIS, EFFRx, Olatec, iBalance (Arthrex), Manna Tree Partners, Stryker, Trimble, 3M, Bristol Myers, Squibb, Pfizer, AbbVie, Johnson & Johnson; and Board Member for the International Society of Hip Arthroscopy, Vail Health Services and Co-Chairman for the Steadman Philippon Research Institute. MJP has also received education support from Smith+Nephew, Inc., ConMed Linvatec, Ossur, Arthrex, Siemens Medical Solutions; speaking fees and consulting from Smith+Nephew, Inc., MIS, Olatec, NICE Recovery Systems; and hospitality payments from Siemens Medical Solutions and Synthes GmbH. JJR is a consultant for Smith+Nephew, Inc. RWS, SMC, JWA having nothing to disclose.

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