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Introduction

While pathology of the acetabular labrum and chondrolabral junction is often studied, lesions and pathology associated with the ligamentum teres and acetabular cotyloid fossa have been clearly elucidated as an important and perhaps overlooked source of nonarthritic hip pain and mechanical symptoms. Tears of the ligamentum teres, synovial disorders such as synovial chondromatosis, and stenosis of the cotyloid fossa are representative of the more commonly encountered pathologies in this area. Although recently defined physical examination tests exist, imaging and arthroscopy remain essential for diagnosis and management of these injuries.

The precise function of the ligamentum teres (LT) remains unclear and multiple theories exist. Some authors' work demonstrated evidence of a role as a static stabilizer of the hip joint with contributions to force and fluid distribution. Other sources diverge from this view, characterizing the LT as a functionless remnant of embryonic tissue [1–3]. Most recently, theories propose the LT as a possible source of proprioceptive and somatosensory feedback [4] and also as a stabilizer of the hip joint. Basic science evidence has shown that the LT resists dislocation and microinstability with

tensile strength comparable to the anterior cruciate ligament (ACL) in a porcine model [1]. This stabilization role seems to be even more pronounced in dysplastic hips [5]. Some have established its role as an important static stabilizer of the joint and have advocated reconstruction in the setting of symptomatic insufficiency [6]. The role of the LT as a source of hip pain is more well established [5–11].

Pathology of the LT includes both traumatic and nontraumatic tears. Gray and Villar introduced a classification of LT tears that is comprised of a type I full-thickness tear, a type II partial-thickness tear, and a type III degenerative tear [12]. Botser et al. proposed a descriptive classification that quantifies the degree of tearing. It includes a type I <50 % partial-thickness tear, a type II >50 % but <100 % partial-thickness tear, and a type III full-thickness tear [13]. Nontraumatic tears of the LT have been associated with developmental dislocation of the hip, Legg–Calve–Perthes disease, and osteoarthritis/degenerative signs [13–16]. Domb et al. have also established associations between acetabular bony morphology and patient age to the presence of LT tears. Tears of the LT were more frequent in those with a diminished lateral acetabular coverage as defined by a decreased lateral center-edge angle of Wiberg in conjunction with an elevated Tonnis acetabular inclination angle. An increased prevalence of LT tears was also associated with age >30 years [17]. Traumatic rupture of the LT secondary to hip dislocation is a well-recognized cause of tears as well, and it is not known how much this may contribute to recurrent instability in these patients [5–7, 9–11].

Clinically, patients with a clear injury mechanism resulting in acute rupture may subsequently report symptoms of instability and pain [7, 18–20]. Diagnosis otherwise may be elusive and challenging as patients will likely describe non-specific symptoms of groin pain, catching, and giving way. Physical examination will often reveal painful range of motion consistent with intra-articular pathology. More specific tests such as the posterior impingement test, the dial test, the apprehension with distraction test, and the LT test as

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described by O'Donnell et al. have been described [20–23]. The incidence of LT tears is varied ranging from as low as 4–17 % [5, 19] in earlier reports to as high as 49–65 % in more recent reports [13, 17, 18]. This variation may be a result of observation bias in some groups with heightened awareness of this pathology and recognition of less severe lesions.

Persistent symptoms of pain or mechanical symptoms following appropriate conservative care constitute potential surgical candidacy. Literature support exists for arthroscopic mechanical and/or radiofrequency debridement when evidence of intra-articular pathology amenable to arthroscopic treatment exists [8, 10, 11, 13, 17, 21]. In those patients with symptomatic instability and both subjective and positive clinical exam findings who have failed prior surgical procedures, LT reconstruction using varying techniques and graft sources has shown promising early results [6, 23–25]. Further study is required to validate the exact role for this procedure.

Synovial chondromatosis (SC) is an uncommon, benign, often monoarticular disease characterized by the formation of sessile or pedunculated collections of cartilage nodules [26]. Histology shows metaplasia of the synovial mesenchymal cells [27]. This can result in cartilaginous loose bodies which can ossify or undergo endochondral ossification [27]. The hip is the second most commonly involved joint after the knee [28]. The main consequence is chondral damage secondary to mechanical abrasion from the loose bodies. Small, cartilaginous loose bodies may result in minimal articular damage, whereas larger and/or ossified bodies may result in more severe, irreversible damage.

Clinically, an early diagnosis is important as a poor prognosis has been associated with retained intra-articular loose bodies [29, 30]. The severity of osteoarthritic changes at the time of treatment is the main predictor of successful clinical outcome [27]. Plain radiographs may show loose bodies in the joint or peripheral compartment but would fail to show noncalcified loose bodies. CT and especially MRI has aided in earlier and more accurate diagnosis [27].

Recommended treatment consists of surgical removal. Conventional open surgery has shown good results but with recurrence rates as high as 15 % with arthrotomy alone versus the morbidity and prolonged rehab associated with surgical dislocation [31, 32]. By contrast, arthroscopic removal of loose bodies and synovectomy has shown satisfactory results with a shortened recovery time and fewer complications [27, 33, 34].

A stenotic cotyloid fossa can be secondary to marginal heterotopic bone formation in the fossa and also abnormal amounts of fibrous and fatty tissue. A theory proposes that this can lead to incarceration of the LT. The space-occupying effect of this can perhaps lead to lateral subluxation of the

femoral head and may have implications to chondrolabral pathology secondary to edge/rim loading [35]. Philippon et al. reported a 7 % rate of cheilectomy for a stenotic cotyloid fossa in a report on 45 professional athletes with femoroacetabular impingement (FAI) and their associated pathologies [18]. Plain radiographic and MRI evaluation can show bony projection into the fossa that is contiguous with the medial margin of the acetabular sourcil [1]. The goal of treatment is to remove any space-occupying tissue from the cotyloid fossa, decompress the constriction upon the LT, and perhaps improve joint congruence [35].

This chapter demonstrates the correlation between MRI and arthroscopic findings for lesions and pathology associated with the LT and cotyloid fossa and their associated diagnosis and management. Three cases will be presented, including:

1. Decompression of a stenotic cotyloid fossa
2. A ligamentum teres reconstruction for symptomatic deficiency
3. Arthroscopic treatment of synovial chondromatosis with associated FAI pathology

Case 1: Cotyloid Fossa Stenosis

History

Male, 39 years old, snowboarder. Patient has had left hip pain for several years. He recalls first feeling hip pain while surfing, which made him limp for a few days. The pain progressed slowly and now prevents him from performing any kind of exercise. He can walk up to four blocks before starting to limp. He also noticed some loss in range of motion and difficulty to put on shoes. His pain is 5/10 at rest and 8/10 at its worst.

Exam

Hip flexion 0–110°. Internal rotation 5°. Abduction 30°. Flexion abduction and external rotation (FABER) 20 cm, knee to table distance. Positive anterior impingement sign. Negative posterior impingement sign. Negative dial test. Thomas test was positive. Ober's test was negative.

Imaging

Radiographs

Decreased joint space (<2 mm), bilateral mild hip arthritis, pistol grip deformity with mild osteophytes; false profile view shows ossified anterior labrum (Fig. 30.1a, b).



Fig. 30.1 (a) Pre-op anteroposterior radiograph; note reduced joint space in the left hip. (b) False profile view: *arrow* shows ossified labrum anteriorly

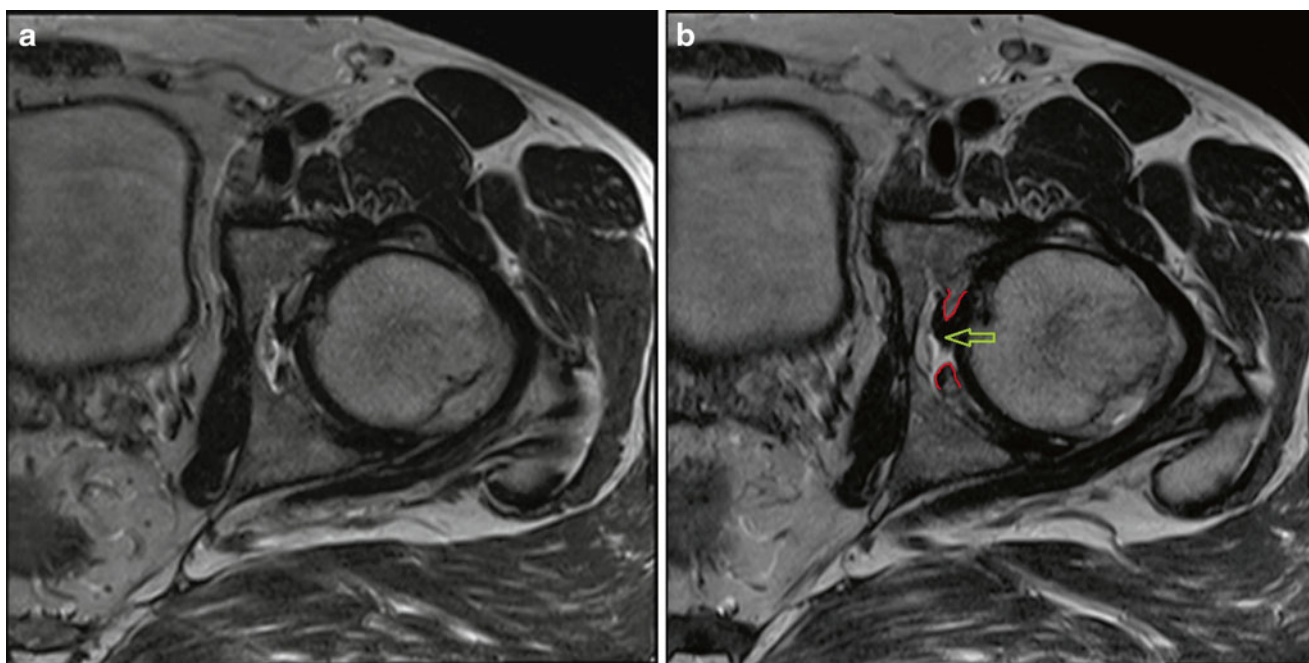


Fig. 30.2 (a, b) MRI of cotyloid fossa stenosis—axial view. *Red lines*: border of osteophytes causing the cotyloid fossa stenosis. *Yellow arrow*: ligamentum teres

MRI

Labral tearing of the anterior and lateral portions of the labrum. Chondral thinning and fissuring; lateral center-edge angle is 23° ; alpha angle is 76° ; femoral retroversion is 4° . Moderate effusion with capsular scarring and extensive synovitis, debris, and possible loose bodies. A prominent osteophyte was observed around the cotyloid fossa (Fig. 30.2a, b).

Arthroscopy

The patient underwent a left hip arthroscopy, synovectomy, rim trimming, neck osteoplasty, removal of loose bodies, cartilage debridement, and chondroplasty.

An ossification of the acetabular labrum from 8:00 to 2:00 position was observed. This was removed with an osteotome

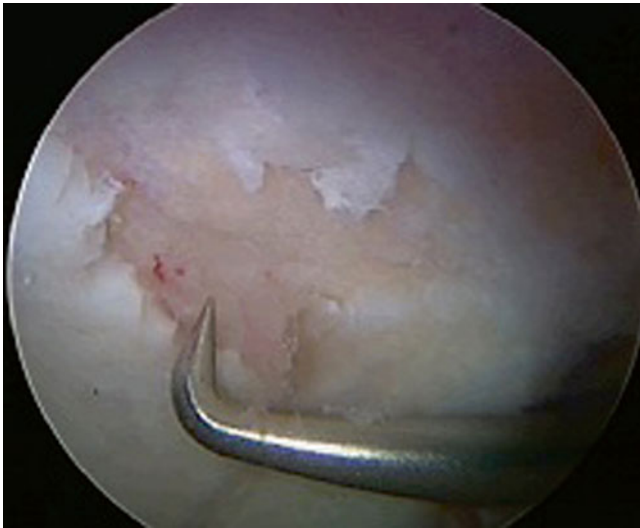


Fig. 30.3 Microfracture using an arthroscopic awl

and a burr. A labral reconstruction was performed with a 50 mm IT band graft and six suture anchors.

There was a grade 4 acetabular chondral defect of roughly 30×15 mm. Microfracture was used to treat this lesion (Fig. 30.3).

With the hip in distraction, the cotyloid fossa was inspected, and osteophytes were removed from this area to allow the femoral head to be well seated within the acetabular cup and to release the ligamentum teres entrapment. To accomplish this, we utilized different instruments such as an osteotome, burr, chisel, and basket punch (Fig. 30.4a–e).

After this, we debrided the ligamentum teres with the RF device, removing the inflamed synovium around it so it would sit better inside the cotyloid fossa (Fig. 30.5).

After the cotyloid fossa stenosis and LT debridement, we observed a better congruence between the femoral head and the cotyloid fossa. Traction was released so a dynamic assessment could be performed, and the femoral head could glide freely inside the acetabulum with restoration of the fluid seal.

Discussion

This case reveals the multifactorial nature of hip dysfunction in the active patient with moderate osteoarthritis. The acetabular rim, sclerotic and with ossified labrum, was a component of lost joint motion. The large cam-style femoral osteophyte also contributed. The theoretical contribution of space-occupying degenerative acetabular fossa contents also appeared to contribute. Attention to the deep central hip compartment arthroscopically was guided by clinical suspicion and the MRI findings of synovitis, effusion, and acetabular fossa stenosis. Further study is required to advocate global acceptance of this type of debridement, but preliminary case-based evidence is promising.

Case 2: Ligamentum Teres Reconstruction

History

Female, 25 years old, softball coach. Patient had already undergone two prior right hip arthroscopies and two left hip arthroscopies.

The last left hip surgery (1 year before) included labral debridement, lysis of adhesions, chondroplasty, synovectomy, psoas lengthening, and a labral reconstruction using an IT band autograft.

The patient complains of left hip pain that continues to bother her daily. She has signs of hip instability. She complains of her left hip giving out multiple times a day, preventing her from performing daily activities. Sometimes she has the feeling that the hip “pops out.”

Past surgical history: positive for multiple surgeries on both hips, both shoulders, and left foot.

Exam

Normal strength of the muscles surrounding the hip joint, including hip flexors bilaterally. Normal sensation throughout lower extremities. Positive anterior impingement sign. Physical exam is consistent with generalized laxity. ROM: flexion 126°, abduction 45°, and adduction 30°. Grossly positive left hip manual distraction test.

Imaging

Radiographs

Joint space was preserved (>2 mm). Sourcil measuring 2–3 mm, anterior sourcil 3 mm. Tonnis angle is 5.5°. Lateral center-edge angle is 29°. Alpha angle is 43°. Sharp angle is 42°. See Fig. 30.6.

MRI

Labral reconstruction appears stable, without any gross separation identified. Diminutive ligamentum teres with signs of scarring and possible prior debridement. A complete LT tear could be present. See Fig. 30.7a, b.

Arthroscopy

Before putting the leg into table traction, a physical exam was performed under anesthesia. The joint could be distracted with manual traction alone, confirming severe hip laxity (Fig. 30.8).

Initial arthroscopic inspection: significant synovitis throughout the joint, significant adhesions especially at the

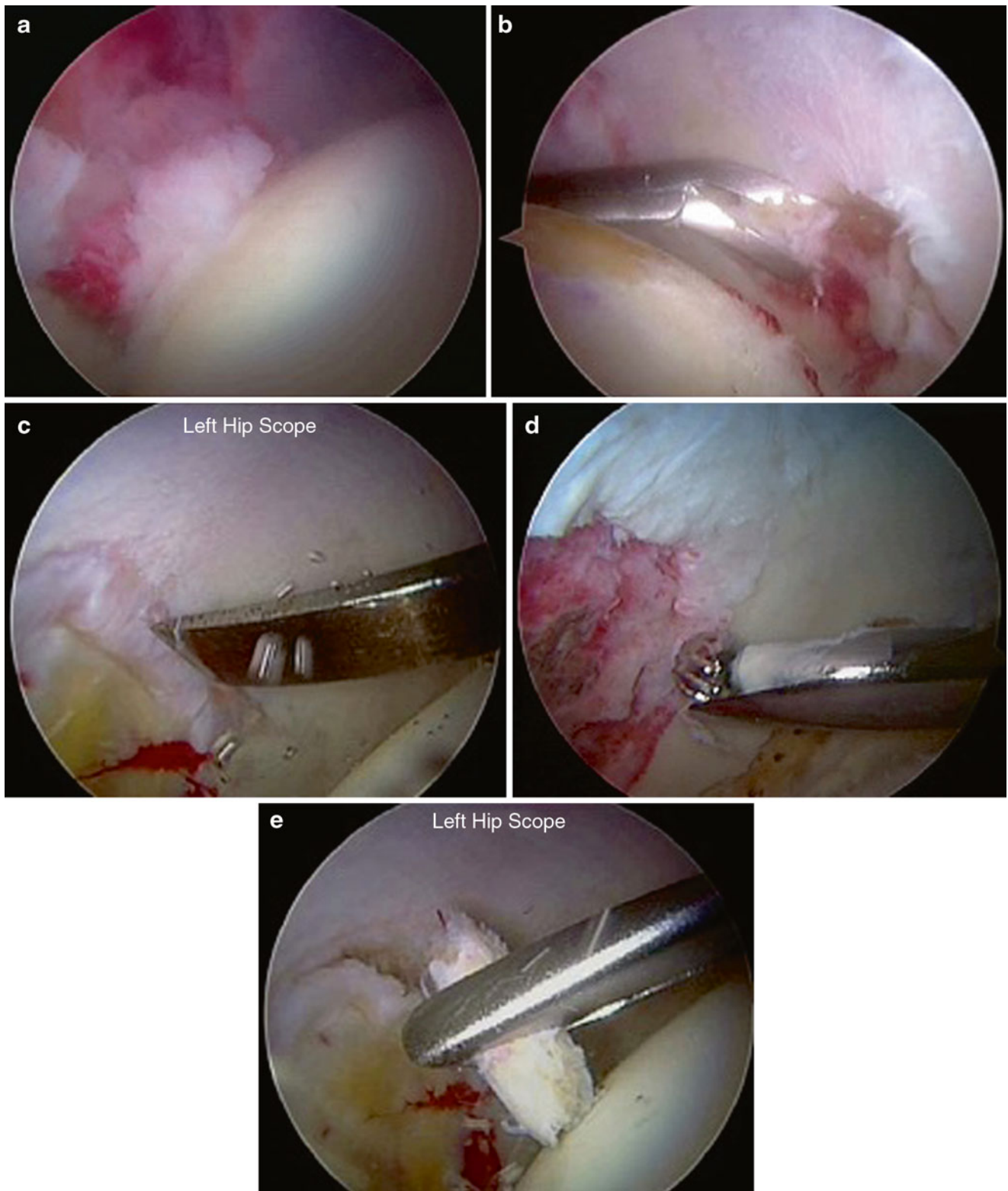


Fig. 30.4 (a) Fossa stenosis; ligamentum teres entrapment. (b) Osteophyte removal with a basket punch. (c) Chisel used for osteophyte removal. (d) Fossa stenosis decompression with burr. (e) Osteophyte removed with grasper

capsulolabral recess, and a visible ligamentum teres tear (Fig. 30.9).

Synovectomy was done using a combination of the electrocautery wand, ablator, and shaver. Hemostasis was assured with the RF device.

We then removed the adhesions by using the RF ablator and shaver, clearing up the capsulolabral recess. The labral reconstruction was intact and the suction seal was restored once the adhesions were removed.

Ligamentum teres: the LT was torn and grossly insufficient. The scar tissue surrounding it was debrided.

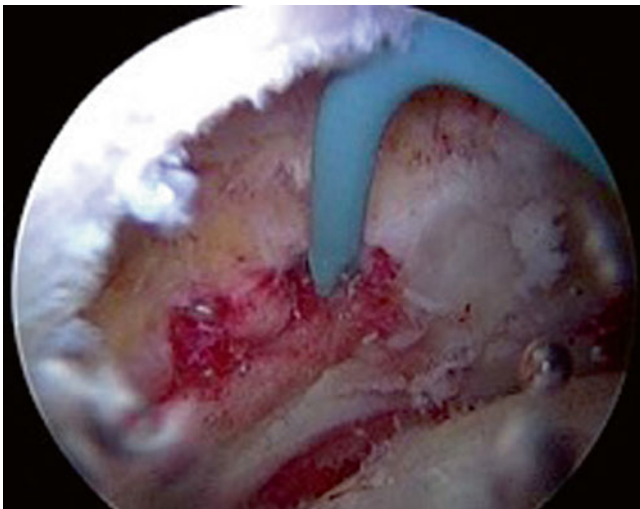
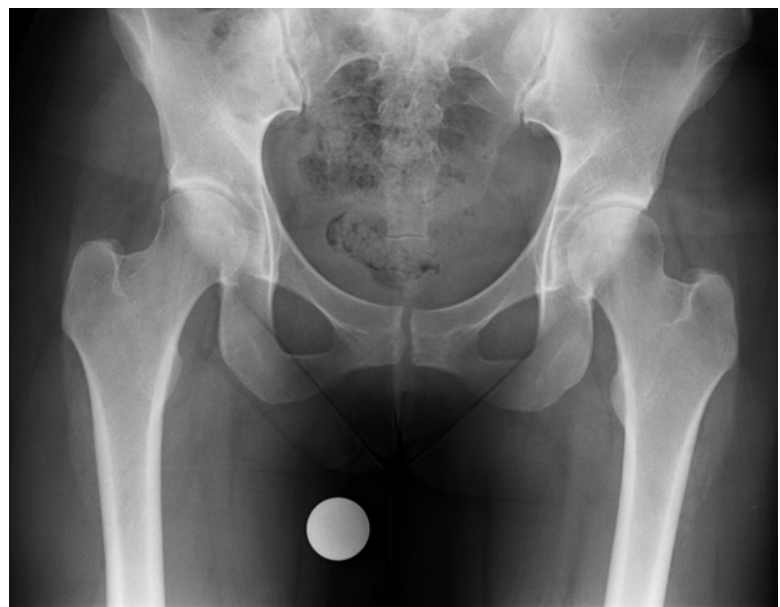


Fig. 30.5 LT debridement

Fig. 30.6 AP pelvis before surgery



To begin the LT reconstruction, the arthroscopic shaver and burr were used to debride the cotyloid fossa, partially removing the pulvinar fat, and to expose the footprint of the ligamentum teres.

Then, using the C-arm, a K-wire was inserted from the lateral cortex of the femur, in line with the femoral neck. The trajectory was 1 cm below the center of the neck, so the tip of the K-wire would exit at the femoral fovea. The correct position of the K-wire was confirmed at both lateral and AP views (Fig. 30.10).

We then drilled a hole using a cannulated 6 mm reamer over this K-wire. This was then enlarged to 7 mm and finally to 8 mm. The tunnel went all the way to the femoral fovea, and a curette was placed inside the joint to protect the acetabular joint surface from damage by the reamer and the K-wire (Fig. 30.11a–c).

The anchor guide was placed through this hole on the femoral neck; a hole was drilled on the cotyloid fossa for insertion of a 2.9 mm anchor loaded with a #2 suture.

This #2 suture was retrieved arthroscopically through the anterolateral portal. One of the suture limbs was passed through a tibialis anterior allograft using a free needle. Then an arthroscopic knot was used to push the graft into the joint through a cannula, until it was compressed against the fossa. A sequence of knots secured this end of the graft at this position (Fig. 30.12a, b).

Then an arthroscopic grasper was inserted through the femoral neck hole to pull the allograft into the femoral neck tunnel. About 2.5 cm of the graft remained between the fovea and the fossa, with the leg in extension and external rotation (Fig. 30.13a, b). This is critical to avoid loss of movement.

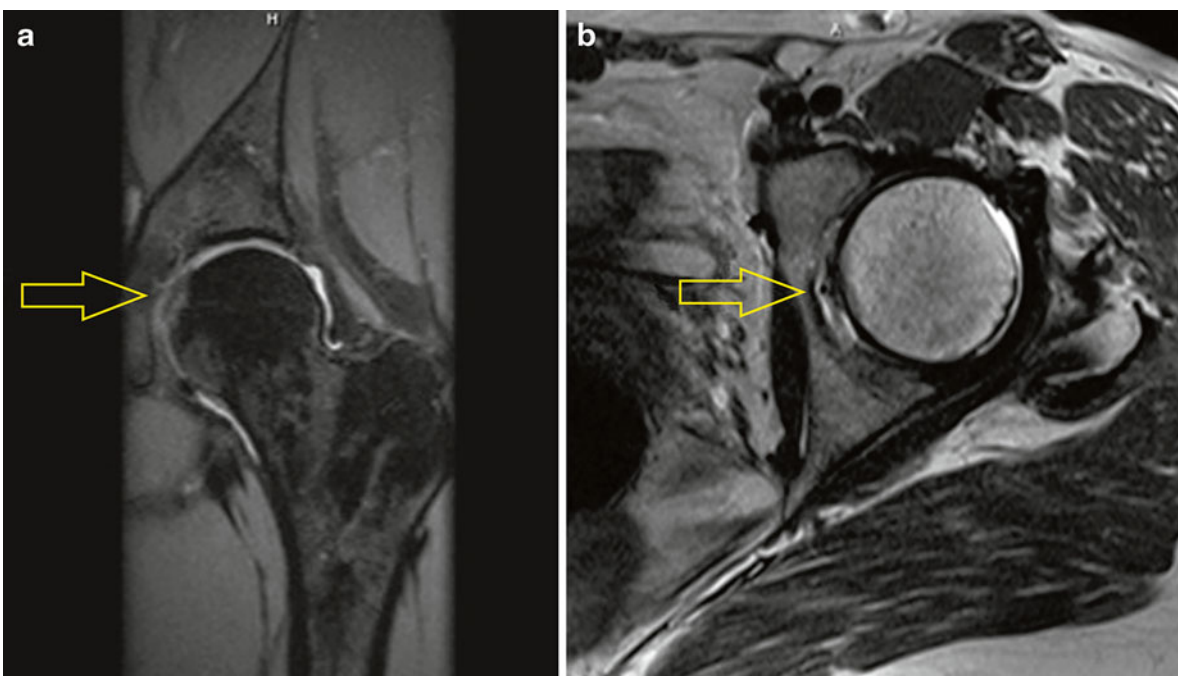


Fig. 30.7 (a, b) MRI pre-op: ligamentum teres is frayed and cannot be precisely outlined, which could be due to a complete tear

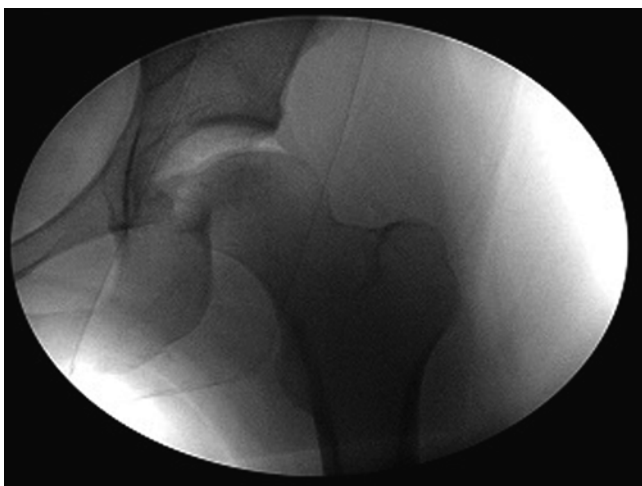


Fig. 30.8 Effortless manual distraction of the hip joint, under anesthesia

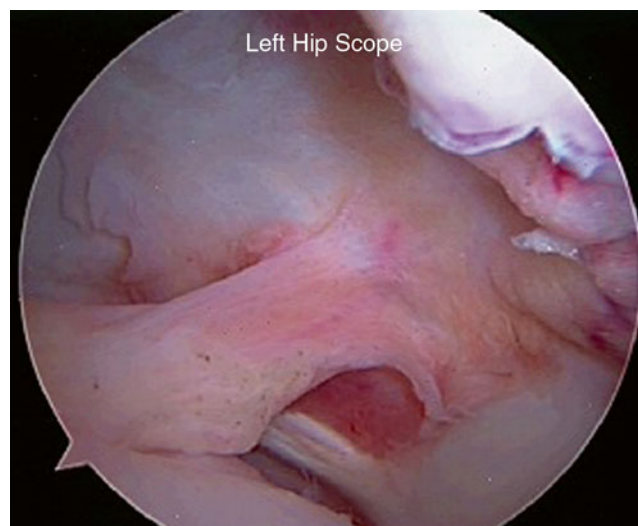


Fig. 30.9 Torn insufficient ligamentum teres

Tension level was verified by checking the hip's range of motion and graft excursion. After reaching the desired tension, an 8×35 mm interference screw was placed to assure fixation of the graft inside the tunnel. After fixation, the hip range of motion was again verified to be satisfactory.

To end the case, we performed a capsular plication to make sure that the capsule would heal with an appropriate amount of tension, restoring its function as a secondary stabilizer.

Discussion

There are conceptual and technical points to consider upon review of this case. Conceptually, we believe that hip microinstability is multifactorial. The ligamentum teres is a secondary stabilizer of the hip joint. LT insufficiency may become symptomatic in patients with dysplasia and/or following surgical LT debridement. Technically, the LT reconstruction with an allograft needs to be associated with other

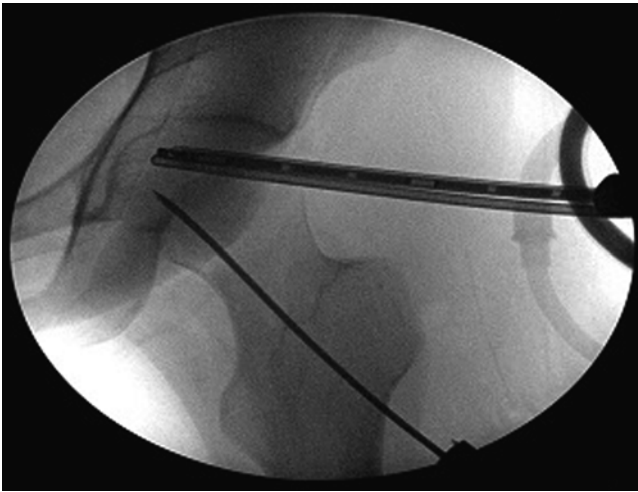


Fig. 30.10 K-wire is drilled into the femoral neck with c-arm and arthroscopic guidance

procedures to address microinstability, such as capsular plication. Meticulous attention should be given to correct allograft length and tension. Reduced length may impair full range of motion. This procedure requires mastery of all advanced hip arthroscopy techniques prior to performance and is presented here as an example of the frontier of treatment in the salvage setting.

Case 3: Synovial Chondromatosis

History/Exam

A 35-year-old male with a desk job who recreationally plays golf and attends spinning classes presented to the orthopedic clinic with 6 months of insidious onset mild, intermittent left groin and lateral hip pain with stiffness as he began undertaking

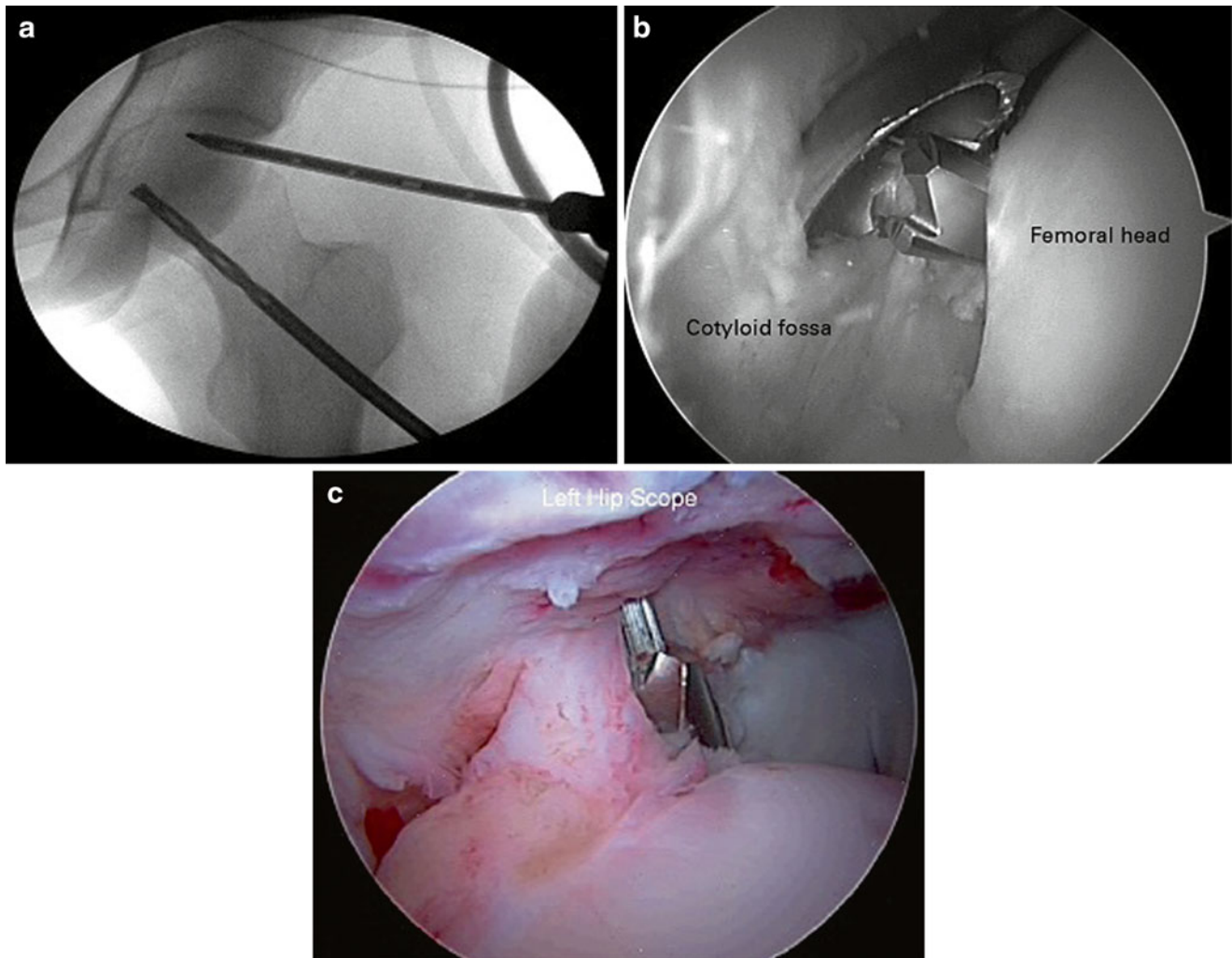


Fig. 30.11 (a) The 8 mm tunnel is made with a reamer. (b) A curette is used to protect the acetabular surface. (c) Drill bit exiting through the fovea

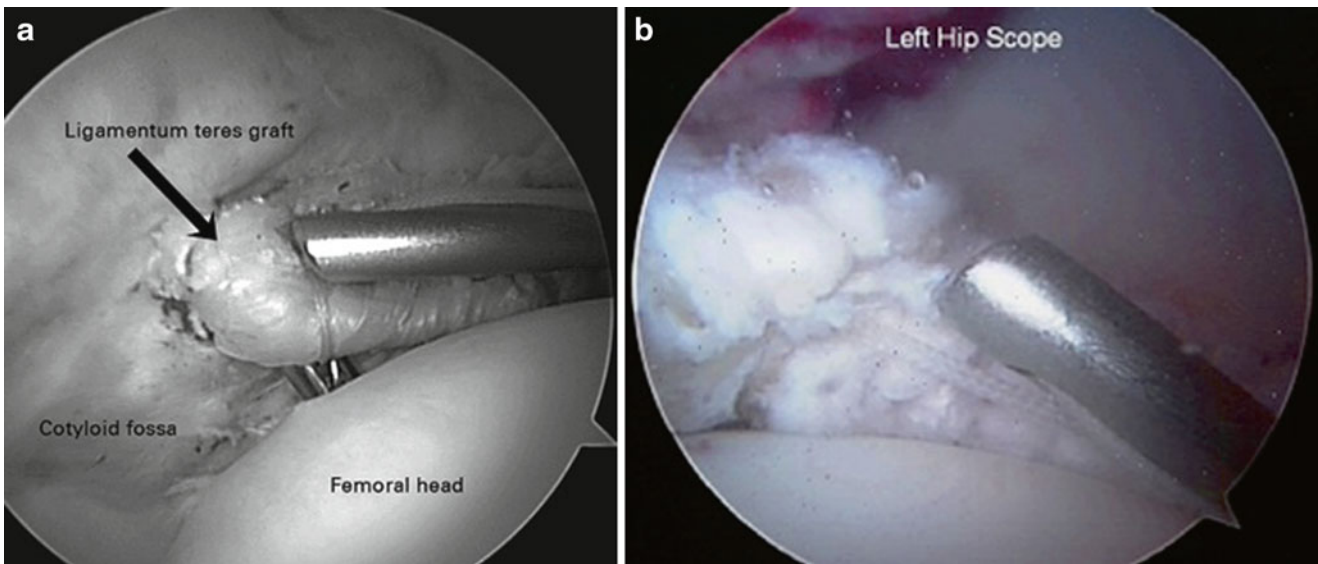


Fig. 30.12 (a, b) Graft is pushed into position with a knot pusher

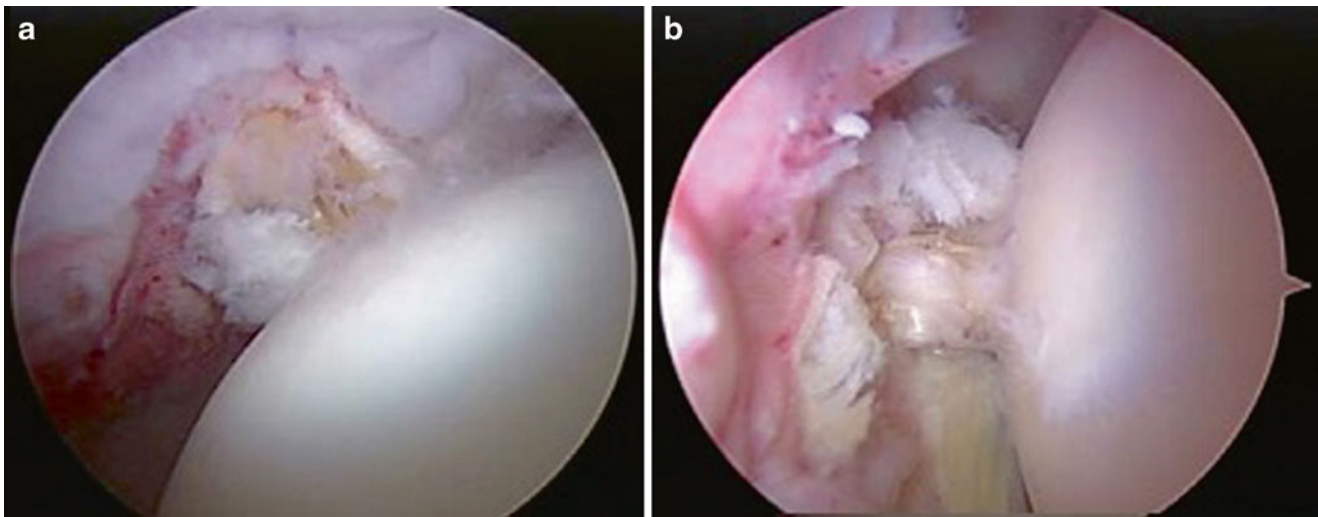


Fig. 30.13 (a, b) Status post fixation of the allograft in position

his spinning classes. He denied any symptoms 6 months ago. The pain was worse with lying on his back, deep hip flexion activity such as putting on his shoes, and twisting or rotation of the hip such as getting in and out of the car.

On physical exam, the patient was found to have full and symmetric range of motion of the hip with pain at the extremes of motion and a positive anterior impingement sign, positive Stinchfield test, and positive FABER test for reproduction of anteriorly based pain. He had no areas of tenderness and no appreciable weakness compared to the contralateral side. He was neurovascularly intact distally.

Imaging

Plain radiographs (Fig. 30.14a, b) revealed maintained joint space with findings of femoroacetabular impingement. Magnetic resonance imaging was obtained to further characterize the extent and character of any chondral or labral pathology and to look for other attendant signs of femoroacetabular impingement given the long-standing symptoms.

A complete exam was obtained and key images are shown. Figure 30.15a, b reveals an effusion with an acetabular labral tear as well as pincer pitting at the femoral head–neck junction.

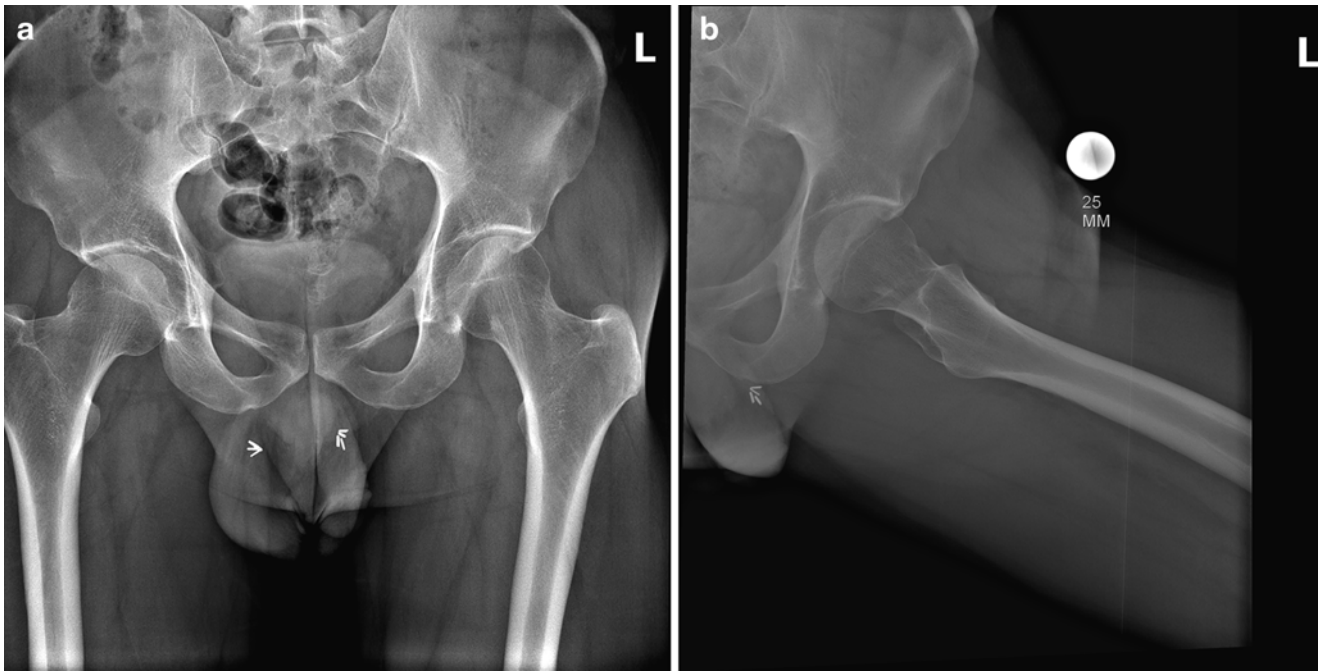


Fig. 30.14 (a, b) AP pelvis and frog leg lateral of the pathologic hip, pre-op

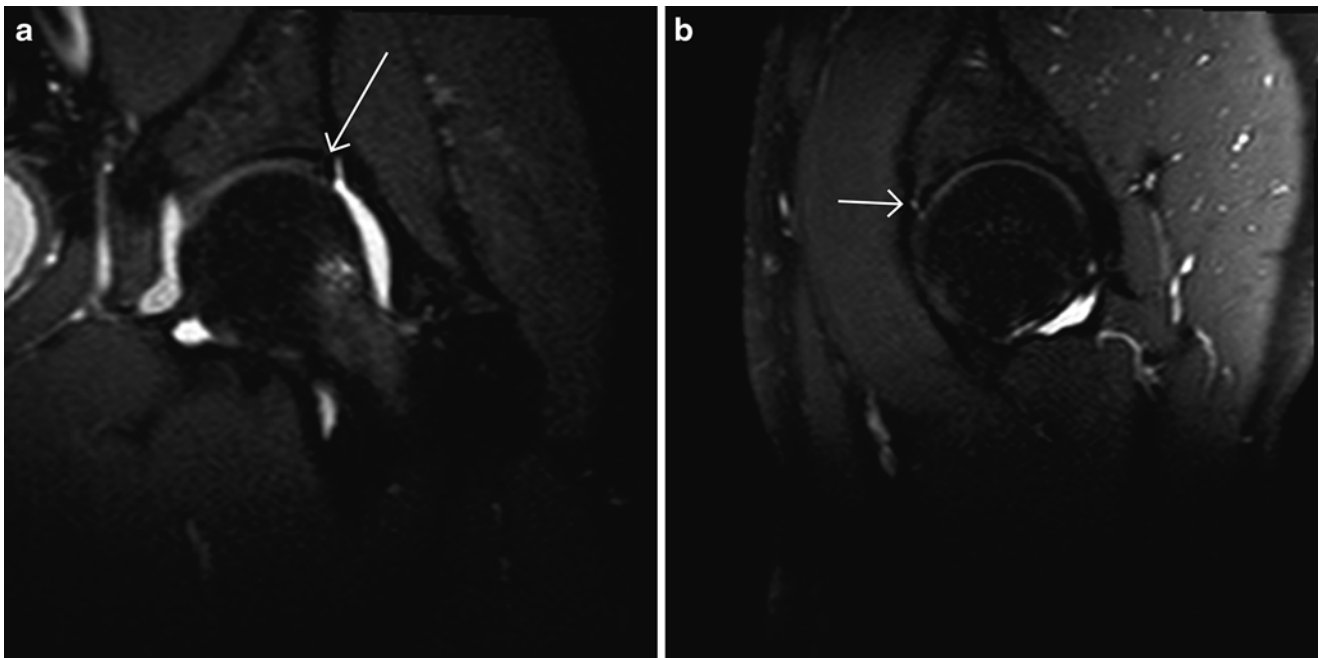


Fig. 30.15 (a) T2 coronal MRI image of the left hip, pincer lesion with labral tear. (b) T2 sagittal MRI image of the left hip, pincer lesion with labral tear

Although at first underappreciated, the imaging clearly shows many intermediate signal loose bodies within the acetabular fossa (Fig. 30.16a, b).

Despite nonoperative management including nonsteroidal anti-inflammatory medications, rest, physical therapy including

low-impact activity and core strengthening, and beneficial, albeit transient, responses to intra-articular corticosteroid injections, his symptoms persisted and worsened slowly over the course of 2 years. Surgical intervention with the intention of treating the femoroacetabular impingement and associated

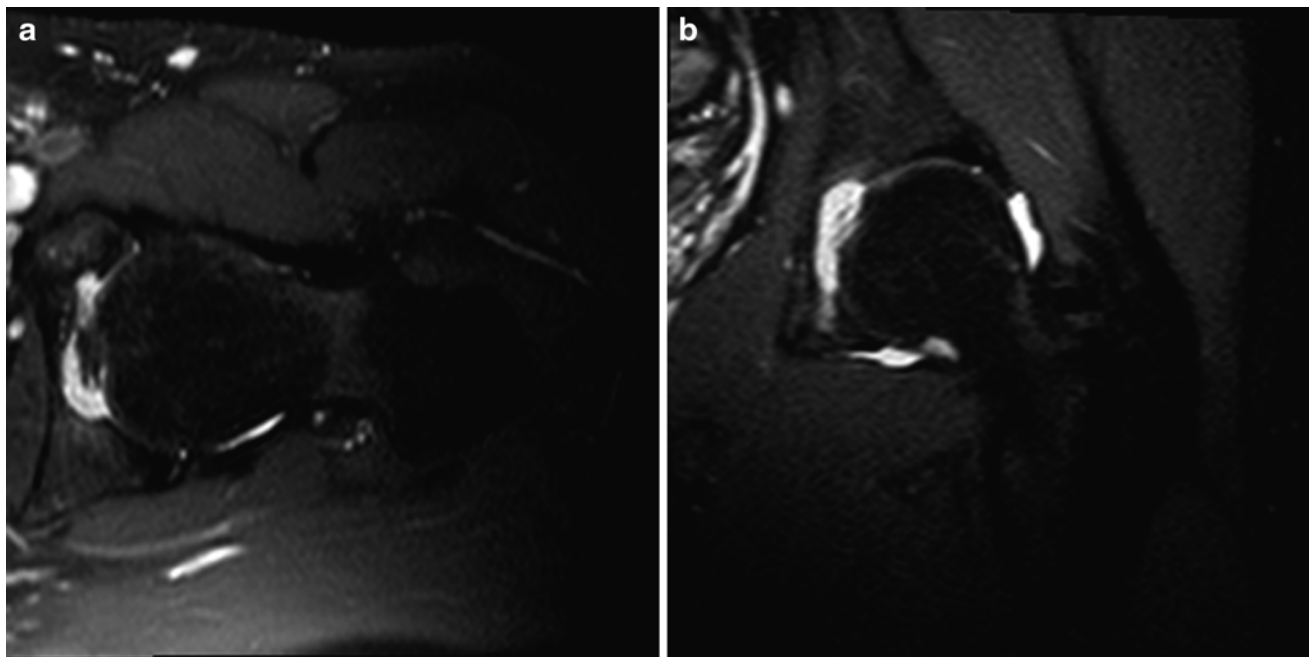


Fig. 30.16 (a) T2 axial MRI images, effusion, and loose bodies in the fossa. (b) T2 coronal MRI images, effusion, and loose bodies in the fossa (gray signal)

chondrolabral pathology was recommended, and the risks and benefits were discussed. He elected to proceed with arthroscopic evaluation of the hip joint with a plan for labral repair, acetabuloplasty, and femoroplasty.

Arthroscopy

The patient was taken to the operating room and placed supine on a Hana traction table. A standard diagnostic arthroscopy of the left hip was completed. The patient had moderate synovitis peripherally. The chondrolabral junction was also found to have high-grade chondrolabral instability with labral detachment from the acetabular margin with junctional attrition and marginal contusion of the labral tissue from about 9:30 to 12 o'clock position (Fig. 30.17). In addition, there was a margin of softened, unstable articular cartilage but no areas of unstable delamination. The femoral head articular cartilage was normal. The ligamentum had some synovitis but otherwise the fovea itself was normal. The fossa on the other hand had accretions of multiple cartilaginous loose bodies completely incarcerating the entirety of the acetabular fossa (Fig. 30.18a–c).

A capsulotomy was then performed and a synovectomy was performed with a shaver. A labral takedown was performed meticulously in a retrograde fashion to maintain the bulk of the labral tissue. The unstable marginal articular cartilage was then debrided with a shaver. After labral takedown and debridement of the unstable marginal articular cartilage,

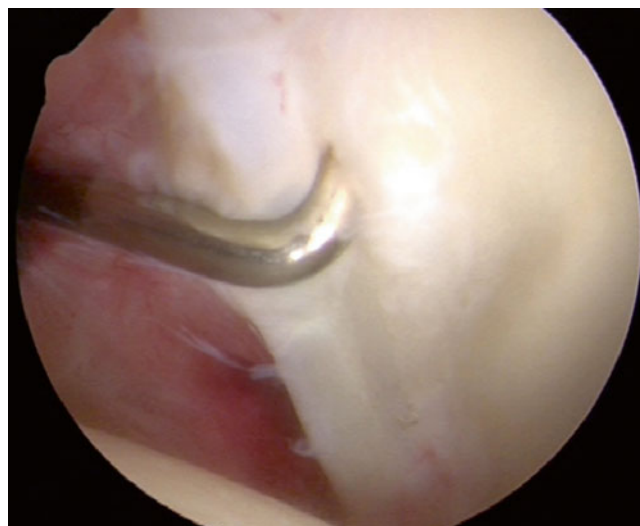


Fig. 30.17 Labral tear

the full extent of the pincer lesion was visualized, and a burr was used to perform an acetabuloplasty negating the anterior crossover sign seen fluoroscopically and removing the grossly visualized pincer lesion. The labrum was then repaired with two #2 nonabsorbable sutures placed circumferentially in a baggage tag fashion and then secured to the acetabular margin via two 2.9 mm-knotless anchors (Fig. 30.19). The chondrolabral stability and suction seal of the joint were confirmed to be reestablished through direct palpation and dynamic evaluation.

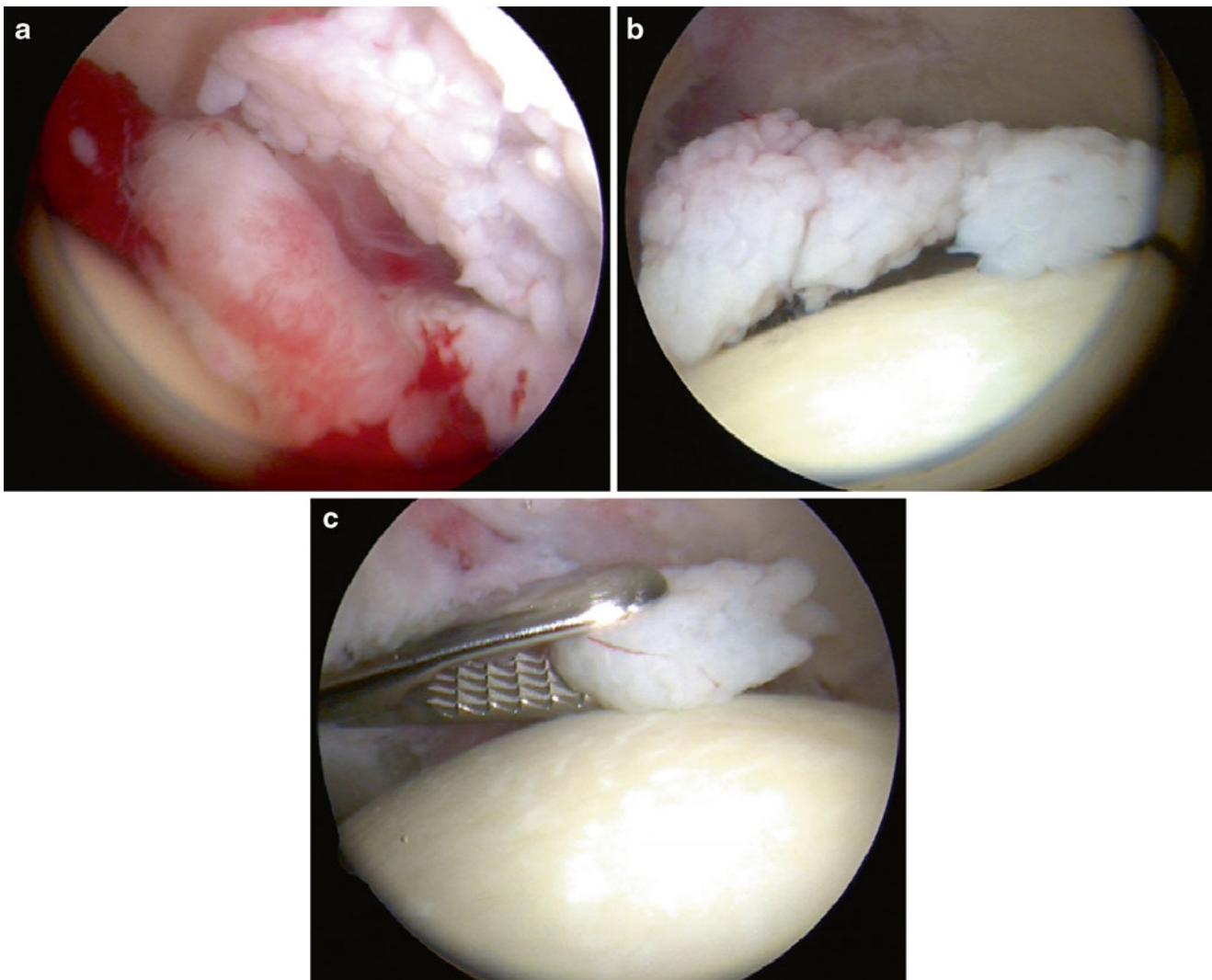


Fig. 30.18 (a–c) Loose bodies in the cotyloid fossa and central compartment; removal of loose body with grasper

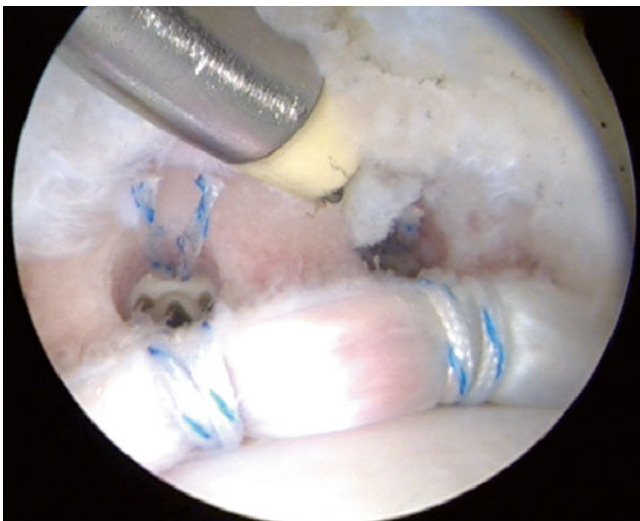


Fig. 30.19 Labral repair with luggage handle fixation through knotless suture anchors

Once the pincer lesion had been debrided and the chondrolabral junction was restored, the loose bodies were removed from the acetabular fossa. The removal was meticulous and required the use of multiple instruments in concert including graspers, the motorized shaver, and intra-articular suction. Some could be retrieved in whole, but others required debridement and suction irrigation (Fig. 30.18a–c).

At the completion of the evaluation and treatment of the pathology in the central compartment, the gross traction was relieved and the peripheral compartment was then examined arthroscopically. Dynamic evaluation revealed residual bony conflict with the acetabulum and pincer pitting with reactive cam lesion at the head–neck junction anteriorly. A T-capsulotomy was performed, and the fibrocartilage was removed from the head–neck junction down to the bony cam lesion with electrocautery. There was also appreciated a loss of head–neck offset down to the area of the cam lesion. A motorized burr was used to perform femoroplasty, therefore

restoring the normal head–neck offset and removing the cam lesion. Further exploration was done to confirm the absence of any more loose bodies. A dynamic exam revealed the successful resolution of the bony conflict.

Discussion

This case illustrates the requirement for thorough consideration of the possibility that loose bodies may contribute to hip complaints. MRI findings of low-signal areas in the acetabular fossa, particularly when arthrogram contrast is added, should raise clinical suspicion. This case being a clear case of secondary chondromatosis, the primary pathology was treated as well in the form of osteochondroplasty and impingement correction. There is little evidence to support or reject the concern that osteoplasty would lead to recurrence of synovial chondroma formation.

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