Surgical Technique: Arthroscopic Core Decompression

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Abstract

Osteonecrosis of the femoral head remains a challenging problem in the young patient population. The pathophysiology of this condition is poorly understood, and often, the underlying etiology is unknown. Several risk factors have been identified, including steroid use, alcohol use, trauma, and a history of hypercoagulable disorders. The management of osteonecrosis is dependent on the stage of the disease, which is typically divided into pre-collapse and post-collapse stages. The management of pre-collapse osteonecrosis of the femoral head is controversial, and both medical and surgical options have been described. The most commonly used surgical options include core decompression as well as bone grafting (vascularized or non-vascularized). Core decompression is a technique that theoretically decreases intraosseous pressure of the femoral head, resulting in a local vascularized healing response, and recently, arthroscopic techniques have been reported.

Introduction

Etiology

Osteonecrosis, also referred to as avascular necrosis, is a condition in which subchondral bone loses its viability, resulting in sclerosis, weakening of the surrounding bone, subchondral collapse, articular incongruity, and, ultimately, resultant osteoarthritis [1, 2]. While a majority of cases are idiopathic without a known cause, several risk factors for the development of osteonecrosis of the femoral head have been identified. These risk factors can be broken down into two categories, including traumatic and atraumatic. Traumatic etiologies include fracture as well as a history of hip subluxation or dislocation. Atraumatic etiologies are more common [3] and include corticosteroid use, alcohol use, sickle cell disease, and hypercoagulable conditions [4], including thrombophilia, protein S deficiency, and protein C deficiency. Other, less common causes are listed in Table 1. In the pediatric patient population, diagnoses including Legg–Calve–Perthes (LCP) disease [8] and slipped capital femoral epiphysis (SCFE) [9, 10] are more common .

Table 1 Less common causes of osteonecrosis of the femoral head

Bone marrow replacing disease processes, such as Gaucher's disease [5]
Dysbaric disorders, such as decompression sickness (Caisson's disease, aka the "bends")
Systemic lupus erythematosus [6]
Inflammatory bowel disease
History of undergoing organ or tissue transplant [7]
Chronic renal failure
Pancreatitis
Pregnancy

Pathophysiology

The pathophysiology is poorly understood, making it difficult to prevent this condition. The pathogenesis can be multifactorial, with a combination of metabolic factors and local/host factors affecting blood supply to the femoral head. The disease process is thought to involve an interruption to the vascular supply of the femoral head, which causes adjacent hyperemia, demineralization of the bone, and trabecular thinning, leading to subchondral collapse. Specifically, coagulation of intraosseous microcirculation occurs, followed by resultant venous thrombosis and retrograde arterial occlusion, which increases intraosseous pressure, leading to decreased blood supply to the femoral head. This ultimately leads to death of osteocytes and osteoprogenitor cells, resulting in subchondral fracture and collapse. In cases with underlying traumatic etiologies, typically injury to the medial femoral circumflex artery is responsible. As such, osteonecrosis is most common in cases of displaced femoral head fractures, followed by basicervical femoral neck fractures; the highest risk being combined intrafoveal femoral head and displaced femoral neck fractures (Pipkin III). Hip dislocations are also associated with the development of osteonecrosis; however, if the joint is reduced within 6–8 h of injury, the rate of osteonecrosis is decreased [11, 12].

Clinical Presentation

History and Physical Examination

Patients with femoral head osteonecrosis can be asymptomatic and the diagnosis can be made from imaging performed for another indication. The vast majority of symptomatic patients complain of progressive pain localized to the groin and general hip region. While pain localized to the groin is most specific for intra-articular pathology, pain in the thigh and buttock is also commonly seen. These symptoms can be exacerbated with weight bearing and with combined flexion and internal rotation. Patients should be asked about prior history of trauma, surgery, corticosteroid use, ethanol use, and personal or family history of blood disorders. Sudden inability to bear weight associated with fevers, chills, weight loss, and/or night sweats warrant further workup for infectious, inflammatory, and or malignant underlying etiologies. Physical examination findings can often be nonspecific. Patients may walk with a mild antalgic gait favoring the affected side, but otherwise the majority of examination findings are normal during early stages of the condition. Any neurovascular deficits or isolated muscular weakness should be further worked up for other underlying etiologies.

Imaging Studies

All patients with suspected osteonecrosis of the femoral head should undergo a complete radiographic workup including an anterior–posterior (AP) view of the pelvis, an AP view of the affected hip, and a cross-table or frog-leg lateral view of the affected hip. Often, it is the lateral view that best demonstrates subchondral collapse and/or collapse. The most commonly utilized classification systems for osteonecrosis are based on radiographic findings and historically include the Ficat classification [13], and more recently the Steinberg classification (Table 2) [14]. Of note, patients who are diagnosed with AVN in another area of the body (i.e., humeral head) should always get hip radiographs as there is an increased risk of having concomitant (but asymptomatic) femoral head AVN [15]. Radiographs can remain completely normal for months after symptoms begin (Fig. 1). Early radiographic findings of osteonecrosis will show mild density changes within the femoral heads due to micro-infarcts with corresponding calcification. In both of these systems, stage III will show a crescent sign radiographically, indicating subchondral collapse (Fig. 2). Typically, advanced imaging with magnetic resonance imaging (MRI) is also employed if there is a high index of suspicion and/or the patient is at risk (i.e., alcoholic) despite normal radiographs. This modality can reveal changes due to osteonecrosis at earlier stages than plain radiographs are capable of showing. MRI has been shown to have excellent specificity and sensitivity with regard

Table 2 Steinberg classification system

0	Normal XR, normal MR
I	Normal XR, abnormal MR (and/or bone scan)
II	Cystic/sclerosis, abnormal MR (and/or bone scan)
III	Crescent sign (subchondral collapse), abnormal MR (and/or bone scan)
IV	Femoral head flattening, abnormal MR (and/or bone scan)
V	Narrowing of joint, abnormal MR (and/or bone scan)
VI	Advanced degenerative changes, abnormal MR (and/or bone scan)

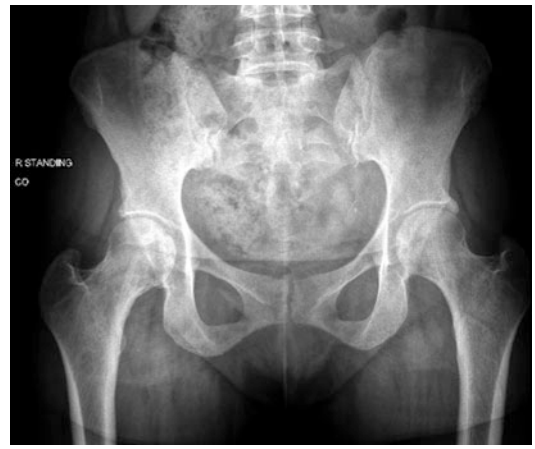

Fig. 1 AP radiograph of the pelvis demonstrating well-preserved joint space without evidence of collapse, cam, pincer, or dysplasia of the bilateral hips

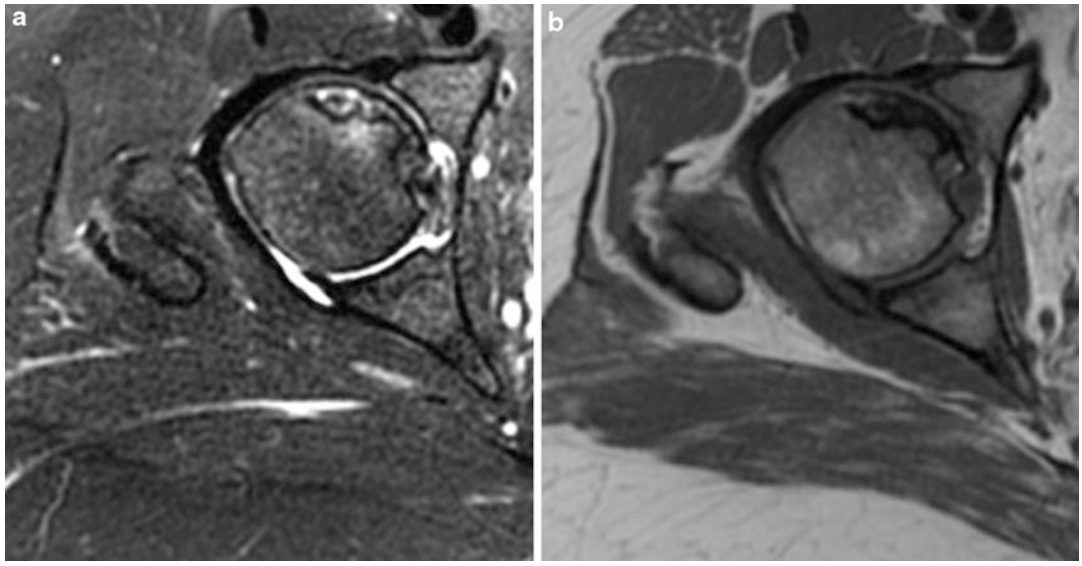

Fig. 2 Axial cuts from MRI demonstrating osteonecrosis (*ON*) of the right anterior superior femoral head (**a**: T2-weighted image, **b**: T1-weighted image)

to osteonecrosis and will typically appear bright on T2-weighted images with corresponding dark findings on T1-weighted images. Bone scan is also another modality that can be used to help diagnose and stage osteonecrosis of the femoral head, but is not as specific as MRI [16, 17].

Management Options

Based on symptoms and imaging findings, osteonecrosis of the femoral head is typically divided into early, or pre-collapse, versus late, or post-collapse, stages. Treatment options vary depending on the stage, with late stage presentations the most difficult to manage (from a non-arthroplasty perspective). Without any intervention, the natural history of osteonecrosis is that of ultimate disease progression, with most patients ultimately progressing to femoral head collapse and end-stage arthritis [18, 19]. Asymptomatic patients typically present in the pre-collapse stages and can be managed nonoperatively with close clinical follow-up. These patients must be watched closely, and the development of pain should prompt a repeat examination with repeat imaging studies.

Table 3 Surgical options for osteonecrosis of the femoral head

Core decompression
Rotational osteotomy
Vascularized graft transfer (free fibula)
Non-vascularized graft transfer
Total hip arthroplasty
Unipolar or bipolar hemiarthroplasty
Hip resurfacing

Patients who are symptomatic and in the pre-collapse stages will most often require intervention. While some medical therapies including marrow/stem cells [20–24] and bisphosphonate [25] use have been advocated, current results are inconsistent, and the majority of patients will undergo surgical intervention. Surgical options for these pre-collapse patients are outlined in Table 3. Non-arthroplasty options include core decompression, vascularized bone graft [26–30], non-vascularized bone graft, and osteotomies. Treatment options for post-collapse patients, however, are more limited, given the advanced stage of the disease. While most post-collapse osteonecrosis patients can be successfully managed with total hip arthroplasty [31], joint replacement may not be the best option in young, active

Table 4 Surgical steps

Establish anterolateral and anterior portals
Evaluate femoral head for subchondral collapse
Evaluate integrity of articular cartilage
Evaluate labrum and bony margin of acetabulum
Interportal or T-capsulotomy to address concomitant labral and/or femoral head–neck pathology
Maintain arthroscope in the joint to assist in avoiding subchondral penetration
Introduce drill and reamer under X-ray guidance, lesion typically in anterior superior femoral head
Introduce arthroscope into socket to confirm integrity of subchondral bone at lesion site
Implant ceramic putty to provide compressive strength
Perform dynamic arthroscopy of femoral head

patients, given the obvious concern for early polyethylene wear with subsequent osteolysis, component loosening, osteolysis, and the potential need for early revision.

Core decompression is a surgical technique in which the necrotic lesion is reamed or drilled to decrease local intraosseous pressure and stimulate a vascularized healing response. Multiple authors have demonstrated effective results with this treatment strategy for pre-collapse osteonecrosis, including arthroscopic-assisted decompression [32–34]. Given recent advances in techniques and instrumentation, hip arthroscopy is now the gold standard for the diagnosis of intra-articular hip pathology [35, 36]. Substantial improvements in hip-specific diagnostic modalities have improved the understanding of bony and soft tissue pathology in patients with intra-articular hip disorders. The association of concomitant soft tissue and/or bony pathology with osteonecrosis of the femoral head is currently unknown. Further, the ability of imaging studies to predict collapse and/or associated hip pathology in addition to osteonecrosis at the time of surgery is also unknown. Thus, arthroscopy at the time of decompression provides an accurate means to confirm the presence or absence of femoral head subchondral collapse, chondral delamination, and associated labral, capsular, and synovial pathology [37–39]. If present, such concomitant pathology can be addressed at the time of decompression and obviate the need for a subsequent surgery. Further, arthroscopy allows for verification and guidance during drilling and/or reaming to avoid penetration of the articular surface. The following section describes the surgical technique [40] for arthroscopic-assisted core decompression of the femoral head (Table 4).

Arthroscopic-Assisted Core Decompression: Surgical Technique

Patient Positioning and Surgical Setup

The patient is positioned supine on a traction table with a well-padded perineal post placed in the groin between the legs (Smith and Nephew hip traction system, Smith and Nephew, Andover, MA). Traction is applied with the leg in neutral extension, axially distraction, and adduction to provide a cantilever moment to the operative hip.

Landmarks

The greater trochanter and anterior superior iliac spine borders are palpable landmarks used to identify appropriate portal placement. These are marked on the skin.

Portals

Under fluoroscopic visualization, a standard anterolateral (AL) portal is created 1 cm proximal and 1 cm anterior to the AL aspect of the greater trochanter. The 70° arthroscope is inserted over a guidewire. While viewing from the AL portal, needle localization is used to establish an anterior portal, penetrating the capsule at the 2 o'clock position. The anterior portal is approximately 1 cm lateral to the line drawn vertically from the anterior superior iliac spine (ASIS) and a line drawn horizontally from the AL portal.

Diagnostic Arthroscopy

Diagnostic arthroscopy begins with the arthroscope in the AL portal (Dyonics Arthroscopy

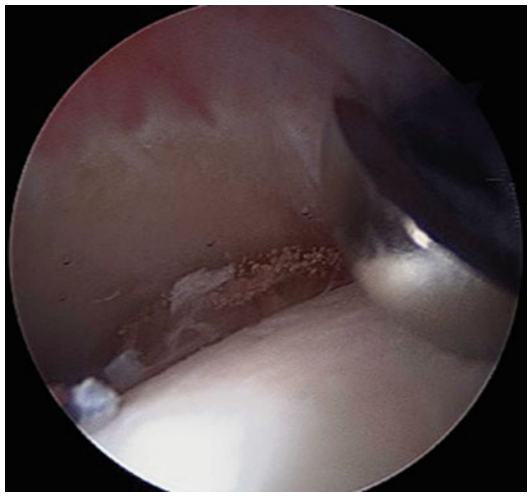

Fig. 3 Femoral head articular surface, visualization through the anterolateral (*AL*) portal

System, Smith and Nephew, Inc., Andover, MA) (Fig. 3). The posterior-superior and posterior-inferior labrum, superior and lateral femoral head, and acetabular articular surface are evaluated for a labral tear or chondral lesion. Any unstable tissue or cartilage is probed through the anterior portal to determine instability. The arthroscope is then switched to the anterior portal and the remainder of the labrum, acetabulum, and femoral head are visualized. The anterior portal is best for visualizing the anterior superior femoral head, the most common site of osteonecrosis. It provides a direct "bird's-eye" view. The site, however, can also be visualized from the AL portal with the 70° arthroscope lens. Interportal capsulotomy is reserved for circumstances where labral or articular cartilage work must be performed such that increased arthroscope and instrumentation mobility is warranted.

Throughout each of the steps (guide pin placement, reaming, curettage, and putty placement), the arthroscope is maintained in the AL portal, focused on the articular side of the area of necrosis, to ensure that the subchondral bone and articular cartilage are not violated. The 70° arthroscope enables visualization of the articular side of the lesion so that combined fluoroscopic imaging can be performed to evaluate debridement and filling of the socket without the radiopaque arthroscope obscuring fluoroscopic visualization if maintained in the anterior portal.

Fluoroscopic Confirmation of the Lesion

Fluoroscopic assistance confirms the location of the osteonecrotic lesion. The articular surface is evaluated and probed for any depression, softening, and/or delamination. If softening is present, a probe is used to measure the size, stability, and depth of the lesion. This is typically performed through the anterior portal while visualizing through the AL portal. The arthroscope is then switched back to the AL portal, while any instrumentation in the anterior portal is removed to allow for adequate visualization of the femoral neck on AP and frog-leg lateral fluoroscopic views. A spinal needle is placed into the anterior portal to allow for outflow and the pressure of the inflow is reduced to 40 mmHg.

Core Decompression

Core decompression is then performed through a separate 3 cm incision distal and posterior to the AL portal. This incision is created with fluoroscopic guidance. Blunt dissection is subsequently carried down to the tensor fascia lata, which is incised longitudinally. The vastus lateralis muscle belly is elevated anteriorly to expose the proximal intertrochanteric portion of the femur.

Guide Pin Placement

The starting point of the guide pin is biased posterior to the midpoint of the proximal femur in the sagittal plane and proximal to the lesser trochanter. With arthroscopic visualization,

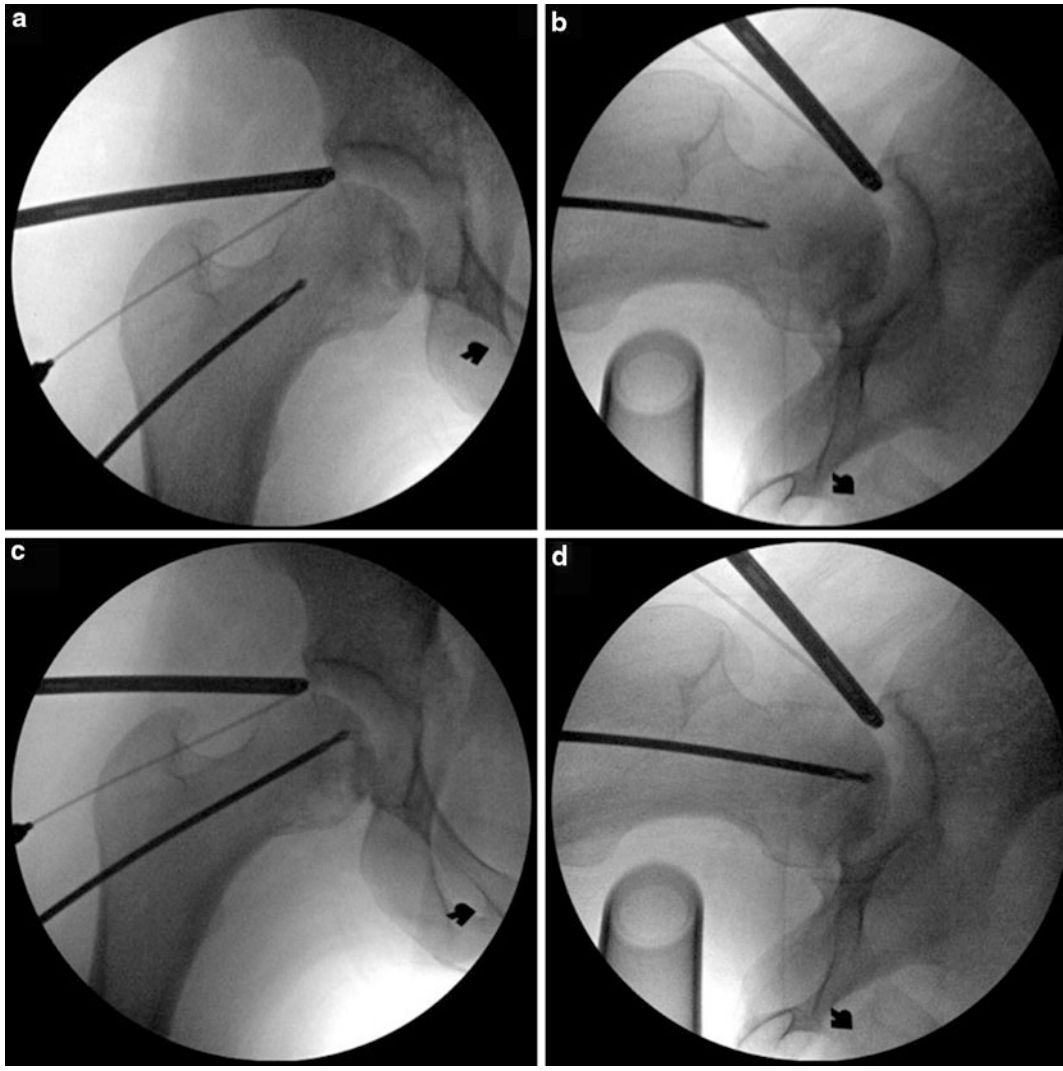

Fig. 4 Intraoperative fluoroscopic imaging demonstrating guide pin placement into the center of lesion (**a** and **b**, AP and lateral, **c** and **d**, AP and lateral after pin advancement)

a probe is positioned over the area of cartilage softening, and the guide pin is directed toward the area of osteonecrosis. The trajectory is posterolateral to anteromedial to penetrate the necrotic lesion. The guide pin advanced to 1–2 mm deep to the subchondral bone (Fig. 3a). Appropriate positioning in the femoral head is confirmed with anterior–posterior (AP) and cross-table lateral radiographs (Fig. 4).

Introduction of Reamer

A soft tissue guide is placed over the guide pin and a reamer is advanced to the same depth (Advanced Core Decompression System, Wright Medical Technology Inc., Arlington, TN). Careful attention is paid not to allow the guide pin or reamer to advance beyond the subchondral bone. Confirmation that the reamer has penetrated the necrotic

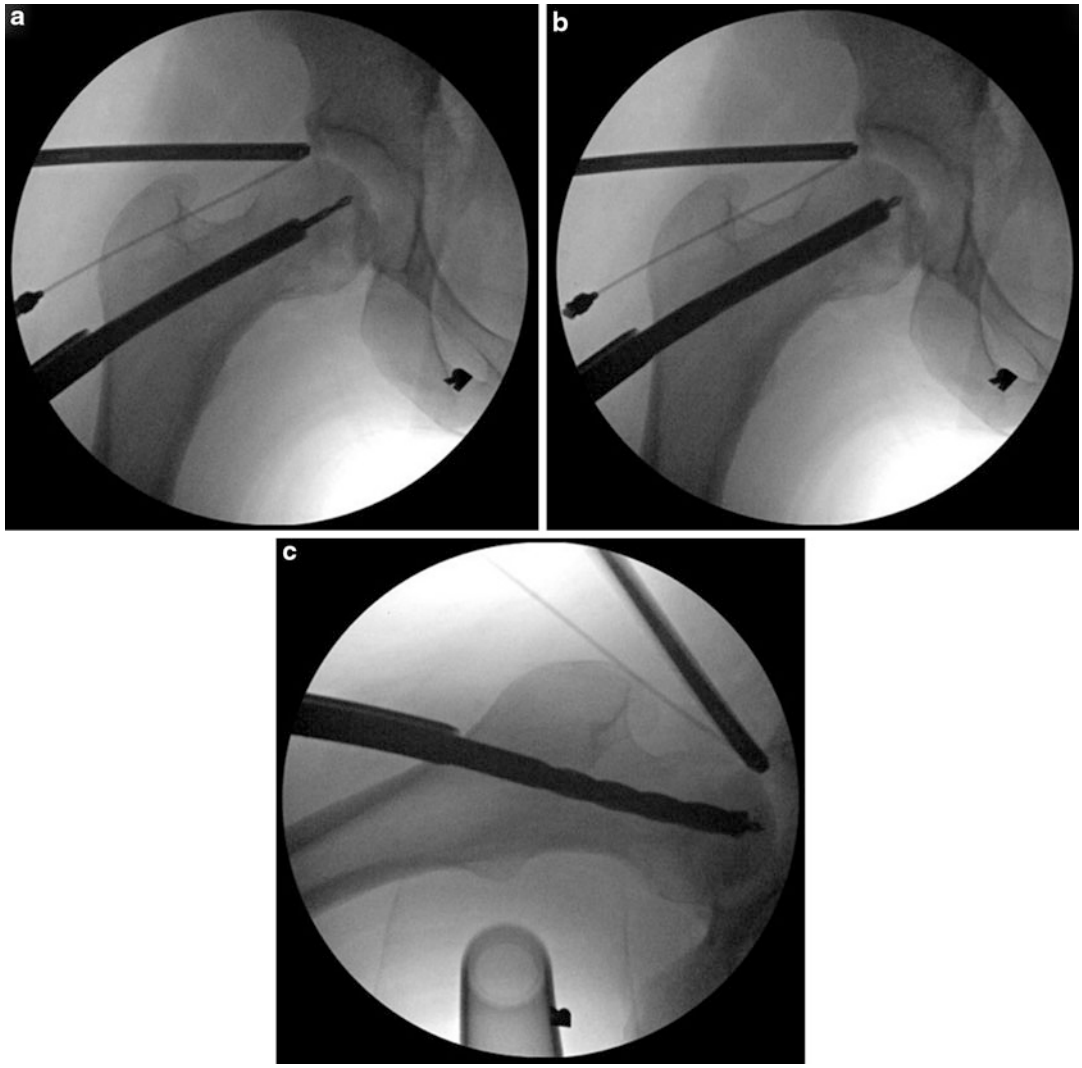

Fig. 5 Intraoperative fluoroscopic imaging demonstrating introduction and advancement of reamer over pin

lesion is obtained fluoroscopically as well as by tactile sensation – the surgeon will feel resistance while reaming the sclerotic bone (Fig. 5).

Introduction of 30° Curved Curette

The pin/reamer is removed and a 30° curved curette is advanced up the socket and the residual necrotic bone is removed (Fig. 6).

Insertion of Syringe into Socket

A syringe is inserted into the socket and combination calcium phosphate (CaPO4)/calcium sulfate (CaSO4) synthetic putty (ProDense®, Wright Medical Technology Inc., Arlington, TN) is injected into the socket [41]. It is injected in a retrograde fashion, allowing the pressure of the putty to guide the syringe (Fig. 7). Intra-articular arthroscopic visualization confirms no

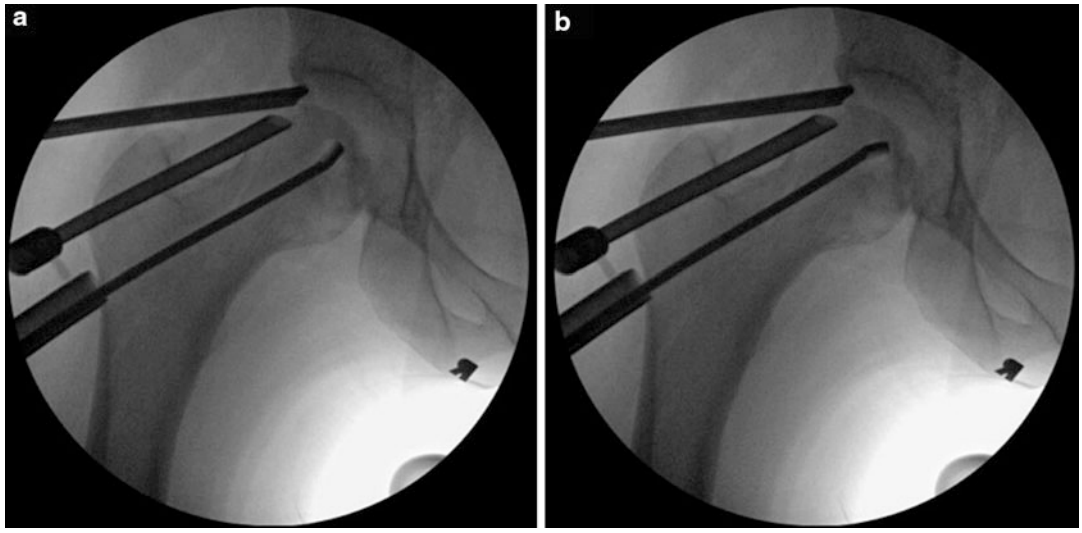

Fig. 6 Intraoperative fluoroscopic imaging demonstrating curettage of lesion

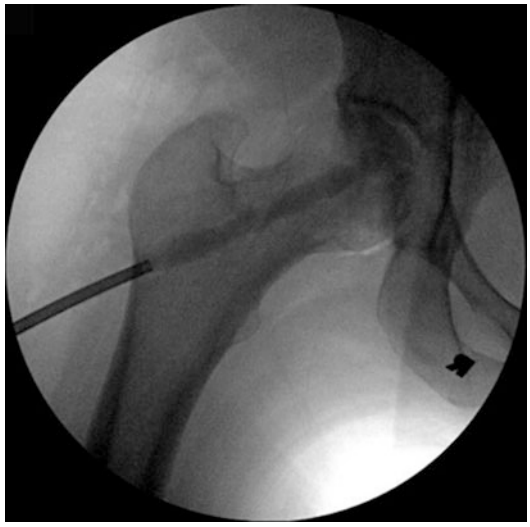

Fig. 7 Intraoperative fluoroscopic imaging demonstrating filling of socket with calcium sulfate and calcium phosphate cement mixture

intra-articular penetration of the putty. Final AP and lateral fluoroscopic views are obtained verifying fill of the socket (Fig. 8). Once the putty is cured (as evidenced on the side table), instruments are removed from the hip, and traction is released. Dynamic fluoroscopy is performed verifying joint reduction.

Closure

The tensor fascia lata is closed with 0 absorbable braided suture, the dermis with 2-0 braided absorbable suture, and the skin (along with portals) with 3-0 monofilament suture.

Postoperative Course

Postoperatively the patient is maintained touchdown weight bearing with crutches for 6 weeks. Immediate passive range of motion is begun the evening of surgery with continuous passive motion. At 6 weeks the patient is progressed to weight bearing as tolerated. Return to athletic activity is delayed for a minimum of 3 months.

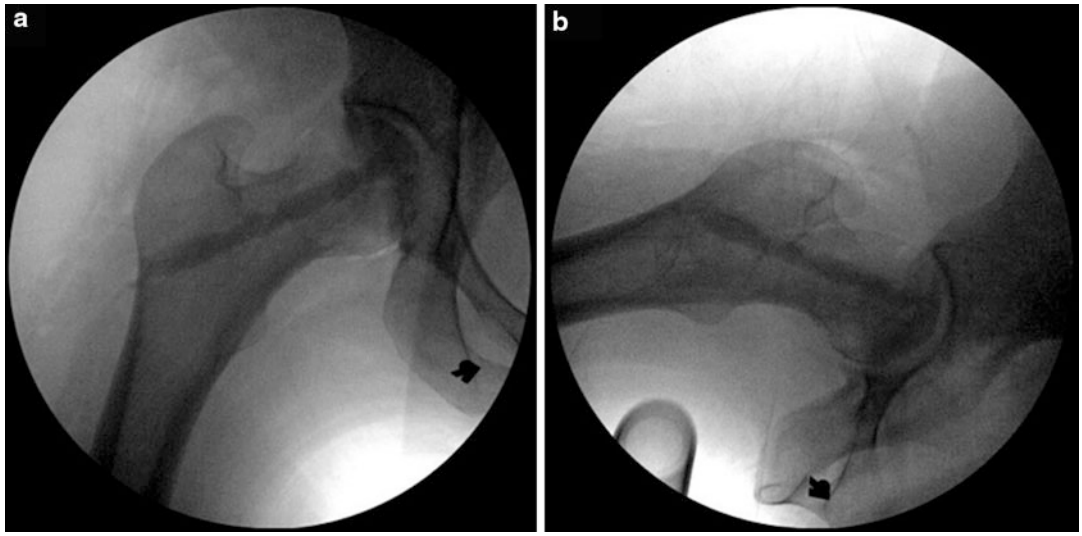

Fig. 8 Intraoperative fluoroscopic imaging verifying fill of the socket

Summary

Core decompression is a technique that theoretically decreases intraosseous pressure of the femoral head, resulting in a local vascularized healing response. Arthroscopic-assisted core decompression of the femoral head is an effective treatment option for pre-collapse osteonecrosis of the femoral head. Arthroscopy at the time of decompression provides the added advantage of intra-articular visualization to confirm the diagnosis, allow for treatment of associated bony and soft tissue pathology, and avoid the risk of joint penetration in carefully selected patients.

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