Arthroplasty Tips & Tricks: Rapidly Progressive Avascular Necrosis of the Femoral Head Following a Single Intra-articular Corticosteroid Injection

Introduction

Avascular necrosis (AVN)—also known as osteonecrosis or aseptic necrosis—is the death of bone due to a disruption in its blood supply.\(^1\) It most commonly involves the femoral head, but may also involve other sites such as the femoral condyles, humeral head, or talus.\(^2\) Males are more likely to be affected than females and it typically occurs in patients 30 to 60 years old.\(^2\)

While the pathophysiology of AVN has been studied extensively, there is no general consensus on the specific etiology. The inciting “event” can be broadly classified as traumatic or atraumatic. Such events ultimately lead to disruption of the bone microcirculation via three primary mechanisms: mechanical vascular interruption, intravascular occlusion, or extravascular compression.\(^1,3\)

Many of the commonly cited risk factors for AVN—such as trauma or hemoglobinopathy—can be easily understood when considering the mechanisms of osseous vascular disruption. For instance, mechanical disruption typically occurs following trauma. The medial circumflex femoral artery, the main blood supply to the adult femoral head, can be disrupted as a result of fractures involving the proximal femur or hip dislocation.\(^4\) Hemoglobinopathies, such as sickle cell anemia, are classically cited as a cause of intravascular occlusion. The sickled erythrocytes may become mechanically sequestered in the low flow areas of the femoral head, thus leading to vaso-occlusion.\(^1,5\)

A lesser understood phenomenon is AVN resulting from corticosteroid use. Numerous theories exist regarding the exact pathophysiology, but the most accepted theory is that the use of corticosteroids leads to accumulation of fat within the bone marrow resulting in intraosseous hypertension and decreased perfusion.\(^1,5,6\) This is most commonly discussed in the context of patients on long term, high dose oral steroids for maintenance of systemic diseases such as systemic lupus erythematosus, irritable bowel disease, and rheumatoid arthritis.\(^8,9\) Less often, AVN can be seen after topical steroid use or intra-articular steroid injection; these instances are limited to case reports.\(^10-16\)

We present a case of rapidly progressive avascular necrosis of the femoral head following a single intra-articular corticosteroid injection.

Case Report

The patient is a 74-year-old female with a past medical history significant for lumbar degenerative disc disease who presented to an outside hospital orthopaedic surgeon’s office with right groin, lateral hip, and buttock pain. Imaging at that time was consistent with moderate osteoarthritis of the right hip as well as degenerative disc disease of the lumbar spine. Given her concomitant lumbar and hip findings on radiographs, the patient underwent a diagnostic and therapeutic image-guided corticosteroid injection to the right hip at that time.

She reported complete resolution of her pain for approximately two weeks, but the pain gradually returned shortly thereafter. Her imaging was repeated at this time and the findings were unchanged from prior (Figures 1 and 2). Three weeks after the injection, her pain was worse than before her injection and she was unable to ambulate. She returned to her surgeon’s office and a bone scan obtained approximately 11 weeks following the injection showed increased uptake in the right hip and pelvis. Magnetic resonance imaging (MRI) of the right hip was then obtained approximately 15 weeks following the injection, showing complete destruction of the femoral head and a fluid collection in the soft tissue anterior to the hip joint (Figure 3). The patient was then referred to our office for further management.

The patient presented to our office approximately 16 weeks following the injection. At that time, plain radiographs demonstrated complete destruction of the right femoral head (Figure 4). Differential diagnoses included septic arthritis, metastatic disease, primary bone malignancy, or Gorham’s disease. An infectious work up that included complete blood cell count, erythrocyte sedimentation rate, C-reactive
protein, and subsequent right hip aspiration were all negative for infection. The patient was also discussed at our institution’s weekly orthopaedic oncology conference, and there was no concern for an oncologic etiology.

Given the patient’s pain and limited function, the decision was made to proceed with right total hip arthroplasty (THA) versus placement of a right hip antibiotic spacer. Despite a negative infectious work-up preoperatively, the patient was advised that intraoperative right hip aspiration and frozen sections would be obtained to guide decision-making.

The patient was taken to the operating room approximately 19 weeks following the injection. Right hip aspiration and intraoperative frozen sections were not consistent with infection. The decision was made to proceed with right THA (Figure 5). The patient’s immediate postoperative course was uneventful. The patient was most recently seen over two years following her THA. She reports 0/10 right hip pain with no symptoms of infection or instability. Her Hip disability and Osteoarthritis Outcome Score for Joint Replacement (HOOS, JR.) improved from 43.34 preoperatively to 92.34 postoperatively.

**Discussion**

Avascular necrosis of the femoral head following intra-articular corticosteroid injection is an uncommon phenomenon. There is a paucity of data surrounding this topic, with case reports comprising the existing literature.11,16

There are seven reported cases in the literature for femoral head AVN following a single intra-articular steroid injection.11 In 2006, Yamamoto et al. reported the first case in a 50-year-old female who received a single intra-articular steroid injection for the management of hip osteoarthritis; the patient developed AVN of the femoral head three months following the injection.12 In each of the reported cases thereafter, patients developed rapidly progressive
As studies uncover more information regarding the pathophysiology of avascular necrosis, researchers have begun looking at pharmacologic interventions to slow or even halt the process. Recent studies have demonstrated the potential benefit from using bisphosphonates, lipid lowering agents, and vasodilators. However, more robust data from randomized control trials are needed to truly elucidate the utility of these treatment options.

Operative intervention can be broadly classified as joint preserving versus joint replacing. If a patient has a small- or medium-sized femoral head lesion without collapse, this most commonly is treated with core decompression. While the technique can vary, the principle of core decompression is to decrease intraosseous hypertension. The two most common techniques involve using either a trephine or percutaneous drilling to decompress the femoral head lesion and increase blood flow to the necrotic lesion. Studies comparing these techniques involve using either a trephine or percutaneous drilling to decompress the femoral head lesion and increase blood flow to the necrotic lesion.

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AVN of the femoral head shortly after a single intra-articular corticosteroid injection with no prior history of steroid use. The previously reported cases developed AVN between four and 14 weeks following the injection. The primary treatment modality for these patients was arthroplasty; six patients were treated with total hip arthroplasty and one patient was treated with hemiarthroplasty.

The management of avascular necrosis varies based on radiographic severity and age. The initial management for patients with femoral head lesions without collapse is to limit weightbearing using a walker or cane. However, nonoperative treatment is rarely used in isolation due to poor success rates. Mont et al. reviewed 21 studies utilizing nonoperative management as the primary treatment modality for AVN and found only 22% of patients avoided surgery at 34 months. As such, weightbearing modification is typically only one facet of a treatment plan.

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two techniques in sickle cell patients with AVN have shown equivalent odds of improvement, and the treating surgeon should perform the technique in which they feel the most comfortable.  

Core decompression can also be combined with autologous bone marrow grafting. A prospective study by Hernigou et al. followed 189 hips in 116 patients for five to 10 years after undergoing core decompression with autologous bone marrow grafting. One-hundred forty-five patients underwent the procedure prior to femoral head collapse; only nine of these patients went on to a total hip replacement. Twenty-five of the 44 hips that underwent the procedure after femoral head collapse required a total hip replacement during the follow up period.  

Bone-grafting—both nonvascularized and vascularized—has shown promise in treating AVN of the femoral head. The theory behind nonvascularized fibular bone grafting was that it would provide structural support to the femoral head and serve as a scaffold for the formation of new bone in the defect. A retrospective review of 39 hips with avascular necrosis treated with nonvascularized bone grafting with supplemental bone morphogenic protein 7 (BMP-7) demonstrated no need for further surgery at 24 months in 80% of small- and medium-sized lesions.  

Advances in microvascular surgery have paved the way for vascularized bone grafting, with the thought that vascularized bone grafting not only provides structural support to prevent femoral head collapse, but also the blood supply to encourage healing. A randomized control trial by Cao et al. comparing vascularized free fibula grafting to core decompression showed that patients who underwent vascularized free fibula grafting had improved vascularity and decreased progression of AVN compared to the patients that underwent core decompression. However, at 36 months there was no difference in the rate of THA between the two groups. Although surgeons are optimistic that vascularized bone grafting will be a reliable joint-preserving procedure in the future, further investigation is necessary to identify appropriate candidates.

For the larger, collapsed lesions or if the patient has radiographic findings consistent with arthritic changes of the acetabulum, joint replacement remains the gold standard of treatment. With AVN occurring in younger, more active patients, there were significant concerns regarding implant survivorship. However, with advances in implant technology and surgical techniques, this has been significantly improved. A study comparing THAs performed for osteonecrosis showed an improvement in revision rates from 17% to 3% before and after 1990, respectively. Results continue to be promising, as a recent comparative study examining outcomes for THA performed for AVN versus osteoarthritis showed no significant difference in functional outcomes, implant survival, and rate of complications at ten years when matched for age and sex. Given our experience with this case, we have several
recommendations for AVN patients with femoral head collapse following an intra-articular corticosteroid injection. The treating physician should utilize all nonoperative modalities to manage the patient's hip osteoarthritis prior to recommending an intra-articular corticosteroid injection. If a patient receives an intra-articular steroid injection and reports worsening pain, radiographs should be obtained at that time. MRI and bone scan can be helpful in making the diagnosis in these cases involving severe bone loss. Infectious etiologies should be ruled out as well. If the patient has persistent pain with radiographic findings consistent with AVN with femoral head collapse, the authors recommend proceeding with operative management. The treating surgeon should obtain intraoperative frozen sections to rule out infection and be prepared to proceed with antibiotic spacer placement if intraoperative frozen section is consistent with infection. If findings are not consistent with infection, we recommend proceeding with total hip arthroplasty.

Given the numerous reports in the literature over the last 15 years, there is a clear link between rapidly progressive avascular necrosis of the femoral head and intra-articular steroid injections. Further studies are required to fully understand this association and the authors advocate for such investigations.

References