SAS Weekly Rounds: Avascular Necrosis

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Abstract Osteonecrosis of the femoral head is a condition that affects upwards of 10,000 individuals in the USA each year. The peak incidence is in the fourth decade of life, and overall, there is a male preponderance. The condition accounts for up to 12% of total hip arthroplasties performed in developed countries. The etiology can be traumatic or non-traumatic, with 90% of atraumatic cases attributed to corticosteroid therapy or excess alcohol consumption. Osteonecrosis of the femoral head reflects the final common pathway of a range of insults to the blood supply and ultimately results in femoral head collapse, acetabular involvement, and secondary osteoarthritis. Currently, conservative treatment options, which aim to correct pathophysiologic features allowing revascularization and new bone formation, appear to be able to delay but not halt the progression of this condition. As a consequence of femoral head osteonecrosis, many individuals undergo surgical treatments including: core decompression, osteotomy, non-vascularized bone matrix grafting, free vascularized fibular grafts, limited femoral resurfacing, total hip resurfacing, and total hip arthroplasty.

Keywords osteonecrosis · avascular necrosis · femoral head

Case report

DM is a 56-year-old man with HIV/AIDS and osteonecrosis of the hip admitted for total hip arthroplasty.

The patient was initially evaluated by rheumatology for acute gouty arthritis of the ankle at the time of an admission to New York Presbyterian Hospital for pneumonia complicated by respiratory failure and *Pseudomonas* sepsis. After his extensive intensive care unit course, as he was mobilized, he developed pain in the left knee, left hip, and left thigh. His symptoms subsided then migrated to the right side. He had been walking independently, but progressed to require a cane, and subsequently a walker. The pain became progressively more severe, prompting him to seek surgical care. He was evaluated in the Surgical Arthritis Service (SAS) clinic.

His past medical history is significant for HIV. He has been on highly active anti-retroviral therapy (HAART) with Viracept 1250 BID and Epzicom 600/300 mg daily. At the time of this SAS evaluation, his CD 4 count was 473 and his viral load was undetectable. His nadir CD4 was six at the time of his admission for pneumonia and sepsis. In the past, he had been treated for hepatitis C with ribavirin, prednisone, and azathioprine. He has insulin-dependent diabetes managed with lispro and glargine insulin. He had a remote history of intravenous drug use and cocaine abuse. He has well-controlled hypertension. Medications at the time of his SAS evaluation included glargine and lispro insulin, Cozaar, Viracept, Epzicom, methadone, and Percocet.

The SAS evaluation revealed that he had difficulty walking due to pain and was using a walker. His pain was greater on the right than the left. Range of motion of the hips revealed limited flexion of the right hip to 40°. Internal rotation was absent, and he had 30° of external rotation. Flexion on the right was painful and restricted with flexion to 60°, absent internal rotation, and external rotation to 60. Distal strength was normal. X-rays were obtained and revealed avascular necrosis with subchondral fracture and collapse of both hips. Total hip arthroplasty was recommended.

He was admitted to the Hospital for Special Surgery for total hip arthroplasty, which he underwent without complication. The surgery was performed with a small posterolateral approach. A Smith and Nephew non-cemented Reflection acetabular component with an elevated highly cross-linked polyethylene liner and an Anthology non-cemented femoral component with a 36-mm Oxinium femoral head were implanted. The femoral head had

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significant avascular changes with collapse and secondary arthritic changes. Viral load at the time of surgery was 7,000. He had a ConstaVac drain placed which was removed on postoperative day 1. He received vancomycin for antibiotic prophylaxis due to a penicillin allergy. He received Coumadin for DVT prophylaxis. Postoperatively, he was instructed to ambulate weight bearing as tolerated and was discharged home on postoperative day 5.

He was discharged home for follow-up in SAS clinic with the expectation of admission in 3 to 6 months for left hip arthroplasty.

Discussion

Osteonecrosis of the femoral head, also known as aseptic or avascular necrosis, was first described in 1738 by Alexander Munro, a professor of anatomy at Edinburgh University. It was not until 1829 that Cruvilhier described how the femoral head pathology seen in this condition was secondary to impaired osseous blood flow [1]. Osteonecrosis affecting the femoral head frequently presents in the third, fourth, or fifth decade, with the mean age being 38 years [2]. With the exception of cases associated with systemic lupus erythematosus (SLE), there is a male preponderance and a male to female ratio of 7 to 3 [3]. The first series of 27 cases of femoral head osteonecrosis was described by Mankin and Brower [4] in 1962, and since then, there has been a steady increase in reported cases, in part due to better diagnostic techniques. Between 10,000 and 20,000 cases are reported annually in the USA with an estimated prevalence of up to 600,000 [2]. Overall, the diagnosis of osteonecrosis accounts for 5% to 12% of total hip arthroplasties (THA) performed each year [5–9].

Neither the etiology nor natural history of osteonecrosis has been defined. Osteonecrosis can be associated with traumatic or non-traumatic insults to femoral head blood supply. The medial circumflex artery is the primary blood supply to the femoral head. Arising from the profunda femoris artery, its course is around the medial side of the femur, passing between pectineus and iliopsoas and then between the obturator externus and adductor brevis. The deep branch then runs close to the tendon of obturator externus before running under the posterior aspect of the hip capsule, the zona orbicularis before passing antro-medially along the neck until it reaches the posterior capsule. Here, the terminal branches, the lateral epiphyseal arteries, enter the femoral head postero-superiorly supplying the epiphysis. In both sub-capital fractures and hip dislocation, the blood supply from deep branches of the medial circumflex artery is at risk of interruption.

Displaced fractures and dislocations of the hip are associated with mechanical interruption to the circulation of the femoral head. Fractures of the femoral neck have been associated with a 15% to 50% prevalence of osteonecrosis depending on fracture type, time until reduction, and accuracy of reduction [8, 10–12]. The prevalence of osteonecrosis following hip dislocation

ranges from 10% to 25% and is related to the duration of dislocation, with prompt reduction halving the prevalence seen if reduction is delayed for 12 h [13, 14].

Risk factors for osteonecrosis can also be classified into those that have a proven direct association where a cause effect relationship has been firmly established, those with a strong association, and those with a probable association (Table 1). Osteonecrosis can occur following a single insult such as a fracture or dislocation as previously described, but in most cases is the result of prolonged episodes of minor damage to the vascular supply to the femoral head.

The two most common risk factors of osteonecrosis are corticosteroid therapy and alcohol consumption, which account for as many as 90% of new cases of atraumatic osteonecrosis [2, 15, 17]. In cross-sectional studies, 10% to 30% of cases of osteonecrosis have been associated with corticosteroid therapy, and longitudinal studies indicate that 8% to 10% of individuals taking corticosteroids will develop osteonecrosis [18]. Doses of greater than 20 mg/day, even for a short period, have been associated with a higher risk. A meta-analysis by Felson and Anderson [18] revealed a 4.6-fold increase in the rate of osteonecrosis for every 10-mg/day increase in mean daily dose. The mechanism of steroid

Table 1 Risk factors for osteonecrosis [16]

Direct association	
Trauma	Cassion disease
Fractures	Gaucher's disease
Dislocations	Major arterial disease
Vascular injury	Sickle cell disease
Thermal injury	Thalassemia
Kienböck disease	Post-irradiation
Strong association	
Alcohol consumption	Corticosteroid therapy
Probable association	
Cigarette smoking	Iatrogenic
Connective tissue disease/ rheumatologic	Hemodialysis
Ankylosing spondylitis	Organ transplantation
Ehlers–Danlos syndrome	Laser surgery
Polymyalgia rheumatica	Highly active antiretroviral therapy
Polymyositis	Infectious
Raynaud's disease	Osteomyelitis
Rheumatoid arthritis	HIV
Sjögren syndrome	Meningococemia
Septic lupus erythematosus	Metabolic and endocrine
Gastrointestinal	Chronic renal failure
Inflammatory bowel disease	Cushing disease
Pancreatitis	Diabetes
Obesity	Fabry's disease
Starvation	Gout and hyperuricemia
Hematologic and vascular	Hypercholesterolemia
Arteriosclerosis	Hyperparathyroidism
Disseminated intravascular coagulation	Lipodystrophies (hyperlipidemia)
Giant cell arteritis	Neurological injury
Hemophilia	Neoplastic
Hypofibrinolysis	Orthopedic
Idiopathic thrombocytopenia	Congenital hip dislocation
Leukemia	Developmental dysplasia of the hip
Polycythemia	Hereditary dysostosis
Post-venous thrombosis	Legg–Calvé–Perthes disease
Thrombophillia	Osteoporosis/osteomalacia
Thrombophlebitis	Slipped femoral capital epiphysis
Vasculitis	Pregnancy

induced osteonecrosis still remains unclear. In 1964, Johnson proposed that lipocyte hypertrophy, induced by steroids, leads to increased pressure within the femoral head resulting in sinusoidal vascular collapse and ultimately necrosis [19, 20]. Others argue that the increased lipid content of the femoral head is a consequence of fat micro-emboli that disrupt the delicate blood supply to the femoral head [21, 22]. More recently, it has been shown that steroids may have a direct detrimental effect on endothelial and smooth muscle cells within the vasculature impeding venous drainage from the femoral head [23]. Lipocyte hypertrophy, fat emboli, and venous malfunction are all seen in osteonecrosis; however, it is still unclear how much each contributes to the pathophysiology of this condition.

Alcohol consumption has also been associated with increased rates of osteonecrosis [2]. A study by Matsuo et al. [17] demonstrated that an intake of greater than 400 ml alcohol per week, around three glasses of alcohol, 6 units, per day, increased the relative risk 9.8-fold compared to non-drinkers. Again, the pathophysiology of alcohol-induced osteonecrosis is not completely understood, but like corticosteroids, it is thought to be associated with changes in lipid metabolism within the femoral head [23, 24].

There is a large group of conditions where there is a probable association with osteonecrosis. The cause effect relationship is more difficult to establish in individual patients due to associated steroid use. Examples include connective tissue disorders including rheumatoid arthritis, SLE and ankylosing spondylitis, vascular disorders including giant cell arteritis, idiopathic thrombocytopenic purpura, and vasculitis orthopedic conditions including developmental dysplasia of the hip, congenital hip dislocation, hereditary dystosis, Legg–Calve–Perthes disease, and slipped capital femoral epiphysis have a likely but unproven association. Some 10% to 20% of cases of osteonecrosis are classified as idiopathic [1]. A study by Wheelless et al. [25] of the vascular anatomy of 99 hips with advanced osteonecrosis demonstrated that 94% had an abnormal vascular supply compared to 31% in the control group. Given that a reduction of femoral head blood supply by 1.6-fold reduces PO_2 by a third, it is possible that individuals with anomalous femoral head blood supply are more susceptible to future insults that may lead to necrosis [26]. Alternatively, individuals carrying genetic mutations which lead to hypercoagulability may develop osteonecrosis when triggered by environmental stimulate.

Patients infected with HIV, such as our patient, have a higher incidence of osteonecrosis than the general population. Although the incidence of HIV-associated osteonecrosis has increased since the advent of HAART, cases have been reported prior to its use. No correlation has been seen with viral load, CD4 cell count, or age. HIV-infected patients with osteonecrosis are likely to have other well-defined risk factors for osteonecrosis. Hypercoagulability is common in the HIV-infected population, given a prevalence of anticardiolipin and antiphospholipid antibodies reported in up to 50% of patients. Corticosteroids are used for multiple indications in HIV-infected patients and have been implicated in the development of osteonecrosis in the HIV.

Alcohol abuse and altered lipid metabolism are also seen in this population and may contribute to the increased incidence of osteonecrosis. The increase in osteonecrosis in the HIV-infected patients may simply reflect the increase in other risk factors in this population, as is demonstrated in this case [178, 179].

Aaron [27] proposed a mechanism that integrates these risk factors demonstrating that osteonecrosis is not a specific diagnostic entity but rather a final common pathway of a series of derangements that produce decreased blood flow leading to cellular death within the femoral head. Vascular interruption secondary to trauma as well as thrombotic occlusion and extravascular compression all result in decreased blood flow which in turn leads to ischemia, osteocyte necrosis, loss of structural integrity, and, ultimately, collapse of the femoral head.

Osteonecrosis can be clinically silent or can present with any number of manifestations. The chief complaint is often pain localized to the groin area but, occasionally, the ipsilateral buttock or knee. It has been described as deep, intermittent throbbing pain with an insidious onset that can be sudden. Physical examination reveals a coxalgic gait and pain with hip range of motion. Pain with internal rotation, clicking of the hip, and a reduced range of motion are often signs that the femoral head has already collapsed [6].

Histologically, in the early stages of the disease, examination of the femoral head shows bone marrow necrosis. Following the initial ischemic insult, the bone marrow cells die at different intervals. Hematopoietic cells die within 6 to 12 h followed by osteocytes at 12 to 48 h and marrow fat cells at day 5 [28]. When the repair process begins, the dead osteocytes are reabsorbed and empty lacunae are seen within the bone. Next, osteoblasts lay down new bone over the necrotic areas leading to the characteristic appearance termed “creeping substitution.” Histologically, this process of reabsorption and repair can be observed as early as 3 days following vascular insult. In small lesions, necrosis and repair continues simultaneously; however, in larger lesions, repair is impeded within the large necrotic avascular core, leading to marked thickening and increased density of its borders. This in turn leads to loss of structural integrity and subchondral collapse.

Important first-line investigations are anteroposterior and lateral hip x-rays; however, due to the relatively slow process of bone remodeling, it is important to note that there may initially be no radiographic changes visible [29, 30] (Fig. 1). Magnetic resonance imaging (MRI) is much more sensitive and can detect changes in femoral head fat content, which is seen as early as 5 days after vascular insult when adipocytes undergo necrosis. This is reflected as loss of the normal high intensity signal (Fig. 2). MRI is considered to be the gold standard diagnostic test with a sensitivity and specificity of 99% [6]. Bone scans have been used in high-risk groups to detect early remodeling but can be misleading, with high levels of false negative between 25% and 45% in cases subsequently confirmed by MRI or histological diagnosis [31, 32]. This is due to the transition period between the initial ischemic insult, seen as cold on bone scan, and the start of remodeling when the lesion is

Fig. 1. Anteroposterior and lateral radiographs of a patient who presented with bilateral osteonecrosis secondary to HIV infection. The right hip demonstrates a reactive sclerotic rim surrounding a lytic necrotic area with segmental articular collapse in keeping with advanced osteonecrosis. The left hip demonstrates widespread articular collapse, joint space narrowing and secondary osteoarthritis

seen as hot lasting up to 14 days [28]. The most important differential diagnosis is that of transient osteoporosis which is seen in the third trimester of pregnancy and in men in the fifth and sixth decade of life. MRI in this condition demonstrates edema extending into the femoral neck which is uncommon in osteonecrosis [33–35]. A thorough examination of the contralateral hip is essential, as a 40% to 80% incidence of bilaterality has been reported with transient osteoporosis [2, 36].

There are over 16 major classification systems used in osteonecrosis. The first, and most widely used system, was described by Ficat and Arlet [37] in the 1960s before the advent of MRI. Other systems have since been described taking into account MRI findings as well as the extent of involvement of the femoral head. These include the classification system of the University of Pennsylvania, described by Steinberg et al. [36], and the classification system of the Association Internationale de recherche sur la

circulation Osseuse [38] (Table 2) Combined, these classification systems have been used in 95% of studies published [39].

Early on in osteonecrosis, there are no changes visible on imaging studies, and this is referred to as stage 0 in all classification systems. This progresses to stage I where a decreased signal intensity is observed in T1-weighted MRI images. Increased uptake of tracer on bone scintigraphy may also be visible. Stage II represents the reparative stage, and radiographic changes are visible in subchondral bone. There may be evidence of osteosclerosis, cyst formation, or osteopenia. There is no evidence of subchondral fracture and the articular surface remains intact. Stage III is characterized by subchondral lucency, referred to as the crescent sign, which indicates subchondral collapse. After stage III, the different classifications diverge, but typically distinguish the amount of head depression, joint space narrowing, acetabular involvement, and advanced degener-

Fig. 2. Coronal T1 (a) and T2-weighted (b) magnetic resonance images of the right hip of another patient presenting with a pain on ambulation. A small osteonecrotic lesion is visible with the loss of the normally high signal intensity reflecting adipocyte necrosis

Table 2 Classification of osteonecrosis

Ficat and Arlet [37]		University of Pennsylvania [36]	
Stage	Radiographic findings	Stage	Criteria
1	None (only evident on MRI)	0	Normal radiograph, bone scan and MRI
2	Diffuse sclerosis, cysts	I	Normal radiograph
3	Subchondral fracture (crescent sign)		Abnormal MRI and/or bone scan
4	Femoral head collapse, acetabular involvement and joint destruction (osteoarthritis)	II	A: Mild (<15% femoral head involvement)
			B: Moderate (15–30% femoral head involvement)
			C: Severe (>30% femoral head involvement)
		III	Cystic and sclerotic changes in the femoral head
			A: Mild
			B: Moderate
		IV	C: Severe
			Subchondral collapse without flattening (crescent sign)
			A: Mild (<15% articular surface involvement)
		V	B: Moderate (15–30% articular surface involvement)
			C: Severe (>30% articular surface involvement)
			Flattening of the femoral head
		VI	A: Mild (<15% and <2 mm depression)
			B: Moderate (15–30% and 2–4 mm depression)
			C: Severe (>30% and >4 mm depression)
		VII	Joint space narrowing or acetabular changes
			A: Mild
			B: Moderate
		VIII	C: Severe
			Advanced degenerative change

ative changes including loss of anatomical sphericity of the femoral head which ultimately leads to destruction of the integrity of the joint [1, 16].

Operative and non-operative approaches have been used in the management of osteonecrosis of the femoral head, and these will now be discussed in turn. Conservative treatments include the maintenance of non-weight-bearing status as well as pharmacological therapies. Weight-bearing modification has been proven ineffective except for the treatment of small asymptomatic lesions located outside the weight-bearing area [1]. Pharmacological treatments have been proposed for the early stages of the disease aiming to correct pathophysiologic features allowing revascularization and new bone formation. Non-operative management needs to be deployed early if it is to alter the physiology of the condition, and treating patients conservatively after the appearance of the crescent sign on x-ray, indicating subchondral collapse, is usually not successful.

As previously mentioned, many patients who have osteonecrosis have altered lipid metabolism leading to a hyperlipidemic state. In one study, 284 patients taking high-dose steroids were treated with lipid-lowering drugs and the incidence of osteonecrosis recorded. After a mean of 7.5 years, only 1% had developed osteonecrosis, which is much lower than the usual incidence of 3% to 20% in this patient group [40].

Bisphosphonates have also been used in the treatment of osteonecrosis. A study by Agarwalla et al. [41, 42] demonstrated a significant reduction in pain and disability scores in patients receiving bisphosphonates and a decrease in bone marrow edema on MRI. There was, however, no evidence of radiographical regression. Lai et al. [43] conducted a small controlled trial comparing 29 osteonecrotic hips with large lesions treated with alendronate to 25 hips that had not been treated. When followed up at 24 to 28 months, the treatment group had fewer incidences of collapse, 7% versus 76%, and the number requiring total hip arthroplasty was also reduced, 3% versus 64%.

Other drugs including anti-hypertensives have been used. The vasodilator Naftidrofuryl has been shown to reduce intraosseous pressure in a small case study; however, its clinical efficacy has yet to be demonstrated [44]. While there is a scientific basis for using some of these therapies, future prospective randomized trials are needed to determine their true efficacy. Some new therapies such as hyperbaric oxygen therapy [45, 46], electrical stimulation [47–49], pulsed electromagnetic field therapy [50–53], and extracorporeal shock wave therapy [54, 55] have shown promising results in animal studies, and early work suggests that they may have a role in the early stages of osteonecrosis. Further long-term outcome studies are needed to evaluate these treatments.

The surgical management of osteonecrosis can be divided into head-preserving procedures and arthroplasty. Patients with pre-collapse are generally treated with head-preserving procedures, whereas collapse of the femoral head and arthritis may require arthroplasty.

Core decompression has been used to reduce intraosseous pressure in the femoral head bone compartment (Table 3). The procedure was originally introduced as a diagnostic tool by Ficat and Arlet [37]. Typically, it is used to treat small to medium lesions and involves introducing an 8- to 12-mm cannulae into the lesion from just proximal to the level of the lesser trochanter to avoid the development of a stress fracture [56]. This technique can include augmentation with supplementary bone grafting [57, 58]. Numerous studies have been conducted to evaluate the effectiveness of this procedure. Mont et al. [59] conducted a meta-analysis of 23 studies published prior to 1995. This analysis of 1,026 hips revealed success rates of 84%, 65%, and 47% for stages 1, 2, and 3, respectively, giving an overall success rate of 63% compared to a 35% success rate in hips treated non-operatively, demonstrating that core decompression is more effective than conservative management. When taking into account the 23 studies published since 1995, the overall success rate of core decompression rises to 70%, with mean follow-up ranging from 2 to 10 years [56].

Table 3 Outcome after core decompression [56]

Authors	Year	Hips	Success (%)	Mean follow-up (range) in months
Radke et al. [60]	2004	65	70	–
Mont et al. [61]	2004	45	71	24 (20–39)
Lieberman et al. [62]	2004	17	82	53 (26–94)
Aigner et al. [63]	2002	45	80	69 (31–120)
Simank et al. [64]	2001	94	69	72
Yoon et al. [65]	2001	39	45	–
Steinburg et al. [66]	2001	312	64	(24–168)
Maniwa et al. [67]	2000	26	66	94 (29–164)
Chen et al. [68]	2000	27	60	>24
Lavernia and Sierra [69]	2000	67	63	>24
Bozic et al. [70]	1999	54	48	120 (24–196)
Simank et al. [71]	1999	94	78	72 (18–180)
Van Laere et al. [72]	1998	51	39	24 (6–47)
Scully et al. [73]	1998	98	71	(21–50)
Iorio et al. [74]	1998	33	70	64
Chang et al. [75]	1997	84	30	57 (24–165)
Powell et al. [76]	1997	29	66	48
Mazieres et al. [77]	1997	20	50	24
Mont et al. [78]	1997	79	73	144 (48–216)
Styles and Vichinsky [79]	1996	13	76	44
Markel et al. [80]	1996	54	35	47 (12–95)
Holman et al. [81]	1995	31	50	>12
Smith et al. [82]	1995	114	44	40 (24–78)
Mont et al. [59] ^a	1996	1,206	63	–

^a Literature review of 23 published studies comparing core decompression to conservative management

Core decompression has also been studied in conjunction with grafting of decalcified bone matrix. A study by Aaron et al. [58] analyzed 118 hips with Ficat stages 2 and 3 and compared the results of core decompression to core decompression augmented with decalcified bone matrix. They found no significant difference in outcome in Ficat stage 2 hips between augmented and control groups. However, in stage 3 hips at 24 months follow-up, there was a 47% success rate in the control group compared with an 88% success rate in those hips augmented with decalcified bone matrix, suggesting that augmentation with bone matrix may prove beneficial in more advanced disease.

Variations on this technique have also been described, including a percutaneous approach utilizing a 3.2-mm Steinmann pin that, in a study of 163 hips by Song et al. [83] with a minimum 5-year follow-up, had a 79% success rate in patients with stage I disease and 77% success rate in patients with stage II disease. The authors concluded that the results of multiple drilling with a small bore pin were comparable with other core decompression techniques. This technique needs further evaluation; however, it has the added benefit of being less invasive with low morbidity and fewer surgical complications. Overall core decompression is recommended for the treatment of early stage osteonecrosis of the hip and has the best results in pre-collapse lesions Ficat stages 1 and 2 that involve less than 30% of the femoral head [56].

Osteotomies have been used to realign the collapsing segment from the principal weight bearing area (Table 4). Two types are used: transtrochanteric rotational and intertrochanteric valgus varus which is combined with flexion or extension. Varying success rates between 56% and 79% have been reported as well as a range of technical

difficulties of converting failed cases to THA. Benke and Barker [84], in a study of 105 THA after failed osteotomy, reported an infection rate of 8.6% and recorded technical difficulties including broken screws and femoral shaft fractures that occurred in 17.1% of cases. Ferguson et al. [85] reporting on 305 THA after failed osteotomy noted operating time to be significantly longer for the conversion compared to a standard hip, possibly explaining the increased infection rate as well as an increased blood loss. Infection rates were 9% in this study and the cumulative probability of failure at 10 years was 20.6%. As a result, femoral osteotomy is not widely accepted as a standard method of treatment.

Non-vascularized bone grafting has also been described as a method of providing support to the articular cartilage surface, preventing joint collapse while promoting bone healing (Table 5). Grafting techniques can be via a core tract [98], trapdoor [99], or lightbulb technique [100]. Necrotic bone is removed and allogenic or autologous bone introduced. The overall clinical success is varied, and generally, this technique is reserved for individuals in which core decompression has been unsuccessful or individuals with post-collapse lesions of less than 2 mm, Pennsylvania stage IVA [99, 101–103]. Several studies including one by Mont et al. [102] have demonstrated excellent outcomes in stage 3 disease, 83% at 5 years.

Free vascularized fibular grafts have the added advantage of supporting subchondral bone with a viable strong bone strut as well as providing revascularization to the femoral head aiding osteogenesis. The ipsilateral fibula is harvested with its peroneal artery with its two veins and inserted into the core tract and stabilized with a Kirschner wire. The ascending branches of the lateral circumflex

Table 4 Outcome after rotational osteotomy

Authors	Year	Hips	Success (%)	Mean follow-up (range) in months
Transtrochanteric rotational osteotomy				
Dean and Cabanela [86]	1993	18	17	60 (18–106)
Sugano et al. [87]	1992	41	56	72 (36–132)
Sugioka et al. [88]	1992	295	78	132 (36–192)
Masuda et al. [89]	1988	52	69	60 (12–120)
Eyb and Kotz [90]	1987	39	59	—
Tooke et al. [91]	1987	17	39	51 (18–63)
Valgus varus osteotomy				
Mont et al. [92]	1996	37	76	138 (60–216)
Scher and Jakin [93]	1993	45	87	65 (36–126)
Jacobs et al. [94]	1989	22	72	63
Gottschalk [95]	1989	17	47	36
Maistrelli et al. [96]	1988	106	71 at 2 years 58 at 8 years	96 (48–180)
d'Aubigne et al. [97]	1965	75	79	(12–72)

artery and vein are then anastomosed to the peroneal vessels of the fibula using microvascular surgical techniques. Urbaniak et al. [107] reviewed the results of 1,523 hips treated with this technique between 1979 and 2000. A success rate of 91% was found at 6 months to 22 years follow-up in individuals with no preoperative evidence of collapse. In cases where there was evidence of collapse or joint space narrowing, success rates were found to be 85% and 73%, respectively [6]. In a small prospective study by Kane et al. [108] analyzing 39 hips followed for 2 years, the results of vascularized fibular grafts were better than that of core decompression 80% to 42%; however, as yet, no randomized control trials exist. Unfortunately, Vail and Urbaniak [109] have reported this technique to have a complication rate of 19%, which includes motor weakness, subjective discomfort in the ankle, as well as sensory abnormalities in the lower limb. Aluisio and Urbaniak [110] also noted the rate of fracture of the proximal femur to be 2.5% in one large series.

Once the femoral head has collapsed, alternative surgical options must be strongly considered. Limited femoral resurfacing can be used in late to mid collapse to salvage the femoral head provided there is no acetabular involvement. In younger patients, it has several potential advantages, including preservation of femoral bone stock, low dislocation rates, simplicity of revision, and deferral

of total hip arthroplasty. Overall, the success of limited femoral resurfacing is approximately 84% at short-term follow-up (Table 6). Mont et al. [111] analyzed the functional outcome of limited femoral resurfacing. They found that at 7 years, there was a significant difference in the number of individuals playing sports when compared to age- and sex-matched subjects who underwent total hip arthroplasty. In this series, both groups had similar success rates of around 90%; however, continuing symptoms of groin pain were higher in the resurfacing group, 20% compared to 6%. Limited femoral resurfacing has the advantage of preserving femoral neck bone stock as well as leaving the femoral canal intact, facilitating future conversion to a total hip replacement. The results of these studies suggest that limited femoral resurfacing may be appropriate for younger patients presenting with more advanced osteonecrosis: Ficat stage 3 hips, lesions involving greater than 30% of the femoral head, and lesions with greater than 2 mm. There must be no evidence of acetabular cartilage involvement, and relative contraindications include large cysts in the femoral neck, osteopenia, and a body mass index greater than 35 [112, 113]. Limited femoral resurfacing should be considered, as an interim procedure as most will ultimately fail and require revision to total hip arthroplasty.

Table 5 Outcome after non-vascularized bone graft [56]

Authors	Year	Hips	Success (%)	Mean follow-up (range) in months
Core tract technique				
Lieberman et al. [62]	2004	17	86	53 (26–94)
Rijnen et al. [104]	2003	28	71	42 (24–119)
Plakseychuk et al. [105]	2003	50	36	60 (36–96)
Trapdoor technique				
Mont et al. [102]	1998	30	58	56 (30–60)
Ko et al. [103]	1995	14	80	56
Lightbulb technique				
Mont et al. [106]	2003	21	86	48 (36–55)
Rosenwasser et al. [100]	1994	—	36	144 (120–180)

Table 6 Outcome after limited femoral resurfacing [113]

Authors	Year	Hips	Success (%)	Mean follow-up (range) in months
Cuckler et al. [114]	2004	59	68	54
Beaule et al. [115]	2004	28	86	60 (28–100)
Adili et al. [116]	2003	29	94% at 1 years 76% at 3 years	34 (24–63)
Beaule et al. [117]	2001	37	79% at 5 years 59% at 10 years 45% at 15 years	78 (24–216)
Mont et al. [111]	2001	30	90	84 (48–101)
Sigüier et al. [118]	2001	37	85	49 (24–89)
Nelson et al. [119]	1997	21	82	>60
Grecula et al. [120]	1995	10	70	96
Tooke et al. [121]	1987	12	92	39 (24–62)
Langlais et al. [122]	1979	86	85	78

In more advanced disease where there is acetabular involvement, hip resurfacing can be considered. Like femoral resurfacing, it has the advantage of preserving femoral bone stock and has similar indications as limited femoral resurfacing. Overall, the success of total hip resurfacing is approximately 84% at early follow-up (Table 7). Studies have also demonstrated improved walking speeds and abductor and extensor movements showing superior hip kinematics when compared with standard hip replacements [123, 124]. As with limited femoral resurfacing, emerging clinical experience suggests that if involvement of the femoral head is greater than 30%, then the success of total hip resurfacing will be decreased.

Bipolar hemiarthroplasty has also been used in the treatment of osteonecrosis. Originally, it was designed to decrease acetabular shear forces by the use of an outer free acetabular cup that articulates with the prosthetic femoral head. Historically, it has always produced suboptimal results (Table 8) and does not circumvent the need for resection of the femoral neck and violation of the femoral canal which may complicate future revisions. The use of a thin polyethylene cup can also lead to extensive wear and subsequent osteolysis, particularly in active patients [130–132]. As a result, bipolar hemiarthroplasty should largely be avoided as a treatment for osteonecrosis, particularly in young patients.

Total hip replacement is the single treatment with the highest likelihood of providing excellent early pain relief

and good functional outcome [6]. However, these advantages must be balanced against the fact that it sacrifices more host bone and narrows future operative options. The main indication is advanced osteonecrosis with secondary degenerative arthritis involving the acetabulum [1]. Relative contraindications to this procedure include the younger patient in which femoral-head-preserving options may be more appropriate or individuals who may be at risk of recurrent dislocation, such as alcoholics [142]. From the available data, it appears that cementless total hip arthroplasty has a better success rate than cemented systems, particularly over the last 10 years. Studies suggest that success rates are comparable with total hip arthroplasty performed for osteoarthritis [115, 143–145], with some series reporting a 98% success rate 11 years postoperatively (Table 9) [146]. Seyler et al. [113] note that factors that contribute to high failure rates include young age, long life expectancy, increased body weight, and poor quality femoral bone.

Despite improved outcomes after total hip arthroplasty, there still are some subpopulations of patients including those with SLE, sickle cell disease and those individuals taking high-dose steroids after undergoing renal transplantation that have less than optimal results (Table 10) [142, 166]. This may reflect the ongoing pathology of these conditions with continuing insults to the vasculature of the bone, delaying bone ingrowth following implantation. Other factors that may influence the failure rates include

Table 7 Outcome after total hip resurfacing [113]

Authors	Year	Hips	Success (%)	Mean follow-up (range) in months
Mont et al. [125]	2005	41	93	36 (24–48)
Amstutz et al. [126]	2004	36	94	42 (26–74)
Mohamad et al. [127]	2004	12	84	18 (17–46)
Beaule et al. [115]	2004	56	95	60 (28–100)
Yoo [128]	2004	40	93	36 (24–48)
Grecula et al. [120]	1995	19	53	96
Grecula et al. [120]	1995	35	15	96
Dutton et al. [129]	1982	42	>76	37

Table 8 Outcome after bipolar hemiarthroplasty [113]

Authors	Year	Hips	Success (%)	Mean follow-up (range) in months
Tsumura et al. [133]	2005	32	86	92 (60–180)
Yamano et al. [134]	2004	29	62	144
Lee et al. [135]	2004	40	95	96
Nagai et al. [136]	2002	12	75	144–216
Ito et al. [137]	2000	48	75	137 (84–216)
Chan and Shih [138]	2000	28	89	77 (48–144)
Sanjay and Moreau [139]	1996	26	57	55 (25–84)
Murzik and McCollum [140]	1994	32	88	(24–216)
Learmonth and Opitz [141]	1993	38	87	56 (42–72)
Takaoka et al. [132]	1992	82	86	66
Cabanela [130]	1990	23	59	110
Lachiewicz et al. [131]	1988	31	48	55

decreased immune status leading to higher infection rates and steroid use which increases infection rates and may result in poorer quality bone and healing ability.

There are a few randomized controlled trials in advanced osteonecrosis. One by Grecula et al. [120] in 1995 compared the outcomes of patients aged under 50 treated with either standard cemented arthroplasty, limited femoral resurfacing, or total hip resurfacing. When followed up at 96 months, results were found to be 80%, 70%, and 15%, respectively.

As previously mentioned, it is important to evaluate the contralateral hip, as bilateral involvement has been reported to be between 40% and 80%. A recent study by Nam et al. [176] investigated the progression of asymptomatic osteonecrosis. Three hundred twelve patients presented with bilateral non-traumatic osteonecrosis, of

which 128 underwent joint-preserving procedures and 184 received no treatment for their asymptomatic hip. Sixty-two (59%) hips were classified stage I and 43 (31%) stage II. Fifty-one percent of cases were related to alcohol abuse, 19% corticosteroid-related, and 30% were idiopathic. At 5 years follow-up, 59% of the asymptomatic hips had progressed to collapse, of these 5% involved less than 30% of the femoral head area, 46% involving 30% to 50%, and 83% involving more than 50%, again demonstrating the importance of the size of the lesion in relation to risk of collapse. Forty-one percent of hips remained asymptomatic without signs of collapse, with 30% progressing from stage I to stage II. These results suggest that in early stage asymptomatic osteonecrosis, regular observation in conjunction with pharmacological therapy may be the most appropriate management.

Table 9 Outcome after total hip arthroplasty [113]

Authors	Year	Hips	Success (%)	Mean follow-up (range) in months
Cemented				
Kim et al. [146]	2003	100	98	122
Fyda et al. [147]	2002	53	83	>120
Ortiguera et al. [148]	1999	188	82	214 (120–304)
Garino and Steinberg [149]	1997	123	96	54 (24–120)
Kantor et al. [150]	1996	28	86	92
Katz et al. [151]	1992	34	97	46 (24–84)
Saito et al. [145]	1989	29	52	84
Cementless				
Schneider and Knahr [152]	2004	57	82	(120–168)
Kim et al. [146]	2003	100	98	122
Taylor et al. [153]	2001	70	96	77
Xenakis et al. [154]	1997	29	96	91
Delank et al. [155]	2001	66	93	65 (58–94)
Xenakis et al. [156]	2001	36	93	136 (120–180)
Hartley et al. [157]	2000	55	79	117
Fye et al. [158]	1998	72	97	84 (>48)
D'Antonio et al. [159]	1997	53	85	82 (60–96)
Gonzalez et al. [160]	1997	40	80	58 (24–108)
Stulberg et al. [161]	1997	98	75	87 (31–134)
Kim et al. [162]	1995	78	79	86 (72–108)
Phillips et al. [163]	1994	20	95	64 (>24)
Piston et al. [164]	1994	35	94	90 (60–120)
Lins et al. [165]	1993	37	81	(48–72)
Katz et al. [151]	1992	34	97	46 (24–84)

Table 10 Total hip arthroplasty after specific etiologies [113]

Authors	Year	Hips	Success (%)	Mean follow-up (range) in months
Systemic lupus erythematosus				
Zangger et al. [167]	2000	26	93	55 (21–124)
Chen et al. [168]	1999	18	100	46 (24–85)
Sickle cell disease				
Al-Mousawi et al. [169]	2002	35	80	114 (60–180)
Hickman et al. [170]	1997	15	67	72 (24–144)
Moran et al. [171]	1993	22	57	56
Acurio and Friedman [166]	1992	25	60	103 (24–216)
Clarke et al. [172]	1989	27	41	66
Hanker and Amstutz [173]	1988	8	38	78 (24–208)
Renal disease and transplantation				
Lieberman et al. [174]	1995	46	19	54
Murzik and McCollum [140]	1994	77	54/100 ^a	(24–216)
Failed free vascular fibular graft				
Berend et al. [175]	2003	89	94 at 5 years 85 at 10 years 82 Overall	110 (60–180)

^a Cemented stem/cementless stem

Conclusion

The pathophysiology and management of osteonecrosis is complex and controversial. A better understanding of the biology behind this condition as well as the factors that put some individuals at a higher risk than others will hopefully lead to earlier diagnosis. This in turn may lead to more effective therapies to correct pathophysiological features. The primary goal of treatment should be to relieve pain, maintain a congruent hip joint, and delay the need for total hip arthroplasty for as long as possible. Four main radiographic features are useful in determining the extent of the disease and selecting the appropriate surgical intervention. First is to assess if there has been collapse of the femoral head, demonstrated by the crescent sign on x-ray. If the head has collapsed, then the success rate of head-preserving procedures decreased significantly [6]. Second is the size and location of the lesion. If the involvement is greater than 30% of the femoral head, or the entire weight-

bearing surface is involved, then the success of head-saving procedures will be decreased [102, 177]. The third evaluation is to assess the extent of head depression. Collapse of greater than 2 mm likely requires arthroplasty, although the effect of bone grafting when collapse is present is an area of some debate. If the acetabulum is involved, then total hip arthroplasty is the only surgical option. Ultimately, the final decision is made following intraoperative inspection of the femoral cartilage and acetabulum. A suggested algorithm is presented in Table 11 [6]. Total hip arthroplasty was indicated in our patient due to collapse of the femoral head.

While current conservative treatment options appear hopeful, it has not been demonstrated that any of these therapies halt progression of this condition and larger randomized control trials are needed to fully evaluate effectiveness [5]. In terms of more conservative surgical management strategies, there may be potential for head-preserving surgery such as percutaneous core decompression

Table 11 Treatment algorithm for osteonecrosis of the femoral head [6]

Pennsylvania stage	Presentation	Treatment
I and II	Asymptomatic	Observation Pharmacological ? Core Decompression ± bone grafting
I ABC and II ABC	Symptomatic	Core decompression ± bone grafting Vascularized graft
I C, II C, III ABC and IV A	Symptomatic	Bone Grafting (vascularized or non-vascularized) Osteotomy Femoral head resurfacing Total hip arthroplasty
IV BC	Symptomatic	Femoral head resurfacing Total hip arthroplasty
V and VI	Symptomatic	Total hip arthroplasty

Femoral head involvement A=<15%, B=15–30%, C=>30

combined with the use of novel adjuvants such as growth factors and cytokines which may help augment repair prior to collapse. Further controlled trials are needed to evaluate the role of limited femoral resurfacing and total hip arthroplasty in the younger population as well as in subgroups of patients in whom total hip arthroplasty has proven less than optimal.

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