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REVIEW

Current concepts on osteonecrosis of the femoral head

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Abstract

It is estimated that 20000 to 30000 new patients are diagnosed with osteonecrosis annually accounting for approximately 10% of the 250000 total hip arthroplasties done annually in the United States. The lack of level 1 evidence in the literature makes it difficult to identify optimal treatment protocols to manage patients with pre-collapse avascular necrosis of the femoral head, and early intervention prior to collapse is critical to successful outcomes in joint preserving procedures. There have been a variety of traumatic and atraumatic factors that have been identified as risk factors for osteonecrosis, but the etiology and pathogenesis still remains unclear. Current osteonecrosis diagnosis is dependent upon plain anteroposterior and frog-leg lateral radiographs of the hip, followed by magnetic resonance imaging (MRI). Generally, the first radiographic changes seen by radiograph will be cystic and sclerotic changes in the femoral head. Although the diagnosis may be made by radiograph, plain radiographs are generally insufficient for early diagnosis, therefore MRI is considered the most accurate benchmark. Treatment options include pharmacologic agents such as bisphosphonates and statins, biophysical treatments, as well as joint-preserving and joint-replacing surgeries. the surgical treatment of osteonecrosis of the femoral head can be divided into two major branches: femoral head sparing procedures (FHSP) and femoral head replacement procedures (FHRP). In general, FHSP are indicated at pre-collapse stages with minimal symptoms whereas FHRP are preferred at post-collapse symptomatic stages. It is difficult to know whether any treatment modality changes the natural history of core decompression since the true natural history of core decompression has not been delineated.

Key words: Osteonecrosis; Femoral head; Conservative treatment; Core decompression; Stem cells; Total hip arthroplasty

Core tip: This paper walks the reader through the most current evidence regarding the etiology, pathogenesis, treatment options and prognosis of patients presenting with osteonecrosis of the femoral head. We emphasize early diagnosis with magnetic resonance imaging,

review surgical and non surgical treatment modalities and provide a personalized management algorithm according to the different stages of the disease.

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INTRODUCTION

Osteonecrosis (ON) of the femoral head (ONFH) is the final common pathway of a series of derangements that result in a decrease in blood flow to the femoral head (FH) leading to cellular death, fracture, and collapse of the articular surface[1,2]. It typically affects relatively young, active people between 20 and 40 years and regularly follows an unrelenting course resulting in substantial loss of function. It is estimated that 20000 to 30000 new patients are diagnosed with ON annually accounting for approximately 10% of the 250000 total hip arthroplasties (THA) done annually in the United States[3]. Spontaneous regression of avascular necrosis is rare, with the vast majority of untreated patients progressing to THA and a collapse rate of 67% in asymptomatic patients and 85% of symptomatic hips[4]. Although many authors have suggested treatment based on patient age, symptoms, stage, and/or medical status, the orthopedic community has not yet adopted a uniform treatment algorithm[5-11]. The lack of level 1 evidence in the literature makes it difficult to identify optimal treatment protocols to manage patients with pre-collapse AVN of the FH, and early intervention prior to collapse is critical to successful outcomes in joint preserving procedures.

ETIOLOGY AND PATHOGENESIS

There have been a variety of traumatic and atraumatic factors that have been identified as risk factors for ON, but the etiology and pathogenesis still remains unclear. The estimated frequency of the most frequent risk factors for ONFH in the United States is: alcohol (20%-40%), corticosteroid therapy (35%-40%), and idiopathic (20%-40%)[12].

Most studies have attributed the disease process to the combined effects of genetic predisposition, metabolic factors, and local factors affecting blood supply such as vascular damage, increased intraosseous pressure, and mechanical stress[3,13,14]. This results in bone ischemia and infarction leading to bone death. The precipitating mechanism which leads to this pathway is variable though (Figure 1). Ischemia can result from external or internal vascular insult typically caused by direct trauma, vascular occlusion, direct cellular toxicity, or altered mesenchymal stem cell differentiation[15].

Several mechanisms leading to vascular occlusion have been proposed as possible underlying causes of necrosis. High doses of glucocorticoids prevalent in systemic diseases such as systemic lupus erythematosus as well as excessive alcohol intake have been associated with alterations in circulating lipids with resultant microemboli in the arteries supplying the bone[16]. In addition increased risk of fat emboli has also been attributed to the increase in bone marrow fat cell size which blocks venous flow. Therefore, fat emboli, adipocyte hypertrophy, and venous stasis have all been implicated as etiologic factors in this disease process. Vascular occlusion can also result from disease processes that increase intravascular coagulation and thrombus formation. Antiphospholipid antibodies, inherited thrombophilia, and hypofibrinolysis have all been associated with altered mechanisms in both the coagulation and fibrinolytic pathways. Occlusion can also occur as a result of red blood cell sickling and bone marrow hyperplasia as seen in sickle cell hemoglobinopathies or may be due to an accumulation of cerebroside-filled cells within the bone marrow as seen in Gaucher's disease[17]. Decompression sickness associated with increased pressure can lead to nitrogen bubble formation that can also cause arteriolar occlusion and necrosis. This has also been shown to result in elevated plasma levels of plasminogen activator inhibitors leading to increased coagulation[18]. Trauma due to fracture or dislocation can lead to damage to the extraosseous blood supply. This is especially specific to fractures in the subcapital region of the femoral neck. Trauma at this location interrupts the anastomosis between the lateral epiphyseal vessels, which are branches from the medial femoral circumflex artery supplying, and the artery of the ligamentum teres leading to compromised blood flow to the FH. Lastly, direct cellular insult may result from irradiation, chemotherapy, or oxidative stress and may lead to a reduction in osteogenic differentiation and physiologic diversion of mesenchymal stem cells toward the adipocytic lineage[15].

DIAGNOSIS AND ASSESSMENT

Early diagnosis is crucial for optimal treatment of ON, as treatment success is related to the stage at which the care is initiated[13]. Current diagnostic modalities available include radiography, scintigraphy, functional evaluation of bone, magnetic resonance imaging (MRI), computer-assisted tomography, and histological studies.

Clinical presentation of ON typically is asymptomatic in early stages, although patients may develop groin pain that can radiate to the knee or ipsilateral buttock. On physical examination, patients usually present with a limited range of motion at the hip and complain of pain particularly with forced internal rotation. A detailed history can identify any associated risk factors (Table 1)[13]. ON must be suspected with presentation of pain

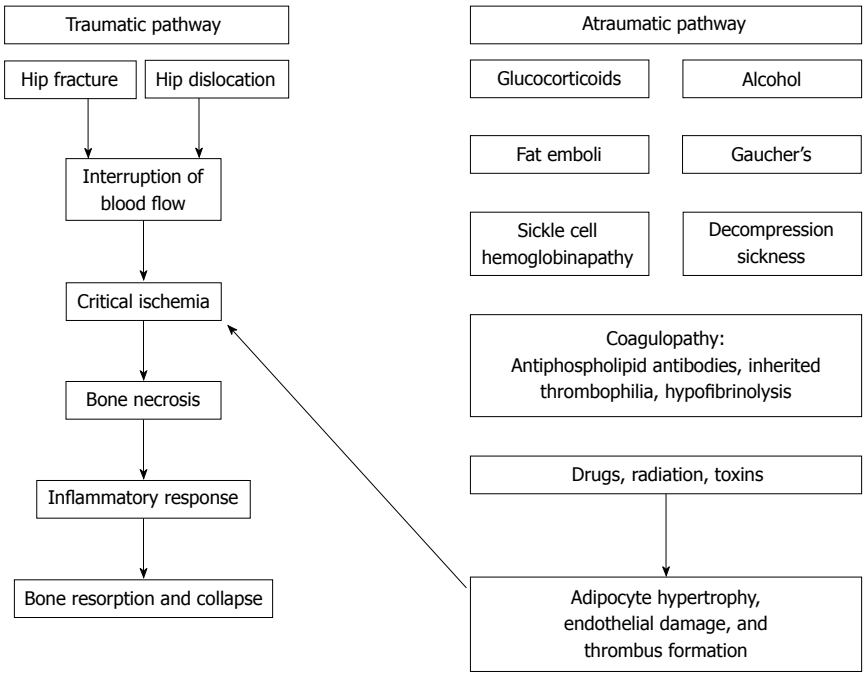

Figure 1 Mechanisms of osteonecrosis.

Table 1 Risk factors for osteonecrosis of the femoral head

Direct	Indirect
Femoral head/neck fracture	Chronic corticosteroid use
Hip dislocation	Excessive alcohol consumption
Slipped capital femora epiphysis	Coagulation disorders
Radiation	Hemoglobinopathies
Sickle cell disease	Dysbaric phenomena
Caisson disease	Autoinmune diseases
Myeloproliferative disorders	Smoking
	Hyperlipidemia

Table 2 Ficat and arlet classification system

Stage	Features
0	Normal radiographs (silent hip)
Ⅰ	Slight abnormality as patchy/opaque areas, minor osteopenia
Ⅱ	Sclerotic or cystic lesions
	Ⅱa: No crescent sign
	Ⅱb: Crescent sign without flattening of the femoral head
Ⅲ	Flattening of the femoral head or femoral head collapse
Ⅳ	Femoral head collapse and osteoarthritis of the hip (joint space narrowing, osteophytes and acetabular changes)

in the hips, negative plain radiographs, and any of these risk factors, since plain radiographs may present as normal in the early stages of necrosis. Patients who have had a history of necrosis must be watched for bilateral ON, as bilaterality has been reported in up to 70%[19].

The two most common classifications used in the diagnosis of ON include the Ficat and Arlet and the Steinberg University of Pennsylvania systems (Tables 2 and 3)[20]. Ficat classification consists of four stages, based on standard radiographs. Stage Ⅰ indicates normal imaging. Stage Ⅱ indicates normal FH contour, but with evidence of bone-remodeling, such as cystic or osteosclerotic regions. Stage Ⅲ indicates evidence of subchondral collapse, or flattening of the FH. Stage Ⅳ indicates a narrowing of the joint space with secondary degenerative changes in the acetabulum, such as cysts, osteophytes, and cartilage destruction. Hungerford[13] described the stage 0, silent hip (preclinical and preradiologic), in which AVN can be suggested if it has been already diagnosed in the contralateral femoral head. In this case bone marrow pressure and histology studies would be abnormal. Although the Ficat classification system has been well established, it is dependent on radiographic imaging and does not allow for quantitation of lesion size, making it impossible to measure disease progression[21]. Steinberg expands the Ficat system into six stages and includes quantification of involvement of the FH within each stage. They defined mild (less than 15% radiographic involvement of the head's articular surface), moderate (15%-30% involvement of the head's articular surface), and severe (greater than 30% involvement of the head's articular surface) stages. In addition, the Association Research Circulation Osseous (ARCO) suggested a new classification system based on the combination of radiographic, MRI, bone scan and histologic findings. However, apparently these two classifications systems, Ficat and ARCO are still not enough reliable to assess the status of ONFH alone[22].

Several studies have shown that the size of the necrotic segment in the FH is a fundamental parameter to determine the prognosis and treatment of this condition. Different methods are currently used to measure the size of the lesion. These include, the tradicional

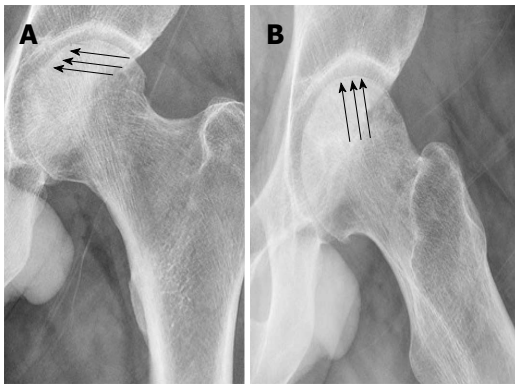

Figure 2 Left hip anteriorposterior and cross leg lateral X-rays showing (arrows) the crescent sing.

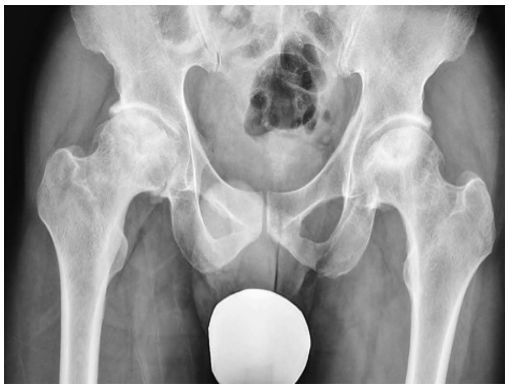

Figure 3 Bilateral osteonecrosis of the femoral head with flattening of the surface and early sings of osteoarthritis.

Table 3 Steinberg staging system

Stage	Features
0	Normal radiograph, bone scan and magnetic resonance imaging
I	Normal radiograph, abnormal bone scan and or magnetic resonance imaging
	I A Mild (involves < 15% of femoral head)
	I B Moderate (involves 15% to 30% of femoral head)
	I C Severe (involves > 30% of femoral head)
II	Cystic and sclerotic changes in the femoral head
	II A Mild (involves < 15% of femoral head)
	II B Moderate (involves 15% to 30% of femoral head)
	II C Severe (involves > 30% of femoral head)
III	Subchondral collapse (crescent sign) without flattening of the femoral head
	III A Mild (involves < 15% of femoral head)
	III B Moderate (involves 15% to 30% of femoral head)
	III C Severe (involves > 30% of femoral head)
IV	Flattening of the femoral head/femoral head collapse
	IV A Mild (involves < 15% of femoral head)
	IV B Moderate (involves 15% to 30% of femoral head)
	IV C Severe (involves > 30% of femoral head)
V	Joint space narrowing and/or acetabular changes
	V A Mild
	V B Moderate
	V C Severe
VI	Advance degenerative joint disease

angular measurements methods described by Kerboul and Koo and Kim and the quantitative volumetric measurement performed by quantitative digital analysis[23,24]. A recent study[25] comparing the efficacy of these systems showed more accurate and reliable measurements using the volumetric measurement method[25]. However, simpler measurement systems, though less accurate, are more commonly utilized since volumetric measurements are technically too demanding for general use. In spite of that, the size of the necrotic region must be determined as part of a comprehensive evaluation of this condition.

Current ON diagnosis is dependent upon plain AP and frog-leg lateral radiographs of the hip, followed by MRI. The AP radiographs will usually demonstrate the primary area of involvement once changes can be viewed. Generally, the first radiographic changes seen by radiograph will be cystic and sclerotic changes in the FH. Subtle osteosclerotic or cystic changes in the subchondral regions may be missed because the anterior and posterior acetabular margins overlap the superior FH, therefore lateral frog-leg radiographs of the FH are necessary. Early delamination of the cartilage from the underlying bone will most likely be demonstrated by the crescent sign (Figures 2 and 3)[15]. Flattening of the FH can also be viewed by radiograph, but may only be visible in one view[15].

Although the diagnosis may be made by radiograph, plain radiographs are generally insufficient for early diagnosis; therefore MRI is considered the most accurate benchmark[5]. A single-density line on T1-weighted images and a high signal intensity line on T2-weighted images represent the early necrotic-viable bone interface and the hypervascular granulation tissue characterizing ON (Figure 4)[13]. However, recently subchondral insufficiency fractures of the FH have been proposed as a new concept regarding FH collapse with a reported incidence of 5%-10% of patients who underwent a hip replacement with a diagnosis of ONFH[26,27]. These entities must be differentiated since AVN represents an irreversible condition, which might lead to permanent joint failure and SIF may either completely resolve or progress toward epiphyseal collapse[28-30]. The characteristic finding of SIF on MRI is a low intensity band on T1 in association with bone marrow edema, however, this finding has also been described in ONFH. A recent study[30] demonstrated that the shape of the low intensity band (on T1-weighted MRI) is helpful for differentiation between the two diagnoses. The low intensity band seen in SIF is generally irregular, serpiginous, discontinuous, and convex to the articular surface, while the band in ONFH tends to be smooth, concave and well circumscribed. However, the shape of the low intensity band is not always diagnostic and further imaging may be required (MRI with gadolinium). Ultimately, both clinical and MRI characteristics need to be evaluated for the critical differentiation of both conditions (Table 4).

Other functional tools for evaluating ON include measuring bone-marrow pressure, venography, and core

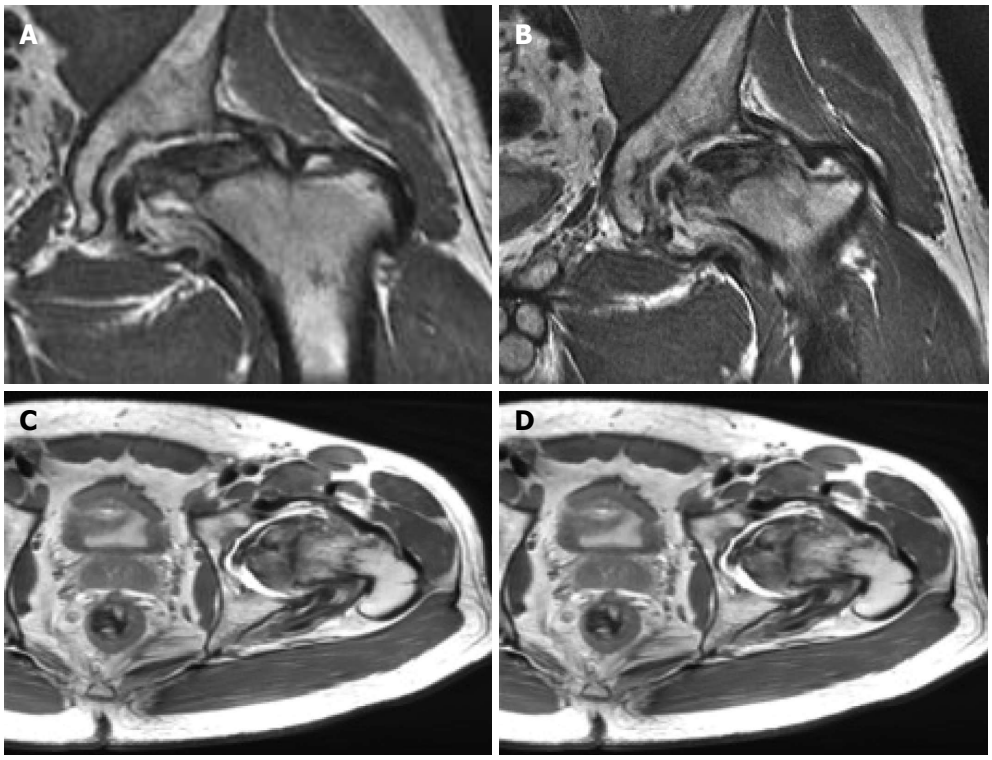

Figure 4 Magnetic resonance imaging of the left hip showing extensive avascular necrosis of the femoral head with collapse and a large area of devitalized bone demonstrating fibrocystic change. There is associated severe arthrosis of the left hip joint with a moderate effusion, synovitis and debris and a marked bone marrow edema pattern on both sides of the joint.

Table 4 Clinical and imaging differences between osteonecrosis femoral head and subchondral insufficiency fracture

	SIF	ONFH
Age/sex	Elderly/female	30 s to 40 s
Etiology	Osteoporosis/obese	Steroid/alcohol
Bilateral	Rare	50%-70%
Shape of the band	Iregular, disconnected	Smooth
High signal of the proximal Segment on gadolimium MRI	Yes	No

From Yamamoto T. In: Yamamoto T. Subchondral Insufficiency Fractures of the Femoral Head. *Clinics in Orthropedic Surgery* 2012: **4**: 3. SIF: Subchondral insufficiency fracture; ONFH: Osteonecrosis femoral head; MRI: Magnetic resonance imaging.

biopsy. While these tests are specific and sensitive, they are invasive and only used when MRI, and radiograph reveal negative findings in a patient where ON is highly likely. Although CT scans can aid in distinguishing between late stages of ON before collapse of the FH, this modality is rarely used due to its high doses of radiation. Characteristic features that define the diagnosis of ON include: collapse of the FH, anterolateral sequestrum, or the crescent sign, or when a double-line sign is demonstrated through MRI on T2-weighted images, or there is a positive histologic finding upon bone biopsy.

Non-surgical management

The aim of treatment of AVN of the hip is to prevent collapse of the FH and may vary depending on the underlying etiology and stage of progression. Treatment options include pharmacologic agents, biophysical treatments, as well as joint-preserving and joint-replacing surgeries. Medical management of AVN has been increasingly used in early stages in attempt to delay the progression of the disease.

Pharmacological management of AVN includes lipid lowering agents, anticoagulants, vasoactive substances, and bisphosphonates. Increases in both the number and size of circulating fat cells have been associated with the development of ON of the hip, therefore lipid lowering agents, such as statins, which reduce the rate of adipogenesis, are beneficial. Statins have been shown to provide protective effects for patients receiving steroids. It is still unclear whether statins have the ability to reverse steroid-induced ON once it has already occurred[31,32]. Anticoagulants such as enoxaparin act through the inhibition of platelets aggregation thereby increasing blood flow to ischemic areas of the bone. These agents are primarily beneficial in patients with underlying coagulopathy disorders, such as thrombophilia or hypofrinolysis[9,33]. Prostacyclin is a vasoactive agent that improves blood flow through its vasodilator effects in the terminal vessels. Although prostacyclin has shown significant improvement in both clinical and radiologic outcomes in early stages of AVN, long term benefits have yet to be established[34].

Bisphosphonates significantly reduce the incidence of collapse of the FH in osteonecrotic hips by reducing

osteoclast activity. Alendronate has been shown to prevent early collapse of the FH in Steinberg stages Ⅱ and Ⅲ non-traumatic ON at 24-28 mo follow up and has been reported to diminish the amount of pain at one year follow up when it is compared with placebo treatment[35,36]. Alendronate has been used as an adjunctive therapy with surgical procedures and has been found to reduce pain and the risk of collapse in early stages of ONFH[37]. Evidence for prevention of THR and reduction of AVN progression still remains controversial[38].

Biophysical treatments include extracorporeal shockwave therapy (ESWT), pulse electromagnetic therapy, and hyperbaric oxygen (HBO) therapy. ESWT has been shown to restore tissue oxygenation, reduce edema, and induce angiogenesis and may offer an alternative to the invasive modalities for FH necrosis in the earlier stages[39,40]. ESWT has also been associated with improvement in both pain and function, and has been found to result in a reduction of lesion size and bone marrow edema at 1-year follow up. Long term (8-9 years) improvement in pain and Harris Hip scores has also been demonstrated in the ESWT group treatment when compared with the core decompression group treatment[41]. Although not as commonly used, pulse electromagnetic therapy is believed to function by stimulating osteogenesis and angiogenesis however its role as early stage ON treatment has not yet been established[42]. HBO increases extracellular oxygen concentration and reduces cellular ischemia and edema by inducing vasoconstriction[43]. Studies have reported radiographic improvement in Steinberg stage I-AVN, as well as pain and ROM improvement in Ficat stage-Ⅱ ON[39,44].

Conservative treatment of AVN may be effective in the earlier stages of the disease. Although medical management may improve pain and functional outcomes, randomized clinical trials are necessary with long term follow up to determine effectiveness of therapy.

Surgical treatment

Currently there is no consensus regarding the treatment of the different stages of ONFH in the adult population[7,10,45,46]. A recent survey of 753 members of the American Association of Hip and Knee Surgeons reported that total hip replacement was the most common intervention for treatment of post-collapse stages of ONFH, whereas core decompression was the most common procedure for symptomatic pre-collapse stages of ONFH. Other less frequently performed treatments include conservative management, vascularized and non-vascularized bone grafts, hemi-arthroplasty, osteotomy, and arthrodesis[47]. ONFH tends to affect younger patients, therefore a variety of joint preserving surgical procedures have been developed to delay the progression of the disease and afford pain relief[5,21,48,49].

The surgical treatment of ONFH can be divided into two major branches: FH sparing procedures (FHSP) and FH replacement procedures (FHRP). In general, FHSP are indicated at pre-collapse stages with minimal symptoms whereas FHRP are preferred at post-collapse symptomatic stages.

FHSP: FHSP aim to preserve the FH and include core decompression (CD), CD combined with different grafting procedures and/or biologic agents and rotational osteotomies. Since all these procedures cannot restore the sphericity of the FH their role in the management of post-collapse stages is very limited[50,51].

CD: CD of the FH is the most common procedure currently performed to treat early stages of ONFH with the goal of decompressing the FH pressure in order to restore normal vascular flow and ultimately relieve pain[5,52,53]. The technique of CD has varied in terms of surgical approaches, number of drillings, and trephine diameter. Small diameter drilling has been proposed as an alternative because it has the advantage of reaching the anterior portion of the FH (most frequently involved region in ONFH) (Figure 5). In addition, small diameter drilling has been associated with minimal morbidity, less risk of weakening the FH and the articular cartilage, and less risk of stress risers that ultimately can lead to a subtrochanteric fracture[54]. Although CD has been shown to delay the progression of ON, its role in complete reconstruction of the necrotic area has not yet been established[55].

Bone grafting procedures: Non-vascularized bone grafts from different sources (allograft, autograft or artificial) have been used to fill the necrotic area in the FH. The grafting can be performed through the core decompression tract, which is the most common technique, but also through a window in the FH or in the femoral neck[56]. This latter technique, also referred to as the trapdoor procedure, requires a surgical dislocation of the hip in order to graft the defect through a cartilage window in the FH.

Vascularized bone grafting combines the benefit of core decompression along with an osteoinductive and osteoconductive graft in the devitalized FH. This procedure was popularized in the 1970's coincidently with the emergence of microsurgical techniques[51]. The variability among the surgical techniques to perform this procedure has however confounded the uniformity of the published data.

The free vascularized fibular grafting (FVFG) has been shown to support the subchondral architecture as well as restore local circulation to the necrotic FH in treatment of ONFH. A study on 470 patients with a mean follow-up of 5.0 years showed an average Harris hip score improvement from 65.0 to 86.9, no radiographic changes in 57.3% of patients, improvement in 33.7% of patients, and necrosis progression in 9.0% of patients respectively. These results show that the modified technique of the use of FVFG for treatment of ONFH yields similar postoperative results in comparison

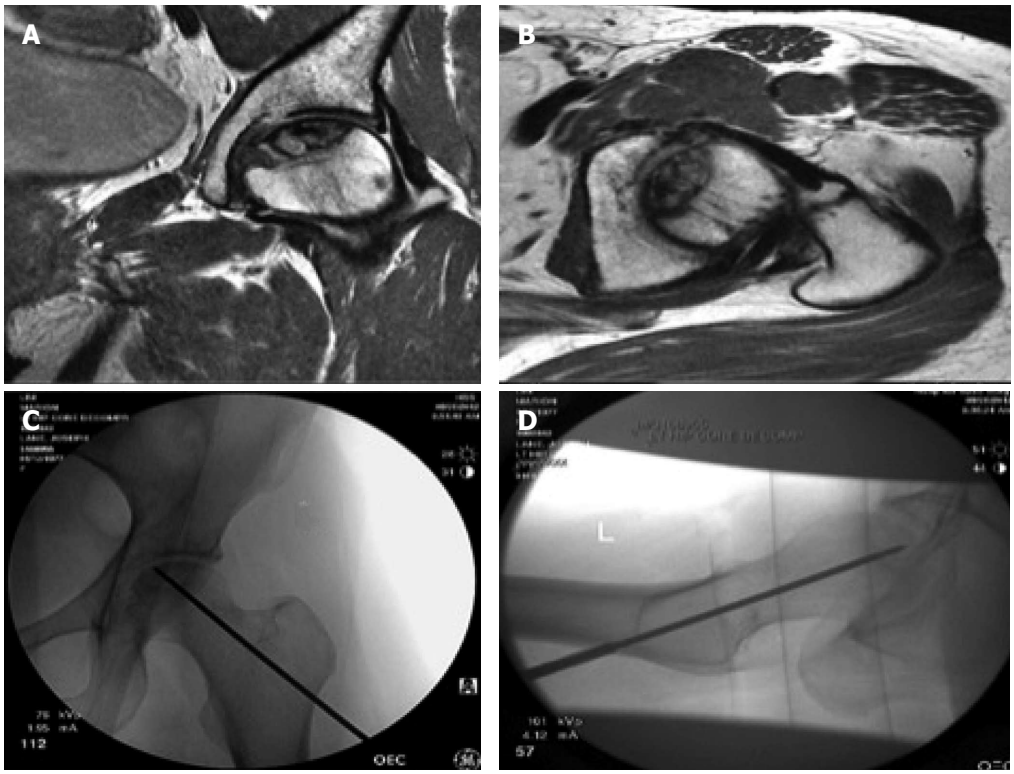

Figure 5 Core decompression of the left femoral head. Preoperative magnetic resonance imaging, above (coronal and axial views) and fluoroscopic imaging during the procedure below.

to the traditional method. Although vascularized fibular grafting has shown promising results, especially in young patients with ONFH, the extensive surgical time, donor-site morbidity, prolonged rehabilitation, and an increased risk of a proximal femoral fracture has limited its use in practice[57-59].

Tantalum implants: Porous tantalum implants in combination with core decompression offers the advantage of providing structural support without the risk of autograft harvest or the infectious complications of bone allograft[60-62]. Veillete *et al*[62] reported an overall survival rate of 91.8% at twenty-four months, and 68.1% at forty-eight months after evaluating fifty-four patients with ONFH treated with core decompression and the insertion of a porous tantalum rod. Although these results appear promising, there are concerns about the origination of metal debris in the joint if a THR becomes necessary as well as a more complicated surgical technique. In addition, previous histologic studies demonstrated little bone ingrowth and insufficient mechanical support of the subchondral bone at the time of conversion from a tantalum rod to THR[63]. Long-term follow up is necessary in order to assess the functional and clinical outcomes of this technique.

Biological agents: There is considerable enthusiasm in the development of biological therapies that can enhance core decompression with osteogenic (mesenchymal stem cells) and/or osteoinductive agents (bone morphogenic protein) that have the potential to produce better results for larger lesions.

It has been hypothesized that there is an insufficient supply of progenitor cells in patients with AVN, which are required to enhance remodeling in areas of ON[64]. For this reason, newer treatment modalities have been developed to introduce stem cells to the areas of necrosis in order to prevent fracture and collapse of the FH. Since 2002, when Hernigou *et al*[65] first described a technique for injecting mesenchymal stem cells into an area of necrosis, four studies have prospectively evaluated the use of stem cells and core decompression. These studies presented consistent findings showing that patients treated with core decompression and stem cells achieved a significantly higher Harris Hip Score at final follow up. Gangji *et al*[66] reported in 2004 the results of a controlled, double blind study comparing core decompression with and without bone marrow aspirate. After 24 mo follow up the survival analysis revealed a significant difference in the time to collapse between both groups and a decreased of 35% of the necrotic lesion in the bone marrow graft group.

The instillation of stem cells into the osteonecrotic region of the FH can be performed through various methods. These include the direct instillation through the core tract, a selective femoral arterial perfusion, or the catheterization of the medial, lateral, or obturator artery. The direct instillation through the core tract is the most commonly performed procedure, however the catheterization of these vessels makes it difficult thereby requiring higher technical skills. However, it is important to maintain the final concentrate of cells

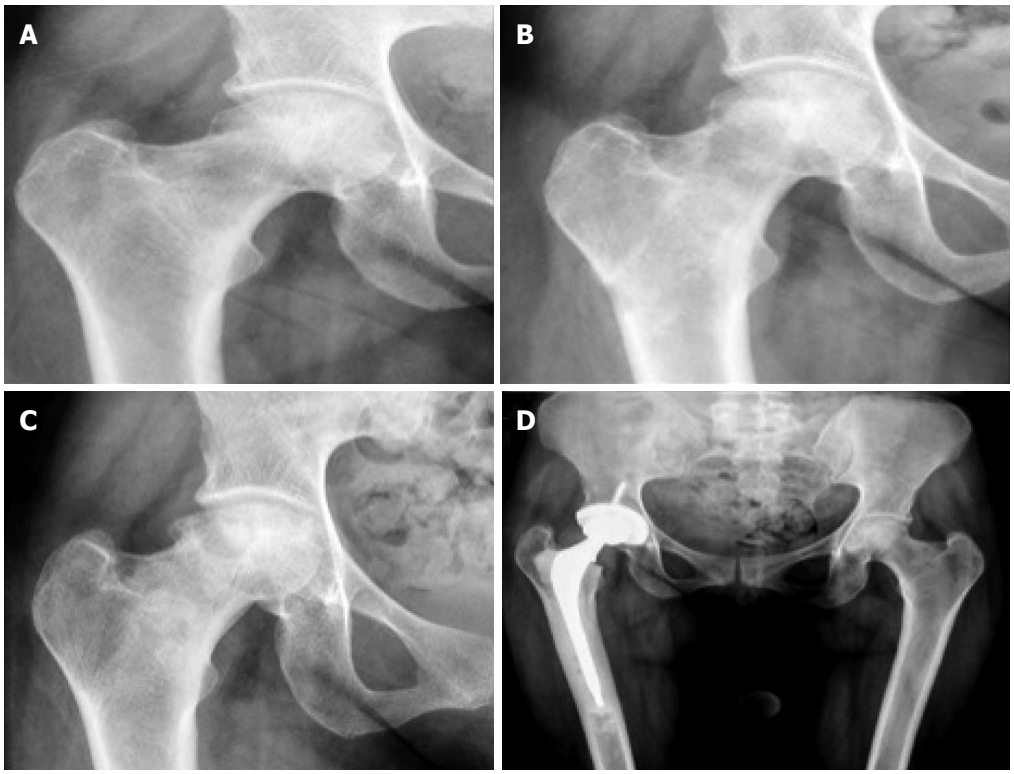

Figure 6 Right femoral head osteonecrosis. Flattening of femoral head progression in 24 mo ending up in a right total hip replacement.

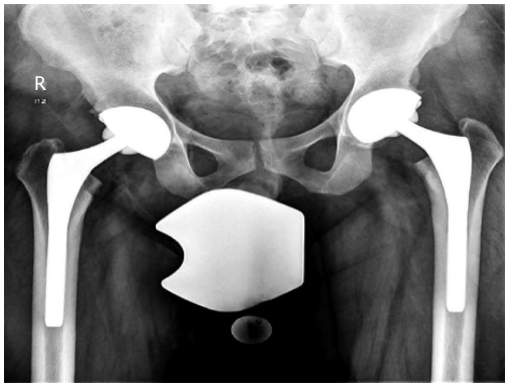

Figure 7 Bilateral total hip replacement in a patient with bilateral hip osteonecrosis of the femoral head.

when doing a direct instillation in order to effectively regenerate the osteonecrotic region (optimum effective dosage minimum necessary concentration 5 × 107 and CD 34 + 5 × 107 cells)[45,67]. This is another factor that has been shown to influence healing of necrotic areas in the FH[64,65]. Additionally, the relationship between the injected volume and the lesion volume needs to be studied. Although these previous studies confirm that bone marrow aspirate concentrate has the potential to induce bone repair in ONFH, the data is preliminary and many questions still need to be addressed[45,64-67].

Osteotomies: Two general types of osteotomies, angular intertrochanteric and rotational transtrochanteric, can be performed to remove the segment of necrotic bone away from the weight-bearing region in the hip[68-72].

The transtrochanteric rotational osteotomy (TRO) for treatment of ONFH was introduced by Sugioka[71] in 1972. The aim of this procedure is to rotate the necrotic region of the FH out of the weight bearing area of the acetabulum. Sugiota[71] reported promising clinical results with a success rate of 78% after 3-16 years. However, their results with this technically demanding procedure have not been reproduced[68-70]. Rotational osteotomies can provide a painless, mobile, and stable hip if there is an unloading of the necrotic area of the FH when it is rotated from the acetabular major bearing surface and if the depth of the necrosis is not bigger than one third of the head diameter[71]. Hisatome *et al*[72] reviewed 25 hips in 21 patients six years after Sugioka's transtrochanteric anterior rotational osteotomy for ONFH. They concluded that although the collapse of a new weight-bearing region can be prevented, the progressive collapse of the transposed necrotic area induces anterior joint instability and subsequent arthritic changes.

Despite the promising results, patients who ultimately require conversion to THA after a proximal femoral osteotomy have a 17% intraoperative complication rate and an 82% survival rate of the implant after 10 years. Osteotomies are a reasonable option when they are performed by experienced surgeons in patients younger than 45 years with a Kerboull angle below 200° and no longer taking steroids.

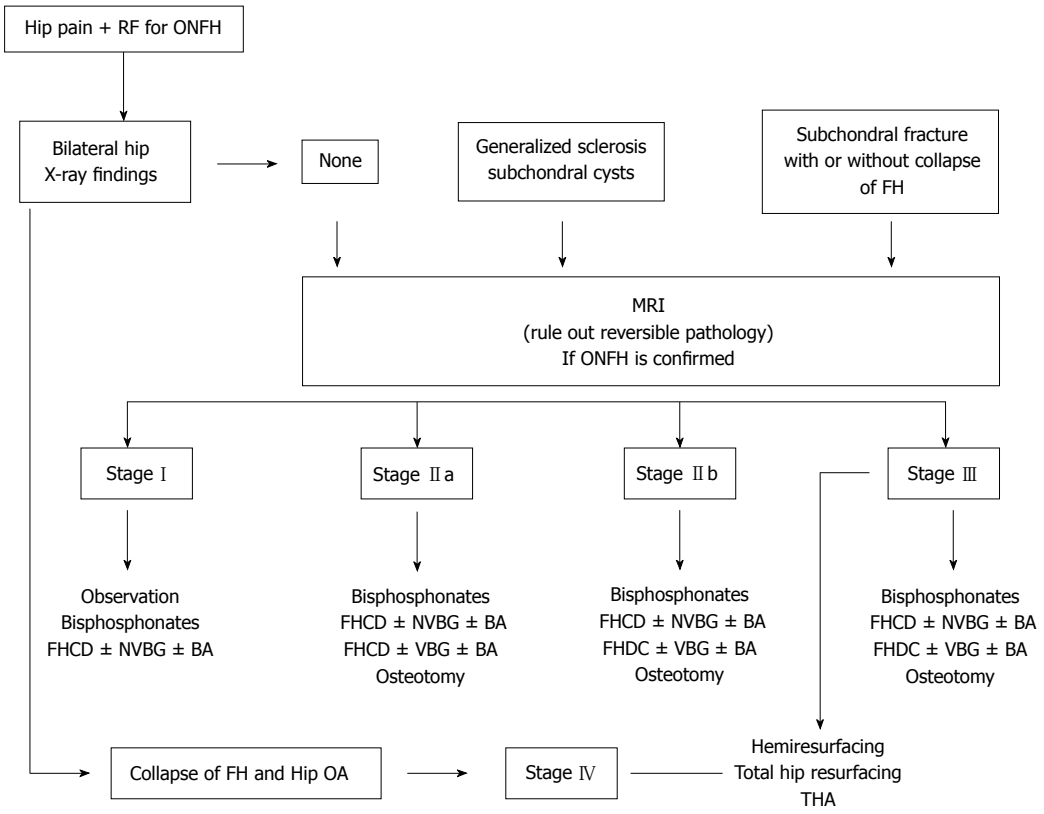

Figure 8 Algorithm for the management and treatment of patients with osteonecrosis of the femoral head. RF: Risk factors; ONFH: Osteonecrosis of the femoral head; FH: Femoral head; MRI: Magnetic resonance imaging; FHCD: Femoral head core decompression; NVBG: Non vascularized bone graft; BA: Biologic agents; VBG: Vascularized bone graft; OA: Osteoarthritis; THA: Total hip arthroplasty.

FHRP

Although FHSP may provide good clinical results in patients with small pre-collapse lesions, these interventions are less predictable in patients with larger lesions or in FH collapse. These patients are therefore better candidates for FHRP.

Hemi-resurfacing arthroplasty and hemipolar/bipolar hip replacement: Hemi-resurfacing arthroplasty is a significant treatment option when the joint surface is still preserved and the articular cartilage is minimally damaged. Possible indications include a Ficat Ⅲ, early stage Ficat Ⅳ, or early failure of a free vascularized fibula graft. With good patient selection and surgical technique this procedure can restore patient function although pain relief may not be as predictable as after THR[73]. Hemi-resurfacing arthroplasty causes little distortion of the anatomy, preserves bone, and produces minimal particle debris. Accurate evaluation of the acetabular articular cartilage and its longevity with this component poses a difficult challenge.

Hemi-arthroplasty replacements are an alternative treatment strategy as they preserve the acetabular bone stock. The major concerns with this procedure are the incidence of protrusion and polyethylene wear that can lead to particle-induced osteolysis and femoral stem loosening[74,75]. Nevertheless, either hemi-resurfacing arthroplasty or proximal femoral osteotomies are preferred to hemi-arthroplasty.

THA: Arthroplasty is typically reserved for patients with late-stage ONFH, as well as older patients and those with more advanced arthritis (Figures 6 and 7)[47]. Arthroplasty is the only treatment that has been proven to reduce pain and restore mobility. In the United States, it is estimated that approximately 10% of all THRs are done in symptomatic hip ON[6,49].

There have been several studies which have shown poor results of THR for ONFH with failure rates between 37% and 53%, but more recent long term follow up studies have reported improved results compared with earlier reports[76-78]. The advances in the past two decades with the advent of surface bearings with low wear rates present promising results when used in patients with an advance stage of necrosis at mid-term follow up[79-81].

Kim *et al*[82] recently reported a 98% stem survivorship and an 85% cementless cup survivorship at 17.3 years of mean follow up. The most common reason for revision was due to cup wear or loosening. Although longer-term follow up studies are needed, promising stem and cup survivorship seems to be feasible.

Overall patients with ONFH present similar failure rates after THA than the general population. However a few ON risk factors, as renal failure and/or transplant and sickle cell disease, have been associated with

worse outcomes[81]. Fortunately these risk factors are present in a small population of patients with ONFH and even in this high-risk group population the outcomes of THR have improved over time[83-85]. Many studies have also shown that the outcomes of primary THR are not affected by previous hip joint preserving procedures[86-91]. However, THAs performed after rotational or angular osteotomies have shown higher complication rates when compared to those who did not have a previous osteotomy because of the disturbed anatomy of the proximal femur after the TRO[86,92-94].

CONCLUSION

Clinical and MRI characteristics need to be evaluated for the critical diagnosis of ONFH (Table 4). The progression of ONFH has not been well established, therefore it is difficult to evaluate whether a specific treatment modality changes the natural course of the disease. Medical management and surgical intervention has demonstrated to provide symptomatic relief, and early intervention prior to collapse has been shown to be critical to successful outcomes in joint preserving procedures. Future research should be directed at delineating whether one treatment strategy can delay the progression of ONFH of the hip thereby preventing collapse and the need for THA. A proposed algorithm for the diagnosis and management of ONFH is given in Figure 8.

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