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Review

Osteonecrosis of the Femoral Head: Current Concepts in Diagnosis, Staging and Treatment

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ABSTRACT

Osteonecrosis of the femoral head (ONFH) is a progressive disorder characterized by ischemia-induced death of the subchondral bone, most commonly affecting young and active individuals. More than one-third of patients have a history of corticosteroid use, while approximately 20% of cases are considered idiopathic. In the early stages, the disease may remain clinically asymptomatic; however, with progression, patients typically develop hip or groin pain accompanied by limitation of hip abduction and internal rotation. Treatment strategies are determined according to the stage of the disease and include conservative management, joint-preserving procedures, and salvage procedures. The aim of this review is to comprehensively summarize the etiology, pathophysiology, diagnostic methods, staging systems, and current treatment approaches for femoral head avascular necrosis.

Keywords: Avascular Necrosis, Bone Marrow Aspirate Concentrate, Biological Augmentation, Osteonecrosis of femoral head

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INTRODUCTION

Avascular necrosis, also known as osteonecrosis, aseptic necrosis, or ischemic bone necrosis, is a degenerative joint disorder characterized by the death of the subchondral components of bone. The condition primarily impacts the epiphyses of weight-bearing long bones.[1] Osteonecrosis of the femoral head (ONFH) is a subtype of osteonecrosis in which impaired blood supply to the femoral head, due to various etiologic factors, results in bone tissue death.[2] As ONFH is an ischemic process, it may lead to substantial pain and functional limitation, adversely affecting quality of life. It may also progress from joint degeneration to coxarthrosis in up to 20% of patients.[2-5]

ONFH predominantly affects young adults (mean age, 33-38 years).[3] In the United States, approximately 10,000-20,000 new cases are reported annually, and subchondral collapse may occur in up to 80% of patients who do not receive adequate treatment.[3,4] The most critical period in the management of ONFH is the pre-collapse stage. [2-4] The Japanese Investigation Committee (JIC) classification may help predict subchondral collapse, guide the selection of appropriate treatment modalities in the pre-collapse stage, and facilitate prognostication of treatment outcomes.[6-8] The primary aim of this narrative review is to comprehensively evaluate and summarize contemporary diagnostic approaches, staging systems, and modern treatment strategies tailored to different stages of ONFH.

Literature Search Strategy

This manuscript was designed as a narrative review aimed at summarizing the current

concepts in the diagnosis, staging, and treatment of osteonecrosis of the femoral head (ONFH). The relevant literature was identified through searches of major medical databases including PubMed/MEDLINE, Scopus, and Web of Science. Keywords such as "osteonecrosis of the femoral head", "avascular necrosis", "core decompression", "bone marrow aspirate concentrate", "stem cell therapy", and "hip arthroplasty" were used in various combinations. Priority was given to recent publications and high-quality studies addressing diagnostic strategies, staging systems, and contemporary treatment approaches. Reference lists of relevant articles were also screened to identify additional pertinent studies.

Etiology and Pathophysiology

More than one-third of patients with ONFH have a history of corticosteroid use. During the COVID-19 pandemic, an increased incidence of ONFH has been reported, with corticosteroid exposure identified as the major contributing factor. [9-11] Alcohol misuse is implicated in 20%-30% of cases, whereas the etiology remains idiopathic in approximately 21%.[9,11] Other associated causes include trauma (femoral head fractures and hip dislocation), metabolic disorders (Gaucher disease, hyperparathyroidism, Cushing disease, and gout), autoimmune diseases (systemic lupus erythematosus), hematologic disorders (sickle cell anemia, bone marrow transplantation, and other hemoglobinopathies), and Legg-Calvé-Perthes disease. [2,9,12]

The most widely accepted mechanism of ONFH development is disruption of subchondral blood flow.[2] All etiologic factors are thought to act through three principal pathways that cause ischemia of bone structures: vascular pathologies, microthrombi formation, and extravascular compression. [1,2,12] In post-traumatic ONFH, injury to the retinacular arteries-which are crucial for the superolateral blood supply of the femoral head-has been emphasized. [13] Corticosteroid use has been associated with diffuse vasoconstriction, reportedly independent of dose.[3] Regardless of the underlying etiology, histological evidence of bone marrow necrosis and osteocyte death typically becomes apparent 24-72 hours after the onset of ischemia. This is followed by the release of free fatty acids and calcium ions into the extracellular matrix and initiation of an inflammatory response.[3] With prolonged ischemia, microfractures develop in the acellular trabecular bone, rendering it unable to withstand even normal loading. The limited proliferative capacity of bone cells during repair, reduction and disruption of the subchondral vascular network, and marked macrophage activation contribute to impaired regeneration.[14]

Diagnostic and Staging Methods

Clinical Evaluation and Physical Examination

In the early stages of ONFH, patients may be asymptomatic. As the disease progresses, pain typically begins in the groin and may radiate to the trochanteric region, gluteal area, and thigh. At this stage, pain intensity may increase during activities such as walking or climbing stairs without a clear restriction in hip range of motion, and may decrease with rest. With further progression, resting pain may occur, and limitation in hip abduction and internal rotation may become prominent. In advanced stages, articular cartilage degeneration may develop, leading to increased pain severity and reduced range of motion. In the terminal stage, acetabular cartilage involvement occurs, and clinical osteoarthritis becomes more evident.[1,2,9,11]

Imaging Modalities

When ONFH is suspected, anteroposterior and frog-leg lateral radiographs should be obtained as initial imaging studies.[1,2] However, the sensitivity of plain radiography is low in early disease [2,15]; therefore, magnetic resonance imaging (MRI), single-photon emission computed tomography (SPECT), and bone scintigraphy may be more valuable for early diagnosis (Fig.1).[2]

On plain radiographs, early-stage disease may appear entirely normal; alternatively, radiolucent or sclerotic areas may be

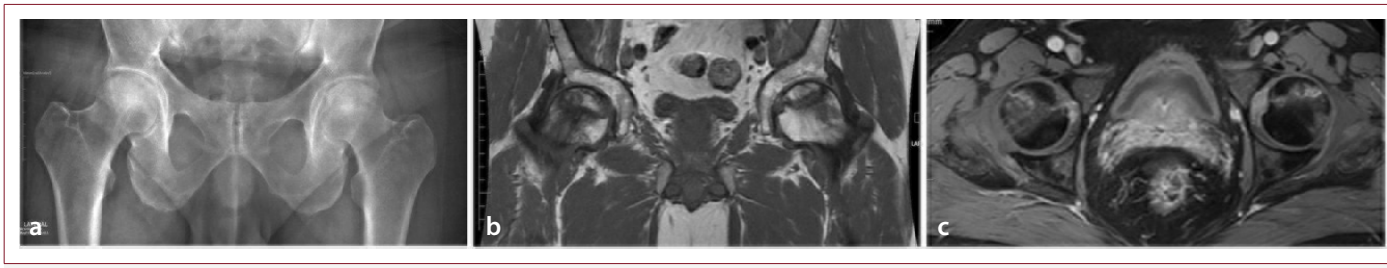

Figure 1. X-ray **(a)** and MRI **(b,c)** images of a 36-year-old male patient presenting with progressive worsening bilateral hip pain, without any underlying cause. **(a)** X-ray failed to demonstrate any pathological signs. **(b)** T1-weighted MRI image slice, showing well-defined lesions in the superior aspect of the femoral heads. **(c)** Bilateral bone marrow edema around the lesion in T2-weighted MRI image slice.

detected.[2,15] The pathognomonic "crescent sign" is observed in more advanced disease with subchondral collapse.[2,3,15] MRI is the most sensitive, specific, and widely used modality in ONFH and is considered the gold standard for imaging.[3,15] MRI provides clinically relevant information across all stages of the disease, including bone marrow changes; the size, location, and depth of the necrotic lesion; and the status of acetabular cartilage (Fig. 2). Moreover, MRI offers the advantage of monitoring treatment response.[1-3] The "double-line sign," described by Mitchell et al. [16] in 1987, remains highly specific for ONFH.[15,16] According to Stoica et al.[15], early-stage class A (fat-like) appearance corresponds to a normal marrow signal with a reactive sclerotic rim surrounding the lesion. Class B (blood-like) appearance may occur in the presence of subacute hemorrhage or insufficient inflammation/hyperemia. Class C (fluid-like) appearance is seen when inflammation, hyperemia, or lipid loss is prominent. When fibrosis and sclerosis predominate, class D (fibrous-like) appearance is observed, characterized by low signal intensity on all sequences.[15]

If contrast-enhanced MRI is not performed in early-stage disease, avascular foci may be overlooked. SPECT has been reported to be highly sensitive in detecting early avascular lesions and may be useful for early diagnosis.[17,18] When MRI is not feasible or provides insufficient information, SPECT can serve as an alternative.[15,17,18] On Tc-99m planar bone scintigraphy, a "donut sign," characterized by central hypoperfusion with peripheral reactive increased uptake, may be observed in the subacute stage of ONFH.[2,3]

Computed tomography (CT) is useful for evaluating morphological changes in ONFH (subchondral sclerosis, cystic areas, and collapse), but its sensitivity is limited in early stages. The "star sign," typically seen during the reparative phase, may be identified on CT. CT is particularly valuable for post-collapse evaluation and preoperative planning.[3,15]

Staging Systems

More than 16 classification systems have been described in the current literature for ONFH.[3,19] The Ficat-Arlet classification incorporates plain radiographs, MRI findings, and clinical features.[19] It consists of five stages (0-4). Stages 0-1 represent the "silent hip" phase with no radiographic findings. Stage 2 is characterized by sclerosis and cystic changes. Stage 3 includes subchondral collapse and the crescent sign. Stage 4 is the final stage, in which osteoarthritis and degenerative changes of the hip are present.[19]

The Steinberg classification, developed by incorporating bone scintigraphy findings, extends the Ficat-Arlet system by additionally considering lesion size and the extent of femoral head involvement.[20] This system includes seven stages, each subdivided into three categories based on the percentage of head involvement: A (<15%), B (15%-30%), and C (>30%).[20] Stage 0 indicates suspected AVN; stage I shows abnormalities on MRI or bone scintigraphy with normal radiographs; cystic and sclerotic changes on radiographs characterize stage II; and stages III-VI demonstrate radiographic signs of progressive osteonecrosis deterioration.[20]

The Association Research Circulation Osseous (ARCO) classification, updated in 2019, further evaluates lesion localization and has been suggested to be more reliable and clinically realistic than other systems; it is currently one of the most widely used classification methods.[21]

The JIC classification is among the most powerful systems for predicting collapse risk and guiding surgical planning in ONFH. The risk of subchondral collapse increases progressively from type A to type C2. Types A and B are associated with the lowest risk of collapse. Takashima et al.[22] reported that the JIC classification may be more effective than other systems in early-stage ONFH (Table 1).[8]

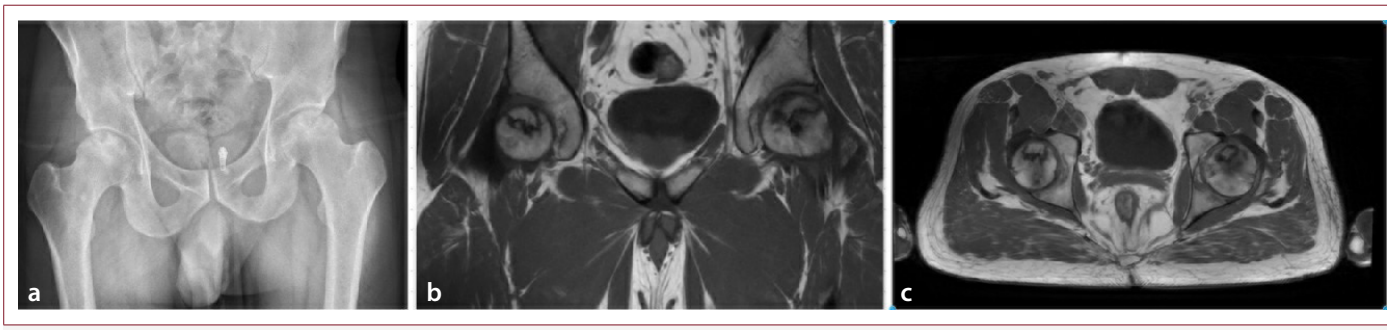

Figure 2. X-ray **(a)** and MRI **(b,c)** images of a 52-year-old male patient presenting with bilateral hip pain due to prior corticosteroid use. **(a)** X-ray demonstrated bilateral mixed sclerosis and cystic areas. **(b)** T1-weighted coronal and **(c)** T1-weighted axial slices showing well-defined anterosuperior lesions, indicating ONFH.

Table 1. Staging and classification systems used in ONFH

	Ficat-Arlet Classification		Steinberg Classification		ARCO Classification		JIC Classification
			Steinberg classification further grades extent of involvement as: A (<15%), B (15–30%), and C (>30%)		ARCO also incorporates lesion size (<15%, 15–30%, >30%) and location (medial, central, lateral), making it highly suitable for research and prognostic stratification.		The JIC system emphasizes location of necrosis and risk of collapse, rather than only disease stage.
0	Clinical symptoms: Asymptomatic X-ray: normal MRI: normal Bone scan: Decreased or heterogeneous uptake	0	Clinical symptoms: Asymptomatic X-ray: normal MRI: normal Bone scan: normal				
I	Clinical symptoms: Hip pain, activity-related X-ray: normal MRI: Bone marrow edema Bone scan: "Cold in hot" pattern	I	Clinical symptoms: Possible Pain X-ray: normal MRI: edema Bone scan: Abnormal Uptake	I	Clinical symptoms: Pain may be present X-ray: normal MRI: edema Bone scan: Abnormal uptake	A	Medial one-third of the weight-bearing area Low risk of collapse
II	Clinical symptoms: Persistent pain X-ray: Sclerosis, Cysts MRI: Well-defined necrotic area ± edema Bone scan: Increased peripheral uptake	II	Clinical symptoms: Hip Pain X-ray: Sclerosis, Cysts MRI: Necrotic lesion visible Bone scan: Increased uptake	II	Clinical symptoms: Progressive pain X-ray:Sclerosis/cysts, no collapse MRI: Clear demarcation of necrosis Bone scan: Increased uptake	B	Medial + central area Moderate risk of collapse
III	Clinical symptoms: Mechanical pain, limp X-ray: Crescent sign, subchondral fracture MRI: Subchondral collapse Bone scan: Intense increased uptake	III	Clinical symptoms: Mechanical pain Xray: Crescent sign MRI: Subchondral fracture Bone scan: Increased uptake	IIIA	Clinical symptoms: Mechanical pain X-ray: Subchondral fracture, collapse <2 mm MRI: Crescent sign, early deformity Bone scan: High uptake	C1	Extends laterally but not to acetabular edge High risk of collapse
				IIIB	Clinical symptoms: More severe symptoms X-ray: Collapse ≥2 mm MRI: Distorted femoral head contour Bone scan: High uptake	C2	Extends to the lateral acetabular edge Very high risk of collapse
IV	Clinical symptoms: Severe pain, stiffness X-ray: Femoral head collapse MRI: Deformity, cartilage degeneration Bone scan: Diffuse increased uptake	IV	Clinical symptoms:Functional limitation X-ray: Femoral head collapse MRI: Femoral head deformity Bone scan: Increased uptake	IV	Clinical symptoms: Advanced pain, limited motion X-ray: Secondary OA, acetabular changes MRI: Degenerative changes Bone scan: Diffuse increased uptake		

Table 1. Continued

Ficat-Arlet Classification	Steinberg Classification	ARCO Classification	JIC Classification
	V Clinical symptoms: Severe pain X-ray: Joint-space narrowing MRI: Secondary OA changes Bone scan: Diffuse increased uptake		
	VI Clinical symptoms: Severe disability X-ray: Advanced OA MRI: Advanced degenerative changes Bone scan: Diffuse increased uptake		

Contemporary Treatment Options

A broad range of treatment options is available for patients diagnosed with ONFH, ranging from conservative management to total hip arthroplasty. Subchondral collapse of the femoral head may occur in up to 80% of untreated patients.[3] Selection of the most appropriate treatment strategy depends on the patient's age, pain severity, lesion location, size and depth, comorbidities, and the presence of cartilage degeneration.[2,3] Outcomes are more favorable when treatment is initiated before femoral head collapse[2,3]; therefore, early diagnosis is the most critical step in the management of ONFH. It should be noted that ONFH secondary to sickle cell anemia, corticosteroid exposure, or immunosuppressive therapy may be associated with a higher risk of a poor prognosis.[2,3,4]

Treatment may be broadly categorized into three groups based on disease stage, lesion size, and patient age/activity level: conservative treatment methods, joint-preserving procedures, and salvage procedures.

Conservative Treatment Methods

The primary goal of conservative management, particularly in early-stage disease (ARCO stages I and II), is to prevent subchondral collapse. Commonly used approaches include activity modification, protected weight-bearing, hyperbaric oxygen therapy (HBOT), pharmacological treatments (e.g., iloprost [PGI2], bisphosphonates, and anticoagulants), and extracorporeal shockwave therapy (ESWT).[3,4,23] Among pharmacological agents, bisphosphonates and deferoxamine have been shown to reduce steroid-induced bone resorption and enhance regeneration.[24] Iloprost (PGI2) has also been reported to be effective when used alone or in combination with joint-preserving procedures in early-stage ONFH (ARCO stages I and II).[25]

Currie et al.[26] reported outcomes of 30 years of experience with HBOT and demonstrated that it significantly prevented progression of ONFH. Guo et al.[27] showed that anticoagulant therapy may be effective in idiopathic ONFH; however, they suggested that it does not provide a protective effect in secondary ONFH. ESWT has been reported to be more effective than core decompression, and the addition of HBOT and bisphosphonates may further enhance the effects of ESWT.[28] Overall, the combined use of conservative modalities is considered reasonable, and these strategies may also serve as adjuncts to surgical treatment in appropriately selected patients.

Joint-Preserving Surgical Procedures

Core Decompression (mechanism and current modifications)

The principal objective of core decompression is to reduce intraosseous pressure within the femoral head and thereby improve perfusion.[3,4] It is the most commonly performed primary treatment option for patients without subchondral collapse, and its effectiveness has been enhanced by combining it with biological and/or cellular therapies.[3,4,29]

Conventional single-track drilling has been used historically for core decompression.[30] Recently, multiple drilling techniques have gained popularity due to lower risks of hip fracture, local wound complications, and revision surgery.[30,31] Additionally, retro-drilling with an expandable reamer through a single core is gaining popularity in terms of reducing complications and allowing application of adjunct biological augmentations.[29] (Fig. 3) Following either technique, avoidance of weight-bearing for 6 weeks and refraining from high-demand activities for up to 12 months are generally recommended.[3,4]

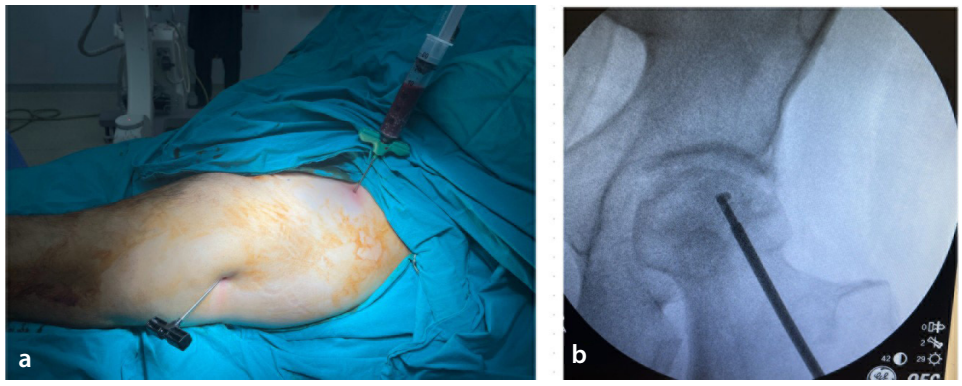

Figure 3. (a) Obtaining BMAC and core decompression using retrograde drill is possible in a minimally invasive manner. **(b)** Retrograde drilling of the femoral head to perform debridement and core decompression of the osteonecrotic lesion.

Grafting Techniques

Non-vascularized bone grafting

Non-vascularized bone grafting has been used for decades to prevent subchondral collapse in ONFH. Surgical techniques such as the Phemister, lightbulb, and trapdoor procedures have been described to support and repair the subchondral bone. [3] Mei et al.[32], in a study comparing allogenic non-vascularized bone grafting with core decompression, reported better femoral head survival in the core decompression group. With devices for mosaicplasty, cylindrical autologous grafts taken from the iliac crest can be utilized for filling the defect after core decompression with ease (Fig. 4).

Vascularized bone grafting (VFG)

Vascularized fibular grafting (VFG) has been recommended as a hip-preserving option in selected patients with ARCO stages I-III disease, particularly in cases of failed core decompression, collapse <3 mm, and femoral head involvement <50%.[3,4] Yoo et al.[33] reported acceptable long-term outcomes at a mean follow-up of 13.5 years after VFG.

Biological Augmentation

Despite the availability of multiple treatment options, no universally accepted gold-standard treatment for ONFH has yet been established. In recent years, interest has increased in orthobiologic therapies-such as mesenchymal stem cells (MSCs), bone marrow aspirate concentrate (BMAC), hematopoietic stem cells, endothelial progenitor cells, platelet-derived products, and growth factors-to prevent collapse and delay or avoid total hip arthroplasty.[5]

Bone marrow aspirate concentrate (BMAC) and stem cell applications

Several studies have suggested that autologous bone marrow-derived mesenchymal therapies and BMAC, used as an adjunct

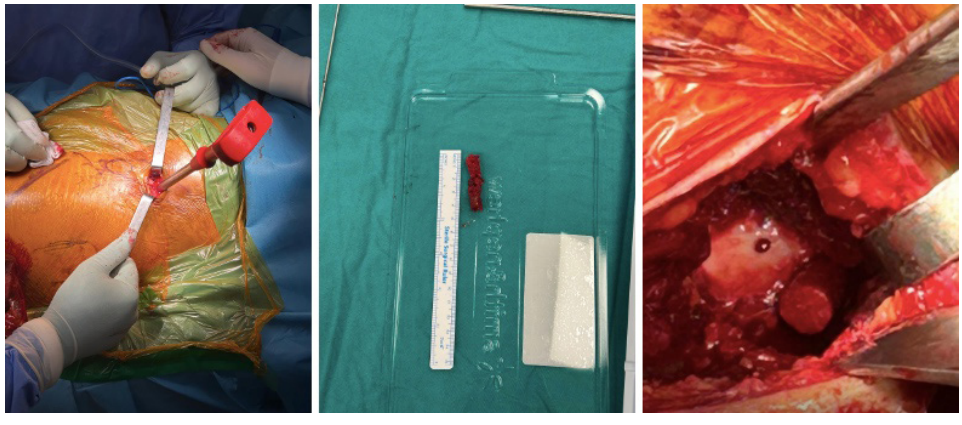

Figure 4. Cylindrical cancellous bone grafts harvested from the iliac crest are inserted through the tunnel created on the lateral aspect of the proximal femur.

to core decompression, may be more effective in preventing the need for total hip arthroplasty and improving clinical outcomes.[5,34,35] BMAC is an autologous bone marrow-derived cellular concentrate typically obtained via iliac crest aspiration and contains MSCs, hematopoietic stem cells, endothelial progenitor cells, platelets, growth factors, and cytokines. Because it is minimally invasive, relatively easy to apply, and supported by preclinical in vitro and in vivo studies demonstrating efficacy in osteonecrosis, BMAC has been increasingly used in combination with core decompression and has become progressively more popular in recent years.[5,29,35]

In addition to decompression, BMAC may support recovery after osteonecrosis through osteoconductive and osteoinductive effects. Zaffagnini et al.[5] found that BMAC was more effective than other orthobiologic strategies in reducing the need for total hip arthroplasty. Wang et al. [36] concluded that MSC therapy was the most effective approach for reducing the need for total hip arthroplasty, followed by BMAC therapy.

Platelet-rich plasma (PRP) and growth factors

Autologous platelet-rich plasma contains high concentrations of platelets and various growth factors (PDGF, TGF-β, FGF, EGF, VEGF), and has been shown to influence tissue repair as well as MSC proliferation and differentiation. In ONFH, PRP is thought to stimulate angiogenesis and osteogenesis and to exert beneficial effects by inhibiting inflammatory reactions and apoptosis. Therefore, PRP has been recommended as an adjunct to core decompression in early-stage disease (ARCO stages I and II).[37] Houdek et al. suggested that, in patients without subchondral collapse and after cessation of corticosteroid use, combining BMAC and PRP with core decompression may represent the most appropriate treatment strategy.[38]

Osteotomies

The goal of osteotomy in ONFH is to redistribute load across the femoral head to prevent subchondral collapse. Intertrochanteric (valgus/varus) and transtrochanteric (rotational) osteotomies have been described. Osteotomy may be considered in selected patients younger than 40 years with BMI <24, ARCO stages II-III, JIC type B lesions, adequate viable bone stock (angle >150° between the central vertical line and the lateral edge of the necrotic region on coronal MRI), no acetabular pathology, and preserved hip range of motion.[3,4,39]

Salvage Procedures

In advanced-stage ONFH (ARCO stage IV) that remains symptomatic despite conservative and joint-preserving interventions, arthroplasty may provide substantial pain relief and functional improvement. Because many patients are young and active, thorough counseling regarding potential complications of arthroplasty is essential.[2,4]

Total hip arthroplasty

With advances in bearing surfaces-particularly ceramic-on-ceramic and highly cross-linked polyethylene (HXLPE)-ceramic articulations-implant survivorship has improved. Zhang et al. reported that, among patients undergoing total hip arthroplasty (THA) for ONFH, medical complications increased with age, whereas revision rates and surgical complication rates decreased. Overall complication rates were higher in ONFH patients than in those with osteoarthritis (OA).[40] Tanaka et al.[41], in a nationwide database study including approximately 25,000 patients, reported significantly higher rates of hip dislocation, mortality, and additional complications after THA performed for ONFH compared with THA performed for OA. In contrast, Osawa et al. reported comparable 10-year survivorship and complication rates between THA performed for OA and THA performed for ONFH when matched for age and sex.[42]

Hip resurfacing arthroplasty

Hip resurfacing has been described as an alternative to conventional THA for young patients with ONFH, with the potential benefits of bone stock preservation and excellent short-term outcomes.[43] However, none of the hip resurfacing implants met the 10-year survivorship benchmark, and concerns about pseudotumor formation, femoral neck fractures, and metal-ion toxicity associated with metal-on-metal bearings have led to a decline in its use in recent years.[44,45]

CONCLUSION

ONFH remains a frequently debated condition in orthopedic practice, particularly with respect to etiology and treatment decision-making. Treatment selection should be guided by patient age, activity level, and the presence or absence of femoral head collapse and/or articular cartilage degeneration. In patients without subchondral collapse, combined application of core decompression with BMAC and/or PRP, along with adjunctive hyperbaric oxygen therapy, may be considered as part of a multidisciplinary joint-preserving strategy.

DECLARATIONS

Ethics Committee Approval: This is a review, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent: Informed consent was not deemed necessary for this study.

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Peer-review: Externally peer-reviewed.

ABBREVIATIONS

ARCO: Association Research Circulation Osseous

AVN: Avascular necrosis

BMAC: Bone marrow aspirate concentrate

BMI: Body mass index

CT: Computed tomography

EGF: Epidermal growth factor

ESWT: Extracorporeal shock wave therapy

FGF: Fibroblast growth factor

HBOT: Hyperbaric oxygen therapy

HXLPE: Highly cross-linked polyethylene

JIC: Japanese Investigation Committee

MRI: Magnetic resonance imaging

MSC: Mesenchymal stem cell

OA: Osteoarthritis

ONFH: Osteonecrosis of the femoral head

PDGF: Platelet-derived growth factor

PGI2: Prostacyclin

PRP: Platelet-rich plasma

SPECT: Single-photon emission computed tomography

TGF-β: Transforming growth factor beta

THA: Total hip arthroplasty

VEGF: Vascular endothelial growth factor

VFG: Vascularized fibular grafting

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