REVIEW ARTICLE

Osteonecrosis of the Hip: A Primer

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ABSTRACT

In this report, we deliver a concise and up-to-date review of osteonecrosis, a pathologic, painful, and often disabling condition that is believed to result from the temporary or permanent disruption of blood supply to an affected area of bone. We will discuss the epidemiology (disease distribution), pathogenesis (mechanism of development), etiology (associated risk factors, causes, and disorders), clinical manifestations (reported symptoms and physical findings), diagnosis and classification, and treatment options for hip osteonecrosis.

INTRODUCTION

The intent of this article is to present an update on osteonecrosis (ON) affecting the femoral head or hip joint and how it can best be managed in the adult population. Specifically, this report will encompass the epidemiology, pathogenesis, etiology, clinical manifestations, diagnosis and classification, and treatment options for hip ON. ON, also referred to as avascular necrosis, aseptic necrosis, or ischemic bone necrosis, is associated with many disorders and risk factors that cause mature bone cells to die, leading to bone destruction (eg, collapse) or end-stage arthritis of the femoral head. The condition can occur in any bone in the body (eg, upper extremity, knees, shoulders, and ankles), or in more than 1 bone at different times, but it most commonly affects the hip joint. When initially diagnosed in an area other than the hip, the hip should simultaneously be evaluated clinically and with radiographic and other imaging studies. The causes of ON are classified as either traumatic (related to an injury) or atraumatic (not related to an injury). Accurately diagnosing and classifying ON are important in helping to direct treatment options. Identification of associated risk factors and patient education are important in successful management of ON. Targeting associated risk factors, pharmacologic management, and/or surgery, including joint preserving procedures and total hip arthroplasty (THA), also play significant roles in the clinical care of patients with ON.

EPIDEMIOLOGY OF HIP OSTEONECROSIS

Although the exact prevalence of ON is unknown, the incidence is estimated to be between 20,000 to 30,000 newly diagnosed patients each year in the US.[1] ON is the underlying diagnosis in approximately 10% of all THA in the US.[2,3] ON affects people of all ages, although it is most commonly seen in patients between the ages of 30 and 65 years.[4] The mean age at diagnosis is typically younger than age 50 years.[3] The male-to-female ratio varies depending on the associated comorbidities. For example, alcohol-associated ON is more common in men, whereas ON associated with systemic lupus erythematosus (SLE) is more common in women.[3]

More than 20,000 people each year require hospital treatment for hip ON.[4] In many of these cases, both hips are affected by the condition. Most commonly, ON affects the proximal end (epiphysis) of the femur (hip bone).

PATHOGENESIS OF HIP OSTEONECROSIS

The mechanism(s) by which hip ON develops remains unclear. For the most part, hip ON is believed to result from the combined effects of genetic predisposition, metabolic factors, and local factors affecting blood supply including vascular damage, increased intraosseous pressure, and mechanical stresses.[2,5,6] Most experts agree that a lack of blood supply to the femoral head and bone marrow, which produces stem cells and platelets, causes death of the osteocytes (cells within mature bone) and/or mesenchymal cells (stem cells that form cartilage, bone, and fat).[7] The result is demineralization or resorption of the dead tissue by new but weaker osseous tissue (trabecular thinning), subsequently leading to subchondral fracture and collapse of the femoral head.

Other proposed mechanisms for the pathogenesis of ON include vasoconstriction-induced changes caused by the adverse effects of excess glucocorticosteroids affecting bone and venous endothelial cells[8,9] and excess glucocorticoid-associated ON involving alterations in circulating lipids believed to cause microemboli in the arteries that supply bone with blood.[10]

ETIOLOGY OF HIP OSTEONECROSIS

A combination of traumatic and atraumatic factors can directly contribute to the etiology of ON (see Sidebar: Etiologic Factors Associated with Osteonecrosis). On the basis of longitudinal cohort studies and meta-analyses, direct risk factors have been discovered that play a definitive etiologic role in the development of ON. Associated risk factors, however, account for most of the links to the eventual development of ON.[11]

Traumatic Causes of Hip Osteonecrosis

Traumatic causes of ON include femoral neck fractures or dislocations as well as direct injury of bone of marrow elements (eg, related to radiation injury, dysbarism, or Caisson disease). The mechanism involved in femoral neck fractures or dislocations is damage to the extraosseous blood vessels, which results in disrupted blood supply to the affected region of the hip. Hip dislocation is another type of traumatic injury, which affects approximately 20% of trauma-related ON patients.[12]

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Caisson disease (eg, decompression in scuba diving) causes the formation of nitrogen bubbles that can occlude arterioles, leading to ON. Patients who develop symptoms can develop hip ON years after exposure to this process. The depth and duration of pressure and number of exposures are important factors in the progression of this disorder.[13]

Atraumatic Causes of Hip Osteonecrosis

Numerous studies report prolonged use of corticosteroids associated with the development of ON can be directly related to duration and total dosage of the medication.[14-16] Patients treated with prolonged high doses of glucocorticoids appear to be at the greatest risk of developing ON; however, these patients often have multiple other risk factors.

Glucocorticoid–induced ON develops in 9% to 40% of patients receiving long-term therapy, and develops much less frequently in patients receiving short-term therapy.[17] One meta-analysis and systematic review identified an incidence of ON in nearly 7% of patients who used < 2 g of corticosteroids.[18] From this meta-analysis, a lower risk was seen in patients treated with doses of prednisone less than 15 mg/d to 20 mg/d.[18] One population-based study of 98,390 patients showed the incidence of hip ON among patients who had received a single short-term, low-dose methylprednisolone taper pack was 0.13%, compared with 0.08% in patients who were not prescribed a methylprednisolone taper pack, thus indicating a number needed to harm of 2041 patients.[19]

Alcohol use has been associated with approximately 31% of patients who develop hip ON.[3,20-22] Excessive alcohol consumption related to ON of the hip is believed to result from the decreased bone genesis caused by excess lipid formation and increased intracellular lipid deposits, leading to osteocyte death and ON.[23]

High doses of corticosteroids and excessive alcohol use together present the highest associated direct risk factors for the development of hip ON[24] and account for more than 80% of cases not related to trauma.[3,6] One study compared 112 patients who had idiopathic hip ON and no history of systemic corticosteroid use with 168 controls.[3,20] An elevated risk for regular alcohol drinkers and a clear dose-response relationship with alcohol were noted, compared with controls. The relative risks were 3.3, 9.8, and 17.9 for current consumers of less than 400 mL/wk, 400 mL/wk to 1000 mL/wk, and more than 1000 mL/wk of alcohol, respectively.[9]

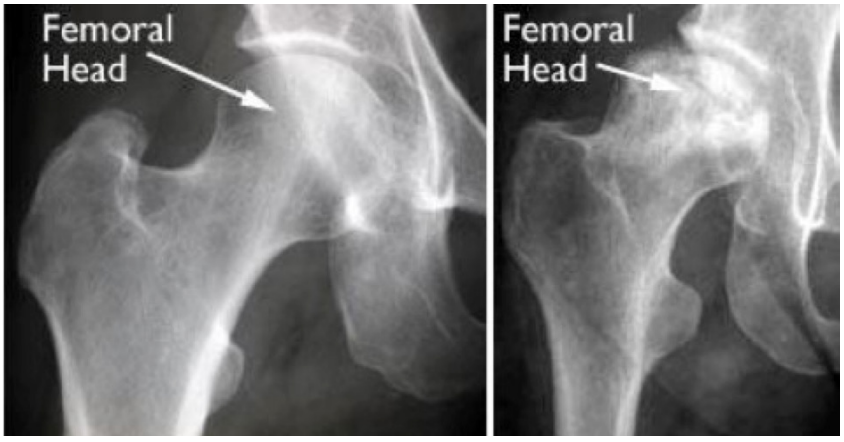

Figure 1. Left, radiograph of a healthy hip joint. Right, radiograph of a hip joint where the osteonecrosis has progressed to collapse of the femoral head.

ON is common in patients with sickle cell disease because of its propensity to cause red blood cell sickling and bone marrow hyperplasia. Approximately 50% of affected patients develop ON by the age of 35 years.[25] Sickle cell hemoglobinopathy can directly cause vascular obstruction and ON.

The development of ON has been reported in 3% to 30% of patients with SLE, and those most at risk are patients who have taken glucocorticoids with regular doses of prednisone greater than 20 mg/d.[3,26-28] ON has been reported in as many as 60% of patients with Gaucher disease (a hereditary disorder) because of its ability to directly obstruct vascular supply.[3,29,30] Gaucher disease is an autosomal recessive inherited disorder of metabolism where a type of fat (lipid) called glucocerebroside cannot be adequately degraded. Normally, the body makes an enzyme called glucocerebrosidase (a normal part of the cell membrane) that breaks down and recycles glucocerebroside.[31]

Other less common but apparent links to hip ON include patients with antiphospholipid antibodies, Cushing disease,[29] and SLE. The development of acute lymphoblastic leukemia, chronic myeloid leukemia, and acute myeloid lymphoma[3,32] places patients at increased risk for ON related to the treatment with steroids for these conditions.

Pancreatitis (usually associated with use of corticosteroids), pregnancy, chemotherapy, smoking, vasculitis, pleuritis, and central nervous system factors such as an inflammatory response resulting in a reduction in the number of sympathetic nerve fibers (as seen in rheumatoid arthritis, Crohn disease, Charcot foot, and inflammatory bowel disease), have been associated with ON.[33]

There is some evidence that hip ON may have a genetic basis underlying associated risk factors.[34] For example, men are affected as much as 3 times more than women when excessive alcohol use is the associated risk factor. However, when lupus or corticosteroid use are the associated risk factors, women are affected more often than men.[26,27,32] SLE is approximately 9 times more common in women than in men.[35] This increased susceptibility may be made possible, at least in part, owing to differences related to hormones and sex chromosomes.[35] Chronic renal failure or end-stage renal disease in patients on hemodialysis, hyperuricemia/gout, HIV infection, hyperlipidemia, organ transplantation, and intravascular coagulation are also linked to the development of ON.[31,32,36-39] Despite the many possible associations and links, an estimated 20% of ON cases are labeled as idiopathic or of unknown etiology.[7]

CLINICAL MANIFESTATIONS OF HIP OSTEONECROSIS

Hip pain is the most commonly reported symptom of later-stage ON, although a small proportion of patients may not have symptoms. Pain in the groin is the most commonly reported symptom, followed by pain referred into the thigh and buttock. Pain can present with weightbearing or joint motion. Pain at rest occurs in approximately two-thirds of patients with ON, and pain at night occurs in approximately one-third of patients.[33] Pain in multiple locations of the body is rare and suggests a multifocal process. Physical findings of hip ON are generally nonspecific but may entail reduced range of motion of the affected joint, painful ambulation, Trendelenburg sign, and/or crepitus.[3,40,41]

Clinical Assessment of Hip Osteonecrosis

ON of the hip is generally addressed by 1) review of a patient's medical history, 2) obtaining appropriate radiologic evaluation, 3) determining the stage of the condition, and 4) developing a plan for treatment options.[42,43] When evaluating a patient for ON, questions should be directed at assessing a history of pain, use of medications (especially corticosteroids), surgery, pregnancy, trauma, chronic medical conditions (especially sickle cell disease, Gaucher disease, autoimmune disease, and leukemia), smoking, and/or alcohol use. When asking about injuries/illnesses, it is important to carefully explore injuries related to hip fractures, dislocations, or scuba diving because Caisson disease is atraumatic.

DIAGNOSIS AND CLASSIFICATION OF HIP OSTEONECROSIS

Diagnosing hip ON in the initial stages of the disorder is important for management[43-46]; at initial stages, the disease may not progress. In most cases, patients with early-stage ON are generally without symptoms and are identified incidentally; unfortunately, most patients do not present for evaluation until the ON has reached later stages. Although there is presently no definitive treatment known to permanently halt ON from progressing to later stages, there are treatment methods, such as lipid lowering agents, anticoagulants, and bisphosphonates, currently being used for this purpose.[36-38]

Table 1. Ficat & Arlet classification system of the femoral head

Classification	Clinical	Radiographs	MRI
Stage 0	No symptoms; preclinical	Normal	Normal
Stage 1	Possible groin pain	Normal or mild osteopenia	Possible edema
Stage 2	Groin pain and stiffness; pain with activity	Osteopenia and/or subchondral cysts; diffuse porosis; precollapse of joint space	Outlines area of involvement of the femoral head
Stage 3	Groin pain, stiffness, radiation of pain; pain with activity	Crescent sign and/or subchondral collapse (flattening) of joint with secondary degenerative changes; loss of sphericity of femoral head	Same as radiographs
Stage 4	Groin pain and limp; pain at rest	End-stage disease with collapse; extensive destruction of joint; reduced joint space	Same as radiographs

MRI = magnetic resonance imaging

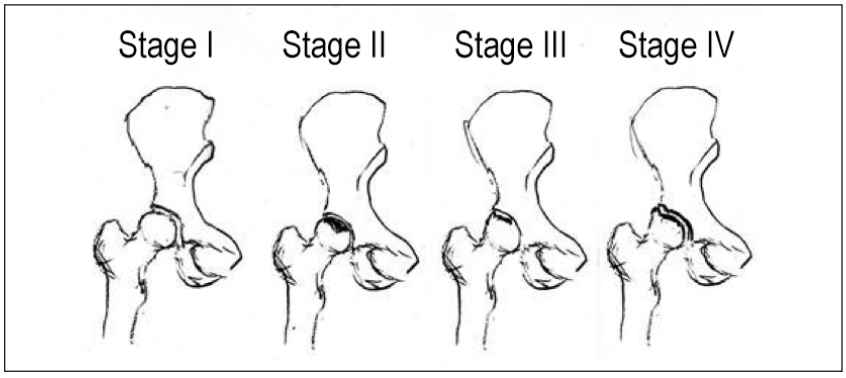

Figure 2. Progression of osteonecrosis using the Ficat & Arlet classification system. Osteonecrosis can progress from a normal, healthy hip (Stage I) to the collapse of the femoral head (Stage IV).

A diagnosis of ON can accurately be made when a patient is symptomatic, imaging findings are compatible, and other causes of pain and bony abnormalities either are unlikely or have been excluded. Beyond the clinical and physical examination, imaging techniques such as radiographs and magnetic resonance imaging (MRI) scanning are also used for diagnosis. Plain radiographic evaluation is performed first, followed by MRI. MRI has been reported to be < 99% specific and sensitive for detecting early ON.[47,48] MRI images can also quantitatively assess the size of the lesion or involvement of the affected bone by digitizing the area of the femoral head occupied by bone with abnormal texture.[49] MRI changes include well-demarcated and homogeneous focal lesions on T_1-weighted images with a single-density line separating normal and ischemic bone, as well as a second high-intensity line on T_2-weighted images (the pathognomonic double-line sign) representing hypervascular granulation tissue.[3] This level of imaging detail is useful because the size and extent of the lesion of the affected bone is important and can help direct treatment. For end-stage disease, however, use of MRI in patients with ON may be unnecessary because treatment options at this stage can be limited.

These findings are often classified using the 4-stage Ficat and Arlet system, which is described here and in Table 1. The plain radiograph can remain normal for months after the onset of symptoms such as groin pain (Stage I). The earliest radiographic findings are usually mild density changes, followed by sclerosis and cysts (Stage II). Findings then progress to the pathognomonic crescent sign (subchondral radiolucency seen in the anterolateral aspect of the proximal femoral head) from subchondral collapse (Stage III), and subsequent loss of sphericity (measurement of the roundness) or collapse of the femoral head with eventual joint-space narrowing and

degenerative changes in the acetabulum that are visible (Stage IV). Key radiographic features to look for include 1) stage (precollapse vs postcollapse), 2) size of lesion, and 3) amount of head depression.

A computed tomography scan producing a 3-dimensional picture of the bone has moderate sensitivity but is nonspecific and can pose a significant radiation burden to patients. Computed tomography can have some specificity if there is already femoral head collapse. Fortunately, most clinicians are assured with their diagnosis of ON without computed tomography scanning, which is generally reserved for distinguishing precollapse and postcollapse disease.

Differential Diagnosis of Hip Osteonecrosis

Because patients with symptomatic hip ON can present with symptoms similar to many other hip pathologies, these should be adequately ruled out before final diagnosis. Bone marrow edema syndrome and subchondral fractures are two of many potential diagnoses that need to also be considered.

Etiologic Factors Associated with Osteonecrosis

Traumatic-associated risk factors
Femoral neck fracture
Dislocation or fracture-dislocation
Sickle cell disease
Hemoglobinopathies
Caisson disease (dysbarism)
Gaucher disease
Radiation

Atraumatic-associated risk factors
Corticosteroid administration
Alcohol use
Systemic lupus erthyematosus
Cushing disease
Hypersecretion of cortisol (rare)
Chronic renal failure/hemodialysis
Pancreatitis
Pregnancy
Hyperlipidemia
Organ transplantation
Intravascular coagulation
Thrombophlebitis
Cigarette smoking
Hyperuricemia/gout
HIV

Other potential risk factors
Idiopathic causes

Bone marrow edema syndrome, also known as transient osteopenia of the hip, may occur in isolation or in association with injuries, particularly those that result in neurologic damage. In the latter situation, chronic pain and transient osteopenia are features of the complex regional pain syndrome (also known as reflex sympathetic dystrophy, causalgia, and other terms).[3] Bone marrow edema syndrome can be differentiated from ON on the basis of histologic and MRI findings.

Subchondral fracture of the femoral head typically occurs in patients with preexisting osteopenia and is generally thought to represent an insufficiency fracture.[50] These fractures may be difficult to visualize with plain radiographs. Subtle flattening is sometimes present with early lesions; collapse of the femoral head is progressive.

CLINICAL MANAGEMENT OF HIP OSTEONECROSIS

Factors to consider when developing an optimal management approach for symptomatic ON of the hip should be aimed at treating the stage and degree of involvement of ON, the extent and location of bony involvement, the presence (or absence) of symptoms, and the patient's comorbidities. The goal of therapy is to preserve the biological hip joint for as long as possible while also taking into consideration quality of life issues such as patient age, mobility, occupation, and lifestyle. Three main therapeutic options for management of hip ON include 1) nonoperative management, 2) joint-preserving procedures, and 3) THA.

The effects of atraumatic causes of hip ON pose special concerns. For those affected, 67% report no symptoms but may eventually go on to have a collapsed joint.[51] The natural history of asymptomatic medium-sized, and especially large, osteonecrotic lesions is progression to worsening of the condition and eventually end-stage disease and collapse of the hip in a substantial number of patients. For those with symptoms, approximately 80% to 85% of cases will result in collapse of the femoral head within 2 years.[6] Early diagnosis of ON may therefore provide the opportunity for early treatment, which can prevent collapse and, ultimately, the need for total joint arthroplasty. However, most patients present late in the course of the disease, and a high index of suspicion is necessary for those with known or probable risk factors, particularly patients with high-dose corticosteroid use.[3]

Similarly for patients with asymptomatic hip ON, the size, extent, and location of the necrotic lesion affecting the femoral head should be considered. Generally, lesions affecting less than 15% of the femoral head are best managed nonoperatively; lesions between 15% to 30% should be managed surgically; and lesions involving more than 30% of the femoral head are likely to progress to collapse, despite surgical intervention, and eventually require THA.[3,52,53]

Nonsurgical Treatment Options in Hip Osteonecrosis

Physical Therapy

Physical therapy may provide relief and alleviate some symptoms but generally will not preclude progressive hip ON from advancing to later stages.[54] Similarly, restricting weight-bearing with the use of assistive devices such as crutches or a cane may be useful to control symptoms of pain, weakness, and antalgic gait. Physical therapy is not appropriate if the goal of treatment is to prevent the hip from requiring THA, and to date there is no evidence that weight-bearing restrictions are helpful in preventing progressive ON disease from advancing to end-stage disease.

Medications

Nonsteroidal anti-inflammatory drugs and acetaminophen may provide temporary relief of pain in symptomatic patients. Opioid medications may be used judiciously and for short periods of time when other agents are ineffective to manage moderate-to-severe pain while surgical options are being considered.

Investigational medication options currently being used but that are not proven or reliably used to treat ON include 1) anticoagulants, 2) bisphosphonate antiresorptive agents, 3) cholesterol lowering statins, and 4) hyperbaric oxygen.

Surgical Options in Early-Stage Hip Osteonecrosis

Core Decompression

Core decompression is a minimally invasive surgical technique performed to

manage symptoms in early stages (precollapse) of the condition (eg, Ficat and Arlet Stages I and II). The procedure involves drilling holes into the femoral head to relieve pressure and create channels for new blood vessels to nourish the affected areas. The published success rates of core decompression vary greatly from 40% to 100%, depending on patient population.[35] Higher success rates after core decompression are seen in patients with the earliest disease stages. Patients with successful core decompression procedures typically return to unassisted ambulation after several months and can have complete pain relief.[55]

Bone Grafting

Core decompression can be combined with bone grafting to help regenerate healthy bone and support cartilage at the hip joint. A bone graft is healthy bone tissue that is transplanted to the area of necrotic or dead bone. A standard technique uses an autograft that involves taking bone from one part of the body and moving it to another part of the body. A bone graft that is harvested from a donor or cadaver is called an allograft and is typically acquired through a bone bank.

Bone Marrow Aspirate Concentration

The bone marrow aspirate concentration injection procedure with core decompression involves the use of concentrated bone marrow that is injected into the dead bone of the hip. This investigational technique harvests stem cells from a patient's bone marrow and injects them into the area of ON.[9] The bone marrow aspirate concentration procedure is hypothesized to prevent further progression of the disease and to stimulate new bone growth.[56]

Percutaneous Drilling

Another surgical option is percutaneous drilling. In this procedure, a hole is drilled percutaneously through the femoral neck to the affected site in the femoral head. One report on 45 hips with a mean follow-up of 24 months reported 24 (80%) of 30 hips with Ficat and Arlet Stage I disease had successful outcomes (defined as Harris Hip Score < 70).[57] A more recent study comparing multiple drilling vs standard core decompression showed favorable results in favor of percutaneous drilling.[28]

Surgical Options in Advanced-Stage Hip Osteonecrosis

Vascularized Bone Graft

A vascularized fibula graft is a more involved surgical procedure in which a segment of bone is taken from the fibula with its blood supply. The graft is then transplanted into a hole created in the femoral neck and head, and the artery and vein are reattached to help heal the area of ON.[55]

Osteotomy

Osteotomy in hip ON can be performed to remove necrotic bone away from primary weight-bearing areas. Although this operation may delay THA surgery, it is most useful in patients with idiopathic ON who demonstrate small precollapse or early postcollapse of the femoral head. A consequence of osteotomies, however, is that they make subsequent THA more challenging and are often associated with an increased risk of nonunion of the bone.

Nonvascularized Bone Graft

There are 3 types of nonvascularized bone grafting surgeries: 1) trapdoor procedure, 2) lightbulb technique, and 3) Phemister technique. The trapdoor procedure is one in which autogenous cancellous and cortical bone grafting have been successful in Ficat and Arlet Stage III hip ON in patients with small- to medium-sized lesions. A review of the results of 30 trapdoor operations performed on 23 patients with Ficat and Arlet Stage III or Stage IV ON of the femoral head performed through a so-called trapdoor made in the femoral head revealed a good or excellent result as determined by the Harris Hip Score system.[11]

Lightbulb Technique

The lightbulb technique uses a cortical window in the anterior aspect of the femoral neck. Necrotic bone can be removed using this window, which can be later packed with nonvascularized bone graft. Wang et al[55] evaluated 110 patients (138 hips) who underwent the lightbulb procedure. At mean follow-up of 25 months, mean Harris Hip Scores improved from 62 to 79 points. A total of 94 hips (68%) were considered to have successful outcomes at latest follow-up. Radiographic improvements were noted in 100% of Association Research Circulation Osseous Stage IIa patients, 77% in stage IIb patients, and 51% in stage IIc and IIIa patients.[55]

Phemister Technique

In the Phemister technique, a trephine is inserted through the femoral neck to create a tract to the lesion. A second trephine is then inserted to create another tract to the lesion site. A cortical strut graft can then be placed in the lesion. A recent review reports this procedure to have a clinical success rate ranging from 36% to 90%.[25]

Total Hip Arthroplasty

Once the femoral head has undergone major collapse, replacing the hip joint is the only practical operative option and offers the most predictable pain relief in advanced ON. THA is successful in relieving pain and restoring function in the majority of patients.[45-47] In THA, the diseased cartilage and bone constituting the hip joint is replaced with artificial implants made of metal and plastic. A prosthetic hip replacement generally lasts 15 years before it might wear out and need to be revised. For the younger age group, a THA may be a suboptimal solution because of possible activity restrictions. Additionally, because prostheses have longevity restrictions—components wear after long-term use—these patients will likely require a revision THA later in life. THA must be carefully considered and balanced against quality of life issues, but it is not absolutely contraindicated for younger patients.

PATIENT EDUCATION ABOUT HIP OSTEONECROSIS

Prevention of Osteonecrosis

Patient education about risk factors, therapies, and management is essential for patients to make better-informed decisions about their condition. The process of ON education involves identification of an individual's associated disorders and risk factors related to ON.

Patients with asymptomatic ON may have a high prevalence of progression to symptomatic disease and femoral head collapse. Education for patients with asymptomatic disease is precautionary and imperative to ensure modification of risk factors and optimization of care. Preventing atraumatic ON requires 1) avoiding excessive use of alcohol defined as < 15 drinks/wk for men and < 8 drinks/wk for women,[10] 2) avoiding smoking, and 3) reducing corticosteroids to the lowest

possible therapeutic dose. Informing patients about the correlation between corticosteroid use and potential development of ON is critical in management of this condition.

Prevention of Progression of Osteonecrosis

Patients diagnosed with early-stage ON should be advised of the aforementioned precautions and should avoid placing excessive pressure on their joints, follow a healthy diet, and maintain an appropriate weight to mitigate progression of ON. Although a healthy diet in itself does not directly reduce pressure on a patient's joints, weight loss (if overweight/obese) will reduce axial loads on the hip joint, which in turn decreases the strain applied to the femoral head/neck (to both the tension and the compression sides).[42]

CONCLUSION

ON is a pathologic and often painful condition involving necrotic areas of tissue that can affect any bony joint in the body. The hip joint is the most common location for ON and should always be properly evaluated, utilizing radiographic screening and MRI scanning, when ON is initially diagnosed in another body part. The earlier a diagnosis of ON is made, the better the opportunity to save the hip joint without surgical intervention or with minimally invasive surgical techniques.

After a diagnosis of ON is made, the size, extent, and location of the lesion and the classification stages are considered to develop an optimal plan of care. The presence or absence of symptoms is important in this process. The goals of treatment involve attempting to preserve the biological hip joint for as long as possible and consideration of a patient's lifestyle and quality of life issues. To date, the 2 main therapeutic options for management of hip ON include joint-preserving procedures and THA. Patient education about potential risk factors and development of ON is essential to prevent the condition and/or to potentially prevent or halt progression of early-stage disease to later-stage disease. ❖

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Connections

Every activity of the living organism is connected with a separate part of the body whence it arises. Therefore, an activity is necessarily damaged when the part which produces it is affected.

— Galen of Pergamon, 130 AD-210 AD, prominent Greek physician, surgeon, and philosopher in the Roman Empire