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# FEMORAL HEAD OSTEONECROSIS, DIAGNOSIS AND MANAGEMENT

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**Abstract:**

*In this review we will discuss epidemiology, pathophysiology and highlight diagnosis methods. We overview non-surgical in early stages and surgical treatment methods. Detailed search was conducted throughout the electronic databases; PubMed, and Embase, for relevant studies discussing Femoral head osteonecrosis. Studies which are published up to the end of 2018 with English language and human subjects were included. Osteonecrosis is a pathology generally seen in younger adults, where collapse of the femoral head and early start of osteoarthritis could eventually necessitate hip arthroplasty when non-operative procedures and joint-sparing treatments fail. Fundamental science study to understand the pathophysiology and to establish treatments that can be translated to clinical application has progressed rapidly, and these developments offer great guarantee for the future therapy of osteonecrosis. Similarly, technological improvements in surgical treatment methods have likewise improved outcomes over the past two decades and will continue to help patients recover from this functionally debilitating joint disease.*

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## INTRODUCTION:

The avascular necrosis (AVN) or osteonecrosis of the femur head (ONFH), an illness with several etiological factors, impacts young populace and otherwise handled prompt, causes the collapse of femur head eventually requiring hip arthroplasty. Early discussion of avascular necrosis of femur head may be painless; nonetheless the supreme presentation hurts restriction of hip motion [1].Passive motions of hip are likewise limited. There is a high opportunity of bilateral discussion. Careful medical history is important to locate any of the danger factors. The Harris hip rating is one of the most usual professional ranges utilized for assessing the hip status.

The antero-posterior radiographs of the impacted hip reveal the principal location of AVN. Nevertheless, since the former and posterior acetabular margins overlap the exceptional part of the femoral head, refined problems in the subchondral area might be missed out on. So good quality side X-rays of the femoral head are essential. A cross table lateral radiograph is less sufficient than a frog leg side to expose the architectural details of the femoral head [2].Technetium 99m diphosphonate imaging (bone scanning) is a beneficial technique for detecting osteonecrosis [3].Several researches have demonstrated that MRI is one of the most precise of all imaging methods [2].Dual line signal on T2-weighted image is practically pathognomic for AVN. Likewise, the solitary thickness line, which is so usually seen laying out the necrotic sore on the T1 weighted photo is believed to be highly certain for AVN. MRI can likewise reveal the re-vascularization front and provides objective proof of tissue adjustments in feedback to treatment allowing sequential evaluation of AVN lesions on follow-up [4] In contrast Computerized tomography (CT) scanning, works only in dividing the late pre-collapse phases of AVN from the very early collapse stage [2].

Ficat defined a four stage (I with IV) category system, which is based on common radiographs [5].In Stage I the radiographs are regular. In Stage II the contour of the femoral head is normal however the radiographs show proof of bone remodeling consisting of cystic and sclerotic regions. Phase III includes flattening of the femoral head. In Stage IV, there is joint space narrowing with second degenerative changes in the acetabulum. Steinberg et alia [6], expanded the Ficat system by dividing Stage III sores right into femoral heads with and without collapse or hips with acetabular participation.

While patients with advanced AVN generally end up with hip arthroplasty, a few of those with very early diagnosis of the lesion (at pre-collapse stage) have been managed with hip salvage surgical treatment. More recent techniques consisting of range of medicines have additionally been made use of for non-operative management of AVN. It is thus considered worthwhile to have a review of healing modalities of AVN femoral head before the lesion reaches the stage when arthroplasty comes to be an unavoidable option.

In this review we will discuss epidemiology, pathophysiology and highlight diagnosis methods. We overview non-surgical in early stages and surgical treatment methods.

## METHODOLOGY:

Detailed search was conducted throughout the electronic databases; PubMed, and Embase, for relevant studies discussing Femoral head osteonecrosis. Studies which are published up to the end of 2018 with English language and human subjects were included. Search strategies used following MeSH terms in searching: “Femoral head osteonecrosis”, “diagnosis”, “management”, “risk factors”.

## DISCUSSION:

- **Epidemiology**

About 5%-18% of all hip arthroplasties are finished on patients with a primary medical diagnosis of osteonecrosis [7].Patients are usually more youthful grownups age 35 years to 45 years, and threat elements for 75%-90% of instances include chronic steroid usage, alcoholism, smoking cigarettes, hip injury consisting of femoral neck fractures and hip dislocations, and prior hip surgical procedure. Various other potential etiologies for osteonecrosis include childhood years background of slipped funding femoral epiphysis (SCFE), deep sea diving or various other hyperbaric conditions, systemic lupus erythematosus (SLE) and various other connective tissue problems, autoimmune illness creating vasculitis, sickle cell anemia, coagulopathy such as thrombophilia or disseminated intravascular coagulation, human immunodeficiency virus (HIV) infection, hyperlipidemia, fat embolus syndrome, treatment of developmental hip dysplasia, chemotherapy and/or radiation, organ transplantation, chronic liver disease, Gaucher illness, gout, and metabolic bone condition [9].Males are affected up to 3 times more than females, and reciprocal femoral head osteonecrosis is discovered in as much as 75% of situations [8].Incidence in the late 1990's was reported to be 10 000 to 20 000 brand-new patients each year, yet this occurrence has probably boosted over the past years [7].

- **Pathophysiology**

The blood supply to the femoral head originates mainly from the basicervical extracapsular articular ring and ascending branch of the medial femoral circumflex artery, along with smaller sized secondary contributions from inferior and superior gluteal arteries and artery of the ligamentum teres [8].The disturbance of this blood supply can be multifactorial, either extravascular or intravascular. Extravascular disruption is commonly attributed to traumatic causes. Proximal femur fractures causing displacement of the neck influence the basicervical arterial ring, whereas hip dislocations can interrupt the ligamentum teres and cause intracapsular hematoma, making the integrity of the extracapsular ring an essential consider the survival of the femoral head. Intravascular embolic matter such as clots, lipids, immune complicateds, or sickle cells can also occlude the terminal arterioles in the subchondral bone of the femoral head [9].

Despite the underlying etiology of osteonecrosis, several research studies suggest a typical pathogenic path involving apoptosis of osteoblasts and osteocytes [10].Adhering to infarction, oxygen- and nutrient-deprived osteocytes and marrow cells pass away unless they can receive blood supply from collateral circulation. As the collateral blood circulation providing the epiphyses is limited, capillary arterialization could not restore adequate blood circulation to the cells. In addition to vascular compromise and programmed cell death, faulty bone fixing is also a key element of osteonecrosis. Adipogenesis has been revealed to be a causal factor in steroid- and alcohol-related osteonecrosis, as it results in compression of venous sinusoids and congestion. The venous congestion enhances intraosseous pressure, protecting against sufficient arterial blood circulation, eventually bring about bone infarction [11].In particular cases, hereditary variables, such as mutations in the COL2A1 gene, have been connected with the pathogenesis of osteonecrosis [12].

Weight bearing during walking generates tons 2 to 3 times body weight on the anterosuperior femoral head articular cartilage and remarkable acetabular dome and 5 to 6 times body weight throughout running or jumping [13] Ischemic disturbance of the weight-bearing surface area in an osteonecrotic hip substantially impacts an individual's capacity to finish pain-free tasks of day-to-day living. Infarcted subchondral bone has trabeculae that come to be thinned by osteoclastic activity, and the hypoxic atmosphere does not enable osteoblastic fixing or remodeling. The location of bone necrosis becomes surrounded by a reactive, sclerotic edge, and the weakened cancellous bone eventually stops working under the recurring lots of weight bearing, bring about subchondral fracture (the "crescent sign" on radiographs). Subchondral collapse at some point brings about articular deterioration.

- **Diagnosis and classification**

Median thigh or groin discomfort with limitation of hip movement in patients less than 50 years of age ought to raise the suspicion of osteonecrosis. Patients generally present with slow-onset, insidious groin discomfort that may be independent or bilateral. Signs are generally magnified with weight bearing and eased with rest. The pain may likewise be in the butts, knees, or anterior and lateral upper leg. Series of movement ends up being restricted, particularly hip abduction and interior rotation, and logrolling (passive internal and exterior turning) generates pain. Early stages of the illness can typically be asymptomatic, and some patients present after articular surface collapse has already happened. Hip prognosis can be substantially boosted with early medical diagnosis, prior to articular collapse [14].

Lab values such as activated partial thromboplastin time (aPTT) and prothrombin time (PT) are generally regular in hip osteonecrosis, although more extensive workup of the etiology might disclose coagulopathy or inflammatory joint illness such as SLE [15]. Plain radiographs ought to consist of anteroposterior and frog-leg side sights of both hips, as the pathology frequently likewise presents in the contralateral hip. Radiographs could show typical findings (Ficat phase I) or subchondral cyst formation and sclerosis (Ficat phase II), however more advanced illness entails femoral head flattening and subchondral collapse, as seen with the "crescent indicator" (Ficat phase III). Osteoarthritic joint space narrowing with osteophyte formation are findings of untreated osteonecrosis (Ficat stage IV) [16] (Table 1). Radiographs are highly specific for advanced osteonecrosis (Ficat II or III) yet not really sensitive for early modifications (Ficat I).

**Table 1.** Osteoarthritic joint space narrowing with osteophyte formation findings of untreated osteonecrosis[16].

| | **Ficat/Arlet** | **Steinberg/U Penn** | **ARCO** |
|---|---|---|---|
| Stage I | Normal radiographs | Normal radiographs | Normal radiographs |
| Stage II | Subchondral cyst formation and sclerosis | Femoral head lucency/sclerosis | Demarcating sclerosis in femoral head, no collapse |
| Stage III | Femoral head flattening, subchondral collapse, "crescent sign" | Subchondral collapse without femoral head flattening, "crescent sign" | Femoral head collapse, "crescent sign", no joint space narrowing |
| IIIA | | | Collapse < 3 mm |
| IIIB | | | Collapse > 3 mm |
| Stage IV | Osteoarthritic joint space narrowing, degenerative changes | Subchondral collapse, femoral head flattening, normal joint space | Osteoarthritic degenerative changes |
| Stage V | | Flattening with joint space narrowing, acetabular changes, or both | |
| Stage VI | | Advanced degenerative changes, secondary osteoarthritis | |

ARCO: Association Research Circulation Osseous.

The advent of magnetic resonance imaging (MRI) and its widespread use generated the Steinberg or University of Pennsylvania osteonecrosis classification system [14], which differentiates subchondral collapse from femoral head articular cartilage collapse (flattening) (Table 1). Phases I via IV are classified by percent of femoral head participation: A < 15%, B 15%-30%, C > 30%. These dimension modifiers are taken into consideration predictors of femoral head collapse. Small lesion size

and more median place are considered prognostically favorable [17].

One more typically utilized classification system that utilizes MRI and various other radiographic techniques is the Association Research Circulation Osseous (ARCO) staging system, which was presented in 1992 and is summarized in Table 1 [18].

MRI has become the imaging modality of choice, as it is very sensitive and specific for osteonecrosis. T1 pictures on MRI generally show a serpiginous "band-like" lesion with low signal strength in the anterosuperior femoral head, and a "double-line sign" can be seen on T2 series, which depicts a high signal strength reparative interface of vascular responsive bone adjacent to necrotic subchondral bone. Radionuclide bone scans are much less sensitive and particular compared to MRI however can be utilized to identify inflammatory task in the femoral head when MRI is contraindicated. CT is additionally much less sensitive than MRI in finding osteonecrosis and has a substantial radiation problem. Angiography and biopsy are intrusive approaches to validate osteonecrosis and consequently are only utilized as research study modalities [17].

- **Nonoperative treatment:**

Nonoperative management of ONFH consists of limited weight-bearing, medicinal agents and biophysical methods of treatment. The objective of medication therapy in the precollapsed phase is to improve hip function, give discomfort relief, protect against radiographic progression to subchondral fracture and collapse, and enable recovery of the necrotic lesions [18].

Restricted weight-bearing using cane, crutches or a walker is effective in early-stages ON hip (Ficat and Arlet Stage-I and II) when the osteonecrotic lesion is <15% and situated far from the weight-bearing dome (medial lesions) [19].Mont et al. [20] evaluated 21 researches (n = 819 patients) based upon limited weight-bearing treatment and observed satisfactory medical outcome (no further surgical treatment) in 22% patients after 34 months. Radiological development was seen in 74% patients. There was no difference in results amongst patients following complete, partial, and nonweight-bearing programs in the research. In a systematic evaluation (degree II evidence), Mont et al. [21] once more reported that 59% (394 of 664 hips )of asymptomatic hips had onset of symptoms or disease progression to collapse after 7 years (array, 0.2-20 years). The detectives reported enhanced threat of collapse in sickle cell illness (73%; 29 of 40 hips) and minimal danger of collapse in systemic lupus erythematous (SLE) (17%; 10 of 59 hips). 32% patients with little or medium-sized lesions (<50% of head involvement) advanced to symptoms or collapse, whereas big lesions had 84% of opportunity of development. It was stressed that development to advanced-stage depends mostly on area, size of the lesion and etiology. Little size lesion could reveal spontaneous regression. In 21st century, this method of treatment could not be accepted as a standard separated modality of treatment and might be an additive therapy to medical or surgical management.

Pulsed electromagnetic therapy is believed to positively impact early-stage ON via stimulation of osteogenesis and angiogenesis much like ESWT. Massari et al., [23] 37 in their retrospective evaluation of 76 hips treated with electromagnetic field stimulation in Ficat Stage-I to III, reported that the 94% of hips in Stage-I and II avoided the requirement for THA with a dramatically higher percentage of hips in Stage-III advancing to THA at a mean followup of 2 years. At present, proof in favor of electromagnetic excitement is restricted and additional study is had to discover its possible duty in early-stage ON.

Hyperbaric oxygen enhances oxygenation, decreases edema by triggering vasoconstriction, and induces angioneogenesis; thus creating a reduction in intra osseous pressure and improvement in microcirculation. Reis et al., [24] observed regular MRI in 13 hips after hyperbaric oxygen treatment (100% oxygen at 2-2.4 atmospheric pressure for 90 min by mask for 100 days) to 12 patients with 16 ONFH, all with Steinberg phase 1 illness. Camporesi et al. [25] also reported clinical enhancement at followup of 7 years in the research study of 19 patients randomized to obtain 30 treatment doses of either hyperbaric oxygen or hyperbaric air for a total duration of 6 weeks. None of the hyperbaric oxygen team patients needed THA till the moment of final followup. As a result of restricted data, using hyperbaric oxygen in ONFH is controversial.

- **Operative treatment**

Surgical treatment for precollapsed stage ONFH involves hip preserving procedures (CD, nonvascularized bone-graft, vascularized bone-graft) whereas prosthetic hip surgical procedure is reserved for advanced-stage of collapse and arthritic hip.

**Core decompression**

Core decompression is the most typically performed surgery for therapy of very early ONFH. It decreases

the intraosseous pressure in the femoral head and increases blood flow to the necrotic area, thus enhancing neobone formation. It has been taken into consideration as the only cost-efficient surgery for ONFH [26], yet the success of the treatment is mainly based on the etiology and radiographic criteria such as lesion size, place or collapse of the lesion [27].The overall success rate as specified by the need for more surgical procedure has varied between 40% and 80% across numerous research studies at 2-7 year followup [26].Conventional core decompression (CD) was carried out utilizing 8-10 mm cannula or trephine which had the prospective threat of subtrochanteric fracture and hip joint penetration.

To augment bony regeneration in the necrotic lesion site, the applications of osteogenic or angiogenic precursor cells with or without development factor is an alluring possibility. Grown-up tissue derived mesenchymal stem cells (MSCs) application represents an extremely appealing alternative for treatment of ONFH in the precollpased stage [28].Many researchers have recorded reduced amount of endothelial progenitor cells and colony creating units in patients suffering from ONFH [29].Besides that, there suffers migratory capacity of endothelial progenitor cells and increased cellular senescence leading to reduced angiogenesis in patients of ONFH [28].All these reasons justify the potential role of stem cells or development factors in treatment of precollapsed ONFH. MSCs implantation has capability to differentiate into numerous cell lineages including the osteoblast, chondrocytes and adipocytes. This property of stem cells has been observed in a speculative dog model while assessing its effect in ON. Nonetheless, the efficacy of stem cells in healing ON lesion is as a result of the osteoblastic distinction capability or additional to release of development factors or cytokines stays vague. In addition, enhancement of neovascularization or angiogenesis property of stem cells have been described by many researchers which ascribes another factor for its possible duty in the therapy of ON.

## CONCLUSION:

Osteonecrosis is a pathology generally seen in younger adults, where collapse of the femoral head and early start of osteoarthritis could eventually necessitate hip arthroplasty when non-operative procedures and joint-sparing treatments fail. Fundamental science study to understand the pathophysiology and to establish treatments that can be translated to clinical application has progressed rapidly, and these developments offer great guarantee for the future therapy of osteonecrosis. Similarly, technological improvements in surgical treatment methods have likewise improved outcomes over the past two decades and will continue to help patients recover from this functionally debilitating joint disease.

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