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Osteonecrosis Of Femoral Head—Overview, Current Management And Treatment Strategies

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Abstract.
Osteonecrosis (ON) is a severe disabling disease that often develops in young people of working age and is associated with the death of bone cells in a certain area of bone tissue, usually caused by a disruption of the local blood supply. ON most often develops in the head of the femur. Factors that can provoke the development of ON of the head of the femur include traumatic injury to the hip joint, surgical interventions on it, dysplasia of the hip joints, immune-inflammatory rheumatic diseases, various coagulopathies, hyperlipidemia, genetic abnormalities, chronic liver diseases, treatment with glucocorticoids (GC), radiation and polychemotherapy, alcohol abuse and a number of others. According to the literature, among the pathogenetic factors associated with COVID-19 and causing bone destruction, there are coagulopathy, endothelial dysfunction and endotheliitis, hyperproduction of cytokines, activation of the complement system, thrombotic microangiopathy, hypoxia and a number of other mechanisms, including the direct damaging effect of the virus on trabecular and cortical bone tissue. The article presents our own clinical observation describing a case of ON development after a new coronavirus infection. Thus, all individuals who have had COVID-19, regardless of the severity of the coronavirus infection and the use or lack of use of GC, should be considered vulnerable to the development of ON and should be included in the risk group for this complication.
Keywords: implants, osteonecrosis, femoral head, avascular necrosis

Introduction.
Osteonecrosis is a progressive disorder in which lack of sufficient blood supply leads to cell death, fracture, and collapse of the affected area. The condition is frequently associated with the femoral head, where progression can be debilitating and can ultimately necessitate total hip arthroplasty (THA). The etiology of osteonecrosis is complex with numerous contributing agents, most markedly trauma, steroid use, and alcohol. Treatment of osteonecrosis is controversial because no option has been overwhelmingly embraced, and little research has compared treatments. Researchers estimate that 20,000 new cases of osteonecrosis are diagnosed in the United States each year.1 The increasing incidence and debilitating progression of osteonecrosis suggest the need for additional investigation of effective and novel treatments, as well as the need for clearer understanding of available treatments. This review characterizes the current knowledge on etiology, pathophysiology, epidemiology, and clinical management of osteonecrosis, with an emphasis on recent developments.

Etiologyof ON.
The exact pathomechanism of ON is often unclear. Each case is probably determined by different factors, including underlying conditions or medication that increase the likelihood of vessel obstruction, alteration of the osteocyte's metabolism, and genetic factors [7].
Most of the blood supply to the femoral head in adolescents and adults comes from the medial and lateral circumflex branches of the profundal femoris artery, which derives from the femoral artery [8]. Obstruction of the subchondral microcirculation, particularly retinacular vessels, leads to bone necrosis. Bone cell necrosis is linked with a high risk of developing secondary osteoarthritis and restrictions in the hip range of motion through an accumulation of microfractures in the osteonecrosis area [2,8].
Non-Traumatic Causes of ON
The most common non-traumatic causes are corticosteroid treatment and alcohol abuse. Corticosteroids alter adipocytes' differentiation, increasing the size and number of adipocytes. This process leads to the

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intracellular accumulation of lipids. As a result of increased pressure inside the bone cells, vascular endothelial cells become damaged, leading to local coagulopathy, vascular thrombosis, and ischemia. A dose-dependent effect was observed in a meta-analysis performed by Mont et al. The incidence of ON was 6.7% with a corticosteroid dose of >2 g prednisone equivalent per day. Every additional 10 mg/day increased the rate of ON by 3.6%. Nonetheless, not all patients receiving corticosteroids develop ON. It is proposed that other factors, including genetic polymorphisms and concomitant diseases, increase individuals' vulnerability [7,10,11]. For instance, in patients treated with corticosteroids due to systemic lupus erythematosus, a higher risk of ON was observed than in patients with other medical diagnoses [6].

Alcohol abuse is reported by 20–30% of the patients with ON. The potential mechanism of induction of ON is unknown. Alcohol may provoke osteocyte death through several pathways, e.g., by increasing intracellular deposition of triglycerides, which leads to pyknosis of osteocytes similarly to corticosteroids, and by decreasing osteogenesis through promoting stromal cell differentiation into adipocytes [12].

Traumatic Factors

Posttraumatic ON occurs when the blood supply to the femoral head is disrupted due to a fracture or dislocation of the femoral head. In most cases, ON is related to fractures in the sub-capital region of the femoral neck. Injury in this region disrupts the anastomosis between the lateral epiphyseal vessels, limiting the blood supply to the femoral head [4,16].

In the systematic review performed by Ghayoumi et al., the overall incidence of avascular necrosis in patients with displaced femoral neck fractures was 17.3% [18]. According to Slobogean et al., displaced fractures were associated with a statistically higher incidence of ON than undisplaced fractures (14.7% vs. 6.4%) [17].

Clinical and Radiographic Examination of the ON

The diagnosis of ON is mainly based on both clinical and radiographic findings. Typical clinical presentation includes increasing pain, stiffness, and crepitus, usually proceeded by a period of minimal symptoms. During the physical examination, patients typically complain of a limited range of motion at the hip and the presence of pain, particularly with a forced internal rotation [4]. Early identification of the disease provides better outcomes. Many imaging techniques were found helpful in detecting bone necrosis signs, including X-ray, magnetic resonance imaging (MRI), computed tomography (CT), and radionuclide examinations. Imaging evaluation of ON should begin with radiography, a non-expensive and widely available technique. Classic radiography may show subchondral radiolucency, called the "crescent sign", indicating subchondral collapse [9]. CT and X-ray are less sensitive than MRI and show the necrotic changes during later stages of ON. Nonetheless, signs of ON are often apparent enough not to warrant additional radiologic evaluation.

MRI is the gold standard for osteonecrosis diagnosis and allows differentiating ON from other diagnoses that may mimic it, such as bone bruises or transitioned osteopenia [1,9]. MRI allows for early ON diagnosis and may help identify patients at risk of femoral head fracture. Identification of bone marrow edema in the proximal femur and joint effusion are critical prognostic factors [3]. T1-weighted images show a limited subchondral linear-shaped low signal intensity, while T2 demonstrates a double-line sign. However, MRI cannot be used after fracture fixation with metallic implants, limiting its utility, especially in patients who develop bone ischemia following surgical procedure [18].

Fan et al. compared single-photon emission computerized tomography and computerized tomography (SPECT/CT) to determine the risk of bone necrosis in patients following femoral neck fracture. The study results revealed that SPECT is most useful for determining the prognosis of ON in patients aged >58 years and with displaced fractures [15]. Diagnostic methods based on nuclear medicine, such as positron emission tomography (PET) or technetium bone scans, may also be used to detect the early stages of ON and help predict the disease progression [1,16].

Management

Conservative treatment of ON aims to improve hip function, prevent the femoral head from collapsing, provide pain relief, and delay necrotic changes [3,48]. Nonoperative management is mainly reserved for the early stages of disease in patients without a history of trauma. Usually, the imaging findings correspond to stages 0 and 1 on the Steinberg scale [1]. Restriction in weight-bearing using a cane, crutches, or a walker is one of the ways to delay disease progression. However, some papers indicate that reducing joint reactive forces does not slow disease progression [49].

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Pharmacological Treatment

Multiple pharmacological agents were proposed as a treatment for ON. These include anticoagulants, statins, vasodilators, bisphosphonates, and other agents currently under investigation [18]. Such treatment is mostly used at the early stages of the disease. Their effectiveness, however, is limited, and there are no clear recommendations for their use in ON due to paucity of evidence. Many patients, after pharmacological treatment, eventually undergo surgery.

1.Bisphosphonates

Bisphosphonates are recommended in the early stages of ON. They act by inhibiting osteoclastic activity and reducing bone turnover, thus preventing woven bone formation [3]. In a randomized controlled trial, the efficacy of alendronate and placebo was compared in patients with non-traumatic AVN at Steinberg stages II–III. Patients in the drug arm experienced 2 collapses out of assessed 29 femoral heads, while 19/25 assessed femoral heads collapsed in the placebo arm [10]. However, another prospective, randomized, placebo-controlled study by Chen et al. did not confirm these findings. There were no significant differences in radiographic outcomes, prevention of THA, and improvement of quality of life between the placebo and treatment arm [11]. The results of available studies are therefore inconclusive. Some of them have limitations in their methodology, including the lack of a control group. The paucity of available evidence does not allow forming guidelines informing the dose and duration of bisphosphonate therapy.

2. Statins

Therapy with statins may inhibit corticosteroid-induced adipogenesis and osteonecrosis of the femoral head. Nonetheless, similarly to bisphosphonate therapy, there are no guidelines on statin use. The results of Ajmal et al. indicate no difference in the occurrence of osteonecrosis between patients on corticosteroids receiving or not receiving statins [22]. On the contrary, Prichett et al. observed a significant reduction in AVN rate in patients on steroids and receiving statins [23].

3. Vasodilators

The beneficial effect of a vasodilator iloprost on radiographic and clinical outcomes in patients with early stages of ON was reported. Claben et al. investigated the effect of iloprost in 108 patients with osteonecrosis; the median follow-up of patients was 49.7 months. Most of the patients (74.8%) noted an improvement in subjective complaints and a decrease evaluated by the visual analog scale. However, patients with a lower stage of disease had better outcomes [14].

Some authors suggest that enoxaparin may delay the progression of osteonecrosis if therapy is implemented in the early stages of the disease [5], but data on its effectiveness remain limited.

4. Other Therapies

Different shockwave devices were studied in ON treatment. Several studies involving extracorporeal shockwave therapy (ESWT) in ON with promising results have been published. The main effect observed was a decrease in pain; some patients had a complete regression of MRI changes. ESWT's proposed mechanism of action is a stimulation of osteoblastic activity, which results in increased density of the bone in the pelvic area. Russo et al. stated that ESWT efficacy is more significant in the early stages of the disease and that ESWT is more effective than core decompression and grafting [27].

Hyperbaric oxygen therapy increases extracellular oxygen concentration and reduces intraosseous hypertension and bone edema. Nonetheless, due to small populations in clinical trials and limitations in their methodology, the efficacy of hyperbaric oxygen therapy has to be confirmed in large randomized controlled trials [28].

Surgical Treatment

Surgical treatment of ON includes joint preserving procedures. These are mostly reserved for young patients in the pre-collapse stage of the disease. THA is indicated for patients with advanced disease.

Core decompression (CD) is the most common procedure performed in the early stages of ON. The principle of this method is to reduce the intraosseous pressure and restore circulation in the femoral head. The procedure is based on drilling holes into the femoral head, which relieves internal pressure and creates space for new blood vessels [29]. CD is recommended as a first-line treatment for patients with early disease. The method is cost-effective and provides excellent results in long-term follow-up [30]. The technique improved over time, and now, the recommended way is multiple drilling.

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Nonvascularized bone grafts derived from different body parts (e.g., tibial autograft, fibular autograft, or allograft) are used to fill the necrotic area in the femoral head. The procedure is most often used at the early stages of the disease after CD fails. There are three main techniques of nonvascularized bone grafting: trapdoor, lightbulb, and Phemister [33]. Many studies have reported excellent outcomes in patients after nonvascularized bone grafting.

Vascularized grafting improves subchondral architecture and also restores circulation in the damaged area of the femoral head. This technique uses part of the fibular bone with a nutrient artery. The graft is inserted into the decompression core with simultaneous anastomosis of the graft to the lateral circumflex femoral artery. This technique is recommended to treat patients with articular collapse <3 mm and <50% involvement of the femoral head [31].

Osteotomies are intended to redistribute the necrotic femoral head tissue away from the load-bearing area and replace it with the portion of the healthy tissue. However, despite protecting the weight-bearing region from collapse, the transferred necrotic area may induce joint instability and arthritic changes [34].

Cellular therapies are mainly based on mesenchymal stem cells (MSCs) derived from bone marrow, adipose tissue, or umbilical cord. MSCs initiate the revascularization process and regenerate bone tissue [32,33]. Li et al. treated 49 hips in the early stage of ON with bone marrow MSCs combined with pravastatin. Results show improvement in hip function and pain. New vasculature was observed in 21 hips after six months from the treatment. While promising results of MSC therapy are observed, more controlled studies should be performed to make recommendations for its use in routine practice [34].

Total hip arthroplasty (THA) should be performed in patients with a significant femoral head collapse, loss of hip function, and severe pain. The procedure involves the removal of the ball and socket of the hip and replacement with an artificial implant. THA is a suboptimal choice for young patients due to activity restriction and possible future revision of the implant [37]. Most patients, however, have good outcomes after THA, particularly pain relief and restoring hip function [35,36].

Conclusions.

ON of the femoral head is a common cause of disability in patients aged between 20 and 40 years. ON may be a complication of surgical treatment of femoral head fractures. ON risk following such procedures is diverse and depends on several factors: the type of internal fixation, type of fracture, Garden classification, preoperative traction, and the time interval between injury and surgery. Untreated ON leads to secondary hip arthritis requiring hip arthroplasty. Early identification of ON offers physicians time to make a relevant treatment decision. Pre-collapse treatment is crucial to obtaining successful outcomes in patients. MRI is the gold standard as it allows for identifying osteonecrosis in its early stages.

Nevertheless, from a practical standpoint, imaging evaluation of ON should begin with traditional radiography because it is a non-expensive and widely available technique. A trial of non-operative management should be performed in patients with early-stage disease, while surgical treatment is routinely used in more advanced stages. Though data on newer therapies are emerging, there is still little evidence to create precise recommendations regarding most treatment methods for patients with ON.

The primary aim of this review was to provide a concise and practical overview of current knowledge of the pathophysiology of ON, as well as clinical aspects, such as diagnosis, staging, and therapeutic options. Selected new ON management methods, which are likely to be included in the routine clinical practice, were also briefly described. As clinicians, we are aware that such comprehensive overviews prepared by practicing specialists, though subject to the flows of narrative review, are of value to other clinicians, as they synthesize the evidence described in multiple systematic reviews and clinical studies, which, by definition, focus on specific populations or types of management. As authors, we hope that this brief overview of clinically essential aspects of ON of the femoral head and its management would help inform practicing specialists. At the same time, we understand that narrative synthesis might not be appealing to, e.g., researchers and academics due to limitations in the methodology of such reviews.

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