



## OSTEONECROSIS OF THE FEMORAL HEAD: ETIOLOGY, PATHOGENESIS, NATURAL HISTORY, AND ARCO STAGING

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### Abstract

Nontraumatic osteonecrosis of the femoral head (ONFH) usually affects young and middle-aged adults and frequently leads to femoral head collapse and subsequent arthritis of the hip. The incidence of the disease is increasing in accordance with the increased use of glucocorticoids for the adjuvant therapy of leukemia and other myelogenous diseases as well as the management of organ transplantation. This review provides up-to-date knowledge on the etiology and pathogenesis, natural history of ONFH, and it also describes Association Research Circulation Osseous (ARCO) classification criteria of glucocorticoids- and alcohol-associated ONFH, and the 2019 ARCO staging system.

**Keywords:** *ONFH; etiology; pathogenesis; ARCO classification*

### INTRODUCTION

Jean Cruveilhier, a French anatomist, first described necrosis of the femoral head after hip trauma around 1830. He presumed that vascular injury was the etiology of the necrosis.<sup>1</sup> In early 20th century, nontraumatic factors were identified to induce necrosis of the femoral head.<sup>2,3</sup> These necrotic lesions were called avascular necrosis, ischemic necrosis, or aseptic necrosis. In 1992, the Committee on Nomenclature and Staging of the Association Research Circulation Osseous (ARCO) decided to use “osteonecrosis” as a uniform terminology.

Nontraumatic osteonecrosis of the femoral head (ONFH) usually affects adults younger than 50 years. The incidence is increasing annually along with the increasing use of glucocorticoids for the management of organ transplantation and the adjuvant

therapy of myelogenous diseases. Thus, this disease became a worldwide socioeconomic burden.<sup>4-6</sup>

This review updated knowledge on the etiology and pathogenesis of ONFH, and it described ARCO classification criteria of glucocorticoids- and alcohol-associated ONFH and the 2019 ARCO staging system.

### ETIOLOGY

In the early and middle 20th century, nontraumatic factors were recognized to cause or to be associated with ONFH. In 1952, bone necrosis was found in divers and this disease entity was known as “Caisson disease” and later as dysbaric osteonecrosis.<sup>7</sup> Osteonecrosis in patients with sickle cell disease was found in the 1960s.<sup>8</sup> Development of ONFH after glucocorticoid use was reported

in 1957.<sup>9</sup> By the 1960s, it was known that those who consumed excessive alcohol had increased incidence of ONFH.<sup>10</sup> Since 1990, thrombophilia, hypofibrinolysis, and hypoangiogenesis were found to be related with ONFH, and familial incidence of ONFH were reported.<sup>10,11</sup>

To date, various polymorphisms and abnormal enzymes were recognized to be associated with ONFH. These genetic factors are implicated in hypercoagulability/hypofibrinolysis and/or hypoangiogenesis. Protein C and Protein S deficiencies,<sup>11-14</sup> mutations in the factor V Leiden or the prothrombin 20210A gene,<sup>15</sup> polymorphisms of the plasminogen activator inhibitor-1 gene (PAI-1),<sup>16,17</sup> presence of antiphospholipid antibodies,<sup>18,19</sup> and decreased activity of 5,10-methylenetetrahydrofolate reductase (MTHFR) have been known to be associated with hypercoagulability/hypofibrinolysis and ONFH.<sup>17</sup> Polymorphism in the endothelial nitric oxide synthase (eNOS) gene and polymorphisms of vascular endothelial growth factor (VEGF) have been known to be associated with defective angiogenesis and ONFH.<sup>20-23</sup>

The use of glucocorticoids, alcohol overuse, smoking, systemic lupus erythematosus, dysbaric disease, pelvic radiation therapy, nonglucocorticoid chemotherapeutics for leukemia and other myelogenous diseases, sickle cell disease, Gaucher's disease, human immunodeficiency virus infection, pancreatitis are known risk factors or conditions associated with ONFH.<sup>24-36</sup>

In 2019, ARCO conducted a Delphi survey to develop a classification scheme of steroid-associated and alcohol-associated ONFH. The ARCO classification criteria of glucocorticoid-associated ONFH included the following: (1) patients should have a history of glucocorticoid use >2 g of prednisolone or its equivalent within a 3-month period; (2) osteonecrosis should be diagnosed within 2 years after glucocorticoid usage, and (3) patients should not have other risk factor(s) besides glucocorticoids.<sup>37</sup> The ARCO classification criteria of alcohol-associated ONFH included the following: (1) patients should have a history of alcohol intake > 400mL/week (320 g/week, any type of alcoholic beverage) of pure ethanol for more than

6 months; (2) ONFH should be diagnosed within 1 year after alcohol intake of this dose; and (3) patients should not have risk factor(s) other than alcohol abuse.<sup>38</sup>

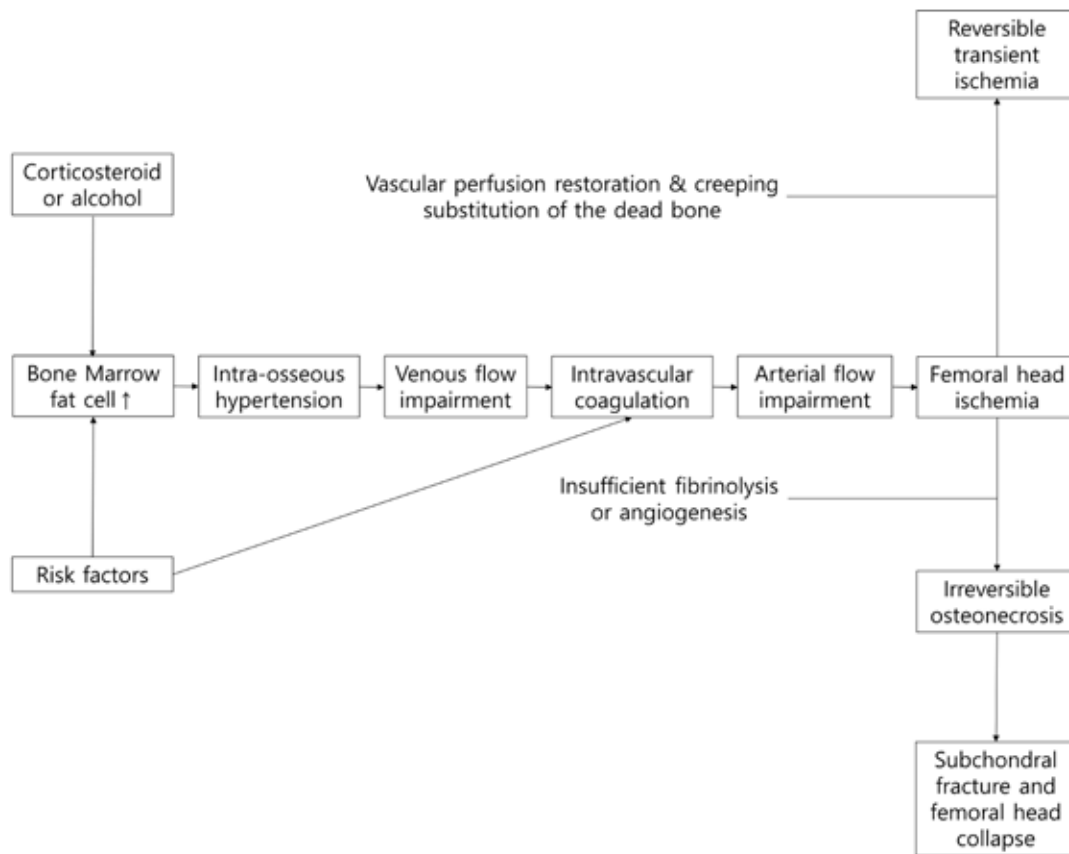
## PATHOGENESIS

The pathogenesis of ONFH is perplexing and continues to be investigated and scrutinized. Nevertheless, there has been considerable progress made in our comprehension concerning the pathogenesis of the nontraumatic ONFH during the last 3 decades. Currently, there are several general consensuses. First, local ischemia due to compromised blood flow is the cause of the disease. Second, alcohol- or glucocorticoid-associated ONFH is not an embolic infarction, even though embolism plays the central role in the development of osteonecrosis in patients with Gaucher's disease, hemoglobinopathies, and dysbaric osteonecrosis. Third, ONFH has a multifactorial etiology including genetic predispositions and exposure to risk factors. In most ONFH patients, these factors reciprocally interact and play roles together in the pathogenesis.<sup>39-41</sup> The genetic predisposition explains why only some of glucocorticoid users and alcohol abusers acquire the disease, while others do not.

The disease is an evolutionary process involving (1) marrow necrosis and death of osteocytes, (2) fibrovascular reparative process around the necrotic zone, and (3) collapse of necrotic bone and subsequent arthritis of the hip.<sup>39-41</sup>

Glucocorticoids and alcohol promote differentiation of mesenchymal stem cells to adipocytes. They also induce hypertrophy of the adipocyte through increasing intracellular lipid synthesis.<sup>42-44</sup>

The increments of number and volume of marrow fat cell induce intraosseous hypertension in the proximal femur. Venous sinusoids are compressed due to intraosseous hypertension, and intravascular coagulation occurs. Then, arterial blood flow is blocked, and an ischemia occurs in the femoral head. Findings of marrow necrosis, death of osteocytes, and encapsulating fibrovascular tissue are the histologic criteria to make a diagnosis of ONFH. Ischemic events do not always progress to definite ONFH. Whether the ischemic lesion progresses or



**Figure 1.** Pathogenesis of nontraumatic osteonecrosis of the femoral head.

not depends on the restoration of vascular perfusion and the creeping substitution of dead bone by new bone. Genetic predispositions of hypercoagulability/hypo-fibrinolysis and/or hypo-angiogenesis play a role in the restoration of vascular perfusion.<sup>39-41</sup>

In 2019, the ARCO task force developed a plausible model to explain the pathogenesis of nontraumatic ONFH (Figure 1).<sup>39</sup>

#### NATURAL HISTORY ACCORDING TO SIZE/ LOCATION OF NECROSIS

The size/location of necrosis predicts further collapse of the femoral head and is the major determinant in the treatment of ONFH. Small lesions seldom collapse even without any medical or surgical treatment, while most of the large lesions are progressive.<sup>45-47</sup> Once ONFH develops, the lesion size does not change with time.<sup>48</sup> Thus, the size of necrotic portion should be measured prior to treating

an ONFH patient, and any treatment should not be done on small lesions. The efficacy of a certain treatment should be determined cautiously considering the different natural courses according to the size of the necrosis.

Various classification systems have been developed to characterize the size/location of the necrosis. Currently, three classification systems, Steinberg classification, Japanese Investigation Committee (JIC) classification, and modified Kerboul classification, are widely used.<sup>45-47</sup>

The Steinberg system categorized the extent of necrosis into three subsets: mild (<15 % of articular surface or head affected), moderate (15–30 %), and severe (>30 %).<sup>46</sup>

In the JIC classification, necrotic lesions were classified into four types: type A lesion < medial 1/3 of the weight-bearing portion; type B lesion < medial 2/3 of the weight-bearing portion;

type C1 lesion > medial 2/3 of the weight-bearing portion but not extending laterally to the acetabular edge; and type C2 lesion extending laterally to the acetabular edge.<sup>47</sup> *The advantage of the JIC classification is its simplicity because only mid-coronal Magnetic Resonance images (MRI) are required. However, accurately dividing the acetabular dome into three zones could lead to low reproducibility.*

In 1974, Kerboul et al. developed a method to quantify the extent of necrosis by measuring the arc of the necrotic portion on anteroposterior and lateral hip radiographs and then calculating the sum of these two angles.<sup>49</sup> In 2006, Ha et al. modified the method by measuring the necrotic arc on the mid-coronal and midsagittal MR images. The authors classified the necrotic lesions into three categories: small lesion (combined necrotic angle <190°), medium-sized lesion (combined necrotic angle between 190° and 240°), and large lesion (combined necrotic angle >240°).<sup>45</sup> *Modified Kerboul method could represent the size of necrotic lesions relatively precisely, but it requires two MR images and calculations.*

However, there is no agreement as to which method is most reliable and valid. Unified classification system of necrotic size/location should be developed and optimal cutoff values on disease progression should be obtained soon.

## STAGING

The first ARCO staging system of ONFH was established in 1994. However, it was known that stage 0 lesion, marrow necrosis with viable osteocytes, does not progress to definite osteonecrosis.<sup>40</sup> Thus, ARCO revised the staging system in 2019. In the 2019 version, stage 0 was deleted; instead, stage III was divided into early (III A) and late stage (III B) according to the depth (2 mm) of head depression, and the subclassification of location/size was not incorporated (Table 1).<sup>50</sup> *Evolution of ONFH staging regarding the prognosis and treatment should be further studied with long-term follow-up and larger cohorts.*

## SUMMARY

Both genetic predispositions and exposure to risk factors are involved in the pathogenesis of ONFH. The size/location of necrosis is the major determinant of the disease progression. The size of the necrotic portion is determined at the initial ischemic attack, and it does not change with time. Small lesions do not progress and thus need no treatment. Any medical or surgical treatment should be done on painful hips with medium-sized to large lesions. ARCO recommends the use of 2019 revised staging system and the classification criteria of glucocorticoid- and alcohol-associated osteonecrosis.

## AUTHOR CONTRIBUTIONS

KHK was involved with the conception and design of the study; YKL provided administrative support; KHK and YKL provided study materials or patients; JWP collected and assembled data; and HSK was concerned with data analysis and interpretation.

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**Table 1.** The 2019 Revised ARCO Staging for Osteonecrosis of the Femoral Head

ARCO Stage	Image Findings
I	X-ray: normal MRI: low-signal band on T1-weighted MRI
II	X-ray: abnormal MRI: abnormal
III IIIA (early) IIIB (late)	Subchondral fracture on X-ray or CT Femoral head depression < 2 mm Femoral head depression > 2 mm
IV	X-ray: osteoarthritis

ARCO: Association Research Circulation Osseous; MRI: magnetic resonance imaging; CT: computed tomography.

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