Chapter 2

Osteonecrosis and Hip Development Disorder

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http://dx.doi.org/10.5772/intechopen.68161

Abstract

Blood vessel branching of the proximal femur by its scheme differs from all other major joints. This scheme changes during the individual's development, dynamically depending on age. Namely, the caliber, blood flow rate, and dominance of certain arteries from the entire network of blood vessels that participate in the vascular supply of the hip are not equally expressed in all stages of development. In each successive stage, blood supply is dominated by a different artery that, after a certain period of time, shifts its major role to another artery. Anastomoses between individual arteries are not constant in all stages of development, and they represent a great importance for compensatory mechanisms. The disturbance of local arterial blood vessels, at a time when they dominate the blood supply and affect the quality of hip development and maturation, leads to reduced perfusion, and consequently, to the lack of development, ossification, and possible osteonecrosis.

Keywords: osteonecrosis, hip developmental disorder, bone, hip vascular supply

1. Introduction

Blood vessel branching of the proximal femur by its scheme differs from all other major joints. This scheme changes during the individual's development, dynamically depending on age. Namely, the caliber, blood flow rate, and dominance of certain arteries from the entire network of blood vessels that participate in hip vascular supply are not equally expressed in all stages of development. In each successive stage, blood supply is dominated by a different artery that, after a certain period of time, shifts its major role to another artery. Anastomoses between individual arteries are not constant in all stages of development, and they represent

a great importance for compensatory mechanisms. The disturbance of local arterial blood vessels, at a time when they dominate the blood supply and affect the quality of hip development and maturation, leads to reduced perfusion, and consequently, to the lack of development, ossification, and possible osteonecrosis.

The problem of the circulation and vascular network supplying bones is exposed as a main problem in the etiology and pathogenesis of hip disorders in children which can affect later stages of life. The authentication of the problem is quite hard since bone biology is inappropriate for the assessment of vascular status. Results and first signs of diseases are visible after a couple of months or even longer.

Acute or chronic ischemia in other tissues has a specific clinical presentation and symptomatology, which is not the case with the hip joint. Also, techniques that are applicable for vascular status assessment in other tissues are almost useless for the estimation of the vascular status on bone tissue.

2. Bone vascular system

As in other tissues, vascular system of the bones can be divided into three levels: afferent and efferent blood vessel and a microvascular network that fills in the space between them (**Figure 1**). Afferent blood vessel is composed of epiphyseal, metaphyseal, nutrient, and periosteal arteries. Microvascular network consists of medullar sinusoid, cortical, and periosteal capillaries, while the efferent blood vessel is formed of epiphyseal, metaphyseal, nutrient and periosteal veins, and collective sinusoids. None of the mentioned vascular elements can be considered independent. There are numerous anastomoses between them and opinions about one's participation in bone nutrition are still divided.

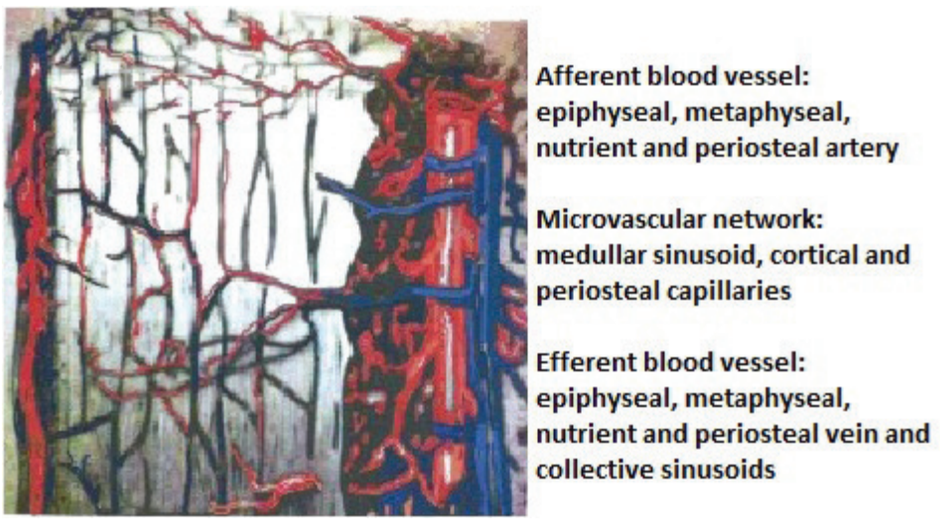

Figure 1. Bone vascularization.

http://dx.doi.org/10.5772/intechopen.68161

The nutrient artery is the main artery supplying the cortex of the bone. Its branches separate under a sharp angle and do not divide further. In medullar cavity, the nutrient artery is divided into ascendant and descendant medullar arteries. Branches separate radially out of these supply cortices. In normal bone structure, there is a centrifugal flow meaning that blood vessel interchange is parallel to Haver's channels and bone axis. These blood vessels drain into venules on the periosteal bone surface. This type of vascular organization has functional meaning allowing relatively high intramedullary pressure that can affect movements of the interstitial fluid in bones [1].

3. Specificity of hip vascular supply

Blood supply of the hip is specific. It is formed of rich vascular network, that is, in great deal, responsible for the development of the joint. Blood vessels that participate in the hip vascularization can be divided into nutritive, retinacular, and foveolar. Retinacular blood vessels are the main source of hip supply and are extremely vulnerable (**Figures 2** and **3**). They can be damaged by infection, joint fluid, trauma, or forced position of the joint. Blood supply of the hip changes and adjusts during joint stages of development, from embryonal to the adolescent stage, when it adopts definite appearance and functional characteristics. Stages of vascular supply of the hip consist of fetal, infantile, intermediary, prepuberty, adolescent, and adult stage [2]. Each stage is different by the role of main nutritive artery of the diaphysis, metaphysis, periosteal, and anastomoses. The only constant factor in all of the mentioned stages is that the supplying blood vessels come from the a. femoralis communis, a. profundae femoris, and branched of the a. iliacae internae—a. gluteae and a. obturatoriae. The most important artery for hip supply is a. profunda femoris with its branches—a. circumflexa femoris medialis and lateralis.

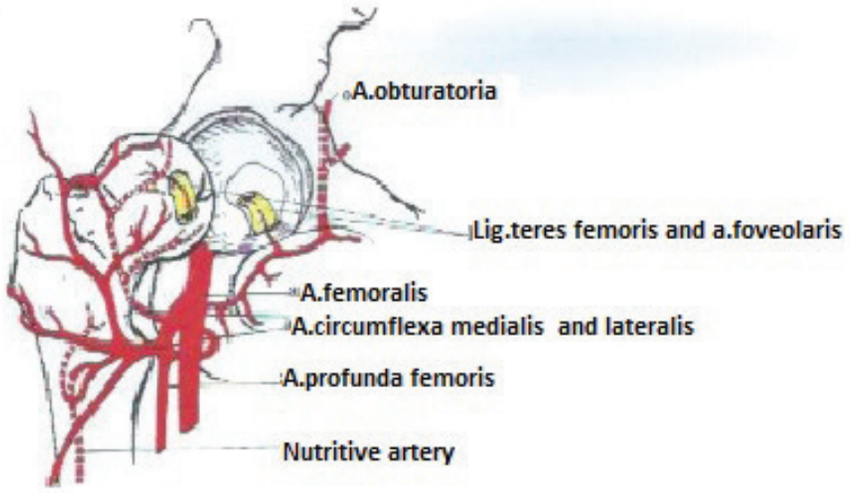

Figure 2. Scheme of arteries of the coxofemoral region.

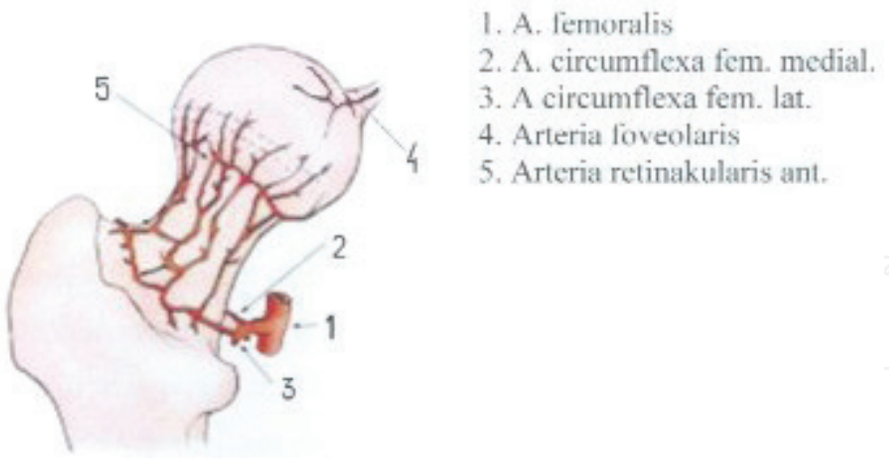

Figure 3. Scheme of arterial blood supply of the femoral head and neck (anterior view).

Final branches of the a. circumflexae femoris medialis and lateralis form anastomoses that together with a. obturatoria and a. glutealis make circulus arteriosus—arterial ring around the femoral neck (**Figure 4**). This ring can be found pericapsullary as well as intracapsullary. From extracapsular arterial ring, retinacular arteries are formed that diverge in regular interval and pass through capsule forming ascendant cervical arteries. These arteries, by their position, can be divided into anterior, posterior, medial, and lateral, and their function is to supply femoral head and neck. Extracapsular ring forms medial metaphyseal and lateral epiphyseal arteries that supply blood to epiphyseal gap. These nutritional arteries pass through the capsule in the lower lateral part of the femoral neck parallel with intertrochanteric line where arterial circulation can be jeopardized leading to avascular necrosis (AVN) of the femoral head.

The intracapsular arterial ring is positioned subsynovially around the distal part of the femoral neck. This ring forms vascular borderline of the joint (circulus articuli vasculorum

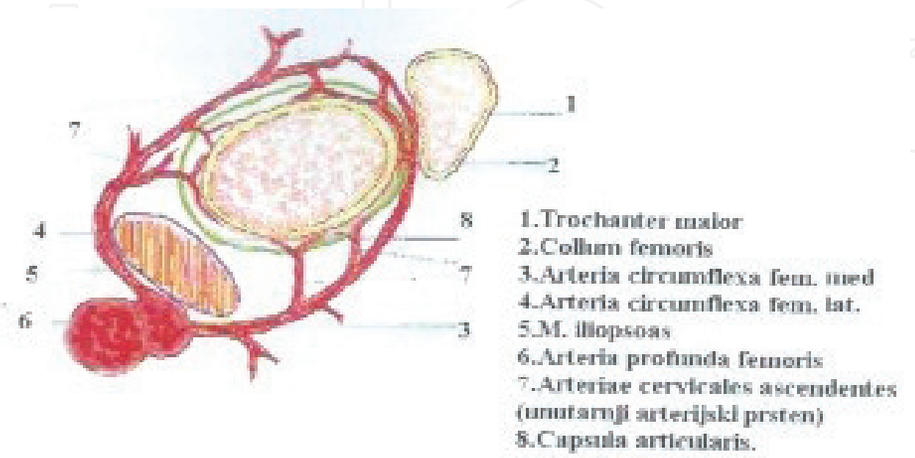

Figure 4. Scheme of extracapsular and intracapsular arterial ring.

Hunteri). Out of this ring, short arteries depart to the bone metaphysis or articular cartilage of the femoral head in which ossification nuclei end. Every artery supplies only one ossification nucleus. In the case where there are multiple secondary ossification nuclei, every one of them is supplied by separate artery that anastomoses in between which can explain partial necrosis of the femoral head [3].

4. Dynamics of development of hip blood supply

Dynamics of hip development blood supply are intensified by movements in the joint through stages of development of a person. The entire dynamic process can be divided into five phases:

(a) *Fetal phase* includes the entire intrauterine period and first 4 months of life. A. circumflexa femoris medialis and lateralis together with a. obturatoria give outer epiphyseal, ascendant, and lower metaphyseal arteries. In this stage morphologically and functionally a. circumflexa femoris lateralis is more developed. Outer epiphyseal arteries are directed horizontally and by the inner part of the femoral head. Lower and ascendant metaphyseal arteries have vertical flow. Their branches are directed toward the inner side of one part of the femoral head, while foveolar arteries pass through ligament capitis femoris supplying the inner part of the femoral head.

(b) *Infantile phase* is considered from 4 months of life to 4 years of age. The most intensive growth and development of the collateral blood vessels that form arterial ring around the femoral neck occur between the sixth and the 18th month of life. This stage coincides with the walking phase that is considered the most intensive period for the establishment of physiological motions in the hip joint. In this phase, epiphyseal arteries get stronger, getting their supply from a. circumflexa femoris medialis that takes over the dominant role for blood supply. There are no arteries that pass through ligament capitis femoris, and anastomoses that amplify supply through arterial ring around the femoral neck are poorly developed.

(c) *Intermediary phase* is considered from 4 to 7 years of age. In this phase, epiphyseal vessels are constant with significant caliber, while metaphyseal vessels fall behind in development. The characteristic of this phase is the strengthening of the arteries of ligament capitis femoris. This phase includes the development of the arterial ring around the femoral neck.

(d) *Prepuberty phase* includes period between the eighth and the 10th years of life where definite development is dominating with stabilization of all, already, formed anastomoses.

(e) *Adolescent phase* is considered on a period between the 11th and the 15th years of life when, regarding vascular system, metaphyseal blood vessel network is established and when growth of the proximal femur is ended. In this stage, outer epiphyseal arteries and a. capitis femoris are leading the blood supply and are basis for arterial network of the femoral head and neck in adults [4–6].

5. Avascular necrosis as a consequence of treatment of developmental hip dysplasia (DDH)

5.1. AVN: complication or inevitable consequence of the treatment of DDH?

AVN in different degrees is a well-known complication of the treatment of DDH. It can be a devastating complication with possible premature development of osteoarthrosis. In a milder degree, it can be manifested through minimal residual deformity and hip dysfunction.

5.2. Definition of AVN

It is quite hard to define AVN. Weinstein, instead of the term AVN, used a term "growth disorder of the proximal femur" [7]. However, when milder degrees of AVN are analyzed, this term is not suitable because there is no actual growth disorder. This is the most serious complication connected with the treatment of hip development disorder. Its sequelae are deformity of the femoral head, permanent acetabular dysplasia with chronic lateral subluxation, relative hypotrophy of the great trochanter, and abbreviation of the lower extremities.

In fact, AVN represents complication of bone blood supply in terms of inadequate perfusion and oxygenation; it is extremely rare in healthy children, while it is quite often in children with diagnosed hip development disorder. AVN is characterized, on a microscopic level, by zones of devitalized bone trabecula and bone marrow that have tendency to spread and affect the subchondral plate. From the entire proximal femur, its head is the most vulnerable spot for the development of AVN. The most common position of the development of AVN is just below the joint cartilage that was affected by ischemia the most or suffers the greatest pressure which is the anterolateral aspect of the femoral head. However, there is no specific part of the femoral head that is spared of the affect of AVN. In adult patients, where in childhood had diagnosed hip development disorder and noted AVN, the affected segment was never totally revascularized. Once detected, collapse of the femoral head usually persists [8–10].

The beginning of disease is inconvincible and practically asymptomatic. AVN usually progresses until complete destruction of the joint that, even before the fifth decade of life, demands radical surgery—hip joint replacement. It is estimated that 10% of 500,000 hip replacement surgeries performed in the USA per year have indication of AVN.

5.3. Pathophysiology of the AVN in DDH

Pathological changes that can lead to the development of the AVN have their beginning in two different categories: vascular and extra vascular factors. Vascular factors can be divided into arterial and vein factors.

5.3.1. Vascular factors

All vascular factors can be divided into arterial and vein factors. Almost all vascular factors are located extraosseally. Arterial factors are, probably, the dominant and most important

factors for the development of the AVN. The femoral head is, because of the arteries that are marked as arteries of the terminal type and their relatively bad developed collateral network, a part of the body that is most liable to development of the AVN. Some kind of trauma that hip suffers during the surgery, in order to achieve optimal relations in coxofemoral joint, can lead toward thrombosis with contusion or even complete interruption of lateral retinacular arteries that in the critical period are basic for blood supply of the femoral head and neck. Compared to other tissues, such incident can be marked as only infarction that covers the irrigational spot of the affected blood vessel. In children with hip development disorder, a less valuable hip is noted and in most cases with weaker developed blood vessels. During treatment of this kind of a hip, possible attenuation of vascular network can be seen. In older patients that have been diagnosed with DDH, atherosclerotic changes can be presented on magisterial as well as on terminal arteries of this region. In middle-aged female patients, vasculitis and Raynaud's disease can lead toward exacerbation of the AVN, while in male patients, AVN is expressed after decompression or because of vasospasm.

Those rare vascular factors that act intraosseously are related to vein blood flow and are less often caused by intraosseal arterial events. Primer etiological factor for the development of the AVN in this group is micro-embolism. Micro-embolism in one irrigational field blocks circulation in one part of the femoral head. This condition can be seen in cases of fat embolism, air embolism, or thrombosis, while steroid therapy is administered. Intraosseal vascular factors refer to all diseases that reduce vein blood outflow causing vein stasis. All types of vein stasis, because of increase of pressure, can indirectly lead to AVN. Some metabolic or hormonal disruption leads to the enlargement of intramedullary fat-loading osteocytes. This directly influences shift in space and reduction of vein capacity resulting in difficulties with vein drainage. Intraosseal phlebography that was performed in patients with AVN showed abnormalities in drainage system emphasizing the fact that vein circulation participated in development and contributed in the progression of this disease. Capability of decompression in space occupied with bone marrow depends on regional anatomical structures especially of vascular outflow and bone architecture. The femoral head, unfortunately, does not have anatomical advantages as other bones because of its formation as a socket on a narrow metaphyseal neck. Only a couple of vein channels go through bone cortex and have the capability of direct decompression. Increased pressure during disproportion in inflow and outflow has to be directed toward narrow metaphyseal neck [11–14].

5.3.2. Extravascular factors

Intraosseal factors are basic factors that influence the development of AVN from this group. Skeletal system in subchondral zone of the femoral head is a closed rigid cortical socket. This system is conditionally sensitive on pressure increase that can result in somewhat like a compartment syndrome. Many authors have described the increase of the pressure in bone marrow of the proximal femur in many patients diagnosed with AVN. First effects of pressure increase can be resulted in vein outflow, sinusoids, and small capillaries. Reflex spasm can even block nutritive arteries before they reach cortex. The subchondral zone of the femoral head that is in most intimate contact with acetabulum is not in a favorable position because

of the mechanical relations. Pressure and friction on one side together with weaker blood supply create a baffle effect that can contribute to further restriction of the decompression of bone marrow in the affected region opposite of subchondral zones with regular perfusion. Trabecular deformity, which can occur because of this kind of development also compresses medullar space and leads to an increase in the intraosseal pressure. This kind of situation can contribute to morphological changes, reduction of trabecula, thinning of bone matrix, and disturbance in dynamics of ossification. An increase of the bone pressure strives to concentrate in the affected zone because of specificity of its architecture. This process, together with an increase of the intraosseal pressure, tends to transform ischemic zone that was marginally affected into the zone of complete bone infarction with functional anoxia [15].

5.4. Pathophysiology of the AVN

Macroscopic changes of the AVN on the femoral head show a thin layer of compact subchondral bone and joint cartilage. Joint cartilage can be supplied with synovial fluid. The only part of cartilage that can lead to necrosis is around the zone of demarcation. Bone part of the femoral head can lead to necrosis in the form of irregular areas of yellowish necrosis, while only some of trabeculae seem vital. In progression of the process, spot-like zones of necrosis start to resorb which can be seen on X-rays. In further development, micro-fractures can be noticed and consequently bone sequesters start to form. The line of the trabecular fracture goes through necrotic part of the bone-causing formation of joint sequester. Because of this, the affected region of the femoral head collapses. After this, progressive destruction of joint cartilage is noted with formation of free joint bodies (corpora libera) and marginal osteophytes.

From pathophysiological aspect, AVN can be divided into phase of cellular necrosis and phase of reparation.

Cellular necrosis first affects hematopoietic elements (6–12 h after insult), after which necrosis of bone cells follows: osteocytes, osteoblasts, and osteoclasts (12–48 h after insult), followed by necrosis of fat cells of bone marrow (2–5 days after insult). Complete absence of osteocytes in localized areas of trabecular bone is real indicator of the existence of AVN. This is a typical finding for AVN that lasts 14 days. Phase of death of fat cells of bone marrow can be recognized by vacuoles inside a cell that do not have a nucleus, thus looking alike to lipoid cysts. Bone infarction can be recognized into four zones: central zone of cellular necrosis surrounded by concentrically circles of ischemia, hyperemia, and zone of normal tissue. Once AVN is noticed, products of necrotic tissue cause initial inflammatory response that is manifested with vasodilatation, fluid transudation, fibrin precipitation, and local leukocytes infiltration. This response is the basis for the development of hyperemia and the basis for the start of reparatory process—reconstruction of infarct zone. In reparatory phase, bone resorption is noted first that is followed by a neoosification process. The reparatory phase does not happen inside of the necrotic and ischemic zone, but between vital and infracted zone, because it demands adequate perfusion. Reparatory response results in progressive growth of reactive border that demarks that part of the bone tissue that is doomed to failure. Mesenchyme cells and capillary proliferation supply macrophages and fibroblasts entrance into "dead zone," which starts the reparatory phase that is presented as osteoporosis. Progressive loss of mechanical support

and destruction of bone architecture are trying to be replaced by the activity of osteoblasts which is usually not enough because this process is followed by micro-fractures on places with least resistance. Fractures, fragmentations, and other disorders on the subchondral bone are visible signs of this process on normal X-ray. Capillary invasion extends to the subchondral part of the bone tissue and resorbs cartilage. This phase used to hold conviction in many orthopedic surgeons for osteochondritis dissecans [16–19].

5.5. Consequences of the AVN

Consequences of AVN can be divided into minimal and manifest changes [19]. If irrigational zone of occluded blood vessel is small and does not cover large amount of space with supply, patients can have minimal changes with insignificant or even no symptoms at all. These changes get detected rarely or accidentally through some diagnostic procedure for some other disease. Because of this, many patients do not get included into studies about AVN and its consequences. On the other side, manifest changes become visible when multiple occlusions on blood vessels are formed. These changes on specific bone regions become visible on X-rays after certain period of time. Signs of bone change are noticeable in the form of necrosis that subjects to slow but limited reparatory process. In the background, reactive zone of reparation is visible that forms sclerotic edge of trabecular thickening. These reparatory attempts can be followed and noticeable through different stages as well as different forms of necrosis and remodeling.

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