International Journal of Pediatrics and Genetics
Vol. 3 No. 6 (2025)
ISSN: 2995-5483

To Evaluate the Effectiveness of The Proposed Method of Surgical Treatment of Patients With Early Stages of Aseptic Necrosis of the Femoral Head from the Standpoint of Evidence-Based Medicine

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Abstract: The relevance of the topic. One of the urgent problems of modern orthopedics is the development of effective treatment methods for patients with aseptic necrosis of the femoral head. This is due both to the high incidence of the disease (from 1.2 to 4.7%) among all destructive dystrophic pathology of the hip joint and to the involvement of young people of working age. The high socio-economic importance of the problem is given by the fact of a significant frequency of early disability in patients of this category and the need for radical surgical interventions (Zulkarneev R.A. and co-authors, 2010; Yakupov R.R. and co-authors, 2016; Zhang Y., 2014). The use of modern diagnostic methods, such as computed tomography, magnetic resonance imaging, and scintigraphy, makes it possible to detect the onset of a pathological process in the femoral head at an early stage, when radiological verification is technically impossible. This, in turn, makes it possible to carry out early and effective surgical treatment (Nazarov E.A. 2013; Ildar F.A., 2014; Bryukhanov A.V., 2014; Venugopal V., Prabhu A., 2014).

Key words: conduct, analytical, necrosis, femoral.

Femoral head cancer is of great socio-economic importance in all countries of the world: for example, in the United States, up to 18 percent of all total arthroplasty (TETS) operations are performed for this pathology annually, and in Asian regions the situation is even more serious - in Taiwan, Korea, Hong Kong, and Japan. There are more than forty TETS operations due to AVN. Aseptic necrosis of the femoral head (ANGBC) is a disabling clinical disease characterized by the death of osteocytes and bone marrow, followed by resorption of necrotized tissues, which leads to progressive destruction of bone architecture, subchondral fracture and collapse of the femoral head, and, finally, loss of congruence in the joint with loss of its basic functions. Without treatment, more than 70% of femoral heads in osteonecrosis (OH) are destroyed within 3-4 years after diagnosis according to X-ray diagnostics, and therefore endoprosthesis surgery is required, which is also painful for many patients due to severe joint pain. In addition, according to a number of authors, asymptomatic avascular necrosis (AVN) can develop up to 5 years in 94% of patients, the manifestation of clinical symptoms in such patients already corresponds to a high stage of the disease. There is no up-to-date epidemiological data on ANGBC in Russia. It is known that IT affects men more often in middle age, and women more often after the age of 50, and at the age of 20-40, men are about 7-8 times more likely than women to get sick; necrotic changes in more than half of those affected affect both TBS. The problem of developing new treatment methods for ANGBI is especially relevant for young patients who lead an active lifestyle. Without timely and effective treatment, coxarthrosis often progresses, leading to unbearable pain and immobility. Thus, early intervention, taking into account the stage and mechanism of pathology development, is key to the success of organ-sparing procedures for non-surgical treatment of ANGBI. It is especially important to create comprehensive treatment methods, including drugs and

methods that affect all parts of the pathological process, relieve pain, stimulate regenerative processes in the head of the TBS and, accordingly, avoid or delay the operation of endoprosthetics.

Clinical diagnosis of ANGBC is extremely difficult, since, with rare exceptions, stages I and II of osteonecrosis do not have clear clinical manifestations. Most often, when diagnosing ANGBC, patients undergo surgical treatment: intertrochanteric detorsion-varicose osteotomy, biostimulation of the femoral neck with cortical auto- and allografts, total endoprosthetics. This approach to the treatment of ANGBI is justified and is due to the fact that the diagnosis is usually made at the stage of necrosis, when the osteonecrosis zone is crushed, accompanied by fractures of the subchondral bone plate with detachment of articular cartilage from the osteonecrosis zone. It is in this situation that the patient experiences persistent pain, which is not relieved by the action of analgesics and anti-inflammatory drugs. Accurate diagnosis of the first stage of ANGBC is possible, as a rule, only with the help of MRI. At stages I and II of aseptic necrosis, organ-sparing surgical treatment is also performed, which is more gentle than endoprosthetics: fenestrating osteotomy of the proximal femur revascularization of the femoral head with an autograft from the large trochanter on the muscular pedicle decompression of the necrosis site and osteosupply with osteosupply materials. A method of combined impact autoplasty of the femoral head, including decompression, intraosseous resection and excision of the lesion Osteonecrosis followed by combined autoplasty of the postresection defect. Reversible stages of aseptic necrosis, which can be treated non-surgically, are characterized by edema of bone tissue or the presence of superficial foci of subchondral necrosis of a certain localization in the absence of bone destruction and while maintaining the shape of the femoral head. Traditionally used conservative therapy for patients with stages I and II of ANGBI is unloading of the affected joint, physiotherapy (electrophoresis, acupuncture, laser therapy, shock wave therapy), complex techniques combining drugs that improve blood circulation and affect osteogenesis. An interesting method of treating patients with ANGBC is performed with prolonged epidural analgesia by injecting a local anesthetic through an epidural catheter at a dosed rate for 6-8 days. However, according to many experts, conservative treatment of ANGBI is not effective enough and provides only short-term improvement; it is believed that the pathological process is not stopped due to the use of ineffective methods, due to the use of drugs with low or unproven efficacy.

Currently, the development of new methods for the treatment of pathologies of the musculoskeletal system should be based on an understanding of the molecular and cellular mechanisms for correcting bone and cartilage metabolism and innovative drug delivery methods that allow targeted delivery of the drug mixture to the area of destructive changes in cartilage and bone tissue.; It is based on a combination of pharmacological and physiotherapeutic approaches that provide a multidirectional effect on the destruction zone, relieve pain and inflammation, have an anti-ischemic effect and stimulate regenerative processes. Based on the above, the treatment of patients with stages I and II of ANGBI, the problem to which this study is devoted, is not only medical, but also socially significant, since progressive osteonecrosis inevitably leads to joint destruction, the search for ways of non-surgical treatment of patients with ANGBI providing a reliable therapeutic effect is relevant. ON TBS is one of the most frequent localities of death of the subchondral femoral head bone, followed by sequestration of the joint head tissue, its collapse and the development of deep destructive processes. In the latest ICD-10, IT is included in the classification with the allocation of idiopathic aseptic bone necrosis and secondary drug related injury (post-traumatic) and other causes. ANGBC, avascular or ischemic necrosis (ICD-10), is a serious pathology of TBS. In this regard, we focus on the main vessels that provide blood supply to the TBS. The blood supply to GBC is mainly due to A. circumflexa femora medialis, which in the area of Fossa trochantarica gives rise to three or four branches, the so-called rr. retinaculares (capsule vessels). They pass dorsocranially along the neck in the synovial layer until they reach the border of the cartilage of the head, where they enter the bone tissue and supply blood to the head. Branches inside the lig. teres belong to A. obturatoria. As a rule, they supply blood to only a

small part of the bone tissue near the lig attachment. teres. The large trochanter is supplied with blood by the ascending branch of the circumflexa femoris lateralis. It anastomoses cranially in the femoral neck area with branches of A. circumflexa femoris medialis. Additional blood supply to the femoral head occurs due to the intraosseous vessels extending from the metaphysis in the cranial direction. With a fracture of the femoral neck, these vessels are, of course, always damaged. The factors influencing the progression of ANGBC, from the appearance of trabecular edema, superficial necrotic lesions to an impression fracture of the subchondral bone with the formation of a zone of collapse and disintegration of the femoral head, are not yet fully understood. There is little epidemiological data on ANGBN. In the United States, 10,000 to 20,000 new cases of ANGBC are diagnosed annually, which, according to various authors, accounts for 10 to 15% of all pathological hip disorders (TBS). It is for this reason that an estimated 5 to 18% of the more than 500,000 total hip replacement (TETS) surgeries are performed annually in the United States. According to the work, 5-7.5 million people in the world are affected by non-traumatic ANGBC. Risk factors such as excessive alcohol consumption and systematic use of corticosteroids have been clearly identified. At the same time, the percentage of patients with excessive alcohol intake is comparable for different regions, although, as follows from Table 1, the number of such patients tends to increase in Russia. In Japan, the highest percentage (60%) of ANGBC responds to patients with systematic use of corticosteroids. The disease progresses rapidly in most young patients, leading to collapse of the femoral head and destruction of the hip joint (TBS). The progression of ANGBC to the final stage, according to the work of Roiua B. is associated with five main mechanisms: hypercoagulation, suppression of angiogenesis, hyperadipogenesis, hereditary predisposition, and excess bone resorption in bone remodeling. Bone cell necrosis is more often the end result of several pathogenic mechanisms acting individually or synergistically. They are the result of ischemia, direct toxic effects on cells and changes in the differentiation of mesenchymal stem cells. Ischemia may be the result of vascular disruption, compression, narrowing, or intravascular occlusion. Destruction of the vascular network around the femoral head is observed in 15-50% of cases of femoral neck fractures and 10-25% of cases of hip dislocation, which usually leads to the development of traumatic osteonecrosis.

Systematic minor microtrauma and overloading of the hip joint (usually in athletes) are also a potential cause of the development of traumatic ANGBC. An analysis of the medical literature on ANGBI indicates that one of the main versions of the mechanism of development of this degenerative process in large joints is considered to be a circulatory disorder that leads to ischemia and subsequent changes in bone structure. As follows from scheme 1, vasoconstriction may be the result of intraosseous hypertension secondary to fatty infiltration of the bone marrow after the use of corticosteroids or alcohol abuse. Vasoconstriction of the epiphyseal arteries of the femur can be enhanced by corticosteroids. Intravascular occlusion may be the result of thrombosis, fatty or gaseous embolization, or aggregation of sickle cell red blood cells. The use of corticosteroids is considered the most common risk factor for angiosis, which, according to various authors, accounts for 10 to 30% of cases of this pathology. However, only 8-10% of patients taking corticosteroids develop IT. The dose of the drug, which is usually associated with the development of ANGBI, is more than 2 g of prednisone, or its equivalent per day, for 2-3 months. According to magnetic resonance imaging (MRI) data, the total dose of corticosteroids taken by the patient before his diagnosis of angiosis ranges from 1800 to 15500 mg (on average, 5928 mg) of prednisone or its equivalent. The same study showed that the period from the start of corticosteroid use to the diagnosis of ANGBI is very individual and ranges from 1 to 16 months (5.3 months on average); in most patients, ANGBI is detected within a year. However, the literature describes cases of ANGBI development after prolonged use of corticosteroids. Thus, in he was diagnosed in a patient with refractory ulcerative colitis only after ten years of prednisone use. On the one hand, predisposing risk factors may not always be identified in patients with ANGBI, but often only a small number of them develop pathology in groups of patients exposed to a certain risk factor.

There are regular reports in the medical literature about hip (as well as knee and shoulder) joint cancer that occurs due to metabolic disorders with prolonged use of corticosteroids after solid organ transplantation. However, in two separate studies by Lieberman J.R. and his colleagues, the percentage of angiosis in patients after exposure to high doses of corticosteroids was estimated, and it was shown that osteonecrosis is rarely detected. In the first study, 3 out of 203 patients (2%) developed symptomatic TBS after liver transplantation. Similarly, in the second study, TBS or knee cancer developed in only 6 out of 204 patients (3%) after heart transplantation. These observations can be explained by the multifactorial nature of the OH process and suggest that additional genetic factors are necessary for the development of symptomatic necrosis in a patient. On an in vivo model, Korompilias A.V. et al. It has been shown that corticosteroids can act as a trigger triggering the OH process against the background of an already existing pathological condition (for example, autoimmune pathology); Thus, a side effect of systemic glucocorticosteroid therapy is the risk of necrotic changes in the joint. The above-mentioned authors also provided evidence confirming that the body's immunological response plays an important role in the pathogenesis of ANGBC. Studies have shown a link between the development of ANGBC and the presence of a history of thrombophilia and hypofibrinolysis in patients. At least abnormalities of blood coagulation factor I were found in 82% of patients with ANGBC compared with 30% in the control group ($p < 0.0001$) [208]. Deviations in the values of two or more coagulation factors were detected in 47% of patients compared with 2.5% in the control group ($p < 0.0001$). An increased level of plasminogen activator type I inhibitor (PAI-1) was found in patients with ANGBC. The same authors showed that hereditary hypofibrinolysis and thrombophilia are more common in women with hyperestrogenism and are the main factors in the pathogenesis of ANGBI in this group of patients; such patients should undergo courses of anticoagulant therapy with enoxaparin.

Patients infected with the human immunodeficiency virus (HIV) are also at risk of developing ANGBC, although it is still not clear whether the cause of osteonecrosis is the virus or the drug therapy used. HIV-associated ANGBC has been observed in patients since the 1990s, and the number of such patients is growing. The destructive process in TBS is usually two-way. Although some reports indicate a link between osteonecrosis and highly active antiretroviral therapy, this version does not have sufficient evidence. Ries M.D. and others. Cases of patients with HIV and ANGBC who have not received highly active antiretroviral therapy are described, which suggests that HIV infection may be an independent cause of osteonecrosis. The development of ANGBC in HIV-infected patients may be associated with other risk factors observed in these patients, including the use of corticosteroids and hyperlipidemia, alcoholism and increased blood clotting, megestrol acetate therapy (chemotherapy with toxic drugs) and immunostimulating drugs. Most of the reported cases of hereditary ANGBC are associated with Gaucher's disease, sickle cell anemia, or familial thrombophilia. The study demonstrated a link between ANGBC and autosomal dominant inheritance of a mutation in the gene encoding type II collagen of class alpha-1 (CO2L1). Based on the analysis of the literature, it should be noted that at the moment, with the development of modern diagnostic methods, the number of etiological factors causative for the development of ANGBI has significantly expanded. At the same time, the theory of ANGBC pathogenesis focuses primarily on the role of ischemia in the joint. Ischemia occurs for various reasons - as a result of injury; any pathology, hereditary or acquired; pharmacotherapy and, as a result, leads to subsequent changes in the bone structure.

Conclusion. The progression of ANGBC is caused by partial revascularization of the infarct site, simultaneous processes of bone regeneration and resorption by osteoclasts, and recurrent vascular disorders of arterial and venous blood flow in the femoral head bone tissue. Dynamic monitoring of the TBS head with impaired blood supply (MRI, X-ray) It revealed a pronounced polymorphism of the morphological manifestations of the disease: there are areas where bone ischemia resolves without necrosis, in other cases typical osteonecrosis develops, often coexisting with bone repair. The dynamics of the development of the ANGBC process is often unpredictable, even during

therapeutic measures, and therefore aseptic necrosis of the TBS head in traumatology and orthopedics is rightfully considered one of the least studied pathologies.

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