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Current View of Diagnosis and Treatment of Avascular Necrosis of The Femoral Head

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Abstract: Avascular necrosis of the femoral head is a serious disease, the relevance of which has not been lost to this day. With early detection and adequate therapy the prognosis is favourable, but late diagnosis and lack of treatment lead to rapid destruction of the joint and disability of the patient. The aim of this study is to provide information on modern diagnostic possibilities and differentiated approach to the choice of treatment method depending on the stage of osteonecrosis.Key words: avascular necrosis of the femoral head, osteonecrosis.

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INTRODUCTION

Avascular necrosis is a severe disease characterised by a disruption of physiological processes of bone tissue regeneration, leading to the destruction of bone cells. Such a process occurs in a certain area of bone tissue [1,2,4]. The most frequent localisation of this pathology is the head of the femur, which according to various authors is observed in persons aged 35 to 55 years, 20% of patients are over 50 years of age [3,5,7]. In terms of the incidence of this pathology, men predominate women, in terms of lesions more than 50% of cases are observed bilateral lesions. Authors distinguish two types of avascular necrosis: medullary - characterised by disruption of blood supply to the medullary canal, destruction of trabecular bone cells and lattice-cortical - with involvement of the joint, with death of trabecular and subchondral bone [19]. A sufficient number of classifications of avascular necrosis have been proposed, but none of them include early stages of the process and postimpression late stages of the disease. Association Research Circulation Osseous (ARCO) classification, which includes the above mentioned stages, is widely used.

Diagnosis of avascular necrosis of the femoral head at early stages has its own certain difficulties. Some authors indicate progressive atrophy of the thigh and gluteal muscles on the affected side early in the disease [18,22]. They also observe restriction of rotational movements, especially internal rotation (80-85% of cases), followed by restriction of hip extension, and lastly, a decrease in hip flexion.

Classification Association Research Circulation Osseous (ARCO 1991)

Stages of the disease	Morphological changes
Stage 0	no lesion centre
Stage I	no radiological changes, the focus is detected only in MRI examination

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stage II	first signs of osteonecrosis without disturbance of the articular surface shape are detected in the femoral head on radiography, MRI, radionuclide study
III stage	subchondral fracture without violation of the sphericity of the femoral head. The 'crescent sign' is observed in the subchondral bone
IV stage	Impression (flattening) of the femoral head area
Stage V	all of the above changes combined with narrowing of the articular gap (secondary necrosis)
stage VI	total degenerative-dystrophic changes of the joint

According to ARCO International, the mandatory diagnosis of osteonecrosis is based on instrumental methods of investigation - radiography and MRI [14].

Radiography should be performed in all patients with joint pain; it allows diagnosis at a late stage of osteonecrosis, detection of bone fracture and concomitant osteoarthritis or arthrosis of the joint. In the absence of radiological changes in the joint, it is recommended to perform MRI, on a device with a magnetic field strength of at least 1.5 Tesla, which allows to detect osteonecrosis at an early stage. To determine the effectiveness of the prescribed treatment and further management of the patient, it is recommended to perform MRI studies 3 months after the start of therapy [13,23]. Angiographic study during radiological examination is recommended to assess blood flow in the area of osteonecrosis when planning surgical treatment with bone grafts [12,17]. Whole-body bone scintigraphy is recommended in the differential diagnosis of osteonecrosis with cancer [10].

Densitometry is recommended for patients over 50 years of age with secondary or idiopathic osteonecrosis. Systemic loss of bone mineral density was observed in 60% of patients with traumatic osteonecrosis, 33% of patients taking glucocorticoids, 27% of patients with idiopathic osteonecrosis, 11% of patients with alcohol abuse and 10% of patients receiving chemotherapy [6,8,9].

Depending on the stage of the disease, conservative or surgical treatment of avascular necrosis of the femoral head is prescribed. Conservative therapy includes unloading of the adjacent joint in combination with the administration of NSAIDs, osteotropic therapy, vascular therapy, intra-articular injections and physiotherapy. Crutch walking with joint unloading for at least three months is recommended. Joint unloading in the early stages of osteonecrosis is necessary to reduce the risk of joint surface impingement, as walking in this case increases the load by 3.5 times, which in the presence of microfractures in the subchondral zone can be critical for the patient in the acute phase of osteonecrosis. Unloading less than three months is insufficient because the remodelling cycle of a certain bone tissue area even under favourable conditions is 3 months [21].

The goal of drug therapy in the early stages of osteonecrosis is to reduce the intensity of bone resorption of the femoral head, stimulate osteoregeneration, which leads to an increase in bone mass, improve its quality, increase strength and prevent impression deformity [7,8].

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Daily administration of calcium-containing preparations (500-1000 mg/day) in combination with colecalciferol is recommended as basic therapy in osteonecrosis from the first days after the disease detection and independent of its localisation [10,11]. Active metabolites of vitamin D3 and calcium preparations influence the proliferation of osteoblast precursors, activate bone formation processes and improve mineralisation of newly formed bone tissue. Improved calcium absorption in the intestine contributes to an increase in skeletal mass and increases the mineral composition of bone, including its cortical component, which determines the strength properties of the femoral head [7,9]. Among the etiological factors of osteonecrosis a certain role is played by the pathology of the blood coagulation system, then the use of anticoagulants is assumed in the therapy of the disease. Disaggregants are prescribed to improve microcirculation in the lesion area [19,20].

When choosing surgical treatment of osteonecrosis of the femoral head, it is recommended to take into account the stage of the disease, the presence of an impression fracture and the patient's age. At stages 1 and 2 according to ARCO classification, tunnelling (decompression) of the affected area of the femoral head is recommended to reduce pain syndrome and improve blood supply. Tunnelling is performed under EOP control, and it is important to pass the channel to the osteonecrosis focus and not to traumatise the hyaline cartilage covering the focus. To enhance the effect of tunnelling the focus of damage, it is recommended to inject biological substances with stimulating effect [24]. But tunnelling of the osteonecrosis focus is not recommended at late stages of the disease in the presence of subchondral bone collapse [16,18]. In early stage 3 according to ARCO classification, bone autograft on a vascular pedicle is recommended. In late stage 3, it is recommended to perform total joint replacement or to use (in young patients) a bone autograft on a vascular pedicle [15]. From stages 4 to 6, after subchondral bone collapse and in the presence of pain, total joint replacement is recommended; cemented constructs are not recommended in young patients because of the high risk of reoperation [20].

Conclusion. Early diagnosis of osteonecrosis determines the tactics and prognosis of treatment. Small lesions (less than 15% of the femoral head) can be completely repaired on the background of treatment. In lesions of more than 50% of the femoral head, with a high probability of progression to collapse, eventually requiring total joint endoprosthesis. However, in most cases it is not possible to prevent the progression of the disease, as the patient's non-compliance with the orthopaedic regime, the presence of concomitant pathology and chronic diseases have a special influence.

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