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MORPHOLOGICAL ASPECTS OF ASEPTIC NECROSIS OF THE FEMORAL CAPITIS AFTER COVID-9

Choriyev B.A., Primov Kh.N., Bakhriev I.I., Saydakhmedov M.K., Mirzamukhamedov O.Kh., Sobirova D.R.

Tashkent Medical Academy, Tashkent, Uzbekistan
Center for Forensic Medical Examination of the Armed Forces of the Republic of Uzbekistan

Abstract. *This study describes the specific pathological changes in aseptic necrosis that developed after COVID-19. In this study, 49 patients underwent surgery and the femoral capitis was removed for examination. According to the results of the study, in the late stages of aseptic necrosis, fibro-, chondro-, osteomatous restructuring, the formation of a "fractured membrane" on the surface of the femoral capitis, the appearance of foci of dystrophic-degenerative and destruction-necrosis (melting), chronic non-specific inflammation, between bone trabeculae and pillars, the formation of granulation tissue and the growth of fibroreticular tissue, osteoclast resorption reactions (lacunar).*

Keywords: *COVID-19, aseptic necrosis, morphology, femoral capitis, destruction.*

Introduction. It is known that COVID-19 has various negative effects on various organs and systems of the human body. Some statistics have shown that about 20-30 thousand new cases of osteonecrosis of the femoral capitis are registered annually in the USA alone, which is more than the total number of patients, which is about 5-5.7 million [5].

Additional studies have shown that the etiology of osteonecrosis is multifactorial, and there is no single etiological factor. Most of the blood supply to the femoral capitis in adolescents and adults comes from a branch of the deep femoral artery, which is considered a branch of the femoral artery. For this reason, disturbances in the blood supply to these vessels, i.e., obstruction of subchondral microcirculation, lead to bone necrosis. Bone cell necrosis is associated S a high risk of developing secondary osteoarthritis and the appearance of microcracks in the area of osteonecrosis S limited range of motion in the hip joint [6, 7].

As an independent disease, E. Bergmanom was first described in 1927 and E. Froyndom in 1939. The disease usually develops at the age of 35-55 years (38 years on average). The ratio of incidence of men and women is 3:1. There is specific information in the scientific literature on the development of osteonecrosis after infection S COVID-19 [1, 4, 5].

According to L. Hui et al., 39% of patients infected and SARS-CoV-2 developed necrosis of the femoral capitis Sin a few months after SARS [4].

Diagnosis of necrosis of the femoral capitis is mainly based on clinical and radiographic findings. The typical clinical presentation includes progressive pain, stiffness, and crepitus. During exercise, patients usually complain of limited range of motion and pain in the hip joint. Early detection of the disease allows you to effectively deal and it. Many imaging modalities have been shown to be effective in detecting evidence of bone necrosis, including radiography, magnetic resonance imaging (MRI), and computed tomography (CT). These methods are the gold standard for diagnosing osteonecrosis and distinguish it from other diagnoses [4].

Osteonecrosis of the femoral capitis, also called avascular necrosis, is a pathological condition characterized by decreased vascularization of the subchondral bone of the femoral capitis, resulting in osteocyte death, demineralization and resorption of bone pore tissue, changes in trabecular architecture included [2, 6].

There are two types of pathological osteonecrotic process: medullary and cortical. Medullary osteonecrosis develops when the blood supply to the medullary canal is disturbed, which leads to the death of trabecular bone cells. Orthical osteonecrosis is more severe.

The risk of vascular disease is usually noted in the proximal part of the thigh, where trabecular and subchondral bones die, and this area does not undergo calcification, as in medullary necrosis [3].

In addition, there is information about the effect of coagulopathy, pancreatitis, bleeding disorders, and some autoimmune diseases on its development.

However, according to different authors, in about 40-50% of cases the disease develops idiopathically Sout etiological factors. According to recent data, among the causes of idiopathic osteonecrosis, blood clotting disorders are of great importance [9].

Information on pathomorphological changes in aseptic necrosis of the bone tissue of the hip joint after COVID-19 in the scientific literature is not enough, and the pathogenesis, morphogenesis and pathohistological changes of aseptic necrosis that developed in the bone tissue in this pathology have not been studied. "learning" is considered an actual problem that needs to be studied today [4,8].

The International Society for the Study of Osteoarthritis has proposed a system for assessing histopathological changes in particular hyaline, according to which it is divided into 4 stages. For stage 0, there is no pathology in the joint. Pathological changes in the joint

begin in the superficial zone and gradually spread to deeper sections. At stage I, the integrity of the cartilage tissue is preserved, but there is a loss of the cellular plate. In chondrocytes, signs of apoptosis, hypertrophy and proliferation are simultaneously observed. At stage II, shallow cracks are observed on the surface of the togai layer. In some areas it will be possible to see the thinning of the uncle. There is a stratification of collagen fibers, they are well distinguishable. In the superficial zone, the fibers are arranged in parallel, and in the central zone, perpendicular to the articular surface. At stage III, vertical cracks also occupy the central zone. Cracks propagate along the periphery and divide the matrix into pieces. Chondrocytes are arranged randomly and form focal accumulations. The border of the ossification zone is blurred, in some places it is not observed.

In stage IV of the pathological process, microvilli are attached to the joint space in the joint where the pathological process takes place. The subchondral bone is thickened and has a porous structure, sometimes devoid of cartilaginous tissue. It is characterized by varying degrees of deformation of the articular surfaces and the activity of bone reconstruction processes. At this stage, it is characterized by a significant absence of an uncle in the form of separate foci or in the form of large areas. In addition, sclerotic thickening of the subchondral bone is observed, as well as the formation of cystic spaces.

Research objective. To present the morphogenesis and histopathological features of aseptic necrosis of the femoral capitis after COVID-19.

Materials and Methods. In this study, a histological analysis of the femoral capitis obtained by surgery was performed in 49 patients and a diagnosis of aseptic necrosis of the femoral capitis requiring surgical intervention, who were treated in the traumatology department of the Central Military Clinical Hospital of the Ministry of Defense of the Republic of Uzbekistan. We checked the defense for 2019-2022.

The patients in our study were male military personnel and civilians aged 30 to 74 years. Thus, out of 49 examined patients, 17 (34.7%) were under the age of 40 years, 18 (36.7%) were aged 40-50 years, and only 14 were over 50 years old, which is 28 of all patients. According to the results of a virtual examination of patients in the femoral capitis, various degrees of aseptic necrosis were revealed. During a surgical operation at III-IV stages of aseptic necrosis, the area of the femoral capitis affected by aseptic necrosis, the outer shell of the bone, the shell of the femur and the neck of the femur were removed together. Pieces of tissue were examined macroscopically, pieces 1.0-1.5 cm long were cut out of each and fixed in 10% formalin and phosphate buffer solution for 72 hours. The bone fragments were decalcified in 10% nitric acid. Then, after washing in running water for 3-4 hours, they were dehydrated in alcohols of increasing concentration, embedded in paraffin, and blocks were prepared. Histological sections of 5-7 μm in size were taken from paraffin blocks, deparaffinized and stained S hematoxylin-eosin.

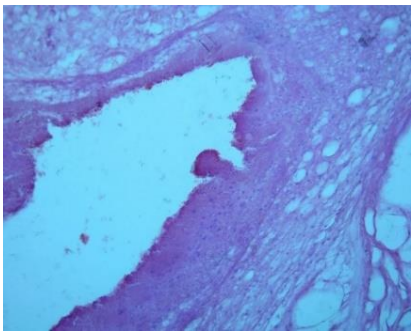

Fig. 1. The appearance of a "cystic center" in place of the necrotic area of the bone head tissue, the cyst wall is composed of fibrochondromatous tissue of uneven thickness, S neurobiotic-dystrophic changes on the inner surface. G-E dye. Cat. 10x40.

Fig. 2. The resected femoral head. The subchondral surface is uneven, small "bone cracks" of various depths have appeared, the chondroid tissue in the subcapsular area is uneven, rough, and the chondroid tissue is sometimes S foci of dystrophy-degranulation.

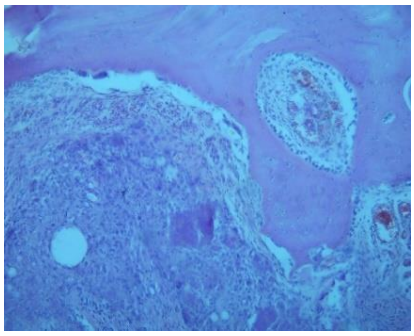

Fig. 3. Bone columns are unevenly and irregularly located, osteoclasts and osteoclast-like multinucleated large cells have appeared in the peripheral part and intertrabecular tissue areas, necrotic and necrobiotic areas (osteolysis) of bone blocks have areas of infiltration mixed S lymphohistocytocytes and plasmocytes, blood vessels are unevenly full. G-E. paint. Floor 10x40.

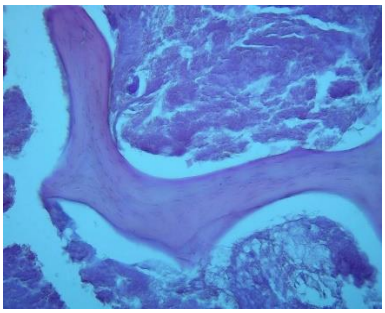

Fig. 4. The uneven and irregular structure of the bone structure, the structure is homogenized, and the interstitial tissue is unevenly stained, the foci of aseptic necrosis in the intertrabecular tissue are homogenized and intensively stained S basophils, the structure is not differentiated. G-E paint. A letter. 10x20.

Fig. 5. The macroscopic appearance of the resected bone head in section: the subchondral surface is unevenly separated from the bone marrow, sometimes unevenly thickened, partially separated from the area of aseptic necrosis (up to 0.4-0.8 cm). Under the subchondral tissue, unevenly located "aseptically necrotic" area in the area of 3.0x2.5x2.0 cm in semi-circular shape and dystrophy-degranulation fluid-colored foci, sometimes uneven hemorrhage foci can be seen in the tissue.

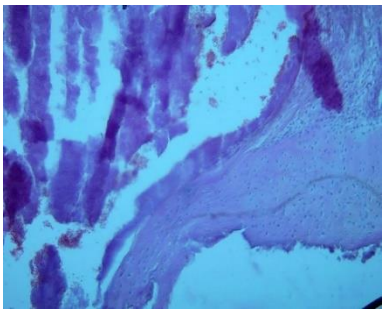

Fig. 6. The uneven and irregular structure of the bone structure, the structure is homogenized, and the interstitial tissue is unevenly stained, the foci of aseptic necrosis in the intertrabecular tissue are homogenized and intensively stained S basophils, the structure is not differentiated. G-E paint. A letter. 10x40

The surface of the resected area of the femur is irregularly oval, unevenly smooth, dark brown in color, the lateral surface, subcapsular tissue are unevenly deformed, sometimes and uneven pits. Along the edge of the lateral surface, a fissured (slit-like) defect 1.5x0.5 cm in size is visible on the bone, the bottom of which has penetrated into the structure (layer) of the bone marrow, and an area of focal necrosis, and destruction (decay) is visible along the edges and bottom and spreads towards the bone fabrics (Figure 5). On the cut (cut): the thickness of the subchondral layer (bone membrane) is 0.4 cm, the width of the detachment of the subchondral layer from the bone tissue around the area of the cracked shell is up to 0.5 cm, and the bone tissue and bone marrow around it are uneven (Figure 2, 6). The hemispherical tissue 3.0x2.5x2.0 cm is necrotic and destroyed, a fibrous cyst (cavity) 0.5x0.5 cm in size was found in this area. serous and grayish detritus and softened tissue is determined in the cavity. When other sections are cut, the subchondral surface (periosteum) has a thickness of up to 0.4 cm, areas of uneven compression (compression) are visible due to the uneven growth of fibro-osteochondromatous tissue around and at its bottom (Figure 1). The surrounding marrow is light brown and yellowish in color. Microscopic examination: on the surface of the subchondral tissue, the fibrochondromatous tissue is sharply thinned, unevenly thickened in places due to the growth of fibrochondromatous tissue. On several transverse sections, the columnar and trabecular structure of the bone is sharply disturbed and lost, predominantly areas of destruction and zones of homogenized necrosis (aseptic) are visible. In these areas, the tissues underwent sharp dystrophic-degenerative changes. Bone columns are unevenly and randomly located, homogenized, intensely stained and basophils. Between the bone columns and the intertrabecular tissue, areas of fibromatous tissue of different thickness and density and granulation tissue of varying degrees of maturity are formed. In the interstitial tissue, infiltrates are determined and predominantly fibrohistiocytic, sometimes rare and predominantly dark lymphoplasmacytic cells. In the deep layers, between the areas of necrosis and destruction, a focus of a cystic structure is visible, the wall of which is thinned by fibrous tissue, and homogeneous necrotic detritus is determined in the cavity. Osteoclast cells (large multinucleated cells prone to absorb decomposed bone tissue) and osteocyte cells are sometimes sparse and, conversely, accumulated along the edge of bone trabeculae and columns, located chaotically (unevenly) and unevenly in separate areas. most of these cells seem to be concentrated around bone trabeculae and columns, as well as around bone fragments and columns and uneven contours, dystrophic-degenerative (Figure 3, 4). In the bone tissue and bone marrow structures, areas of focal necrosis are visible, surrounded by areas of rarefied and dark lymphoplasmacytic infiltration, newly formed capillary-type vessels in the emerging focal granulation tissue, focal fibrous tissue, and fibrohistiocyte cells are also visible. A mixture of hyaline and fibrous tissue along the edge and along the edge of the bone tissue, the interstitial tissue is unevenly colored and edematous, in some areas the cells of the fibrous and chondromatous tissue underwent focal proliferation, in some areas the cells are scattered cell-free areas are also visible. On some

sections, the chondromatous tissue is sharply thickened, sometimes cell-free areas are also visible. On the surface of the joint, foci or areas of dystrophic-degenerative changes are determined.

Results and Discussion. To date, two mechanisms have been proposed for the pathogenesis of aseptic necrosis after infection and COVID-19. The first is the defeat of the bone tissue vessels by the virus, and the second is the negative effect of glucocorticoids used in the treatment of infection on bone tissue.

It should be noted that foci of aseptic necrosis can be detected in several bones in one patient, of which 13% of cases occur in the femoral capitis and ankle. Aseptic necrosis, the death of osteocytes and bone marrow cells occurs as a result of bone infarction, the cause of which is the absence of collateral vessels in the arteries that supply the bone, and the development of thrombosis against the background of COVID-19.

Aseptic necrosis, necrobiotic changes develop as a result of ischemia due to vasoconstriction and thrombosis of the periosteum surrounding soft tissues. Initially, the interstitial osteoid becomes necrobial and cavities appear in which degraded osteoblasts, osteoclasts, and fibroblasts can be seen. At the last stage of necrosis, sequesters can be seen from solid bone columns, and structureless dendrites from intermediate osteoid structures.

On microscopic examination, bone trabeculae were damaged by necrotic involutive processes, fragmented, separated from each other, which led to the appearance of large spaces filled and connective tissue rich in adipocytes. In some bone spaces, microfractures of the bone appeared. Due to chronic ischemia, a large number of osteoblasts appear Sout content in the bone trabeculae, which indicates the death of internal osteocytes. In addition, very little vascularity can be noted in the residual bony trabeculae and sclerotic connective tissue. In osteonecrosis, macrophages are the predominant inflammatory cells present in the femoral capitis, indicating the presence of a large amount of tissue and cellular debris requiring phagocytosis.

Conclusion. The studied surgical material revealed pathomorphological signs corresponding to the last stage of deforming coxarthrosis. Fibro-, chondro-, osteomatous restructuring, the formation of a “cracked shell” on the surface of the femoral capitis, the appearance of foci of dystrophic degeneration and destruction-necrosis (melting), chronic non-specific inflammation, the formation of granulation tissue between bone trabeculae and columns and the growth of fibroreticular tissue, signs such as osteoclast resorption reactions /lacunar/ and cyst formation.

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СУДЕБНО-МЕДИЦИНСКАЯ ЭКСПЕРТИЗА ВЕЩЕСТВЕННЫХ ДОКАЗАТЕЛЬСТВ (АШЁВИЙ ДАЛИЛЛАР СУД-ТИББИЙ ЭКСПЕРТИЗАСИ)