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Aseptic Necrosis of the Femoral Head

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A B S T R A C T
Aseptic femoral head necrosis is a multifactorial disease characterized by the occurrence of femoral head collapse with progressive development of severe secondary coxarthros is. Clinically, the disease is manifested by progressive pain and limited range of motion, Trendelenburg's symptom, crepitation and instability of the femoral head. Treatment includes surgical interventions and correction of cytoactivity. There is evidence of the ability of bisphosphonates to prevent femoral head collapse. Key words: aseptic femoral head necrosis, osteonecrosis, Leiden factor V, interleukin, cross-linked N-terminal telopeptide, tumor necrosis factor α.

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Aseptic necrosis of the femoral head (ANGBC), avascular necrosis (International Classification of Diseases, 10th revision) is a multifactorial disease affecting mainly young people, leading to the development of secondary severe osteoarthritis of the hip joints with subsequent disability [2]. As an independent disease, it was first described by E. Bergman in 1927 and E. Freund in 1939. Initially, ANGBC was compared to Perthes disease in children, suggesting that adults use the same term, but in children, unlike adults, the disease often ends with the restoration of bone tissue with the preservation of the femoral head. The disease usually develops at the age of 35-55 years (mean age 38 years). Data on the prevalence of the disease in the general population are not provided. However, it is noted that in the United States, between 10,000 and 50,000 new cases are diagnosed each year [4, 6,]. Only 20% of individuals develop angioedema at the age of over 50 years [3]. The ratio of men to women is 3: 1, and half of the patients have a bilateral lesion. Approximately 5-18% of all hip replacement surgeries are performed for primary angioedema [3, 4, 16]. There are two types of pathological osteonecrotic process: medullary and ethmocortical involving the joint. Medullary osteonecrosis develops when the blood supply to the bone marrow canal is disrupted, resulting in the death of trabecular bone cells. Ethmocortical osteonecrosis involving the joint has a more severe course (Figure 1). The risk of vascular disorders is usually noted in the proximal femur, both the trabecular and subchondral bones die, and this area is not able to calcify, as in medullary necrosis [19]. Aseptic necrosis of the femoral head occurs due to impaired blood flow and necrosis of the bone marrow elements of the femoral head. Long-term use of glucocorticosteroids (corticosteroids) and autoimmune diseases are the main risk factors

Contact therapy, alcoholism, smoking, injuries and surgical interventions on the joint. There are also data on the effects on the development of the disease of coagulopathies (thrombophilia and disseminated intravascular coagulation), pancreatitis, coagulation disorders, certain autoimmune diseases, systemic lupus erythematosus, ionizing radiation, sickle cell anemia, hyperlipidemia, fat embolism syndrome, hip dysplasia, chemotherapy and / or radiation therapy, organ transplantation, chronic liver diseases. diseases of the liver and metabolic diseases of the bone tissue. However, according to various authors, approximately 40-50% of cases of angioedema are described as idiopathic without etiological factors [2, 16, 35]. According to recent data, among the causes of idiopathic angioedema, great importance is attached to blood clotting disorders, in particular, thrombophilia in such patients is several times more common [10]. Some authors suggest that ANGBC is the result of genetic abnormalities. Another working

hypothesis states that cell death occurs as a result of both an increase in intraosseous pressure of the femoral head, leading to a deterioration in blood supply, and a mechanism similar to the compartment syndrome [12]. According to recent data, approximately 40% of patients receiving corticosteroid therapy develop varying degrees of angioedema [45]. It should be noted that in addition to the fact that the use of corticosteroids is one of the etiological factors of angioedema, they stimulate apoptosis of osteocytes. It is assumed that corticosteroids disrupt the blood circulation of the femoral head, causing tissue ischemia. Bone marrow adipocyte hypertrophy is also induced, which increases intraosseous pressure [4]. In order to identify biomarkers of GCS-induced ANGBC, a comparative study of the expression of individual serum proteins in patients with osteonecrosis and in healthy volunteers was conducted. A significant decrease in the expression of C3-and C4-components of complement, inter-α-heavy trypsin inhibitor H4 and α2-macroglobulin was revealed in patients with osteonecrosis. These proteins are actively involved in the processes of intra-vascular coagulation, apoptosis, and redox reactions [7]. In addition, since angioedema is a multifactorial disease, it is necessary to take into account predisposing factors for the development of GCS-induced osteonecrosis. In experimental studiesxon rats, L. Tian et al. The disease was associated with activation of osteoclasts via Toll-like receptor-4 (TLR4) signaling pathways [34]. The mouse genome is 90% similar to the human genome, which suggests similar patho genetic characteristics of osteonecrosis [34]. According to recent data, TLR stimulation is involved in the pathogenesis of inflammatory and autoimmune diseases. When corticosteroids are used to treat these diseases, anti-inflammatory activity is realized through the suppression of nuclear factor kB (NF-kB), which, in turn, contributes to the development of angioedema. Effects on TLR7 or TLR9 and on interferon

Type I contributes to the development of systemic autoimmune diseases. Nuclear factor kB and interferon regulatory factor-7 are signaling transcription factors that implement a pro-inflammatory response by acting on TLR7 or TLR9. In experiments on rats, exposure to TLR7 or TLR9 in combination with the use of corticosteroids or NF-kB and regulatory factor-7 interferon contributed to the development of angioedema after treatment with corticosteroids. This study proved that innate immune signals via TLR underlie the pathogenesis of aseptic necrosis [30]. It is suggested that impaired differentiation of mesenchymal cells may be a potential mechanism of angioedema pathogenesis. Arterial occlusion is considered the main predisposing factor; there are two main mechanisms: thrombosis and embolism. Arterial thrombosis can develop in two ways, acting synergistically: this is the primary damage to the artery wall and the primary pathology of coagulation. Some authors describe thrombosis caused by endothelial damage due to atherosclerosis or other diseases that cause vascular occlusion. In healthy people, the balance between thrombosis and fibrinolysis is controlled; in the presence of hereditary or acquired thrombophilia and / or hypofibrinolysis, this balance can change, leading to thrombosis. Another possible cause of angioedema is embolic disease. Impaired lipid metabolism leads to an increase in the amount of circulating lipids with reduced lipolytic activity of enzymes. Circulating lipids accumulate in the liver, contributing to fat degeneration and the formation of fat cysts. Rupture of cysts or spontaneous diffusion of fat droplets from the liver leads to fat embolism. The subchondral region of the femoral head is an area of localization of fat emboli both due to the small diameter and due to the location of terminal segments of arterial vessels in this zone. Intraosseous fat emboli cause stasis by mechanical occlusion, as well as damage to the endothelium by products of hydrolysis of free fatty acids. Another mechanism involved in pathogenesis is external capillary compression by hypertrophied bone marrow fat and cells with increased intracerebral pressure and circulatory disorders [19]. Blood supply to the femoral head is provided by the basicervical extracapsular articular vascular ring and the ascending branch of the medial circumflex femoral artery, as well as by small additional arterial tributaries of the lower and upper gluteal arteries of the round ligament [16]. In any case, the main cause of necrosis is acute ischemia caused by acute circulatory disorders of the femoral head (both arterial and venous). According to some researchers (Hungerford D., Leung P., 1983), the cause of these disorders is the peculiarity of blood supply to the femoral head in the form of a closed compartment (Fig. 2). Thus, the development of angioedema is described as a complication in femoral artery stenting [3].

In addition to the vascular factor, great importance is attached to the features of reparative bone regeneration in angioedema, which is disrupted in this disease. In the general scheme, the pathogenesis of this disorder can be presented as follows. Damage to the bone structure and violation of mechanical

properties occur during the beginning of repair processes. For the formation of a new bone with effective mechanical properties, it takes 3 months, but for damage to the mechanical structure of the trabecular bone by osteoclasts, 3 weeks is enough. Thus, reparative processes become ineffective for the mechanical properties of the femoral head. Femoral head collapse occurs under mechanical stress (Fig. 3) [38]. Despite the fact that in the Russian-language literature the term “osteonecrosis”is used to denote the disease, emphasizing the aseptic nature of the pathology, in the English-language literature the term "osteonecrosis" is used. Moreover

Clinical signs and symptoms of angioedema are very diverse and depend on the stage of the disease. In the initial stages, there may be minor bouts of pain for no apparent reason, often with a normal range of motion; pain is noted with internal rotation of the hip. With the progression of the disease, discomfort can be replaced by sudden attacks of severe pain. Such attacks can signal the presence of a collapse or fracture of the femoral head, leading to the final stage of degenerative changes. Further development of the disease is typically determined by increasing mechanical symptoms, including reduced volume and painful movements, Trendelenburg's symptom, crepitation and instability of the femoral head [12]. Clinically, angioedema is characterized by symptoms similar to those of coxarthrosis: 1) pain in the groin area, passing along the front and side of the thigh

Diagnostics Laser Doppler flowmetry and microsensory intraosseous pressure transducers are used to study the femoral head blood circulation [25]. Histological examination of bone tissue samples taken during the operation of endoprosthetics of the affected joint is of great importance. Histological examination allows you to diagnose the disease, differentiate it with other pathologies and determine the pathogenetic pathways of the process. With the progression of osteonecrosi, the bone structure changes significantly. Osteoclast activity increases in the subchondral zone and necrosis zone, while osteoblast activity increases in the sclerosis zone (Figs. 5, 6) [39]. Immuno histochemical examination of bone samples revealed that the subchondral and necrotic zones were positive for the NF-kB receptor activator (RANK)

Osteoblasts (positive for alkaline phosphatase) are detected around trabeculae in the sclerosis zone with a decrease in their number in the subchondral and necrotic zones in comparison with healthy bone. Osteoclasts (positive for tartrate-resistant acid phosphatase).

They appear around the trabeculae of the subchondral and necrotic regions with a decrease in their number in the sclerotic zone compared to healthy bone (see Figs. 5, 6) [39]. Microscopically, osteonecrosis is characterized by the formation of empty lacunae with vascular fibrous tissue around in combination with adjacent bone tissue (Fig. At higher magnification under a microscope, foci of newly formed bone tissue can be seen next to empty lacunae (Fig. In most cases, laboratory parameters, such as prothrombin time, activated partial thromboplastin time, are normal in angiotensin-angiotensin syndrome [16]. However, when comparing the parameters of patients with non-traumatic angioedema and healthy volunteers, significant deviations in the concentration of clotting factors in blood plasma were revealed. Thus, patients with angioedema showed a significant decrease in ghrelin levels, an increase in the levels of Von Willebr and factor, plasminogen activator inhibitor-1 (PAI-1), and C-reactive protein in comparison with the control group, which indicates the involvement of these factors in the pathogenesis of the disease [20]. As a diagnostic marker of angioedema development, the following definition is proposed:

the level of interleukin-33 in the blood plasma. In a study conducted in 125 patients with angioedema, they showed a significant increase in interleukin-33 levels (174.33 pg/ml) compared to healthy individuals (90.5 pg/ml), regardless of the cause of osteonecrosis [48]. Complex biomarkers for the early diagnosis of angioedema are proposed: levels of OPG, RANKL, cross-linked N-terminal telopeptide (NTX), C-terminal procollagen I peptide (PICP), tumor necrosis factor α, and interleukin-1β1 for enzyme-linked immunosorbent assay. Examination of patients with angioedema revealed statistically significant differences in this complex in comparison with healthy individuals

Literature

1. Mardanov J. J. The results of surgical treatment of pathological spinal fracture during extradural tumor of spinal cord //Europaische Fachhochschule. – 2014. – №. 4. – С. 21-24.

2. Марданов Ж. Ж., Юлдашев Р. Р. Вертебропластика в хирургическом лечении опухолей позвоночника-оценка результатов лечения //Врач-аспирант. – 2012. – Т. 53. – №. 4. – С. 9-13.
3. Марданов Ж. Ж. Задне-боковой доступ при хирургическом лечении экстрадуральных опухолей спинного мозга //Вопросы науки и образования. – 2021. – №. 22 (147). – С. 4-13.
4. Mirzamurodov H. H. New approaches to treatment of patients with coxovertebral syndrome //Asian journal of Pharmaceutical and biological research. – 2021. – Т. 10. – №. 2. – С. 9-19.
5. Mirzamurodov H. H. et al. OPTIMIZATION OF TOTAL HIP ARTHROPLASTY FOR DYSPLASTIC COXARTHROSIS //Новый день в медицине. – 2020. – №. 4. – С. 667-672.
6. Мирзамродов Х. Х. У. УДЛИНЕНИЕ КОНЕЧНОСТЕЙ ПРИ АХОНДРОПЛАЗИИ. СОВРЕМЕННЫЕ ПРЕДСТАВЛЕНИЕ ОБ АХОНДРОПЛАЗИИ //IJTIMOIY FANLARDA INNOVASIYA ONLAYN ILMIY JURNALI. – 2022. – Т. 2. – №. 7. – С. 21-27.
7. Ахмедов Ш. Ш. и др. ЭНДОПРОТЕЗИРОВАНИЕ ТАЗОБЕДРЕННОГО СУСТАВА ПРИ ДЕГЕНЕРАТИВНО-ДИСТРОФИЧЕСКИХ ЗАБОЛЕВАНИЯХ У ВЗРОСЛЫХ //КОЛОНКА РЕДАКТОРА.–2008. – 2018.
8. Ахмедов Ш. Ш. и др. The peculiarities of prophylaxis of pulmonary thromboembolism after total hip endoprosthesis in dysplastic coxarthrosis //Новый день в медицине. – 2020. – №. 2. – С. 53-55.
9. Khamraev B. U., Sh A. S. Two-stage revision hip replacement patiens with severe acetabulum defect (case report) //Asian journal of Pharmaceutical and biological research. – 2021. – Т. 10. – №. 2.
10. Khamraev B. U., Sh A. S. Our experience of treatment of femor fractures by the method of intramedular locking osteosynthesis //Asian journal of Pharmaceutical and biological research. – 2021. – Т. 10. – №. 2.
11. Акрамов В. и др. Эндопротезирование тазобедренного сустава при переломах шейки бедренной кости //Журнал проблемы биологии и медицины. – 2017. – №. 3 (96). – С. 14-16.
12. Акрамов В. Р. Некоторые проблемы эндопротезирования ранее оперированного тазобедренного сустава.“ //БЮЛЛЕТЕНЬ АССОЦИАЦИИ ВРАЧЕЙ УЗБЕКИСТАНА” Узбекистан Ташкент. – №. 2-2011. – С. 110-113.
13. Зиядуллаев А. Х. ЭКСПЕРИМЕНТАЛЬНАЯ МОДЕЛЬ ОСТЕОАРТРОЗА КОЛЕННОГО СУСТАВА У КРЫС НА ФОНЕ ВНУТРИСУСТАВНОГО ВВЕДЕНИЯ ОБОГАЩЕННОЙ ТРОМБОЦИТАМИ АУТОЛОГИЧНОЙ ПЛАЗМЫ //Scientific progress. – 2022. – Т. 3. – №. 4. – С. 1312-1319.
14. Ziyadullaev A., Nurmonov S., Parmonov A. Study of the catalytic reaction of acetylene with cyanuric acid //Journal of science. Lyon. – 2020. – №. 8-1. – С. 11-14.
15. Nematov D. et al. Molecular dynamics simulations of the DNA radiation damage and conformation behavior on a zirconium dioxide surface //Egyptian Journal of Chemistry. – 2019. – Т. 62. – №. The First International Conference on Molecular Modeling and Spectroscopy 19-22 February, 2019. – С. 149-161.
16. Amrilloevich N. D. et al. INTRA-ARTICULAR PLATELET-RICH PLASMA INJECTIONS INTO THE KNEE IN PATIENTS WITH EARLY OSTEOARTHRITIS //Asian journal of pharmaceutical and biological research. – 2021. – Т. 10. – №. 3.
17. Amrilloevich N. D. et al. APPLICATION OF EXPERIMENTAL SIMULATION WHEN STUDYING THE PATHOGENESIS OF OSTEOARTHROSIS //Asian journal of pharmaceutical and biological research. – 2021. – Т. 10. – №. 3.
18. Ozodovich N. S., Halimovich M. H. Morphological Changes In Bone Tissue In Chronic Osteomyelitis On The Background Of Application Of Plate Concentrate //The American Journal of Medical Sciences and Pharmaceutical Research. – 2021. – Т. 3. – №. 04. – С. 160-164.

19. Ozodovich N. S. Analysis of morphological changes in the bones after osteomyelitis and features of treatment methods //Asian journal of Pharmaceutical and biological research. – 2021. – Т. 10. – №. 2.
20. Yunusovich Y. S. Traumatic Significance of Determining the Level of Antibiotic Activity in Fatty Biosynamens //Central Asian Journal of Medical and Natural Science. – 2022. – С. 112-117.
21. Юнусович Ю.С. ДИНАМИЧЕСКАЯ РАБОТА МЫШЦ ПРИ ФИЗИЧЕСКОМ НАПРЯЖЕНИИ //Журнал травматологии и инвалидности. – 2022. – Т. 1. – №. 5. – С. 8-14.
22. Gafforov A. U., Saodat A. U. Improvements in surgical treatmet for diaphysealfractures of the lower leg bones //European Journal of Molecular & Clinical Medicine. – 2020. – Т. 7. – №. 3. – С. 3914-3919.
23. Gafforov A. U., Asilova S. U., Teshaev A. A. ANALYSIS OF REPARATIVE AFTER SURGICAL TREATMENT OF DIAPHYSEAL FRACTURES OF THE SHIN BONES //Art of Medicine. International Medical Scientific Journal. – 2021. – Т. 1. – №. 3.
24. Mirzohidovna H. E. OBESITY AS A RISK FACTOR FOR RECURRENT POLYCYSTIC OVARY DISEASE //Asian journal of pharmaceutical and biological research. – 2021. – Т. 10. – №. 3.
25. Халимова Е.М., Каримова Н.Н. Дисгормония и ее коррекция при преждевременной недостаточности яичников //МЕЖДУНАРОДНЫЙ ЖУРНАЛ СИСТЕМ ЗДРАВООХРАНЕНИЯ И МЕДИЦИНСКИХ НАУК. – 2022. – Т. 1. – №. 4. – С. 408-412.
26. Халимова Э. М., Нурханова Н. О., Сулейманова Г. С. СОМАТИЧЕСКИЙ СТАТУС ЖЕНЩИН С МАСТОПАТИЕЙ В ПЕРИОД ПЕРИМЕНОПАУЗЫ //Молодежь, наука, медицина. – 2015. – С. 359-361.
27. Ходжанов И. Ю., Хакимов Ш. К., Касымов Х. А. Выбор способа хирургического лечения воронкообразной деформации грудной клетки у детей на основе критериев эластичности грудино-реберного комплекса //Травматология и ортопедия России. – 2013. – №. 3 (69). – С. 130-135.
28. Ходжанов И. Ю., Хакимов Ш. К., Касымов Х. А. Некоторые ортопедо-косметические аспекты лечения воронкообразной деформации грудной клетки у детей и подростков //Врач-аспирант. – 2012. – Т. 52. – №. 3.4. – С. 531-539.
29. Fayziev X. B. et al. Morphological aspects of the spleen of white mongrel rats after severe traumatic brain injury caused experimentally in the form of a road accident //International Journal of Pharmaceutical Research. – 2021. – Т. 13. – №. 2. – С. 998-1000.
30. Файзиев Х. Б., Тешаев Ш. Ж. Черепно-мозговая травма и иммунитет //Новый день в медицине. – 2020. – №. 2. – С. 577-579.
31. Ilkhomovna K. D. Morphological Features of Tumor in Different Treatment Options for Patients with Locally Advanced Breast Cancer //International Journal of Innovative Analyses and Emerging Technology. – 2021. – Т. 1. – №. 2. – С. 4-5.
32. Khodzhaeva D. I. Changes in the Vertebral Column and Thoracic Spinecells after Postponement of Mastoectomy //International Journal of Innovative Analyses and Emerging Technology. – 2021. – Т. 1. – №. 4. – С. 109-113.
33. Khodjayeva D. I. MORPHOLOGY OF IDIOPATHIC SCOLIOSIS BASED ON SEGMENT BY SEGMENT ASSESSMENT OF SPINAL COLUMN DEFORMITY //Scientific progress. – 2022. – Т. 3. – №. 1. – С. 208-215.
34. Ilkhomovna K. D. Modern Look of Facial Skin Cancer //BARQARORLIK VA YETAKCHI TADQIQOTLAR ONLAYN ILMIY JURNALI. – 2021. – Т. 1. – №. 1. – С. 85-89.
35. Sanoeva M. et al. Comparative analysis of cognitive function at vascular complications of migraine (diagnosis and clinical approach) //Journal of Critical Reviews. – 2020. – Т. 7. – №. 3. – С. 425-430.

36. Саноева М. Ж., Саидвалиев Ф. С. Мигрень-вчера, сегодня, завтра. Современный взгляд на проблему //Международный неврологический журнал. – 2016. – №. 8 (86). – С. 72-78.
37. Sanoeva M. et al. Peculiarities of clinical and hemodynamic manifestations of migraine strokes //International Journal of Psychosocial Rehabilitation. – 2020. – Т. 24. – №. 2. – С. 350-358.
38. Саноева М. Ж., Жураева Г. Б., Мухидова Г. Х. Клинические особенности развития депрессии как предиктора осложненных форм мигрени //Вестник Совета молодых учёных и специалистов Челябинской области. – 2018. – Т. 1. – №. 3 (22). – С. 29-36.
39. Atayevich R. O. Xviii–Xix-Asrlarda O‘zbekistonda Tibbiyotning Rivojlanishida Solih Ibn Muhammad Qandaxoriyning QoShgan Hissasi //AMALIY VA TIBBIYOT FANLARI ILMIY JURNALI. – 2022. – Т. 1. – №. 5. – С. 74-78.
40. Ходжаева Д. И. АНАЛИЗ СРАВНЕНИЯ МОРФОТОПОМЕТРИЧЕСКИХ ПАРАМЕТРОВ СТРУКТУР ПОЯСНИЧНОГО ОТДЕЛА ПОЗВОНОЧНОГО СТОЛБА В НОРМЕ И ПРИ ДЕГЕНЕРАТИВНО-ДИСТРОФИЧЕСКИХ ИЗМЕНЕНИЯХ //Uzbek Scholar Journal. – 2022. – Т. 5. – С. 192-196.
41. Khodzhaeva D. I. Modern Possibilities of Ultrasounddiagnostics of Skin Cancer //IJTIMOIY FANLARDA INNOVASIYA ONLAYN ILMIY JURNALI. – 2021. – Т. 1. – №. 1. – С. 101-104.