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LABORATORY PARAMETERS OF CHRONIC OSTEOMYELITIS

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Abstract
Chronic osteomyelitis (CS) is a long-term infectious and inflammatory disease of bone tissue, often complicated by the formation of sequestration and purulent fistulas. Diagnosis of CS is difficult due to the nonspecific clinical manifestations and the need to differentiate with other pathologies of the bone system. Laboratory parameters play an important role in confirming the diagnosis, assessing the activity of the inflammatory process and monitoring the effectiveness of treatment. This article discusses the key laboratory markers used in the diagnosis and monitoring of CS. These include indicators of a general blood test (leukocytosis, a shift in the leukocyte formula to the left, an increase in ESR), biochemical parameters (C-reactive protein, procalcitonin), as well as microbiological studies (seeding of purulent discharge on bacterial flora and determination of sensitivity to antibiotics).

Keywords: Hematological osteomyelitis, leukocytes, clinic, diagnosis, recurrence, amputation.

Introduction

Special attention is paid to the dynamics of laboratory parameters during the treatment of CS, since their normalization indicates the effectiveness of the therapy. The possibilities of using new laboratory markers, such as determining the level of cytokines and markers of bone metabolism, are also discussed for a more accurate assessment of the activity of the inflammatory process and predicting the outcome of the disease. The article presents the results of a study of a number of immunological parameters in acute and chronic hematogenous osteomyelitis in comparison with their features of the course of the disease. A characteristic feature of acute hematogenous osteomyelitis (CGO) is the high frequency of chronization of the inflammatory process, which is caused by the development of secondary immunodeficiency. A significant difference to the

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surgical rehabilitation of patients with bone defects after hematogenous osteomyelitis, Chronic osteomyelitis is characterized by the presence of a focus of infection or non-purulent inflammation of the bone tissue, a recurrent course and is considered an incurable disease. [1,3,9,10,11,13].

Chronic osteomyelitis is characterized by the presence of a focus of infection or non-purulent inflammation of bone tissue, a recurrent course and is considered an incurable disease (P. Miettunen et al., 2009). Unfavorable treatment results (relapses, amputations, functional disability of limbs) still reach 20-30%, which leads to disability of the child (Mader J.T. et al., 2013; Verhelle N. et al., 2003; Gonsalez M.H. et al., 2005; Cierny G. et al., 2016). The transition of osteomyelitis to a chronic form is due to a number of reasons, of which the main ones are late treatment, insufficiently radical surgery, and errors in antibiotic therapy (Grinev M.V., 1977, Gostischev V.K., 2007). Atypical forms of osteomyelitis occur mainly in children, which immediately acquire a sluggish, chronic course, without an acute phase (Mikhailov M.K. et al., 1985; Akzhigitov G.N. et al., 1986; Cierny G. et al., 2006; Hofmann S.R. et al., 2012). The causes of the development of a chronic process at once, bypassing the acute stage, were seen differently by different authors. The most untenable theories have to be recognized for the development of chronic osteomyelitis with prolonged unjustified use of antibiotics, the so-called "antibiotic" osteomyelitis, in which, allegedly, the virulence of microorganisms decreases, resulting in the development of erased, atypical forms (Akzhigitov G.N. et al.; Prokopova L.V. et al., 1984; Harris N.H. and Kirkaldy-Willis W.H.; Kozlovski K., 1983). Another theory that has not been confirmed attaches too much importance to endogenous and exogenous anthropogenic environmental factors (Chirkin V.V., 1991). [1,5,9,15,16].

There is still a high frequency of unsatisfactory results after surgical treatment and recurrence of chronic osteomyelitis from 9% to 56% (Nikitin G.D. et al., 1990; Eremin A.V., 2006; Linnik S.A., 2012; Shtofin S.G., 2009). In this regard, patients often undergo surgical interventions 5-10 or more times, and remain untreated for decades (Goryunov C.B., Romashov D.V., Butyvshchenko I. A., 2004; Usyk S.F., Fedoseev M.M., Brattichuk A.N., 2007; Bhavan, K.P., 2009; J. Kumar et al. al., 2010). At the present stage, the organization of medical care for patients with this serious disease remains imperfect. There is a late hospitalization of patients, which can range from 77% to 86.2%. The level of timely pre-hospital diagnosis remains low and there is a misunderstanding of the urgency of providing urgent care for this serious disease. Diagnostic errors during the initial examination of a patient by a surgeon are observed in 25.1-58.3%. In a number of regions of the country, this disease has a mortality rate from 0.5% to 3.7% (Bordiyan S.G., 2006).

K.S. Ternovoy and co-authors, I.S. Vengerovsky attach great importance to the body's defenses in the development of primary chronic forms of osteomyelitis. They believe that the body's reactivity and tissue resistance play an important role in the clinical course of hematogenous osteomyelitis. Constant exposure to a microbial agent in some cases can cause a decrease in the protective forces of a macroorganism, lead to the development of acute osteomyelitis (Vengerovsky I.S., 1965; Grinev M.V., 1977). In other cases, on the contrary, the protective properties of the macroorganism improve, immunity and resistance to the causative agent of the disease are developed, therefore, inflammation either does not develop at all, or the disease takes a subacute or chronic course (Harris N.H., 1965; Udeka K., 1975). [1,3,9,17,18,20].

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Isolated exposure to only a polluted environment or only incorrect antibiotic therapy cannot lead to the development of chronic atypical osteomyelitis, since many forms of this disease were described back in the 19th century, before the discovery of antibacterial drugs and pronounced anthropogenic environmental pollution. Chronic osteomyelitis is a widespread disease, covering up to 10% of all inpatient patients with purulent pathology and occupying up to 6% in the structure of pathology of the musculoskeletal system (Nikitin G.D. et al., 2002; Leshchenko I.G. et al., 2003; Fedorov V.D. et al., 2003; Linnik S.A., 2012; Shtofin S.G., 2009; Malcius D. et al., 2009). The number of patients with hematogenous osteomyelitis does not tend to decrease (Bordiyan S.G., 2006). In Russia and the CIS countries, a high level of transition from the acute stage to the chronic remains - 3.1%-30% of cases (Barskaya M.A. et al., 2000; Abaev Yu.K. et al., 2004; Ferreira G.F. et al., 2012). In 1832, Benjamin Brody first described the picture of a localized tibial abscess of an amputated limb of a patient suffering from severe pain in the affected limb, which is not a manifestation of a systemic process that occurred without acute illness and had no previous infections (Stephens M.M. et al., 1988). Garre described sclerosing "non-purulent" osteomyelitis in 1893 (Harris N.H., 1965). In our country, cases of various forms of primary chronic osteomyelitis were described before the introduction of antibiotics into medical practice (Vengerovsky I.S., 1969; Gurevich I.B., 1939; Dieterichs M.M., 1932; Rosenfeld V.E., 1941; Rosenzvit A.I. 1936; Sviridov S.A., 1946). [17, 18, 20].

In 1889, A.A. Bobrov at the III Congress of Russian Physicians and in 1894 E. Lexer proposed an embolic theory, according to which bacterial embolism with slow bone blood flow settles in one of the terminal vessels of the bone (epiphyseal, metaphyseal, diaphyseal). Embolus subsidence is facilitated by the narrowness of the terminal arteries and the slowing of blood flow in them. The deposited microorganisms cause swelling of the surrounding tissues, and a complete blockage of the lumen of the intraosseous vessel occurs, which causes hypoxia and, subsequently, bone necrosis. The authors believe that in early childhood, the vessels of the metaphysis end blindly, and this explains the onset of the inflammatory process more often in the metaphysis area (Grinev M.V., Mikhailov M.K. et al., 1985). [17, 18, 20].

A significant contribution to the development of the theory of osteomyelitis pathogenesis was made by the experiments of Smolensk pathologist Professor S. M. Derizhanov in 1837-1840. The author caused sensitization of rabbits with horse serum. Then, by injecting a permissive dose of serum into the bone marrow cavity, aseptic allergic osteomyelitis was obtained. Based on these experiments, S.M. Derizhanov believed that bacterial emboli do not play any role in the pathogenesis of osteomyelitis. The disease develops only on the basis of sensitization of the body and the occurrence of aseptic inflammation in the bone, which occurs from a variety of causes. In osteomyelitis, proliferative changes in the periosteum and haversov channels compress the vessels from the outside, and the swelling of the walls of the vessels themselves reduces their lumen from the inside. All this makes it difficult and disrupts blood circulation in the bone, contributing to the occurrence of osteomyelitis (Derizhanov S.M.). Indirect signs of autoallergic inflammation against the background of previous sensorization were revealed in their studies by A.A. Gorevoy et al., namely, an increase in leukocytes, ESR, a shift in the leukocyte formula to the left, an increase in the number of eosinophils, signs of hypercoagulation, an increased number of circulating antibodies with a decrease in phagocytosis, characteristic changes in the protein composition and

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physico-chemical properties of blood plasma. (Gorevoy A.A. et al., 2002). The biofilm population is thus a constant source of virulent pathogens. (Ciemy G., 2011; Walter G., 2012). [17,18,20].

Currently, a number of foreign authors classify chronic nonbacterial osteomyelitis as an autoinflammatory disease (McGonagle D. and McDermott M. F., 2006; Hofmann S.R et al., 2012; Miettunen P. et al., 2009). Autoinflammatory diseases are rare disorders characterized by recurrent episodes of fever and inflammation in the absence of high titers of autoantibodies, autoreactive T-lymphocytes, and underlying infection. The association with other autoimmune and autoinflammatory diseases such as palmar-plantar pustulosis, chronic inflammatory bowel disease, psoriasis, c-ANCA positive vasculitis, Takayasu arteritis, and lack of IL-1 receptor antagonists is being discussed. (Hamel J. et al., 2011; Iyer R.S. et al., 2011; Aksentijevich I. et al., 2009). The autoinflammatory mechanism is believed to be based on a violation of cytokine regulation, which can be at different levels: transcription disruption (as a result of gene variability) and epigenetic modifications. Cytokine imbalance can cause disruption of immune homeostasis, increasing or decreasing inflammatory responses, which in turn can cause susceptibility to infection or autoimmune disorder. [7,8,10].

A theory of the occurrence of bone necrosis in relapses of CRHO, based on the occurrence of thrombosis and embolism in the microcirculatory system resulting from disorders in the coagulation and fibrinolytic systems of the blood was proposed. They published the results of surgical treatment of 69 patients with CRHO of various localization in combination with heparin and streptokinase. According to the authors, the proposed method gives positive results and reduces the frequency of recurrence of HRT.

In order to correct the increased blood viscosity, hypercoagulation phenomena, accompanied by a violation of microcirculation processes in CRH, the introduction of hyperosmotic solutions is recommended, as well as accelerated diuresis in the postoperative period.

However, these attempts are not systemic in nature and do not affect all links in the chain of pathogenesis of the recurrence of HRT. In the literature available to us, we have not found any works describing, much less indicating, the correction of the POL-antioxidants system in the treatment of HRGO. There is no information about such a systemic treatment of HCG, which would have a complex and consistent effect on all links of the pathological process associated with the formation of bone necrosis.

A significant difference to the surgical rehabilitation of patients with bone defects after suffering from hematogenous osteomyelitis is the school. Ilizarova [1,2,3,4].

He gave a quantitative description of RVH depending on the time of the disease. The author showed that in patients who were admitted in the first 3 days of the disease, there was a shift in the quantitative indicators of RVG. These data allowed the author to conclude that in the early stages of the development of CSF, there were functional circulatory disorders, which later developed into organic ones with the formation of avascular zones and foci of necrosis.

Pronounced circulatory disorders in the paraossal tissues (vascular spasm, hypertonia, increased volume pulse, etc.) in the affected limb, which occur much later than the intraosseous ones, were indicated [12].

In acute renal failure, the greatest changes in the coagulogram are observed in patients on days 6-12 from the onset of the disease and manifest themselves in a shortening of whole blood

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coagulability [13]. Plasma tolerance to heparin and thromboplastin activity increases with inhibition of heparin content and fibrinolytic activity of blood.

Disorders in the hemostasis system in acute respiratory syndrome in the form of hypercoagulation were found[14].

Research and experimental work by a number of authors has shown that bone necrosis in osteomyelitis is the result of damage to the intraosseous circulation resulting from disorders in the coagulation and fibrinolytic systems of the blood.

According to some authors, an increase in the overall activity of the blood coagulation system was found in the clinic and experiment in the septicopyemic form of CSF, with the local form of the disease, disturbances in the coagulation system were insignificant. Other authors point to the possibility of developing DIC in acute respiratory viral infections and even suggest the occurrence of this syndrome in one anatomical space without significant changes in the coagulation and fibrinolytic systems of the blood [13,14,15].

Many authors attribute the recurrence of chronic osteomyelitis and unsuccessful outcomes in the treatment of chronic osteomyelitis to the fact that bone, consisting of 70% inorganic substance and equipped with specific microcirculation, is unable to provide collateral microcirculation. This contributes to the formation of thrombosis and embolism, which, by obstructing the blood supply to various areas of bone tissue, are the cause of bone necrosis [15.16.17].

Disturbances in the microcirculatory system lead to a decrease in oxygen saturation of tissues, disrupt its normal metabolism, which in turn leads to the suppression of all other metabolic processes in ischemic tissues, which is a fundamental moment in the development of the pathological process. Oxygen metabolism in a purulent wound has been studied in more detail due to more favorable methodological possibilities. On the other hand, there are many common mechanisms in the pathogenesis of osteomyelitis and purulent wounds of soft tissues, since their biological essence is the same - the process of inflammation, which occurs with excessive migration of macrophages and leukocytes into the tissue damage zone. Naturally, the clinical and biochemical changes in the body in osteomyelitis are qualitatively similar to other processes with an inflammatory component [17].

The importance of the required amount of oxygen for the healing of a clean wound has been shown in experimental studies, etc. At the same time, it was found that a significant part of the damaged tissue is in suboptimal conditions of low oxygen pressure and the wound healing process occurs under hypoxic conditions.

In conditions of an infected wound, hypoxia is further aggravated and reaches anoxia. This conclusion was made by those who found a lack of oxygen in the dead space of infected wounds, starting from day 10. A decrease in critical oxygen pressure, which is not the same for different types of cells, causes a deterioration in tissue metabolism. [17,18,20].

Thus, based on experimental data, it was shown that there is a hypoxic gradient in wounds with incomplete blood circulation, i.e. the lesion center relative to the wound edges and surrounding tissues is hypoxic. The wound edges and surrounding tissue show abnormally high oxygen pressure, approaching in many places the pressure measured in the arteries. Based on this, it was suggested that low oxygen pressure in the center of a purulent wound may be biologically beneficial to the wound [18.19.20]. As for oxygen metabolism in bone tissue, it has been shown

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that at low oxygen pressure in atmospheric air, fracture healing occurs more slowly. Studies have shown that acute tissue hypoxia slows down bone regeneration, reducing both the synthesis of collagen intercellular tissue and mineralization. Hyperbaric oxygen saturation has been found to stimulate fracture healing. Low oxygen pressure in bone tissue was found in osteomyelitis. They also showed that a decrease in the partial pressure of oxygen in the tibia in osteomyelitis can be caused by three reasons: microorganisms use oxygen in the course of their vital activity, infection increases the inflammatory process, resulting in increased oxygen consumption and microorganisms, penetrating the microcirculation system, reduce oxygen supply. [17, 18, 20].

Conclusions: Thus, a comprehensive assessment of laboratory parameters is an integral part of the diagnosis and monitoring of chronic osteomyelitis, which makes it possible to optimize treatment tactics and improve the prognosis for patients.

Most cases of osteomyelitis are caused by the spread of infection from adjacent areas or open wounds, and the infection is often polymicrobial and/or involves S. aureus. Osteomyelitis should be assumed in patients complaining of local pain in peripheral bones, fever, malaise, as well as local, resistant spinal pain and weakness, especially in the presence of risk factors for recent bacteremia. CT or MRI scans should be performed, because in osteomyelitis, X-ray results may be inconsistent for > 2 weeks after the onset of the disease. Initial therapy should include broad-spectrum antibiotics. To achieve the best effect, treatment should be based on the results of bone seeding.

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