



CLINIC, DIAGNOSTICS AND TREATMENT OF ACUTE HEMATOGENOUS OSTEOMYELITIS IN CHILDREN

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ABSTRACT

This article discusses acute hematogenous osteomyelitis (AHO) in children, a serious condition involving purulent inflammation of bone marrow and surrounding tissues. It details the challenges in early diagnosis and the importance of timely treatment to prevent complications. The article covers etiology, pathogenesis, classification, clinical presentation, diagnostic methods, and treatment principles. Key aspects include the significance of early surgical intervention, rational anti-inflammatory therapy, and addressing antibiotic-resistant microorganisms to improve outcomes in pediatric patients.

Actuality. Acute hematogenous osteomyelitis (AHO) is a purulent-inflammatory process of the bone marrow tissue that spreads to all components of the bone structure (panostitis) and often affects adjacent tissues. AHO is one of the most serious purulent-septic pathologies of childhood, characterized by a severe clinical course, difficulties in the diagnostic workup, and a significant risk of developing complications. Delayed hospitalization of patients is observed in 77-86.2% of cases, and diagnostic errors during the initial examination reach 25.1-58.3%. Chronicity of the process occurs in 3.1-21.8% of cases. Epiphyseal osteomyelitis leads to orthopedic complications in 23-58.3% of patients.

Objective of the study – to standardize the basic principles of diagnosing clinical symptoms and treatment strategies for AHO in pediatric practice.

General concept. Mentions of pathological conditions associated with bone tissue damage are found even in the works of Hippocrates, but the term "osteomyelitis" was proposed by Raynod in 1831. For a long period, the concept of "panostitis" was used, reflecting the rapid involvement of all structural elements of the bone in the pathological process. In modern practice, abortive forms of the disease are increasingly observed – rapid suppression of the inflammatory process with timely (up to 3 weeks) surgical intervention. In such situations (about 30% of observations), even with thorough X-ray examination, it is not possible to visualize macroscopic changes in bone tissue and periosteal reactions. This suggests that osteitis and periostitis are not independent diseases, but complications of osteomyelitis that arise due to untimely initiated complex treatment.

Thus, AHO should be considered as an acute purulent inflammatory process affecting the bone marrow, bone tissue, and surrounding soft tissues of various etiologies. In pediatric



practice, this pathology is accompanied by various pathomorphological changes and can lead to the formation of orthopedic deformities.

Etiological factors. The causative agents of AHO are pyogenic microorganisms, among which *Staphylococcus* (up to 90%) or its associations with *Escherichia coli*, *Proteus*, *Pseudomonas aeruginosa* occupy a dominant position. Non-clostridial anaerobes can also act as etiological agents, especially in conditions of impaired blood supply, acidosis, and tissue destruction. These microorganisms are identified in 40% of cases, with *Bacteroides* accounting for 72% and *Peptococci* for 28%. In 97% of the studied samples, anaerobes are found in combination with aerobic microorganisms (mainly *Staphylococcus*, *Proteus*, *Pseudomonas aeruginosa*).

Infection occurs through damaged skin, mucous membranes, and lymphoid structures of the pharynx. Pyodermas, inflammatory processes of the nasopharynx, and latently occurring infections are of significant importance. In infants, the umbilical wound often becomes the portal of entry for infection.

In some cases, osteomyelitis develops as a result of the spread of purulent inflammation from adjacent tissues and organs (for example, odontogenic osteomyelitis in case of carious lesions of teeth, osteomyelitis of the ribs in case of pleural empyema, osteomyelitis of the phalanges of the fingers in case of panaritium).

Pathogenetic mechanisms. The pathogenesis of AHO remains not fully understood. The inflammatory process, localized within rigid bone walls, causes sequential compression of venous and then arterial vessels. An indirect confirmation of this mechanism is the pain syndrome that occurs due to increased pressure in the intramedullary space (intraosseous pressure in AHO increases to 300-500 mm H₂O at physiological values of 60-100 mm H₂O).

In the absence of early diagnosis, starting from the 4th-5th day of the disease, purulent exudate migrates through the Haversian canals under the periosteum, causing its detachment. In later stages (8-10 days), the purulent content breaks through into the surrounding soft tissues with the formation of phlegmonous inflammation. Untimely diagnosis significantly complicates the therapeutic process. The intensity of the pain syndrome significantly decreases after spontaneous drainage of the subperiosteal abscess.

In newborns, the main risk factors include: unfavorable premorbid status (maternal infectious diseases, intrauterine hypoxia), lesions of the central nervous system, as well as invasive medical procedures. Intrauterine infection can contribute to the generalization of the inflammatory process with a potentially lethal outcome. The main portals of entry for infection are the umbilical wound, the vascular bed, the skin, and mucous membranes. Most often, the pathological process is initiated by bacteremia developing after catheterization of the umbilical vein.

In children older than one year, the development of AHO is provoked by chronic foci of infection (carious lesions of teeth, tonsillar hypertrophy, adenoid vegetations).

Classification. The first systematization of AHO was proposed by T.P. Krasnobaev (1925), who, based on the characteristics of the clinical course, identified the following forms of the disease: 1) toxic, or adynamic; 2) septic; 3) local (localized). Among the classification systems based on the principles of T.P. Krasnobaev, the classification of I.S. Vengerovsky



(1953), which takes into account the latest data on the etiology and pathogenesis of this disease, has gained the greatest practical significance. According to this systematization, the following forms of AHO are distinguished:

1. Supercute forms with a predominance of systemic intoxication: septicotoxic form, characterized by the dominance of signs of acute intoxication; septicopyemic form with metastatic lesions of vital internal organs and cavities.

2. Forms with a more favorable course, accompanied by metastasis (multiple lesions of bones, joints, intermuscular and subcutaneous phlegmons and abscesses predominate): with metastases in other bone structures; with involvement of joints; with metastases in subcutaneous tissue, muscle tissue, lymph nodes, and other organs and tissues.

3. Localized forms with a dominance of local symptoms: with a lesion of a single bone; with subperiosteal localization without visible involvement of bone tissue.

4. Atypical forms, rarely diagnosed in pediatric practice: acute diffuse osteomyelitis; sclerosing osteomyelitis; albuminous osteomyelitis; intraosseous abscess.

The classification of osteomyelitis proposed by V.A. Shalygin et al. (2002) is based on the addition and unification of the systematizations of I.S. Vengerovsky (1953), G.N. Akzhigitov et al. (1998), as well as ICD-10 (2000).

According to the classification of V.A. Shalygin, osteomyelitis is differentiated by the following criteria: etiological factor (monoculture, microbial association, or unidentified pathogen); clinical form (acute hematogenous, post-traumatic, iatrogenic, firearm, post-radiation, chronic); phase and stage of the pathological process (acute, subacute, chronic); anatomical localization (lesion of tubular bones: epiphyseal, diaphyseal, metaphyseal, mixed, diffuse; lesion of flat bones); features of the clinical course (generalized – septicotoxic and septicopyemic; focal – fistulous and non-fistulous); nature of complications (local and systemic).

Clinical Presentation. The symptomatology and intensity of AHO in pediatric patients are characterized by significant variability due to numerous factors: the body's immune status, the pathogenicity of the infectious agent, the patient's age-related characteristics, the anatomical localization of the pathological process, the duration of the disease, and prior therapeutic interventions. The degree of the body's sensitization plays a crucial role. In cases where the initiation of the osteomyelitis process coincides with the peak of the hyperergic phase of the immune response in a sensitized organism, a pronounced systemic reaction dominates, bearing a certain resemblance to anaphylactoid shock. In alternative situations, systemic manifestations may be less pronounced.

In clinical practice, three main forms of acute hematogenous osteomyelitis are distinguished: toxic (adynamic); septicopyemic; and local.

Toxic (Adynamic) Form of AHO. This form is characterized by a rapid development of the clinical picture with a symptom complex of endotoxic shock. Typical manifestations include a collaptoid state, impaired consciousness, delirium, hyperthermia (up to 40-41°C), possible convulsive seizures, and emetic syndrome. Tachypnea is noted in the absence of clear auscultatory and percussion findings indicative of pneumonia. The cardiovascular system demonstrates signs of central and peripheral circulatory failure, hypotensive syndrome,



followed by the development of heart failure and myocarditic changes. The skin often shows petechial hemorrhages. The tongue is dry with a brownish coating. The abdominal status is characterized by meteorism, tenderness in the epigastric region, and hepatomegaly.

The predominance of general clinical manifestations of severe toxicosis significantly complicates the localization of the primary bone focus. Local symptoms can only be identified after the patient's overall condition stabilizes. In such cases, moderate swelling of the affected area, painful contracture of the adjacent joint, local hyperthermia, and sometimes increased subcutaneous vein pattern are revealed. The detection of these changes serves as an indication for a diagnostic puncture of the suspected focus. Upon verification of the AHO diagnosis, an increase in intraosseous pressure is registered, although purulent contents in the bone marrow canal are usually absent during osteoperforation.

This form of osteomyelitis is characterized by high mortality, despite intensive infusion therapy including broad-spectrum antibiotics, immunomodulatory drugs, and surgical debridement of the focus.

Septicopyemic Form of AHO. This form manifests with pronounced septic symptoms, but bone lesions are diagnosed significantly earlier. The onset of the disease is also acute, with hyperthermia up to 39-40°C, progressive intoxication, and dysfunction of vital organs and systems. Cognitive impairment, delirium, and euphoria are possible. From the first day of the illness, an algic syndrome in the affected limb is noted, the intensity of which is due to the development of intraosseous hypertension. The development of septic complications due to metastasis of purulent foci to various organs (pulmonary tissue, myocardium, renal parenchyma, as well as other bone structures) is characteristic.

Local Form of AHO. This form is characterized by the dominance of local symptoms of purulent inflammation over general clinical manifestations. Atypical osteomyelitis forms also belong to this category.

The onset of the disease in a typical course is acute. Intense pain in the limb occurs against a background of apparent well-being. Older children usually accurately localize the area of maximum tenderness. The patient tends to immobilize the affected limb, as any motor activity intensifies the pain syndrome. With para-articular localization of the focus, the ligamentous apparatus and periarticular tissues are involved in the pathological process, leading to the formation of a persistent joint contracture.

The temperature reaction from the onset of the disease is elevated and remains at high values (38-39°C). The child's general condition deteriorates rapidly, and anorexia and polydipsia are observed, indicating the progression of intoxication syndrome.

An objective examination of the affected limb reveals primary signs of the inflammatory process: local swelling, diffuse tissue infiltration, and increased venous pattern of the skin. The key diagnostic signs of osteomyelitis are: pronounced local tenderness upon palpation and especially upon percussion over the affected area. Swelling and tenderness extend to adjacent anatomical structures.

Symptoms such as skin hyperemia and especially fluctuation in the affected area are late diagnostic signs and indicate the advanced stage of the pathological process.



The success of AHO therapy in children, especially young children, is determined by timely diagnosis and adequate surgical intervention. Surgical treatment should be performed before the development of septic complications. Early debridement of the primary focus (within the first 72 hours) allows for the prevention of septicopyemia, and subsequent comprehensive therapy ensures recovery in 95% of patients.

Diagnosis. Traditional diagnostic methods for osteomyelitis demonstrate insufficient informativeness in the early stages of the disease due to the heterogeneity of the clinical picture, atypical course, and absence of pathognomonic symptoms, especially in early childhood pediatric practice. This leads to a high frequency of diagnostic errors at various stages of examination.

Radiological examination in the first 24 hours has limited value and is primarily used for differential diagnosis with traumatic bone injuries, tuberculous lesions, neoplastic processes, and cystic formations. Initial radiographic signs (linear periostitis, focal destruction, osteoporotic changes) manifest only by the 10th to 21st day of the disease, making early radiographic diagnosis ineffective.

Subsequent radiographic examinations are necessary for verification of the diagnosis, determining the topography and extent of the lesion, as well as monitoring therapeutic effectiveness.

A significant diagnostic role is assigned to microbiological examination methods, including bacterioscopy and culture diagnosis with the determination of microbial load and antibiotic susceptibility. Diagnostic inaccuracies at this stage contribute to a reduction in the effectiveness of complex therapy, chronification of the pathological process, or progression with the development of generalized forms and multiple organ failure.

In the diagnosis of AHO in children, methods of layer-by-layer thermometry of the affected limb segment and radioisotope gamma scintigraphy using technetium pyrophosphate 99 are highly informative. Angiographic techniques (angiography, phlebography, rheovasography, arterial oscillography, longitudinal tetrapolar rheoplethysmography) are used less frequently.

Ultrasound echolocation can be of decisive importance in the diagnosis of acute hematogenous epiphyseal osteomyelitis, as it allows visualization of the inflammatory process in the early stages, preceding the appearance of radiographic changes. Key early sonographic markers of osteomyelitis include: reduction of acoustic density of bone tissue, manifested by an increase in the thickness of the cortical layer (predominantly more than 1.5 mm), an increase in periosteal thickness (over 1 mm), a decrease in its echogenicity, edema of the subcutaneous adipose tissue, and structural changes in muscle tissue. Later ultrasound signs include infiltrative and phlegmonous changes in soft tissues, dilation of the joint cavity with the accumulation of heterogeneous exudate, and thickening of capsular and synovial structures.

Thus, the most accessible and informative methods for diagnosing AHO in pediatric practice are: anamnestic data and the results of an objective clinical examination; comprehensive laboratory diagnostics; identification of the etiological agent; ultrasound examination of the affected and intact (contralateral) limbs; and radiographic diagnosis.



Significant diagnostic difficulties arise in osteomyelitis lesions of the bone structures of the hip joint. In the initial phase of the disease, local symptoms may be blunted due to the massive muscle layer of this anatomical region. Careful examination reveals moderate flexion of the lower limb at the hip joint. Abduction and external rotation are also noted. The motor function of the hip joint is limited and accompanied by pain syndrome. Joint structures and supra-axial skin are characterized by moderate swelling.

In cases of uncertain diagnosis, expanded use of diagnostic bone puncture with subsequent cytological analysis of the aspirate is recommended.

Treatment. The fundamental principles of comprehensive therapy for AHO in children, formulated by T.P. Krasnobaev in 1925, retain their clinical significance to this day. The therapeutic approach includes three main directions: 1) local intervention – surgical debridement of inflammatory foci; 2) antibacterial therapy, taking into account the identified pathogen and its susceptibility to antimicrobial drugs; 3) systemic impact on the patient's body – normalization of the disturbed homeostasis.

Local therapy involves timely debridement of the osteomyelitic focus. Despite discussions regarding the technique and extent of surgical intervention, burr hole osteoperforation remains the leading surgical method. The key objective of osteoperforation is the elimination of pathologically increased intraosseous pressure. In cases of metepiphyseal osteomyelitis in newborns and infants, a puncture method for treating purulent arthritis is used, including evacuation of exudate and irrigation of the joint cavity with an antibiotic solution. Among specialists, there is no consensus regarding the necessity of opening the intraosseous focus in metaphyseal localization of the process in children under 5 years of age. However, osteoperforation performed in the early period of the disease (up to 3 weeks) in children older than one year can be effective. It is important that the surgical intervention is carried out as gently as possible, with minimal damage to the joint and bone structures.

Immobilization is an integral component of the comprehensive treatment of AHO, especially in patients of the younger age group. With a favorable disease progression, strict immobilization should be limited in time (4-8 weeks) to avoid circulatory disorders, muscle atrophy, the development of osteoporosis, and the slowing of reparative processes.

In the context of **antibacterial therapy**, the choice of antimicrobial agent is determined by the sensitivity of the pathogen, the drug's tropism, its compatibility with other medications, and potential toxicity for pediatric patients. Before pathogen identification, it is recommended to prescribe third- or fourth-generation cephalosporins in combination with nitazole preparations. Modern clinical practice faces an increase in the number of antibiotic-resistant microorganisms, which explains the atypical course of acute hematogenous osteomyelitis in young children. This phenomenon is characterized by a subacute course, which significantly complicates timely diagnosis in the absence of pronounced clinical and radiological manifestations.

Normalization of homeostasis includes the restoration of systemic hemodynamics and tissue perfusion (inotropic and volemic support to improve cardiac output and oxygen delivery); respiratory support; nutritional support; correction of immunological status; and intensive detoxification therapy.



Within the framework of detoxification measures, a wide range of extracorporeal therapy methods is used. The preference for certain methods of gravitational surgery is due to many factors, among which the main ones are the availability and technical equipment of the medical institution. A significant role is played by the traditions of scientific medical schools: the use of exchange (cytapheresis, plasmapheresis) or membrane technologies (hemodialysis, ultrafiltration, hemodiafiltration, etc.), physical and chemical methods of intracorporeal and extracorporeal destruction of toxic substances – from photomodification of blood and biological fluids to indirect electrochemical oxidation of blood.

The therapeutic complex can be supplemented by physiotherapy procedures and differentiated immunocorrection aimed at various components of the anti-infectious resistance system, which helps to reduce the exudation phase, stimulates proliferative processes, activates the T-system of lymphocytes, and normalizes the kallikrein-kinin system. As a targeted correction of the activation of free radical oxidation processes and the antioxidant system, vitamins E, C, unithiol, and membrane-stabilizing drugs (fat emulsions, essentielle, and others) are included in the complex of therapeutic measures.

Conclusions. Acute hematogenous osteomyelitis should be defined as an acute purulent inflammation of the bone marrow, bone tissue, and adjacent soft tissues of various etiologies.

Osteitis and periostitis are complications of the initial stage of osteomyelitis (phlegmon of the bone marrow) arising for various reasons, among which the key factor is the untimely initiation of complex therapy.

Early diagnosis of AHO, timely surgical intervention, and rational anti-inflammatory therapy can improve treatment outcomes, reduce the likelihood of chronicization of the process, and decrease the frequency of generalized forms of the disease in pediatric patients.

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