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ETHIO-PATHOGENESIS OF POUND DESTROYMENT IN CHILDREN

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Abstract
Infectious diseases are one of the most serious problems because they are the most widespread and the leading cause of death. Although significant advances have been made over the past decades in addressing this problem, this statement is still true today. Sepsis is the leading cause of death in critical care patients. It develops in 750000 people each year and more than 210000 of them die. Progress in the study of the pathophysiology and genetic basis of the individual response to sepsis has changed the common understanding of this syndrome, and some therapeutic interventions used in recent years have demonstrated efficacy; however, the problem of intensive care is still quite acute and requires further research.

Keywords Sepsis, purulent-destructive diseases, paediatric age.

INTRODUCTION

In paediatric patients, against the background of antibiotic resistance of heterogeneous pus flora, morpho-functional immaturity of organs and tissues, insufficiency of local and general immunity, a high level of purulent-septic pathology with frequent outcome in sepsis remains [1]. That is why in February 2002 at the International Pediatric sepsis consensus conference IPSCC a modern classification of sepsis in paediatrics was adopted, allowing to start intensive therapy at the earliest possible time of the septic process [2].

In bacteriological cultures of biological media of patients with severe purulent-septic diseases, opportunistic microflora and fungi of the genus Candida, which are recognized as pathological agents and are not symbionts of the human body, are increasingly detected [3].

Children with purulent-septic pathology developed against the background of chronic somatic pathology have an unfavourable course of the disease with a high level of chronicity, disability and lethality.

Etiology of sepsis

Etiology of early neonatal sepsis

Streptococcus agalactiae (beta-haemolytic streptococcus group B)

Escherichia coli

Listeria monocytogenes

Etiology of late neonatal sepsis

Representatives of the family Enterobacteriaceae (E. coli, Klebsiella spp., Serratia marcescens, Proteus spp., Citrobacter diversus and others)

Rarely occurring: Pseudomonas aeruginosa, Flavobacterium meningosepticum, Staphylococcus aureus, Staphylococcus epidermidis, Enterococcus spp.and fungi of the genus Candida

Extremely rare: Streptococci belonging to serological groups A, D and E; and Streptococcus pneumoniae, which are highly sensitive to natural penicillins and all other betalactams

Pathogenesis of sepsis

The development of organ systemic damage in sepsis is primarily associated with uncontrolled spread of proinflammatory mediators of endogenous origin from the primary focus of infectious inflammation with subsequent activation under their influence of macrophages, neutrophils, lymphocytes and a number of other cells in other organs and tissues, with secondary release of similar endogenous substances, endothelial damage and reduced organ perfusion and oxygen delivery.

Dissemination of microorganisms may be absent at all or be short-lived and difficult to detect. However, even this slippage can trigger the release of proinflammatory cytokines at a distance from the focus. Bacterial exo- and endotoxins can also activate their hyperproduction from macrophages, lymphocytes, and endothelium.

The cumulative effects exerted by mediators form the syndrome of systemic inflammatory response.

Three main stages can be distinguished in its development.

Stage 1 - local production of cytokines in response to the action of microorganisms. Cytokines act in the focus of inflammation and in the territory of the reacting lymphoid organs, performing as a result a number of protective functions, participating in the processes of wound healing and protection of body cells from pathogenic microorganisms.

Stage 2 - release of small amounts of cytokines into the systemic bloodstream. Small amounts of mediators are able to activate macrophages, platelets, release of adhesion molecules from the endothelium, production of growth hormone. The developing acute-phase reaction is controlled by pro-inflammatory mediators (IL 1,6, 8, TNF (tumour necrosis factor) and anti-inflammatory cytokines (IL 4, 10,13, TGF (transforming growth factor)).

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In paediatric patients, against the background of antibiotic resistance of heterogeneous pus flora, morphofunctional immaturity of organs and tissues, insufficiency of local and general immunity,

a high level of purulent-septic pathology persists, with frequent outcome in sepsis [8]. That is why in February 2002 at the International Pediatric sepsis consensus conference IPSCC a modern classification of sepsis in paediatrics was adopted, allowing to start intensive therapy at the earliest possible time of the septic process.

In bacteriological cultures of biological media of patients with severe purulent-septic diseases, opportunistic microflora and fungi of the genus Candida, which are recognised as pathological agents and are not symbionts of the human body, are increasingly detected[9]. In children with purulent-septic pathology developed against the background of chronic somatic pathology, an unfavourable course of the disease with a high level of chronicity, disability and lethality is noted.

Rapidly changing pathomorphosis of suppurative infection with increasing antibiotic resistance is described, which makes it necessary to purposefully monitor suppurative flora in children with purulent-septic pathology, to create databases containing information about the features of pathogenic flora in patients from different profile departments of surgical hospitals [10].

In connection with the implementation of the National Health Care Project in Russia and the introduction of modern technologies for diagnosing the infectious process in children, it has become possible to create a register of the most virulent and frequently occurring purulent flora for doctors in each region. The need for timely identification of the causative agent of purulent infection in children is considered in practical healthcare together with the need for early diagnosis of septic manifestations, determination of the degree and nature of antibiotic sensitivity and antibiotic resistance. Prevention of septic complications of purulent infection in children, etiopathogenetic therapy, will reduce the frequency of surgical sepsis, its late diagnosis, reduce the chronicisation of the process, disability and mortality [12].

Purulent inflammatory skin lesions or pyoderma are divided into staphyloderma and streptoderma, although some of these forms can be caused by both microorganisms.

Vesiculopustulosis is a superficial neonatal staphyloderma. The process is localised in the mouths of eccrine sweat glands (staphylococcal periporitis).

Pseudofurunculosis (multiple abscesses) is a staphylococcal lesion of the entire eccrine sweat gland. It differs from a furuncle by the absence of a dense infiltrate and a characteristic necrotic "rod"

Pemphigus - epidemic neonatal vesicles - one of the types of staphylococcal skin lesions, occurring in benign and malignant forms.

- The benign form is characterised by the appearance at the end of the first week of life or later on the background of redness of vesicles and flaccid blisters 0.2-0.5 cm in size, filled with translucent fluid containing pus. Localisation - the lower abdomen, arms, legs, inguinal, cervical and other skin folds, less often - other parts of the body. Affected all layers of the skin up to the granular. More often pustules are multiple, rashes in groups, but may be single. Nikolsky's symptom is negative.

- Malignant form also develops at the end of the first week of life, but it is observed multiple flaccid blisters (flectenes) ranging in size from 0.5 to 2-3 and more cm in diameter, the skin between them sloughs off. Nikolsky's symptom is positive. Temperature above 38 degrees C, the condition is severe - in addition to lethargy, lack of appetite, intoxication phenomena are pronounced - pallor, respiratory rate, palpitations, vomiting. The disease is highly contagious and usually ends in septicaemia.

Pemphigus must be differentiated from bullous epidermolysis and syphilitic neonatal vesicles. Epidermolysis bullosa is a hereditary disease; blisters occur from birth on apparently healthy skin, mainly on protruding areas exposed to friction, filled with serous, serous-purulent or haemorrhagic contents. In dystrophic forms, scarring atrophy remains at the sites of the blisters.

Syphilitic vesicles can be detected at birth or appear in the first days of life. The vesicles are

localised on the palms and soles, and occasionally on other areas of the skin. At the base of the blisters is a specific infiltrate, so the blisters are surrounded by a reddish purple rim. Opening the blisters reveals an eroded surface.

Ritter's exfoliative dermatitis is a severe form of epidemic neonatal vesicular vesicular disease. It is caused by hospital strains of Staphylococcus aureus producing exotoxin exfoliatin. It begins at the end of the 1st and beginning of the 2nd week of life with reddening, wetting of the skin and formation of cracks, then flaccid blisters. Nikolsky's syndrome is positive. The child has the appearance of being burnt by boiling water. The process runs septic with a decrease in body weight, toxicosis, gastrointestinal disorders, anaemia, dysproteinaemia. Staphylococcal burnt skin syndrome (SBSS) occurs similarly to this disease, but in older children. Pemphigus must be differentiated from bullous epidermolysis and syphilitic neonatal vesicles. Epidermolysis bullosa is a hereditary disease; blisters occur from birth on apparently healthy skin, mainly on protruding areas exposed to friction, filled with serous, serous-purulent or haemorrhagic contents. In dystrophic forms, scarring atrophy remains at the sites of the blisters.

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The contagiousness of neonatal staphyloderma is high. Infection is possible in the presence of nosocomial infection, as well as in utero through the placental circulation.

Epidemic vesicles, epidermal vesicles, epidermal epidermolysis, and SSOC should be differentiated from congenital hereditary skin diseases with epidermal detachment (bullous epidermolysis, congenital ichthyosis), where the process affects all layers of the skin, including the basal layer, and non-purulent skin lesions (Stevens Johnson syndrome and Lyell's syndrome - toxic epidermal necrolysis), which have an allergic or toxic-allergic etiology. These diseases can occur at any age.

Impetigo is a form of pyoderma, a very contagious disease, it is caused by both streptococci and staphylococci. To the development of impetigo predispose eczema, pediculosis, scabies and fungal infection. Purulent blisters first appear on the face - around the mouth and nose - and very quickly spread to other parts of the body. The blisters dry up with the formation of crusts. Streptococcal impetigo differs from staphylococcal impetigo by the golden colour of the crusts. Streptococcus pyogenes is usually the causative agent of ordinary (non-bullous) impetigo, but in this case staphylococci can also cause superinfection.

Impetigo can occur as a primary infection (on clean skin) and as a secondary infection (on the background of another dermatosis). It is characterised by superficial pustules, which are covered with yellowish-brown (honey-coloured) crusts after opening. Staphylococci sometimes cause bullous impetigo.

Bullous impetigo is a superficial skin infection (tense blisters with clear contents) caused by Staphylococcus aureus. Under the action of Staphylococcus aureus exfoliatins, the epidermis is detached and blisters 1-2 cm in diameter are formed, with neutrophils and staphylococci in their contents. At first the rash appears around the nose and mouth, then quickly spreads to other parts of the body, blisters with purulent contents appear.

After opening the blisters, crusts form. Children under 5 years of age are particularly susceptible to the disease; dissemination of infection can lead to death.

One of the manifestations of impetigo is slit-like impetigo (angular stomatitis, zajeda).

Intertriginous streptoderma - occurs on the contiguous surfaces of large skin folds. In mild cases - a fairly common finding during preventive examinations, requiring nothing more than good hygiene of the folds and simple preparations based on zinc paste.

If poorly cared for, children often have mixed staphylococcal streptococcal intertrigo, characterised by a greater degree of hyperaemia and swelling of the affected skin.

So it is very important to view the folds carefully, especially in children with paratrophy, excessive sweating, diabetes mellitus.

Ecthyma is an ulcerative form of streptoderma, sometimes of mixed etiology. The disease is favoured by a decrease in the general reactivity of the body due to infections and severe general diseases.

Ecthyma develops as a result of penetration of streptococci deep into the skin, under the epidermis. In this regard, the formation is not a phlyctena, but a deep, on the background of the inflammatory infiltrate, a blister or epidermo-dermal pustule the size of a large pea or larger. The vesicle or pustule quickly shrivels into a serous-haemorrhagic or purulent-haemorrhagic crust, immersed in the thickness of the skin and bordered by a zone of dim hyperaemia. After removal of the crust, an ulcer with sheer edges is found, filled with granulation over time. In the natural course of ecthyma, granulations develop under the crust, which is gradually displaced from the ulcer, then falls off, leaving a scar surrounded by a border of hyperpigmentation.

The penetration of streptococci deep into the skin is caused by microtraumas, pruritic dermatoses. Ecthyma are usually multiple, often linear (along the course of scabies).

Rye is an acute recurrent infectious disease of the skin and subcutaneous tissue caused by streptococcus. In children, the course of the swelling in children is similar to that of adults.

In newborns, it starts most often in the navel or finger area. Very quickly, the rash migrates ("travelling rash", "travelling rash"). Erythema in newborns may not be as intense as in older children or adults, but swelling, infiltration of the skin and subcutaneous tissue is always present. The edges of the lesion have a zigzag contour, but the limiting roll is not expressed. In newborns can be and the so-called "white" rye, when there is no redness, and in the area of the lesion sometimes appear blisters, subcutaneous abscesses and necrosis.

The disease begins with high fever and chills. Simultaneously, dense areas of reddened skin appear, warm to the touch and with irregular edges.

Sometimes the beginning of the disease can be insidious - without fever or with a slight increase in temperature. Then the general condition progressively worsens, body temperature is kept at 39-40 ° C, the child becomes lethargic, refuses to breast, there are digestive disorders, phenomena of myocarditis, nephritis, meningitis, septicaemia develops. Infectious diseases of the umbilical wound run in the form of catarrhal omphalitis (wet umbilicus), purulent omphalitis - bacterial inflammation of the bottom of the umbilical wound, umbilical ring, subcutaneous fatty tissue around the umbilical vessels. Positive symptom of Krasnobaev.

Phlegmon of newborns - one of the most severe purulent-inflammatory diseases of the skin and subcutaneous fatty tissue of newborns, which begins with the appearance of a red spot on a small area of skin, usually dense to the touch, further in its development can be distinguished 4 stages. The initial stage is characterised by a rapid spread of the process deep into the subcutaneous fatty tissue, purulent dissolution of which outstrips the rate of skin change. Alternative-necrotic stage occurs after 1-1.5 days - the colour of the affected skin area

acquires a purple-blue tinge, softening occurs in the centre. The rejection stage is characterised by the deadness of the detached skin. In the repair stage, granulation develops, epithelialisation of the wound surface with subsequent scar formation.

Mastitis of newborns is a severe disease that begins against the background of physiological engorgement of the breast glands. Clinically manifested by an increase in one gland, its infiltration, redness, then there is fluctuation. Purulent discharge appears from the discharge ducts.

Osteomyelitis is an inflammation of the bone marrow that spreads to the compact and cancellous substance of the bone. Staphylococcus aureus enters the bone haematogenously from foci in the skin or nasopharynx, often the gate of infection cannot be identified. Osteomyelitis more often affects children, the usual localisation is metaphyses of long tubular bones, in newborns the epiphyses of the femur and humerus are more often affected. A predisposing factor is recent trauma to the limb. Initially, symptoms of intoxication with high fever and confusion predominate; later, there is severe pain in the affected limb, increasing with movement. In the early stage, the number of leukocytes in the blood is within normal limits or lower, later developing neutrophilic leukocytosis. Radiological changes usually appear 2-3 weeks from the onset of the disease, bone scintigraphy allows a diagnosis to be made more quickly. When pus breaks through from under the periosteum into soft tissues, skin hyperaemia and fluctuation appear.

Sepsis.

- Generalised form of purulent-inflammatory infection with an acyclic course caused by opportunistic bacterial microflora, the basis of pathogenesis of which is the rapid development of systemic (generalised) inflammatory response of the organism (SIR) in response to the primary septic focus.

- SIR is a general biological non-specific immunological reaction of the human body in response to the action of damaging endogenous or exogenous factor.

Systemic inflammatory reaction is characterised by an increase in the production of proinflammatory cytokines and, to a lesser extent, anti-inflammatory cytokines produced by virtually all cells of the human body, including immunocompetent cells. This orientation of the mediator response of SVR to a stimulus is labelled as SVR with a predominantly proinflammatory orientation.

The main clinical manifestations of adequate SVR include: increase in body temperature (hyperthermia), increase in the number of heart contractions (tachycardia), increase in the number of breaths (tachypnoea), hyperventilation of the lungs, increase in the number of leukocytes in the peripheral blood (leukocytosis), increase in the number of immature leukocytes (metamyelocytes, myelocytes, bacilli) in the peripheral blood.

Along with it, SVR with predominantly anti-inflammatory orientation of mediator response can be observed. One of the most severe and least curated is a mixed antagonistic reaction or dysregulation of SVR, the so-called "mediator storm", "mediator chaos". Rapid, inadequate to the action of the damaging factor, the development of SVR eventually contributes to induced apoptosis and in some cases necrosis of cells, which determines the damaging effect of SVR on the organism.

Regulation of SVR is carried out by activation of hypothalamic-pituitary-adrenal system, which normally provides the body's response to stress. In this regard, pronounced manifestations of SWO are accompanied by increased secretion of adrenocorticotropic hormone (ACTH) in the adenohypophysis with a corresponding stimulation of hormonal activity of the adrenal cortex and an increase in the level of cortisol in the blood.

Thus, sepsis is a systemic inflammatory response of the body to infection. In children of high school age and adults are sepsis syndrome (sepsis without PON), severe sepsis as a manifestation of sepsis with multi-organ failure, septic shock (sepsis with hypotension). Newborns and young children due to

physiological features of the immune system are prone to generalisation of the body's reactions to excessive exposure to damaging factors (infection), sepsis always occurs with multi-organ failure. In newborns, congenital sepsis is also isolated, which is divided into early, which occurred in the first 72 hours from birth and late, whose symptoms appeared on the 4th – 6th day, as well as acquired – with the onset after 7 days. Downstream, lightning –fast is isolated – 1 to 7 days, acute 4-8 weeks and prolonged for more than 8 weeks.

The frequency of sepsis.

According to T. E. Ivanovskaya, in 1978-1982, sepsis was detected in 4.5% of deceased children, among them newborns (92.3%) and, in particular, premature infants prevailed.

According to the results of autopsies conducted at the Leningrad Regional Children's Pathology and Anatomical Bureau (LODPAB), the incidence of sepsis in the structure of total infant mortality was 1% in 2000, 1.4% in 2001 and 1.9% in 2002, while the proportion of newborns among children who died from sepsis did not exceed 50%.

According to foreign authors, the incidence of sepsis among newborns ranges from 0.1 to 0.8%. Of particular concern are children in intensive care units and premature newborns, among whom the incidence of sepsis is on average 14% (from 8.6% among premature infants with gestational age from 31 to 38 weeks. up to 25% among premature infants with gestational age from 28 to 31 weeks).

In Uzbekistan, sepsis has occupied the 4th-5th place in the structure of neonatal mortality over the past decades, averaging 4-5 cases per 1,000 live births. The mortality rates from sepsis are also quite stable and amount to 30-40%.

Among older children, sepsis occupies 7th – 10th place in the mortality structure. Features of the status of newborns that cause increased sensitivity to infections:

1. Reduced chemotaxis, low bactericidal activity of phagocytes, low levels of properdin, C3, IgM, IgA
2. Low expression of HLA-2 class molecules→immaturity of presentation mechanisms, including by dendritic cells.
3. Low content of T-helpers ("naive" T-helpers of newborns are less mature than in adults)
4. Tendency to differentiate in the direction of T-x2→IL4, IL13→hypersensitivity to infections
5. Low production of IL12, IL15 in response to stimulation→ low production of IL2, yIF by T cells→low cellular cytotoxicity
6. Low production of TNFa, GM-CSF, M-CSF
7. The NK function is suppressed.
8. Low CD21 expression on neonatal B lymphocytes

High risk factors for neonatal sepsis:

- Death of previous children in the family from systemic bacterial infections before the age of 3 months (suspected hereditary immunodeficiency).
- Multiple abortions in the anamnesis, gestosis in the mother, which lasted more than 4 weeks.
- Clinically detected bacterial vaginosis in the mother during pregnancy and childbirth.
- Clinically pronounced bacterial infectious processes in the mother immediately before and during childbirth, including pyelonephritis, chorioamnionitis.
- Detection of streptococcus B or its antigens in the mother's birth canal.
- Anhydrous interval of more than 12 hours.
- The birth of a child with a very low and especially extremely low body weight.
- Tachycardia in the fetus without fever in the mother, hypotension, blood loss or the administration of drugs that cause tachycardia.
- Asphyxia at birth or other pathology that required intensive care and prolonged abstinence from enteral nutrition.
- Surgical operations, especially with extensive tissue injury.
- Congenital malformations with damaged skin,

burns.

- Type 1 SDR and pulmonary edema.
- Multi-day catheterization of the umbilical and central veins.
- Intrauterine infections.
- Multiple malformations or stigmas of dysembriogenesis

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