Treatment Tactics Of Sepsis In Purulent-Necrotic Diseases Of Soft Tissue

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ABSTRACT

The article analyzes the work carried out to create a new algorithm for the diagnosis and complex treatment of sepsis in patients with purulent-necrotic diseases of soft tissues. In the departments of purulent surgery of the ASMI clinic and the regional adult health center of the Andijan region, 28 patients with purulent-necrotic diseases of soft tissues underwent a study, where the tasks were set to create a new step-by-step algorithm for the diagnosis and complex treatment of sepsis in patients with purulent-necrotic diseases of soft tissues.

Keywords: Sepsis, purulent-necrotic diseases of soft tissues, facial carbuncle, neck adenophlegmon.

Sepsis is a systemic response to infection that can be life-threatening. However, it is not fully understood what effects sepsis has on organs at the cellular and molecular level. Using mouse models of sepsis, scientists from the United States and Japan characterized the pathogenesis of sepsis by assessing changes in gene expression in various organs over time. It turned out that almost the same effect on gene expression can be achieved by the combined action of tumor necrosis factor (TNFα) and one of the cytokines: IL-18, IFN-γ or IL-1β. Neutralizing these cytokines allowed mice given a lethal dose of lipopolysaccharide (LPS), which causes sepsis, to survive and protected them from a decrease in body temperature[16,17]. These experiments determined the importance of pro-inflammatory cytokines in the pathogenesis of sepsis and septic shock, which is confirmed by numerous data on an increase in the serum concentration of pro-inflammatory cytokines against the background of endotoxin administration and the ability of TNFα to induce the clinic of septic shock in experimental animals. At the same time, the ability to prevent the development of shock in animals with the introduction of neutralizing anti-TNFα antibodies, or soluble receptors and TNFα, allowed TNFα as a mediator that has an adverse toxic effect in severe bacterial infections [8,16].

Sepsis can occur acutely, sometimes almost instantly (when, in the absence of proper treatment, death occurs within a few hours or days), or chronically. Currently, the nature of the course of sepsis is changing significantly as a result of early antibiotic therapy. Despite the highest development of antibiotic therapy methods in developed countries, the incidence of sepsis tends to increase, and the mortality rate from this pathology averages 45-50%. In the USA, up to a million people fall ill with sepsis every year and 28-42% of these patients die; this figure also corresponds to the CIS countries - in Russia, Uzbekistan, Belarus, the mortality rate is up to 34-40%, and up to 90% of patients[1, 4, 5]. A revision of the opinion about the frequency of sepsis, which arose through the efforts of the French scientist Bonet, led to a

rapid increase in these indicators, in addition, it gave us the opportunity to take a broader look at this problem and use all possible treatment methods that are in our arsenal. New methods and comprehensive programs for the treatment of sepsis have been developed, especially in patients with purulent-septic complications in diabetes mellitus and purulent soft tissue infections [2, 6, 9].

The causative agents of sepsis can be pathogenic, opportunistic microorganisms: cocci (staphylococci, pneumococci, meningococci), Escherichia coli, Pseudomonas aeruginosa, Mycobacterium tuberculosis, Klebsiella, etc.; viruses of the herpetiform group, etc.; fungi such as Candida, Aspergillus[7, 9, 12, 15].

Generalization of infection is due to the predominance of the pathogen over the bacteriostatic capabilities of the body as a result of massive invasion (for example, a breakthrough of an abscess into the blood from an infected thrombus, when trying to squeeze out a boil, from an infected platelet mass and purulent melting of the infiltrate with phlegmon, etc.) or congenital or acquired decrease in immunity . Immune disorders preceding sepsis, as a rule, remain undetectable, except in cases of hematopoietic depression. However, sepsis does not arise as a result of immunity disorders in general, but as a result of a breakdown in one of its links, leading to a disruption in the production of antibodies, a decrease in phagocytic activity or the activity of lymphocyte production. Therefore, in most cases, sepsis is caused by a single pathogen, the reproduction of which is normally prevented by the immune response, i.e., a certain part of it that turns out to be genetically or acquired damaged; a change in pathogens during one disease is the exception, not the rule[9,11,15].The simultaneous coexistence of several pathogens, their change is observed in immunosuppression caused by the use of cytostatics, depression of hematopoiesis as a result of bone marrow aplasia or leukemic lesions, the action of intense insolation and tanning, which grossly suppress the immune response at several levels. Recurrent septic bacterial infection is observed with hereditary defects of complement C2, properdin and other factors of the complement system. With severe defects of immunity, so-called opportunistic infections often occur, caused by opportunistic flora and saprophytes. Approximately 10% of septic conditions are caused by a combination of pathogens. Polymicrobial sepsis occurs in disorders of the immune system associated with the absence of the spleen, disorders of the cellular (T-helper) immune system in AIDS[9, 12]. In adults and children without obvious causes of immunodeficiency (cytostatic, steroid therapy, etc.), most often the causative agent of sepsis is staphylococcus or pneumococcus, less often meningococcus[4,6, 13].Against the background of cytostatic therapy (especially in conditions of nosocomial infection), gram-negative microflora (intestinal or Pseudomonas aeruginosa, Proteus) plays an important role. The causative agent of sepsis from an infected thrombus of the aortic aneurysm, the inferior vena cava system (distal to the filter installed in it), the subclavian vein (if a catheter is left in it for a long time) can be staphylococci, Pseudomonas aeruginosa, and pneumococcus. Lymphopropiferative tumors and lymphophanulomatosis are accompanied by impaired antiviral immunity, which leads to generalized herpetic infections (chickenpox, herpes zoster, herpes simplex) up to sepsis. When neutrophilopoiesis is impaired as a result of hereditary neutropenia, recurrent staphylococcal infections occur, sometimes with the development of staphylococcal sepsis. With long-term use of steroid hormones, chronic or acute bacterial septic processes and tuberculous sepsis are possible[4, 6, 11].

After removal of the spleen (for any reason), a predisposition to septic conditions occurs, most often of meningococcal or pneumococcal etiology. The spleen is able to phagocytose non-opsonized, non-antibody-associated bacteria, encapsulated bacteria (particularly pneumococci and meningococci), whereas only well-opsonized bacteria are phagocytosed in the liver. In order for the liver to take over the function of destroying encapsulated, non-opsonized bacteria after removal of the spleen, large volumes of fresh plasma containing opsonins must be

administered. After splenectomy, the plasma content of opsonins such as properdin and tuftsin, produced mainly by the spleen and necessary for the phagocytosis of microorganisms by neutrophils, decreases. Properdin is also a factor that triggers an additional pathway of activation of the complement system (from the S3 component) - one of the important links of humoral immunity. Finally, the spleen produces mainly immunoglobulin M. The lack of all these factors contributes to the development of fatal post-splenectomy syndrome[12, 14, 15].

The formation of disseminated intravascular coagulation (DIC) syndrome plays an important role in the spread of infection. Massive purulent infection serves as the basis for tissue decay, the release of kinins and proteolytic enzymes into the blood, which contribute to impaired vascular permeability, stasis, and thrombus formation in the microcirculatory system. Multiple thromboses become an environment for the growth of microflora. In the development of DIC syndrome in sepsis, a significant role is played by endotoxin - lipopolysaccharide from the wall of Escherichia coli, capsular polysaccharide of pneumococcus, coagulase produced by the staphylococcal capsule, and other products of the bacterial cell. One of the most studied ways of excitation of DIC syndrome in sepsis is the activation of coagulation factor XII (Hageman factor). Influencing the vascular wall, endotoxin activates factor XII, which leads to increased coagulation, the formation of kallikrein and its precursors, and with them the activation of fibrinolysis (conversion of plasminogen into plasmin), the formation of kinins, and activation of the complement system. The accumulation of bradykinin leads to the development of shock - a drop in blood pressure, increased vascular permeability and microcirculation disorders[8].

DIC syndrome and shock are permanent complications of sepsis caused by gram-negative microorganisms, meningococcemia, acute pneumococcal and staphylococcal sepsis. The accumulation of kinins in sepsis and disseminated intravascular coagulation syndrome is facilitated by the depletion of enzymes such as kininase, a kallikrein inhibitor, usually found in the plasma of healthy individuals. Fibrinolysis, activated at the beginning of DIC, then sharply decreases due to the depletion of Hageman factor, kallikrein, and plasminogen itself. Inhibition of fibrinolysis is a characteristic sign of DIC syndrome complicating sepsis. When microthrombi become infected, DIC inevitably leads to severe multiorgan pathology, in the pathogenesis of which the infection itself plays a major role initially, and after 2-3 weeks, the pathology of immune complexes plays a major role. There is no clear boundary between septic organ pathology itself and immune complex syndromes after the elimination of the main bacterial and microthrombotic processes. Inactivated but non-phagized bacteria in blood clots can retain their activity and cause relapses of the disease, contributing to its transition into a chronic, often single-organ process. DIC syndrome is practically obligatory in the pathogenesis of sepsis; the disappearance of its laboratory and clinical signs indicates successful treatment[10].

Thrombocytopenia and decreased coagulation may not only be due to the consumption of platelets and clotting factors in blood clots. In connection with infection, the formation of antibodies, immune complexes, phagocytosis is activated (in particular, phagocytosis of neutrophils); at the same time, the enzymes elastase and chemotrypoin are released from neutrophils. An excess of these proteolytic enzymes contributes to tissue damage (in particular, the vascular wall), lysis of platelets and some coagulation factors, which, in turn, leads to the development of hemorrhagic syndrome and acute respiratory distress syndrome[9, 10].

The clinical picture of sepsis depends on the pathogen, the source of infection and the state of immunity. The onset of the disease can be violent with stunning chills, hyperthermia, myalgia, hemorrhagic or papular rash, or gradual with slowly increasing intoxication and a gradual increase in body temperature. Frequent but nonspecific signs of sepsis include enlarged spleen and liver, severe sweating after chills, severe weakness, physical inactivity, anorexia, and constipation. In the absence of

antibacterial therapy, sepsis usually ends in death from multiple disorders of all organs and systems. Thrombosis (especially of the veins of the lower extremities) in combination with hemorrhagic syndrome is characteristic[8, 9].

With adequate antibacterial therapy against the background of a decrease in temperature and a decrease in intoxication, 2-4 weeks from the onset of the disease, arthralgia appears (up to the development of polyarthritis), signs of glomerulonephritis (protein, red blood cells, casts in the urine), symptoms of polyserositis (pleural friction murmur, pericardial friction murmur) and myocarditis (tachycardia, gallop rhythm, transient systolic murmur at the apex or on the pulmonary artery, expansion of the boundaries of relative dullness of the heart, reduction or even negativity of the T wave and downward displacement of the ST segment mainly in the anterior thoracic leads). These symptoms, which occur against the background of improvement in the main indicators of the septic process and related to the pathology of immune complexes, should not be confused with signs of septic, bacterial pathology itself. The main manifestations of the latter occur in the first days of the disease and are characterized by all the signs of a purulent-septic process in a particular organ (purulent myocarditis, endocarditis, variants of septic damage to the lungs and kidneys). Massive antibacterial therapy and the fight against disseminated intravascular coagulation play a decisive role in the treatment of sepsis.

In severe disseminated intravascular coagulation syndrome and respiratory distress syndrome, multiple disc-shaped atelectasis and unstable polymorphic shadows in the lungs caused by interstitial edema are observed. Similar changes are observed in severe sepsis, regardless of the causative agent, and on single radiographs they are almost indistinguishable from pneumonia. However, shadows of an inflammatory nature are characterized by persistence, while shadows of interetitial edema are characterized by ephemerality. During auscultation of the lungs, interstitial edema may be indicated by silent fine rales and crepitus.

Diagnosis. The onset of any severe inflammatory process, accompanied by chills and high body temperature, at first glance, is not easy to distinguish from the onset of sepsis. However, a rapid rise in temperature to 39-40 (C, stunning chills, a general serious condition without pronounced monoorgan pathology, high leukocytosis with a band shift of up to 20-30%, clinical and laboratory signs of DIC syndrome are sufficient grounds for diagnosing sepsis and providing appropriate intensive care. It is extremely important to associate the diagnosis of sepsis with a specific pathogen, since bacteriostatic, antiviral or antifungal therapy is strictly specific. Establishing an etiological diagnosis is very difficult and not always possible. Blood culture and identification of specific bacterial antigens in 50-60% of cases do not answer the question about the nature of the pathogen in the first days of the disease, when specific treatment tactics are determined. Diagnosis of sepsis with identification of the nature of the pathogen involves daily blood cultures, regardless of negative responses in the first days of the disease and and ongoing antibacterial therapy, making the possibility of positive culture results less and less likely. An important role in identifying the causative agent of sepsis is played by the characteristics of the clinical picture of the disease and its first symptoms.

Staphylococcal sepsis is characterized by tremendous chills, high fever, and pain in the muscles and bones. Muscle pain can be almost morphine-like in intensity. Usually, in this case, single papules of a non-hemorrhagic nature can be seen on the skin, sometimes with the formation of tiny bubbles at the top of the papule. The general condition of the patients is serious, but there is no deep general depression, the consciousness is clear, the patients clearly talk about their feelings [3, 5, 12]. X-rays of the lungs often reveal multiple cloud-like shadows of almost the same size and density, which subsequently merge, forming uneven foci and zones of disintegration. At the beginning of the process, a dry cough is noted, then it becomes wet with the discharge of copious yellowish sputum. The resulting lung abscess can break into the pleura with the development of

empyema. Myalgia is often a consequence of microabscesses in the muscles. In the future, the formation of multiple phlegmons, foci of osteomyelitis, abscesses of the liver, kidneys and other organs is possible. Meningococcal sepsis is often characterized by a rapid onset with very severe intoxication and enteropathy, which within a few hours can lead to the development of shock; characterized by progressive workload and rapid loss of consciousness. In a number of patients, profuse polymorphic or monomorphic papular hemorrhagic rashes appear on the skin[6]. The observation of this rash becomes the basis for the assumption of the meningococcal nature of sepsis and the immediate administration of large doses of penicillin intravenously. Hemorrhagic rashes indicate severe disseminated intravascular coagulation syndrome; they involve not only the skin, but also the subcutaneous tissue, so the necrosis that develops in their place can be quite deep. A severe microthrombotic process contributes to the rapid formation of deep bedsores; it also underlies the clinical picture of glomerulonephritis (up to the development of anuria) and hepatitis (moderate increase in bilirubin levels, hypertransaminasemia against the background of liver enlargement). A severe complication of meningococcal sepsis is hemorrhage in both adrenal glands (due to DIC syndrome), causing the clinical picture of shock. Against the background of improvement of the patient's condition and normalization of temperature, meningococcal sepsis can be complicated by symmetrical gangrene (dry or wet) of the toes, and with insufficiently active therapy for DIC - more extensive gangrene requiring amputation of the limbs. The hemogram often shows hyperleukocytosis with a band shift of up to 20-40%. Clinical improvement and the dynamics of the blood picture may not coincide: leukocytosis and band shift sometimes persist against the background of normal body temperature, which, under the influence of powerful antibacterial therapy, decreases within several days, and multiple organ pathology and deep necrosis remain for several weeks. Along with high leukocytosis, thrombocytosis also occurs (sometimes up to 1 million or more platelets in 1 μl of blood), in particular, due to the activation of colony-stimulating factors of hematopoiesis under the influence of interleukin I produced by macrophages that process the pathogen antigen.

An increase in the level of interleukin I (as an endogenous pyrogen) is associated with fever, neutrophilia, proliferation of T-helper cells, and the production of antibodies.

Pneumococcal sepsis is characterized by the usual onset of sepsis: tremendous chills, a rise in body temperature to 39-40 °C. However, in these cases, severe intoxication occurs with adynamia, but without loss of consciousness and shock. Patients answer questions in monosyllables and quickly become exhausted. Skin rashes, myalgia, phlegmon and other manifestations of septicolemia are not characteristic of pneumococcal sepsis. The absence of pronounced organ pathology against the background of an extremely difficult general condition is indicative. A distinctive feature of the disease is often the persistence of a small percentage of eosinophils in the blood, while other types of bacterial sepsis are characterized by aneosinophilia. Leukocytosis in pneumococcal sepsis is moderate, but the band shift can be pronounced. Hemorrhagic syndrome is usually absent. The course of pneumococcal sepsis is not as violent as meningococcal sepsis (there may be exceptions!), but improvement under the influence of antibacterial therapy also does not occur as quickly as with meningococcal sepsis.

The first signs of adequacy of treatment are a decrease in weakness, the disappearance of chills, and the appearance of appetite, although body temperature may remain elevated for several days, only showing a downward trend. Underestimating the subjective improvement rate is very dangerous, since the absence of laboratory signs of improvement against the background of persistent febrile temperature may create a misconception about the ineffectiveness of antibacterial therapy, while it is penicillin (and not broad-spectrum antibiotics) that is indicated for pneumococcal sepsis throughout the entire disease, lasting many weeks, and

sometimes months (for example, with an infected blood clot in a large vessel). Premature discontinuation of penicillin is indicated by relapse of fever, deterioration of general condition, and resumption of chills. All this requires not changing the antibiotic, but returning to treatment with penicillin in large doses (the usual doses of penicillin for pneumococcal and meningococcal sepsis for adults, amounting to 20,000,000-24,000,000 units/day, should not be significantly increased, since at doses of 30,000 000-40,000,000 units/day, severe hemolysis, pancytolysis or hemorrhagic syndrome caused by platelet disaggregation may develop). A feature of pneumococcal sepsis is the low severity or complete absence of clear organ manifestations of the disease, although this type of sepsis, like others, can be complicated by an immune complex syndrome of one kind or another.

Sepsis caused by gram-negative microorganisms (Escherichia coli, Proteus, Pseudomonas aeruginosa) occurs either in the presence of large entrance gates (postoperative abscesses in the abdominal cavity, abscesses in the pelvis after gynecological interventions, an infected blood clot in the aneurysmal dilated aorta), or with severe suppression of the immune system (cytostatic therapy, lymphoproliferative tumors of the blood system, acute leukemia). In the diagnosis of these forms of sepsis, the most important role is played by bacteriological analysis - culture of blood, urine, sputum, bacterioscopy of discharge from wounds and prints of wound surfaces. One of the manifestations of pseudomonas sepsis (sometimes staphylococcal) is necrotic hemorrhage: rashes (sometimes isolated) of a rich dark red, almost black color, surrounded by a dark red shaft and rising above the skin surface. These sometimes painful (especially at first) formations gradually grow; body temperature remains febrile; daughter screenings occur in other areas of the skin and in internal organs (discovered during pathological examination). Necrotic hemorrhages are practically resistant to conventional types of antibacterial therapy due to the dense thrombotic shaft surrounding them, but inside these formations there is an active pathogenic flora. The mechanism of formation of necrotic hemorrhages is apparently close to the pathogenesis of noma and gangrene, in which the decisive role is played by the focus of necrosis, surrounded by a gradually expanding zone of thrombosis: the infection provokes thrombus formation, the blood clots constitute a nutrient medium for the growth of microorganisms and block the flow of antibiotics into the necrotic focus. The process turns out to be self-sustaining due to the depletion of the fibrinolysis system. The main means of breaking this vicious circle is the local use of dimethyl sulfoxide with an antibiotic against the background of conventional antibacterial therapy and increasing the fibrinolytic activity of the blood using massive transfusions of fresh frozen plasma.

Sepsis caused by Pseudomonas aeruginosa, against the background of immunosuppression (with cytostatic therapy, tumors of the blood system) is characterized by extreme severity and rapidly developing shock. The same sepsis, which arose with normal blood counts as a result of a breakthrough of infection from an infected thrombus, can proceed torpidly; the patient's condition worsens gradually; Antibacterial therapy has some positive, but unstable effect. In general, sepsis caused by gram-negative microflora, without an entrance gate, with a normal composition of leukocytes and without taking immunosuppressants, is very unlikely. For sepsis, rapid development of shock (sometimes literally within 2-3 hours from the onset of febrile temperature).

Diagnosis of sepsis caused by gram-negative microflora in hematology and oncology hospitals often falls on the shoulders of the doctor on duty, who, however, before starting antibacterial therapy (simultaneously with the first administration of the antibiotic), must take blood for culture in any sterile sealable container and place it in a thermostat at 37 "C. In the diagnosis of sepsis, one should not neglect any sign that allows one to assess the nature of the pathogenic flora. So, if the source of sepsis is some kind of suppurating cavity (pleural empyema, interintestinal abscess, etc.), then

they try to determine the nature of the microflora to a certain extent by smell[9,11].

A clinical sign of a change in pathogen against the background of the current septic process is a change in the clinical picture of the disease: against the background of progressive improvement in the condition, the temperature suddenly rises, chills appear, leukocytosis increases and a pronounced band shift is detected again. Similar changes are also possible due to the formation of a septicopyemic cavity. Therefore, simultaneously with the search for a new pathogen, it is necessary by all available means to exclude the presence of an abscess of internal organs (intrahepatic abscess, renal carbuncle, etc.).

In **order to create** a new algorithm for complex diagnosis and treatment of sepsis in patients with purulent-necrotic diseases of soft tissues, we conducted this study.

We were given the following tasks:

- to create a complex of therapeutic measures for surgical sepsis in patients with purulent-necrotic diseases of soft tissues;

- to develop stages of algorithms for the use of complex treatment of sepsis in these diseases.

Material and methods. In the departments of purulent surgery of the ASMI clinic and the regional adult health center of the Andijan region, the present studies were carried out on 28 patients with purulent-necrotic diseases of soft tissues. These patients were –

carbuncles – 10 patients;

necrotic phlegmon of soft tissues – 10 patients;

Fournier's disease - purulent-necrotic melting of the tissues of the scrotum and perineum - 4 patients;

putrefactive processes in postoperative wounds - 4 patients.

These patients were divided into three groups:

6 patients received traditional treatment (retrospective group);

10 patients received complex treatment;

12 patients received complex treatment with step-by-step rehabilitation.

The patients underwent general clinical examinations of blood and urine, studies of biochemical parameters, the dynamics of daily changes in blood sugar, determination of pathogens of purulent-necrotic processes by culture and wound discharge, as well as the leukocyte intoxication index (IL), phagocytic activity of leukocytes (FAL)[1,2].

Laboratory studies before treatment showed that all patients had secondary, toxic anemia from moderate to severe severity, hypoproteinemia, hypoalbuminemia, dysproteinemia of the globular fraction and proteins due to hypergammaglobinemia, minor toxic hyperglycemia from 6 to 9 mmol/l., a significant decrease indicators FAN, IL.

Patients who received traditional treatment, who received standard short-term preoperative therapy, including intravenous and intramuscular administration of antibiotics, detoxification agents and vitamin therapy, second group, 10 (35,7%) patients, along with antibiotic therapy, received conservative infusion therapy 5-8 days before surgery, including antiplatelet, protein, antioxidant, membrane-tropic, immunocorrective, detoxification, and vitamin therapy. Membranotropic therapy consisted of tocopherol acetate (200-300 mg intramuscularly), Essentiale (10.0 ml per 200 ml of 5% glucose solution intravenously), rheosorbilate 250.0 each, ascorbic acid (10.0 ml of 5% glucose solution 2-3 times intravenously with insulin). Detoxification therapy included infusions of a 5% glucose solution with insulin and saline solutions, and forced diuresis. In the third group, curantil (0.15 g/day), trental (0.5 g/day), rheopolyglucin intravenous drops (400 ml/day) in courses of 3-4 days were used for disaggregate therapy. Correction of immunological disorders was carried out by the introduction of immunoglobulin, T-activin, and in the third group, specially highly immunized anti-staphylococcal plasma and gamma globulin were used. Vitamin therapy was carried out with the introduction of vitamin C, vitamins A and B, in both groups (control and main) Surgical treatment of the primary purulent focus of sepsis, as well as some septicopyemic secondary lesions and external drainage of purulent cavities were performed. Patients in both groups subsequently underwent rehabilitation measures to restore impaired functions of organs and systems.

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Conclusions:

1. For surgical sepsis in patients with purulent-necrotic diseases of soft tissues, an individual approach to the treatment of each patient is necessary.

2. When treating surgical sepsis in patients with purulent-necrotic diseases of soft tissues, antibiotic therapy should be carried out until the end of treatment in high doses of two types of synergistic antibiotics.

3. When treating surgical sepsis in patients with purulent-necrotic diseases of soft tissues, it is necessary to develop stages of algorithms for the use of complex treatment.

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