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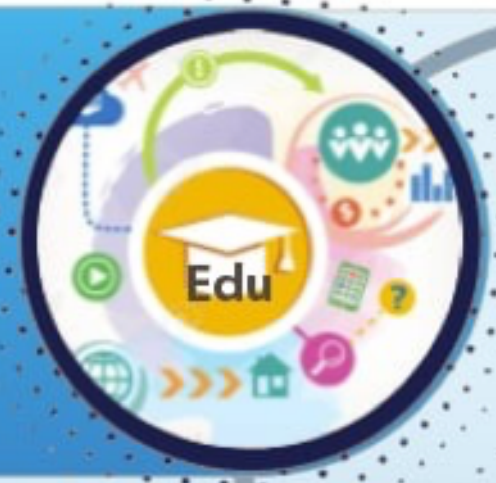

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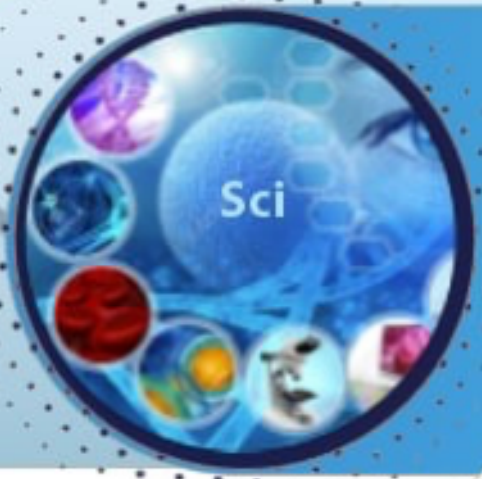

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Review Article

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Fundamental Issues of Purulent Mediastinitis

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ABSTRACT

This review article presents information on the pathological anatomy of purulent mediastinitis, on the specificity of the pathogenesis of purulent mediastinitis, in particular the local process and the mechanisms of development of its complications. Modern information about the system of general and anti-infective resistance of the organism in the development of purulent mediastinitis is described in detail. It is concluded that purulent mediastinitis is a severe pathological process affecting vital organs and leading to the development of multiple organ dysfunction or insufficiency. It is lesions of this nature that act as the basis for the development of severe sepsis and septic shock, leading to death.

Keywords: Purulent mediastinitis, pathomorphology and pathogenesis of purulent mediastinitis

Morphological changes in fiber in the development of purulent mediastinitis occur in stages, as in other forms of purulent surgical infection of soft tissues. However, this staging is not absolute, and it is often more correct to speak from the very beginning about the form, and not about the stage of the process. In addition, the morphology of the disease is significantly influenced by the mechanisms of primary alteration and the biological characteristics of the causative microorganisms [1,47].

Acute inflammation of the mediastinal lymph nodes is always secondary to the underlying disease of the chest or neck. Macroscopically, the lymph nodes sometimes increase to the size of a walnut, juicy and hyperemic on the incision; Microscopically, small ulcers can be detected in them. In principle, complete purulent melting of these lymph nodes is possible, followed by the development of a nonspecific purulent process in the mediastinal tissue, and it is very likely that this is the case with the lymphogenous occurrence of secondary mediastinitis [2,49].

In the pulmonary form of anthrax, hemorrhagic necrosis of the mediastinal lymph nodes develops with the blockade of the lymph flow pathways and the subsequent development of hemorrhagic mediastinitis [3,51].

Serous inflammation of mediastinal tissue is characterized by the development of impregnation of adipose tissue with exudate with a small amount of cellular elements - neutrophils and lymphocytes. With perforation of the wall of the esophagus, mediastinal emphysema develops simultaneously with inflammation, the severity of which depends on the mechanism of injury. The influence of mediastinal emphysema on the course of the purulent process is assessed by different authors in different ways - some believe that it has no independent significance, others assign it almost a leading role [4,53].

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The transition of serous inflammation to purulent inflammation can occur at different times, depending on the mechanism of mediastinal infection. With perforations of the esophagus, an average period of 6 - 8 hours is considered, but it is significantly reduced, for example, with an extensive rupture when trying to eliminate a long-existing "meat" blockage and, on the contrary, can increase, for example, with so-called "point" perforations and in cases where perforation occurs during anesthesia [5,55].

The macromorphological manifestation of serous inflammation is a dense infiltrate, in the center of which purulent melting subsequently occurs. Infiltrates from the surface are yellow. Microscopically there is vasodilation, cellular infiltration, the presence of fibrin deposits and fiber sclerosis [6,57].

With the progression of the process, the mediastinal fiber acquires a dirty gray, sometimes brownish-brown color. Delimited accumulations of pus (abscesses) are observed relatively rarely, purulent impregnation of tissues occurs more often, that is, phlegmons develop, which can spread in isolation in the posterior or anterior mediastinum or be total [7,59].

Mediastinal abscesses are mainly localized in the upper parts of the anterior mediastinum and in the root zone of the lungs, can be single and multiple. As a rule, a strong capsule is not formed and there is a tendency to break through ulcers [8,61].

In addition, in odontogenic and tonsilogenic mediastinitis, the spread of the purulent process often occurs as necrotizing fasciitis, which practically negates the protective role of connective tissue formations of the neck and mediastinum [9,63].

With adequate treatment and a favorable course, the process enters the repair phase, which can last up to 3 months. At the same time, in the emerging scar tissue, it is possible to preserve foci of "dormant" infection (especially around non-removed foreign bodies), which can lead to a relapse of the acute purulent process. Such recurrent purulent mediastinitis occurs, as a rule, in a delimited form. In addition, in some cases, for reasons that are not entirely clear, acute inflammation turns into chronic inflammation [10,48].

The pathogenesis of purulent mediastinitis significantly distinguishes this disease from other forms of purulent surgical infection. The main features of the local process are as follows [11,50].

As already noted, the location of fascial formations of the mediastinum and neck does not actually prevent the spread of pus through the anterior and posterior mediastinum. At the same time, the transition of the process from the anterior parts of the mediastinum to the posterior and vice versa usually does not occur, although the individual variability in the structure of the mediastinal fascia can lead to the most bizarre spread of purulent streaks. However, these provisions are valid mainly in primary purulent mediastinitis [12,52].

In secondary purulent mediastinitis, when the primary purulent focus is localized in the organs and cellular spaces of the head and neck, there are at least two important additional circumstances. The first is the anatomical possibility of the spread of pus, both in the anterior and posterior mediastinum from the pharyngeal and pharyngeal tissue spaces of the neck. The second is the predominance of secondary purulent mediastinitis among the causative agents highly pathogenic microbes, the distinctive features of which are tropism for connective tissue and high histolytic activity of waste products. Due to the latter, secondary purulent mediastinitis often proceeds according to the type of necrotizing fasciitis, in which the protective role of fascial formations is negated, and the process in the mediastinum often acquires a total character from the very beginning [13,54].

A certain importance in the development of purulent mediastinitis belongs to mediastinal emphysema, which occurs in primary mediastinitis due to suction of air from the external environment, and in secondary as a result of the vital activity of the gas-forming flora. The delamination of fiber by gas can undoubtedly contribute to a more rapid progression of the purulent process [14,56].

The high histolytic activity of wound contents is also observed in primary purulent mediastinitis developing as a result of perforation of the esophagus. This is explained (in addition to the biological characteristics of pathogens) when the damage is localized in the lower parts by throwing acidic gastric contents into the esophagus and from it into the mediastinum, and when the damage is localized in the upper sections, by entering the purulent cavity of saliva [15,58].

Due to the high histolytic activity of purulent exudate in purulent mediastinitis, cases of purulent destruction of the walls of organs and formations of the mediastinum and neck - esophagus, trachea, mediastinal pleura with the formation of various fistulas; aorta, large arteries and veins with the development of arrosive bleeding; diaphragm with the formation of abdominal abscesses and diffuse peritonitis is often observed [16,60].

In addition, the rapid development of purulent-necrotic changes in mediastinal tissue is facilitated by signifi-

cant mediastinal mobility due to the functioning of the heart, lungs, and large vessels [17,62].

Lesions of internal organs and systems in purulent mediastinitis also have a number of significant qualitative and quantitative differences from those in other forms of acute purulent surgical infection [1,18].

The location of many lymphatic formations in the mediastinum and the place of direct fusion of the lymphatic and venous systems in combination with negative pressure cause a high absorption capacity of mediastinal tissue, which in turn causes a rapid increase in intoxication in common forms of purulent mediastinitis [6,19].

Also, due to their location, vital organs (heart and lungs) and large nerve trunks (recurrent laryngeal nerves, phrenic and vagus nerves, etc.) are affected not only by intoxication but also directly (throughout) involved in the inflammatory process [11,20].

Inflammatory fiber infiltration can lead to compression of the unpaired and semi-unpaired veins located in the mediastinum, which in turn leads to impaired venous outflow from the heart and pleura [16,21].

All of the above features lead to a high incidence of various lesions of the heart, lungs, and other organs, and very often multiple organ failure syndrome in patients with purulent mediastinitis [21,22].

The defeat of the cardiovascular system of toxic genesis in one form or another is observed in all cases without exception. Almost all patients have severe tachycardia. In the early stages of the disease, transient bradycardia may sometimes occur for a short time [23,26].

As a rule, with purulent mediastinitis, the contractile function of the myocardium is also impaired. This is objectively confirmed by electrocardiography and ultrasound scanning. The severity of myocardial changes is also evidenced by the detection of a significant increase (two or more times the norm) in patients with purulent mediastinitis of the level of the first and second fractions of lactate dehydrogenase. The incidence of endocarditis and myocarditis in secondary purulent mediastinitis reaches 56%. There are isolated reports of the development of myocardial infarction in patients with purulent mediastinitis [24,31].

The causes of damage to the cardiovascular system, along with the effect of resorbable toxic products on the walls of blood vessels and myocardium, is a violation of the venous outflow from the heart due to compression of the unpaired and semi-unpaired veins by inflammatory infiltrate. The latter circumstance not only leads to a violation of one's own cardiac perfusion but is also the initiating moment in the development of effusion pericarditis. The accumulation of fluid in the pericardial cavity in purulent mediastinitis can be very significant - up to the development of cardiac tamponade [25,36].

A complex set of pathological processes developing in purulent mediastinitis in almost all patients leads to serious lesions of organs that provide the function of external respiration [26,41].

Compression of the upper respiratory tract by inflammatory infiltrate, coupled with an attached secondary lesion of the mucous membrane, can lead to stenosis of the larynx and trachea. These phenomena usually increase at the time of the provision of operational assistance, as a result of which it is often necessary to impose a tracheostomy [27,46].

A number of reasons explain the high incidence of pleurisy in patients with purulent mediastinitis. The mediastinal pleura is involved in the inflammatory process by contact, it is possible to spread the lesion to other parts along or due to lymphogenous and hematogenous infection. In cases of simultaneous perforation of the esophagus and mediastinal pleura, purulent pleurisy develops as a result of direct infection of the pleural cavity. Finally, in some cases, the initiating role is played by the above-mentioned compression of the vessels that ensure the outflow of blood and lymph from the pleura. Developing at the same time, pleurisy is bilateral and rapidly progressing [28,51].

The development of pneumonia, the incidence of which in patients with secondary purulent mediastinitis reaches 84%, and lung abscesses is mainly due to aspiration. The probability of the latter naturally increases with the occurrence of various respiratory fistulas. A significant (up to 60% of normal) decrease in the content of alpha-1-antitrypsin, a natural inhibitor of both endogenous and exogenous proteases, observed in patients with mediastinitis, also plays a role. Favorable conditions for the development of pneumonia are created due to the restriction of respiratory excursions of the lungs and hypostasis due to the patient's prolonged stay in a forced position, secondary inflammatory lesions of the intercostal muscles and diaphragm and a violation of the chest's own hemodynamics [29,56].

All the above leads in some cases to the development of respiratory failure, which sometimes makes it necessary to resort to long-term respiratory support. In a particularly severe course of the disease, the development of respiratory distress syndrome, usually fatal, has been described [30,61].

Changes in indicators characterizing nonspecific resistance in patients with mediastinitis are common for

severe forms of purulent surgical infection. In the blood, leukocytosis, a shift of the leukocyte formula to the left, toxic granularity of neutrophils, lymphomonocytopenia, and an increase in ESR are detected. Indicators characterizing the functions of the mononuclear phagocytic system are reduced [31,62].

The state of the immune system in most patients can be characterized as a pronounced secondary immunodeficiency. A decrease in the total number of lymphocytes, populations of T- and B-lymphocytes and a subpopulation of T-suppressors with an increase in the number of zero cells and an imbalance of immunoglobulins are characteristic. According to a number of authors, there is a marked decrease in the absolute and relative number of T-lymphocytes and a very moderate B-lymphocytes, a decrease in the number of immunoglobulins of classes G and M, with an unchanged amount of immunoglobulins A. The severity of changes in the immune system correlates with the severity of the condition and the prevalence of the affected area [32,63].

Studies of the hemostasis system have shown that patients with acute mediastinitis against the background of severe endotheliosis develop DIC syndrome with activation and subsequent depletion of components of the blood coagulation and anticoagulation system, antithrombin III, prekallikrein and plasminogen, blockade of microcirculation and increased fibrinolysis in the focus of inflammation. As the severity of the disease increased, an increase in the orthophenanthroline test was noted, which made it possible to quantify the degree of thrombinemia, an increase in the "depression" of the fibrinolytic system according to dependent fibrinolysis and streptokinase-induced euglobulin lysis. In severe purulent mediastinitis, activation of the kallikrein-kinin system is noted, which is manifested by an increase in the level of kallikrein and a decrease in prekallikrein. Thus, the dynamics of changes in hemostasis in purulent mediastinitis corresponds to the phase of development of disseminated intravascular coagulation syndrome [33].

In the early stages of the disease, hypercoagulable phenomena usually predominate. Examinationof patients with purulent mediastinitis prior to surgery with thromboelastography revealed a reduction in coagulation time, a decrease in clot retraction time, and a significant increase in fibrinogen content [34].

Clinically, violations of the antisystem of intravascular coagulation are manifested in severe cases of the disease with a detailed picture of DIC syndrome with massive hemorrhages. In addition, there are cases of local venous thrombosis, such as jugular veins [35].

It should be noted the difficulties of laboratory diagnosis of DIC syndrome in purulent mediastinitis, which are due to the fact that coagulographic studies are usually performed against the background of massive infusion therapy, blood and plasma transfusions [36].

There are three main types of disorders of the functions of the organs of the gastrointestinal tract that occur in purulent mediastinitis. In terms of frequency of occurrence, these are liver dysfunctions, acute erosions and ulcers of the stomach, intestines, and enteral insufficiency syndrome [37].

Violation of protein-synthetic function is manifested by a decrease in the level of total protein and albumin, dysproteinemia. Evidence of severe damage to the liver parenchyma is a significant (2-3 times) increase in the blood of patients with severe forms of purulent mediastinitis of the content of enzymes of cytoplasmic membranes of hepatocytes - aspartate aminotransferase, alkaline phosphatase; especially hepatospecific - histidase and urocaninase. However, the most obvious and permanent violations of the detoxification function of the liver - hyperbilirubinemia is observed in 70% of patients with purulent mediastinitis [38].

Acute erosion and ulcers develop in 75-80% of patients with purulent mediastinitis. The most likely mechanism of their occurrence is stressful. Some patients have complications of acute ulcers in the form of bleeding. The latter can sometimes be fatal [39].

Clinically pronounced enteral insufficiency syndrome, manifested by severe and persistent paresis of the gastrointestinal tract, is rarely observed in lower and total mediastinitis, always in combination with other severe complications. The most likely cause of this complication is a severe inflammatory lesion of the vagus nerves - "inflammatory vagotomy". Toxic damage to the nervous apparatus of the intestine may also be of some importance. The phenomena of dynamic intestinal obstruction in purulent mediastinitis may also be a consequence of the development of retroperitoneal phlegmon [40].

Renal function in patients with purulent mediastinitis is usually impaired quite late, in the terminal stages of the disease (of course, if patients did not have a pre-existing pathology of the urinary system). Oliguria, observed in severe cases of the disease, is due to low renal perfusion due to hypotension, which develops as a manifestation of infectious-toxic shock. The development of acute renal failure in Purulent mediastinitis is quite rare [41].

The manifestation of renal failure in the early stages of the disease is an unfavourable prognostic sign [42].

Disorders of the neuropsychiatric status in patients with purulent mediastinitis are not uncommon, indicating a significant severity of the disease [43].

The excitement observed sometimes in the first days of the disease, in most cases is replaced by deep prolonged inhibition. Among the various psychopathological symptoms of reactive genesis, asthenic disorders prevail. Less commonly, delirious, amentive and hallucinatory-delusional disorders are noted. With an increase in the severity of the condition, depressive manifestations, at first close to purely neurotic, acquire a dysphoric color, combined with other psychoorganic symptoms [44].

Premorbid personality traits (alcoholism, neurosis, psychopathy, traumatic brain injuries) play a significant role in the formation of psychogenic disorders [45].

The course of the widespread purulent process in the mediastinal tissue is inevitably accompanied by significant losses of fluid, electrolytes and protein, the presence of negative energy and plastic balances. In patients with purulent mediastinitis, increasing hypovolemia, a significant decrease in the volume of circulating protein, hyperglobulinemia, and serious electrolyte disorders are detected. Difficulties in the correction of nutritional disorders are due to the unusual duration of the severe period of the disease for most other forms of purulent surgical infection. Especially serious problems arise in cases where the possibility of feeding through the mouth is excluded - in the presence of damage to the esophagus, phlegmon of the bottom of the oral cavity and the absence of a gastrostomy or nasogastric tube, as well as in the development of enteral insufficiency syndrome in a patient [46].

Thus, from the presented material it can be concluded that purulent mediastinitis is a severe pathological process affecting vital organs and leading to the development of multiple organ dysfunction or insufficiency. It is lesions of this nature that act as the basis for the development of severe sepsis and septic shock, leading to death.

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YIRINGLI MEDIASTINITNING FUNDAMENTAL ASOSLARI

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ABSTRAKT

Ushbu maqolada yiringli mediastinitning patologik anatomiyasi, yiringli mediastinit patogenezining o'ziga xosligi, xususan, mahalliy jarayon va uning asoratlarini rivojlanish mexanizmlari haqida yozilgan. Yiringli mediastinit rivojlanishida organizmning umumiy va infektsiyaga qarshi chidamliligi tizimi to'g'risidagi zamonaviy ma'lumotlar batafsil bayon etilgan. Yiringli mediastinit hayotiy organlarga ta'sir qiluvchi og'ir patologik jarayon va ko'p a'zolar disfunksiyasi yoki etishmovchiligi rivojlanishiga olib keladi. Bu tabiatning zararlanishi og'ir sepsis va septik shokning rivojlanishiga asos bo'lib, o'limga olib keladi.

Kalit so'zlar: yiringli mediastinit, patomorfologiya va yiringli mediastinit patogenezi

ФУНДАМЕНТАЛЬНЫЕ ВОПРОСЫ ГНОЙНЫХ МЕДИАСТИНИТОВ

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Абстракт

В данной обзорной статье представлены сведения относительно патологической анатомии гнойных медиастинитов, особенности патогенеза гнойных медиастинитов, в частности местного процесса и механизмов развития его осложнений. Подробно изложены современные сведения о системе общей и противоинфекционной резистентности организма при развитии гнойных медиастинитов. Сделано заключение, что гнойные медиастиниты – это тяжелые патологические процессы поражающие жизненно-важные органы и приводящие к развитию полиорганной дисфункции или недостаточности. Именно поражения такого характера выступает в качестве основы развития тяжелого сепсиса и септического шока, приводящие к летальному исходу.

Ключевые слова: гнойный медиастинит, патоморфология и патогенез гнойного медиастинита