

# Role of Endogenous Intoxication in Development and Course of Paratonsillar Abscess

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**Abstract** The most dangerous local complication of acute and exacerbations of chronic inflammatory diseases of the pharynx is the formation of abscesses in the structure of the cellular spaces of the neck. Peritonsillar abscess (PTA) is the most common abscessing lesion. In turn, parapharyngeal abscess (PFA) is often found in patients with PTA as its complication. The purpose of this review is to study the role of endogenous intoxication in the development and course of peritonsillar abscess. The review material consisted of scientific publications over the past 10 years, published in the international databases E-library, Scopus and Web of Science. Thus, it follows that the study of issues of modern views on the etiopathogenesis of peritonsillar abscess is relevant and diverse, in the development and course of peritonsillar abscess there is endogenous intoxication.

**Keywords** Peritonsillar abscess, Pathogenesis, Endogenous intoxication, Inflammation

## 1. Introduction

Peritonsillar abscess (PTA) is a complication of acute tonsillitis and occurs in 2.6% of patients and is determined by a number of clinical features, such as sore throat, trismus of the masticatory muscles, asymmetry of the pharynx [2,11,15,17]. However, many issues related to the etiology, clinical picture, laboratory and differential diagnostics, therapy and surgical treatment of PTA remain unresolved to this day [1,5,7,10,14,16,19]. The lack of generally accepted terminology, as well as standardized approaches to the management of patients with peritonsillar abscesses is due to multiple problems, since this type of complication of acute tonsillitis is the subject of study in several areas of medicine - otolaryngology, pediatrics, microbiology, immunology. Further analysis of the etiology and pathogenesis of peritonsillar abscess carries a solution to one of the main problems at the present stage: excessive and non-systemic use of antibacterial drugs, especially since the problem of microorganism resistance to antibiotics is becoming increasingly widespread; the absence of clear principles of diagnosis, as well as developed regulations for treatment and prevention [2,6,9,13,16].

## 2. Results

The fundamental factor in the treatment of peritonsillar abscesses is that the administration of antibacterial drugs from the first day of treatment is a necessary condition, but due to

the difficulty of diagnosis, the absence of a clear treatment algorithm, even in a hospital setting, leads to an increase in the duration of this disease [3,4,8,12,15,18].

**The purpose of this review** is to study the role of endogenous intoxication in the development and course of peritonsillar abscess.

**The review material** included scientific publications over the past 10 years, published in the international databases E-library, Scopus and Web of Science.

**Results of the review and their discussion.** Peritonsillar abscess often occurs against the background of angina or a few days after its completion. First, hyperemia of the vessels, a violation of their permeability appear in the peritonsillar tissue, edema develops and small-cell infiltration occurs. Absorption of bacterial toxins and products of the inflammatory reaction causes fever, intoxication, and changes in blood composition. Compression of the nerve trunks causes severe pain, disorders of the central and autonomic nervous systems. An abscess is formed as a result of purulent melting of the paratonsillar infiltrate. The disease begins with pain when swallowing, which gradually becomes constant and sharply increases when trying to swallow saliva, radiating to the ear and teeth. Headache, general fatigue, body temperature rises to febrile numbers, patients refuse food and drink. Pronounced trismus of the masticatory muscles occurs. Speech becomes slurred and nasal. As a result of inflammation of the pharyngeal muscles and cervical lymphadenitis, a painful reaction occurs when turning the head, the patient holds his head to the side and turns it, if necessary, together with the whole body.

The general condition of the patient becomes severe not only because there is purulent inflammation in the pharynx and intoxication, but also due to excruciating pain in the

throat, sleep disturbances, inability to swallow liquid and starvation.

According to the routes of infection penetration into the peritonsillar tissue, the following variants of PTA can be distinguished:

1. Tonsillogenic.
2. Odontogenic (the source of infection is carious teeth).
3. Traumatic.
4. Hematogenous (with generalized bacterial infections).

There are several localizations of paratonsillar abscess:

1. Supratonsillar localization (anteroposterior), occurs in more than 70% of cases (the abscess is localized between the capsule of the tonsil and the upper part of the anterior palatine arch);
2. Posterior localization, occurs less frequently and accounts for about 15% of cases (the abscess is localized between the palatine tonsil and the posterior arch);
3. Lower localization, occurs in slightly more than 5% of cases (the abscess is localized between the lower polys of the tonsil and the lateral wall of the pharynx);
4. Lateral localization, occurs in less than 5% of cases (the abscess is localized between the middle part of the tonsil and the lateral wall of the pharynx).

According to clinical and morphological changes, three forms of paratonsillitis are distinguished: edematous, infiltrative and abscessing. The edematous form of inflammation is very rare, infiltrative - in 15-20% and in most cases the abscessing form is observed - in 75-90% of patients.

There are several localizations of paratonsillar abscess:

1. Supratonsillar localization (antero-superior), occurs in 73% of cases (the abscess is localized between the capsule of the tonsil and the upper part of the anterior palatine arch);
2. Posterior localization, occurs in 16% of cases (the abscess is localized between the palatine tonsil and the posterior arch);
3. Inferior localization, occurs in 7% of cases (the abscess is localized between the lower polys of the tonsil and the lateral wall of the pharynx);
4. Lateral localization, occurs in 4% of cases (the abscess is localized between the middle part of the tonsil and the lateral wall of the pharynx).

In anterior or anterior paratonsillitis, there is a sharp bulging of the upper pole of the tonsil together with the palatine arches and soft palate towards the midline, the surface of which is tense and hyperemic, the uvula is displaced to the opposite side, the tonsil is also pushed down and back. Posterior paratonsillitis, localized in the tissue between the posterior arch and the tonsil, can spread to the arch and the lateral wall of the pharynx. During pharyngoscopy, swelling is noted in the same area. The palatine tonsil and anterior arch may be slightly changed, the uvula and soft palate are usually edematous and infiltrated.

Lower paratonsillitis has less pronounced pharyngoscopic signs. Only swelling and infiltration of the lower part of the

anterior arch are noted, but subjective manifestations of the disease in this localization are significant. The adjacent part of the root of the tongue is usually involved in the process, sometimes there is swelling of the lingual surface of the epiglottis. External, or lateral paratonsillitis is less common than other forms, but is the most severe, with this localization, swelling and infiltration of the soft tissues of the neck on the affected side, torticollis, trismus are expressed. On the part of the pharynx, inflammatory changes are less. Moderate bulging of the entire tonsil and swelling of the surrounding tissues are noted. The problem of treating acute inflammatory processes of the pharynx, the prevalence of which does not decrease, continues to arouse great interest in both theoretical and practical terms. The severity of purulent-inflammatory processes depends on the severity of endogenous intoxication associated with the presence of decay products of necrotic tissue, exo- and endotoxins, microorganisms and other biologically active substances. An increase in the number of medium-weight molecules (MWM) in the biological environments of the body indicates an increase in catabolism and the development of endotoxemia. The choice of a detoxification therapy method is determined by the severity of the endogenous intoxication syndrome and the state of the physiological detoxification systems. Efferent treatment methods, which are based on the extraction of toxic substances of exogenous or endogenous origin from the internal environments of the body, have determined a separate direction in modern medicine. Despite the fact that surgical dentistry has a large number of different detoxification methods, they are not always used in the treatment of outpatients with acute purulent-inflammatory diseases. Enteral sorption therapy is one of the most accessible ways to eliminate the symptoms of endogenous intoxication. In many chronic diseases, a decrease in the adaptation resource is a predictor of process decompensation at a certain stage of disease progression. When exposed to an external factor (for example, a microbial one in PTA), the "cost of adaptation" is too high, and the resource of adaptive reactivity is insufficient, which can lead to an adaptation "breakdown" that defines the pathological process as decompensated. There are various methods for assessing adaptive reactivity, including using the lymphocyte index (or adaptation index). Depending on the value of the lymphocyte index (LI), one can indirectly judge the stress or anti-stress type of adaptation reactions. The lower the LI value, the greater the probability of the occurrence of the stress type of adaptation reactions.

It is of interest to study the relationship between intoxication load and adaptive reactivity - to what extent does intoxication load affect the development of the stress type of adaptation. The absence of such information in the available scientific literature served as the basis for conducting this study. In patients with PTA, moderate intoxication prevails (69.9%) with predominant LIIO values in the range of 2.8-3.8 units. Mild intoxication was detected in 16.4%, severe intoxication - 13.7% of cases. The increase in the intoxication index from moderate to severe values was accompanied by a reliable deepening of stress types of adaptation: with moderate

intoxication, the "stress-2" reaction prevailed (64.7%), and with severe intoxication, the "stress-3" reaction prevailed (80.0%), and the probability of the latter was 40 times more reliable (OR = 0.03; CI 95% 0.002–0.211;  $p < 0.001$ ), which can be regarded as an unfavorable prognostic sign. Modern knowledge regarding the morphofunctional organization of the palatine tonsils as a lymphoepithelial organ allows us to consider them as regional centers with immunoregulatory functions in relation to the mucous membranes, which consists in the creation and maintenance of the local immunity system at an adequate level. Evaluation of the role of local and general immunity in chronic tonsillitis is one of the pressing problems, taking into account the immunopathological mechanisms of disease development. When a purulent-inflammatory focus occurs, the first stage of protection is the reaction of local immune factors. The main class of immunoglobulins in the secretion of the oral mucosa are immunoglobulins of class A, produced by plasma cells of the connective tissue layer of the mucous membrane. It has been established that secretory immunoglobulins A (sIgA), fixed on the mucous membranes, reduce their permeability and promote the "immune elimination" of pathogens.

### 3. Discussion

Studying the functioning of the immune system in patients with chronic tonsillitis is necessary to reduce the number of relapses of PA.

Currently, the palatine tonsils are considered an immunocompetent organ, and diseases associated with pathological changes in the palatine tonsils are classified as an immunopathological profile of disorders with a leading toxic-allergic mechanism, accompanied by autoimmune and immune complex reactions. Of course, the first stage of protection when a purulent-inflammatory focus occurs is the reaction of local immune factors. It has been established that secretory immunoglobulins A (sIgA), fixed on mucous membranes, reduce their permeability and promote "immune elimination" of pathogens. In connection with these data, further study of factors of local immunological protection of the palatine tonsils in paratonsillitis is of unconditional interest.

Scientific research has shown that the IL-6 content in saliva in children diagnosed with peritonsillar abscess is significantly elevated compared to the control group. The IL-1 $\beta$  content in saliva changes earlier than other acute phase inflammatory mediators in response to local action of a damaging factor.

Analysis of the immunological examination results showed that in the group of patients with APA there is an increase in the phagocytic activity of neutrophils, the CD4+/CD8+ index, the content of cells expressing HLA-DR+ markers, complement activity, serum concentrations of IgA, IgM, IgG, fibronectin levels, proinflammatory cytokines IL-8, IL-1 $\alpha$ , IL-1 $\beta$  and a decrease in the level of TNF- $\alpha$ , myeloperoxidase activity and the levels of cells with CD8+, CD4+, CD16+,

CD20+, CD25+. The development of a peritonsillar abscess is possible both in the acute period of angina and during the period of inflammation subsidence, with any course of angina. Informative for the diagnosis of a formed abscess were only the total number of leukocytes, absolute values of neutrophils, basophils, eosinophils and indicators of the acute phase response and endogenous intoxication: ESR, albumin, urea. Therefore, it is justified to study the resistance of the body of patients with angina to predict the formation of a peritonsillar abscess by determining the type of immune reactions and the level of cells: HLA-DR+, CD3+, CD4+, CD8+, CD16+, CD20+, CD25+ and CD95+. The results of cluster and discriminant analyses allow us to consider the resulting classification of types of immune reactions of the body adequate. Lipid peroxidation (LPO) is a process of direct oxygen transfer to a substrate with the formation of peroxide compounds. Currently, there is a large amount of experimental and theoretical material on the basis of which it can be stated that lipid peroxidation is a physiological process. This is due to the recognition of the decisive role of biomembranes in the vital activity of the body, in the structure of which lipids with a high content of unsaturated fatty acids occupy an important place. The first reaction in chain oxidation is the origin of a chain as a result of the formation of a free radical that oxidizes the lipid. This radical is formed as a result of single-electron oxidation, i.e., when a hydrogen atom or electron is detached from the reacting molecule. This process is the limiting link in the entire chain reaction and the speed of the processes as a whole depends on its speed. Diene conjugation in molecules of polyunsaturated fatty acids and their hydroperoxides appears in the very initial stages, and they are classified as primary products of LPO.

At the second stage of oxidation, dienes and trienes break down to intermediate products - aldehydes and ketones, one of the compounds is malonic dialdehyde (MDA). Secondary products, in particular MDA, interact with amino groups (NH<sub>2</sub>) of phospholipids that are part of the membranes, forming polymeric fluorescent compounds known as Schiff bases (SB). In addition to SB, gaseous components - heptane, ethane, octane - are also considered final products. High biological activity of LPO determines 2 opposite types of their action in the body. Primary products of LPO, normally present in the body in relatively low concentrations, have a physiological effect, while secondary products of LPO have a damaging effect. Lipid peroxidation within its physiological level is an integral component of cell activity. Peroxidation processes are an active mediator of cell metabolism, participate in the biosynthesis of hormones, prostaglandins, progesterone, cholesterol synthesis, modification of the phospholipid composition of membranes, phagocytosis. A decrease in the physiological level of LPO processes is a stress factor for the body. Under the influence of active oxygen species (hydrogen peroxide, hydroxyl radical, superoxide anion radical), polyunsaturated fatty acids are destroyed, the lipid environment of membrane-bound phospholipids changes, and new ion conductivity channels are created. Malonic dialdehyde oxidizes sulfhydryl groups

of proteins (SH groups) and protein complexes in the active centers of membrane-bound proteins, which leads to a loss of the functional properties of biomembranes. The physiological level of LPO products is controlled by various regulatory systems. Normally, all cells contain a wide range of compounds that prevent the production of excess free oxygen species. Conventionally, a distinction is made between physiological and biochemical (actually antioxidant) systems of body defense. Strict regulation of LPO reactions is ensured by the coordinated functioning of enzymatic and non-enzymatic mechanisms of the antioxidant defense system. The coordinated action of these links of the antioxidant system ensures non-specific resistance of the body, its protection from the effects of various pathogenic factors, including microbial ones.

Enzymatic and non-enzymatic mechanisms implementing antioxidant protection (low- and high-molecular substances) primarily prevent damage to cell membranes and disruption of the functional activity of enzymes, which plays a significant role in maintaining homeostasis. The central place in the enzymatic link of the body's antioxidant protection is occupied by superoxide dismutase (SOD). Superoxide dismutase has a pronounced protective effect in inflammatory and reperfusion lesions. This is the only enzyme that forms a chain of oxygen-dependent free-radical reactions. Superoxide dismutase very effectively converts superoxide anion radical into hydrogen peroxide, prevents spontaneous dismutation, the rate of which is 3 orders of magnitude lower than enzymatic reactions. Due to such efficiency of action, the superoxide anion radical in the cell is contained in a concentration of  $10^{12}$ - $10^i$  M. Thus, SOD is a limiting link in the entire LPO process. Non-enzymatic antioxidants are represented by fat-soluble and water-soluble forms. Tocopherols occupy a central place in the non-enzymatic link of the body's antioxidant protection. Vitamins of group E (tocopherols) are compounds synthesized in plants and enter the body of animals and humans with food. Alpha-tocopherol exhibits the greatest antioxidant activity. In the biological system, alpha-tocopherol protects polyunsaturated fatty acids from oxidation only in combination with water-soluble antioxidants: vitamin C, glutathione, etc. Ascorbic acid restores the alpha-tocopherol radical, returning its antioxidant properties, while simultaneously ensuring the removal of radicals from the hydrophobic phase of the lipid bilayer into the aqueous phase. Alpha-tocopherol is concentrated in the membranes of mitochondria, erythrocytes, plasma membranes, that is, where high partial oxygen tension is determined. Due to its ability to easily give up and capture electrons, alpha-tocopherol can act as a reducing agent for metals of variable valence. Thus, (vitamin E) in the ascorbate /a-tocopherol pair is an effective radical quencher in biological membranes, provided that there is no Me11 in the medium.

The role of synergists is played by substances that have a low oxidation-reduction potential and easily transform from the oxidized form to the reduced form (ascorbic and citric acids). The presence of two enol groups in the structure of the ascorbic acid molecule allows the latter to participate in oxidation-reduction transformations, acting as a donor and

acceptor of hydrogen. Under conditions of moderate production of prooxidants, characteristic of the intracellular environment, ascorbic acid is reversibly oxidized to dehydroascorbic acid. Further oxidation to diketogulonic acid (irreversible form of oxidation) occurs at low pH values ( $\text{pH} < 4$ ). Irreversible degradation of ascorbic acid is inhibited by thiosulfate, thiourea, and uric acid. Dehydroascorbic acid has the same high antioxidant activity in biological systems as ascorbic acid, since it is very quickly restored in the presence of reduced glutathione. It is known that the body's need for ascorbic acid in many animals is covered by endogenous synthesis. However, for humans, monkeys, guinea pigs, and bats, it is a vitamin that they receive from outside, and the lack of which in the diet leads to the development of pathological changes. A decrease in the content of ascorbic acid in tissues is explained by its oxidation to diketogulonic acid, which accelerates under conditions of oxidative stress. Superoxide radical, hydrogen peroxide, and other active forms of oxygen (ROS) can migrate from the cell to the intercellular space and into the blood plasma. Extracellular free radicals cannot be destroyed by enzymes, since serum and tissue fluids are poor in SOD, catalase, and glutathione enzymes. In these fluids, the main antioxidant role belongs to the copper-containing protein ceruloplasmin, which is part of the alpha globulin fraction of blood plasma. Ceruloplasmin functions in the blood, where it intercepts free radical forms of oxygen, thus protecting lipid-containing biological structures from damaging effects. Currently, most researchers consider ceruloplasmin to be the main endogenous antioxidant of blood plasma.

Thus, there are a number of interconnected antioxidant systems in the body, the main purpose of which is to maintain enzymatic and non-enzymatic reactions at a steady-state level. At each stage of the development of peroxide reactions, there is a specialized system that performs these functions.

Thus, with the expansion of the range of biochemical studies, it has been established that free-radical processes occupy far from the last place in the pathogenesis of inflammatory reactions. The accumulation of free-radical oxidation products in the body has a pro-inflammatory, demetabolizing effect, increases the violation of the immune status, contributes to the chronicity of the inflammatory process, as well as the formation of a complicated course of diseases.

In this regard, the study of the manifestations of oxidative stress in purulent-inflammatory processes in the throat and its correction seems relevant and significant, and the appointment of drugs with antioxidant action is pathogenetically justified.

Paratonsillar abscess often occurs against the background of tonsillitis or a few days after its completion. At first, hyperemia of vessels, impaired permeability, edema and small-cell infiltration appear in the peritonsillar tissue. Absorption of bacterial toxins and products of the inflammatory reaction causes fever, intoxication, and changes in blood composition. Compression of nerve trunks causes severe pain, disorders of the central and autonomic nervous systems.

An abscess is formed as a result of purulent melting of the infiltrate that occurs in the paratonsillar space. The disease begins with the appearance of pain when swallowing, which gradually becomes constant and sharply increases when trying to swallow saliva, radiating to the ear and teeth. Characteristic symptoms are headache, general fatigue, an increase in body temperature to febrile numbers, while patients refuse to eat or drink. Trismus of the ingestive muscles gradually occurs and increases. Speech becomes slurred and nasal. As a result of inflammation of the muscles of the pharynx and cervical lymphadenitis, a painful reaction occurs when turning the head; the patient holds his head to the side and turns it, if necessary, along with the entire body.

The general condition of the patient becomes severe not only because there is purulent inflammation in the pharynx and systemic intoxication, but also due to excruciating pain in the throat, sleep disturbance, inability to swallow liquid and hunger.

In addition, a certain specificity of clinical symptoms is characteristic of different localizations of the peritonsillar abscess.

With an anterior superior or anterior peritonsillar abscess, there is a sharp bulging of the upper pole of the tonsil together with the palatine arches and soft palate to the midline, the surface of which is tense and hyperemic, the uvula is displaced to the opposite side, the tonsil is also pushed down and back.

A posterior peritonsillar abscess, localized in the tissue between the posterior arch and the tonsil, can spread to the arch and the lateral wall of the pharynx. During pharyngoscopy, swelling is noted in the same area. The palatine tonsil and anterior arch may be slightly changed, the uvula and soft palate are usually edematous and infiltrated.

The lower paratonsillar abscess has less pronounced pharyngoscopic signs. Only edema and infiltration of the lower part of the anterior arch are noted, but the subjective manifestations of the disease in this localization are significant. The adjacent part of the root of the tongue is usually involved in the process, sometimes there is edema of the lingual surface of the epiglottis. It is possible for collateral edema to spread to the upper part of the larynx, which can lead to its stenosis.

External, or lateral paratonsillar abscess is less common than other forms, but is the most severe. The process develops in the tissue filling the tonsillar niche outside the tonsil, so the conditions for spontaneous opening with a breakthrough of pus into the pharyngeal cavity are the least favorable here. With this localization, swelling and infiltration of the soft tissues of the neck on the affected side, torticollis, and trismus are expressed. On the part of the pharynx, inflammatory changes are less. Moderate bulging of the entire tonsil and swelling of the surrounding tissues are noted.

The main cause of peritonsillar abscess is the penetration of pathogenic microflora into the tissues that surround the palatine tonsils. Predisposing factors are bacterial lesions of the pharynx. Most peritonsillar abscesses occur as a complication of angina or exacerbation of chronic tonsillitis.

Also, a peritonsillar abscess can have an odontogenic origin - the cause is dental caries, periostitis of the alveolar processes, chronic gingivitis.

In very rare cases, an abscess is formed after infection of the wounds of the mucous membrane as a result of trauma.

According to the phase of development of the pathological inflammatory reaction, three main forms of peritonsillar abscess are distinguished, which act as stages of the pathological process.

Edematous - which is characterized by swelling of the peritonsillar tissues without signs of inflammation.

Infiltration - manifested by damage to the mucous membrane, local increase in temperature, the appearance of pain syndrome.

Abscessing - develops 5-7 days after the onset of the infiltration stage. Severe deformation of the pharynx occurs.

Depending on the localization of the pathological process, the peritonsillar abscess is divided into anterior, posterior, lower and external.

The main symptom of a peritonsillar abscess is a sharp pain in the throat, which intensifies when chewing, swallowing and opening the mouth. Lateralization to the lower jaw and ear is possible. Also noted is an increase in temperature to 39-40 °C, trismus of the masticatory muscles, pronounced salivation, a sharp unpleasant odor from the mouth, swelling of the lateral surface of the neck, forced position of the head (tilted to the affected side).

Diagnosis of acute inflammatory diseases of the pharynx, especially its phlegmonous forms, presents certain difficulties due to their deep location in the para- and retropharyngeal spaces. Meanwhile, these diseases that are not diagnosed in a timely manner and inadequately treated sometimes lead to death due to the development of severe complications in the form of mediastinitis, sepsis, thrombosis. In this regard, timely and early diagnosis of these diseases plays an important role in choosing further tactics for treating this pathology. According to indications and clinical studies, the analysis of the results of the general (clinical) blood test showed significant differences in leukocyte indices both between groups and in absolute and relative indices of leukocyte fractions in patients with CP, CT and PA. When interpreting the results of a clinical blood test in general and in CP, CT and PA in particular, it is necessary to rely only on the absolute indices of the number of cells of various leukocyte fractions, and not on the leukocyte formula data, which are usually used in routine practice. It has been shown that the most informative in diagnosing the course of CP and CT were the indices of acute phase response and endogenous intoxication in both the general blood test (ESR), coagulogram (fibrinogen and RFMC), and in the biochemical analysis (albumin, creatinine, urea). According to some authors, in neutrophils of patients with lacunar tonsillitis complicated by paratonsillar abscess, before treatment, an increase in the activity of the entire enzymatic spectrum was observed. After a course of standard therapy, the activity of all the enzymes studied was suppressed. At the same time, a qualitative redistribution of the reacting cells occurred.

Pharyngoscopy reveals a protrusion and hyperemia of the soft palate above the tonsil. The abscess causes asymmetry of the pharynx, displacing the uvula to the opposite side and pushing back the tonsil.

Scientists have also developed an original method of intraoperative diaphanoscopy of the paratonsillar space in patients with chronic tonsillitis, which allows intraoperative detection of tonsillar vessels during surgery, which in turn makes it possible to perform simultaneous intraoperative preventive hemostasis.

A sufficient number of patients with acute tonsillitis and paratonsillar abscess in the blood serum were found to have an increased content of lipid peroxidation products (malonic dialdehyde, biochemiluminofamm indicators).

## 4. Conclusions

When studying groups of patients, an increase in the concentration of medium-molecular peptides in the blood plasma was found, which reflects the severity of endogenous intoxication at the height of the inflammatory process. The prescription of a complex of antioxidant drugs to patients with acute tonsillitis leads to a more rapid normalization of the concentration of malonic dialdehyde and medium-molecular peptides in the blood plasma of patients with angina compared to the group receiving traditional treatment. The inclusion of antioxidant drugs in the treatment regimen for patients with peritonsillar abscess along with traditional therapy allows to increase the effectiveness of the treatment, which is confirmed by laboratory parameters. In patients with peritonsillar abscess, significant changes in the immunological status were also revealed, manifested in the suppression of cellular immunity, as well as in an increase in the level of immunoglobulins IgA and IgM during the manifestation of the process. Thus, the level of mature T lymphocytes (SVZ+) at the time of admission was  $41.27 \pm 1.7944\%$  compared to the control group - donors 47-56%. And the IgA and IgM indicators were  $3.2 \pm 0.12$  and  $2.7 \pm 0.03$  g/l compared to the control group - IgA donors  $2.2 \pm 0.6$  g/l and  $1.5 \pm 0.05$  g/l. In the complex therapy of patients with peritonsillar abscess, the use of the drug polyoxidonium and the local antiseptic solution octenisept leads to a faster clinical recovery, as well as to an acceleration of the normalization of immunological and biochemical blood parameters.

Thus, it **follows** that the study of the epidemiology and prevalence of peritonsillar abscess is relevant and requires further research.

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