



Fundamentals of Immunopathogenesis and Pathophysiology of Exudative Otitis Media

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Abstract: According to statistical data ear diseases occupy the second place in the structure of general otorhinolaryngological pathology giving way only to the diseases of nose and paranasal sinuses. Most ear diseases are accompanied by the development of different types of hearing loss, which affects patients not only in the acute phase of the disease, but also takes a chronic form. The social importance of the treatment of this pathology is given by the fact that more than half of all patients with hearing loss are of working age.

Key words: exudative media otitis media, neoplasms, complex therapy.

Introduction

Exudative media otitis media (EOM) is a common inflammatory disease of the middle ear characterized by an accumulation of serous-mucous fluid in the cavity behind the tympanic membrane [2; 5; 8]. According to the literature, EOM is most common in children [1; 10; 13]. However, recent data indicate an increasing incidence of EOM in the structure of adult otolaryngological pathology [3; 16; 21].

For decades, numerous studies have been devoted to describing the mechanisms of exudative otitis media. One such study, conducted in 1878 by A. Politzer, proposed the "hydrops ex vacuo" theory, according to which the cause of EOM is the factors leading to negative pressure in the middle ear [24]. Also, the formation of secretions in the tympanic cavity is a consequence of inflammatory processes in the middle ear mucosa and occurs under the influence of a number of factors [9; 17]. These theories of EOM development reflect part of a single pathological process for the development of chronic middle ear inflammation [6; 10; 18].

The progression of the inflammatory process from the nasopharynx to the pharyngeal orifice is accompanied by the spread of inflammation and the development of auditory tube dysfunction, which is accompanied by impaired middle ear outflow [2; 4; 7]. This results in negative pressure and increased carbon dioxide content in the tympanic cavity. As a result, a discharge is formed in the tympanic cavity, and bacteria from nasopharynx attach to it [19; 23; 25; 28]. In addition, microbial invasion in the tympanic cavity is also facilitated by a disruption of the ventilation and drainage function of the Eustachian tube, caused by an imbalance between the opening and closing mechanisms of the Eustachian tube. Increased exudation leads to increased blood flow, vein compression and impaired drainage. Exudation as a component of the inflammatory response reduces the concentration of toxins and incoming serum antibodies promote phagocytosis, which is supported by the inflammatory response and stimulation of leukocyte release in the inflammatory focus [20; 27; 33].

The immunological element of research into middle ear diseases in particular EOM is central as the most important component. The immunological response is a synergistic interaction between the humoral and cellular responses of the immune system to antigenic stimulation. Immunoglobulins of various classes produced by immunocompetent cells B-lymphocytes are the hallmarks of the humoral response.

In cellular immunity, T-lymphocytes are crucial and their subpopulations, such as killer cells, helper cells and immunological memory cells, are divided based on their mode of action and involvement in immunity. Cytotoxic T lymphocytes of T helper cells can induce lysis of target cell membranes. Under the influence of T-helper cells, B-lymphocytes take part in proliferation and differentiation upon antigen activation [27; 31].

"Local immunity" as a term is a set of defence adaptations and serves to protect the body from the external environment and is specific to the mucous membranes and skin of the body. Non-specific defence mechanisms are also included in this complex. Such mechanisms include the mucociliary system and the synthesis of active proteins such as properdin and interferon [1; 27; 33]. This type of local immunity is usually defined by modern immunology as specific responses to local lymphoid tissue, such as infiltrates of lymphoid and plasma cells and localised clusters of varying density in mucoid tissue [31].

Studies on the morphology, histology and immunomorphology of the middle ear mucosa have supported and refined the idea of middle ear immune defence, which was originally based on the detection of exudates in different types of otitis media. The mucociliary system of the middle ear mucosa is regarded as one manifestation of a synergy of specialized and non-specific defence mechanisms [27; 29].

In order to describe the immunopathogenesis of EOM, it is now proposed to identify types of immune damage based on four types of immunopathological reactions. If there is an elevated concentration of IgE in the middle ear secretion, immediate-type hypersensitivity, also known as type-I, can be suspected. Cytotoxic reactions may be seen in patients with tympanosclerosis (type II). The presence of immune complexes in the exudate of secretory otitis media indicates that the synthesis of immune complexes occurred in the presence of type III complement. The presence of a significant number of T cells in the mucoid exudate diagnoses delayed-type hypersensitivity (type IV) [30; 32].

According to this interpretation, the hypothesis of hypersensitivity distinguishes between a specifically immune stage and a non-specific inflammatory stage of immune inflammation. The development of the immune response occurs simultaneously with the operation of tissue resistance mechanisms, which are not tissue-specific. This particular form of immune response is associated with the production of secretory antibodies, which are responsible for the protective action of epithelial secretions [29]. Laboratory tests of middle ear exudate in EOM have shown the presence of secretory immunoglobulins. The finding of antibody-producing cells in the lamina lymphoid-plasma infiltrate [29; 30] supports the hypothesis that the middle ear mucosa is responsible for the local production of immunoglobulins.

It is widely known that inflammatory disease occurring in the middle ear cavity is characterised by an accumulation of exudate consisting of soluble and insoluble components. The soluble components are similar to serum, while the insoluble ones consist of carbohydrate glycoproteins that are bound to proteins and are similar to mucins. The exudate may also contain various inflammatory cells that are involved in the middle ear's immune defence against infection. These cells include leukocytes, lymphocytes and monocytes; various oxidative and hydrolytic enzymes of lysosomal origin; complement and its fractions; inflammatory mediators, proteinase inhibitors, including antibacterial and antiviral antibodies - immunoglobulins [14].

Neutrophils, monocytes, macrophages and lymphocytes are the most common types of inflammatory cells found in the middle ear exudate of patients diagnosed with EOM [29]. Eosinophilic leukocytes are less common in individuals with EOM. The observed variations in cellular composition suggest that EOM is an active process, with changes in the cellular composition of the exudate varying according to the phase of inflammation. The presence of an exudate indicates either a proliferative phase or a chronic course of the disease. Changes in the proportion of inflammatory cells present in the exudate in ESRD have been linked to immunological processes that influence the nature and progression of the inflammatory process [32; 33].

All the information presented indicates an increase in the specific and non-specific resistance of the mucosa and its epithelial cover in the middle ear and auditory tube. As a result of ESR, the number of secreting cells in the mucosal epithelium sharply increases, the capillary network increases, the enzymatic and immunological activity of the covering epithelium and lymphoid cells increases, cell proliferation in the subepithelial layer sharply increases [15; 35].

The presence of immunoglobulins in the middle ear exudate, whose concentration is significantly higher than in blood serum, is another indicator of the proper functioning of the local immune system. The concentration of immunoglobulins in the middle ear exudate is significantly higher than in blood serum. Patients with ESR have elevated levels of specific immunoglobulins, especially IgA, while the levels of IgM and IgE remain stable. The duration of illness in a patient correlates with an increased likelihood of detecting IgA in the middle ear exudate. In addition, there is a correlation between an increase in secretion viscosity and an increase in IgA frequency [32; 33].

Studies of the morphology, histochemistry and immunology of the middle ear mucosa confirm the presence of immune defence mechanisms in this area. The mucociliary system of the middle ear mucosa is the site of specific and non-specific defence mechanisms, which are responsible for the expression of local immunity [27; 29; 30].

According to a review of the relevant literature, the immunopathogenesis of ESR is not yet fully understood and requires further research. In order to find a solution to this problem, local immunopathological processes occurring at the level of the middle ear mucosa need to be investigated.

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