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ESSENTIALS OF EXUDATIVE OTITIS MEDIA IMMUNOPATHOGENESIS AND PATHOPHYSIOLOGY

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Abstract
Based on statistical data, ear disorders are ranked second in terms of general otorhinolaryngological pathology, with only paranasal sinus and nose diseases ahead of them. Many forms of hearing loss arise in conjunction with most ear illnesses, affecting individuals not only during the acute stage of the illness but also in its chronic form. Given that over half of all patients with hearing loss are of working age, treating this illness is socially significant.

Keywords: exudative media otitis media, neoplasms, complex therapy.

Introduction

A frequent inflammatory middle ear illness called exudative media otitis media (EOM) is characterized by a buildup of serous-mucous fluid in the space behind the tympanic membrane [2; 5; 8]. The evidence indicates that children are most likely to experience EOM [1; 10; 13]. Recent data, however, show that the structure of adult otolaryngological pathology is showing an increasing prevalence of EOM [3; 16; 21].

The mechanics of exudative otitis media have been the subject of countless study for decades. In 1878, A. Politzer conducted a study that presented the "hydrops ex vacuo" theory, which states that the variables causing negative pressure in the middle ear are the source of EOM [24]. Additionally, a variety of causes, including inflammation in the middle ear mucosa, contribute to the development of secretions in the tympanic cavity [9; 17]. A portion of a single pathogenic mechanism for the development of chronic middle ear inflammation is reflected in these hypotheses of EOM development [6; 10; 18].

The development of auditory tube dysfunction and the spread of inflammation are linked to the progression of the inflammatory process from the nasopharynx to the pharyngeal orifice, which is accompanied by reduced middle ear outflow [2; 4; 7]. As a result, the tympanic cavity experiences negative pressure and a rise in carbon dioxide levels. Bacteria from the nasopharynx cling to the discharge that consequently forms in the tympanic cavity [19; 23; 25; 28]. Furthermore, an imbalance between the Eustachian tube's opening and shutting mechanisms can produce a disruption in the tube's ventilation and drainage function, which in turn might enable microbial invasion in the tympanic cavity. Increased blood flow, venous constriction, and poor drainage are all caused by increased exudation. The inflammatory reaction and stimulation of leukocyte release at the inflammatory focus increase phagocytosis, while exudation as a part of the inflammatory response lowers the concentration of toxins[20; 27; 33].

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The most crucial and core aspect of study on middle ear illnesses, and specifically EOM, is the immunological component. The humoral and cellular immune system responses to antigenic stimulation work in concert to produce the immunological response. The signatures of the humoral response are immunoglobulins of different classes made by B-lymphocytes, which are immunocompetent cells.

T-lymphocytes play a key role in cellular immunity, and their subpopulations—killer cells, helper cells, and immunological memory cells—are categorized according to how they function and contribute to immunity. T helper cell-derived cytotoxic T lymphocytes have the ability to cause target cell membrane lysis. B-lymphocytes participate in antigen-induced proliferation and differentiation when T-helper cells are present [27; 31].

The phrase "local immunity" refers to a group of defense mechanisms that are unique to the body's mucous membranes and skin and function to shield the body from the outside world. This complex includes defense mechanisms that are not specific. The production of active proteins like properdin and interferon, as well as the mucociliary system, are examples of these mechanisms [1; 27; 33]. Modern immunology typically defines this kind of local immunity as particular reactions to local lymphoid tissue, such as localized clusters of variable densities in mucoid tissue and infiltrates of lymphoid and plasma cells [31].

The concept of middle ear immune defense, which was initially predicated on the identification of exudates in various forms of otitis media, has been validated and improved by research on the morphology, histology, and immunomorphology of the middle ear mucosa. It is believed that the middle ear mucociliary system is one example of how specialized and non-specific defense systems work together [27; 29].

It is currently suggested to categorize immune damage according to four categories of immunopathological reactions in order to explain the immunopathogenesis of EOM. Immediate-type hypersensitivity, or type-I, can be suspected if the middle ear discharge has an elevated concentration of IgE. Tympanosclerosis (type II) individuals may experience cytotoxic responses. The existence of immune complexes in the secretory otitis media exudate suggests that type III complement was present during the immune complex production process. Delay in diagnosing delayed-type hypersensitivity (type IV) is indicated by a notable quantity of T lymphocytes in the mucoid exudate [30; 32].

This view states that the notion of hypersensitivity makes a distinction between the non-specific inflammatory stage of immune inflammation and the particularly immunological stage. Tissue resistance mechanisms function in tandem with the development of the immune response and are not specific to any one tissue. The generation of secretory antibodies, which underpin the protective function of epithelial secretions, is linked to this specific type of immune response [29]. Secretory immunoglobulins have been detected in middle ear fluid from EOM patients in laboratory studies. The idea that the middle ear mucosa is in charge of the local generation of immunoglobulins is supported by the discovery of antibody-producing cells in the lamina lymphoid-plasma infiltrate [29; 30].

It is commonly recognized that an accumulation of soluble and insoluble exudate is a characteristic of inflammatory diseases that affect the middle ear cavity. The insoluble components are made up of glucose glycoproteins that are attached to proteins and resemble mucins, whereas the soluble

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components are comparable to serum. Different inflammatory cells that are involved in the middle ear's immunological defense against infection may also be present in the exudate. Leukocytes, lymphocytes, and monocytes are among the cells in question, as are other lysosomal-derived oxidative and hydrolytic enzymes, complement and its fractions, inflammatory mediators, proteinase inhibitors, and immunoglobulins, which are antiviral and antibacterial antibodies [14].

In the middle ear exudate of individuals diagnosed with endometriosis (EOM), the most often observed inflammatory cell types are neutrophils, monocytes, macrophages, and lymphocytes [29]. In those with EOM, eosinophilic leukocytes are less prevalent. The observed cellular composition fluctuations imply that exudate cellular composition varies according to the stage of inflammation, indicating that exudate cellular composition is an active process. An exudate's existence suggests that the disease is either in its proliferative phase or is progressing slowly. Immunological mechanisms that affect the course and character of the inflammatory process have been connected to variations in the percentage of inflammatory cells found in the exudate in end-stage renal disease (ESRD) [32; 33].

Everything that has been shown points to an increase in the middle ear and auditory tube mucosa's and its epithelial cover's particular and non-specific resistance. ESR causes a dramatic rise in the capillary network, enzymatic and immunological activity of the covering epithelium and lymphoid cells, cell proliferation in the subepithelial layer, and the amount of secreting cells in the mucosal epithelium [15; 35].

Immunoglobulins are another clue that the local immune system is working well; the concentration of these substances in middle ear exudate is much higher than in blood serum. Compared to blood serum, the middle ear exudate has a much higher immunoglobulin concentration. Furthermore, there is a relationship between an increase in IgA frequency and an increase in secretory viscosity [32; 33].

Immune defense systems are present in the middle ear mucosa, as confirmed by studies of its morphology, histochemistry, and immunology. The expression of local immunity is brought about by both non-specific and specific defense mechanisms, which are found in the middle ear mucociliary system [27, 29; 30].

A review of the pertinent literature indicates that more research is necessary to fully understand the immunopathogenesis of ESR. Investigation of local immunopathological processes at the level of the middle ear mucosa is necessary to find a solution to this issue.

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