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ACUTE AND CHRONIC SINORITIS, ETIOLOGICAL FACTORS

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Summary
This article provides information about the etiological factors of acute and chronic sinusitis.

Keywords: acute and chronic sinusitis, etiological factors, odontogenic sinusitis.

Relevance

Sinusitis should be understood as inflammation of the mucous membrane of the paranasal sinuses (SNP). The term "sinuitis" is used less and less frequently. The diagnosis of “rhinosinusitis” reflects an inflammatory process in the mucous membrane of the SNP and the nasal cavity, which, in terms of infectious pathology, characterizes an acute respiratory viral infection (ARVI) more than sinusitis itself. In practice, the terminology that reflects the inflammatory process according to its localization is more common: inflammation of the maxillary sinus is diagnosed as sinusitis, ethmoid labyrinth - ethmoiditis, frontal sinus - frontal sinusitis, sphenoid - sphenoiditis. The terms are also used: hemisinusitis - inflammation of all SNPs on one side, polysinusitis - damage to several sinuses and pansinusitis - inflammation of all SNPs. As for the actual term “sinusitis”, it is currently more used in chronic non-infectious processes (for example, polyposis sinusitis). This publication will focus on acute and chronic sinusitis of viral, bacterial and fungal etiology.

The basis of the inflammatory lesion of the SNP is an infectious onset. The causative agents of sinusitis are viruses, bacteria and fungi that enter the sinus mainly exogenously, but infection with opportunistic flora that is constantly present on the mucous membrane of the upper respiratory tract is possible. For exogenous infection, the aerogenic route of infection is characteristic, and besides it, the hematogenous and (very relevant) odontogenic route of infection. For OS, viral and bacterial etiology is more characteristic, for CS - bacterial and fungal. Rhinoviruses, which number over 100 serotypes, enter the mucous membrane and bind to intercellular adhesion molecules (ICAM-1), a receptor that is constantly expressed on the epithelium of the nasal cavity and nasopharynx. Approximately 90% of rhinoviruses enter the body in this way, in the region of the pharyngeal tonsil, which is rich in the mentioned receptors [1]. After cell invasion and replication, rhinovirus spreads to the nasal cavity and SNP. Unlike influenza viruses and adenoviruses, which cause extensive damage

and desquamation of the ciliated epithelium, rhinoviruses are less invasive, and therefore the main part of the ciliated epithelium remains relatively intact.

Depending on the etiology and pathogenesis of sinusitis, there are: rhinogenic, hematogenous, traumatic and odontogenic ways of infection of the maxillary sinus. The transition of inflammation from the nasal mucosa is observed in acute rhinitis, influenza. Hematogenous sinusitis is possible with typhoid, pneumonia, diphtheria, scarlet fever and other common infections. Traumatic sinusitis occurs with gunshot and other injuries of the maxillary sinus, sometimes as a result of a violation of the integrity of the sinus during tooth extraction. The occurrence of odontogenic sinusitis is associated with the spread of infection from diseased teeth, which is facilitated by anatomical features in the form of a close fit of the bottom of the maxillary sinus to the tops of the roots of the teeth. Normally, the maxillary sinus is located above the sockets of the second premolar and the first and second molars. When expanding it, the supralunar region of the third molar (wisdom tooth) is captured from behind, and the region of the first premolar and less often the canine is captured anteriorly. The roots of the teeth located in the alveolar process are separated from the bottom of the maxillary sinus by a bone septum of unequal thickness in different individuals; in some it is massive, in others it is so thin that the reliefs of the tops of the roots protrude into the cavity, and sometimes the tops of the roots are covered only by the periosteum and directly by the mucous membrane of the cavity. Such an intimate connection of the teeth with the maxillary sinus makes it clear the occurrence of odontogenic sinusitis as a complication as a result of the disease of the periapical areas of the teeth. Particularly evident is the role of odontogenic infection, when granulating periodontitis or granuloma, having destroyed the bone septum between the bottom of the maxillary sinus and the periapical space, involves the sinus mucosa adjacent to the tooth cells in the process. In the case of accession to this rhinogenic infection, the process becomes aggravated and takes on a spilled character. It is possible to spread the infection through the system of the venous plexus, which connects the tissues of the alveolar process of the periodontium with the mucous membrane of the maxillary sinus.

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