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MODERN APPROACHES TO THE TREATMENT OF POLYPOSIS RHINOSINUSITIS

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ABOUT ARTICLE

Key words: polypoid rhinosinusitis, nose, paranasal sinuses, edema.

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Polyposis Abstract: rhinosinusitis (PRS) is a widespread disease prone to frequent recurrence. The quality of life of patients with this pathology is sharply reduced and is comparable to that of patients with moderate bronchial asthma. What causes problems? such serious **Polyposis** rhinosinusitis is a chronic inflammatory disease of the mucous membrane of the nasal cavity and paranasal sinuses, characterized by the formation and recurrent growth of polyps, edematous consisting mainly of tissue infiltrated by eosinophils.

POLIPOZ RINOSINUSITNI DAVOLASHGA ZAMONAVIY YONDASHUVLAR

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MAQOLA HAQIDA

Kalit so'zlar: polipoid rinosinusit, burun, paranasal sinuslar, shish.

Annotasiya: Polipoz rinosinusit (PRS) tez-tez takrorlanishga moyil bo'lgan keng tarqalgan kasallikdir. Ushbu patologiya bilan og'rigan bemorlarning hayot sifati keskin pasayadi va o'rtacha bronxial astma bilan og'rigan bemorlar bilan solishtirish mumkin. Bunday jiddiy muammolarga nima sabab bo'ladi? Polipoz rinosinusit - burun bo'shlig'i va paranazal sinuslarning shilliq qavatining surunkali yallig'lanish kasalligi bo'lib, asosan eozinofillar infiltratsiyalangan shishgan to'qimalardan iborat poliplarning shakllanishi va takroriy o'sishi bilan tavsiflanadi.

СОВРЕМЕННЫЕ ПОДХОДЫ К ЛЕЧЕНИЮ ПОЛИПОЗНОГО РИНОСИНУСИТА

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О СТАТЬЕ						
Ключевые	слова: полипозный		Аннотация	я: По	Полипозный	
риносинусит, нос,	околоносовые	пазухи,	риносинусит	(ПРС) —	широко	
отек.			распространенное заболевание, склонное к			
			частым рециді	ивам. Качество	ингиж с	
			больных с да	нной патологи	ей резко	
			снижено и сравнимо с таковым у больных бронхиальной астмой средней степени			
			тяжести. Что вызывает такие серьезные проблемы? Полипозный риносинусит — хроническое воспалительное заболевание		серьезные	
					инусит —	
					болевание	
	слизистой оболочки полост		лочки полости	носа и		
			околоносовых пазух, характеризующееся образованием и рецидивирующим ростом			
			полипов, состоя	щих преимущест	твенно из	
			отечной ткан	ии, инфильтри	ированной	

INTRODUCTION

The main complaints in ORS are: nasal congestion, impaired sense of smell, frequent headaches, fatigue, sleep disturbance (Botirov A. J. et al, 2020). The main diagnostic methods in the diagnosis of PRS are anterior rhinoscopy, endoscopic examination and computed tomography. These research methods make it possible to establish the prevalence of the pathological process and possible anatomical defects that contribute to its development. Of the additional methods, it is necessary to investigate the respiratory function (acoustic rhinometry, spirometry), smears-prints, allergological testing. Until today, the etiology and pathogenesis of this disease are not completely clear. Among the causes contributing to the development of the polyposis process are allergies, bronchial asthma, chronic inflammation of the paranasal sinuses, various anatomical anomalies that contribute to the disruption of aeration and the maintenance of the inflammatory process (Djuraev J. A. et al, 2021).

As a rule, the disease occurs at the most able-bodied age - 25-35 years. Men suffer from this pathology on average 2 times more often than women. On average, 4-5% of patients who seek help from an otolaryngologist are diagnosed with PRS. However, these figures refer only to clinically manifesting forms of the disease. The real prevalence of ORS, taking into account subclinical forms, is much higher. Back in 1892, E. Zuckerkandl reported that polyps were found in the paranasal sinuses (PNS) at every eighth autopsy (12.5%). P.L. Larsen and M. Tos examined autopsy nasoethmoid blocks removed from 19 people who died of cardiovascular and

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neurological diseases and found polyps in the ethmoid labyrinth in 5, i.e. in more than a quarter of all cases. This disease is often one of the manifestations of the systemic pathology of the respiratory tract, and its pathogenesis is closely related to the pathogenesis of bronchial asthma, with disorders of the metabolism of arachidonic acid and water-salt metabolism in the body, in particular with cystic fibrosis. So, with cystic fibrosis, polyposis changes in the mucous membrane of the nasal cavity occur even in childhood and are prone to more frequent recurrence. In patients with bronchial asthma associated with aspirin intolerance, the number of relapses of ORS approaches that of cystic fibrosis. Polyps that develop in an allergic background are also prone to more frequent recurrences compared to ORS without a history of allergies. All of the above indicates that ORS is only a syndrome of a systemic disease that requires a serious and balanced approach to treatment.

THE MAIN RESULTS AND FINDINGS

Morphologically, polyps consist of damaged, sometimes metaplastic epithelium, located on a thickened basement membrane, and edematous stroma, containing a small number of glands and vessels and practically devoid of nerve endings (Azizova F. H. 2001). The stroma of a typical polyp contains fibroblasts that form a supporting frame, pseudocysts and cellular elements, the main of which are eosinophils located around the vessels, glands and directly under the integumentary epithelium. Eosinophils play a key role in the pathogenesis of PRS, but it is not yet known exactly how eosinophilic inflammation leads to the formation and growth of polyps. It is suggested that the recruitment of eosinophils may be regulated by cytokines. Cytokines produced by Th2-lymphocytes (in particular, interleukin-3 and interleukin-5) can cause eosinophilia by stimulating the proliferation of eosinophils in the bone marrow and their release into the bloodstream, as well as inhibiting their apoptosis (programmed death). Recent studies suggest that interleukin-5 is a key factor in the pathogenesis of ORS, inducing the processes of eosinophil homing, their migration into tissues, and degranulation (Tuxtaev K. R., Rasulev K. I., Azizova F. X, 2008). One hypothesis suggests that activated eosinophils migrate into the mucosa to kill fungi that enter the PNS cavity during normal air exchange.

Another pathogenetic mechanism of ORS, intensively studied in recent years, is a violation of the metabolism of arachidonic acid and intolerance to non-steroidal anti-inflammatory drugs. In patients with ORS, the cyclooxygenase enzyme is inhibited, which leads to the activation of an alternative pathway of arachidonic acid metabolism catalyzed by 5-lipoxygenase.

The products of the lipoxygenase pathway of arachidonic acid breakdown, leukotrienes, are powerful pro-inflammatory mediators hundreds and thousands of times more active than histamine and prostaglandins.

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In the last decade, research into the pathogenesis of ORS and testing of new methods of treatment has become a priority in otorhinolaryngology, but it has not been possible to completely prevent the recurrence of ORS. It became clear that the problem of ORS should be considered primarily from a therapeutic standpoint, and not as a disease that initially requires surgical intervention (Azizova F.H at all, 2010). Unfortunately, currently existing medications can only stop the growth of polyps and increase the intervals between relapses, but not cure the disease itself. Therefore, surgical removal of polyps from the nasal cavity and SNPs remains a forced but necessary measure in the arsenal of ORS treatment. Unfortunately, surgical maximalism remains the dominant trend in the treatment of ORS. Many otolaryngologists in our country offer a patient an operation, having discovered even small polyps in the middle nasal passage during anterior rhinoscopy, and they do this without any serious examination and medical preparation. According to P. Clement (2004), 5-7% of all cases of polypous rhinosinusitis are resistant to conservative treatment with steroids and do not recur after surgery. As a rule, they are combined with anatomical defects (curvature of the nasal septum, ridges and spines of the septum, conch bullus, additional anastomosis, etc.).

All of the above clearly defines a group of drugs, the use of which will allow influencing the known links in the pathogenesis of ORS. These are glucocorticosteroids (GCS) - drugs whose effectiveness is not in doubt and has been repeatedly confirmed in controlled clinical trials. At the moment, they are the only means capable of slowing down the growth of polyps and prolonging the periods of remission in ORS.

GCS have long been widely used in the treatment of ORS. These drugs have a pronounced and rapidly manifesting anti-inflammatory and immunosuppressive effect. GCS reduce the number of mast cells and their mediators, as well as the number of eosinophils, T-lymphocytes and Langerhans cells in the mucous membrane of the respiratory tract.

By inhibiting the synthesis of arachidonic acid, corticosteroids reduce the production of prostaglandins and leukotrienes, thereby reducing plasma extravasation and tissue edema. GCS reduce the secretion of the glands of the mucous membrane, the sensitivity of the receptors of the nasal mucosa to histamine and mechanical stimuli. Thus, corticosteroids affect almost all links in the pathogenesis of ORS. For the treatment of ORS and the prevention of polyp growth after surgery, both systemic and topical corticosteroid therapy can be used.

A short course of systemic therapy of corticosteroids, called "medicated polypolytomy", is widely used in the treatment of PRS, and its effectiveness in some cases is not inferior to that of instrumental polypotomy performed using a loop (Khakimov A.M. at all, 2011) For "medicated polypotomy", prednisolone per os is prescribed at a dosage of 0.5-1 mg per 1 kg of the patient's body weight. To prevent side effects, two thirds of the daily dose should be taken

early in the morning, one third - during lunch. The specified dose is prescribed for 10 days, formeanwhile, it is gradually reduced until it is completely canceled on the 14-16th day of

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treatment.

Such a course can be prescribed if there are contraindications to surgical intervention. If polyps recur in a very short time, when both the patient and the doctor are disappointed with the results of repeated operations, short courses of systemic corticosteroid therapy, prescribed no more than 2-3 times a year, can also be an alternative.

Optimal in the treatment of PRS, especially associated with bronchial asthma, aspirin intolerance, nasal and bronchial hyperreactivity, is a combination of corticosteroid therapy and surgical treatment. In these situations, we always perform the intervention against the background of a short course of systemic corticosteroids, prescribing prednisolone 30–40 mg/day (20–30 mg 8 times a day + 10 mg daily) for 3 days before surgery and 3 days after it. We have used this treatment regimen in more than 300 patients. None of them had an exacerbation of bronchial asthma in the postoperative period. Treatment with corticosteroids before surgery reduces the size of polyps, reduces swelling and bleeding of tissues, and allows the intervention to be performed with minimal trauma, while maintaining the anatomical structures and healthy mucosa (Kh A, F., Kh B, D., Kh A, 2001).

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CONCLUSION

Taking into account the fact that the use of THCS in polypous rhinosinusitis both in the pre- and postoperative period requires a long time of use, the drug should have a high safety profile and not cause systemic effects and atrophy of the nasal mucosa. Mometasone furate (MF) is such a drug, the efficacy and safety of which in the treatment of ORS has been proven in numerous randomized placebo-controlled trials. The systemic bioavailability of MF is the lowest among all TGCS and is not detected in the systemic circulation using conventional methods, which makes it possible to prescribe MF from the age of 2 years. In addition, it was proved that

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with prolonged use of MF for 52 weeks, the effectiveness of the drug did not decrease, which indicates the absence of "addiction". The anti-inflammatory effect of Nasonex (MF) contributes to the normalization of the mucous membrane - a decrease in infiltration by inflammatory cells and an increase in the number of ciliated epithelial cells.

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