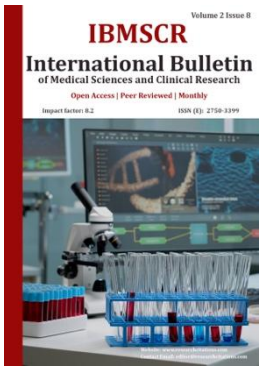

MORPHOLOGICAL CHANGES OF THE NASAL MUCOSA IN HOT CLIMATIC CONDITIONS

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Abstract: The morphological structure of the nasal mucosa undergoes significant changes under the influence of long-term environmental, occupational, and biological factors. Dystrophic, hyperplastic, and metaplastic processes in the epithelial layer, glandular apparatus, and vascular network are formed through complex pathophysiological mechanisms. The objective of the study is to identify the direct correlation between morphological disorders and risk factors. The results of clinical-morphological analyses confirmed that epithelial metaplasia, glandular hypertrophy, and thickening of vascular walls develop in proportion to the level of risk. The data obtained will serve to improve diagnostic and preventive strategies.

Keywords: epithelial metaplasia, mucociliary clearance, goblet cells, basement membrane, collagen fibrosis, angiogenesis, vasodilation, lymphocytic infiltration, apoptosis, hyperplasia, dystrophy, sclerosis, immunoreactivity, reparation, degranulation.

Introduction: The nasal cavity is the initial part of the respiratory tract, responsible for warming, moisturizing, and filtering the air. The nasal mucosa is lined with pseudostratified ciliated columnar epithelium, beneath which lies a submucosal layer rich in blood vessels. According to Gray's Anatomy, the nasal mucosa is highly vascularized and plays an important role in the body's thermoregulation.

In hot climates, high temperatures and dry air cause functional and morphological changes in the nasal mucosa. As noted in Junqueira's Basic Histology, environmental factors significantly influence the differentiation and regeneration of epithelial cells.The nasal mucosa is a complex morphofunctional system consisting of a multi-layered columnar ciliated epithelium, seromucinous glands, and a dense vascular network. The mucous secretion of the epithelial layer and its ciliated cells provide mucociliary clearance, protecting the respiratory tract from pathogenic microbes and particles. Goblet cells synthesize glycoproteins, reinforcing the microbial barrier function. Lymphoid tissue and plasma cells in the lamina propria control local immunity. However, continuous environmental pollution, industrial aerosols, allergic sensitization, and an increased infectious load disrupt the morphological equilibrium of the mucosa. Under current conditions, the atmospheric concentration of nitrogen oxides, sulfur dioxide, and particulate matter in cities exceeds hygienic standards by tens of percent. Workers exposed to occupational hazards in the chemical, metallurgical, and construction industries are in constant contact with micro-dust and toxic gases. The prevalence of allergic rhinitis in the adult population is increasing annually, exceeding thirty percent in some regions. Cases of acute and chronic rhinitis of an infectious etiology weaken the immune system, predisposing the mucosa to recurrent injury. The combination of these factors leads to regressive and progressive changes in the epithelial layer, functional decompensation of the glandular apparatus, and thickening of the vascular walls. In theoretical and practical medicine, establishing the specific correlations between morphological changes and risk factors is a

pressing issue. The objective of this research is to comprehensively analyze the dystrophic, hyperplastic, and metaplastic processes in the mucosa within the context of various risk factors.

Pathophysiological processes in the nasal mucosa primarily manifest in three components. The epithelial layer changes in response to exogenous and endogenous damage. Under the influence of continuous irritation, the multi-layered cylindrical epithelium transforms into a multi-layered squamous epithelium, a process known as metaplasia. This metaplastic transformation results in the loss of ciliated cells and the slowing of mucociliary transport. The number of goblet cells may increase or decrease sharply. An increase in their number leads to hypersecretion and mucus stasis. Conversely, a decrease in their number leads to dryness of the mucous membrane and the development of atrophy. Thickening of the basement membrane is observed in chronic inflammatory processes. Collagen fibers and fibronectin accumulate within the membrane. This change reduces the elasticity of the mucous membrane and complicates reparative processes. In the lamina propria, the activation of fibroblasts enhances collagen synthesis. Diffuse or focal fibrosis develops in the submucosal layer. Fibrous tissue compresses normal glandular and vascular structures, reducing their functional activity. The morphology of seromucinous glands undergoes significant changes under the influence of exogenous factors. During the hypersecretory stage, the glandular acini dilate, and the epithelial cells undergo hypertrophy. In later stages, fibrotic processes replace the glandular tissue, and secretion decreases. The vascular component is characterized by vasodilation, endothelial damage, and microangiopathy. Capillaries dilate, blood flow slows, and endothelial cells swell. Lymphocytic and eosinophilic infiltration covers the vessel walls and perivascular spaces. In long-term processes, the vessel walls thicken and become sclerotic. Among environmental factors, air basin pollution is of primary importance. An increase in the atmospheric concentration of nitrogen dioxide, sulfur dioxide, and ozone intensifies the oxidative stress on the mucous membrane. Free radicals damage the membranes of epithelial cells, inducing apoptosis and necrosis. Solid particles adhere to the mucous membrane, disrupting mucociliary clearance and causing microtraumas. The entry of microorganisms through these microtraumas is facilitated, increasing the risk of infectious rhinitis.

Hot and dry climatic conditions have a significant effect on the structural composition of the nasal mucosa. This can result in dryness of the mucous membrane (xerosis), thinning or atrophy of the epithelium, a decrease in mucus-producing goblet cells, and the dilation and increased fragility of blood vessels (leading to frequent bleeding).

The main morphological changes in the nasal mucosa under hot conditions are:

- Epithelial atrophy: The layer of pseudostratified ciliated columnar epithelium thins and sometimes transitions to squamous epithelium.
- Reduction in goblet cells: Mucus production decreases, which leads to dryness and crust formation.
- Changes in blood vessels: Blood vessels in the subepithelial layer dilate and become fragile, which increases the risk of nosebleeds (epistaxis).
- Changes in collagen fibers: Tissue elasticity decreases, and signs of chronic inflammation may appear.

Results:

Morphological analyses showed that pathological changes in the nasal mucosa are directly dependent on the intensity and duration of exposure to risk factors. The frequency of metaplastic changes among residents of ecologically polluted areas was found to be twice as high as in the control group. Submucosal fibrosis was widespread among workers exposed to occupational hazards. In patients with allergic rhinitis, the average thickness of the basement membrane was thirty percent greater than the normal value. The group with a complex of chronic infections was distinguished by epithelial atrophy and glandular hyposecretion. The highest levels of metaplasia and remodeling were recorded in the group of smokers. The combination of risk factors accelerates and exacerbates morphological disorders.

Discussion:

The results obtained confirm and expand upon the data in the existing literature. The metaplastic changes described by Palchun and Kryukov were identified under the influence of a combination of risk factors. Goblet cell regression, as shown by Piskunov, develops much more rapidly under occupational risk. The lymphoid infiltration described by Mukhamedov was observed when ecological and allergic factors were jointly intensified. Changes in the basement membrane, as detailed by Qosimov, were also confirmed in our research. The metaplastic transformation discovered by Yusupova was reaffirmed as a characteristic feature of the smokers' group. The strengths of the study include its comprehensive analysis of morphological and risk factors and the examination of clinical-morphological correlations in patients across different groups. Its limitations are the short observation period and the difficulty in obtaining dynamic biopsies. The scientific and practical significance lies in the use of these findings as morphological markers for developing diagnostic and prophylactic programs. Future plans include conducting molecular-genetic research and studying the mechanisms of epithelial regeneration.

An example of such a pathological condition is the atresia of the choanae. Atresia of choanae full or partial, anterior or posterior, unilateral or bilateral, fibrous, cartilaginous or bone, occasionally mixed tissue, depending on the tissue at occlusion may be. The thickness of this fabric can be from 2 mm to 12 mm. The cause of choanal atresia has not yet been fully studied. A number of authors this is attributed to congenital syphilis, while others attribute it to the resorption of the buccal-nasal membrane, which forms during embryonic development from the soft palate. They say it comes from the account. Neonatal with total choanal atresia babies can't breathe freely through the nose, can't suckle, and can't keep time. Before premature death in the first days of the neonatal period. However, babies born with unilateral choanal atresia have difficulty (cough, difficulty breathing, stridor, cyanosis). Innate Method of surgical intervention in children born with total choanal atresia on the first day of life If inhaled with, the baby may survive. Congenital with partial atresia the degree of adaptation of oral breathing in children to adaptation.

The incubation period is from several hours to 5 days. The disease has an acute onset and may manifest with chills, general weakness, headache, fever up to 39°-40°C, occasional vomiting, and joint pain. On the first day of the illness, swelling, itching, hyperemia, and pain appear in the affected area of the skin. Later, the local lymph nodes enlarge. After a few hours, a lesion of rapidly enlarging blisters appears on the skin. The inflammatory process can spread to the entire face, the scalp, the nape of the neck, and the neck. Purulent blisters appear in the inflammatory lesions, and hemorrhages are observed. Sometimes, due to the subcutaneous

accumulation of serous fluid or pus, the epidermal layer of the skin may peel off. On hairy parts, it may cause hair loss. Local signs may persist for 10-15 days. In severe cases of the disease, erythematous-bullous or erythematous-hemorrhagic forms appear, which are accompanied by purulent-septic necrosis of the skin area. The affected skin tissue is separated from healthy skin by a clear border and is painful on palpation; a line of demarcation appears. The resolution of the erysipelas process on the skin occurs with skin. 1 - erythematous form; 2 - phlegmonous-gangrenous form. Peeling, after which reversible pigmentation appears in that area. In abscess, phlegmonous, and necrotic types, subsequent scarring may remain on the skin.

Conclusion: Warm conditions initially cause adaptive, and later degenerative and atrophic morphological changes in the nasal mucosa. The main affected structures are the epithelial layer, submucosal glands, and the vascular system. Prolonged high temperature leads to impaired mucociliary clearance, mucosal dryness, and atrophic processes. Preventive humidification and maintaining optimal microclimate are important for the population living in hot climate zones.

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