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Morphofunctional Polymorphism of the Laryngeal Mucosa in Chronic Laryngitis

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Annotation: Chronic laryngitis is a chronic inflammation of the mucous membrane of the larynx, characterized by irreversible damage to the mucous membrane of the larynx. Reactive and reparative processes in the larynx represent the main pathogenetic factor, which can remain at one level if the etiological factors have ceased to act. Inflammation, edema, hyperemia and infiltration and proliferation of the mucous membrane can be represented by various levels of the in situ reaction.

Keywords: morphofunctional polymorphism, laryngeal mucosa.

Introduction. Chronic laryngitis accounts for 8.4% - 10% of all ENT pathologies. Reinke-Hajek COPD accounts for 5.5 - 7.7% of all laryngeal diseases that lead to voice disorders. CGL is more common in men aged 40-60 years, and Reinke-Hajek COPD is more common in

The inflammatory process damages the ciliated epithelium of the larynx, especially the posterior wall. This impairs the important function of mucus movement from the tracheobronchial tree: when the beating movement of the epithelial cilia is impaired, the resulting mucus stasis in the area of the posterior wall of the larynx and around the vocal folds causes reactive cough. Mucus on the vocal folds can manifest laryngospasm. Significant changes can occur in the epithelium of the vocal folds in the form of hyperkeratosis, dyskeratosis, parakeratosis, acanthosis and cellular atypia.

Chronic laryngitis is an inflammatory disease and when studying the microflora, Staphylococcus aureus or its association with viridans streptococcus are most often isolated; less frequently, growth of Candida fungi is detected [3].

The development of the pathological process in the larynx is facilitated by many factors: a person's predisposition to inflammatory diseases of the upper respiratory tract, recurrent acute laryngitis, prolonged and excessive voice strain, alcohol and smoking abuse, dust inhalation and some other occupational hazards (local irritants) that have an adverse effect on the mucous membrane of the larynx and its neuromuscular apparatus. An important role in the development of chronic laryngitis is played by impaired nasal breathing (rhinitis, polyps, deformation of the external nose, curvature of the nasal septum) and unfavorable external climatic conditions of human habitation and work (low and high ambient temperatures, dryness, humidity, dust, gas pollution) in which the patient is forced to constantly breathe through the mouth, which adversely affects the condition of the mucous membrane of the larynx.

Inflammatory phenomena in chronic laryngitis are less pronounced and widespread than in acute catarrhal laryngitis. They develop mainly in the area of the vocal folds and in the interarytenoid space [11].

Depending on the nature of the inflammatory process, there are three main forms of chronic laryngitis: catarrhal, hypertrophic and atrophic. Microscopic examination reveals the following changes: hypertrophy of the laryngeal mucosa, round cell infiltration, vasodilation, changes in the secretory glands, and epithelial metaplasia. In the catarrhal form, congestion is observed, with the mucosa thickening due to round cell infiltration rather than serous impregnation. The flat epithelium of the vocal folds is thickened, the glands of the vestibular folds are enlarged and secrete more secretion. In the hypertrophic form, both the epithelial cover and the submucosal layer are subject to hyperplasia. If these changes occur in individual areas of the larynx, this form is called "limited laryngitis". The changes are expressed in the form of individual nodules, epithelial thickenings, which consist of thickened epithelium and elastic tissue. In chronic hypertrophic laryngitis, attention is drawn to the frequent detection of various reactive changes in these patients, up to the formation of pachydermia, leukoplakia and micropapillomas, the development of adenomatosis, and the loss of protein masses [12].

Hyperplastic processes are most often observed in the area of the posterior vocal folds and the interarytenoid region - pachydermia. However, they can also develop in other areas, including the laryngeal ventricles, and lead to the formation of mucous membrane ridges that extend beyond the ventricles and cover the vocal folds. Hyperplasia of the stratified squamous epithelium without keratinization or with keratinization of the superficial layers contributes to the development of pachydermia, leukoplakia and keratosis. Keratosis is a manifestation of pathological keratinization, corneal dystrophy, which can be caused by trauma, viral infection, vitamin deficiency, chronic inflammation. Pachydermia is based on powerful papillary and strand hyperplasia of the integumentary epithelium in combination with inflammation, reactive proliferation and coarsening of the underlying connective tissue [6].

Leukoplakia is characterized by flat or papillary hyperplasia of the epithelium with a significant increase in the number of layers. The number of cells in the basal and spinous layers increases mainly. In the atrophic form of laryngitis, the mucous membrane of the larynx becomes thin and dry due to the loss of its glandular apparatus. Histologically, pronounced small-cell infiltration is detected in the mucous membrane and submucous layer, especially near the epithelium, around the vessels and glands. As the process develops, the connective tissue thickens, the glands disappear and the lumen of some of the vessels is obliterated, as a result of which the mucous membrane of the larynx becomes thinner, loses its usual color, natural moisture, thick mucus appears on its surface, after drying, which forms crusts.

Chronic laryngitis is divided into catarrhal, hyperplastic, atrophic and edematous-polypous (Reinke-Hayek disease). Specific laryngitis is distinguished separately and, according to some authors, pachydermia of the larynx is defined separately. Limited and diffuse forms of hyperplastic laryngitis are distinguished. Vocal fold nodules, according to some authors, are a form of hyperplastic laryngitis, however, most authors tend to define this pathology as an independent one. Chronic laryngitis is also divided into specific and non-specific [3,7].

The microscopic picture of chronic hyperplastic laryngitis is a combination of reactive changes in the integumentary epithelium and inflammatory, reparative and sclerotic changes in the proper plate of the mucous membrane. The inflammatory infiltrate is characterized by the predominance of mononuclear elements and plasma cells over segmented leukocytes. The predominance of cells of the productive phase of inflammation is the main distinguishing feature of chronic inflammation. The second morphological component is reactive changes in the integumentary and glandular epithelium in the form of its hyperplasia, acanthosis and keratinization disorders, dysplasia of the respiratory epithelium - in the form of basal cell hyperplasia, an increase in the number of goblet cells and squamous cell metaplasia. Pronounced acanthosis of the stratified squamous epithelium is the main pachydermia.

In chronic edematous polypous laryngitis, the signs of inflammatory infiltration do not come to the fore, but rather pronounced interstitial edema, often with stromal myxomatosis, damage to the vascular wall, and thickening of the basement membrane of the integumentary epithelium.

Polypous growths of the vocal cords represent the most common group of benign formations of the vocal folds and account for up to 70% of all causes of voice disorders [1].

The morphological basis of polypoid growths is the formation of persistent edema caused by a high degree of permeability of the capillary bed. In the pathogenesis of these disorders, a significant place is occupied by local inflammation of the vascular wall, of ischemic-hypoxic genesis, caused by increased vocal load (vocal trauma) [10].

The morphological features of chronic edematous polypous laryngitis (Reinke-Hayek disease) include reactive transformation of the epithelium in the form of hyperkeratosis, the appearance of a keratohyalin layer with the presence of pronounced edema of the stroma against the background of a small number of dilated microcavities filled with edematous fluid containing lymphoid cells. Subepidermal and intraepidermal blisters filled with transparent contents are often found, which, in combination with koilocytic transformation of cells, indicates the viral nature of the pathology [1].

One of the pathological manifestations of chronic hypertrophic laryngitis is edema of the tissues that make up Reinke's space (Reinke's edema). Edematous hypertrophy of the vocal folds (Reinecke-Hajek disease) is characterized by damage to their anterior 2/3. The posterior third of the vocal folds is never involved in the process. This is explained by the fact that it is formed by the vocal processes of the arytenoid cartilages and the structure of the mucous membrane here is different. The anterior 2/3 of the vocal folds are represented by edematous tissue, resembling a polyp in appearance, yellow-white or gray in color, freely balloting during phonation and inhalation. The polypous edge of the folds sags into the subglottic space during breathing. The folds are not always symmetrically altered. Diagnosis of edematous hypertrophy of the vocal folds does not cause difficulties due to the characteristic laryngoscopic picture. In Reinke's edema, the medial, inferior and partially superior surfaces of the vocal folds are predominantly affected. In the pathogenesis of this disease, as well as edematous hyperplasia of the vocal folds, vascular disorders in combination with irritating factors (tobacco, alcohol, increased vocal load, bacterial allergy) are of great importance.

In chronic hypertrophic laryngitis, epithelial thickening occurs not only in the vocal folds, but also in other parts of the larynx. Secretion disorders are noted: dry mucous membrane, sometimes accumulation of viscous secretion in the lumen of the larynx. In advanced stages, keratinization of the epithelium is observed. The symptom of selective fluorescein deposition during fluorescence microlaryngoscopy is of great importance for identifying areas of malignancy in foci of laryngeal dyskeratosis (pachydermia, keratosis, leukoplakia), the vascular pattern of which is not determined microlaryngoscopically. In chronic hypertrophic laryngitis, erosion of the epithelium and especially atypical forms of vessels detected against the background of smooth hyperplasia, elevated leukoplakia always raise suspicion of malignancy [5].

An important differential diagnostic sign, indicating the benign nature of the process, is the symmetry of changes in the larynx, while malignant neoplasms in the larynx are always unilateral. It is necessary to differentiate chronic catarrhal laryngitis from primary infiltrative tuberculosis of the larynx, syphilis, scleroma, benign and malignant neoplasms of the larynx. In children, chronic hypertrophic laryngitis is differentiated from laryngeal papillomatosis. Chronic atrophic pharyngitis is differentiated from ozena of the larynx [9].

Consultation. In chronic hypertrophic laryngitis (polyps of the vocal folds, Reinke-Hajek disease, epithelial nodules, intubation granulomas), the microlaryngoscopic picture is characterized by strict localization of the pathological formation against the background of a pale pink epithelial cover with an unchanged vascular pattern. The color of the formations depends on (the nature of the integumentary epithelium and the state of blood circulation). The integumentary epithelium is smooth, "capillaryized", sometimes slightly edematous due to circulatory disorders or thickened due to moderate hyperplasia, keratosis, which, as a rule, indicates the long history of the disease. A microlaryngoscopic feature of the above-mentioned limited proliferative formations was revealed - the transition of vessels from the surrounding mucous membrane to the neoplasm.

References

1. Быкова В.П., Кочесокова Э.А., Иванченко Г.Ф., Ротова И.Д. Морфология полипов и слизистой оболочки голосовых складок при болезни Рейнке // Архив патологии. 2015. Т.77 № 1. С. 30-37. DOI: 10.17116/patol201577130.
2. Дайхес Н.А., Быкова В.П.,. Пономарев А.Б,. Давудов Х.Ш. «Клиническая патология гортани. Руководство-атлас».- М.- Медицинское информационное агентство.2009.- 160с.
3. Донецкая Э.Г. «Клиническая микробиология: руководство».-М.-2011. С.131
4. Иванченко Г.Ф., Демченко Е.В., Быкова В.П., Григорян С.С. Клинические особенности и терапия больных хроническим гиперпластическим ларингитом: Метод. Рекомендации.- М., 1998.- 9 с.
5. Иванченко Г.Ф., Демченко Е.В., Быкова В.П., Григорян С.С. Клинические особенности и терапия больных хроническим гиперпластическим ларингитом: Метод. Рекомендации.- М., 1998.-9 с.
6. Кунельская В.Я. «Микозы в оториноларингологии».- М.-«Издательство «Медицина».-1989.- 225 с.
7. Крюков А.И., Кунельская Н.Л., Романенко С.Г., Павлихин О.Г.,. Елисеев О.В, Яковлев В.С., Красникова Д.И., Лесогорова Е.В. Терапия воспалительных заболеваний гортани». РМЖ . - 2013. -№ 2. - с.38-41
8. Романенко С.Г. Острый и хронический ларингит. Оториноларингология. Национальное руководство. Краткое издание под ред. В.Т. Пальчуна. М.:ГЭОТАР-Медиа, 2012, с. 541-547.
9. Самиева Г. У. Современные методы лечения острого стенозирующего ларинготрахеита у детей (обзор литературы) //Молодой ученый. – 2014. – №. 11. – С. 149-151.
10. Самиева Г. У. Дисбиотические расстройства верхних дыхательных путей у детей с острым стенозирующим ларинготрахеитом //Медицинские новости. – 2015. – №. 7 (250). – С. 70-71.
11. Самиева Г. У., Карабаев Х. Э. Клинические особенности течения рецидивирующих стенозирующих ларинготрахеитов у детей //Академический журнал Западной Сибири. – 2013. – Т. 9. – №. 2. – С. 6-6.
12. Robert P. Ossof, Stanley M. Shashay et. All. The Larynx, Lippincott Williams & Wilkins, 560 p.