Etiopathogenetic mechanisms of vocal nodules and Reinke's edema development and their morphopathological manifestations

Sumerags Dins [1], Pilmane Māra [2], Segliņa Gunta [3]

[1] *Cesu Klinika Hospital, Latvia*
[2] *Institute of Anatomy and Anthropology, Rīga Stradiņš University, Latvia*
[3] *Department of Otorhinolaringology, Rīga Stradiņš University, Latvia*

Objectives. Vocal nodules and Reinke's edema are common benign vocal fold diseases associated with vocal fold overuse, smoking, and other environmental factors. The precise etiopathogenetic mechanisms are not fully understood. Thus, our aim was to study proliferation, programmed cell death, inflammation, and ischemia markers to reveal complex cellular and tissue-level interactions contributing to the pathogenesis of these conditions.

Materials and methods. Vocal nodules were obtained from 10 females (aged between 17 and 56) and five Reinke's edema-affected vocal cords (58 to 71 years old). Controls were obtained from the vocal cords of seven cadavers (aged 40–70 years). Ki-67, PGP 9.5, IL-10, IL-1α, EGFR, VEGF, and apoptosis were detected by the biotin–streptavidin immunohistochemistry.

Results. In the case of vocal nodules, a significant increase in proliferation markers (Ki-67), growth factors (EGFR), ischemia (VEGF), and inflammation (IL-1α) have been observed. These findings indicate a persistent inflammatory and ischemic environment, leading to hyperplastic changes in the tissue. Simultaneously, the intensification of apoptosis and its strong correlation with proliferation and inflammation suggest complex interactions maintaining the formation of vocal nodules.

Studies on Reinke's edema reveal an intense proliferation of the surface epithelium, associated with chronic inflammatory processes due to smoking and other external factors. Increased IL-10 marker expression indicates a pronounced anti-inflammatory response, attempting to compensate for the epithelial damage caused by inflammation. Furthermore, higher IL-1α and PGP 9.5 expression is associated with inflammation, proliferation, and tissue remodeling processes in Reinke's edema.

Conclusions. These studies suggest that developing vocal nodules and Reinke's edema in the vocal folds are linked to similar etiopathogenetic mechanisms, where inflammation, cellular growth, and ischemic changes dominate. Further analysis of these markers could provide new opportunities in diagnosing and treating these diseases, potentially leading to more targeted therapeutic approaches that address pathological processes at the cellular level.