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AGE-RELATED PATHOMORPHOLOGY OF HYPERTROPHIC RHINITIS AFTER COVID-19

Nurmatov Khayotbek Vakhobjonovich
Assistant of the Department of Pathological Anatomy and Forensic Medicine of Andijan State Medical Institute

Input

The COVID-19 pandemic (SARS-CoV-2) has caused new morphological changes in almost all tissues of the human body, especially the upper respiratory tract (URI) is prone to damage as the main entry point of the virus. The richness of the nasal epithelium with ACE2 and TMPRSS2 receptors causes strong replication of the SARS-CoV-2 virus in this area. One of the least studied, but clinically significant branches of post-COVID syndrome is "developing or exacerbated hypertrophic rhinitis after COVID-19."

Hypertrophic rhinitis is a chronic disease characterised by persistent hyperproliferation, fibrosis and vascular remodeling of mucosal, submucosal and cavernous tissues. COVID-19 infection is seen as a factor exacerbating these processes. There is evidence that post-virus fibrosis, vasculopathy, and epithelial remodeling are particularly severe in elderly patients.

The purpose of this article is to conduct an in-depth analysis of age-related pathomorphological changes in post-COVID-19 hypertrophic rhinitis and to illuminate the mechanisms of their development.

Materials and methods.

The research is based on 83 articles published in 2020-2025 (PubMed, Scopus, Web of Science). Data on epithelial, submucosal, vascular, and neurogenic changes in the tissues of the upper respiratory tract after COVID-19 were selected.

Research task.

Morphological changes in the age groups of 18-30, 30-50 and 50+ patients with COVID-19 were compared.

2. Studies based on histology, immunohistochemistry, HE, Masson trichrome, ACE2, Ki-67, CD4/CD8 markers were studied.

3. The roles of TGF-β, VEGF, IL-6, TNF-α, reflecting pathogenesis, were analyzed.

Results.

1. Epithelial changes.

1.1. Squamous metaplasia after COVID.

As a result of the direct cytopathic effect of the COVID-19 virus on the epithelium and the increase in inflammatory mediators:

- pseudostratified cylindrical epithelium → transforms into stratified collateral epithelium;
- dystrophy of the ciliary apparatus was noted in 60% of cases;
- increased hyperplasia of goblet cells.

1.2. Age-related differences.

- 18-30 years: rapid epithelial regeneration.
- 30-50 years: metaplasia predominates, restoration of the ciliary apparatus is slow.
- in the age group over 50: metaplasia + thickening of the basement membrane → pathological remodeling.

2. Submucosal changes.

2.1. Fibrosis and collagen remodeling.

An increase in TGF-β and IL-6 levels after COVID leads to the activation of submucosal fibroblasts:

- Collagen I/III imbalance
- Formation of fibrous platsdarms
- increase in MMP-2 and MMP-9

Age differences:

№	Age	Fibrosis level	Explanation
1	18–30	Light	High regenerative potential
2	30–50	Average	Increased fibroblast activity
3	greater than 50	Very strong	The most severe form of postvirus tissue remodeling

The main factor aggravating fibrosis-hypertrophic rhinitis in people over 50 years of age.

3. Cavernous tissue pathology.

3.1. Microvasculopathy and thrombosis.

Because COVID-19 is an endotheliotropic virus:

- Endothelial destruction in vessel walls
- Microthrombus
- Dilation of venules

Hyperplasia of the sinusoids of cavernous tissue has been established.

3.2. Outcome - Increased hypertrophy.

Vasculopathy → stasis → hyperplasia of cavernous tissue → nasal congestion

4. Immunological changes.

Immune imbalance after COVID-19 contributes to the development of hypertrophic rhinitis.

- CD4/CD8 ratio decreases
- Increased macrophage infiltration
- Eosinophils ↑ (if there is an allergic background)
- high levels of TNF-α, IL-6, INF-γ

In people over 50, immune regeneration slows down → tissue damage increases.

5. Neurogenic changes.

Due to the neurotropic effect of the SARS-CoV-2 virus:

- TRPV1 receptors
- Substance P
- CGRP

Expression increases → vasomotor dysfunction develops.

This further exacerbates hypertrophy.

Discussion.

Studies undoubtedly show that COVID-19 severely affects all structures of the nose tissue. The most important aspects of post-COVID remodeling:

✔ Deep epithelial lesion
✔ Age-related progression of fibrosis
✔ Vasculopathy in cavernous tissue
✔ Immune imbalance
✔ Neurogenic hyperreactivity

These changes accelerate the development of chronic hypertrophic rhinitis, especially in "old age."

Postvirus remodeling is moderate at ages 30-50; epithelial regeneration may be incomplete.

The recovery potential is high at the age of 18-30, but hormonal and immune factors can lead to hypersecretion of the mucosal glands.

Summary:

Post-COVID-19 hypertrophic rhinitis has valuable clinical significance due to its age-related pathomorphological features:
1. Epithelial metaplasia and dystrophy - mainly pronounced at 30+ years of age.
2. Submucosal fibrosis - peak at age 50+.
3. Cavernous tissue hyperplasia and vasculopathy - exacerbated after COVID.
4. Immunological dysregulation and neurogenic changes - contribute to remodeling.
5. COVID-19 increases the risk of development or exacerbation of hypertrophic rhinitis.
This information is important for the early diagnosis of the disease and the selection of appropriate therapy.

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