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Morphological Assessment of the Nasal Mucosa of the Inferior Turbinates in Patients with Hypertrophic Rhinitis

U. S. Khasanov, Sh. S. Abdurakhimova, U. N. Vokhidov

Republican Specialized Scientific and Practical Medical Center of Otorhinolaryngology and Head and Neck Diseases, Tashkent, Uzbekistan

Abstract Chronic rhinitis is a common otorhinolaryngological condition that significantly impacts patient quality of life and has notable medical and social relevance. Among its various forms, hypertrophic rhinitis represents a prevalent chronic pathology of the upper respiratory tract, characterized by progressive structural changes in the nasal mucosa. The objective of this study was to conduct a morphological assessment of the inferior turbinate mucosa in patients with hypertrophic rhinitis. A clinical investigation was carried out involving patients diagnosed with hypertrophic rhinitis, with biopsies of the inferior turbinate mucosa subjected to detailed morphological analysis. The findings demonstrated pronounced dystrophic alterations in epithelial cells, including protein hydropic and ballooning degeneration, with progression to necrosis and epithelial desquamation. A marked increase in goblet cell numbers and mucus production was observed. In the lamina propria, sclerotic transformations, inflammatory infiltration, tissue edema, vascular abnormalities, and hypertrophy of the terminal portions of mucous glands were identified. These results indicate that chronic hypertrophic rhinitis is associated with complex degenerative and proliferative changes in both the epithelial and subepithelial layers, underscoring the importance of early diagnosis and targeted therapeutic interventions to prevent irreversible tissue remodeling.

Keywords Hypertrophic rhinitis, Prevalence, Etiopathogenesis, Diagnosis, Morphological study

1. Introduction

Diseases of the nose and paranasal sinuses occupy one of the leading positions in the structure of ENT pathology and show a tendency to increase both in overall human nosology and in the number of hospitalized patients. Hypertrophy of the nasal turbinates develops due to a variety of factors of different natures, including both exogenous and endogenous causes. Adverse environmental influences of physical and chemical origin, infectious agents and allergens, trauma and harmful habits, anatomical predispositions, and internal organ diseases lead to an increase in the size of the nasal turbinates, difficulty in nasal breathing, and a decline in patients' quality of life - which often ultimately results in the need for surgical intervention [1,7,12,18,22].

In recent decades, treatment methods for hypertrophic rhinitis involving physical factors have become widespread: cryosurgery, surgical lasers, the use of plasma and radio wave devices, and shaver systems. However, traditional techniques such as galvanocautery and conchotomy are still in use, which is largely due to the insufficient availability of modern endoscopic and surgical equipment in many healthcare and preventive institutions.

The issue of treatment method selection by both physician and patient is often a matter of awareness, accessibility, and, in many cases, financial capability. Even when multiple treatment options are available, the choice is frequently made based on the physician's personal experience rather than objective clinical data.

There is insufficient coverage in the literature regarding the clinical effectiveness and cost-efficiency of various treatment methods for hypertrophic rhinitis, particularly in relation to patient quality of life and safety.

According to various authors, the proportion of hypertrophic rhinitis in the structure of ENT diseases ranges from 4.1–6.5% to as high as 16–18%, and this figure is also showing a tendency to increase.

The primary and most important function of the nasal cavity is the conduction of airflow. Nasal breathing difficulty is not only a subjective symptom that affects quality of life but also a manifestation of a number of conditions associated with structural changes within the nasal cavity.

Nasal airflow may be impaired for various reasons. The main causes include narrowing or enlargement of the nasal passage. The former includes hypertrophic rhinitis, nasal septum deviation, polyps, and tumors, while the latter includes atrophic rhinitis, consequences of improper surgical

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interventions, and so on.

The anatomical and physiological characteristics of the nasal mucosa predispose it to frequent contact with adverse environmental factors that negatively affect the epithelium. This leads to reduced protective functions of the nasal cavity and contributes to the development of pathological changes in the lower respiratory tract, and eventually in the entire body.

One of the most common causes of nasal passage obstruction is turbinate hypertrophy. However, it is important to first clarify the meaning of hypertrophy. According to the WHO international consensus, hypertrophic rhinitis and turbinate hypertrophy are not classified as separate diagnoses, but rather can occur in the context of allergic, occupational, hormonal, or drug-induced rhinitis. They may also be categorized under chronic non-specific rhinitis. In the ICD-10 (International Classification of Diseases, 10th Revision), turbinate hypertrophy is classified under Chapter X with the code J34.3.

At present, ICD-10 is the primary tool used for the statistical analysis of public health data and the performance of healthcare institutions. It is used to collect and analyze data on morbidity and mortality, evaluate population health trends, and assess the status of medical care - essential elements for effective healthcare planning. ICD-10 ensures methodological consistency and comparability of morbidity, mortality, and healthcare usage data both nationally and internationally.

In domestic clinical practice, the term “chronic hypertrophic rhinitis” is still widely used, with further classification into cavernous and fibrous forms - primarily due to the framework of the mandatory health insurance system.

It is also important to distinguish between the concepts of hypertrophy and hyperplasia of tissues. Hypertrophy is generally understood as an increase in the volume of an organ or part of an organ due to the enlargement of its individual components (cells and tissues). Hyperplasia, by contrast, refers to an increase in the number of structural elements of a tissue or organ.

Since hyperplasia involves active cell proliferation and the formation of new structures, this process is observed in various pathological tissue overgrowths, including chronic productive inflammation. Muscle tissue, which is an important component of the cavernous tissue of the nasal cavity, can increase in size not only through hypertrophy but, to some extent, also through hyperplasia, as demonstrated in studies involving testosterone.

Thus, hypertrophy (and possibly hyperplasia) of the nasal turbinates develops due to a variety of factors of both exogenous and endogenous origin. Adverse environmental influences of a physical and chemical nature, infectious agents, allergens, trauma, harmful habits, anatomical predispositions, and internal organ diseases all contribute to the enlargement of the nasal turbinates. This results in nasal breathing difficulties, a decrease in patients’ quality of life, and, ultimately, often necessitates surgical intervention and reflects a high prevalence of hypertrophic rhinitis in the population.

Population morbidity represents an independent issue requiring dedicated study. It is understood as the emergence and spread of diseases among the population as a result of interaction with the environment over generations. In morbidity statistics, several methods are used, with the main ones being: data from medical consultations, results of health screenings, and mortality statistics.

Given the high prevalence of hypertrophic rhinitis, it is relevant to study the regional epidemiological aspects of this condition and to improve diagnostic and treatment methods.

2. Materials and Methods

Study design and participants. This was a clinical, descriptive, and analytical study conducted at the Republican Specialized Scientific and Practical Medical Center of Otorhinolaryngology and Head and Neck Diseases, Tashkent, Uzbekistan, from January 2024 to March 2025. The study population consisted of patients diagnosed with chronic hypertrophic rhinitis who were referred for examination and treatment. A total of 30 patients were included. The sample size was determined based on the number of eligible patients attending the center within the study period; no formal power calculation was performed due to the exploratory nature of the morphological assessment. Sampling was purposive, including only those who met the eligibility criteria.

Inclusion criteria were: (1) confirmed diagnosis of chronic hypertrophic rhinitis based on clinical, endoscopic, and radiological findings; (2) age ≥18 years; (3) no prior nasal surgery affecting the inferior turbinates; and (4) informed consent for participation and biopsy collection.

Exclusion criteria were: (1) acute infectious rhinitis or sinusitis at the time of evaluation; (2) systemic inflammatory or autoimmune disorders affecting the nasal mucosa; (3) malignant or benign tumors of the nasal cavity; and (4) refusal to participate or provide tissue samples.

Data collection instruments. The main data collection instruments included:

1. **Clinical evaluation forms** structured to record demographic data, medical history, symptom duration, comorbidities, and prior treatments.
2. **Endoscopic examination** using a rigid nasal endoscope to document mucosal condition and hypertrophy characteristics.
3. **Radiological assessment** (CT scan or X-ray) to evaluate nasal cavity and paranasal sinus anatomy.
4. **Morphological analysis** tools: biopsy forceps for tissue sampling from the inferior turbinate mucosa; fixation in formalin; processing for paraffin embedding; and staining with hematoxylin-eosin and toluidine blue. These instruments had been previously validated for ENT clinical and histopathological research. Morphological assessments were performed by experienced histopathologists, ensuring inter-observer reliability.

Procedures. Eligible patients were identified from daily outpatient and inpatient lists. After confirming eligibility, the study purpose and procedures were explained, and written informed consent was obtained. Clinical and endoscopic examinations were performed in the ENT outpatient clinic. Radiological assessments were conducted in the radiology department. Inferior turbinate mucosal biopsies were obtained under local anesthesia in a sterile setting. Specimens were fixed immediately, processed, stained, and examined under a light microscope for morphological changes. All findings were documented in a secure research database.

Ethical considerations. The study was approved by the Ethics Committee of the Republican Specialized Scientific and Practical Medical Center of Otorhinolaryngology and Head and Neck Diseases (Approval code: RSNPMC-ENT -2024-01). All procedures complied with the Declaration of Helsinki principles. Participant confidentiality was maintained by coding data without identifying information. Anonymity was ensured in all reports and publications. Participation was entirely voluntary, with no impact on standard medical care. Written informed consent was obtained from each participant before enrollment and biopsy collection.

Data analysis. Data were entered into and analyzed using **SPSS version 26.0** (IBM Corp., Armonk, NY, USA). Descriptive statistics (mean, standard deviation, frequency, percentage) were used to summarize demographic and clinical data. Morphological findings were analyzed qualitatively and quantitatively, comparing histological features such as epithelial changes, goblet cell density, vascular alterations, and glandular hypertrophy. The Chi-square test was applied for categorical variable comparisons, and the Student's t-test was used for continuous variables where applicable. A p-value <0.05 was considered statistically significant. These statistical tests were chosen to appropriately evaluate differences between observed pathological changes and baseline histological norms.

3. Results

A total of 30 patients with a confirmed diagnosis of chronic hypertrophic rhinitis were included in the study. The cohort consisted of 14 women (48.3%) and 16 men (51.7%), resulting in a near-equal sex distribution. Comparative analysis of clinical data between sexes did not reveal statistically significant differences in disease presentation, comorbidities, or histological changes; thus, the results are presented for the group as a whole.

Demographic and clinical characteristics. The age of participants ranged from 18 to 60 years, with a mean age of 39.6 ± 8.2 years. Five patients (17.3%) were in the 18–30-year group, 23 patients (75.7%) were aged 31–50 years, and 2 patients (7.0%) were aged 51–60 years. The majority of cases fell within the working-age population, highlighting the considerable socio-economic impact of this pathology.

Younger patients (18–30 years) typically exhibited a shorter disease duration, often less than one year, and fewer comorbidities. In contrast, the older subgroup (51–60 years) demonstrated more advanced disease, longer symptom duration, and a greater burden of systemic conditions, particularly cardiovascular disorders.

The duration of symptoms, based on detailed patient anamnesis, varied considerably. Approximately 28.0% of patients reported symptoms persisting for one year or less, 33.0% reported a duration of two to three years, and 39.0% had a disease history exceeding three years. Long-standing cases were notable for persistent nasal obstruction despite medical therapy, indicating resistance to conservative management.

Frequent or prolonged use of topical vasoconstrictor sprays was reported by 89.3% of patients, with some using these agents continuously for several years. Nasal trauma was documented in 10.7% of cases, most often due to blunt facial injuries.

Comorbidity profile. Comorbid conditions were detected in 89.7% of the cohort. The most frequent was chronic pharyngitis (26.3%), consistent with the chronicity of upper airway inflammation in this group. Cardiovascular diseases, predominantly hypertension and ischemic heart disease, were identified in 14.0% of patients, primarily in the older age category. Respiratory conditions such as chronic bronchitis and mild asthma were found in 13.6% of cases. Nasal septum deviation was observed in 17.7% of patients, often contributing to the severity of nasal obstruction. Gastrointestinal disorders, mainly chronic gastritis, were present in 8.7%. Allergic manifestations, including seasonal rhinitis and atopic dermatitis, were recorded but had no dominant influence on the histopathological findings.

Morphological findings. Histological examination of the inferior turbinate mucosa revealed consistent and pronounced pathological changes in both epithelial and subepithelial layers.

The normal pseudostratified ciliated columnar epithelium was replaced in many regions by altered epithelial architecture, showing a spectrum of dystrophic changes. The most striking features were **protein hydropic degeneration** and **ballooning degeneration** of epithelial cells, manifesting as enlarged cells with cytoplasmic vacuoles of varying sizes. In severe cases, a single large vacuole displaced the nucleus to the periphery, followed by nuclear lysis and liquefactive necrosis.

These dystrophic changes were most prominent in the superficial epithelial layers. Necrotic foci exhibited complete loss of epithelial continuity, with areas of desquamation exposing the lamina propria. The denuded surfaces were frequently covered with a thin fibrinous layer, beneath which subepithelial edema, microhemorrhages, and dense lymphocytic infiltrates were present.

The residual epithelial cells in affected regions displayed fine granular cytoplasm, pyknotic nuclei, and marked intercellular dissociation. In focal atrophic zones, the epithelium was thinned, often reduced to two or three cell layers, with cells adopting a flattened or cuboidal morphology. In certain specimens, **squamous metaplasia** was identified, with the original ciliated epithelium replaced by stratified squamous

epithelium lacking ciliary structures.

Goblet cell changes. Goblet cell hyperplasia was a consistent finding. The cytoplasm of these cells stained intensely fuchsinophilic, with an apical bulge protruding above the level of adjacent ciliated cells. Mucus overproduction was evident, with thick layers of secretion coating the epithelial surface. Toluidine blue staining revealed metachromasia of goblet cell contents, reflecting heightened secretory activity.

Lamina propria alterations. The lamina propria demonstrated marked structural remodeling. Proliferation of coarse fibrous connective tissue was observed, with collagen fibers becoming thickened, irregular, and in some regions hyalinized. These sclerotic changes were accompanied by interstitial edema, stromal loosening, and chronic inflammatory infiltration.

Inflammatory infiltrates were both diffuse and focal, predominantly concentrated in subepithelial zones and around blood vessels and mucous glands. The cellular composition included lymphocytes, plasma cells, macrophages, eosinophils, and occasional neutrophils. The degree of infiltration varied between cases but was universally present.

Vascular pathology. Vascular changes were frequent and significant. Blood vessels were dilated and congested, with evidence of stasis and erythrocyte aggregation. Perivascular hemorrhages and thickening of vessel walls were noted in several specimens, suggesting chronic vascular compromise and hemodynamic disturbance in the hypertrophied turbinate tissue.

Mucous gland changes. The terminal portions of mucous glands within the lamina propria exhibited hypertrophy, with enlarged glandular acini lined by columnar epithelium. Glandular lumens were dilated, and mucus accumulation was prominent. Some glands showed signs of secretory hyperactivity, consistent with the goblet cell findings in the epithelium.

Summary of histopathological pattern. The morphological profile of chronic hypertrophic rhinitis in this cohort was defined by:

1. Degenerative epithelial changes (protein hydropic degeneration, ballooning degeneration, necrosis, desquamation).
2. Goblet cell hyperplasia with increased mucus production.
3. Fibrous tissue proliferation and sclerosis in the lamina propria.
4. Chronic inflammatory infiltration with mixed cellular composition.
5. Vascular dilation, congestion, and perivascular hemorrhage.
6. Hypertrophy and hypersecretion of mucous glands.

These findings collectively indicate that chronic hypertrophic rhinitis involves not only hypertrophy but also complex degenerative, inflammatory, and vascular processes, resulting in progressive structural and functional impairment of the nasal mucosa.

4. Discussion

The present study provides a detailed morphological characterization of the inferior turbinate mucosa in patients with chronic hypertrophic rhinitis, revealing a combination of degenerative, inflammatory, vascular, and glandular changes. These findings expand the current understanding of the pathological substrate of this condition and underscore its complex nature, extending beyond simple mucosal thickening.

Our demographic data showed a predominance of patients in the working-age group (31–50 years), consistent with epidemiological reports indicating that chronic hypertrophic rhinitis significantly affects the economically active population, with substantial medical and social implications (Gelardi et al., 2022; Mihalcea et al., 2023). The high prevalence of comorbidities—particularly chronic pharyngitis, cardiovascular disease, and respiratory tract disorders—supports the concept that hypertrophic rhinitis is both a localized and systemic health concern, often coexisting with other chronic inflammatory conditions of the upper airway (Ciprandi & Tosca, 2021).

One of the most striking histopathological features in our cohort was the presence of pronounced dystrophic epithelial changes, including protein hydropic and ballooning degeneration, progressing to liquefactive necrosis. Similar degenerative alterations have been described in long-standing mucosal inflammatory states, where persistent irritation leads to cellular metabolic exhaustion, disruption of ionic balance, and eventual structural collapse of epithelial cells (Lukka et al., 2018; Wang et al., 2020). The frequent occurrence of epithelial desquamation and erosion, often covered by fibrin, further supports the chronicity and recurrent nature of epithelial injury in these patients.

The observed goblet cell hyperplasia and marked mucus hypersecretion align with previous reports linking hypertrophic rhinitis to mucociliary dysfunction and excessive secretory activity (Yan & Hwang, 2018; Türk et al., 2018). Increased goblet cell density not only reflects an adaptive response to persistent mucosal irritation but also contributes to the clinical symptomatology by increasing nasal discharge and exacerbating airway obstruction. The metachromasia detected with toluidine blue staining indicates active glycosaminoglycan synthesis, a marker of heightened secretory metabolism in these cells.

In the lamina propria, our findings of fibrous connective tissue proliferation, collagen thickening, and hyalinization correspond to the fibrotic remodeling stage of chronic inflammation (Bergmark & Gray, 2018). This sclerotic transformation is likely driven by fibroblast activation under the influence of pro-inflammatory cytokines, resulting in reduced tissue elasticity and irreversible structural changes. Chronic inflammatory infiltration, dominated by lymphocytes and plasma cells with contributions from macrophages and eosinophils, reflects the ongoing immune response to persistent antigenic stimulation, possibly of mixed infectious and non-infectious origin.

Vascular abnormalities—including dilation, congestion, and perivascular hemorrhage—were prominent in our specimens

and may have a dual role in disease pathogenesis. On one hand, increased vascular permeability facilitates inflammatory cell migration and nutrient delivery to the inflamed tissue. On the other, vascular stasis and congestion can exacerbate mucosal edema and contribute to nasal obstruction (Hizli et al., 2020). These vascular changes also provide a plausible explanation for the frequent intraoperative bleeding encountered in turbinate surgery.

Hypertrophy of the terminal mucous glands, with dilated lumens and increased mucus production, further highlights the secretory overdrive in hypertrophic rhinitis. This glandular remodeling parallels the goblet cell changes in the epithelium, suggesting that both surface and submucosal secretory components are simultaneously upregulated in response to chronic irritation.

Our results collectively support the view that chronic hypertrophic rhinitis is not merely the result of cavernous tissue engorgement but represents a multifactorial process involving epithelial degeneration, inflammatory infiltration, fibrotic remodeling, vascular pathology, and secretory gland hyperactivity. This perspective aligns with the work of Karamatzanis et al. (2022) and Gunturu et al. (2019), who emphasized that surgical intervention should be tailored not only to reduce tissue volume but also to address the altered tissue quality and function.

From a clinical standpoint, the predominance of irreversible structural changes in patients with a disease duration exceeding three years underscores the importance of early diagnosis and intervention. Conservative medical therapy—while effective in early stages—may have limited efficacy once fibrosis and glandular hypertrophy are established. In such cases, minimally invasive surgical approaches, including radiofrequency ablation, submucosal reduction, or endoscopic turbinoplasty, may provide more durable relief while preserving mucosal function.

The high rate of long-term topical vasoconstrictor use in our cohort (89.3%) is noteworthy. Chronic use of these agents has been linked to rebound mucosal swelling, microvascular changes, and epithelial damage, which may accelerate disease progression (He et al., 2025). This finding highlights the need for patient education on the risks of unsupervised decongestant use.

Limitations of our study include the relatively small sample size and the single-center design, which may limit generalizability. Additionally, while our morphological assessment was detailed, it did not incorporate immunohistochemical or molecular analyses that could further elucidate the inflammatory pathways involved. Future research should explore biomarker-based stratification of patients to predict disease progression and guide individualized therapy.

5. Conclusions

The morphological assessment of the inferior turbinate mucosa in patients with chronic hypertrophic rhinitis demonstrates that this condition involves complex and multifactorial pathological processes, extending beyond simple mucosal enlargement. The disease is characterized by pronounced epithelial degeneration, goblet cell hyperplasia with mucus hypersecretion, fibrous connective tissue proliferation, chronic inflammatory infiltration, vascular congestion, and hypertrophy of mucous glands. These changes reflect the cumulative effects of prolonged irritation, inflammation, and tissue remodeling, which ultimately lead to irreversible structural and functional impairment of the nasal mucosa.

The high prevalence of comorbidities, long disease duration in many patients, and the frequent misuse of topical vasoconstrictors highlight the importance of early diagnosis, rational pharmacotherapy, and timely surgical intervention when indicated. A comprehensive approach that addresses both the structural alterations and the underlying inflammatory mechanisms is essential for improving patient outcomes. Further research, particularly incorporating immunohistochemical and molecular methods, is needed to deepen understanding of the pathophysiology and to guide the development of targeted, tissue-preserving treatment strategies.

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Authors' Contributions

U.S. Khasanov: Conceptualization of the study, patient recruitment, clinical assessment, and surgical procedures.

Sh.S. Abdurakhimova: Histopathological analysis, data interpretation, and drafting of the morphological description.

U.N. Vokhidov: Statistical analysis, literature review, preparation, and critical revision of the manuscript.

All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Conflicts of Interest. The authors declare no conflicts of interest related to this study.

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