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Chronic Nasal Congestion

Toshmamatov B. N., Djumanova N. E
Zarmed University, Samarkand, Uzbekistan

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Annotation: Chronic nasal congestion is a long-lasting condition characterized by persistent obstruction of the nasal passages due to inflammation, mucosal swelling, or structural abnormalities. It is not a disease itself but a symptom commonly associated with various underlying disorders such as chronic rhinitis, sinusitis, nasal polyps, deviated nasal septum, or allergic reactions.

The main pathophysiological mechanisms include prolonged inflammation of the nasal mucosa, vascular dysregulation, and mucous hypersecretion. Common causes are allergic rhinitis, non-allergic (vasomotor) rhinitis, chronic sinus infections, environmental irritants, and prolonged use of nasal decongestant sprays.

Clinical manifestations include difficulty in nasal breathing, mouth breathing, snoring, reduced sense of smell, and postnasal drip. Chronic congestion significantly impairs sleep quality, cognitive function, and overall quality of life.

Diagnosis is based on clinical evaluation, nasal endoscopy, allergy testing, and imaging studies when structural causes are suspected. Treatment depends on the etiology and may include intranasal corticosteroids, antihistamines, saline irrigation, surgical correction, or allergen immunotherapy.

Timely identification and management of chronic nasal congestion are essential to prevent complications such as chronic sinusitis, otitis media, and sleep-disordered breathing.

Introduction

Chronic nasal congestion is one of the most common conditions encountered in otorhinolaryngology practice. It is characterized by persistent difficulty in nasal breathing caused by inflammation, mucosal edema, or structural abnormalities within the nasal cavity.

This condition has significant **medical and social importance**, as it leads to a decrease in quality of life, sleep disturbances, reduced concentration, and lower work productivity. In adults, chronic nasal congestion is often associated with chronic rhinitis, sinusitis, nasal polyps, or a deviated nasal septum.

The **etiology** of chronic nasal congestion is multifactorial and includes **allergic reactions**, **non-allergic (vasomotor) mechanisms**, **infectious processes**, exposure to **environmental irritants**, and **drug-induced causes**, such as prolonged use of topical decongestants.

Timely diagnosis and appropriate treatment are essential to prevent complications such as **chronic sinusitis**, **eustachian tube dysfunction**, and **sleep-related breathing disorders**, including **obstructive sleep apnea**.

An active chemical mediator called pharmacollagrcall is released during nasal allergic reactions, and this can contribute to the etiology of persistent rhinitis. Furthermore, rejection mechanisms most likely have a significant impact on the start and persistence of chromosomal abnormalities. The most effective method of diagnosis is a thorough history that pays close attention to exposures from the family, workplace, and environment. Additionally, a selective epicutaneous skin test and repeated smears of nasal secretions are used to look for eosinophils. The treatment entails avoiding any dust, fumes, sprays, and similar substances as well as paying attention to minimizing occupational and home environmental exposures.

The most typical indications and symptoms of chronic rhinorrhea, postnasal drip, nasal itching, nasal congestion, and occasionally headache, especially if sinusitis is causing complications. In severe cases, patients develop a persistent mouth breathing habit and experience mucous membrane dryness in the oropharynx. Some patients develop a neurotic reaction to their nasal symptoms, see doctors regularly, and experiment with a wide range of over-the-counter nasal treatments, including topical nasal decongestants, to which they may develop a tolerance and develop rhinitis medicamentos as a result.

Chronic allergic rhinorrhea. Individuals who suffer from perennial allergic rhinitis are susceptible to allergens, which cause chronic symptoms, through an IgE-dependent mechanism of mast cell pertubation. Dust mites, cockroaches, feathers, pet animals (including those exposed to animals at work, like veterinarians and lab researchers), insect debris, various mold spore and pollen allergens, and other occupational allergens may trigger an allergic reaction in these people. A physical examination will reveal nasal turbinate mucosa congestion, airflow restriction, and frequently some mucoid nasal discharge.

Individuals diagnosed with persistent allergic rhinitis typically have positive skin test reactions, such as wheals and erythema, to a variety of allergens, along with high levels of total serum IgE. Examining nasal cells frequently (but not always) reveals that eosinophils make up the majority of the cells (>10%).

Eosinophilia along with persistent nonallergic rhinitis. Eosinophilic nonallergic rhinitis (ENR)5 and non-allergic rhinitis with eosinophilia (NARE) are other names for the recently identified condition perennial nonal-lergic rhinitis with eosinophilia. Six It is typified by persistent symptoms of rhinorrhea and nasal congestion, negative skin test sensitivities to common aeroallergens, and nasal secretions containing eosinophils. Physical manifestations typically don't differ all that much from chronic allergic rhinitis. It is unknown what the underlying

etiology of this condition is. We assume that the entry of eosinophils into the nasal cavity is caused by an eosinophilic chemotactic chemical originating from mast cells (or other cells). The edema, mucous hyperproduction, and vasodilation are all explained by mast cell and basophil products, which are produced through an unclear process and may operate directly or indirectly (reflexively).

Vasomotor rhinitis. Although the name "vasomotor rhinitis" has been around for a while, the etiology of this condition is unknown. Chronic nasal congestion and mild rhinorrhea, a negative rapid skin test reactivity to common allergens, and the lack of eosinophils in nasal secretions are its defining characteristics. Vasomotor rhinitis has been hypothesized-based on scant evidence-to be associated with autonomic dysregulation with cholinergic predominance in the mucous membranes, or membrane congestion brought on by vasodilation in conjunction with excessive mucus production. Generally, applying a cold stimulation to the upper back skin results in a momentary decrease in nasal mucosal temperature, while applying a warm stimulus generates.

Anatomical Structure of the Nasal Cavity

The **nasal cavity** is a complex anatomical structure that plays an essential role in respiration, filtration, humidification, and olfaction. It extends from the **nostrils (anterior nares)** to the **choanae**, which open into the **nasopharynx**. The cavity is divided into two symmetrical halves by the **nasal septum**, which consists of both **cartilaginous** and **bony** parts.

Main Anatomical Components

1. **Nasal Septum**

✓ Formed by the **perpendicular plate of the ethmoid bone**, the **vomer**, and the **septal cartilage**.

✓ Deviation or deformation of the septum is a common cause of chronic nasal obstruction.

2. **Nasal Conchae (Turbinates)**

✓ There are three pairs: **inferior**, **middle**, and **superior turbinates**.

✓ They increase the surface area of the nasal cavity, helping to warm, humidify, and filter inhaled air.

✓ **Hypertrophy** (enlargement) of the turbinates is a frequent cause of chronic nasal congestion.

3. **Nasal Meatuses**

✓ Spaces located beneath each turbinate: **inferior**, **middle**, and **superior meatus**.

✓ They serve as drainage pathways for the paranasal sinuses and the nasolacrimal duct.

4. **Paranasal Sinuses**

✓ Include the **maxillary**, **frontal**, **ethmoidal**, and **sphenoidal** sinuses.

✓ Inflammation or blockage of these sinuses can lead to chronic congestion and sinusitis.

5. **Nasal Mucosa**

✓ Lined by **pseudostratified ciliated columnar epithelium** with **goblet cells**.

✓ The mucosa is rich in **blood vessels** and **glands**, which secrete mucus to trap dust and microorganisms.

✓ Chronic inflammation leads to **mucosal edema**, **hypersecretion**, and **obstruction of airflow**.

6. **Vascular Supply and Innervation**

- ✓ Supplied by branches of the **maxillary**, **facial**, and **ophthalmic arteries**.
- ✓ Venous drainage occurs through the **pterygoid plexus** and **facial veins**.
- ✓ Innervation comes from branches of the **trigeminal nerve (CN V)** and the **autonomic nervous system**, which regulate vascular tone and glandular secretion.

Pathophysiology

Rhinomanometric investigations reveal decreased conductance (increased nasal airway resistance) in patients with persistent rhinitis and airflow obstruction. Rhinomanometry is a difficult task that can lead to several mistakes. It is essentially useless in clinical practice and only useful as a research tool.

Most forms of rhinitis result in increased mucus production; however, measuring this phenomena is solely a research process. An further characteristic of chemical mediator release and allergy- or cold-induced nasal symptoms is an increase in blood vessel permeability to albumin and allergenic macromolecules in the nasal mucous membranes. Chronic rhinitis is associated with a greater sensitivity of the nasal mucous membranes to histamine, methacholine, and other stimuli, which is suggestive of autonomic dysfunction. The autonomic innervation of the nasal airways is removed via a vidian neurectomy release of nasal inflammatory mediators triggered by allergens during both the early and late phases of responses. In allergic rhinitis and possibly in some types of chronic rhinitis, altered eustachian tube function can occur.

A key factor in the altered physiology of rhinitis is nasal reflexes. The afferent limb, cranial nerve V, is responsible for transmitting the sensations of itch, burning, and pain when subepithelial receptors are stimulated. The reflex's efferent limb symbolizes the autonomic innervation of the nose, where sympathetic activity drives vasoconstriction but has little influence on mucous gland function, while parasympathetic cholinergic discharge induces vasodilation and mucous gland secretion.

Therapy

Certain steps should be taken (in addition to the more targeted pharmacologic and immunologic treatments listed below), regardless of the kind of chronic rhinitis. To lessen nasal airway hyperirritability, irritants such dusts, fumes, scents, powders, sprays, and tobacco smoke should be avoided both at home and at work.

Identification and avoidance of possible etiologic agents, including both specific allergens and nonspecific irritants, is the first step in the treatment of chronic rhinitis. Pollen exposure can be decreased using air conditioning. Commercial cleaners that include hypoclorous acid, like chlorine bleach, may be able to stop the formation of mold in musty basements and other places. Measures for controlling dust should be implemented. Nasal decongestant abuse must be avoided, and topical corticosteroid therapy-which is frequently brief-is necessary to treat mediastinal rhinitis mediamentosa. Topical (and systemic) corticosteroids, immunotherapy, cromolyn, antihistamines, decongestants, and pharmacotherapy of the various rhinitides are covered elsewhere. It is necessary to quickly address the anticholinergic medicines, which are another family of antirhinitis medications. It has long been known that drugs like atropine are beneficial in treating rhinitis, particularly when it comes to lowering mucous gland hypersecretion. This latter effect has been observed in cases of hypersecretion linked to vasomotor rhinitis, rhinorrhea accompanying viral upper respiratory tract infections, and allergic rhinitis.24'26 It is hoped that medications like ipratropium bromide, which is now licensed for use as an inhalation therapy for chronic bronchitis and asthma, may soon be accessible for use in treating the many types of rhinitis.

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