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RHINITIS MEDICAMENTOSA: PATHOPHYSIOLOGY, CLINICAL MANIFESTATIONS, AND CURRENT MANAGEMENTS STRATEGIES

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ABSTRACT

Background: Rhinitis medicamentosa (RM) is a non-allergic condition caused by the prolonged use of topical nasal decongestants. Despite its prevalence, it remains frequently underdiagnosed in clinical practice.
Objectives: This review synthesizes current knowledge on the pathophysiology, diagnostic criteria, and multifaceted treatment approaches for RM.
Methods: A comprehensive literature search was conducted across PubMed (up to 2026) focusing on alpha-adrenergic agonist-induced mucosal changes and therapeutic interventions.
Results: Chronic administration of sympathomimetics, such as xylometazoline, leads to adrenoceptor downregulation, secondary vasodilation, and permanent mucosal remodelling. Clinical presentation is characterized by rebound congestion and tachyphylaxis. Management centers on immediate or tapered decongestant cessation, supplemented by intranasal corticosteroids. In refractory cases involving turbinate hypertrophy, surgical intervention (e.g., conchoplasty) remains a definitive option.
Conclusions: RM represents a significant clinical challenge. Increasing clinical awareness and patient education regarding the risk of decongestant overuse are crucial for prevention and successful long-term outcomes.

KEYWORDS

Rhinitis Medicamentosa, Nasal Decongestants, Rebound Congestion, Turbinate Hypertrophy, Intranasal Corticosteroids, Drug-Induced Rhinitis

CITATION

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Introduction: Rhinitis medicamentosa is a nasal breathing dysfunction resulting from uncontrolled use of locally acting nasal decongestants like xylometazoline, oxymetazoline, tramazoline and phenylephrine. Alpha agonists cause constriction of the nasal mucosa vessels causing relieve with nasal congestion caused by various factors like, rhinitis, acute upper respiratory tract infection, influenza or in allergic rhinitis [1]. Usage of local decongestants should be limited to 3-5 days. Prolonged use leads to downregulation of alpha receptors, chronic mucosal edema, epithelial damage, impaired mucociliary transport and, in the long term, to nasal turbinate hypertrophy. It is an extremely urgent public health problem [2]. Rhinitis medicamentosa significantly reduces the quality of life, [3] leads to serious health problems and also can cause irreversible changes in the nasal mucosa. Effective management is challenging, as it requires high patient adherence; in refractory cases, surgical intervention may be necessary [2,4].

Methods: This study comprises a comprehensive narrative review of current literature, including original research, clinical guidelines, consensus statements, and peer-reviewed articles. The search was conducted via the PubMed database (up to 2026). The analysis focuses on the epidemiology, pathophysiology, clinical presentation, diagnostic criteria, and management strategies of rhinitis medicamentosa.

Epidemiology: Approximately 30 percent of the world's population suffers from chronic rhinitis [5]. Rhinitis medicamentosa, also known as ‘rebound congestion’ often arises from attempts to alleviate symptoms of other types of rhinitis like allergic rhinitis. Imidazoline-based nasal decongestants, such as xylometazoline and oxymetazoline, are commonly sold over the counter and rank among the most frequently used medications, largely because they provide rapid relief from nasal congestion and are strongly promoted in the marketplace. RM is seen most often in young and middle-aged adults, affecting men and women at comparable rates. Reported prevalence ranges from about 1% to 9% of patients attending ENT clinics, although the true rate of the problem is likely higher because these medications are readily available over the counter and most patients

delays seeking help from a specialist [6,7,8]. Clinical evidence indicates that patients typically present with a history of decongestant overuse ranging from two weeks to several decades, with 30.8% reporting use exceeding several years [9].

Etiology: The nasal mucosa contains two primary types of blood vessels: resistance and capacitance vessels. Resistance vessels, including small arteries, arterioles, and arteriovenous anastomoses, regulate blood flow into the capacitance vessels, which predominantly consist of venous sinusoids [10,11]. These venous sinusoids are densely supplied by sympathetic nerve fibers. When these nerves are activated, they release norepinephrine, which interacts with prejunctional α2 as well as postjunctional α1 and α2 receptors [12,13,14]. As a result, blood flow to the nasal mucosa decreases and the sinusoids empty more effectively, leading to a reduction in nasal congestion [10,15]. Nasal congestion is also driven by parasympathetic, sensory, and nonadrenergic noncholinergic peptidergic nerves, which promote vasodilation, increase secretions, and trigger symptoms like rhinorrhea and sneezing. In addition, inflammatory cells release mediators like histamine, prostaglandins, leukotrienes that further enhance vascular swelling and obstruction [10].

There are two main groups of nasal decongestants which are sympathomimetic amines and imidazolines. The first group includes drugs such as, pseudoephedrine which is taken orally and despite local action has also systemic effect, and phenylephrine which is used the most in ophthalmology, additive to topical hemorrhoid medications and also but no as often as imidazolines as local nasal decongestant [16]. The second group which is imidazolines comprises drugs like oxymetazoline, xylometazoline, naphazoline, tramazoline, and clonidine, which are the main focus of this paper [17]. Imidazolines act mainly as α2-adrenergic agonists. By stimulating postsynaptic receptors, they induce profound vasoconstriction [18], reducing blood flow within the venous sinuses and providing immediate relief from congestion. At the same time, they reduce the release of endogenous norepinephrine through negative feedback which modulates vascular tone and stabilizes the vascular response. Overall, this also causes a decrease in vascular permeability and a decrease in plasma transudate into the mucosa [8,17,19].

Excessive and long-term use leads to continuous activation of alpha receptors, which results in a decrease in their number and sensitivity which is called “down regulation” [20]. This results in a "rebound" effect, i.e., vasodilation and chronic passive congestion [21]. Ischemia during use and secondary passive congestion lead to hypoxia and mucosal damage. This manifests itself through chronic nasal congestion, dry mucosa, and impaired mucociliary transport [14,17,22]. The changes described are primarily functional. Unfortunately, over time, structural changes occur leads to tissue remodeling such as connective tissue hyperplasia, mucous gland hyperplasia, squamous cell metaplasia, epithelial edema, epithelial cell denudation,, increased expression of the epidermal growth factor receptor, inflammatory cell infiltration and vascular dilation. The final stage may even be ossification of tissue. Rhinoscopy or endoscopy reveals hyperemic, swollen mucosa, often hypertrophy of the inferior nasal turbinates, dry or paradoxically excessively moist mucosa, and a lack of typical allergy symptoms like pallor, and watery discharge. [2,17,23].

The term RM also known as rebound or chemical rhinitis can also refer to nasal congestion that occurs as a side effect of medications other than topical decongestants. These include oral beta-blockers, antipsychotic drugs, oral contraceptives, and antihypertensive agents. However, the mechanisms by which these oral medications cause congestion differ from those associated with topical nasal decongestants [4,24].

Diagnosis and differentiation: The diagnosis of rhinitis medicamentosa requires correlation of symptoms with uncontrolled and prolonged use of nasal decongestants. It also requires ruling out other possible causes of chronic nasal congestion, such as allergic rhinitis, eosinophilic rhinitis, gustatory rhinitis, atrophic rhinitis, senile rhinitis, hormonal rhinitis, vasomotor rhinitis, or rhinitis related to nasal cocaine use [2,4,25]. Nasal and sinus polyps, chronic sinusitis, nasal septum deviation, adenoid hypertrophy, and abnormalities in the structure or function of the ciliary tract should also be considered. Diseases such as vasculitis, sarcoidosis, cystic fibrosis, and functional nasal defects, such as nasal valve collapse during breathing, should be considered. All of the above should be considered in the differential diagnosis, although their presence does not rule out the possibility of concomitant rhinitis medicamentosa if accompanied by overuse of nasal decongestants, which can often occur as patients seek relief from their symptoms [6,25].

Symptoms and clinical presentation: The clinical picture is distinctive and results from secondary vascular and structural changes in the nasal mucosa. The most typical and often initial symptom is progressive, chronic nasal obstruction, usually bilateral, although it can begin unilaterally, which worsens particularly after discontinuation of the medication, and the increasingly weaker response to subsequent decongestant doses [17]. Increasingly frequent use of the medication becomes necessary to achieve short-term improvement, reflecting the phenomenon of tachyphylaxis and the "rebound effect'' [26]. A characteristic element of the clinical picture is secondary dependence on intranasal preparations, manifested by a subjective feeling of "inability to breathe without drops" and a shortening of the duration of action of the medication, which begins to be used repeatedly throughout the day, often also at night. Unlike infectious or allergic rhinitis, discharge is usually sparse or moderate, and a feeling of dry mucosa is more common than severe rhinitis. [27] As a result of chronic inflammation, thick discharge may subsequently develop. Patients often report a burning sensation in the nose, dryness, discomfort, or pain, and sometimes epistaxis resulting from epithelial damage [4]. Chronic swelling and structural changes can lead to hyposmia and, less commonly, anosmia, which is usually reversible, though in severe cases it may persist. Chronic nasal obstruction leads to sleep disturbances, mouth breathing, daytime fatigue, and a reduced quality of life [26,27,28].

Adverse systemic effects: Although intranasal imidazoline medications, such as xylometazoline and oxymetazoline are intended for local action, they can cause systemic effects under certain conditions. This is due to partial absorption through the nasal mucosa, which increases with damage to the nasal mucosa, and swallowing of a portion of the dose [28]. Imidazolines act as α-adrenergic receptor agonists. Once absorbed into the circulation, they cause generalized vasoconstriction (α1) and can affect the central nervous system via (α2) receptors, resulting in increased sympathetic tone or modulation [29]. The most clinically significant adverse effects include arterial hypertension, increased peripheral resistance, reflex tachycardia or bradycardia, and rarely, cardiac arrhythmias [28,30]. The risk is greater in patients with hypertension, coronary artery disease, and the elderly due to increased vascular sensitivity [31]. Possible neurological symptoms include agitation, anxiety, insomnia, headaches, dizziness, seizures, stroke and less frequently, sedation, especially in children, [32] due to increased bioavailability and the risk of central nervous system depression [26,27,33]. Although systemic effects are rare when used appropriately, their significance increases with overdose, chronic use, and mucosal damage [26].

Treatment: The mainstay of treatment for rhinitis medicamentosa is complete discontinuation of topical nasal decongestants, such as xylometazoline and oxymetazoline [17,26]**.** Abrupt discontinuation is usually most effective, although in selected cases, gradual reduction may improve treatment tolerance. Intranasal glucocorticosteroids, such as mometasone and fluticasone, play a key role in therapy by reducing inflammation and vascular permeability, leading to reduced mucosal edema and improved nasal patency [8,17]**.** Their use has been shown to alleviate symptoms and shorten the duration of symptoms during the decongestant withdrawal period. Adjunctive therapy includes nasal irrigation with saline solutions, which improves mucociliary transport and mechanically reduces mucosal edema, alleviating symptoms [26,28]. In more severe cases, short-term use of systemic glucocorticosteroids may be considered to control severe inflammation and facilitate decongestant withdrawal [28].

In patients with persistent turbinate hypertrophy and structural changes that are resistant to conservative treatment, surgical treatment may be indicated. Turbinate reduction procedures aim to improve nasal patency while preserving mucosal function [**17**]**.** One of the most commonly used methods is submucosal ablation using radiofrequency energy, which leads to coagulation of the submucosal tissues, followed by fibrosis and shrinkage [34]. This technique is minimally invasive, causes minimal bleeding, and has a short recovery period, although its effects may be partial and may require repeat procedures [35]. An alternative is turbinoplasty, which involves the removal of a portion of the submucosal tissue while maintaining the continuity of the mucosa. This provides a more lasting effect and better control of turbinate volume. However, it is associated with greater invasiveness and the risk of bleeding [35,36]. In cases of significant hypertrophy, partial turbinate resection is also used, sometimes including bony elements. This is highly effective but increases the risk of functional impairment [37,38]. An additional option is laser therapy, which relies on vascular and tissue coagulation, similar to radiofrequency techniques [38]. However, it should be emphasized that excessive turbinate resection can lead to the development of "empty nose syndrome," in which, despite wide and clear nasal passages, the patient experiences subjective nasal obstruction [39]. This phenomenon results from impaired airflow and damage to sensory receptors and manifests as nasal congestion, dryness, burning, and

sleep disturbances. For this reason, modern nasal surgery is conservative in nature. The indications for surgical treatment include primarily the ineffectiveness of conservative treatment lasting at least 2–3 months, chronic nasal obstruction, persistent structural hypertrophy of the turbinates, and drug-induced rhinitis with irreversible changes in the mucosa [38,39].

In recent years, attempts have also been made to use less conventional therapeutic methods in the treatment of chronic rhinitis, including rhinitis medicamentosa, particularly in cases refractory to standard treatments. One approach being explored is the use of xylitol, which has a potential protective effect on the nasal mucosa by improving its barrier function and influencing the mucosal microenvironment. Animal models have demonstrated its beneficial effects on the regeneration of damaged epithelium and reduced inflammatory changes [40]. Another approach is the use of capsaicin, which, by desensitizing C-type sensory fibers, reduces nasal mucosal hyperresponsiveness and symptoms such as congestion and rhinorrhea. This mechanism is based on the depletion of neuropeptides such as substance P, which limits the neurogenic component of inflammation [41]. Procedures aimed at modulating nasal innervation, such as posterior nasal nerve neurectomy, which aims to limit the influence of the parasympathetic nervous system on secretion and vasodilation, are also gaining interest. This method, although primarily used for allergic rhinitis, is also being considered for other forms of chronic rhinitis as a potential option for patients resistant to pharmacological treatment [42]. Furthermore, experimental studies indicate that physical factors, such as lower temperature, can influence the tissue tension of the nasal mucosa, leading to a reduction in its swelling through a vasoconstrictive effect. While these results are promising, their clinical significance remains limited and requires further study [43].

In summary, alternative treatment methods represent an interesting avenue of research, but are currently complementary and do not replace standard therapeutic management based on discontinuation of decongestants, intranasal steroids and invasive methods.

Conclusions: Rhinitis medicamentosa is a significant and still underestimated public health problem, resulting primarily from the widespread availability and overuse of intranasal decongestants. Despite their initial effectiveness in relieving symptoms, chronic use leads to complex vascular dysregulation, epithelial damage, and tissue remodeling, thereby causing persistent nasal obstruction and a significant decline in patients' quality of life. In advanced cases, these changes may be partially or completely irreversible. This disease can lead not only to severe local symptoms but also to systemic complications, such as hypertension and central nervous system disorders, further underscoring its clinical significance. Nevertheless, patients often consult a specialist only in the advanced stages of the disease. The mainstay of therapy is discontinuation of decongestants and the use of anti-inflammatory therapy, primarily intranasal glucocorticosteroids. In refractory cases, surgical treatment is possible, which is sparing and focuses on preserving mucosal function. A key element in reducing the incidence of rhinitis medicamentosa is educating the public and medical personnel. It is essential to increase awareness of the safe use of intranasal medications, particularly limiting their use to a few days. Regulations regarding the availability of these medications could significantly reduce the scale of the problem. Despite advances in understanding the pathophysiology and treatment of the disease, further research into treatment methods remains necessary.

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