

*Clinical pearls***Rhinitis medicamentosa and the stuffy nose**

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“Rhinitis medicamentosa (RM) is a drug induced, non-allergic form of rhinitis that is associated with prolonged use of topical vasoconstrictors, i.e. local decongestants.”¹ Rhinitis medicamentosa and drug-induced rhinitis are differentiated in this Clinical Pearls article, with the former being caused by sympathomimetic amines and imidazolines and the latter associated with antihypertensives, erectile dysfunction drugs, hormones, nonsteroidal inflammatory drugs, psychotropic drugs, and cocaine. The adverse effects associated with these latter drugs usually occur immediately, whereas exacerbation associated with chronic nasal obstruction from topical decongestants might take days. Similarly, discontinuing oral medications that cause drug-induced rhinitis results in almost immediate relief of symptoms, whereas discontinuing nasal decongestants might not relieve chronic nasal obstruction for prolonged periods of time.

All over-the-counter topical decongestants have a general warning that they should be discontinued after several days of use, even though the cumulative dose or time period to cause rhinitis medicamentosa has not been conclusively determined. This disease is usually characterized by nasal congestion with or without an underlying upper airway problem.

Rhinitis medicamentosa occurs most commonly in young and middle-aged adults of both sexes and has an incidence range from 1% to 9% of visits to otolaryngology clinics or allergy clinics. Nasal decongestants are also commonly started after a viral upper respiratory track infection leading to the syndrome. The appearance of the nasal mucosa cannot distinguish rhinitis medicamentosa from infectious or other types of rhinitis.

Most individuals who have overused topical nasal decongestants do so because of chronic nasal obstruction, a clinical problem that can complicate one's social interactions, reduce the capacity to smell and taste, cause avoidance of exercise, and, in particular, exacerbate or cause snoring and even be involved in the causation of sleep apnea, insomnia, or both. Rhinitis medicamentosa might predispose to chronic sinusitis, otitis media, nasal polyps, and atrophic rhinitis.^{2,3}

Sympathomimetic amines mimic the actions of the sympathetic nervous system, ultimately stimulating the α receptors and resulting in vasoconstriction. They also are mild β receptor agonists and cause rebound nasal dilatation after the α effect has waned. The imidazolines are primary α_2 -agonists, causing vasoconstriction. The decongestive effect of these medications initially lasts approximately 7 to 9 hours but decreases with prolonged use. Benzalkonium chloride, used as a preservative in some nasal sprays, has also been incriminated in causing nasal mucosa swelling.

The reasons why rhinitis medicamentosa occurs are obscure, but a popular hypothesis is that the α receptors become refractory to stimulation and actually result in even more nasal obstruction, necessitating the added use of additional intranasal decongestants. Histologic changes in human subjects are conflicting, with some studies showing destruction of nasal cilia and epithelial cell mitochondria and others showing no morphologic changes in the intracellular space, basal membrane, or tunica propria after 6 weeks of treatment with a decongestant. Histologic studies in rabbits revealed ciliary loss, epithelial cell damage, and edema within 2 weeks of use of these drugs. Over a period of 6 to 7 weeks, additional pathology was observed, including subepithelial layer fibrosis and hypertrophy, increased mucous production, cellular disorganization, and eventually blood vessel damage in these animals.

Many underlying conditions can be associated with rhinitis medicamentosa, such as allergic rhinitis, nonallergic rhinitis, acute and chronic sinusitis, nasal polyps, rhinitis caused by pregnancy, drug-induced rhinitis, or nasal obstruction caused by severe septal deviation. These individuals should have a detailed history, physical examination, and appropriate diagnostic studies to identify the underlying condition.

Other individuals begin intranasal decongestants after a respiratory tract infection and continue them indefinitely because of the self-induced rebound phenomena. Stopping their nasal decongestants with appropriate treatment usually resolves this disease.

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The first goal in the treatment of rhinitis medicamentosa is the immediate discontinuation of topical nasal decongestants. However, their abrupt cessation might result in more swelling and congestion, causing even greater frustration for the patient, as well as the treating physician. Various treatment programs have been suggested, including nasal cromolyn, sedatives/hypnotics, saline nasal sprays, and intranasal glucocorticosteroids, only the latter of which have been shown to be effective. Nasal glucocorticosteroids decrease nasal edema, inflammation, and congestion associated with rhinitis medicamentosa in both animal models and several small, randomized, controlled human trials. However, these trials are not adequately powered, and it is questionable whether all subjects actually had rhinitis medicamentosa.⁴

If a treatment program is not effective, treat the patient with oral prednisone, 15 mg 3 times a day for 5 days, and gradually withdraw the nasal decongestant. Continue high doses of intranasal glucocorticosteroids while identifying the underlying problem. This treatment usually resolves rhinitis medicamentosa regardless of its underlying cause.

The primary reason that a patient begins a topical nasal decongestant is because of nasal obstruction and not sneezing, itching, or rhinorrhea. Medications used for various forms of rhinitis include oral and nasal antihistamines, which have minimal or no effect on nasal obstruction; oral sympathomimetics, which have some effect but which often lead to undesirable side effects; and intranasal glucocorticosteroids, which are most effective. Others include leukotriene antagonists and blockers and cromones. These are minimally effective. In some cases none of these medications adequately relieve the chronic nasal obstruction, causing the patient to use the over-the-counter market and topical intranasal decongestants.

Patients with chronic nasal obstruction can be diagnostic dilemmas for which there is no clear-cut treatment. Turbinectomies or submucosal resections are usually not very helpful and can result in other complications, such as atrophic rhinitis.

Most individuals can tolerate the use of intranasal decongestants as long as they continue intranasal glucocorticosteroids, up to 2 puffs in each nostril, twice daily. They can use intranasal glucocorticosteroids, followed by an intranasal decongestant 2 times a day, enabling them to breathe through their nose more comfortably both during the day and night. There is some evidence in the literature that intranasal budesonide (32 µg per spray), 2 sprays in each nostril daily, versus placebo, used with oxymetazoline, increased the nasal volume and minimal cross-sectional area.⁵

I prefer not to mix the decongestant into the same nasal dispenser with the topical glucocorticosteroid because of the inability to adjust one medication and not the other and because, at times, patients are able to discontinue the decongestant and subsequently even the intranasal glucocorticosteroid, at least temporarily.

As William Osler, the father of American medicine, said "The practice of medicine is an art based on science." The science for this form of treatment is lacking and needs to be confirmed in appropriately controlled studies.

In summary, chronic nasal obstruction remains a major problem for many patients. Intranasal decongestants seem to be the best drugs to relieve such congestion, and using these drugs with intranasal glucocorticosteroids often enables the patient to do so. Again, scientific evidence is lacking, but in my opinion, it works.

REFERENCES

1. Graf P. Rhinitis medicamentosa: a review of causes and treatment. *Treat Respir Med* 2005;4:21-9.
2. Toohill RJ, Lehman RH, Grossman TW, Belson TP. Rhinitis medicamentosa. *Laryngoscope* 1981;91:1614-21.
3. Black MJ, Remsen KA. Rhinitis medicamentosa. *CMAJ* 1980;122: 881-4.
4. Hallen H, Enerdal J, Graf P. Fluticasone propionate nasal spray is more effective and has a faster onset of action than placebo in treatment of rhinitis medicamentosa. *Clin Exp Allergy* 1997;27:552-8.
5. Ferguson BJ, Parameswaran S, Rubinstein E. A study of the effect of nasal steroid sprays in perennial allergic rhinitis patients with rhinitis medicamentosa. *Otolaryngol Head Neck Surg* 2001;125:253-60.