Rhinitis medicamentosa

By

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Introduction:

Rhinitis medicamentosa is a condition characterised by nasal congestion without rhinorrohea or sneezing. This condition is caused by the use of topical nasal decongestants for a prolonged period of time. Use of these topical decongestants for more than a week is sufficient to cause this problem. This condition should be differentiated from rhinitis caused by use of drugs like oral contraceptives, antihypertensives and psychotrophic drugs.

History:

The term rhinitis medicamentosa was coined by Lake in 1946.

Synonyms:

Rebound rhinitis / chemical rhinitis

Pathophysiology:

The nasal mucous membrane is rich in resistance blood vessels draining into capacitance venous sinusoids. These resistance blood vessels include small arteries, arterioles and arteriovenous anastomosis. The capacitance vessels (venous sinusoids) are innervated by sympathetic fibers. Sympathetic stimulation causes activation of alpha 1 and alpha 2 receptors present in the walls of the capacitance vessels which leads to decreased blood flow and constriction of venous sinusoids causing nasal decongestion. Parasympathetic stimulation causes release of acetyl choline which increases nasal secretions. Parasympathetic stimulation also causes release of VIP (vasoactive intestinal polypeptides) causing vasodilatation of the resistance blood vessels leading on to dilatation of sinusoids there by causing nasal congestion. In addition to sympathetic and parasympathetic innervation the nasal mucosa is richly

endowed with sencory type c fibers. These sensory fibers on stimulation releases neurokinin A, calcitonin gene related peptide and substance P. These substances cause down regulation of sympathetic vasoconstriction causing nasal congestion. The exact pathophysiology of rhinitis medicamentosa is still not clear. Various hypothesis exist. Almost all of them focus on dysregulation of sympathetic / parasympathetic tone by exogenous vasoconstriction molecules.

Possible mechanisms of rhinitis medicamentosa include:

1. Secondary decrease in the production of endogenous norepinephrine through a negative feed back mechanism
2. Sympathomimetic amines used as topical decongestants have effects on both alpha and beta receptors. Their alpha effects predominate over beta effects causing nasal decongestion. This beneficial alpha effect is short lived while beta effect is more prolonged. After cessation of alpha stimulation the sympathomimetic amines still keep stimulating beta receptors causing rebound nasal congestion.
3. Rebound increase in parasympathetic activity causing increased nasal secretion and nasal mucosal congestion

Types of topical nasal decongestants in use:

Two types of nasal decongestants are used.

1. Sympathomimetic amines – (pseudoephidrine, amphetamine, phenylephrine mescaline). These drugs activate sympathetic nerves by presynaptic release of endogenous norepinephrine, which binds to alpha receptors causing vasoconstriction leading on to nasal decongestion. Rebound vasodilatation may be caused due to weak affinity of these drugs to beta receptors leading on to vasodilatation and nasal congestion.
2. Imidazolines – (zylometazoline, oxymetazoline, naphazoline). These drugs cause vasoconstriction due to its effect on alpha 2 receptors. These drugs also cause a decrease in the endogenous secretion of norepinephrine via a negative feedback mechanism. This reduction in the endogenous norepinephrine secretion causes rebound vasodilatation and nasal congestion.

Benzalkonium chloride the preservative commonly used in nasal drops have been known to exacerbate rhinitis medicamentosa. The exact mechanism is still not known.

It should be borne in mind that use of nasal decongestants is due to the presence of pre existing pathology in nasal mucosa causing nasal block. Pathologies can be infections, polypi, allergic rhinitis etc.

Symptoms:

Symptoms are usually confined to the nose.

1. Nasal block without significant rhinorrhoea and sneezing
2. These symptoms dont exhibit seasonal variations
3. Patient feels compelled to use nasal topical decongestants
4. Usage of these decongestants become more frequent

Physical examination of nose shows:

1. Nasal mucous membrane appears beefy red
2. Nasal mucosa is boggy, granular, friable and bleeds on touch
3. These patients snore and have sleep apnoea
4. Dry mouth and throat are common findings

Histological features of rhinitis medicamentosa:

1. Nasal epithelium shows severe hyperplasia
2. There is loss of cilia
3. Increase in the number of goblet cells and submucosal glands

Epidermal growth factor receptor:

This is a 70 kilodalton membrane glycoprotein which is usually expressed in fetal airways. This receptor plays a vital role in epithelial cell proliferation, differentiation and airway branching in fetus. In healthy adult airways this receptor is usually not expressed. It is seen only in patients with malignancy involving airway. In patients with rhinitis medicamentosa this epidermal growth factor receptor is found to be expressed in large quantities. They play a vital role in proliferation of goblet cells and mucous secretion by these glands.

Treatment:

The first goal in management of these patients is making them discontinue the use of topical nasal decongestant. It should be borne in mind that sudden cessation of use of topical nasal decongestants will cause more nasal congestion making patient's compliance that much difficult.

Oral prednisalone:

Patient with rhinitis medicamentosa is treated with oral prednisolone in doses of 15 mg thrice a day for 5 days, while the nasal decongestant is simultaneously withdrawn in a phased manner. The patient is weaned from steroid by tapering the dose.

Use of intranasal steroids:

This is becoming popular because it causes fewer side effects than systemic steroids. It can be safely administered for long durations. These patients may derive significant benefit by using intranasal steroids as it helps in simultaneous control of nasal allergy and also reduces the nasal mucosal inflammation and oedema.

Nasal saline douching:

Douching the nose with isotonic saline will help in clearing the nasal cavity of thick mucoid secretions thus enabling the steroid spray to permeate the nose fully.