Rhinitis Classification and Management

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Otolaryngology online

Contents

Rhinitis and its classification

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Introduction:
In broad terms rhinitis is defined as inflammation involving mucosal lining of the nasal cavity [1]. This disorder is rather common in primary care and speciality clinics. This common condition affects nearly 20 - 25% of general population [2] . This figure could increase to 40% of patients attending ENT clinics. Inflammation of nasal mucosa can be caused by various factors which include:

1. Infections
2. Allergy
3. Irritants
4. Medications
5. Hormones

It is hence imperative to classify various types of rhinitis according to their causative factors in order to optimise the treatment modality.
If the following symptoms of rhinitis persist for a duration of more than 3 weeks then it is known as chronic rhinitis. Symptoms of chronic rhinitis include:

1. Excessive discharge from nasal cavity
2. Nasal congestion
3. Pain
4. Pressure symptoms due to secretions daming inside sinuses
5. Sneezing
6. Itchy nose

Classification of chronic rhinitis:

Chronic rhinitis can be classified into [3]:

Allergic – Perennial and seasonal types
Infectious
Non allergic rhinitis: This category can be subclassified into:
Rhinitis caused by surgery
Rhinitis due to cocaine abuse
Rhinitis due to aging
Emotional rhinitis
Exercise induced rhinitis
Gustatory rhinitis
Hormone induced rhinitis: Hypothyroidism, pregnancy, menstural cycle and oral contraceptives
Idiopathic – Vasomotor rhinitis

Non allergic Non infectious

Allergic rhinitis:
This is defined as IgE mediated inflammation of nasal mucosa after exposure to offending allergen. This is acually not a life threatening condition but can significantly impair the quality of life of the patient [4]. Allergic rhinitis is rather common in children and adolescents. Studies reveal that it can occur in any age. Prevalence of allergic rhinitis could be very high in children, as high as 50% [9] in some studies. Allergy tests could help in the diagnosis of this condition. Skin testing, serum specific IgE antibody testing could be very useful in diagnosing this condition. Nasal mucosal inflammation in these patients is caused by complex interaction with inflammatory mediators which are triggered by IgE mediated response to extrinsic allergen [5]. This allergic tendency has a genetic component to it. In genetically susceptible individuals three phases of reactions have been identified. Allergic rhinitis can be subclassified into Perennial and seasonal rhinitis. Seasonal rhinitis is caused by exposure to seasonal allergens like pollen. Perennial rhinitis is caused by exposure to antigen like cat dander, and dust mite antigen which are present throughout the year. Symptoms of perennial allergic rhinitis are more subtle when compared to seasonal ones. Perennial allergic rhinitis more commonly present with typical late phase symptoms like nasal congestion and nasal discharge. Itching and sneezing are rather less when compared to that of seasonal allergic rhinitis.

Phase of allergic sensitization:
This phase is characterised by production and release of IgE directed against the offending allergens (proteins).

Phase of IgE coating nasal mast cells:
In this phase IgE against specific allergens get attached to mast cells which can be seen in large numbers over the nasal mucous membrane.

Phase of mediator release:
When the offending allergen attaches to sensitised IgE present over nasal mast cells, this leads to release of immune mediators from mast cells. These mediators are responsible for nasal mucosal inflammation in these patients [6].

Mediators which are immediatly released include:

1. Histamine
2. Tryptase
3. Chymase
4. Kinins
5. Heparin

Mediators which are released from the mast cells [7] in a delayed fashion include:

1. Leukotrienes
2. Prostaglandin D2

These mediators are responsible for the classic symptoms of allergic rhinitis which include:

1. Congestion of nasal mucosa
2. Sneezing caused by stimulation of sensory nerves
3. Nasal itching
4. Redness / swelling of eyes
5. Mucous glands present in the nasal mucosa are stimulated to increase their secretion
6. Increased submucosal vascular permeability

All these reactions just takes minutes / hours to begin and hence are known as immediate / intermediate allergic reactions. The next phase is late phase reaction which are sustained by mediators released from mast cells through an complex interplay of events. The most important feature of this late phase is recruitment of other inflammatory cells to the nasal mucosa. This late phase reaction is responsible for continuing inflammation of nasal mucosa. Symptoms of late phase reactions are more or less similar to that of early phase. Important aspect of late phase reaction is that there is reduced sneezing and nasal mucosal itching [8] and increased nasal mucosal congestion and secretions. This late phase reaction can continue for hours – days.

Systemic effects of allergic rhinosinusitis include:

Fatigue
Sleepiness
Malaise

Infectious rhinitis:

Viruses have been known to be the common cause for this problem. Rhinovirus which is the cause for common cold has been commonly implicated. Another virus which is commonly implicated in common cold is coronavirus. Viral rhinitis can predispose to bacterial infections which could result due to loss of nasal mucosal ciliary activity. This inturn could lead to secretions filling up the paranasal sinuses. Viral rhinitis can be managed symptomatically, but if the same condition persists for more than a week then super added bacterial infection should be suspected and anitbiotics should be prescribed.

Non allergic rhinitis:

This condition is indistinguishable from allergic rhinitis. A careful history will help in differentiating these two conditions. Probable causes of non allergic rhinitis include:

1. Irritants
2. Medication induced
3. Hormonal
4. Atrophic
5. Non allergic rhinitis with eosinophilia syndrome (NARES)

Hormone induced rhinitis:

This has been described in many hormonal disorders. Neurogenic mechanism has been proposed as the probable factor. Disturbances involving thyroid gland (commonly hypothyroidism) and growth hormone (acromegaly) are characterised by congestion of nasal mucosa and rhinorrhoea.
Oestrogen / progesterone level changes can also cause rhinitis. This type of rhinitis is commonly seen in menstrual cycle, pregnancy etc. Pregnancy induced rhinitis is the classic example of this condition. This condition can be diagnosed with a certain degree of accuracy if seen in pregnant mothers.

Pregnancy induced rhinitis: [10]

Nasal congestion is common in pregnancy. It goes by the name pregnancy rhinitis. This condition is so common that it is seen in one in five pregnancies.

Clinical features:

Nasal congestion

Rhinorrhoea

These symptoms are aggravated by using nasal decongestant nasal sprays.

Definition:

Ellegard defined pregnancy rhinitis as nasal congestion which occurs during the last 6 weeks of pregnancy without other signs of upper respiratory infections / allergy. This disappears completely within 2 weeks after delivery.

Etiology:

1. Could be due to hormone effects
2. Elevated placental growth hormones have been implicated
3. Smoking is considered to be a risk factor
4. Autonomic nervous system imbalance as it occurs in vasomotor rhinitis

Diagnosis:

1. Watery rhinorrhoea
2. Nasal congestion
3. Secondary infections of paranasal sinuses

Medicine induced rhinitis: [11]
This is probably caused due to neurogenic mechanisms or due to local inflammatory effects caused by the offending agent. A wide range of drugs have been implicated in this condition. The following categories of medications are known to cause rhinitis.

1. Antihypertensives
2. Antidepressants
3. Psychotropics
4. Phosphodiesterase type 5 inhibitors (Sildenafil, vardenafil)
5. Anti-inflammatory drugs – This is caused due to an increase in Leukotriene production. This can also lead to reactive airway disorder. Aspirin is the common culprit in these situations (AERD) [12] *Aspirin exacerbated respiratory disease*.
6. Topical use of ά adrenergic medications can cause rebound congestion of nasal mucosa. This condition is known as *Rhinitis medicamentosa*.

Aspirin exacerbated upper airway disorder:

This condition goes under the name *"Samter's triad"* [13]. Features of this triad include:
Aspirin sensitivity
Nasal polyposis
Bronchial asthma.

Synonyms:

Acetylsalicylic acid triad
Widal's triad
Francis's triad

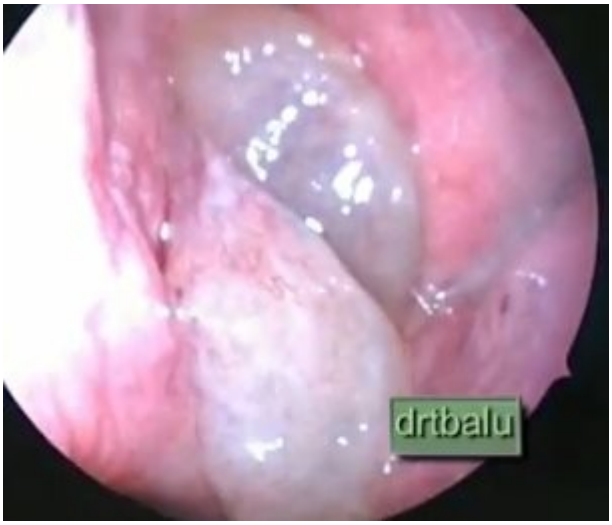
Aspirin triad

This is a medical condition affecting patients of young and middle age groups. These patients need not necessarily give history of nasal allergy.

Chronologically the first symptom to occur after ingestion of aspirin is rhinitis (with symptoms of sneezing, running nose and congestion). Typically this disorder gradually progresses to asthma, nasal polyposis and aspirin hypersensitivity(which comes rather at the fag end of the disease spectrum). Typically these patients are also anosmic because inflammation does not spare the olfactory mucosa also.
This disorder is commonly caused due to an anomaly involving the arachidonic acid cascade causing increased production of leukotrienes. These chemicals are characteristically involved in the inflammatory cycle seen in the nasal mucosa and lower airway. There is a classic over production of Leukotriene 4 (LT4) because normal prostaglandin production is blocked by aspirin and aspirin like drugs. The intermediaries of arachidonic acid cycle then preferentially produces LT4 which is known to the be cause for inflammatory reactions seen in these patients.

Management:

The main focus in managing these patients is directed towards alleviating the symptoms. On an immediate basis nasal decongestants and nasal topical steroids could play a vital role.
Desensitization to aspirin: This can be performed only in specialized clinics. This is ideal in long term remission of symptoms.
Leukotriene antagonists like Monteleukast / zafirleukast can be useful in blocking the harmful effects of LT4.
If nasal polyi are extensive then surgical removal should be resorted to in order to alleviate troublesome nasal obstruction.



Endoscopic image of a patient with nasal polyposis following aspirin ingestion

Dietary restrictions:
A diet low in omega – 6 oils which are precursors of arachidonic acid could be of help in these susceptible patients. Diet rich in omega – 3 oils could be of some help. Low salicylate diet (*Feingold diet*) could really help these patients. Organic food are supposed to contain more salicylates because plants are known to produce more salicylates when attacked by pests. This is actually a protective mechanism [14].

Rhinitis medicamentosa [15]:

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 bedifferentiated 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.

Rhinitis caused due to exposure to irritants:

This is also known as irritant rhinitis. This condition should be considered to be an occupational / environmental disorder. In these patients the noxious agents inhaled through the nose causes irritation rather than an allergic response. Relationship between exposure and symptoms should be sought before a correct diagnosis could be made. This is rather difficult to seek. Agents involved include:

1. Industrial chemicals
2. Wood dust
3. Tobacco smoke
4. Paint fumes
5. Hair spray
6. Perfumes

Diagnosis can be confirmed only by performing nasal provocation tests using the offending irritant. This should be performed under controlled conditions. Gustatory rhinitis [16] which occur following consuming spicy food could be termed as irritant rhinitis. Nasal pretreatment with atropine blocked food induced rhinorrhoea. This explains pathophysiology of this condition i.e. Stimulation of atropine inhibitable muscarinic receptors present in the nose by spicy food [17].

Managment:

This condition can be managed by avoiding exposure to the offending irritant. Use of nasal douches with isotonic saline would provide soothing releif to these patients. If acute symptoms are present then nasal topical steroids can be used.

Atrophic rhinitis [18]:

Atrophic rhinitis is defined as a chronic nasal disease characterised by progressive atrophy of the nasal mucosa along with the underlying bones of turbinates. There is also associated presence of viscid secretion which rapidly dries up forming foul smelling crusts. This fetid odor is also known as ozaena. The nasal cavity is also abnormally patent. The patient is fortunately unaware of the stench emitting from the nose as this disorder is associated with merciful anosmia.

Aetiology: The etiology of this problem still remains obscure. Numerous pathogens have been associated with this condition, the most important of them are 1. Coccobacillus, 2. Bacillus mucosus,

3. Coccobacillus foetidus ozaenae, 4. Diptheroid bacilli and 5. Klebsiella ozaenae. These organisms despite being isolated from the nose of diseased patients have not categorically been proved as the cause for the same.

Other possible factors which could predispose to this disease are:
1. Chronic sinusitis
2. Excessive surgical destruction of the nasal mucosa and turbiantes
3. Nutritional deficiencies
4. Syphilis.
5. Endocrine imbalances (Disease is known to worsen with pregnancy / menstruation)
6. Heredity (Autosomal dominent pattern of inheritance identified)
7. Autoimmune disease

The triad of atrophic rhinitis as described by Dr. Bernhard Fraenkel are 1. Fetor, 2. crusting and
3. atrophy.

Age of onset: Usually commences at puberty.

Females are commonly affected than males. Heredity is known to be an important factor as there appears to be increased susceptibility among yellow races, latin races and American negro races. Poor nurtrition could also be a factor. Bernat (1965) postulated iron deficiency could be a cause of this disorder.

Recently immunologists have considered atrophic rhinitis to be an autoimmune disorder. Fouad confirmed that there was altered cellular reactivity, loss of tolerance to nasal tissues. This according to him could be caused / precipitated by virus infection, malnutrition, immunodeficiency.

Pathology:

1. Metaplasia of ciliated columnar nasal epithelium into squamous epithelium.
2. There is a decrease in the number and size of compound alveolar glands
3. Dilated capillaries are also seen

Pathologically atrophic rhinitis has been divided into two types:
Type I: is characterised by the presence of endarteritis and periarteritis of the terminal arterioles. This could be caused by chronic infections. These patients benefit from the vasodilator effects of oestrogen therapy.
Type II: is characterised by vasodilatation of the capillaries, these patients may

worsen with estrogen therapy. The endothelial cells lining the dilated capillaries have been demonstrated to contain more cytoplasm than those of normal capillaries and they also showed a positive reaction for alkaline phosphatase suggesting the presence of active bone resorption. It has also been demonstrated that a majority of patients with atrophic rhinitis belong to type I category.

Once the diagnosis of atrophic rhinitis is made then the etiology should be sought. Atrophic rhinitis can be divided in to two types clinically:
1. Primary atrophic rhinitis - the classic form which is supposed to arise denovo. This diagnosis is made by a process of exclusion. This type of disease is still common in middle east and India. All the known causes of atrophic rhinitis must be excluded before coming to this diagnosis. Causative organisms in these patients have always be Klebsiella ozenae.

2. Secondary atrophic rhinitis: Is the most common form seen in developed countries. The most common causes for this problem could be:
1. Extensive destruction of nasal mucosa and turbinates during nasal surgery
2. Following irradiation
3. Granulomatous infections like leprosy, syphilis, tuberculosis etc

Clinical features:
The presenting symptoms are commonly nasal obstruction and epistaxis. Anosmia i.e. merciful may be present making the patient unaware of the smell emanating from the nose. These patients may also have pharyngitis sicca. Choking attacks may also be seen due to slippage of detached crusts from the nasopharynx into the oropharynx. These patients also appear to be dejected and depressed psychologically.
Clinical examination of these patients show that their nasal cavities filled with foul smelling greenish, yellow or black crusts, the nasal cavity appear to be enormously roomy. When these crusts are removed bleeding starts to occur.
Why nasal obstruction even in the presence of roomy nasal cavity?
This interesting question must be answered. The nasal cavity is filled with sensory nerve endings close to the nasal valve area. These receptors sense the flow of air through this area thus giving a sense of freeness in the nasal cavity. These nerve endings are destroyed in patients with atrophic rhinitis thus depriving the patient of this sensation. In the absence of these sensation the nose feels blocked.

Radiographic findings:
Are more or less the same in both primary and secondary atrophic rhinitis. Plain xrays show lateral bowing of nasal walls, thin or absent turbinates and hypoplastic maxillary sinuses.

CT scan findings:
1. Mucoperiosteal thickening of paranasal sinuses

2. Loss of definition of osteomeatal complex due to resorption of ethmoidal bulla and uncinate process
3. Hypoplastic maxillary sinuses
4. Enlargement of nasal cavity with erosion of the lateral nasal wall
5. Atrophy of inferior and middle turbinates

Management:
Conservative:
Nasal douching - The patient must be asked to douche the nose atleast twice a day with a solution prepared with:

Sodium bicarbonate - 28.4 g
Sodium diborate - 28.4 g
Sodium chloride - 56.7 g
mixed in 280 ml of luke warm water.

The crusts may be removed by forceps or suction. 25% glucose in glycerin drops can be applied to the nose thus inhibiting the growth of proteolytic organism.
In patients with histological type I atrophic rhinitis oestradiol in arachis oil 10,000 units/ml can be used as nasal drops.

Kemecetine antiozaena solution - is prepared with chloramphenicol 90mg, oestradiol dipropionate 0.64mg, vitamin D2 900 IU and propylene glycol in 1 ml of saline.

Potassium iodide can be prescribed orally to the patient in an attempt to increase the nasal secretion.
Systemic use of placental extracts have been attempted with varying degrees of success.

Surgical management:

1. Submucous injections of paraffin, and operations aimed at displacing the lateral nasal wall medially. This surgical procedure is known as Lautenslauger's operation.
2. Recently teflon strips, and autogenous cartilages have been inserted along the floor and lateral nasal wall after elevation of flaps.
3. Wilson's operation - Submucosal injection of 50% Teflon in glycerin paste.
4. Repeated stellate ganglion blocks have also been employed with some success
5. Young's operation - This surgery aims at closure of one or both nasal cavities by plastic surgery. Young's method is to raise folds of skin inside the nostril and suturing these folds together thus closing the nasal cavities. After a period of 6 to 9 months when these flaps are opened up the mucosa of the nasal cavities have found to be healed. This can be verified by postnasal examination before revision surgery is performed. Modifications of this procedure has been suggested (modified Young's

operation) where a 3mm hole is left while closing the flaps in the nasal vestibule. This enables the patient to breath through the nasal cavities. It is better if these surgical procedures are done in a staged manner, while waiting for one nose to heal before attempting on the other side.

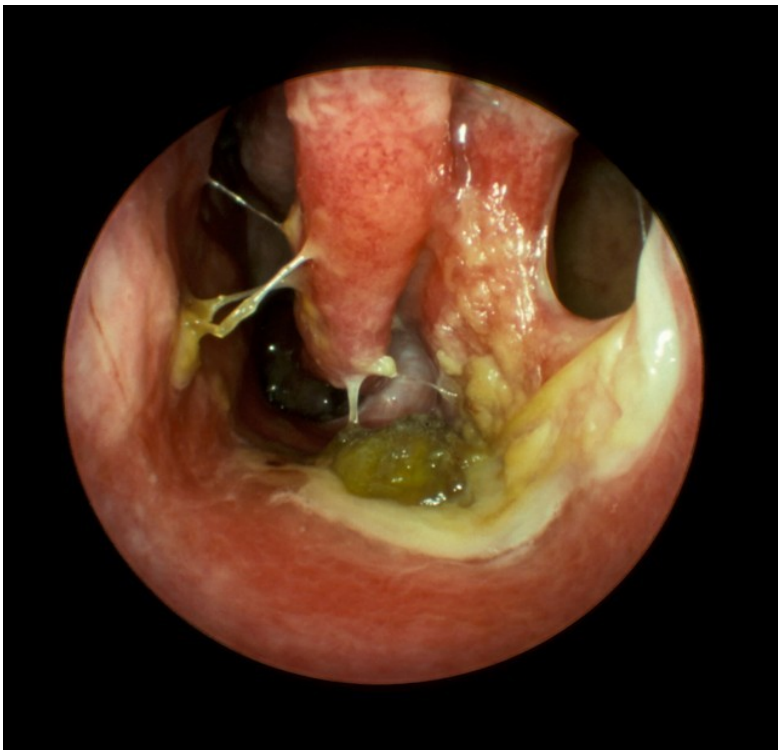


Image showing atrophic rhinitis

Systemic conditions causing rhinitis:
Many systemic disorders can affect the nasal mucosa causing rhinitis. These disorders can be classified under the following subheadings:

Granulomatous disorders: Wegner's granulomatosis, sarcoidosis, chrug strauss syndrome
Autoimmune disorders: Lupus, sjorgren syndrome, Pemphigoid
Cystic fibrosis
Tuberculosis

Signs indicating granulomatous lesions involving nasal mucosa include:

a. Persistent inflammation and crusting of nasal mucosa (Wegner's granuloma)
b. Ulceration, nasal mass, submucosal nodules, cobblestoning, extranasal manifestations and systemic symptoms (sarcoidosis)

NARES:

Non allergic rhinitis with eosinophilia syndrome. Symptoms of this condition is more or less similar to that of allergic rhinitis. Allergy test is negative. Diagnostic feature of this condition is the presence of eosinophils in the nasal smears to the extent of 10 – 20%. Aspirin sensitivity is also common in these patients.
Diagnosis is made usually by the presence of typical symptoms, nasal eosinophilia and negative allergy skin tests. Nasal turbinates appear pale and boggy in these patients.

Management:

Topical nasal steroids play a vital role in management of this condition. This is the vital difference between NARES syndrome and other types of non allergic rhinitis [19].

Intrinsic rhinitis / Vasomotor rhinitis [20]:

Synonyms: Non infective rhinitis, Non allergic rhinitis, Vasomotor rhinitis, Perenial rhinitis..

Definition:Intrinsic rhinitis is defined as a non infective and non allergic condition characterised by nasal block, rhinorrhoea and hyposmia. This is purely a medical condition.

Intrinsic rhinitis encompasses two separate disease entities. These entities show 1. inferior turbinate hypertrophy and 2. nasal polyp formation.

Clinical presentation: Rhinitis is generally characterised by 6 main symptoms: They are

1. Congestion

2. Sneezing

3. nasal itching

4. rhinorrhoea

5. hyposmia

6. post nasal discharge

Among these main symptoms nasal itching and sneezing are features of allergic rhinitis and hence are not seen in intrinsic rhinitis. All the other symptoms are manifested in intrinsic rhinitis.

Seebohm identified two groups of patients amongst those suffering from perenial rhinitis. One group had eosinophils in their nasal secretions while the other did not have any eosinophils in their nasal secretions. Accordingly he classified intrinsic / perenial rhinitis into eosinophilic and non eosinophilic types.

Eosinophilic group: This group is characterised by marked nasal congestion, profuse rhinorrhoea, hyposmia, inferior turbinate hypertrophy and mucoid nasal secretion. Nasal polyposis frequently occurred in this group of patients.

Non eosinophilic group: In these patients nasal obstruction is very mild, rhinorrhoea is very severe. They donot have significant mucosal swelling. Inferior turbinate hypertrophy is not significant. Tendency of nasal polyp formation is rare in this group.

Symptom	Eosinophilic	Non-eosinophilic
Obstruction	Moderate / severe	mild
Rhinorrhoea	Mild / moderate	severe
Sneezing/pruritis	Minimal	Minimal
Hyposmia	Usual	Rare
Mucosal swelling	Marked	Mild
Inf turbinate enlargement	Marked	Mild
Polyps	Common	Never
Sinus mucosal thickening	Common	Rare

Table showing the differences between eosninophilic and non eosinophilic types of intrinsic rhinitis

Aetiology of intrinsic rhinitis:

Theories regarding aetiology of intrinsic rhinitis are:

1. Autonomic imbalance

2. Airway hyperreactivity

3. Allergic reaction to unidentified allergen

4. Disturbances of Beta receptor function

Mechanisms of Beta receptor dysfunction:

1. Down regulation caused by excess endogenous noradrenaline stimualtion.

2. Down regulation and uncoupling of adenylate cyclase produced by the inflammatory mediator induced activation of protein kinase.

3. The action of Beta receptor inhibitory factor presumed to be an anti beta receptor autoantibody.

4. Dysfunction of Beta receptor kinase causing short term desensitisation of beta receptors after exposure to beta agonists.

Role of autonomic nervous system in causing intrinsic rhinitis:

The autonomic nervous system exerts its effects by secreting neurotransmitters ar their nerve endings. The neurotransmitters secreted are adrenaline, noradrenaline, vasoactive intestinal polypeptide, acetylcholine and neuropeptide Y.

The following transmitters are secreted by parasympathetic nerve endings: Acetyl choline, vaso active intestinal polypeptide.

The following transmitters are secreted by sympathetic nerve endings: adrenaline, noradrenaline, neuropeptide Y.

The nasal resistance to air flow is controlled by sympathetic system, whereas the nasal glands are innervated by parasympathetic nerves. Increased parasympathetic outflow causes glandular hypersecretion. Vaso active intestinal polypeptide has been known to cause this effect. The vasodilatation caused due to the effects of vaso active intestinal polypeptide is resistant to the effects of atropine.

Management:

Majority of patients with intrinsic rhinitis benefit from medical management. Only a few require surgical management.

Medical management of intrinsic rhinitis:

Eosinophilic type:

Steroids - Topical e.g. fluticasone, budesonide. A short course of systemic steriods can be administered.

Alpha receptor agonists - Systemic e.g. pseudoephidrine Topical e.g. xylometazoline (short course)

Mast cell stabilisers - Topical cromoglycate solution.

Non eosinophlic type :

Anti cholinergic - Topical e.g. ipratropium Hyosine administered orally or as a patch.

Anti cholinergic / sympathomimetic - Imipramine orally, chlorpheniramine orally.

Symptom	Type of procedure	Procedure
Nasal obstruction	Turbinate reduction	Submucosal diathermy Cryosurgery Laser cautery
	Turbinate resection	Partial resection Submucosal turbinectomy Radical turbinectomy
Rhinorrhoea	Vidian neurectomy	Excision of vidian nerve Endoscopic vidian neurectomy

Table showing the surgical indications for treatment of intrinsic rhinitis

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