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Management of VMR and AR patterns and its impact on sleep.

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Abstract

This report summarizes the scientific evidence for diagnosing and treating allergic and nonallergic rhinitis. This topic was selected by the District Ayurvedic hospital Lunawa, Moratuwa. The report provides summaries of evidence for use by different groups, including primary care practitioners, specialists and researchers. Recognizing the different interest and approaches of these groups, this report focuses on the Management of Vasomotor Rhinitis and Allergic Patterns (Nuzla or Zukam) and Its Impact on Sleep. I sought evidence on diagnostic methods that can help to get a vast idea of this project. Also I have summarized the evidence on the efficacy of Management for these conditions and causes.

Keywords: rhinitis, allergic and nonallergic, Nuzla or Zukam.

1. Introduction

The term "rhinitis" denotes nasal inflammation causing a combination of rhinorrhea, sneezing, congestion, nasal itch, and/or postnasal drainage. Allergic rhinitis is the most prevalent and most frequently recognized form of rhinitis. However, nonallergic rhinitis (NAR) is also very common, affecting millions of people. By contrast, NAR is less well understood and less often diagnosed. Nonallergic rhinitis includes a heterogeneous group of conditions, involving various triggers and distinct pathophysiologies. Nonallergic vasomotor rhinitis is the most common form of NAR and will be the primary focus of this review. Understanding and recognizing the presence of NAR in a patient is essential for the correct selection of medications and for successful treatment outcomes.

Nonallergic rhinitis (NAR) is not a single disease with 1 underlying mechanism but is instead a collection of multiple distinct conditions that cause similar nasal symptoms. Nonallergic rhinitis is at times almost indistinguishable from allergic rhinitis (AR), although typically nasal and palatal itch, sneezing, and conjunctival irritation are less prominent. Non-allergic rhinitis can and frequently does exist simultaneously with AR, a condition known as "mixed rhinitis." The most clinically prevalent form of NAR is vasomotor or idiopathic rhinitis, characterized by sporadic or persistent perennial nasal symptoms that are triggered by environmental conditions, such as strong smells; cold air; changes in temperature, humidity, and barometric pressure; strong emotions; ingesting alcoholic beverages; and changes in hormone levels. These triggers do not involve immunoglobulin E cross-linking or histamine release.

Categories of Rhinitis

According to a study by, Carl Rudolf (2009), Infectious rhinitis, Allergic rhinitis and Vasomotor rhinitis (VMR) are the main three types of identified rhinitis categories. Infectious rhinitis is commonly

caused by a viral or bacterial infection, including the common cold, which is caused by Rhinoviruses, Coronaviruses, and influenza viruses, others caused by adenoviruses, human parainfluenza viruses, human respiratory syncytial virus, enteroviruses other than rhinoviruses, metapneumovirus, and measles virus, or bacterial sinusitis, which is commonly caused by Streptococcus *pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis. Symptoms of the* common cold include rhinorrhea, sneezing, sore throat (pharyngitis), cough, congestion, and slight headache.

Vasomotor rhinitis refers to runny nose that is not due to allergy. One very common type of non-inflammatory, non-allergic rhinitis that is sometimes confused with allergy is called vasomotor rhinitis, in which certain nonspecific stimuli, including changes in environment (temperature, humidity, barometric pressure, or weather), airborne irritants (odors, fumes), dietary factors (spicy food, alcohol), sexual arousal, exercise and emotional factors trigger rhinitis. There is still much to be learned about this, but it is thought that these non-allergic triggers cause dilation of the blood vessels in the lining of the nose, which results in swelling and drainage. Vasomotor rhinitis can coexist with allergic rhinitis, and this is called "mixed rhinitis. The pathology of vasomotor rhinitis appears to involve neurogenic inflammation and is as yet not very well understood. Vasomotor rhinitis appears to be significantly more common in women than men, leading some researchers to believe that hormones play a role. In general, age of onset occurs after 20 years of age, in contrast to allergic rhinitis which can be developed at any age. Individuals with vasomotor rhinitis typically experience symptoms year-round, though symptoms may be exacerbated in the spring and autumn when rapid weather changes are more common. An estimated 17 million Universe citizens have vasomotor rhinitis. (Darlaet et al, 2012) The antihistamine azelastine, applied as a nasal spray, may be effective for vasomotor rhinitis.

Fluticasone propionate or budesonide (both are steroids) in nostril spray form may also be used for symptomatic treatment. Allergic rhinitis or hay fever may follow when an allergen such as pollen, dust, or Balsam of Peru is inhaled by an individual with a sensitized immune system, triggering antibody production. These antibodies mostly bind to mast cells, which contain histamine. When the mast cells are stimulated by an allergen, histamine (and other chemicals) is released. This causes itching, swelling, and mucus production. VMR can occur at any age, although it tends to be more common as people get older. Common triggers of vasomotor rhinitis include changes in temperature, barometric pressure, or humidity. Strong odors such as perfumes, colognes, smoke and dust can also be triggers for vasomotor rhinitis. Some patients will find that eating causes significant nasal drainage or congestion. Others will experience more difficulties during the spring and fall due to the changes in temperature and humidity that occur during these times of the year.

Rhinitis and Its Classification

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

1.2. Classification of chronic rhinitis: Chronic rhinitis can be classified into

3: Allergic – Perennial and seasonal types
1. Infectious Non allergic rhinitis: This category can be subclassified into:

2. Rhinitis caused by surgery
3. Rhinitis due to cocaine abuse
4. Rhinitis due to aging
5. Emotional rhinitis
6. Exercise induced rhinitis
7. Gustatory rhinitis
8. Hormone induced rhinitis: Hypothyroidism, pregnancy, menstrual cycle and oral contraceptives
9. Idiopathic – Vasomotor rhinitis

1.3. Non allergic -Non infectious Allergic rhinitis:

+This is defined as IgE mediated inflammation of nasal mucosa after exposure to offending allergen. This is actually not a life threatening condition but can significantly impair the quality of life of the patient 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% 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 .

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.

Mediators which are immediately 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 a 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:

1. Fatigue
2. Sleepiness
3. Malaise

1.4. 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 in turn 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 superadded bacterial infection should be suspected and antibiotics 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)

1.5. 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.

1.6. Pregnancy induced rhinitis:

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.

1.6.1. Clinical features:

Nasal congestion
Rhinorrhoea
These symptoms are aggravated by using nasal decongestant nasal sprays.

1.6.2. 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.

1.6.3.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

1.6.4.Diagnosis:

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

1.7.Medicine induced rhinitis:

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.

1.8.Aspirin exacerbated upper airway disorder:

This condition goes under the name "Samter's triad". Features of this triad include:

1. Aspirin sensitivity
2. Nasal polyposis
3. Bronchial asthma.

1.8.1.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.

1.8.2.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 Montelukast / zafirlukast can be useful in blocking the harmful effects of LT4. If nasal polypi are extensive then surgical removal should be resorted to in order to alleviate troublesome nasal obstruction.

1.8.3.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 .

1.9.Rhinitis medicamentosa:

Rhinitis medicamentosa is a condition characterised by nasal congestion without rhinorrhea 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 psychotropic drugs.

1.9.1.History:

The term rhinitis medicamentosa was coined by Lake in 1946.
Synonyms:
Rebound rhinitis / chemical rhinitis

1.9.2.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 alpha1 and alpha2 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 acetylcholine 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 thereby causing nasal congestion. In addition to sympathetic and parasympathetic innervation the nasal mucosa is richly endowed with sensory 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 hypotheses exist. Almost all of them focus on dysregulation of sympathetic / parasympathetic tone by exogenous vasoconstriction molecules

1.10.Possible mechanisms of rhinitis medicamentosa include:

1.Secondary decrease in the production of endogenous norepinephrine through a negative feedback 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.

1.11. Types of topical nasal decongestants in use:

Two types of nasal decongestants are used.

1.Sympathomimetic amines – (pseudoephedrine, 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 – (xylometazoline, 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, polyps, allergic rhinitis etc.

1.11.1.Symptoms:

Symptoms are usually confined to the nose.
1.Nasal block without significant rhinorrhoea and sneezing
2.These symptoms do not exhibit seasonal variations
3.Patient feels compelled to use nasal topical decongestants
4.Usage of these decongestants become more frequent

1.11.2.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

1.11.3.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

1.12.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.

1.12.1.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.

1.12.2.Oral prednisolone:

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.

1.12.3.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.

1.12.4.Nasal saline douching:

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

1.13.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 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 .

1.13.1.Management:

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

1.14.Atrophic rhinitis:

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. Diphtheroid 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 turbinates
3. Nutritional deficiencies
4. Syphilis.
5. Endocrine imbalances (Disease is known to worsen with pregnancy / menstruation)
6. Hereditary (Autosomal dominant 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.

1.14.1.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 nutrition 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

1.14.2.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 into two types clinically: 1. Primary atrophic rhinitis - the classic form which is supposed to arise de novo. 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 ozaenae.

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

1.14.3.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.

1.14.4.Radiographic findings:

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

1.14.5.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

1.14.6.Management:

Conservative:
Nasal douching - The patient must be asked to douche the nose at least 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 lukewarm 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.

Kemicetine anti ozaena solution - is prepared with chloramphenicol 90 mg, 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.

1.14.7.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 post nasal 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.

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: Wegener's granulomatosis, sarcoidosis, churg strauss syndrome.

Autoimmune disorders: Lupus, sjogren's syndrome,Pemphigoid ,Cystic fibrosis ,Tuberculosis

Signs indicating granulomatous lesions involving nasal mucosa include:

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

1.15.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.

1.15.1.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

Intrinsic rhinitis / Vasomotor rhinitis: Synonyms: Non infective rhinitis, Non allergic rhinitis, Vasomotor rhinitis, Perennial 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 perennial 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 / perennial rhinitis and 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 do not have significant mucosal swelling. Inferior turbinate hypertrophy is not significant. Tendency of nasal polyp formation is rare in this group.

Table 1 showing the differences between eosinophilic and non eosinophilic types of intrinsic rhinitis.

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

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 stimulation.
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.

1.16.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: Acetylcholine, vasoactive intestinal polypeptide.

The following transmitters are secreted by sympathetic nerve endings: adrenaline, noradrenaline, neuropeptide Y.
The nasal resistance to airflow is controlled by sympathetic system, whereas the nasal glands are innervated by parasympathetic nerves. Increased parasympathetic outflow causes glandular hypersecretion. Vasoactive intestinal polypeptide has been known to cause this effect. The vasodilatation caused due to the effects of vasoactive intestinal polypeptide is resistant to the effects of atropine.

1.16.1.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 steroids can be administered.
Alpha receptor agonists - Systemic e.g. pseudoephedrine Topical e.g. xylometazoline (short course)
Mast cell stabilisers - Topical cromoglycate solution

Non eosinophilic type :

Anti cholinergic - Topical e.g. ipratropium Hyoscine administered orally or as a patch. Anticholinergic / sympathomimetic - Imipramine orally, chlorpheniramine orally.

Table 2 showing the surgical indications for treatment of intrinsic rhinitis:

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

2. Unani perspective

According to Unani theory ,there are two main types of rhinitis (nuzla or zukam). First one is Nuzla involve with throat inflamation, this is due to hot (nuzla – har) or cold (nuzla -barid) .

Second one is one is Zukam involve with nose (there is no inflammation). Nuzla also can occur due to intrinsic factors(asbab- dakhila) and extrinsic factors (asbab –e- khariji).

Nuzla e-dakhila hot, cold etc and Nuzla-e- khariji onset due to behaviors like bathing in evening and early morning, late sleeping patterns,very early wakening patterns, and excessive consumption of foods like chilly, ginger, milk products,excessive consumption of cool drinks.

Nuzla can occur due to mada(abnormal humor) involvement
If involve with damavi mada called Nuzla –e-damavi
If involve with bulghami mada called Nuzla –e-bulghami
If involve with safravi mada called Nuzla-e-safravi
If involve with sawdavi mada called Nuzla-e-sawdavi

Vasomotor rhinitis isn't life-threatening. For those affected with the condition, the symptoms can be annoying, but they aren't serious. Rhinitis is defined as an inflammation of the nasal mucosa and it is characterised by nasal obstruction, runny nose (rhinorrhea), sneezing, and (itching) pruritus.

The causes of rhinitis can be broadly categorised into 3 headings:

2.1. Allergic Rhinitis

Seasonal – Hay Fever
Persistent (perennial) eg House dust mite-induced

2.2. Infectious Rhinitis

(eg the common cold). Children, particularly young children in school or day care centres, may have from eight to 12 colds each year. Viral infections are self-limiting and usually last 7-10 days.

2.3. Non-allergic, Non-Infectious eg Vasomotor Rhinitis (or Irritant rhinitis).

"Vaso" means blood vessels and "motor" refers to the nerves, which innervates nasal tissue and the blood vessels. This is sometimes referred to as idiopathic nonallergic rhinitis. It is estimated that up to 10% of the population suffers from non-allergic rhinitis.

Mixed Allergic and nonallergic Rhinitis probably account for the majority of cases. This is an important category to recognise, as allergen avoidance measures only, will give sub-optimal improvements.

2.4. Mixed Allergic and Vasomotor

probably account for the majority of cases. This is an important category to recognise, as allergen avoidance measures only will give sub-optimal improvements.

3. Features of Vasomotor Rhinitis

Vasomotor Rhinitis is chronic rhinitis that is characterised by intermittent (coming and going) episodes of sneezing, watery nasal drainage (rhinorrhea), and blood vessel congestion of the nasal mucus membranes. There appears to be a hypersensitive response to stimuli such as a dry atmosphere, air pollutants, spicy foods, alcohol, strong emotions, and some medications.

Indeed any particulate matter in the air, including pollens, dust, mould, or animal dander can bother people with VMR, even though they are not actually allergic to these things.

People with VMR are unusually sensitive to irritation and will have significant nasal symptoms even when exposed to low concentrations of irritants. Thus, vasomotor rhinitis seems to be an exaggeration of the normal nasal response to irritation, occurring at levels of exposure, which doesn't bother most people Subjects with vasomotor rhinitis fall into two general groups: **"runners"** who have "wet" rhinorrhea, and **"dry"** subjects with predominant symptoms of nasal congestion and blockage to airflow, and minimal rhinorrhea. These reactions can be provoked by non-specific irritant stimuli such as cold dry air, perfumes, paint fumes, and cigarette smoke. Subjects with predominantly rhinorrhea (sometimes referred to as cholinergic rhinitis) appear to have enhanced cholinergic glandular secretory activity, since atropine effectively reduces their secretions.

It is important to understand that VMR is a **nonspecific response** to virtually any change or impurity in the air, as opposed to allergic rhinitis (or hay fever), which involves a response to a specific protein in pollen, dust, mould, or animal dander.

3.1.Key features of VMR

There is usually no history of allergies and an irritant may or may not be identified by the patient

There is no infection causing these symptoms.

Vasomotor Rhinitis can have a variable presentation.

Most patients seem to be older than the typical patients with hay fever.

Can sometimes present with a seasonal pattern due to changes in temperature and humidity.

Patients present with rhinorrhea (thick or scanty), frontal headaches, and congested turbinates but usually no (itching) pruritus.

Some patients will find that eating (especially, spicy foods) causes more nasal dripping or congestion. The autonomic nervous system controls the blood supply into the nasal mucosa and the secretion of mucus. The diameter of the resistance vessels in the nose is mediated by the sympathetic nervous system while the parasympathetic nervous system controls glandular secretion and to a lesser extent, exerts an effect on the capacitance vessels. Either a hyperactive sympathetic nervous system or a hypoactive parasympathetic nervous system can engorge these vessels, creating an increased swelling of the nasal mucosa, and thus congestion. Activation of the parasympathetic nervous system can also increase mucosal secretions leading to excess runny nose. Overactive irritant receptors may also play a role in Vasomotor Rhinitis.

3.1.Triggers of Vasomotor Rhinitis

Causes for Vasomotor rhinitis are nonspecific stimuli, including changes in environment (temperature,humidity ,barometric pressure or weather),air born irritants(odors ,fumes),dietary factors(spicy food, alcohol),sexual arousal and emotional factors (McGraw-Hill et al, 2002)

In Ayurvedic litreture it is said that controlling the urges of passing stool;urine;vomit;tear ,indigestion,expose to dust and smoke ,excessive talking,anger,changing of weather, excessive expose to sun,not enough sleep, excessive dreaming,cold water consumption, excessive intercose causes to develop vasomotor rhinitis Also pathyapathya theory (good and bad behaviors,good and bad food:drink pattern) is very important in traditional medicine.It is said that “paththium allima” is very important and it is a main event.Honarable Lolimbaraj has mentioned that *as,”shoudn’t need medicines for patients to prevent,when there are wholesome and unwholesome foods and behaviors to prevent”(vidyajeewanaya)* In Ayurveda classics have described as,”one desires of well being in this world and *the world beyond should try his level best to follow the principles of health relating to diet,conduct and action”*

Vasomotor rhinitis occurs when the blood vessels inside your nose dilate, or expand. Dilation of the vessels in the nose produces swelling and can cause congestion. Mucus may also drain from the nose. It’s not known what causes the blood vessels in the nose to swell. Some common triggers that may produce this reaction include:

irritants in the environment such as perfumes, odors, smog, or secondhand smoke

changes in the weather and particularly dry weather

viral infections such as those associated with a cold or flu

hot or spicy foods or drinks

medications such as aspirin or ibuprofen

Many cases are associated with a specific agent or condition. Examples of such agents/conditions are:

Changes in temperature or barometric pressure, turbulent air

Perfumes, strong cooking odours, smoke

Inorganic dust (which is separate from house dust mite), air pollution

Spicy foods, alcohol

Some medications, like some blood pressure tablets

Sexual arousal

Stress (emotional or physical).

3.2.What Are the Symptoms of Vasomotor Rhinitis?

The symptoms of vasomotor rhinitis may come and go throughout the year. The symptoms may last several weeks or may be constant. Common symptoms of the condition include:

stuffy nose
runny nose
mucus in the throat, or postnasal drip

If you develop vasomotor rhinitis you typically will not have the following symptoms:

itchy nose
itchy or watery eyes
scratchy throat

These symptoms are common with allergic rhinitis, which is due to an allergy.

3.3.Other causes of non-allergic rhinitis are:

Nonallergic rhinitis with eosinophilia syndrome (Nares). Eosinophilic rhinitis (ie, perennial intrinsic rhinitis) accounts for up to 20% of rhinitis diagnosis. Some researchers believe that this may be a precursor to the aspirin triad of intrinsic asthma, nasal polyposis, and aspirin intolerance. Abnormal prostaglandin metabolism also has been implicated as a cause of NARES. Elevated eosinophil counts are present in approximately 20% of the general population's nasal smears. However, not everyone with eosinophilia has symptoms of rhinitis. A distinguishing feature of NARES is the presence of eosinophils, usually between 10-20%, on nasal smear. Generally, patients with NARES present with nasal congestion, sneezing, rhinorrhea, nasal pruritus, and hyposmia.

Occupational rhinitis is usually caused by an inhaled irritant or allergen (eg, laboratory animal antigens, grains, wood dusts, and chemicals). Frequently patients with occupational rhinitis present with concurrent occupational asthma.

Hormonal rhinitis is caused by hormonal imbalances such as pregnancy, hypothyroid states, puberty, and oral contraceptive use, conjugated estrogen use.

Drug-induced rhinitis is caused by several medications including angiotensin-converting enzyme inhibitors, reserpine, guanethidine, phentolamine, methyldopa, beta-blockers, chlorpromazine, gabapentin, penicillamine, aspirin, nonsteroidal anti-inflammatory drugs, inhaled cocaine, exogenous estrogens, and oral contraceptives.

Rhinitis medicamentosa is considered a drug-induced rhinitis and results from prolonged use (ie, longer than 5-10 days) of over-the-counter topical nasal decongestants. Typically, these patients present with extensive nasal congestion and rhinorrhea, resulting from loss of sympathetic nerve tone, rather than from the original cause of rhinitis. Normal nasal function should resume within 7-21 days following cessation of decongestants. Symptoms usually improve with nasal steroids.

Gustatory rhinitis occurs following consumption of hot and spicy foods. This is a "wet" (profuse watery) runny nose, secondary to nasal vasodilatation (dilated blood vessels) and it is due to stimulation of the vagus nerve, generally occurring within a few hours of eating the food.

3.4.Conditions often confused with non-allergic rhinitis include:

Nasal polyps
Previous trauma to the nose
Structural abnormalities eg deviated nasal septum

3.5.Diagnosis of VMR

VMR is usually diagnosed by taking a careful history and performing a thorough exam of the nose and throat. In addition, allergy testing (skin prick test) should be performed to make sure there is no allergic basis for some of the symptoms, since this would affect our treatment approach. In some cases a CT scan of the sinuses may be required to exclude chronic sinusitis or polyps. Occasionally, (few usually mild) positive skin prick test reactions are found in patients with VMR, but it does not fit the history and is therefore not relevant to the cause of the rhinitis.

4. Treatment of Vasomotor Rhinitis

Treatments for VMR

Initial treatments for mildly affected patients use single entities, but patients with more severe disease who have failed monotherapy should be tried on combination therapies. Most patients will ultimately respond to the use of combinations of nasal sprays plus an oral medication. Once under control, stepping the therapy down to the lowest effective dose of medications is suggested.(Shustermanet al 2008)

Non-drug, non-surgical

Normal saline nasal douches

Drug Therapy

Antihistamines-

Antihistamines have a variable response. They seem to help a few patients whose main symptom is runny nose, and usually when the rhinitis is mixed vasomotor and allergic.

Anticholinergic agents

Atrovent (Ipratropium bromide) nasal spray is effective in patients who have runny nose as their main symptom.

Nasal steroids

Decongestants

Decongestants, or sympathomimetic agents, are used mostly for congestion.

Examples include:

Pseudoephedrine (Sudafed) tablets. Systemic adverse effects include nervousness, insomnia, irritability, and difficulty urinating in elderly males. They are contraindicated in persons with labile or overt hypertension. Decongestants have not been shown to have an effect on blood pressure in normotensive patients.

Oxymetazoline (Drixine) nasal sprays

Xylometazoline (Otrovine) nasal spray

Topically, these drugs can cause Rhinitis Medicamentosa (a rebound congestion which occurs after taking topical formulations of these drugs for more than five days).

4.1.Treatment

Although each form of NAR should be treated individually, VMR is the most well-studied and clinically important form of NAR and the only type of NAR for which clinical studies have led to approved treatments. In the following discussion, treatment of NAR will focus on VMR, but some mention will be made of other forms of NAR where appropriate. The medications used for treating VMR have been studied less extensively than those for AR, but there are still multiple therapeutic options available. In the following table the algorithm is based on separation of VMR into 3 clinical presentations: congestion predominant, rhinorrhea predominant, and mixed form of VMR where patients experience both rhinorrhea and congestion.

	CLINICAL PRESENTATION	CONGESTION-PREDOMINANT	MIXED CONGESTION AND RHINORRHEA	RHINORRHEA-PREDOMINANT
SYMPTOM SEVERITY	MILD	NAH or NCCS	NAH or NCCS	Ipratropium (IB)
	MODERATE	NAH + NCCS	NAH + NCCS	IB + NCCS or NAH
	SEVERE	NAH + NCCS + Oral decongestant	NAH + NCCS + Oral anticholinergic & decongestant	IB + NCCS or NAH + Oral anti-cholinergic

Figure 1- Algorithm for the treatment of nonallergic VMR.

Once a patient is categorized as VMR, the predominant symptom complex determines initial treatments based on symptom severity. Initial treatments for mildly affected patients use single entities, but patients with more severe disease who have failed monotherapy should be tried on combination therapies. Most patients will ultimately respond to the use of combinations of nasal sprays plus an oral medication. Once under control, stepping the therapy down to the lowest effective dose of medications is suggested.

4.1.1.Nasal corticosteroids

Nasal corticosteroids treat inflammatory conditions regardless of etiology. There is substantial evidence that corticosteroids benefit AR, some forms of NAR including VMR, and chronic rhinosinusitis. In a study of 983 patients with NARES and non-NARES, fluticasone propionate (FP) at both 200 and 400 μg significantly improved total nasal symptoms scores when compared with placebo, and no difference was noted between the 2 concentrations. In the United States, of all the NCCSs approved by the Food and Drug Authority (FDA) available today, only FP is approved for the treatment of both AR and NAR.

Although none of the other current NCCS has received US FDA approval for use in NAR, there is some supportive data for the efficacy of intranasal budesonide and mometasone in some patients with perennial rhinitis. There is also 1 published study demonstrating that there was no benefit from FP in NAR. In that study, NAR patients receiving 200 μg of daily FP showed a reduction in inflammatory mediators but no improvement in symptoms as compared with placebo. By contrast, clinical experience suggests that all NCCSs have some effectiveness in treating VMR.

In VMR, the scent of fluticasone is sometimes a negative feature in patients for whom scent is a trigger. However, as a class, NCCS treats the broadest spectrum of NAR symptoms and seems to have at least some degree of efficacy in all NAR variants, including VMR. Thus, for the treatment of NAR, NCCSs are considered a first-line therapy.

4.1.2.Antihistamines

It is quite likely that all NAR patients have tried oral antihistamines, either in the form of over-the-counter medications or as prescribed by physicians who assume that the symptoms are caused by allergies. Histamine release has not been seen in NAR and is specifically not seen in VMR other than cold-air-induced rhinitis . Thus, the use of oral antihistamines makes little sense, and these medications have rarely been studied in VMR. A 1982 study does show that first-generation antihistamines can improve VMR symptoms when combined with a decongestant.

It is predictable that first-generation antihistamines might reduce rhinorrhea through anticholinergic actions, whereas second-generation nonsedating antihistamines have minimal anticholinergic activity. Typically, second-generation oral antihistamines are of no benefit in NAR. Oral antihistamines are generally ineffective in reducing congestion in AR and thus would not be expected to work in NAR either.

The combination of an antihistamine and a decongestant might help reduce the congestion seen in VMR, but no such indication has been approved by the US FDA. Clinical experience suggests that antihistamine/decongestant combinations are somewhat effective in VMR.

By contrast, intranasal antihistamines are very effective in treating AR (both azelastine and olopatadine are approved for treating SAR). Azelastine is also approved by the FDA for treatment of nonallergic VMR. Although azelastine is primarily an antihistamine, it is unlikely that its efficacy in VMR is due to histamine receptor blockade. Instead, it is probably azelastine actions as an anti-inflammatory and neuroinflammatory blocker that makes this medication useful in treating VMR or NAR. Azelastine has been shown to deplete inflammatory neuropeptides in the nasal mucosa; to reduce levels of proinflammatory cytokines, leukotrienes, and cell adhesion molecules; and to inhibit mast cell degranulation .

In 2 multicenter, randomized, double-blind, placebo-controlled, parallel-group clinical trials, azelastine showed considerable efficacy in the treatment of each of the symptoms of VMR or NAR, including congestion Treatment over 21 days caused a significant reduction in the total VMR symptom score from baseline when compared with placebo (P = 0.002), and every nasal symptom was effectively reduced. Symptom improvement was rapid with most patients experiencing relief within 1 week. There were no serious adverse events, although a bitter taste was experienced by some in the azelastine group. In studies of AR, onset of effect with nasal azelastine is seen in 15 to 30 minutes.

A meta-analysis has suggested that NCCSs are slightly more effective than azelastine in the treatment of AR, but no such analyses exist comparing these products in treating NAR. When NCCS and azelastine were combined in the treatment of AR, the effects of the combination were additive. In a randomized double-blind trial comparing FP alone versus azelastine alone versus the two in combination for the treatment of AR, the combination produced a further 40% reduction in

total nasal symptom scores as compared with either FP or azelastine alone. The combination of FP and azelastine reduced congestion by 48% compared with the individual components. The combination has yet to be studied in VMR or NAR, but extensive clinical experience suggests that this combination is highly effective in VMR as well. On the basis of both published clinical studies and extensive clinical experience, the use of azelastine (and possibly olopatadine) alone and in combination with NCCS is a preferred first-line treatment of VMR/NAR as well as AR.

4.1.3.Anticholinergics

Ipratropium bromide (IB) is a potent intranasal anticholinergic with utility in the treatment of rhinorrhea in AR and NAR. It has been studied in both adults and children. Ipratropium bromide specifically treats rhinorrhea and does little to improve congestion. Intranasal anticholinergics work best for rhinorrhea predominant NAR variants such as cold-air-induced rhinitis (skier's nose) and gustatory and senile rhinitis. In 28 patients with cold-air-induced rhinitis, IB reduced the symptoms and the number of tissues required during and after cold exposure (P = 0.0007 and 0.0023, respectively). In children with perennial AR or NAR, the effect of IB was superior to placebo and equivocal to beclomethasone dipropionate (BD) for the treatment of both rhinorrhea and congestion. However, IB was less effective than BD for controlling sneezing .

Similar to the nasal antihistamines, there seems to be an additive effect when IB is used in conjunction with NCCSs . In a study comparing beclomethasone versus IB versus the two combined, the combination group had better symptom control of rhinorrhea. Beclomethasone monotherapy was found to better treat sneezing and congestion than IB monotherapy. Both medications were very well tolerated.

Oral anticholinergics such as methscopolamine have not been studied in NAR but likely improve symptoms particularly in rhinorrhea predominant disease or in patients with significant postnasal drainage. Many first-generation antihistamines and decongestants also have strong anticholinergic properties. However, side effects such as dry mouth, sedation, and urinary hesitancy limit the usefulness of these drugs. Clinical experience suggests that oral methscopolamine combined with an oral first-generation antihistamine is helpful in treating patients with postnasal drip and that adding this combination to nasal IB, nasal antihistamines, or NCCS is useful.

4.1.4.Decongestants

Both oral and topical decongestants effectively treat congestion regardless of cause; however, none have been studied for NAR. Oral pseudoephedrine is an effective decongestant and can be considered for chronic use. However, side effects such as neurogenic and cardiac stimulation, palpitations, and insomnia affect a significant number of patients. Furthermore, the medication is relatively contraindicated in patients with hypertension. Thus, pseudoephedrine must be used cautiously. Phenylephrine is also an oral decongestant. It has been studied far less than pseudoephedrine and is considered a generally less potent medication.Topical decongestants such as oxymetazoline and phenylephrine are fast acting potent local decongestants. These medications cannot be used chronically because continual use for more than 3 to 10 days leads to rhinitis medicamentosa. For NAR patients with intermittent nasal congestion, a topical decongestant can be used for short-term relief of congestion.

4.1.5.Other NAR Therapies

Although NAR effects many patients, very few medications have been adequately studied for the treatment of this condition. In patients who do not adequately respond to NCCSs, intranasal antihistamines, or IB, other agents can be considered. There are a few limited studies examining intranasal capsaicin in NAR. In theory, repetitive capsaicin application depletes certain neuroinflammatory chemicals. Van Rijswijk et al did demonstrate decreased nasal symptoms in VMR patients treated with capsaicin. Similarly, botulinum toxin A injected into the inferior and middle turbinates of patients with NAR has been shown to decrease congestion, sneezing, rhinorrhea, and nasal itch.In patients with congestion-predominant NAR and turbinate hypertrophy, surgical reduction of the inferior turbinates may be of some benefit. Nasal washing with isotonic or hypertonic saline has a demonstrated benefit particularly in chronic rhinosinusitis and seems to benefit some NAR patients. Antileukotrienes have not been studied in NAR, but there is at least some theoretical benefit in patients with aspirin sensitivity and/or nasal polyposis. One controlled trial has demonstrated some efficacy using acupuncture in NAR .

4.1.6.Surgery

If the rhinitis does not respond to drug therapy, very rarely surgical procedures can be considered. Some of the procedures that have been performed in the past include:

Cryosurgery affects the mucosa and submucosa, making it a quite successful procedure for congestion. However, there is sometimes prolonged postoperative nasal congestion and the possibility of damage to the nasal septum.

Vidian neurectomy disrupts both sympathetic and parasympathetic fibers to the mucosa and it mainly diminishes rhinorrhea.

If chronic hypertrophic changes appear in the mucosa, a number of surgical procedures can be tried. These include:

Cauterization can be accomplished via silver nitrate or electrical current, however it only affects the mucosa.

Cryosurgery is considered superior to cauterization because it also affects the submucosa.

Submucosal resection of the conchal bone is a difficult procedure with much post-operative bleeding. Partial or total inferior turbinate resection works well for nasal congestion but can give post-operative bleeding and crusting.

4.2.How Is Vasomotor Rhinitis Diagnosed?

4.2.1.Diagnosis of VMR

VMR is characterized by sporadic or persistent nasal symptoms that are triggered by environmental conditions, such as strong smells; exposure to cold air; changes in temperature, humidity, and barometric pressure; strong emotions; ingesting alcoholic beverages; and changes in hormone levels. The diagnosis of VMR is primarily made by clinical history. If a patient has appropriate nasal symptoms (usually rhinorrhea, congestion, postnasal drip, headaches, throat clearing, and coughing) triggered by one or more environmental irritants, then VMR is present. Concomitant ocular symptoms tend to be minimal, and the symptoms of nasal and palatal itch as well as sneezing spells are not common. Unlike AR, VMR is usually of adult onset. *(Blackwell et al, 2008)* The diagnosis of VMR is based solely on the patient's history of symptoms and their triggers

The doctor can diagnose vasomotor rhinitis after ruling out other causes of patient's symptoms.If you have symptoms of vasomotor rhinitis, your doctor will first perform different tests to see if your rhinitis is due to an allergy or other health problem. To determine if you have an allergy, your doctor may order a skin test to identify any allergies that you may have or a blood test to see if your immune system is functioning normally. The doctor may also order tests to see if you have any sinus problems that may be causing your rhinitis. Tests may include a nasal endoscope to look inside your nose or a CT scan of your sinuses.

4.3.What Is the Long-Term Outlook?

If you develop vasomotor rhinitis, your outlook will depend on the severity of your symptoms. Treatment with over-the-counter or prescription medication may help reduce or eliminate your symptoms. Correcting underlying health conditions such as a deviated septum may also help reduce your symptoms and improve your prognosis.

Table 3 Treatment Recommendations for Vasomotor Rhinitis: A Stepwise Approach

Medication class	Product	Effect	Side Effects
Topical antihistamines	Azelastine (Astelin)	Improvement in rhinorrhea, sneezing, postnasal drip, and nasal congestion	No serious or unexpected adverse events; bitter taste
Topical corticosteroids	Mometasone furoate (Nasonex)	Improvement in nasal obstruction and congestion scores	Epistaxis, nasal irritation
Topical corticosteroids	Budesonide (Rhinocort), beclomethasone (Beclovent), triamcinolone acetonide (Kenalog)	Improvement in nasal obstruction and congestion scores	Epistaxis, headache, nasal congestion
Topical cromoglycate	Cromolyn sodium (Intal)	Decrease in sneezing and congestion scores	Nasal irritation, headache, nasal congestion
Topical anticholinergics	Ipratropium (Atrovent)	Reduced rhinorrhea only	Minor adverse effects; nasal dryness and irritation
Other agents not recommended by Research			
Oral antihistamines	Sedating and nonsedating	Research outcome not identified	Somnolence, dizziness, dry mouth, headache
Oral sympathomimetics	Only phenylpropanolamine (not available in the United States) was studied.	Withdrawn from the market; no other oral decongestant was identified or specifically studied.	
Leukotriene modifiers	Not identified in any trial on nonallergic rhinitis		
Other agents not discussed by Research: evidence for use lacking, empiric use possible			
Topical decongestants	Oxymetazoline (Nezeril, Afrin, Dristan)	Improvement in congestion	
Oral decongestants	Pseudoephedrine	Improvement in congestion	

Note: Use of topical antihistamines and corticosteroids is approved by the World Food and Drug Administration.

4.3.1.Special Populations

4.3.1.1.Children

Preventive and nonpharmacologic approaches should be tried before beginning medication in children. Approved for use in patients six years and older, nasal anticholinergics such as ipratropium (Atrovent) often reduce rhinorrhea without the undesirable side effects of sedation and fatigue sometimes associated with oral antihistamine use. However, anticholinergics have no effect on the other symptoms of vasomotor rhinitis. Investigators conducted a multicenter, double-blind, placebo-controlled, parallel-group trial 13 in 204 children (six to 12 years of age) and adolescents (13 to 18 years of age) with allergic or nonallergic perennial rhinitis.

Patients with nonallergic perennial rhinitis who used ipratropium had a 41 percent mean decrease in severity and a 37 percent decrease in duration of rhinitis with excellent tolerability, compared with decreases of 15 and 17 percent in severity and duration, respectively, in the placebo group.

Certain nasal corticosteroids, such as mometasone furoate (Nasonex), are approved by the World. Food and Drug Administration for children older than two years and improve the symptoms of congestion and nasal obstruction. Investigators conducted a randomized, double-blind, placebo-controlled, 12-month study to monitor growth in children during treatment with mometasone furoate. A total of 82 patients, three to nine years of age, completed the study. There was no evidence of growth retardation or hypothalamic-pituitary-adrenal axis suppression. Although short-term use studies purporting safety are quoted in the literature, budesonide (Rhinocort), beclomethasone (Beclovent), and triamcinolone acetonide (Kenalog) are not recommended for children younger than six years because of continued concern over possible long-term growth suppression by these older agents. Cromolyn sodium (Intal) can be used to manage symptoms of sneezing and congestion in children older than two years. As in adults, traditional oral antihistamines and newer less-sedating antihistamines have no established beneficial effects on vasomotor rhinitis in children. Prolonged use of topical nasal decongestants can cause irritation and rhinitis medicamentosa without proven benefit. If a therapeutic trial of one of these agents is attempted because of treatment failures with recommended agents, judicious and time-limited use should be considered.

4.3.1.2.Athletes

Topical antihistamines, topical corticosteroids, and topical anticholinergics are treatments permitted by the International Olympic Committee. As of January 1, 2016, the World Anti-Doping Code no longer bans the use of pseudoephedrine, but systemic decongestants are included in the 2016 monitoring program. The code does not prohibit the use of topical decongestants. The stepwise approach to manage athletes should be the same as that used with other populations. A topical antihistamine (e.g., azelastine [Astelin]), topical corticosteroids (e.g., budesonide), and topical anticholinergics (e.g., ipratropium) may be tried. The 2016 World Anti-Doping Code requires an Abbreviated Therapeutic Use Exemption form to notify relevant agencies about the use of topical corticosteroids. Empiric short-term treatment with topical decongestants may be considered if these agents fail.

4.3.1.3.Pregnant women

Symptoms of rhinitis can increase during pregnancy. This increase is thought to be caused by progesterone- and estrogen-induced glandular secretion, augmented by nasal vascular pooling from vasodilation and increased blood volume. Vasomotor rhinitis in pregnancy responds well to intranasal saline instillation. Potential risks versus benefits should be considered in the use of FDA-approved topical anticholinergics (pregnancy category B), topical antihistamines (pregnancy category C), and topical corticosteroids (pregnancy category C). Topical decongestants (pregnancy category C) can provide good short-term relief. Exercise appropriate for physical condition and gestational age also may reduce symptoms.

4.3.1.4.Older Adults

Three types of nonallergic rhinitis commonly occur in older patients. The first, vasomotor rhinitis, is thought to be caused by increased cholinergic activity and is similar to that occurring in younger patients. The second type, gustatory rhinitis, is associated with profuse, watery rhinorrhea that may be exacerbated by eating. The third form is believed to arise from alpha-adrenergic hyperactivity, stimulated by the regular use of antihypertensives. All three types respond well to ipratropium nasal spray. Narrow-angle glaucoma is a relative contraindication to the use of ipratropium.

4.3.1.5.Prognosis and Additional Therapies

Although no single agent is uniformly effective in controlling the many and varied symptoms of vasomotor rhinitis, available evidence supports a stepwise application of several agents after a careful history and physical examination. Additional therapies, for which Research felt there was no strong evidence base, may be tried if the approved approaches fail. These therapies include topical decongestants, oral decongestants, and local application of silver nitrate solutions by an otolaryngologist. Sphenopalatine blocks, also performed by otolaryngologists, are reserved for seriously affected patients who do not respond to other interventions and whose lives are altered significantly by their symptoms. The submucosal injection of botulinum toxin type A (Botox) has been studied in dog models and may yet prove to be of value.

The incidence of VMR varies from study to study. Almost all publications on VMR are found in literature review. Thus, it is unclear whether the incidence or the age and sex distribution applies to populations not yet studied elsewhere in the world. In 1 survey of ***Sri Lankan medical practices,*** the classification of patients with rhinitis was 43% AR, 23% VMR, and 34% mixed rhinitis (rhinitis with both AR and VMR features). These data suggest that at least 57% of rhinitis patients have some contribution from VMR causing their rhinitis symptoms. Similar world studies have found that approximately 1 in 4 patients complaining of nasal symptoms have pure VMR. Recent estimates suggest that 5 million people in Sri Lanka have VMR, with a total prevalence of greater than 200 million worldwide. VMR rhinitis tends to be adult onset, with the typical age of presentation between 30 and 60 years. Once symptoms begin, they frequently last a lifetime. If VMR is present in pediatric populations, it is more likely to be anatomic in nature and to be caused by either adenoid or turbinate hypertrophy, leading to persistent nasal obstruction. In adults, most studies report a clear female predominance, with estimates ranging from 58% to 71% of those affected being female.

In a study classifying a population of both adults and adolescents, female predominance held true with approximately double the prevalence of VMR in women. The financial impact of VMR has not been studied directly, but numerous studies have looked at the direct and the indirect costs of AR. It is likely that because most studies indicate that at least 1 in 4 patients with nasal symptoms have pure VMR, the rough cost of the condition is approximately one third of AR.

4.4.Present Study in Sri Lanka

Many studies were conducted under clinical importance, differential diagnosis, and effective treatments of VMR in world. But in this field of study, researchers didn't put much consideration on causes of VMR. There can be various identified & unidentified causes for vasomotor rhinitis. There is still much to be learned about this, but it is though that these non allergic triggers cause dilation of the blood vessels in the lining of the nose, which results in swelling and drainage. This is a really disturbing condition for patients who have vasomotor rhinitis which disturbs their normal daily life routine. It mainly affects personal life as well as government production, development and economy.

It is observed that many vasomotor rhinitis patients who come to Ayurvedic hospitals have critical sleeping history ,when considering about the patient's history. The symptoms causing really disturbance to the patient , even to his or her personality. Government has to spend thousands of thousand money for medicines to control symptoms of vasomotor rhinitis. But unfortunately there is no survey has done so far to find out the relationship between vasomotor rhinitis and sleeping pattern. If we can do a research on this topic it will really important to develop a healthy community which will enhance the productivity of the country.

When considering the bad effect of VMR patients, still not enough consideration given to socio demographic distribution, sleeping pattern, occupation,lifestyle patterns etc, there can be a relationship between above mentioned reasons. Present study mainly focused on sleeping pattern and vasomotor rhinitis.

5.Allergic Rhinitis and Its Consequences on Quality of Sleep

Background

Allergic rhinitis (AR) is common and has been shown to impair social life and sleep. Patients with severe symptoms may have more sleep disturbances than those with a mild form of the disease, but this has never been assessed using a validated tool. The

objective of our study was to assess, in patients with AR, whether duration and severity of AR are associated with sleep impairment.

Methods

A nationwide controlled cross-sectional epidemiological study was carried out. A representative sample of 260 French ear, nose, and throat and allergy specialists enrolled 591 patients with AR of at least 1 year's duration. Sleep disorders, sleep quality, and AR were assessed using validated tools (Sleep Disorders Questionnaire, Epworth Sleepiness Scale, and Score for Allergic Rhinitis). The severity of AR was assessed using the Allergic Rhinitis and its Impact on Asthma classification.

Results

All dimensions of sleep were impaired by AR, particularly by the severe type. Sleep was significantly more impaired in patients with severe AR than in those with the mild type. The duration of AR (intermittent or persistent) had no effect on sleep.

Conclusion

These data underline the close relationship between AR and sleep and highlight the need for clinicians, particularly general practitioners, to be attentive in this respect. Allergic rhinitis (AR) is a common condition that affects, on average, 20% to 50% of the general population. Sleep disorders are also very common in the general population. Insomnia affects from 20% to 30% of adults, and severe insomnia about 10% of adults.Sleep apnea was objectively found in 9% of women and 24% of men, and hypersomnia, defined as the occurrence of episodes of sleepiness during daily life, affects 4% to 6% of the general population in its severe form and from 15% to 20% in its moderate form.

Allergic rhinitis has been shown to impair quality of life, according to generic tools such as the Short-Form 36-Item Health Status questionnaire, or more disease-specific tools, such as the Rhinoconjunctivitis Quality of Life Questionnaire. Sleep disorders have an impact on patients' quality of life. According to the affected patients, the most important problems related to their condition are the impairment of sleep quality and its consequences, such as daytime sleepiness and impaired concentration. Several studies have shown the relationships among AR and nasal obstruction and abnormal breathing during sleep, snoring, and sleep apnea. In a European cross-sectional survey that investigated the prevalence of sleep disturbances and daytime sleepiness, a positive relationship was found between asthma and daytime sleepiness and apnea; among asthmatic patients, 71% also had AR, which was found to be independently related to increased difficulty in falling asleep, and daytime sleepiness. Poorly controlled symptoms of AR may also contribute to sleep loss or disturbance, resultant daytime fatigue, and decreased overall cognitive functioning. Insomnia also needs to be considered, and, surprisingly, we could not find any study that specifically addressed the epidemiology of sleep disorders in patients with AR. Allergic rhinitis and sleep disorders are therefore 2 prevalent complaints in the general population, and data on their co-occurrence is accumulating. Yet, to more knowledge, there has been no attempt to explore this link.

Therefore, we conducted the *Dreams* to assess the importance of sleep impairment in AR according to its severity and duration.

5.1.Methods

The Dreams was a controlled cross-sectional study performed with a sample of patients with AR who were being treated by an allergist or ear, nose, and throat specialist. The participating clinicians were randomly selected from the overall population of their respective specialization in Sri Lanka.These specialists were selected from all regions of Sri Lanka to rule out any geographic or seasonal parameter and to take into account the population distribution in the country (stratification for geographical area). Each clinician had to include the first patients (at least 2 patients) who presented with AR and who met the inclusion criteria.

5.2.Subjects

Inclusion criteria were that patients—men or women—had to be aged 18 to 50 years, have AR of at least 1 year's duration, and have a score of 7 or higher on the self-administered Score for Allergic Rhinitis questionnaire (see the following subsection). Exclusion criteria were the presence of grade III or IV nasal polyps and/or major nasal septum deviation.

A control group of individuals without AR was matched for age, sex, and geographical area with the first 2 patients selected by the physicians. They were selected from among men and women who were being treated at general practitioners' clinics.

5.3.Establishing ER

The Score for Allergic Rhinitis is a validated diagnostic tool. A score of 7 or greater, the threshold used in the study described herein, has been shown to provide satisfactory discrimination between patients with AR and those without AR. The Score for Allergic Rhinitis questionnaire was completed by patients, and a score was assigned by the clinician. Clinicians completed a standardized questionnaire that covered demographics (their own and those of their patients) and patient characteristics.

Information recorded for each patient included the following: socioeconomic status category, smoking habits, type of AR (according to the Allergic Rhinitis and Its Impact on Asthma [ARIA] classification), symptoms and duration of AR, concomitant allergic disorders (ie, previously diagnosed), therapeutic treatment of patients with AR, and ongoing treatment of concomitant diseases (responses included use of anxiolytic drugs, hypnotics, antimigraine agents, and nonsteroidal anti-inflammatory drugs).

5.4.Assessment of Sleep

Sleep disorders and sleepiness were assessed by self-administered questionnaires: the Sleep Disorders Questionnaire, and the Sleepiness Scale score. The questionnaire based on the Sleep Questionnaire and Evaluation of Wakefulness. This version has been validated in several epidemiological studies. It covers sleep habits; sleep disorders; alertness during the daytime; and psychobehavioral items on mood, memory, and sexual behavior.

Sleep disorders were defined using the categories *items* and *criteria* as shown in the following figure.. All but 1 of the items are derived from questions and possible responses. The selection of sleep disorders was based on following reference document:

The *International Classification of Sleep Disorders* and the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Revised (DSM-IV-R).*

Items Used to Define Sleep Disorders	Criteria for Defining Sleep Disorders
Item A How many minutes does it take for you to fall asleep? (response: >30 min)	Criterion 0 At least 1 yes to the question, "Do you have sleep problems?"
Item B How many times do you wake up each night? (response: ≥2)	Criterion 1 Items (A or B) and (C and D)
Item C "Once I wake up, I cannot fall asleep." (response: every night or every week)	Criterion 2 Duration of Sleep Disorder Longer Than 1 Month
Item D "After a normal night's sleep, I feel..." (response: a bit tired or very tired)	Criterion 3 Items E or F
Item E "I fall asleep during the day, during work, while listening to the radio or music, while traveling, in front of the TV." (response: every night/every week)	Criterion 4 Regular Use of Sedatives
Item F Does it seem that your memory has suddenly gotten worse? (response: yes)	Criterion 5 Hypersomnia: Items E or G
Item G Epworth Sleepiness Scale score >10	Criterion 6 Sleep Apnea Syndrome: Items E, H, and I
Item H Has anyone ever told you that you snore loudly? (response: yes)	Criterion 7 Stop Breathing for Several Seconds During Sleep
Item I Do you snore? (response: often or almost every day)	Criterion 8 Insomnia: Criteria 0 and 1 and Criterion 2 or 3
	Criterion 9 Severe Insomnia: Criteria 0-3 or Criteria 4
	Criterion 10 Observed Apnea and Sleepiness: Criterion 7 and Item E

Figure 3. Definition of sleep disorders. Criteria for defining sleep disorders were based on the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Revised, and the International Classification of Sleep Disorders classification criteria. Items A to H were compared with these criteria to assess minimum criteria for the most common sleep disorders.

This also collected data on 5 specific sleep complaints: difficulties in falling asleep, frequent nocturnal awakenings, early awakenings, poor-quality sleep, and feeling a lack of sleep. The sleep complaints are essentially drawn from the *DSM-IV-R* definition of insomnia. Severe insomnia was defined as the presence of at least 2 sleep complaints according to the *DSM-IV-R* definition. In addition, the minimum criteria from the *International Classification of Sleep Disorders* for insomnia, idiopathic hypersomnia, snoring, and sleep apnea were used. Information was also collected on duration of sleep disorders.

The Epworth Sleepiness Scale, a self-administered questionnaire, is a subjective tool constructed and tested in the early 1990s it has been validated against objective tests such as the Multiple Sleep Latency Test.

5.5.Statistical Analysis

I performed data management and statistical analysis by using the SAS software package (version 8; SAS Institute Inc, Cary, NC). Descriptive analyses for qualitative variables included number, frequency, and the 95% confidence interval, whereas quantitative variables were analyzed in terms of mean value, standard deviation, and median and extreme values. Collected data were analyzed for the total population and by subgroups of type and severity of AR (intermittent or persistent; mild or moderate to severe).

I created a logistical model for each type of sleep disturbance. A general linear model was used to study the relation between severity of AR and sleep symptoms. For description of quantitative variables, the Kruskal-Wallis test with Bonferroni-Dunn post hoc analysis was used.

I have determined predictive factors for sleep impairment by using logistic regression analysis after adjustment for potential confounders (sex and age). The associations were then expressed using odds ratios. A step-by-step selection of sociodemographic and clinical criteria was performed by using a univariate approach ($\chi 2$ or Fisher exact test for non ordinal qualitative variables, Kruskal-Wallis test for ordinal data, and analysis of variance or Kruskal-Wallis test for quantitative variables)

5.6.Results

From April to September 2016, a total of 800 patients were evaluated by clinicians. Following verification of criteria, 440 of these patients, recruited by 12 clinicians, were retained for the analysis and thus constituted the DREAMS population. There were 502 subjects in the control group.

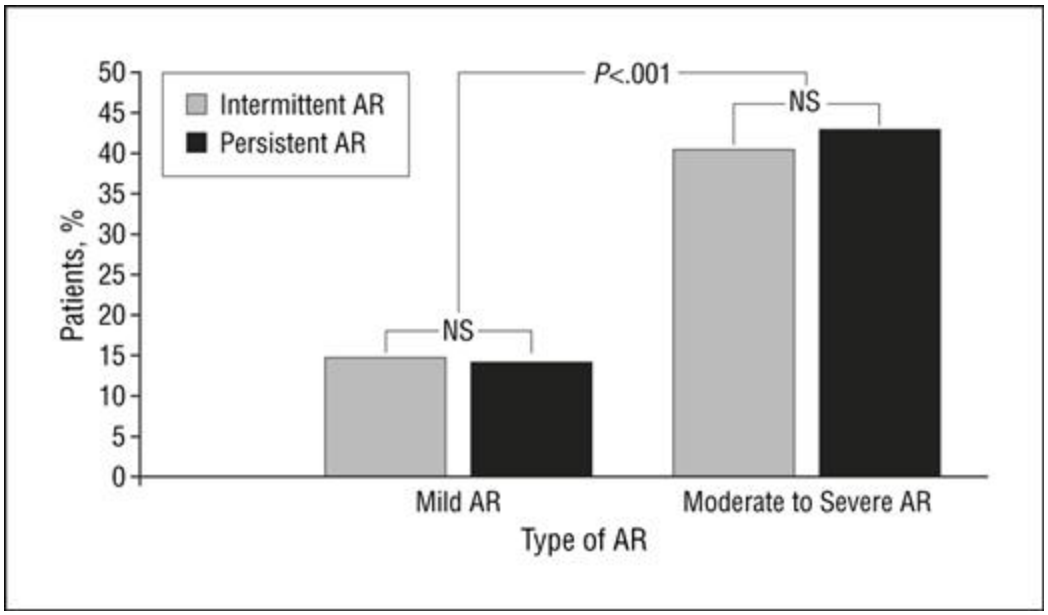

Figure 4Distribution of the population of patients with insomnia who also have allergic rhinitis (AR) (n = 19) according the type of Allergic Rhinitis and Its Impact on Asthma classification. NS indicates nonsignificant impact of AR frequency on insomnia.

Table 4 Prevalence of Sleep Complaints and Sleep Disorders According to the Type of Allergic Rhinitis (AR) (ARIA Classification)

	Patients With AR, %					
Complaint or Disorder	59 With Mild Intermittent AR	81 With Mild Persistent AR	100 With Moderate to Severe Intermittent AR	351 With Moderate to Severe Persistent AR	591 Total	502 Patients in Control Group*
Sleep complaints						
Difficulty in falling asleep	19.6	14.6	49.5	49.6	41.6	18.3
Nocturnal awakening	14.0	15.9	53.9	51.3	42.8	20.5
Early awakening	18.2	12.4	33.3	33.2	28.7	12.8
Nonrestorative sleep	22.8	18.3	56.2	55.4	46.8	19.6
Feeling of lack of sleep	47.4	48.8	61.3	98.8	63.2	25.4
Snoring	28.1	32.5	34.0	46.0	40.3	27.1
ESS score >10	6.4	16.2	23.4	27.3	23.3	17.2
Sleep disorders						
Insomnia	14.6	14.3	40.5	42.8	35.8	16.0
Severe insomnia	10.6	10.3	27.0	27.2	23.2	10.4
Sleep apnea syndrome	1.8	1.2	0	5.8	3.8	0.5
Hypersomnia	15.6	22.7	33.7	36.9	32.6	24.3†
Other						
Regular use of sedatives	3.5	4.8	14.4	7.2	7.7	3.4†

Abbreviations: ARIA, Allergic Rhinitis and Its Impact on Asthma; ESS, Epworth Sleepiness Scale.
*Except where noted, $P<.001$ for total patients with AR vs control group.
†$P=.003$ for total patients with AR compared with control group.

Table 5. Predictive Factors for Sleep Disturbances in Allergic Rhinitis (AR)*

Table 2. Predictive Factors for Sleep Disturbances in Allergic Rhinitis (AR)*

Characteristic	Insomnia OR (95% CI)	*P* Value	Severe Insomnia OR (95% CI)†	*P* Value	Hypersomnia OR (95% CI)	*P* Value	Sleep Apnea OR (95% CI)	*P* Value
Severity of AR (moderate to severe vs mild)	4.04 (2.37-7.22)	<.001	2.67 (1.46-5.24)	.002	2.11 (1.29-3.58)	.004	NS	
Type of AR (persistent vs intermittent)	NS		NS		NS		9.58 (1.88-175.80)	.03
Treatments‡								
Anxiolytics	5.44 (2.30-14.42)	<.001	6.31 (2.69-15.76)	<.001	NS		NS	
Hypnotics	NS		NS		3.97 (1.68-10.14)	.002	NS	
Antimigraine agents	NS		NS				6.14 (1.12-25.62)	.02
Asthma	NS		1.75 (1.07-2.84)	.03	NS		NS	

Abbreviations: CI, confidence interval; NS, nonsignificant; OR, odds ratio.
*Patients with moderate to severe AR have a higher risk of insomnia, severe insomnia, and hypersomnia than those with mild AR. Patients with chronic AR have a higher risk of obstructive apnea than those with intermittent AR.
†Adjusted OR for sex and age (profile likelihood, 95% CI); profile $>\chi^2$.
‡Treatment with nonsteroidal anti-inflammatory drugs was a nonsignificant predictive factor.

5.6.1. Impact of AR on Snoring and Apnea

Snoring was significantly more often reported in patients with AR than in the control group ($P<.001$). Based on the minimum subjective criteria (snoring loudly everyday and daytime sleepiness), sleep apnea syndrome seemed also to be more prevalent in patients with AR than in the control group (3.8% vs 0.5%; $P<.001$).

5.6.2. Impact of AR-Induced Poor Quality of Sleep on Everyday Living

A total of 43.7% of patients with AR reported a feeling of fatigue on awakening despite a normal night's sleep. Headache at awakening, anxiety, and depression as contributing factors of sleep problems and daytime somnolence were significantly more frequently reported by patients with AR than by the control group ($P<.001$).

Severity of AR had an adverse effect on memory ($P = .01$) and mood ($P = .003$). Compared with the control group, significantly more patients with AR reported memory impairment and decreased sexual activity ($P<.01$).

The severity of AR significantly influenced the mean duration of nocturnal sleep during the week and weekends (P = .001 and P = .002, respectively), the frequency of daytime sleepiness (P = .04), the time necessary to fall asleep (P<.001), and the necessary intake of sedative drugs (P = .009).

5.6.3.Predictive Factors for Sleep Disturbances in Patients With AR

The analyses identified several factors predictive for sleep disorders in patients with AR. Above tables presents the factors with significant predictive value for insomnia, severe insomnia, hypersomnia, and sleep apnea.

The severity of the disease was found to be highly correlated with all types of sleep disorders whereas some concomitant medications were predictive for certain types (use of anxiolytic drugs, for example, showed strong correlation with insomnia and severe insomnia).

Male sex showed a high correlation with sleep apnea (odds ratio, 5.9; 95% confidence interval, 2.0-22.4;P = .003), whereas asthma was correlated with severe insomnia (odds ratio, 1.75; 95% confidence interval, 1.07-2.84; P = .03). Age, smoking habits, and residence (urban vs rural) did not have any predictive value.

The present controlled epidemiological study provides important information on the relationship of AR to sleep complaints and sleep disorders. The study was conducted following strict methods and using validated tools (questionnaires) that allowed precise characterization of the study population in terms of respiratory status and sleeping status. However, questionnaires may be insufficient to assess accurately some sleep disorders (eg, sleep apnea) that require confirmation by night polygraphy or polysomnography.

It is known that AR adversely affects quality of life, but to our knowledge, prior to the study described herein, the frequency and pattern of sleep disorders in the population with AR had never been determined. Moreover, this study also addresses the consequences of sleep disorders on everyday living.

The results show a significant impact of AR on all dimensions of sleep quality and, consequently, a lower quality of life as reflected by more somnolence; daytime fatigue and sleepiness; and impaired memory, mood, and sexuality, with a significantly increased consumption of alcohol and sedatives in cases compared with the control group. My findings are consistent with other published observations.

A limitation of this study is that I did not consider the potential influence of cofactors such as anxiety and depression or comorbidities such as asthma on sleep quality. The focus of this study was not on the mechanisms that link AR with altered sleep but rather to examine whether frequency and severity of AR were associated with sleep impairment. Disorders other than AR, such as comorbid disorders (eg, obstructive sleep apnea and asthma) may also have an impact on sleep quality. Therefore, comprehensive treatment of sleep disturbances in patients with AR will likely require a multimodal approach. Although the duration of AR (intermittent or persistent) had no effect on most of the evaluated parameters, the severity of the disease had a significant influence on all considered aspects.

It is important to state that possible bias in the selection and in the interview of patients may have influenced the results of the study. Patients who have AR and who have been specifically interviewed about sleep may recall their troubles more intensively than the control group interviewed in the general population. However, it seems difficult to avoid this bias in a study focused on AR, which demands a high quality of diagnosis made by ear, nose, and throat and allergy specialists. Another possible bias is that patients seen by specialists may have more medical disorders and therefore more sleep disorders than patients seen by general practitioners. But the French social security system allows patients to visit specialists as easily as general practitioners, and sleep disorders are one of the main complaints expressed by patients of general practitioners. It seems thus unlikely that patients of ear, nose, and throat specialists have more sleep problems due to other diseases than do patients of general practitioners.

General practitioners, as well as pulmonary; allergy; and ear, nose, and throat specialists, have to be made aware of the relationship between AR and sleep disorders. My findings suggest that patients consulting for their AR should be routinely questioned about their sleep quality and existing daytime somnolence. This could lead to early detection and treatment of sleep disorders in these patients. The onus is on health care

professionals to make the link between AR and sleep problems in their patients. Treating AR or other nasal symptoms may improve dramatically the quality of sleep. In the long term, such a strategy would have positive repercussions on a societal level; for example, the numbers of road and work accidents would be reduced. Considering the high incidence of AR and the high rate of associated sleep disorders, the issue is one of public health.

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7. Appendix

7.1.Questionnaire 1 (Translated)

Which of the following options describes you? (You may choose more than one)

A. My doctor has told me that I have asthma. S

B. My doctor has told me that I have allergic rhinitis.

C. I sometimes have breathing problems like wheezing and shortness of breath, but I've never been diagnosed with asthma by my doctor.

D. I sometimes have symptoms like a runny nose, sneezing, and/or itchy, watery eyes, but I've never been diagnosed with allergic rhinitis by my doctor

7.2.Sleep Pattern Questionnaire 2

1st Survey This is a sleep pattern survey for research purposes. Your feedback for the following questions will be of invaluable assistance to my research.
Personal details:
Name: _____
Address: _____
Age: _____
Sex:

Female Male Other

Instructions:
1. Please read each question very carefully before answering.
2. Each question should be answered independently of others.
3. Some questions have a selection of answers. For each question place a cross alongside ONE answer only.
4. Some questions have a scale instead of a selection of answers. Place a cross (X) at the appropriate point along the scale.
5. Please answer each question as honestly as possible. Both your answers and the results will be kept in strict confidence.

Questions:

1. Using the PAST 7 DAYS as an example, how long would it take you to fall asleep (in minute)?

2. Using the PAST 7 DAYS as an example, how many hours of ACTUAL SLEEP do you get on an average each night? (This may be different from the number of hours you spend in bed.)

3. At what time in the evening do you feel tired and as a result, in need of sleep?

4. During the PAST 7 DAYS, how often have you had trouble sleeping because you...

(a) Cannot get to sleep within 30 minutes of getting to bed …

Not at all …

Once or twice …

Three or four times …

More than four times

(b) Wake up in the middle of the night or early morning …
Not at all …

Not at all …

Once or twice …

Three or four times …

More than four times

(c) Feel too cold or hot

Not at all …

Once or twice …

Three or four times …

More than four times

(d) Have had dreams

Not at all …

Once or twice …

Three or four times …

More than four times

(e) Have pain

Not at all …

Once or twice …

Three or four times …

More than four times

(f) Other reasons, please describe

How often during the past week have you had trouble sleeping because of this?

Not at all …

Once or twice …

Three or four times …

More than four times

5. During the PAST 7 DAYS, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Not at all …

Once or twice …

Three or four times …

More than four times

6. During the PAST 7 DAYS, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

Not at all …

Once or twice …

Three or four times …

More than four times

7. Using the PAST 7 DAYS as an example, how would you rate your sleep quality overall?

Very good

Fairly good

Fairly bad

Very bad

8. Using the PAST 7 DAYS as an example, how often have you taken medicine to help you sleep? If taken, please specify the name of the medicine(s

Not at all

Once or twice per week

3 or 4 times per week

More than 4 times per week

9. Indicate the number of Allergenic foods equivalents you consumed on the PAST 7 DAYS.

DAY 1	DAY 2	DAY 3	DAY 4	DAY 5	DAY 6	DAY 7

10. If you do not consume the above allergic -contained beverages, what is the reason?

I am very allergic-sensitive.

Just do not like the taste.

The Allergic content is not healthy.

Other reason(s), please state below

11. During the PAST 7 DAYS, Do you smoke habitually? If yes, what is the average number of cigarettes that you smoke in a day?

Not at all

5 or less

6 – 10

More than 10

7.3. Allergic Rhinitis Symptom Questionnaire 3

1. How troublesome are your nasal allergy symptoms (stuffy nose, runny nose, sneezing, itchy nose)?
a) Not at all troublesome—You barely notice the symptoms
b) Mildly troublesome—They're easily tolerable
c) Moderately troublesome—Symptoms are hard to tolerate
d) Severely troublesome—Symptoms are so bad it's hard to function

If your patient answered C or D, he or she has moderate to severe allergic rhinitis.
If your patient answered A or B, then continue with the following questions:

2. Do your allergy symptoms interfere with sleep?

Yes

No

3. Do allergies interfere with your ability to go to school or work at your job?

Yes

No

4. Are your nasal allergies keeping you from doing the things you love, like activities or sports?

Yes

No

If a patient answered "YES" to any of these questions, he or she has moderate to severe allergic rhinitis, and may be an appropriate patient for Allergy. If a patient answered "NO" to all of these questions, then he or she has mild allergic rhinitis, and should consider an antihistamine to treat his or her symptoms.

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