Allergic and Non-allergic Rhinitis

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22.1 Allergic Rhinitis

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Key Points

- After reviewing this chapter, students should be able to:
- Identify the different variants of allergic rhinitis
- Understand the proposed pathogenesis of allergic rhinitis
- Mention the diagnostic tools used for allergic rhinitis
- Describe the different therapeutic options used in managing allergic rhinitis

22.1.1 Introduction

Prevalence of allergic rhinitis (AR) has been generally reported in a range from 10 to 30% with an approximate 7–10% belonging to non-allergic rhinitis (NAR) [1–4]. Among patients presenting with symptoms of allergic rhinitis, a prevalence for local allergic rhinitis (LAR) can be expected in 7–30% of patients [5–8]. There are a number of physiological, functional, and immunological relationships between the upper (nose, nasal cavity, paranasal sinuses, pharynx, and larynx) and lower (trachea, bronchial tubes, bronchioles, and lungs) respiratory tracts. Therefore, AR is frequently associated with asthma, which is found in 15–38% of patients with AR [9]. Furthermore, AR is considered as a risk factor for the development of asthma [9].

22.1.2 Definition

AR is an inflammatory, IgE-mediated disease characterized by rhinorrhea, sneezing, nasal congestion, and/or nasal itching. The condition is frequently accompanied by conjunctivitis; allergic rhinoconjunctivitis, and symptoms reverse spontaneously or after treatment. Other associated symptoms include itching of the palate, postnasal drip, and cough [10].

Clinical history is an important part of assessment of AR patients and should include family,

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environmental, and occupational information and questions regarding loss of smell (hyposmia or anosmia), snoring, sleep problems, postnasal drip or chronic cough, sedation, asthma, and conjunctivitis. A record of frequency, severity, duration, persistence or intermittence and seasonality of symptoms should be included as well. Color and lateralization of rhinorrhea, timing and lateralization of nasal obstruction, intake of other drugs, and concomitant symptoms must be explored. Assessment of quality of life should be evaluated including potential indoor/outdoor allergic triggers as well as effect of previous therapy [11].

Clinical history alone is not a good predictor to determine the clinical relevance of a certain allergen, being patients' own assessment of AR or clinical history alone inferior to the combination of skin prick test (SPT) and clinical history [12]. Standard questions to match the clinical symptoms with a specific allergen have a high specificity (>80%) but a decreased sensitivity (11–56%) when SPT is the only diagnostic method used [13].

AR is classified by many ways depending on clinical symptoms. It can be classified according to:

1. Temporal pattern of exposure to a triggering allergen as seasonal (e.g., pollens), perennial/year round (e.g., dust mites). Recently, the terms of intermittent (symptoms <4 days/week or for <4 consecutive weeks) and persistent (symptoms >4 days/week or for >4 consecutive weeks) AR are used instead [10].
2. Episodic (environmental from exposures not normally encountered in the patient's environment, e.g., visiting a home with pets).
3. Frequency of symptoms.
4. Severity of symptoms: mild (no disturbance of sleep; no impairment of daily activities, leisure, or sport; no impairment of school or work; symptoms present but are not troublesome), moderate or severe (disturbance of sleep; impairment of daily activities, leisure, or sport; impairment of school or work; troublesome symptoms) [10].

22.1.3 Pathophysiology

Numerous inflammatory cells infiltrate the nasal mucosa lining of AR patients once they are exposed to an allergen (most commonly airborne are house dust mite, cockroaches, animal dander, molds, and pollens). These cells include mast cells, CD4-positive T cells, B cells, macrophages, and eosinophils. Helper (Th2) CD4 cells release cytokines (mostly IL-4, IL-5, IL-13) that promote immunoglobulin (IgE) production by plasma cells. Cross-linking of IgE molecules on mast cells and basophils, on second allergen exposure, results in clinical symptoms of AR: itching, rhinorrhea, and mucous secretion.

Patients who have a symptomatic sensitization to aeroallergens seem to present with immediate allergic reactions that are not followed by the typical late-phase response. It has been observed that low levels of IL-5 confirmed by mRNA testing but not of IL-4 or IFN-γ on allergen stimulated peripheral blood mononuclear cells from SPT positive asymptomatic patients and a reduced IL-5 inhibition driven by Treg cells was described in symptomatic SPT positive patients but not in asymptomatic or non-atopic patients [14–17]. This laboratory findings correlate with the in vivo findings of a decreased intradermal delayed response to skin test, decreased eosinophils in nasal mucosa, and decreased blood eosinophil-to-lymphocyte ratio in asymptomatic SPT positive patients [18–20].

A higher number of asymptomatic patients is found in polysensitized patients [21]. Neither the extent of SPT patterns nor levels of specific IgE (sIgE) can make an efficient differentiation between symptomatic and asymptomatic patients [22–24]. Symptomatic AR patients have much higher levels of sIgE and skin reactivity, compared to asymptomatic ones, but these values do not lead to a significant difference [25, 26]. A family history of atopy has been related with a 15–30% increased possibility of presenting respiratory symptoms among patients presenting with positive SPT [21, 25, 27].

22.1.4 Diagnosis

The basic diagnosis of AR consists of a detailed medical history followed by confirmation of sensitization with in vivo SPT and/or in vitro sIgE test. If properly performed, they yield confirmatory evidence for the diagnosis of specific allergy in patients with AR. Nasal examination including endoscopic examination is essential to confirm the diagnosis of allergic rhinitis and rule out other pathologies. Anterior rhinoscopy often shows hypertrophied turbinates with pale or bluish mucosa. Allergic shiners (blue-gray discoloration below the eyelids) and a transverse nasal crease are typical in AR patients. As AR is a risk factor for the development of asthma, the clinical examination of patients should include screening for asthma [10, 11].

SPT is an essential test to confirm sensitization in IgE-mediated allergic disease like AR. A recent meta-analysis showed that this technique is reasonably accurate in identifying patients with suspected AR symptoms (sensitivity range of 68–100% and specificity range of 70–91%) [28]. There are some reported factors that may affect the accuracy of SPT like type of testing device, skill of the tester, skin reactivity, stability of test reagents and its potency [25, 28].

In vitro tests, on the other hand, assess antigen-specific IgE by testing the patient's serum. It is also a safer option if the patient is unable to do SPT, like in the case of treatment with antihistamine medication. However, these tests are expensive compared with skin testing.

A problem can be encountered, as not only non-allergic rhinitis patients can present with positive sensitization to allergens that mismatch with the clinical symptoms and sensitization, but also patients with local allergic rhinitis (LAR) can present with clinical symptoms and negative SPT [11, 29, 30].

The prevalence of asymptomatic sensitization in general population ranges from 1 to 5% for a single allergen and up to 8–30% when a panel of aeroallergens is used, with a range of 10–50% of individuals presenting with positive SPT being asymptomatic [24]. Asymptomatic sensitization is also denoted as latent and it is a risk factor for a later development of symptoms including asthma, with studies showing that 20–60% of these patients become symptomatic during a follow-up period of 2–24 years [24, 25].

In a national USA survey, including individuals between 6 and 59 years and testing with 10 allergens, up to 71.3% of patients with positive SPT were positive to more than one allergen [31].

Among possible causes of false-positive SPT are staff-related causes as violent technique, puncture vs. prick technique and pressure over the lancet among others, lancet characteristics, toxic reactions to the extract (allergen, impurities, additives) or concomitant physical urticarial [24, 32, 33]. The role of cross-reactivity with allergens that are clinically irrelevant (i.e., profilin) must also be considered [24, 34].

The rate of allergen sensitization increases with age in pediatric population reaching a peak before the age of 20–34 years, and this is opposite to the burden of rhinitis symptoms in sensitized patients that decrease with age [35–37].

22.1.5 Diagnostic Scenarios When Standard Testing Is Not Enough

1. **False-positive results in patients with only non-allergic rhinitis with or without asthma**:

   If discordance between SPT or sIgE and clinical history is found, further testing including the time-consuming and specialized staff required nasal provocation test (NPT) may be indicated. Over the last decade, an increasing number of studies on local allergic rhinitis has proven the need to reconsider patients classified as non-allergic rhinitis by means of NPT [29, 38–40].
2. **Polysensitized patients with a mix of real allergy and only sensitization**:

   As discussed above, both SPT and sIgE determination alone is only able to detect

allergen sensitization. A recent meta-analysis using NPT as gold standard has shown an estimate sensitivity and specificity for skin prick testing of 85% and 77%, respectively [28]. Although trained and specialized staff are required for the time-consuming NPT, it is the option of choice in allergic rhinitis to confirm clinical relevance to a certain allergen [29, 41, 42]. A recent EAACI position paper has extensively discussed the present recommended methodology for NPT performance [41]. Polysensitized patients who have a positive SPT results might be the result of a subclinical cross-sensitization. This issue has been shown by means of NPT to multiple allergens (Cupressus Vs cypress pollen), when only the monosensitized patients presented NPT as opposed to the 64 polysensitized [38]. Relevant clinical history is important in patients sensitized to panallergens such as profilin or polcalcin, as they can have no real clinical relevance on the patient symptoms [43].

3. **Patients with local allergic rhinitis diagnosed as non-allergic**:

Despite the time-consuming and specialized staff required, NPT needs to be implemented especially in patients where clinical symptoms and SPT or sIgE mismatch and this has been proved both for indoor and outdoor allergens: There is a large number of publications proving the underdiagnosed prevalence of LAR for indoor allergens as HDM and also for molds or cats that are considered as outdoor sensitization [5, 39, 40, 44–46]. Moreover, there is evidence as well for pollen-induced allergic patients [5, 38, 47, 48]. In a recent review on AR or NAR patients subjected to diagnostic local nasal provocation from 1946 to 2015, it was concluded that positive NPT was shown in 26.5% of patients previously considered non-allergic, and that AR defined by SPT or sIgE may lead to 13.7% of patients without accurate sensitization to allergens or non-allergic etiologies [49]. A pediatric study showed that LAR was present in 29.2% (7/24) of the patients presenting with negative SPT or sIgE, leading to the conclusion that LAR is substantially present in chronic, difficult-to-treat rhinitis patients presenting with negative SPT or sIgE [5].

22.1.6 Clinical Approach to Improve Diagnosis

22.1.6.1 Component-Resolved Diagnosis

The implementation of novel molecular diagnostic testing may improve the sensitivity and specificity of sIgE testing for AR and LAR patients, with in vitro results being progressively closer to the results of nasal and bronchial testing but still not being able to replace them as confirmatory test [26, 50]. Given the difficulties, and the time consumption in performing NPT, component-resolved diagnosis is gaining field on this topic [51]. This improvement may be important due to the increased regulatory demands in many countries, like the EU [52].

Component-resolved diagnosis (CRD) will continue gaining role on a proper decision regarding the need and composition of allergen immunotherapy (AIT) for the future, especially at the time that component-based AIT becomes a routine practice. This topic has been recently reviewed and there are World Allergy Organization (WAO) and European Allergy, Asthma and Clinical Immunology (EAACI) consensus document and guidelines [53–55]. It has been proved useful not only in identification of clinically relevant allergens but more important to determine the presence of panallergens such as profilin or polcalcins that can be a confounding factor for proper AIT leading therefore to significant changes in the decision to implement AIT [56, 57].

Recently, a sort of “atopic molecular march” has been described with an evolution from monomolecular to polymolecular sensitization determined as molecular spreading, and it has been described for children presenting with starting molecules Phl p1 or Der p1/p2/p23 [58, 59]. Furthermore, it has been hypothesized that early allergen immunoprophylaxis targeting these initiator molecules might prevent AR and asthma on preclinical stages [53].

22.1.6.2 Basophil Activation Test

Basophil activation test (BAT) measures basophil response to allergen cross-linking IgE on basophil granulocytes. In addition to clinical history, SPT, and specific IgE determination, BAT can be a part of the diagnostic evaluation of patients with IgE-mediated allergic response like AR [60]. Furthermore, BAT has proven useful in LAR, as well, as more sensitive and less time consuming alternative to detection of nasal specific IgE for HDM, being able to diagnose at least 50% of LAR to HDM with one study was a determinant factor to differentiate symptomatic and asymptomatic patients sensitized to HDM [22, 61].

22.1.7 Treatment

Treatment of allergic rhinitis should be tailored to each patient's condition taking into considerations several factors including age of the patient, nature and severity of symptoms, presence of comorbidities, and quality of life. Guideline-directed treatment plan (such as ARIA recommendations) proved to be effective [10]. Therapeutic measures for managing AR include education and environment control, pharmacotherapy, and immunotherapy:

Education and Environmental Control: It is essential to explain to the patient the nature of his disease, underlying etiology, and the need for extended medical care. Suggestions to avoid and minimize exposure to allergens and irritant factors such as tobacco smoke are discussed with the patients. Measure such as bedding and pillow covers, high efficiency vacuuming of carpets may be useful for house dust mite control. The potential role of pets should be highlighted [10, 11].

Pharmacotherapy: The most effective treatment of allergic rhinitis includes the use of modern generation **intranasal corticosteroid (INC) sprays** with minimum bioavailability and/or non-sedating **oral second generation H1 antihistamines**. While INC sprays are efficacious for all allergic rhinitis symptoms, oral H1 antihistamines relieve mostly rhinorrhea, sneezing, and itching but not nasal blockage. Intranasal corticosteroid (INC) sprays have a slow onset of action and it may take few days for their therapeutic effect to be felt by the patient. Hence, they may be initially prescribed in combination with oral antihistamine. Long-term use of INC sprays may be associated with minor side effects such as nasal dryness and bleeding and can be minimized by educating the patient the proper technique of application. **Oral decongestants** such as pseudoephedrine combined with an antihistamine and/or INC sprays are used in patients with nasal congestion. However, they can cause significant side effects such as irritability, tremors, insomnia, palpitations, and hypertension. They should be avoided in patients with heart disease, hypertension, glaucoma, urinary retention problems, and thyrotoxicosis. **Antileukotrienes** such as montelukast may be used in patients with asthma associated with allergic rhinitis. Short burst of **oral corticosteroids** may be used in patients with severe allergic flare-up. **Intranasal H1 antihistamines** such as azelastine are effective for controlling nasal symptoms. They need to be applied twice daily and their main side effect is inducing a bitter taste. **Nasal anticholinergics** such as ipratropium bromide 0.03% are effective in controlling rhinorrhea, but do not relief other nasal symptoms. They block muscarinic receptors, leading to a decrease in the parasympathetic function. They are usually used in combination with INC sprays or with an antihistamine. Minor side effects include headache, epistaxis, and nasal dryness. They should be used with caution in patients with narrow-angle glaucoma and in prostatic hypertrophy. **Intranasal sodium cromoglycate** is a mast cell stabilizer and was shown to be effective in the prevention and treatment of allergic rhinitis but is less effective than INC sprays and oral antihistamines. They require application four times daily and ocular formulas are helpful in treating allergic conjunctivitis [10, 11].

Two to four weeks after use of the initial therapy, patients should be reevaluated for the efficacy of this treatment in controlling their symptoms and for the presence of side effects. Based upon this evaluation, maintenance treatment is designed. During follow-up visits, and based upon the patients' response, treatment may be either maintained or stepped down. Modification of the maintenance treatment will

also be required during allergic flare ups or respiratory tract infections [10, 11].

Immunotherapy: AIT is a highly effective treatment modality for patients with AR. Unlike other pharmacological treatments, AIT is considered an immune modulating therapy with long-lasting efficacy in the management of AR [62]. It has a well-established efficacy for inhalant allergen sensitization, including patients presenting with LAR [29].

AIT prescription should be based on the clinically relevant allergens and not merely on potentially irrelevant SPT or sIgE sensitization [63]. In polysensitized patients, single-allergen products are recommended when an allergen is clearly responsible for the main symptoms load. The use of AIT for 2 products as single-AIT formulations in parallel is the preferred form of administration when 2 allergens are responsible for the main impact [51, 64]. The fine-tuning of the clinically relevant allergens in polysensitized patients can therefore increase the implementation of these recommendations.

Take Home Messages

- Allergic rhinitis is a common disease with a prevalence as high as 30%.
- It is the commonest chronic rhinitis encountered and has a significant impact on the patients' health, quality of life, and work performance.
- The diagnosis of AR is attained mainly through a detailed medical history. In vivo SPT and/or in vitro sIgE test confirm the diagnosis.
- Therapeutic measures for managing AR include education and environment control, pharmacotherapy, and immunotherapy:
- Treatment should be tailored to each patient's condition taking into considerations age of the patient, nature and severity of symptoms, presence of comorbidities, and quality of life. Guideline-directed treatment plan (such as ARIA recommendations) proved to be effective.

22.2 Non-allergic Rhinitis

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Key Points

- After reviewing this chapter, students should be able to:
- Identify the different variants of non-allergic rhinitis
- Understand the proposed pathogenesis of the different variants of non-allergic rhinitis
- Describe the therapeutic options used in managing the different variants of non-allergic rhinitis

Non-allergic rhinitis includes a heterogeneous group of patients with rhinitis resulting from different pathophysiologic mechanisms other than allergy or infection.

Prevalence: Although the prevalence of allergic rhinitis is well investigated, the prevalence of non-allergic rhinitis is less known. Non-allergic rhinitis tends to occur more in adults, with the typical age of presentation between 30 and 60 years. It is suggested that it is responsible for 50% of cases of chronic rhinitis. A Danish study found that 25% of rhinitis was non-allergic. The disease was found to be more prevalent in women. In the United States, non-allergic rhinitis is estimated to affect around 19 million people [65–68].

The variants of non-allergic rhinitis include:

22.2.1 Idiopathic (Vasomotor) Rhinitis

This is the most frequent form of non-allergic rhinitis encountered. All chronic rhinitis patients without an obvious underlying etiology are grouped together under this variant. Accordingly, this variant is a diagnosis of exclusion. To make this diagnosis, all other causes of non-allergic rhinitis have to be excluded. The patients suffer from perennial nasal congestion, rhinorrhea, and/

or sneezing with no identifiable etiology. It is non-IgE-mediated, non-infectious, and not associated with nasal eosinophilia. The exact pathophysiology of this variant remains to be poorly understood. It is thought to be due to autonomic imbalance with parasympathetic hyperactivity and sympathetic hypoactivity. Parasympathetic hyperactivity results in an increase of acetylcholine and vasoactive intestinal peptide (VIP). Sympathetic hypoactivity results in a decrease of norepinephrine and neuropeptide Y. This imbalance results in excessive nasal secretions and nasal congestion. Other hypotheses suggest hyperactivity of C-fiber, or the release of excessive amounts of neuropeptides (substance P and neurokinins) that promote nasal congestion and nasal secretion production. Topical steroids are initially given for a period of 6 weeks. Ipratropium bromide 0.03% is effective against anterior rhinorrhea, but does not affect other nasal symptoms. This molecule blocks muscarinic receptors, leading to a decrease in the parasympathetic function. Minor side effects include headache, epistaxis, and nasal dryness.

If symptoms persist, intranasal capsaicin has been shown to be an effective treatment (Fig. 22.1) [69–72].

22.2.2 Drug-Induced Rhinitis

1. **Rhinitis Medicamentosa**: This is not an infrequently encountered condition where an individual overuses local vasoconstrictor drops or sprays (oxymetazoline, xylometazoline, naphazoline, etc.) to maintain his nasal airway patent. Initially, the instillation of the vasoconstrictor drops/spray, which is alpha adrenergic agonist, shrinks and decongests the nasal mucosa by constricting the blood vessels. However, rebound congestion occurs after a few hours resulting in nasal blockage and driving the patient to further instill more drops or spray. Over time, increasing amounts of the local medication will be needed to decongest the nasal mucosa and relieve the blockage and the patient becomes addicted to its use. As a result of this, the nasal mucosal blood vessels lose the alpha adrenergic tone and the normal nasal cycle is suppressed. The cilia are decreased in number and squamous metaplasia occurs.

Diagnosis: The patient gives a typical history of nasal blockage and congestion that is only relieved by the instillation of vasoconstrictor drops/sprays. This problem may have been running for weeks, months, and even years. On examination, the nasal mucosa is usually dry, reddish, and thinned out. Occasionally, an underlying primary cause of nasal obstruction, such as a significantly deviated nasal septum, is detected.

Treatment: It is critical to explain to the patient the cause of the problem and importance of getting off the use of these local vasoconstrictor agents. Short burst of systemic steroids, topical intranasal steroids, and systemic decongestant drugs may be prescribed

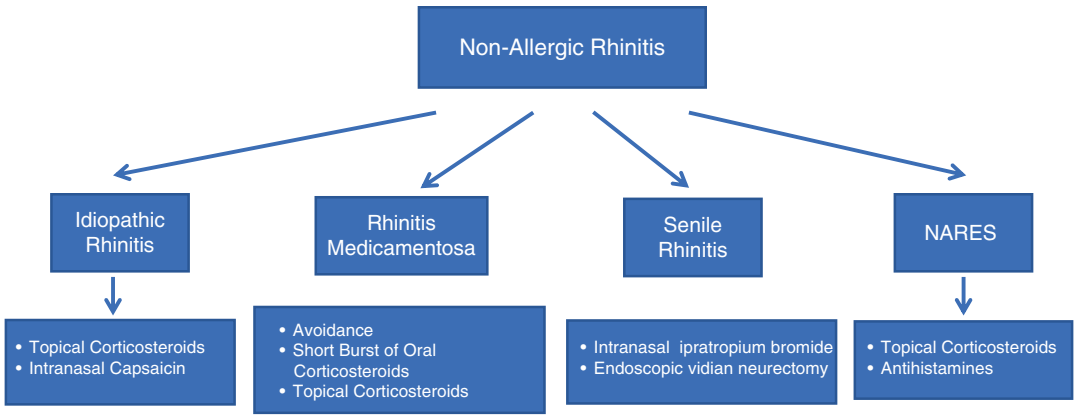

Fig. 22.1 Main therapeutic measures in different forms of non-allergic rhinitis

to help to wean the patient off the local vasoconstrictors. In the subset of patients with an obvious cause of nasal blockage, such as a significantly deviated nasal septum, a septoplasty may be considered but only several weeks after the patient has stopped using the local vasoconstrictors. Similarly, radiofrequency reduction of the inferior turbinate or an inferior turbinoplasty procedure may be considered in select cases (Fig. 22.1) [66, 67, 73].

2. **Cocaine Abuse**
3. **Aspirin and NSAIDs Intolerance**: This may result in an intense eosinophilic rhinitis with the patient suffering from profuse watery rhinorrhea. In the variant of Aspirin Exacerbated Respiratory Disease (AERD), the patient has eosinophilic rhinosinusitis with nasal polyposis, asthma, and aspirin intolerance. The patient's symptoms and asthma are worsened by aspirin and/or NSAIDs intake. The disease results from a defect in the arachidonic acid metabolism with inhibition of the cyclooxygenase pathway and preferential increase in the lipoxygenase pathway with excessive leukotriene production. Avoidance of these drugs is important in this group of patients [65–67].
4. **Others**: Antihypertension medications such as ACE inhibitors, beta blockers, methyldopa, reserpine, guanethidine, phentolamine, chlorpromazine, oral contraceptives, and neostigmine (used in treatment of myasthenia gravis) can produce nasal obstruction [66, 67].

22.2.3 Occupational

This arises in response to airborne particles in the work environment. Occupational rhinitis can result from allergic or irritant responses, which elicit eosinophilic or neutrophilic inflammation. Numerous chemical agents have been identified. These include high molecular weight irritants such as grain dust, flour, fish and seafood proteins, latex, and low molecular weight irritants such as isocyanates, aldehydes, ninhydrin, chlorine, wood dust, and many others. Complete avoidance is the most essential measure in managing this variant of rhinitis [74].

22.2.4 Hormonal

- **Rhinitis of Pregnancy**: During pregnancy, the elevated levels of estrogen may induce an inflammatory process in the nasal mucosa causing bothersome nasal obstruction. Saline irrigations may help in alleviating this condition, which resolves spontaneously after delivery [75].
- **Honeymoon rhinitis**, **epistaxis of vicarious menstruation**, and rhinitis associated with the use of contraceptive pills all are associated with hormonal pathophysiology [75].
- **Hypothyroidism and acromegaly may be associated with inflammatory changes in the nasal mucosa** [66, 67].

22.2.5 Non-allergic Rhinitis with Eosinophilia Syndrome (NARES)

This was first described by Jacobs et al. in 1981. In this rhinitis variant, patients present with perennial symptoms of paroxysmal sneezing, profuse watery rhinorrhea, and itching in the nasopharynx. Their nasal smears show profound eosinophilia while their allergy testing (skin prick test, total and specific IgE) is negative. These patients respond well to topical corticosteroids [76–78].

22.2.6 Senile Rhinitis

Typically, this occurs in the elderly where the patient suffers primarily from persistent watery rhinorrhea with clear watery drops trickling down from the nose. Intranasal ipratropium bromide given up to six times daily is reported to control the condition. Its parasympatholytic effect decreases submucosal gland secretion. Ipratropium bromide should be used with caution in patients with narrow-angle glaucoma, prostatic hypertrophy, or bladder neck obstruction. Injection of botulinum toxin type A (BTA) into the nasal cavities is another suggested treatment modality. Endoscopic vidian neurectomy has been reintroduced and seems to have excellent

results, although the treatment is not without potential serious side effects (Fig. 22.1) [79–81].

22.2.7 Gustatory Rhinitis

This is a condition characterized by sudden onset of excessive bilateral watery rhinorrhea occurring immediately after the ingestion of foods (often, hot and spicy). This is usually not associated with sneezing or itching or any other symptoms. Intranasal ipratropium bromide is usually an effective treatment [82].

22.2.8 Atrophic Rhinitis

This could be either primary or secondary. In **primary atrophic rhinitis**, there is atrophy of the nasal mucosa and underlying bone, with the nasal cavity becoming wide and containing foul-smelling crusts. Typically, the condition occurs in young females. Interestingly, the patients complain of nasal obstruction, cacosmia, and possibly mild epistaxis with separation of the crusts. It has been suggested to result from Klebsiella ozaenae infection, although its role as a primary pathogen is not confirmed. **Secondary atrophic rhinitis** can result from nasal granulomas, radiation, and trauma including surgical resection [66, 67].

Take Home Messages

- Non-allergic rhinitis includes a heterogeneous group of patients.
- It roughly constitutes 50% of patients with chronic rhinitis.
- It includes drug-induced rhinitis, hormonal rhinitis, senile rhinitis, occupational rhinitis, gustatory rhinitis, NARES, atrophic rhinitis, and idiopathic rhinitis.
- Treatment plan of non-allergic rhinitis varies from one variant to another.

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