REVIEW

Gustatory rhinitis

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Purpose of review
This review aims to characterize gustatory rhinitis using recent advances in pathophysiology and novel surgical and medical management strategies.

Recent findings
A significant amount of research has recently focused on the role of capsaicin and its receptors (TRPV1 and VR1), which can be found on sensory c-fibers in human nasal mucosa and play a critical role in the development of nasal hyperresponsiveness to environmental factors. Blocking the nasal sensory nerve stimulation (via the use of capsaicin desensitization) may control nasal hyperresponsiveness and therefore prevent the induction of rhinitis symptoms. Vidian neurectomy.

Summary
Gustatory rhinitis is a conspicuous type of food-associated rhinorrhea, which can occasionally be associated with significant quality-of-life impairment. It results from an abnormal gustatory reflex associated with a hyperactive, nonadrenergic, noncholinergic, or peptidergic neural system. The use of nasal ipratropium bromide may be effective, if avoidance is not possible or successful. We have had excellent results with the use of intranasal capsaicin or endoscopic vidian neurectomy (including removal of 4–5 mm of the nerve between pterygopalatine fossa and the sphenoid floor) in patients with nonallergic rhinitis, and these could potentially be used as a last resort in patients with intractable gustatory rhinitis.

Keywords
capsaicin, gustatory rhinitis, gustatory rhinorrhea, ipratropium bromide, rhinitis, rhinorrhea, vidian neurectomy

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INTRODUCTION

Gustatory rhinitis belongs to the nonallergic, noninflammatory group of rhinitis [1,2▪]. It is a syndrome of food-induced nasal hypersecretion, characterized by the acute onset of copious watery or, occasionally, mucoid rhinorrhea, occurring immediately after the ingestion of certain foods (often, hot and spicy) [1,3–5]. Characteristically, excessive rhinorrhea occurs exclusively during food ingestion and begins within a few minutes of eating the involved food, usually with no associated sneezing, pruritus, congestion, or facial pain [1,6].

Hot and spicy food is most often associated with gustatory rhinorrhea as reported by 49–73% of patients [3,5]. More specifically, hot chilli peppers, red cayenne, tabasco sauce, red pepper, horseradish, black pepper, hot and sour soup, onion, chilli, vinegar, and mustard have been often implicated in gustatory rhinitis [3,5].

Capsaicin (8-methyl-*n*-vanillyl-6-nomamide) is the pungent agent in chili peppers, red cayenne, tabasco sauce, red pepper, horseradish, and black pepper. It stimulates afferent sensory nerves in oral and oropharyngeal mucosa, thus triggering gustatory rhinorrhea [3,7,8,9▪].

Patients with gustatory rhinitis do not have any chemosensory (taste and olfaction) disturbances [7]. There are predilection for either sex and no association with atopy [3,5]. Gustatory rhinitis is observed in all age groups, with peak prevalence between 20 and 60 years [5]. The symptoms sometimes become worse with age [7].

Gustatory rhinitis is classified into four subcategories: idiopathic, posttraumatic, postsurgical, and gustatory rhinorrhea associated with cranial nerve neuropathy [6]. Idiopathic gustatory rhinitis is always bilateral, whereas the other types may be one-sided or bilateral [6].

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KEY POINTS

- Nonadrenergic, noncholinergic, or peptidergic neural system including capsaicin and its receptor (TRPV1) play a significant role in nonallergic as well as in gustatory rhinitis.
- Gustatory rhinitis should initially be managed by avoidance.
- If avoidance fails, the use of ipratropium nasal spray can be helpful for the symptoms of gustatory rhinitis.
- We have had excellent results with endoscopic vidian neurectomy in patients with intractable nonallergic rhinitis.

Boddie *et al.* [10] was the first to describe postsurgical gustatory rhinorrhea in 1976, as a complication of radical parotidectomy. Idiopathic gustatory rhinitis was initially studied and described in detail by Raphael *et al.* [3] in 1989.

Gustatory rhinitis is not uncommon. However, in most cases, it is well tolerated and not associated with major quality-of-life impairment, so the vast majority of patients do not seek treatment. Almost half of the patients describing symptoms of gustatory rhinitis (46%) state that they are never bothered about it, whereas 65% chose not to avoid the causative food [5]. However, it can be socially embarrassing and occasionally troublesome. About 2% of the patients claim to be 'very bothered' and 1% 'extremely bothered' by their symptoms [5].

CAUSE

Gustatory rhinitis forms part of the group of abnormal gustatory reflexes. This group includes Frey's syndrome or gustatory sweating, 'crocodile tear syndrome' or gustatory lacrimation, and gustatory otorrhoea. These are congenital or acquired (posttraumatic or postsurgical) conditions, in which a sensory gustatory input is followed by a normal physiological salivary reflex as well as an abnormally strong sensory-parasympathetic reflex involving parasympathetic innervation (of the nasal mucosa – in the case of gustatory rhinitis) [6,7,11–13].

The cause of idiopathic gustatory rhinorrhea is unknown. However, postsurgical, posttraumatic, and gustatory rhinorrhea associated with cranial nerve neuropathy (as seen in leprosy) are all thought to result from concurrent interruption of salivary gland and secretory nasal mucosa fibers and consequent abnormal re-innervation [11,13–21].

Posttraumatic gustatory rhinorrhea occurs as a rare, late complication of skull base trauma [11–13], whereas gustatory rhinorrhea has also been described as a manifestation of cranial nerve neuropathy in leprosy [19].

Postsurgical gustatory rhinorrhea most commonly occurs after total parotidectomy [10,14,16]; however, it has also been described after hemimaxillectomy [18], total maxillectomy [15,20], septoplasty [17], and oral surgery (difficult dental extraction) [21].

PATHOPHYSIOLOGY

The exact pathophysiology of gustatory rhinitis is still under debate. Recent studies suggest that it is a purely neurogenic event, with no immunological basis, associated with overstimulation of the parasympathetic system [22–24].

Ingestion of the offending food causes stimulation of trigeminal sensory nerve endings located in the upper part of the aerodigestive tract. This is via the activation of postganglionic, cholinergic, muscarinic, parasympathetic fibers, as it has been shown that gustatory rhinorrhea can be significantly reduced by the application of topical atropine [3,4,6,24].

The neural regulation of the upper airways includes complex interactions between afferent sensory fibers and efferent sympathetic and parasympathetic nerves that, along with other mechanisms, regulate epithelial, vascular, and glandular processes in the nasal mucosa [22,25–27].

However, a hyperactive, nonadrenergic, noncholinergic (NANC), or peptidergic neural system is considered today as the most likely underlying pathophysiologic factor in idiopathic rhinitis [2▪,22,25]. Capsaicin produces a distinct taste sensation via the activation of taste receptor cells (TRCs) and on transients receptor potential vanilloid receptors subtype 1 (TRPV1) [8]. TRPV1, the capsaicin receptor, is a nociceptive transducer, which exists in neuronal as well as nonneuronal cells – such as nasal mucosa and oral epithelium [28]. Although the precise role of TRPV1 in goblet cells and submucosal glands is unclear, it is possible that capsaicin may directly influence secretory function through direct activation of TRPV1 on goblet and submucosal glands of the human upper airways [28].

Sensory nerve fibers, including C and Aδ-fibers, have also TRPV1 receptors in trigeminal nerve endings in the mouth and oropharynx which can be stimulated and/or upregulated by capsaicin (producing in this way a burning sensation) [8,9▪,22]. The unmyelinated, peptidergic, sensory

C-fibers or pain receptors are particularly sensitive to capsaicin [25]. This sensory neural stimulation of C-fibers may produce effects either through an orthodromic, central neural reflex, associated with efferent, predominantly parasympathetic, neurotransmission, or via an antidromic, local release of multiple neuropeptides from sensory neurons [22,23▪,25,29,30]. Through the orthodromic reflex, the efferent, predominantly parasympathetic signal, results in increased secretion and vasodilatation [25]. The local release of neuropeptides [including substance P(SP) and calcitonin gene-related peptide (CGRP)] from sensory nerves in the nasal mucosa (antidromic reflex) also results in increased vasodilatation, vascular permeability, and secretion [25]. Sensory nerve endings have a limited amount of neuropeptides available. Following continuous exposure to capsaicin, the available amount of secretory peptides decreases and desensitization occurs. Desensitization of sensory nerve endings could be characteristic of those who add capsaicin to their everyday diet without experiencing any discomfort [25]. Such desensitization forms also the basis of the therapeutic use of capsaicin in nonallergic rhinitis. However, although this neurogenic pathway explains the reactions to capsaicin, it fails to account for patients reporting similar symptoms in the presence of foods that do not contain capsaicin [6].

After the stimulation of afferent sensory nerves, a neural reflex arc is initiated that stimulates parasympathetic efferent nerves supplying the nasal mucosa, especially submucosal glands, resulting in rhinorrhea [3]. As it is proven that there are tight connections between trigeminal sensory nerve fibers and postganglionic parasympathetic neurons present in the sphenopalatine ganglion, it is most likely that sensory nerve stimulation is associated with a parasympathetic reflex and activation of postganglionic, cholinergic, muscarinic, parasympathetic fibers, known to be sensitive to atropine [3,4,6,31]. Disruption of this reflex following trauma, surgery, or inflammation may form the basis for the abnormal gustatory reflex associated with rhinorrhea.

Interaction between sympathetic and parasympathetic nerves may also be involved in the pathophysiological mechanisms of gustatory rhinitis [6,27]. Strong sympathetic nerves stimulation induces the release of both noradrenaline and neuropeptide Y (NPY) [26,32–34]. NPY is a potent vasoconstrictor and neuromodulator of both sensory and parasympathetic nerves activity [26,33–35]. A marked reduction of NPY inhibitory activity on parasympathetic activity could be the mechanism underlying gustatory rhinitis [6,26,27,32,33,35].

DIAGNOSIS

The diagnosis of gustatory rhinitis is based on history and exclusion of other types of chronic rhinitis. The step-by-step exclusion of other types of rhinitis should follow the guidelines established for the diagnosis of idiopathic rhinitis [2▪,4,25]. A food-reaction questionnaire, assessing implicated substances and avoidance behavior, as suggested by Weibel, should be used [5].

There are no generally accepted objective tests for the diagnosis of gustatory rhinitis: Franceschini *et al.* [11] stimulated the tip of the patients tongue with lemon juice and confirmed the initiation of rhinorrhea. It is important to use skin prick tests in order to exclude an allergic reaction. Food challenges are obviously the cornerstone of diagnosis [3].

DIFFERENTIAL DIAGNOSIS

There is a spectrum of food-intake associated disorders (immunologic and nonimmunologic), including food allergy/hypersensitivity and food intolerance [1,24,29]. Rhinitis can be caused by true IgE-mediated food allergy, but it is almost never an isolated manifestation of food allergy [1,3,4,24].

Drug-induced rhinitis has a long list of offending medications. The symptoms include primarily nasal blockage, potentially associated with watery nasal secretions, postnasal drip, and sneezing [2▪,25, 29,36]. Aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) can be associated (in addition to asthma attacks in sensitive individuals) with predominantly watery rhinorrhea [6,29,36,37].

One of the most important underlying mechanisms in both allergic and idiopathic rhinitis is nonspecific nasal hyperreactivity. It is defined as an increased nasal response to a normal stimulus resulting in sneezing, nasal congestion, and nasal hypersecretion, in various combinations [29]. It occurs after many different types of nonspecific stimulation including consumption of hot drinks, hot and spicy foods, and alcoholic beverages. If food is the only trigger and rhinorrhea the only manifestation, gustatory rhinitis should be high in the differential diagnosis; however, in the majority of patients, there is more than one trigger and more than one nasal symptom [4,29].

The type of rhinorrhea seen in allergic rhinitis patients is typically clear, profuse, and watery, so it can be confused with gustatory rhinorrhea [3,6]. However, gustatory rhinorrhea is present only during food ingestion, there is no associated conjuctival, nasal, or oropharyngeal pruritus, nasal

congestion, or sneezing all of which are characteristics of allergic rhinitis [3]. Skin tests are negative in patients with gustatory rhinitis [3].

Increased cholinergic hyperreactivity resulting in profuse rhinorrhea has been documented in patients with recent upper respiratory tract infections (URTIs) [4].

Senile rhinitis or rhinitis of the elderly is one of the best examples of nasal hyperresponsiveness of the parasympathetic system and can mimic gustatory rhinorrhea [1,4,25,29]. However, in contrast to gustatory rhinorrhea, which occurs exclusively during meals, senile rhinitis occurs throughout the day, although it may be aggravated by eating [1,4,29,38].

Other abnormal gustatory reflex syndromes can appear as a complication of either trauma or surgery, including auriculotemporal nerve syndrome (Frey's syndrome) [39–41].

TREATMENT

The initial treatment option should (obviously) always be the avoidance of the offending substance.

However, in the case where avoidance is impractical or insufficient, intranasal anticholinergics (usually ipratropium bromide) are the drugs of first choice [3,6,29,38]. Intranasal atropine and intranasal oxitropium bromide are also proven to be effective; however, they have more side-effects and their use is not routinely recommended. [2▪,3,4,29,42]. Ipratropium should be used prophylactically, just prior to the intake of the offending food [4,7,38,42]. The combination of ipratropium bromide and an intranasal corticosteroid is more effective than either drug alone [4].

Surgical therapy in the form of vidian nerve neurectomy or modified vidian neurectomy – the resection of the posterior nasal nerve (PNN) – is rarely indicated, but can be used as a last option [6,43,44]. Golding-Wood introduced vidian neurectomy in 1960 for the management of intractable rhinitis [44]. However, following its original popularity, it was subsequently almost completely abandoned, because of its lack of long-term effectiveness and it frequent unpleasant side-effects [44,45▪]. Better understanding of the anatomy of the vidian nerve (resulting from three dimensional imaging) and the superior visualization offered by the endoscopes has renewed interest in the technique of vidian neurectomy. We feel that the previously described cases of failure or complications were often the result of inadequate visualization of the vidian nerve and incomplete resection. We now have significant experience in endoscopic vidian nerve (removing 4–5 mm of vidian nerve coursing from the pterygopalatine fossa toward the floor of the sphenoid sinus), and together with others, feel it is a well tolerated, effective technique with long lasting, potentially up to 7 years [45▪,46–48] (see Figs 1–3).

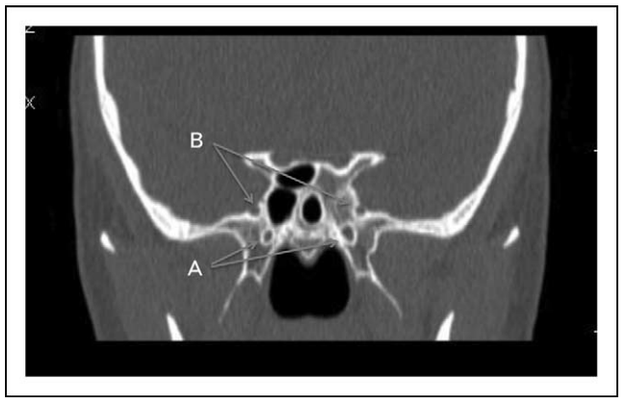

FIGURE 1. Coronal computed tomography scan demonstrating vidian nerve (a) and V2 (b) in the sphenoid sinus.

We have also accumulated significant experience in the use of capsaicin for the use of nonallergic rhinitis. Pathophysiology of gustatory rhinorrhea suggests that capsiacin may be a potential treatment option for intractable gustatory rhinorrhea [6]. Topical capsaicin is efficient in producing long-term reduction of symptoms in nonallergic, persistent, rhinitis patients [9▪,25]. It reduces nasal hyperreactivity through the desensitization and occasionally degeneration of C-fibers [9▪,22,25].

Botulinum toxin type A (BTX-A) is among the treatment choices that are increasingly being used in the symptomatic treatment of nasal

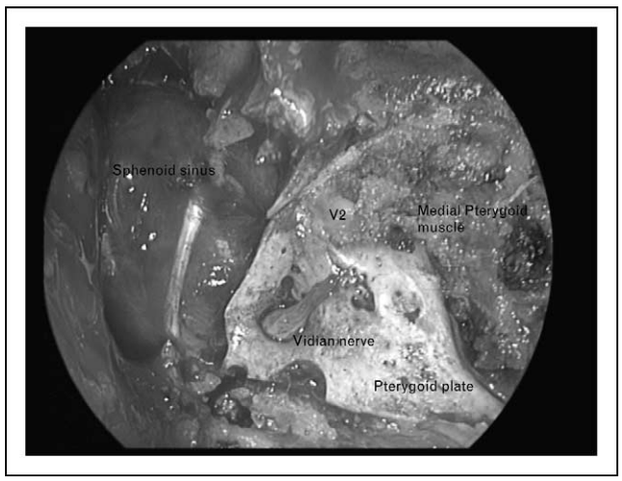

FIGURE 2. Endoscopic view of the vidian nerve and the V2 (maxillary) nerve in a patient with a malignant tumor of the pterygopalatine fossa.

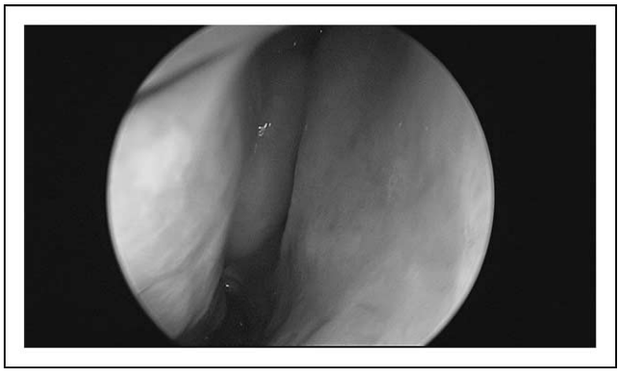

FIGURE 3. Postoperative endoscopic view of the nasal mucosa of a patient following vidian neurectomy.

hypersecretion [49]. BTX-A blocks the acetylcholine release in neuromuscular joint and cholinergic autonomic nerves by binding peripheral cholinergic terminals [49]. Shaari *et al.* [50] in 1995 first suggested that BTX-A can be effective on rhinorrhea. So far, BTX-A in the treatment of rhinorrhea has been applied via injections in the middle and inferior turbinate, septum injections and through application in the nose with a sponge [49,51,52]. It has been shown to be effective in the short term (8–12 weeks), with no significant adverse effects [51,52] and represents a promising treatment option for gustatory rhinitis, which could be explored in future clinical trials.

CONCLUSION

Diagnosis of gustatory rhinorrhea depends on clinical history, including focused questions on allergy, food ingestion, and exclusion of other types of nonallergic rhinitis. Provocation testing as well as skin prick test may be useful to confirm the diagnosis. Management should be based on avoidance of offending substances; if that is impractical or not effective, the use of nasal ipratropium, capsaicin desensitization, or (as a last resort) vidian neurectomy are potential therapeutic options. The use of botilinum toxin injections needs to be further explored in future studies.

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Conflicts of interest

None of the authors has any conflicts of interest to declare.

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Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 000–000).

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