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Anatomical and Clinical Correlations Between Nasal Pathologies and Ophthalmologic Symptoms: A Systematic Review

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Abstract

Background: Close anatomical, neural, and vascular relationships exist between the nasal cavity/paranasal sinuses and the orbit, leading to frequent ophthalmologic symptoms in patients with primary nasal pathologies.

Objectives: To systematically review the anatomical basis and clinical correlations of ophthalmologic manifestations arising from nasal and paranasal sinus diseases, and to evaluate emerging diagnostic and therapeutic implications, including sphenopalatine ganglion stimulation and causes of nasolacrimal duct obstruction.

Methods: A systematic literature search was conducted in PubMed, Scopus, Web of Science, and Embase from January 2000 to June 2026. Search terms included combinations of "nasolacrimal duct", "pterygopalatine ganglion", "sphenopalatine ganglion stimulation", "allergic rhinoconjunctivitis", "nasolacrimal duct obstruction", "cavernous sinus thrombosis", and "herpes zoster ophthalmicus". Inclusion criteria: original studies, anatomical reviews, clinical trials, and meta-analyses. After screening 1,312 records, 92 studies were included following PRISMA guidelines.

Results: Key pathways include nasolacrimal duct continuity (with nasal septal deviation, turbinate hypertrophy, and chronic inflammation as major causes of obstruction), trigeminal and pterygopalatine ganglion neural connections, and valveless venous drainage. Hutchinson's sign strongly predicts ocular involvement in herpes zoster ophthalmicus. Stimulation or blocking of the sphenopalatine ganglion has proven to significantly help with the nasal and eye symptoms of allergic rhinoconjunctivitis.

Our findings support the connection between the anatomy and potential treatment options for the sphenopalatine ganglion and include strong evidence for using multiple disciplines in treating individuals with allergic rhinitis, which is critical.

Keywords: Nasolacrimal Duct Obstruction, Pterygopalatine Ganglion Stimulation, Allergic Rhinoconjunctivitis, Cavernous Sinus Thrombosis, Systematic Review

Introduction

The intricate anatomical and functional relationships between the nasal cavity, paranasal sinuses, and the orbit have long been recognized in clinical practice. Patients presenting with primary nasal pathologies such as allergic rhinitis, chronic rhinosinusitis, nasal septal deviation, or acute sinus infections frequently report concurrent ophthalmologic complaints, including epiphora, ocular itching, redness, excessive tearing, periorbital pain, and in severe cases, visual disturbances or proptosis. These overlapping symptoms are not coincidental but arise from well-defined anatomical pathways that allow direct communication between the two regions.

Tears drain from the eye (or eyes) through a structure called the nasolacrimal duct, which opens into the nasal cavity. The trigeminal nerve and the pterygopalatine (sphenopalatine) ganglion, which innervate both the nose and eye, both have a close relationship due to their common parasympathetic areas of distribution. In addition, the venous blood vessels of the midface are valveless, potentially providing pathways for infectious materials to travel to the cavernous sinus. As such, infectious, inflammatory, or allergic processes in the nasal cavity can as quickly affect the eyes, and vice versa.

Despite the clinical significance of these connections, both medical professionals and patients often do not discover or treat problems quickly or effectively due to the fact that rhinology and ophthalmology are separate specialties. As a result, knowing the direct anatomical relationship between these structures is critical to proper diagnosis and treatment in a timely manner. With ongoing advancements in imaging technologies, endoscopic methodologies, and neuromodulation, the need for a current

evidence-based review of the literature remains essential. The aim of this systematic review was threefold. One goal is to map clinically significant anatomical pathways that link nasal pathologies with ocular symptoms. Another goal was to explore in depth nasal-related causes and risk factors for nasolacrimal duct obstruction. The final goal was to evaluate and review the potential therapeutic use of sphenopalatine ganglion stimulation for conditions that have both nasal and ocular manifestations. To provide clinicians with a complete understanding of these interrelated conditions, current evidence from anatomical, clinical, and interventional studies was used to develop a complete framework.

Methods

Search Strategy and Information Sources

A comprehensive systematic search of the literature was conducted in four major electronic databases: PubMed/MEDLINE, Scopus, Web of Science, and Embase. The search identified articles published between January 1, 2000 and June 17, 2026. The search strategy combined Medical Subject Headings (MeSH) and free-text keywords using Boolean operators. Core search strings included: ("nasolacrimal duct" OR "nasolacrimal duct obstruction" OR NLDO) AND ("nasal pathology" OR "septal deviation" OR "turbinate hypertrophy" OR "allergic rhinitis"); ("pterygopalatine ganglion" OR "sphenopalatine ganglion" OR SPG) AND ("stimulation" OR "block" OR "neuromodulation") AND ("allergic rhinitis" OR "rhinoconjunctivitis" OR "ocular symptoms" OR "lacrimal"); and ("cavernous sinus thrombosis" OR "Hutchinson sign") AND ("sinusitis" OR "nasal infection"). Additional terms such as "anatomical correlation", "orbital complication", and "trigeminal autonomic cephalalgia" were incorporated. The reference lists of all included articles and relevant review papers were manually screened to identify any additional eligible studies.

Eligibility Criteria

Studies were included if they met the following criteria: (a) original research articles, anatomical studies, clinical trials, systematic reviews, or meta-analyses; (b) focused on anatomical relationships, clinical correlations, diagnostic imaging, or therapeutic interventions linking nasal pathologies to ophthalmologic symptoms; (c) published in English; and (d) involved human subjects. Exclusion criteria were: single case reports or case series with fewer than five patients, conference abstracts, letters to the editor, animal or *in vitro* studies, and articles without accessible full text.

Study Selection and Data Extraction

Two independent reviewers screened titles and abstracts, followed by full-text assessment of potentially eligible articles. Disagreements were resolved through discussion and consensus. Study design, study population, key anatomical findings, and clinical outcome data were all collected through a standardized extraction form. Also recorded were any relevant information regarding SPG stimulation treatment given to the patient and any complications resulting from the treatment. Thematic synthesis was performed due to heterogeneity in study designs and outcomes.

Quality Assessment

The methodological quality of included observational studies was assessed using the Newcastle-Ottawa Scale. Randomized controlled trials were evaluated with the Cochrane Risk of Bias tool. Systematic reviews and meta-analyses were appraised using the AMSTAR 2 checklist. No studies were excluded based solely on quality scores; instead, quality was considered during data interpretation.

PRISMA Flow and Study Characteristics

The initial search yielded 1,312 records. After removal of 387 duplicates, 925 articles were screened by title and abstract. Full-text review was conducted on 248 articles, of which 92 met the final inclusion criteria. These comprised anatomical studies, cross-sectional imaging research, clinical cohort studies, randomized trials on SPG interventions, and relevant systematic reviews.

Results

1. Physical Continuity via the Nasolacrimal Duct and Nasal Causes of Nasolacrimal Duct Obstruction

The nasolacrimal drainage system begins at the lacrimal puncta and canaliculi, passes through the lacrimal sac, and continues as the nasolacrimal duct, which opens into the inferior meatus beneath the inferior turbinate at the valve of Hasner. This anatomical continuity allows more than 80% of tear volume to drain directly into the nasal cavity [1, 2]. As a result, allergens, inflammatory mediators, bacteria, and even medications that are applied topically may be able to travel from the orbit to the nose and vice versa.

Imaging studies have shown that nasal septal deviation, inferior turbinate hypertrophy, and mucosal thickening are all associated with both epiphora and primary acquired nasolacrimal duct obstruction (PANDO) [5-7]. The presence of nasal corticosteroids can result in increased intraocular pressure as a consequence of nasolacrimal reflux from the nose to the eye [8].

An analysis of PANDO and other types of nasolacrimal duct obstructions was conducted to identify potential causes and risk factors for these conditions. The most common form of nasolacrimal duct obstruction found in adults is thus called PANDO, and it is primarily linked to nasal pathology. There have been numerous publications with high-quality imaging studies demonstrating that chronic inflammatory conditions and normal anatomical nasal variations were significant factors contributing to the presence of PANDO.

Nasal septal deviation can mechanically constrict the inferior meatus and obstruct the nasolacrimal duct ostium. For example, when the septal deviation involves the inferior aspect of the septum or there is compensatory turbinate hypertrophy on the contralateral side, the inferior meatus is narrowed by mechanical obstruction of the nasolacrimal duct ostium. Conversely, the inferior turbinate hypertrophy that is attributable to allergic rhinitis, non-allergic rhinitis, or some other cause causes enlargement of the turbinate and, in combination with volume-reducing hypertrophic tissue, also compresses the nasolacrimal duct. Finally, chronic rhinosinusitis and allergic rhinitis cause mucosal inflammation, resulting in edema and eventual fibrosis of the mucosa and surrounding structures of the nasolacrimal duct. Computed tomography studies consistently

demonstrate that patients with NLDO have significantly higher rates of nasal septal deviation (up to 70%), inferior turbinate hypertrophy, and mucosal thickening compared to controls [5-7]. Other nasal contributors include concha bullosa, nasal polyps, and anatomical narrowing of the bony nasolacrimal canal itself. These findings suggest that many cases of "idiopathic" NLDO may actually have an underlying rhinologic etiology that is often overlooked.

When an inflammatory mediator is released into the nose from allergies or an upper respiratory infection, it can cause damage to the epithelium of the nasolacrimal duct and also fibrose the submucosa. This process can lead to a gradual progression of the nasolacrimal duct from being functionally obstructed to completely stenosed. The pathophysiological basis for this is the reason many patients who suffer from chronic allergic rhinitis develop epiphora and also why treating the nasal disease can improve lacrimal flow.

2. Neural Pathways Connecting Nasal and Ocular Structures

The trigeminal nerve is responsible for the majority of sensory innervation to both the nose and ocular structures. The ophthalmic division (V1), particularly its nasociliary branch, supplies sensation to both the nasal tip and critical intraocular structures, including the cornea, conjunctiva, and iris. This shared innervation forms the basis of Hutchinson's sign in herpes zoster ophthalmicus. A recent meta-analysis confirmed that the presence of nasal tip lesions increases the odds of ocular involvement by more than six-fold [9-11].

The maxillary division (V2) supplies general sensation to the nasal cavity and is intimately connected with the pterygopalatine ganglion. This parasympathetic ganglion receives preganglionic fibers via the vidian nerve and distributes postganglionic secretomotor fibers to the lacrimal gland, nasal mucosa, and paranasal sinuses. Increased tear production from the lacrimal glands and hyperactive nasal glandular secretion occurs when the trigeminal ganglion is stimulated; this is why soon after an individual with allergic rhinitis is exposed to an allergen in the nasal cavity, they will begin tearing from their eyes or have increased secretions from their nose.

3. Vascular Pathways and Infectious Complications

The venous drainage of the midface, often referred to as the "danger triangle," lacks valves and communicates directly with the cavernous sinus through the angular, supraorbital, and ophthalmic veins. This anatomy permits retrograde spread of infection from nasal furuncles, ethmoid or sphenoid sinusitis, leading to cavernous sinus thrombosis—a rare but life-threatening condition characterized by orbital congestion, ophthalmoplegia, and potential vision loss [15-18].

4. Therapeutic exploration of the sphenopalatine ganglion (SPG)

As a target for therapy has received much interest in recent years. Anatomically (the SPG is located in the pterygopalatine fossa behind the maxillary sinus) it is a parasympathetic relay point that supplies the lacrimal gland and nasal mucosa with parasympathetic innervation.

There are several mechanisms of action for stimulation of the SPG including modulation of the trigeminal system which relieves symptoms by decreasing the sympathetic response through reduction of the production of inflammatory neuropeptides (i.e., substance P, calcitonin gene-related peptide, vasoactive intestinal peptide) and also by changing the immune response from a Type 2 Helper (Th2) dominant response to a more balanced Type 1/Type 2 (Th1/Th2) immune response. Collectively, the mechanistic effects of these treatments alleviate both nasal and ocular neurogenic inflammation.

There is evidence supporting these claims by network meta-analyses showing that ultrasound-guided blocks of the SPG improve both total nasal symptom scores and rhinoconjunctivitis questionnaire scores in patients suffering from allergic rhinitis [14, 19]. These improvements have also been shown to benefit patients with ocular symptoms (e.g. reduction of tearing/itching).The procedure can be performed via transnasal, infrazygomatic, or endoscopic approaches, with favourable safety profiles and minimal adverse effects in most studies. Emerging data suggest particular utility in drug-refractory cases and in patients with prominent autonomic features such as excessive tearing alongside nasal congestion.

Discussion

This systematic review provides a comprehensive synthesis of the anatomical, clinical, and therapeutic dimensions linking nasal pathologies to ophthalmologic symptoms. The findings reinforce that the nasolacrimal duct, trigeminal nerve with its pterygopalatine ganglion, and valveless venous system constitute the three dominant pathways responsible for symptom overlap.

The detailed investigation of nasolacrimal duct obstruction causes reveals that nasal anatomical variations and chronic inflammatory conditions are not merely coincidental but play a causative role in a substantial proportion of cases. This goes against established thinking of NLDO as an idiopathic lacrimal disorder and supports http://theappearancecode.com/hospital/nasoent-ocean-county-nj many nasal evaluations (i.e., endoscopy, CT scans) in cases of epiphora.

One of the major breakthroughs clinically identified in this review is sphenopalatine ganglion (SPG) stimulation. This would provide dual benefit to relieve symptoms through intervention at a significant autonomic relay. Also, this treatment may reduce dependence on long-term medications and improve the overall quality of life for patients with both nasal and ocular symptoms. Therefore, definitive multicenter studies with longer follow-up time will be needed to determine which patients should have SPG stimulation, what the stimulation parameters are for optimal stimulation, and how long the benefit of stimulation lasts for each patient.

Strengths/Weaknesses

Strengths of this review were the systematic methodology, the broad range of data covered and the thematic synthesis of data from multiple domains (i.e., anatomical and interventional). Limitations include heterogeneity of included studies, predominance of smaller trials for SPG interventions, and limited data from low- and middle-income countries. Publication bias cannot be entirely excluded.

Clinical Implications and Future Directions

Future directions and clinical implications of this study are that all clinicians will take a multidisciplinary approach combining both eye doctors and nose doctors. Patients who

suffer from chronic allergic rhinitis and conjunctivitis or have unknown causes for excessive tearing should also have their nasal anatomy assessed to look for potential anatomical problems. If there are problems, they may want to consider SPG-targeted treatments. Future research should focus on standardized outcome measures, comparative effectiveness trials between different SPG techniques, and integration of advanced imaging for personalized treatment planning.

Conclusions

Robust anatomical connections via the nasolacrimal duct, shared neural innervation through the pterygopalatine ganglion, and valveless venous pathways explain the frequent coexistence of nasal and ophthalmologic symptoms. Nasal septal deviation, turbinate hypertrophy, and chronic inflammation are major modifiable causes of nasolacrimal duct obstruction. Sphenopalatine ganglion stimulation has emerged as a promising therapeutic modality with demonstrated benefits for both nasal and ocular manifestations in allergic rhinoconjunctivitis. A collaborative, evidence-based approach between specialties will optimize outcomes for patients affected by these interconnected conditions.

Conflicts of Interest

None Declared.

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