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Nasal Septal Perforation: A Guide for Clinicians

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ABSTRACT: Nasal septal perforation, a communication between the nasal passageways, is usually discovered incidentally during clinical or radiological examination. A broad variety of presumed etiologies have been described, making the diagnostic approach heterogeneous. In this review, we present the reported causes and updated diagnostic and therapeutic management recommendations.

KEYWORDS: Nasal septum, perforation, septoplasty, granulomatosis polyangiitis, relapsing polychondritis, cocaine, rhinitis medicamentosa

A nasal septal perforation (NSP) is a communication between nasal passageways via the cartilaginous or bony portions of the nasal septum. The clinical presentation is indolent for the most part, and most patients with NSP are asymptomatic. For this reason, the prevalence has been inferred from reported radiographic incidental findings. For example, a study of a Swedish population demonstrated that 1% of patients screened had radiographic evidence of NSP,[1] whereas a US study demonstrated an NSP prevalence of 2% in an urban population undergoing computed tomography scans for other indications.[2]

Nasal crusting and epistaxis are commonly present in children with NSP, while in adults rhinorrhea and obstruction have also been described.[3] The size and location of an NSP influence its presentation: Whistling is a more common sign of a small anterior perforation, whereas a posterior perforation is more likely to form asymptomatic scar tissue.

While most cases of NSP are iatrogenic after a variety of nasal procedures or surgeries, other etiologies are possible, and in some cases NSP is a harbinger of systemic diseases. In this article, we review the reported causes and present diagnostic and therapeutic management recommendations.

ANATOMY

The nasal septum is a trapezoidal plate of bone and cartilage with mucosal overlay that extends posteriorly from the nasal vestibule to the choanae and lies perpendicular to the hard palate in the sagittal plane. An anterior cartilaginous portion abuts the posterior bony portion of the septum; anteriorly, the septal quadrangular cartilage lies superior to the small vomeronasal cartilage, whereas the perpendicular plate of the ethmoid bone sits above the vomer bone posteriorly. Bony crests from the maxilla and palatine bone contribute to the posterior-inferior bony septum (**Figure**). Histologically, the septum comprises the nasal mucosa overlying the anterior and posterior perichondrium.[4]

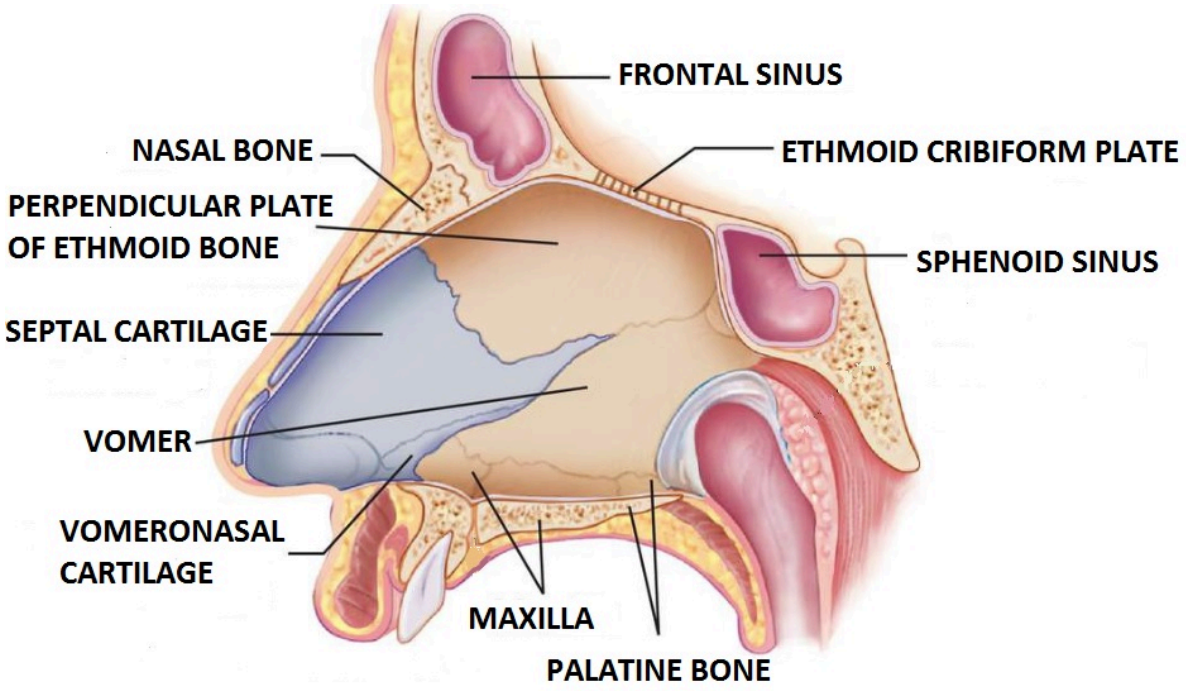

Figure. *Anatomy of the nasal septum.*

PHYSICAL EXAMINATION

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The best way to diagnose an NSP is with direct visualization of the perforation. Caudal nasal perforations can be seen directly with anterior rhinoscopy. Furthermore, illumination of one nostril with a flashlight and observation of the light beam in the other nostril increases the yield of this finding.[5]

ETIOLOGY

Any insult that separates the mucosa from the perichondrium or interferes with the tenuous blood supply to these structures can lead to degeneration, and eventual perforation of the septum. The **Table** presents a list of reported causes of NSP.

Table. Reported Causes of Nasal Septal Perforation

Trauma	Surgical complications[6]
	Nose-picking, rhinotillexomania[7-11]
	Foreign objects, including inhalers[12-15]
Drugs and Toxins	Cocaine[16-19]
	Methamphetamine[20]
	Ketamine[21]
	Vasoconstrictors (rhinitis medicamentosa)[22]
	Intranasal corticosteroids[13-15]
	Industrial irritants (eg, copper, arsenic, chromium, nickel)[23-25]
	Soda ash[26]
	Bevacizumab[27]
Neoplasms[28]	Nasal carcinoma
	Multiple myeloma
	Non-Hodgkin lymphoma
	Natural killer cell/nasal cell lymphoma

Autoimmune and Inflammatory Causes	Systemic lupus erythematosus[28]
	Granulomatosis with polyangiitis[28-31]
	Relapsing polychondritis[32,33]
	Immunoglobulin G4-related disease[34,35]
	Pyoderma gangrenosum[36]
	Takayasu arteritis[36]
	Rheumatoid arthritis[36]
	Mixed connective tissue disorder[36]
	Psoriatic arthritis[36]
	Systemic sclerosis[36]
	Cryoglobulinemia[37]
	Crohn colitis[38]
	Dermatomyositis[39]
	Sarcoidosis[40]
Infections	Leprosy[41,42]
	Tuberculosis[43]
	Nontuberculous mycobacteria[44,45]
	HIV/AIDS[46]
	Histoplasmosis[47]
	Aspergillosis[48]
	Mucor[48]
	Environmental molds[48]

	Mucocutaneous leishmaniasis[49]
	Syphilis[50]
Congenital Defects of Nasal Bones[51]	

Although the largest group of adult patients with symptomatic perforation have no obvious NSP etiology, iatrogenic causes, especially septoplasty and nasal cautery/packing, account for most cases where a cause is identified.[6] Outside of the operating room, nose-picking and its associated compulsive behavior, rhinotillexomania—described in up to 17% of certain adolescent populations—can result in extensive damage not only to the nasal septum, but also to surrounding structures such as the ethmoid sinuses, nasal alae, hard palate, and inferior orbital wall.[7-9] Traumatic septal hematomas and posttraumatic abscesses are found predominantly in children.[10] This may be due to looser connections between the perichondrium and periosteum and underlying cartilage and bone, allowing for blood pooling between these layers and eventual septal dissolution.[11] Foreign bodies, which are common cause of pediatric nasal trauma and obstruction, rarely cause NSP.[12]

Numerous pharmacological substances are linked with NSP. The mechanisms of septal damage caused by these substances range from vasoconstrictive ischemia to direct cytotoxicity. Overall, cocaine use is the most common etiology of NSP.[16] Lateral nasal wall destruction or involvement of the hard palate have been described as a cocaine-induced midline destructive lesion syndrome.[17,18]

Vasoconstriction is the underlying mechanism that leads to NSP. Over time, chronic ischemia results in tissue necrosis of the perichondrium and periosteum. Chemical irritation by a variety of substances used to cut cocaine (eg, talc, mannitol, lactose, borax) may further contribute to septal damage.[19] Other vasoactive drugs of abuse, such as methamphetamine and ketamine, can also lead to NSP.[20,21]

Nasal decongestants such as phenylephrine and oxymetazoline may also cause local vasoconstriction and septal perforation. Longer than 5 days of use can induce rhinitis medicamentosa, and chronic use can induce nasal polyps and ethmoiditis.[22]

The use of intranasal corticosteroids is also linked with NSP, notably in female patients within 12 months of initiation of therapy.[13] Beclomethasone is the intranasal corticosteroid observed to most commonly cause crusting and/or epistaxis preceding perforation.[14] Numerous mechanisms linking local corticosteroids and NSP are postulated but have yet to be confirmed, ranging from local vasoconstriction by inhibition of inflammatory vasodilators to mechanical trauma caused by fluid jets.[15]

Industrial exposure to irritants such as chromium compounds, copper, arsenic, and nickel has been described as a cause of NSP.[23,24] Rhinitis leading to epistaxis is the most common clinical presentation.[25] Workers chronically exposed to large quantities of soda ash without protective equipment had rates of perforation reportedly as high as 12%.[26]

The use of bevacizumab, a monoclonal antibody that inhibits the expression of vascular endothelial growth factor A, approved to treat hereditary hemorrhagic telangiectasia and some advanced solid tumors, is also associated with NSP.[27]

Non-Hodgkin lymphoma, skin basal and squamous cell carcinoma, and melanoma have been described as causes of NSP. Also, patients with the very rare natural killer/nasal cell lymphoma, prevalent in Asia and Latin America, can present with epistaxis and ulceration mimicking an invasive fungal infection, leading to NSP and death within 5 years in 80% of patients.[52] Tissue biopsy is essential for the diagnosis of these diseases.

The nasal mucoperichondrium and periosteum, rich in vascular structures, are subject to vasculitic insults, whereas the cartilaginous structures of the nose itself can be the target of autoimmune diseases. Among the autoimmune etiologies of NSP, granulomatosis with polyangiitis (GPA) is the leading cause of perforations (48% of cases), followed by relapsing polychondritis (26%) and systemic lupus erythematosus (6%). Besides NSP, GPA is associated with destructive lesions of the paranasal sinuses, pharynx, and larynx, and vasculitis affecting the lungs, kidneys, and upper aerodigestive tract.[28] Nasal manifestations range from nasal crusting, sinusitis, obstruction, and epistaxis in most patients; epiphora, nasal perforation, and orbital pseudotumor occur in rare cases.[29] In extreme cases, saddle-nose deformity, an extreme form of NSP whereby nasal bridge support mechanisms are eroded and external deformity results, can be observed.[30] Additionally, sinonasal GPA can present in association with relapsing polychondritis, an autoimmune condition in which auricular, nasal, laryngeal, and joint cartilage becomes periodically and painfully inflamed and destroyed.[32] As many as 25% of patients with relapsing polychondritis demonstrate antineutrophil cytoplasmic antibody (ANCA) positivity.[33] Tissue pathology, not always used for diagnosis, allows some differentiation of these conditions. Treatment is directed at GPA, as this is generally considered more severe than relapsing polychondritis.[31]

Immunoglobulin G4-related disease (IgG4-RD) is characterized by infiltrative IgG4-positive plasma cells, obliterative phlebitis, and fibrosis, and it represents a common disease process for a number of conditions previously described as distinct entities.[34] Sinonasal compromise in IgG4-RD includes palatal erosion and vomero-ethmoid destruction.[35]

Mycobacterium leprae is the most common mycobacterium causing NSP, with the nasal mucosa being involved in up to 95% of patients with lepromatous leprosy.[41,42] Clinical manifestations can range from epistaxis to NSP to total destruction of the nasal septum. The

manifestations can range from epistaxis to NSP to total destruction of the nasal septum. The overall clinical picture of lepromatous leprosy usually supports the diagnosis. Primary nasal tuberculosis has been rarely reported.[43] Nasal mucosa biopsy is necessary for diagnosis of this form of disseminated tuberculosis. Radiological evaluation of lung involvement is essential. Other mycobacteria, such as *Mycobacterium kansasii* and *Mycobacterium marinum*, are rarely reported causes of NSP.[44,45]

Although some case reports have described NSP in patients with AIDS and chronic rhinosinusitis, the mechanism of NSP development in these patients is unclear.[46] Furthermore, in patients with AIDS, invasive fungal infections including with *Histoplasma capsulatum* can cause nasal ulcers and perforation.[47] Mucocutaneous leishmaniasis, which is most common in South America, can cause tumors and eventually perforation of the septum, months to years after a frequently unapparent fly bite carrying the parasite. Histopathological analysis of the nasal mucosa is essential for diagnosis, and new polymerase chain reaction techniques add some diagnostic help.[49] Rarely, tertiary syphilis can destroy the cartilaginous portion of the septum. A combination of serology and histology (endarteritis and plasma cell infiltrates) are diagnostic.[50]

DIAGNOSIS AND WORKUP

Perhaps because of its infrequent presentation, diagnostic guidelines for NSP are lacking. History and physical examination are the most important tools in a clinician's arsenal to diagnose its presence. When NSP is suspected, a detailed history should be obtained regarding prior surgeries, trauma to the nose, drug use, and any rheumatologic symptoms. Direct visualization with the help of a flashlight is usually enough to find a caudal NSP. In contrast, when no obvious cause is found, when rapid enlargement of the NSP occurs, and when the condition appears in immunocompromised hosts, an examination by an otolaryngologist with tissue biopsy when indicated is recommended.

Biopsy of the edge of a septal perforation has demonstrated value but can sometimes be nondiagnostic. In two retrospective studies, the correlation between clinical and histological findings in patients with NSP or ulcers was poor.[53,54] When the suspicion for malignancy is high, and in cases in which serological evidence supports GPA, tissue analysis had demonstrated a higher diagnostic yield. Although imaging studies can show NSP, they seldom identify the cause. In the right clinical setting, chest radiography (to evaluate for sarcoidosis or tuberculosis), rheumatological or infectious diseases laboratory tests, and serum levels of IgG4 may be valuable to diagnose the etiology of NSP.

TREATMENT

Therapeutic strategies for NSP consist of prevention of further progression of the initial disease process and surgical repair of the septal defect. Asymptomatic NSP with stable size usually

requires no intervention.

Treatment with cyclophosphamide and prednisone can frequently cause disease remission in patients with sinonasal and systemic manifestations of GPA.[55] Similarly, the use of high-dose induction with prednisolone in combination with oral corticosteroid maintenance has resulted in response in patients with IgG4-RD, although addition of corticosteroid-sparing immunosuppression is often required.[56] Whereas medical therapy for systemic causes of NSP cannot reverse the anatomical defect, it can often reduce associated symptoms. For example, antitubercular therapy reduces nasal crusting and airway obstruction in patients with perforations secondary to mycobacterial disease.[57]

Surgical repair of septal perforation is typically reserved for patients with obstructive symptoms, airflow disturbances, or nasal whistling in the absence of active septal disease or recently treated head and neck carcinoma. Although description of specific surgical techniques is beyond the scope of this review, the spectrum of septal repair ranges from conservative therapy with nasal saline, topical petroleum jelly, saline irrigations, and nasal buttons to aggressive repair with vascularized local flaps, all with the goal of restoring continuity of intranasal contours and reducing turbulent airflow.[58,59]

CONCLUSIONS

NSP represents a diagnostic dilemma for the clinician due to its broad etiological differential diagnosis. The prognosis is mostly determined by the underlying process. Diagnosis usually requires a thorough investigation and use of multiple tissue specimens, complemented with some laboratory tests. Despite the introduction of novel molecular tests, no cause is found in many cases, which explains the need for further investigation in this field.

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