Nasal septal perforation secondary to rhinitis medicamentosa

Harold F. Keyserling, MD; John D. Grimme, MD; Daniel L.A. Camacho, MD; Mauricio Castillo, MD

Abstract

Nasal septal perforation is a rarely reported complication of rhinitis medicamentosa. We describe such a complication in a 54-year-old man, and we discuss the clinical, pathologic, and imaging aspects of this case.

Introduction

Nasal septal perforation is an uncommon condition. When it does occur, its cause is most often idiopathic or traumatic. Nasal septal perforation may also be the presenting sign of drug addiction or a potentially life-threatening or serious systemic illness, even in an asymptomatic patient. In this article, we describe a case of nasal septal perforation secondary to rhinitis medicamentosa. We also briefly review other common causes of nasal septal perforation.

Case report

A 54-year-old man was referred to us by his primary care physician for evaluation of nasal obstruction with facial pain and pressure. The patient's symptoms had been present for several years. He also reported having used over-the-counter (OTC) nasal sprays for many years. He did not report epistaxis. His medical history was significant for severe asthma, for which he used albuterol and theophylline inhalers. He denied smoking, alcohol abuse, and illicit drug use.

On physical examination, the patient appeared well. He was afebrile and in no acute distress. Findings on a cranial nerve examination were normal, and no cervical adenopathy was present. However, sinonasal endoscopy revealed a near-complete nasal septal perforation and an obstructing mass in the posterior right nasal cavity. The left nasal passage was clear. A biopsy of the mass was performed at endoscopy.

Histopathologic evaluation of the biopsy specimen demonstrated Schneiderian mucosa with scattered submucosal eosinophils. A fragment of fibrinopurulent, eosinophil-rich debris was also present. These findings suggested an allergic etiology. No neoplastic cells were present, and no fungal elements were seen. Laboratory work demonstrated a normal serum angiotensin-converting enzyme (ACE) level and normal serum calcium. A chest radiograph was normal, with no evidence of adenopathy or interstitial lung disease.

The patient was diagnosed with rhinitis medicamentosa and resultant nasal septal perforation secondary to his prolonged use of OTC nasal sprays. He was treated with a steroid taper and intranasal saline drops. Approximately 3 weeks later, computed tomography (CT) of the paranasal sinuses found no evidence of the obstructing mass in the posterior right nasal cavity, suggesting that it had represented inflamed mucosa and had since responded to the steroid taper (figure). Thinning of the medial walls of the maxillary sinuses and nasal turbinates was seen, but no mucosal thickening or air-fluid level was present.

At the 1-month follow-up, the patient reported some alleviation of his symptoms, but he admitted to occasionally using a nasal spray despite instructions not to do so. At the time this report was written, the patient was stable.

Discussion

Rhinitis medicamentosa. Rhinitis medicamentosa is characterized by a nonallergic hyperreactivity of the nasal mucosa that leads to mucosal swelling and congestion.1 It has been reported to affect approximately 1% of all patients seen in otolaryngology clinics, usually young and middle-aged adults.2 There is no predilection for either sex.2 In a series of 130 patients with rhinitis medicamentosa, Toohill et al found that patients had used the causal medication for an average of 21.4 months.2

Rhinitis medicamentosa is caused by the protracted use of nasal sprays, typically OTC decongestants. The active ingredients in these sprays are typically topical vasoconstrictors. Other common offending agents are sympathomimetic amines, such as phenylephrine, and...
selective α2 receptor agonist imidazoles, such as oxymetazoline and xylometazoline. The recommended duration of use for these decongestants is typically limited to 3 to 5 days. Another substance that has been implicated in the development of rhinitis medicamentosa is benzalkonium chloride (BAC), an antimicrobial preservative often found in nasal sprays. BAC’s role as a causal agent in rhinitis medicamentosa has been debated in the literature. However, a recent report revealed that BAC, in concentrations lower than those found in OTC nasal spray preparations, is toxic to human neutrophils. The relevance of this finding to the development of rhinitis medicamentosa has not been established.

Rhinitis medicamentosa is a clinical diagnosis that usually does not require imaging unless it is complicated by a superimposed inflammatory or neoplastic process or nasal septal perforation. Once rhinitis medicamentosa is diagnosed and the offending agent is stopped, patients typically experience rebound nasal congestion. The pathophysiology of this rebound phenomenon is poorly understood. A commonly accepted theory is that rhinitis medicamentosa is a form of vasomotor rhinitis in which rebound arteriolar dilation is caused by disturbances in the autonomic control of the nasal mucosal vascular bed. However, one study has implicated the development of interstitial edema as the primary underlying abnormality.

Patients with rhinitis medicamentosa can be difficult to treat. They become dependent on the nasal sprays that induce their condition in order to relieve the rebound mucosal congestion that develops when they stop it. Weaning patients off a nasal spray can be challenging, as was the case with our patient. Weaning is usually accomplished by administering systemic or intranasal steroids and intranasal saline drops.

Nasal septal perforation. Nasal septal perforation should alert radiologists and clinicians to the possibility of serious underlying systemic or local disease. Published cases of nasal septal perforation secondary to rhinitis medicamentosa are rare, but early reports linked nasal perforations to the prolonged use of topical vasoconstrictors. The persistent deprivation of oxygen to the nasal septum during long-term vasoconstrictor use is believed to induce osteocartilaginous necrosis in a manner similar to the way cocaine does, but possibly with a less rapid onset. Perforations secondary to cocaine use, which have been widely reported in the literature, can occur as quickly as 3 weeks following the commencement of regular intranasal cocaine use. Although we cannot exclude the possibility that cocaine was responsible for the septal perforation in our patient, he did deny using it.

More recently, abuse of intranasal formulations of prescription hydrocodone and codeine have been implicated in nasal septal perforation, with some of these cases being complicated by fungal rhinitis. Nasal septal perforation is also a known complication of systemic vasculitides, including Wegener’s granulomatosis and systemic lupus erythematosus; sarcoidosis; malignancies such as nasal T cell lymphoma and squamous cell carcinoma; infections such as tuberculosis, lepromatous leprosy, and rhinosporidiosis; sinonasal surgery; and traumatic insults such as chronic nose picking and piercing injuries.

The diagnosis of nasal septal perforation is usually made clinically by direct endoscopic examination of the...
nasal passages. Many patients undergo a biopsy at the site of the perforation, although the usefulness of this procedure has been questioned in the literature. Some authors have recommended biopsies only for clinically malignant or polypoid lesions because they had found that most biopsies yielded no additional or clinically relevant information. Additional clinical workup for nasal septal perforation may include an antineutrophil cytoplasmic antibody (ANCA) test to look for Wegener’s granulomatosis, measurements of serum ACE and calcium levels, and chest radiography to screen for sarcoidosis. Urine and serum toxicology screens may prove helpful when cocaine or narcotic abuse is suspected.

Treatment of nasal septal perforation centers on alleviation of symptoms and treatment of the underlying cause or, in the case of intranasal drug abuse or dependency, removal of the offending agent. Although patients with nasal septal perforation may be asymptomatic, many present with intermittent epistaxis, crusting, pain, and rhinorrhea. This symptomatology is believed to occur as a result of decreases in intranasal humidity and temperature control, aberrations that are more commonly seen in anterior perforations than in posterior ones. One aim of surgical closure of the defect is to address these conditions. Effective surgical techniques typically include the use of an interposed connective tissue graft between bilateral intranasal mucosal advancement flaps. Cross-sectional imaging of nasal septal perforation may help determine the size of the bony defect or the extent of a malignant process prior to surgical repair or resection. Imaging is most helpful when inflamed soft tissue or a mass limits rhinoscopic visualization. When evaluating the size of a perforation, the most important determinant of surgical success is the height of the defect from the dorsum of the nose to the floor of the nasal cavity because the tension of the closure flaps and graft is in this plane, not in the anterior or posterior plane. Therefore, coronal image reformations play an important role in presurgical planning. CT is also helpful in determining osseous involvement of the skull base, paranasal sinuses, and orbits by a tumor or an inflammatory process.

Although CT is generally superior to magnetic resonance imaging (MRI) for demonstrating fine bony detail within the nose and paranasal sinuses, MRI may provide better resolution of the soft-tissue structures in this area in a patient with a malignant or aggressive process. This is especially true when perineural spread of disease is suspected. MRI may also help distinguish nasal secretions and mucosal inflammation from tumor masses of the nasal cavity; secretions and inflamed mucosa typically exhibit a hyperintense signal on T2-weighted imaging, in contradistinction to highly cellular tumor masses, which typically exhibit a hypointense T2 signal. Complex proteinaceous nasal secretions may exhibit unusual signal characteristics, but they do not typically demonstrate enhancement.

References