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Palatal Perforation Associated with Intranasal Prescription Narcotic Abuse

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Abstract

Background

Palatal perforation resulting from insufflation of cocaine has been well documented. In comparison, reports of destructive orofacial lesions resulting from intranasal abuse of prescription narcotics are rare. We present the clinical and histologic findings in a case of palatal perforation arising in a patient abusing a prescription opioid drug. The patient denied any history of cocaine use but admitted to habitually crushing and snorting a hydrocodone/acetaminophen preparation.

Study design

The patient presented to our clinic seeking resolution of speech difficulties associated with an oroantral fistula. Surgical repair of the defect had been attempted unsuccessfully in the past. In addition to blood and chemistry panels, endoscopic examination was conducted, with removal of several biopsy specimens for histologic evaluation and flow cytometry. Biopsy specimens included both lesional and perilesional tissue from within the oral and nasopharyngeal cavities. Culture and cytology for fungal organisms were also performed.

Results

Histopathologic examination revealed normal mucosa with diffuse and focal inflammatory changes and no evidence of malignancy. Polarizable foreign material was noted in the specimens. The absence of lymphoid neoplasia was confirmed by flow cytometric analysis. The toxicology panel was positive for the presence of opiates in the blood. Culture and cytology were positive for candidal organisms. A palatal obturator was fabricated for the patient, producing significant improvement in the quality of speech.

Conclusions

This may represent a case of palatal perforation resulting from abuse of a drug other than cocaine. The potential for drugs other than cocaine to produce destructive orofacial lesions should be considered.

1. Case report

A 31-year-old man presented to our clinic complaining of speech difficulties associated with a perforated palate. The patient's speech had a nasal quality and was essentially unintelligible, but he was in no pain or discomfort. The lesion appeared anterior to the junction of the hard and soft palate as a well-demarcated 8 × 11 mm ovoid defect with smooth and distinct margins (Fig 1). Xerostomia was also evident. Surgical repair of the defect had been attempted unsuccessfully 1 year earlier, with the fistula reopening within months after palatoplasty. Upon presentation, it was revealed that the patient had a history of schizophrenia. The patient initially denied any drug abuse, but reluctantly admitted to

habitually snorting a crushed preparation of hydrocodone and acetaminophen. He claimed to have stopped the habit several months earlier. According to the patient, the medication was initially prescribed by a physician, but he could not recall the nature of the condition for which it was prescribed. The patient's physician was no longer in practice and the relevant medical records could not be obtained for review.



Fig 1. Clinical image demonstrating palatal perforation secondary to intranasal narcotic abuse.

Advanced imaging studies, which included head and neck CT and MRI, were performed at another institution a year earlier. These studies revealed a bony defect in the hard palate associated with mucosal thickening throughout the nasopharynx, with no other abnormalities noted. Endoscopic examination was subsequently performed, with removal of biopsy specimens from 12 different lesional and perilesional sites in the nasopharyngeal cavity. This was done in order to establish a definitive diagnosis and to rule out the possibility that the perforation was secondary to malignancy or another pathologic process. Endoscopic evaluation demonstrated a perforation in the nasal cavity, adjacent to a perforated septum, and extending into the palatal aspect of the oronasal cavity (Fig 2). Histologic examination revealed diffuse and focal inflammatory changes in otherwise normal oroantral mucosa, with no evidence of neoplasia, granulomas, or vasculitis (Fig 3). The inflammatory infiltrate was characterized by a dense plasmacytic infiltrate with scattered eosinophils. Polarizable foreign material was also observed in some of the specimens. Flow cytometric analysis revealed no evidence of monoclonal activity, thus ruling out the possibility of lymphoid neoplasia. A complete blood count and syphilis serology were noncontributory. Toxicology screening revealed the presence of opiates in the blood, but no evidence of cocaine. Culture and cytology were positive for candidal organisms of undetermined species.

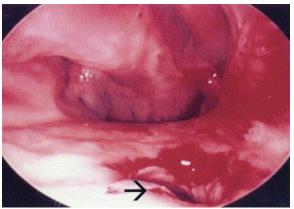


Fig 2. Endoscopic image from the nasal cavity showing the same palatal perforation seen in Fig 1. The oroantral fistula (perforation) is indicated by the black arrow.

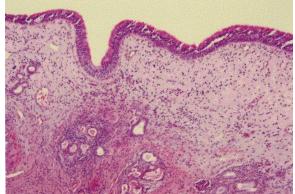


Fig 3. Low-power microscopic view of normal sinonasal mucosa demonstrating a diffuse pattern of chronic inflammation, focal ductal inflammation, and no evidence of malignancy. (H&E, 40× original magnification).

An acrylic palatal obturator was fabricated for the patient. This was done in an attempt to cover the defect and address the patient's chief complaint of speech difficulty. Surgical repair of the fistula was not attempted at this time because the previous attempt was unsuccessful, and the possibility existed of continued drug abuse by the patient. Upon delivery of the obturator, the patient's speech showed significant improvement. The patient was satisfied with the result and was scheduled for follow-up.

2. Discussion

Intranasal drug abuse appears to be a growing trend. In addition to cocaine, insufflation of heroin and other opioids, stimulants, benzodiazepines, and diet pills has been reported.¹¹ Among heroin abusers, insufflation has become a popular method of drug administration, with injecting routes of administration declining dramatically.^{12, 13.} This shift toward intranasal administration may be due in part to increasing awareness of HIV and AIDS among drug abusers.^{11, 12.}

Intranasal abuse of opioid drugs may have similar local complications as intranasal cocaine abuse. Mucosal dryness and septal perforation are reported complications of regular heroin insufflation.^{4., 13.} Xerostomia and septal and palatal perforation were discernible in the patient in this case. Erosion of the soft palate and nasal turbinates are more recently reported complications of intranasal prescription narcotic abuse.³ Though palatal perforations and destructive orofacial lesions are uncommonly seen in abusers of drugs other than cocaine, clinicians should be aware that a variety of causative agents and pathologic conditions may be associated with this clinical presentation, as illustrated in Table I.

Table I. Potential causes of palatal perforation

Adapted from Cottrell et al.²

The pathogenetic mechanisms responsible for opioid-induced damage, as opposed to cocaine-induced damage, remain unknown. With respect to cocaine-associated lesions, it has been suggested that the local vasoconstrictive effects of cocaine may lead to ischemic necrosis of tissue and ultimately nasal or palatal perforation.^{14., 15., 16.} Direct trauma to mucosa anesthetized by cocaine, and irritation by contaminants in the drug, have also been suggested as possible etiologic factors in cocaine-associated lesions.^{2., 16.} Local irritation may further result in stasis of mucociliary activity, crusting, and bacterial or fungal colonization—ultimately leading to necrosis and ulceration.²

As already mentioned, though, the mechanisms of tissue damage with opioid abuse are unknown. One possible explanation for tissue injury with opioid abuse may lie in the effects of opioids on the immune system. Lymphocytes and macrophages are known to possess opioid receptors.^{3, 17.} Opioid drugs may exert immunosuppressive effects through the inhibition of cell-mediated immunity, allowing for the development of invasive bacterial or fungal infections in otherwise healthy opioid abusers.^{3, 18, 19.} In 2 of the 3 previously reported cases of destructive lesions resulting from intranasal prescription narcotic abuse, patients were found to have invasive fungal rhinosinusitis. This finding is somewhat surprising, because invasive fungal rhinosinusitis is typically exclusive to immunocompromised individuals.²⁰ Fungal rhinosinusitis is usually caused by opportunistic pathogens, such as *Phycomycetes* and *Aspergillus* species, and may result in necrosis of nasal mucosa.³ In the present case, candidal organisms of undetermined species were noted on biopsy. This organism is not characteristically associated with necrotic or destructive lesions and was most likely saprophytic or superficially invasive. Though invasive fungal rhinosinusitis may be a serious complication of intranasal opioid abuse, it did not appear to be a factor in the present case.

The relative scarcity of cases of destructive orofacial lesions arising in opioid abusers raises some suspicion that these patients may have used cocaine in the past. In the present case, as well as in previously reported cases, patients denied any prior cocaine use. Toxicology screening, additionally, revealed no evidence of recent cocaine use in our patient. While the possibility of past cocaine use cannot be completely excluded, the potential for opiates or other drugs to produce destructive orofacial lesions should be considered. Finally, we feel a multidisciplinary approach would be the most effective means of managing patients such as the one in this case. This may involve drug counseling and behavior modification in addition to regular chemistry panels to assure discontinuation of the drug habit prior to surgical reconstruction. This approach may increase the likelihood of treatment success.

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