Total necrosis of the intranasal structures and soft palate as a result of nasal inhalation of crushed OxyContin

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Abstract
Nasal inhalation of substances such as cocaine has long been linked to septal necrosis and more recently to palatal perforation. This report describes the case of a 37-year-old man who was addicted to crushed OxyContin (sustained-release oxycodone HCl) tablets and who presented with total necrosis of the septum, sinuses, and soft palate. To the best of the author’s knowledge, this is the first reported case of nasal and palatal necrosis linked to nasal inhalation of crushed OxyContin, which is a relatively new form of drug abuse. The pathophysiology of drug-induced sinonasal disease and a review of the literature are also presented.

Introduction
The link between intranasal cocaine abuse and necrosis of the nasal, sinus, and palatal structures is well known. The highly vascular mucosa of the nasal cavity provides a rapid entry site for drugs of abuse, and cocaine is currently the most widely used such agent. Reportedly, 22 million Americans have tried cocaine, and 4 million use cocaine at least once a month. According to Schweitzer, the incidence of cocaine-induced nasal and sinus complications is 4.8%.

A search of MEDLINE revealed several descriptions of cocaine-induced septal or palatal perforation. These include reports by Vilensky, Schweitzer, Deutsch and Millard, Mattson-Gates et al, Wang et al, Sastry et al, and Gendeh et al. The first descriptions of palatal necrosis were reported by Deutsch and Millard in 1989 and by Mattson-Gates et al in 1991. In both of the latter two cases, cocaine was responsible for the tissue destruction, which occurred as a result of chronic mucosal ischemia and chronic irritation.

Recently, an epidemic of intranasal OxyContin (sustained-release oxycodone HCl) abuse followed the discovery that crushing this long-acting opiate renders it rapidly absorbable and highly intoxicating when inhaled nasally. This epidemic was made possible by the wide availability of OxyContin as a treatment for chronic opiate-resistant pain (e.g., intractable cancer pain) and for other types of pain (e.g., chronic back pain). OxyContin abuse was first reported in the poorer areas of Kentucky, and it has since become a nationwide problem.

Intranasal complications of oxycodone nasal inhalation have hitherto not been reported. In this article, the author describes what to the best of his knowledge is the first such case of a noncocaine narcotic inhalant being implicated in nasoseptal and palatal necrosis. This patient presented with significant necrosis of the nasal and palatal structures, which resulted in the destruction of the septum and turbinates and the loss of a substantial amount of the soft palate. No other such case was found on a search of MEDLINE. Given the epidemic of intranasal oxycodone abuse, more such cases are expected.

Case report
A 37-year-old man presented with complaints of nasal deformity, congestion, postnasal drip, sinusitis symptoms, and reflux of liquid into the back of his nose. He denied having undergone surgery of the nose, sinuses, tonsils, or palate. Other than a nasal fracture he incurred during his youth, he had no significant history of nasal problems and no history of autoimmune or granulomatous disease. He admitted to extensive intranasal abuse of crushed OxyContin, although he said he had never used cocaine. He had become addicted to narcotic analgesics during treatment for severe and intractable back pain subsequent to trauma he experienced during an industrial accident. He had been prescribed oral OxyContin for his pain. As his addiction deepened, he began crushing the tablets and inhaling the powder in increasing amounts.

Physical examination revealed that the patient had a severe saddlenose deformity secondary to a collapse of...
the dorsum, a complete absence of the cartilaginous septum, and a large well-healed cavity within the nose. Rigid nasal endoscopy confirmed the absence of the septum and extensive erosion of the turbinates (figure 1, A). Oral examination revealed a very large defect of the soft palate (figure 1, B). Computed tomography (CT) confirmed the damage to the nasal structures (figure 2). The patient declined surgical treatment for the defects.

Discussion
The problem of nasal necrosis secondary to cocaine abuse is familiar to most practicing otolaryngologists. To the best of the author’s knowledge, the present case is the first reported occurrence of damage to the septum and palate linked to noncoca ine intranasal narcotic abuse. The ubiquity of crushed OxyContin inhalation will likely prompt many other victims to seek otolaryngologic care.

The approach to the intranasal drug-abusing patient requires an understanding of the differential diagnosis of septal perforation, its evaluation, and the mechanism of tissue necrosis secondary to inhaled substances. The best-understood intranasal drug is cocaine. The mechanism of tissue necrosis in cocaine abuse is related to vasoconstriction that occurs as a result of sympathetic stimulation caused by the inhibition of norepinephrine and epinephrine reuptake. In addition, cocaine induces additional vasoconstriction by stimulating vascular smooth muscle contraction. Chronic vasoconstriction leads to mucosal ischemia, atrophy, and ultimately necrosis. In addition, the additives with which cocaine is diluted (mannitol, talcum powder, etc.) serve as mechanical and chemical irritants, leading to giant-cell granulomatous reactions that may be destructive and mimic other granulomatous diseases.1-4

The mechanism by which a nonvasoactive compound
such as OxyContin causes tissue necrosis is not clear, but it is most likely a result of the inflammatory response to the crushed tablets. This response is more similar to the reported reaction to substances used to dilute cocaine than it is to the action of cocaine itself.

The complex of findings in nasal cocaine abuse—and now intranasal noncocaine narcotic abuse—includes nasal collapse, septal perforation, palatal retraction, and pharyngeal wall ulceration. The irritation induced by chronic inflammation can mimic sinusitis and both allergic and nonallergic rhinitis. Once the condition has progressed to septal perforation and tissue necrosis, the clinical findings can mimic midline granulomatous disease, severe bacterial infection, allergic fungal sinusitis, and cancer. Because addicts often conceal their drug use, many of these disorders are evaluated and treated before the clinician discovers that they were caused by intranasal drug abuse.

The differential diagnosis of nasal septal perforation and midline nasal granulomatous disease includes Wegener’s granulomatosis, sarcoidosis, T-cell lymphoma and other malignancies, autoimmune disease, vasculitis, and trauma. Ideally, evaluation should include a biopsy, measurement of the antineutrophil cytoplasmic antibody (c-ANCA) level, and other laboratory evaluations to rule out sarcoidosis and autoimmune disease. Notably, c-ANCA may be elevated in cocaine-induced septal perforation. Unfortunately, drug addiction is often accompanied by noncompliance with medical advice, which can hinder the completion of a full work-up.

Reconstruction of damaged nasal structures often requires repair of the nasal septum and the framework of the nasal tip and valve. Unfortunately, the success of reconstruction is dependent on the patient refraining from further drug abuse. Urine testing and collaboration with a drug addiction treatment program is critical in this regard.

References