

From The New York-Presbyterian Hospital, Columbia-Presbyterian Center

Cocaine-Induced Centrofacial Ulceration

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Cocaine use has increased to epidemic proportions during the past 2 decades. Although recent surveys suggest that cocaine use has decreased over the past 5 years,¹ complications associated with cocaine abuse continue to threaten the health of millions of Americans. An estimated 11% of Americans have used cocaine, and 4 million admit to having used it within the past year.² Myocardial infarction, stroke, cardiac arrhythmias, hypertensive crises, and acute psychosis are only a few of the more serious complications of recreational cocaine use well-known to emergency room physicians.^{3,4} Perhaps the most common sign of habitual cocaine use recognized by dermatologists is nasal septal perforation.⁵ Less common is the far more serious midfacial necrosis reported almost exclusively in the otolaryngology literature. We describe a case of extensive ulceration of the upper lip, nose, and paranasal sinuses induced by chronic nasal insufflation (“snorting”) of cocaine.

Case Report

A 44-year-old woman with a history of chronic sinusitis and asthma was admitted to the hospital with acute upper airway compromise and fever. One week earlier, she began experiencing chills, a nonproductive cough, nasal congestion, and progressive dyspnea, culminating in the development of hoarseness and neck swelling. Plain roentgenography and computed tomography of the neck showed diffuse soft-tissue swelling with significant airway narrowing, while chest roentgenograms showed clear lung fields. Marked edema of the upper airway walls was confirmed by fiberoptic laryngoscopy. She was treated with intravenous antibiotics for cellulitis of



FIGURE 1. An ulcer extends from the upper lip into the floor of the nasal cavity.



FIGURE 2. The base of the columella has been partially destroyed.

the neck, and dexamethasone was administered to protect the airway.

The dermatology service was consulted to evaluate an upper lip ulceration that the patient reported had begun as a small erosion 2 weeks earlier. On physical examination, she had a minimally tender ulcer with ragged, raised borders and a loosely adherent exudate (Figure 1). The ulcer extended from the philtrum of the upper lip to the floor of the nasal cavity, with partial destruction of the base of the columella (Figure 2). Computed tomography showed complete obliteration of the nasal septum with erosion of the turbinate walls and opacification of the sinuses (Figure 3).

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FIGURE 3. Computed tomography shows the cartilaginous nasal septum has been obliterated. The walls of the paranasal sinuses are eroded.

Tzanck smear, potassium hydroxide, and acid fast stains of specimens from the base of the ulcer were negative as were cultures for viral, fungal, and mycobacterial pathogens. Biopsy was refused by the patient.

Initially, it was suspected that the ulceration was self-induced, as part of a Munchausen syndrome. The patient was a former nurse with a history of heavy alcohol and intranasal cocaine use and a complicated past medical history. She had undergone multiple gynecologic, gastrointestinal, and orthopedic procedures and had been hospitalized on numerous occasions for asthma, chronic pancreatitis, episodes of upper gastrointestinal hemorrhage, and recurrent deep venous thrombosis of the thigh. In addition, there was a long history of psychiatric care for depression, self-mutilating behavior, and suicide attempts. On admission, she claimed to have been abstinent from cocaine use for more than 6 months. However, when confronted with the finding of co-

caine metabolites in her urine, she admitted to continued daily intranasal cocaine use.

Within a matter of days, the upper lip ulcer was healing and the patient was breathing more comfortably. Referral for drug rehabilitation was strongly recommended, but she was unwilling to acknowledge her addiction. She was discharged on oral antibiotics with a tapering dose of prednisone, and was lost to follow-up.

Comments

The differential diagnosis of extensive ulceration of the nose (i.e., "rhinophagic" ulceration) covers a broad variety of infectious, collagen-vascular, autoimmune, and neoplastic conditions (Table I), many of which may prove fatal in the absence of appropriate therapy.

In cases for which no specific etiology can be found during initial evaluation, sinonasal lymphoma should

Table I.

Differential Diagnosis of Rhinophagic Ulceration**Infectious**

tuberculosis
 syphilis
 yaws
 leishmaniasis
 leprosy
 herpes simplex virus
 cytomegalovirus
 rhinoscleroma
 noma
 mucormycosis
 paracoccidioidomycosis
 histoplasmosis
 coccidioidomycosis
 blastomycosis
 aspergillosis
 alternariosis

**Collagen-vascular,
Inflammatory & Autoimmune**

Wegener's granulomatosis
 Churg-Strauss disease
 Crohn's disease
 sarcoidosis
 relapsing polychondritis
 lupus erythematosus

Neoplastic

sinonasal lymphoma
 squamous cell carcinoma
 basal cell carcinoma
 adenocarcinoma
 melanoma

Traumatic

cocaine abuse
 iatrogenic
 factitial

be considered. The majority of cases of centrofacial ulcerative lesions, which were previously called "lethal midline granuloma," idiopathic midline destructive disease, polymorphic reticulosis, and lymphomatoid granulomatosis,⁶ are now recognized to be examples of nasal cavity lymphoma^{7,8} (or angiocentric immunoprolifera-

tive lesions).⁹ Multiple biopsies and consultation with an experienced immunopathologist may be required to secure the diagnosis of sinonasal lymphoma, particularly in the early stages when most of the cells may be benign or benign-appearing.¹⁰

The pathogenesis of cocaine-induced nasal septal perforation and centrofacial necrosis likely relates to the complex interplay between the direct vasoconstrictive effects of cocaine and over-the-counter topical decongestants, chemical irritation from the adulterants in cocaine, and local trauma from nose-picking and the use of instruments to snort the drug. Superinfection of the altered nasal structures may further augment the tissue destruction.¹¹⁻¹⁵

Depending on the extent of the inflammation and necrosis, the chronic cocaine user may present with only recurrent epistaxis, headache, or chronic nasal congestion.^{4,16} However, cases of nasal cartilage collapse and saddle-nose deformity,^{13,16-21} perforation of the palate,^{12,15,16,19,20,22} pharyngeal ulceration,^{14,18,23} nasal septal necrosis with ulceration of the lip,²⁴ preseptal cellulitis with orbital wall destruction,²¹ optic neuropathy,^{22,25} cervical emphysema,^{17,26} pneumomediastinum,^{17,26,27} cerebrospinal fluid rhinorrhea,²⁸ and fatal brain abscess²⁹ have all been reported in association with the inhalation of cocaine.

The management of centrofacial ulceration may require reconstructive surgery and the fabrication of prostheses. Efforts to treat the addiction should be undertaken, but unfortunately, as in our case, the nature of the addiction may preclude optimal therapy.

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Errata

In the article by Ackerman *et al.* titled, "New Insights into Old, Common Problems," published in the December issue of *Cutis*[®] (1999; 64:371-373), the statement (twice) in the third from the last paragraph (p.373) should have been that "only uncommonly are Clark's nevi larger than 1.5 cm in greatest diameter," not 1.5 mm as was printed. Miescher's name was misspelled in the second paragraph of the same page.