

Oronasal fistula associated with narcotic substance abuse: a case report

Aldo Vangjeli¹
Erda Qorri¹
Nasira Kopliku²

¹ Department of Oral and Maxillofacial Surgery, Albanian University

² A.V. Implant Dental Clinic, Tirana, Albania

Corresponding author: Aldo Vangjeli
e-mail: aldo.vangjeli@yahoo.it

Abstract

An oronasal fistula, defined as an abnormal communication between the oral and nasal cavities, most frequently results from surgical trauma, infection, or neoplasia. Nevertheless, chronic abuse of intranasal narcotics, especially cocaine, is an increasingly acknowledged etiological factor. This case report intends to present a clinical instance of a cocaine-induced oronasal fistula and to discuss the pathophysiology, diagnostic procedures, and surgical management approaches based on a review of current literature.

Case Presentation: We present the case of a 45-year-old male with a history of chronic cocaine abuse who developed a symptomatic oronasal fistula. Diagnostic computed tomography (CT) revealed destruction of the vomer bone and a fistulous tract. Under general anesthesia, the defect was successfully repaired via a two-layer palatal plastic surgery technique.

Conclusion: This case highlights the serious orofacial consequences associated with substance abuse. The effective management of drug-induced palatal perforations requires a multidisciplinary approach, involving surgical intervention and addressing the underlying addiction to prevent recurrence. Early detection and intervention are vital for ensuring optimal outcomes.

Keywords: Oronasal fistula, cocaine, midline destructive lesion, palatal perforation, palatoplasty, case report.

Introduction

Oronasal fistulas constitute a pathological communication between the oral and nasal cavities, most frequently occurring in the hard palate. They are associated with significant morbidity, including regurgitation of fluids, hyponasal speech, chronic rhinitis, and recurrent sinus infections (1). Common etiologies encompass iatrogenic causes (e.g., post-tooth extraction), trauma, disease, and neoplasia; the role of chronic intranasal narcotic abuse, particularly cocaine, is increasingly documented in the literature (2, 3).

Cocaine use constitutes a considerable public health concern, particularly within the Western hemisphere, where its concurrent use with alcohol has become normalized in specific social and high-intensity occupational settings. This normalization frequently conceals the substantial harm associated with the substance, notably in relation to nasal health. It is extensively acknowledged that intranasal cocaine consumption causes direct injury to the nasal mucosa, resulting in immediate effects such as congestion, epistaxis, and chronic rhinitis. Over time, the corrosive characteristics of the drug and its potent vasoconstrictive effects may lead to more severe consequences, including inflammation, ulceration, and perforation of the nasal septum.

A severe and debilitating complication associated with chronic cocaine use is the development of an oronasal fistula, defined as a pathological communication between the oral and nasal cavities, most frequently occurring in the hard palate. These fistulas



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result in significant morbidity, including the regurgitation of fluids, hypernasal speech, and recurrent sinus infections (1). Common etiologies include iatrogenic causes (e.g., post-tooth extraction), trauma, disease, and neoplasia; notably, the role of chronic intranasal narcotic abuse, particularly cocaine, is increasingly documented in the literature (2, 3).

The pathophysiology involves cocaine-induced tissue ischemia from vasoconstriction, coupled with a local anesthetic effect that masks early symptoms. This combination allows progressive mucosal necrosis and osteolysis (4), often resulting in midline destructive lesions (MDLs) that can perforate the palate. Clinicians must know this etiology, as the presentation can mimic other conditions. This article presents a detailed case report of a cocaine-induced oronasal fistula, its surgical management, and a review of the relevant pathophysiology and treatment strategies.

This article presents a comprehensive case report of a cocaine-induced oronasal fistula, including its surgical management and a concomitant review of pertinent pathophysiology and treatment strategies.

Case Presentation:

A 45-year-old male, Mr. E. M., presented to the maxillofacial surgery department with complaints

of liquids passing into his nose during drinking and significant hypernasality of speech. The patient has a long-term history of intranasal cocaine use. Intraoral examination revealed a palpable defect in the midline of the hard palate. Endoscopic evaluation confirmed the oronasal communication.

Diagnostic Work-up

A non-contrast computed tomography (CT) scan of the head was performed. The imaging demonstrated destruction of the vomer bone. It revealed a distinct fistulous tract in the midline of the hard palate, with a slight deviation to the left, extending from the nasal cavity through the hard palate into the oral cavity (Figure 1).

Blood glucose levels were observed at the upper limit of the normal range. The leukocyte profile revealed mild leukocytosis, without indications of an active infectious process, although the findings remain consistent with a potential diagnosis of bilateral sinusitis (Figure 2).

Treatment

The patient was advised to cease all narcotic use and was scheduled for surgical repair. The procedure was performed under general endotracheal anesthesia. After thoroughly debriding the fistulous margins, the defect was closed in two layers: a nasal layer



Figure 1. Maxillofacial CT: destruction of the vomer and a midline palatal fistulous tract (slightly deviated to the left) creating a oronasal communication.

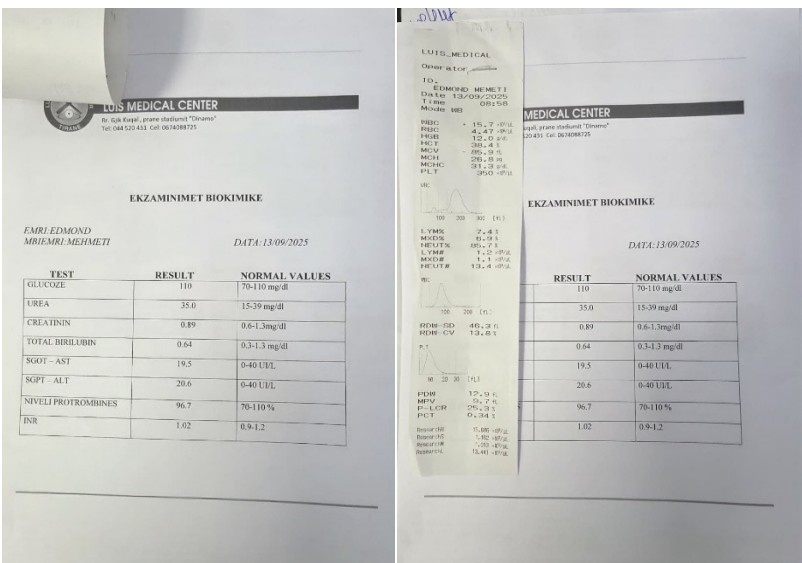


Figure 2. Sinonasal imaging: bilateral mucosal thickening of the paranasal sinuses consistent with sinusitis; laboratory panel showing mild leukocytosis.

was established first, followed by a robust palatal layer, achieving a watertight closure. Bilateral palatal releasing incisions were made to minimize tension on the suture line (Figure 3).

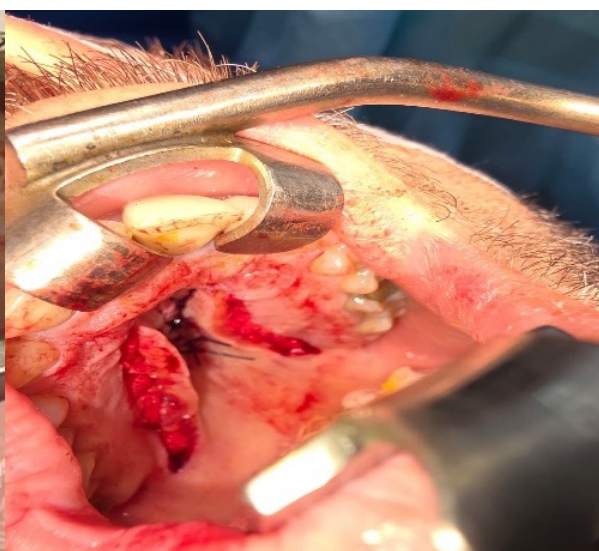
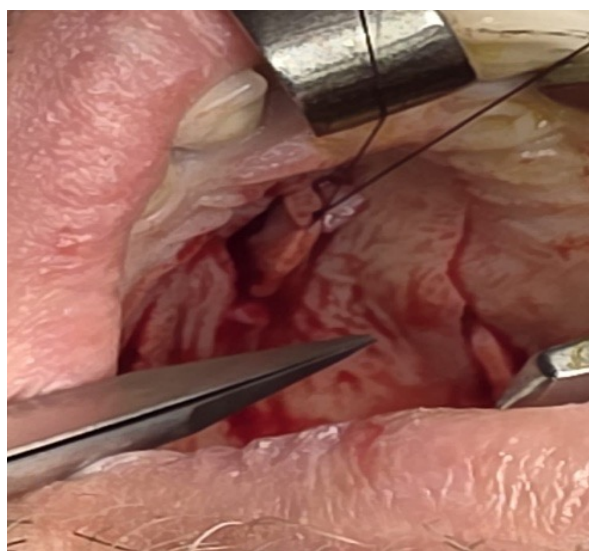


Figure 3. Two-layer palatoplasty: debridement of fistulous margins, closure of the nasal layer followed by a watertight closure of the oral palatal layer, with bilateral palatal releasing incisions to reduce tension.

Postoperative Care and Outcome

The patient's immediate recovery was uneventful. He was hospitalized for 24 hours and managed with: Ceftriaxone 1.0 g IV every 24 hours (antibiotic prophylaxis). Diclofenac (Voltaren) 75 mg/3ml IV twice daily (analgesia/anti-inflammatory). Prednisolone 4 mg orally once daily (to reduce postoperative edema). The patient was discharged in excellent condition with detailed instructions for outpatient care. Follow-up involved wound inspection after 48 hours and scheduled suture removal after 10 days. The patient provided informed consent for the procedure and the publication of this case report.

Discussion

The presented case exemplifies a classic instance of a cocaine-induced midline destructive lesion resulting in an oronasal fistula. The pathophysiological mechanisms are well-documented: cocaine hydrochloride induces severe vasoconstriction of the mucosal blood supply, which leads to tissue ischemia, necrosis, and ultimately bony sequestration (4, 5). The combined anesthetic effect contributes to delayed patient presentation, thereby permitting substantial tissue damage to occur before seeking medical assistance. Diagnosis depends on a high index of clinical suspicion, particularly in patients presenting with unexplained palatal perforations. A thorough patient history is of utmost importance, although substance abuse is frequently underreported (2). Imaging, especially computed tomography (CT), is crucial for delineating the extent of bony involvement and for surgical

planning, as exemplified in our case.

The cornerstone of management is twofold:

1. Addressing the Underlying Cause: Cessation of drug use is non-negotiable to prevent recurrence.

This often requires a multidisciplinary approach involving addiction specialists and psychiatrists (3).

2. Surgical Repair: The selection of an appropriate surgical procedure depends on the size and anatomical location of the defect. Small fistulas may be addressed using local flaps, such as palatal rotation or buccal fat pad flaps. Larger defects, exemplified by the present case, frequently necessitate a two-layer closure complemented by relaxing incisions to achieve a tension-free repair. In extensive tissue loss, vascularized free flaps may be indicated (1, 5).

Our successful outcome with a two-layer palatal repair aligns with techniques described by da Silva Freitas et al. (1) and Aras et al. (5), emphasizing the importance of meticulous surgical technique and tension-free closure.

Conclusion

Cocaine abuse constitutes a considerable risk factor for the development of severe oronasal fistulas. Healthcare professionals must maintain vigilance in identifying the clinical manifestations indicative of this etiology. Achieving successful long-term management presents considerable challenges and depends on a dual approach: definitive surgical correction of the anatomical defect coupled with a comprehensive, multidisciplinary intervention to address the patient's underlying addiction. Neglecting to manage substance abuse invariably results in recurrence and additional tissue destruction.

Statement of Informed Consent

The patient gave informed written consent to publish this case report and accompanying images.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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