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CASE-BASED REVIEW

Dual Palatal Perforation as a Consequence of Chronic Cocaine Abuse: A Case Report with Literature Review

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Main Points

- A rare occurrence of palatal perforation can be a direct consequence as an infrequent complication associated with chronic cocaine abuse that is snorted through the nasal cavity.
- Cocaine powder when lodged within the nasal epithelium can result in oronasal communication, which would subsequently lead food and drink to reflux and cause nasal speech.
- To detect palatal bone erosion and perforation of the soft or hard palate, evaluation of the clinical and radiological examination is required.

Abstract

Palatal perforation can be either congenital or acquired, both of which may be related to a disease. However, a rare occurrence of palatal perforation can be a direct consequence as an infrequent complication associated with chronic cocaine abuse that is snorted through the nasal cavity. Cocaine powder when lodged within the nasal epithelium can result in oronasal communication, which would subsequently lead food and drink to reflux and cause nasal speech. This article presents a rare and a unique case of a 41 years' adult male patient with two palatal perforations in the hard palate induced by chronic cocaine abuse for last 6 years. The patient had developed serious and annoying dysfunction in swallowing and phonation. The patient was advised to discontinue use of cocaine before starting the definitive prosthetic treatment. A brief review of the literature is also presented that focuses on the etiopathogenesis, differential diagnosis, and treatment to shed light on its oronasal effects.

Keywords: Cocaine, oronasal communication, palatal perforation, rhinitis, snorting, euphoria

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Introduction

The drug cocaine (benzoyl methylecgonine) is primarily synthesized/derived from the leaves of the *Erythroxylum coca* plant (Turner et al., 1981). Indian origins of South America have consumed the leaves of this plant for thousands of years to experience its addictive euphoria. Cocaine is mostly administered by snorting it through the nose. A little while after inhalation, a euphoric "high" feeling starts to develop and lasts for 20 – 90 minutes (Brand et al., 2008). The urge to get a quick euphoric high makes the intranasal route one of the primary methods of administering cocaine, over other methods (Maloney, 2011). The nose, as a result, is often frequently exposed to the destructive effects of cocaine powder with untoward effects of it on the nasal passage becoming persistent (Seyer et al., 2002). The growing usage of cocaine intranasally has been linked to a variety of medical problems. Due to the

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effects of both the active ingredient and adulterants, cocaine inhaled directly causes local vasoconstriction and irritation in the nasal mucosa (Rilo et al., 2015). The effects of repeated snorting include ischemia, inflammation, necrosis (micro and macro), and eventually chronic infection, which results in perforation.[8] The most frequent side effect of snorting cocaine is nasal septum perforations, which affect roughly 5% of users (Di Cosola et al., 2007). It has been well-documented that cocaine addiction can lead to epistaxis, chronic rhinitis, hyposmia, palate perforation, midface damage, reduced mucociliary transport, and nasal infection, excruciating facial discomfort and cosmetic nasal abnormalities (Cohen et al., 2008; Silvestre et al., 2010; Trimarchi et al., 2013). A very rare consequence is cocaine-induced palatal perforation, and very few cases have been reported in the literature. For this complication to be diagnosed, a thorough clinical history is necessary, including any prior cocaine addiction, which most people tend not to reveal. To detect palatal bone erosion and perforation of the soft or hard palate, evaluation of the clinical and radiological studies is also required (Di Cosola et al., 2007).

There is a difference in opinion regarding the management in such cases. Lack of motivation from the patient's side for quitting the habit prompts medical professionals to take a more conservative approach to therapy, sometimes hesitating to recommend surgery. Different surgical options and a variety of palate reconstruction techniques have been documented. Local flaps, regional flaps, and free flaps are some of them. The use of palatal obturators enhances patient quality of life by preventing embarrassing situations such as the ingress of liquids and solids into the maxillary sinuses or a nasal tone (Di Cosola et al., 2007; Goodger 2005). Most cases involving cocaine abuse and palatal perforation have been reported as solitary perforations. We present a patient who had developed dual palatine perforation with an intervening mucosal separation due to cocaine addiction and discuss this condition in the light of reported literature.

Case Presentation

A 41-year adult male patient reported to the Department of Oral Medicine, Subharti Dental College, Meerut, with the chief complaint of improper masticatory and speech functions as a result of a perforation in the roof of the mouth. The complaints included liquids passing from the oral cavity into the nasal cavities, purulent discharge from the nose, nasal regurgitation of food for the past 6 months, hypernasal speech, and a diminished sense of taste. Social/personal history revealed a smoker for 15 years, alcoholic, and a 6-year history of cocaine use, snorting cocaine at least two to three times a day. Medical history did not reveal any significant abnormality while the drug history was related to chronic cocaine consumption. Extraoral examination revealed normal parameters for face, temporomandibular joint, and lymph nodes. Functional examination revealed hypernasal voice with no evidence of saddle-nose deformity. On intraoral examination, two palatal perforations with a large perforation measuring 2 cm × 2 cm situated posteriorly and a small anterior perforation measuring 0.5 cm × 0.5 cm with intervening mucosal separation devoid of bone support were observed (Figure 1). The patients' oral hygiene was poor and presented with a Kennedy class 3 modification one partial edentulous arch with one of the arches having a temporary partial denture (Figure 1). The patient

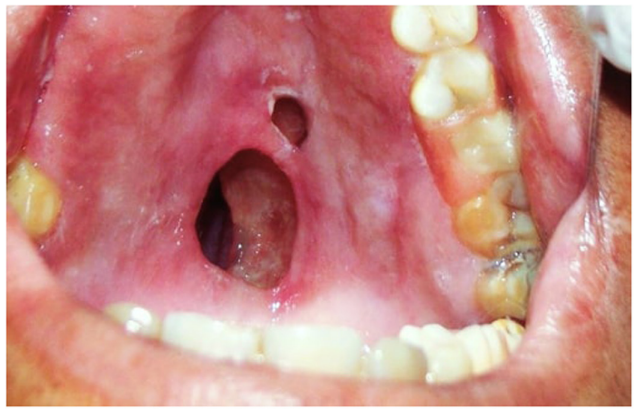

Figure 1. Dual perforation of the hard palate with two perforations (large and small) separated by unsupported mucosal tissue.

provided informed consent. Biopsies from the edge of the septal perforation and palatal perforation showed only acute and chronic inflammation with necrosis but no evidence of vasculitis to rule out malignancy (Figure 2). Routine laboratory tests were within normal ranges, and venereal disease research laboratory and rheumatoid factor tests were negative. Cocaine-induced palatal perforation diagnosis was made based on the patient's clinical examination and social history, with the biopsy taken to rule out dysplastic changes. The patient was advised for a palatal obturator and later surgical repair when his cocaine addiction ceases. The patient failed to return for an appointment.

Discussion

This article in the form of a case report presents dual perforation of hard palate associated with cocaine abuse. The unique feature being two distinct different sized perforations with a thin unsupported mucosal barrier. Cocaine is derived in the usable powder form from the coca plant (*Erythroxylum coca*), and contains benzoyl methylecgonine, a relatively new narcotic among the psychoactive compounds utilized by humans over time (Muakad, 2012).

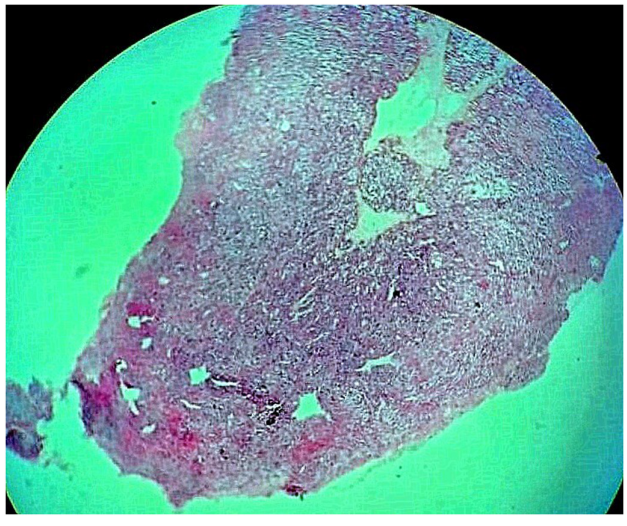

Figure 2. Histopathology of biopsy showing chronic inflammatory cell infiltration mainly lymphocytes.

Cocaine is currently categorized as a psychoactive drug that belongs to stimulants that change brain function and make the central nervous system more active (Blanksma & Brand, 2005). Cocaine can be consumed in a variety of ways, with intranasal administration being one of the more common methods. For a short time after inhaling it, euphoria sets in, lasting for more than an hour depending upon the body metabolism (Maloney, 2011). Mucosal membrane absorption of cocaine results in systemic effects (Silvestre et al., 2010). Clinical effects of the drug include tachycardia, mydriasis, and hyperthermia (Vasica & Tennant, 2002). Intranasal inhalation can result in nasal and oral mucosal irritation and dryness. Damage to the nasal septum and lateral walls of nasal passages has also been reported (Yewell et al., 2002), and less frequently, midline perforation leading to an oronasal fistula or perforation (Rachapalli & Kiely, 2008).

Demography and Clinical Presentation

Silvestre et al., (2010) in their systematic review on palatal perforation among cocaine abusers documented 36 cases of palatine perforation in cocaine users, with females accounting for slightly more than half of all cases (58.33%) and the average age of the patients was close to 40 years, and the majority of them were young adults with a history of cocaine addiction (Silvestre et al., 2010). The manifestation of chronic cocaine abuse varies as reported in the literature. More than 20 reports presented with cocaine-induced midline destructive lesions (septal perforation, turbinate necrosis, hard/soft palate necrosis, and sinus destruction) (Faelens et al., 2021). Reports in the range of 5 – 20 have observed oral ulcers also in the form of oral sores, pemphigus-like lesions, pinna/facial/nasal necrosis, and subcutaneous emphysema (Faelens et al., 2021). Rare or uncommon manifestations reported in the literature include hearing loss, invasive fungal sinusitis, Pott's puffy tumor, and sudden airway obstruction (Faelens et al., 2021). Seven cases of nasolacrimal duct obstruction associated with cocaine usage intranasally have been reported in a case study (Alexandrakis et al., 1999). In such cases, the symptoms vary accordingly and may present as a lack of sensitivity to sensation, anosmia, pain in the nasal passages, headaches, and halitosis (Silvestre et al., 2010).

Patients who have specifically cocaine-induced palatal perforations typically have nonspecific signs and symptoms that are similar to those of other systemic illnesses such as infections, neoplasms, autoimmune, or granulomatous diseases (Trimarchi et al., 2013). Chronic nasal blockage, hyposmia, epistaxis, and excruciating facial discomfort are typically the predominant complaints (Trimarchi et al., 2013). Cocaine has a strong vasoconstrictor effect, it is likely that using it would cause ischemia of the oral cavity's soft and hard tissues, which will then result in their necrosis (Bains & Hosseini-Ardehali, 2005). This destructive process can result in perforations or ulcers on the hard and soft palate, leading to oronasal communication and a nasal voice as a consequence, difficulties with eating and drinking, and even nasal regurgitation (Chaparro-González et al., 2018). Oral symptoms generally include a higher predisposition to periodontal disease, primarily gingivitis, damage to oral tissues, the presence of caries, destructive lesions of the facial midline, xerostomia, and ageusia (Melo et al., 2022). Other independent oral symptoms include dental erosions, hyposalivation, temporomandibular disorders, bruxism,[24] and thermal burns in the oral and pharyngeal cavity (smoking of cocaine). Mouth ulcers, pemphigus-like lesions, laryngeal edema, or rhinitis have been reported to be the less common symptoms (Singh et al., 2017).

Pathogenesis and Differential Diagnosis

Cocaine's local vasoconstrictive effects cause ischemic necrosis of tissue, which eventually results in nasal or palate perforation (Deutsch & Millard, 1989). Both direct injury to the cocaine-anesthetized mucosa and irritation from the drug's impurities have also been proposed as potential causative causes in cocaine-associated lesions (Cottrell et al., 1999). Local inflammation might result in crusting, bacterial, or fungal colonization, mucociliary activity stasis, which may lead to necrosis and ulceration (Cottrell et al., 1999). Vasoconstriction can also be due to central nervous system effects if the drug is taken in moderate-to-large doses. Midline destructive lesions can result from a variety of conditions other than cocaine usage, including infections, neoplasms, systemic disorders, and chemical exposure (Table 1). In order to exclude traumatic causes such as surgery, trauma due to accidents, or self-inflicted lesions, the patient's history should be carefully examined (Wiesner et al., 2004). Midline destructive lesions due to infections like leprosy, tertiary syphilis, mucormycosis in immunocompromised patients, and tuberculosis are also ruled out on the basis of history and other systemic symptoms associated with such diseases (Kuriloff & Kimmelman, 1989). The diagnosis of bacterial or fungal nasal perforations is typically

Table 1.
Differential Diagnosis of Midline Palatal Perforations

Infections	Traumatic	Neoplastic	Autoimmune	Reactive
Tertiary syphilis	Mechanical,	Melanoma	Lupus	Necrotizing
Tuberculosis	Chemical (legal/	Minor salivary gland	Sarcoidosis	sialometaplasia
Leprosy	illicit drugs)	Malignancy	Crohn disease	
Candidiasis	Electrical	Midline destructive Granuloma	Wegener	
Diphtheria	Thermal	Metastatic carcinoma antral	granulomatosis	
Rhinoscleroma	Trauma	malignancies		
Leishmaniasis	Surgery	Cysts and tumors of the		
Mucormycosis	Radiation	oronasopalatal complex		
Actinomycosis				
Histoplasmosis				
Blastomycosis, Coccidiomycosis				

Adapted from Cottrell et al., 1999

simple and relies on examination and laboratory investigations. Numerous immune system conditions, such as systemic lupus erythematosus (Reiter & Myers, 1980), sarcoidosis, rheumatoid arthritis, polyarteritis nodosa, mid-facial granuloma syndrome, and granulomatosis with polyangiitis (GPA) (Trimarchi et al., 2013), can result in midline destructive lesions.

Investigations

When a patient exhibits nonspecific signs and symptoms which resemble those of other systemic disorders, a positive test for cocaine metabolites in the urine, blood, or hair might be a helpful hint and raise suspicion of cocaine-induced palatal perforation (Trimarchi et al., 2013). Indirect immunofluorescence microscopy reveals anti-neutrophil cytoplasmic antibodies frequently in cocaine-induced palatal perforation lesions. These antibodies particularly and principally react with human neutrophil elastase, also known as human leukocyte elastase, when evaluated using antigen-specific solid assays, and they exhibit a perinuclear staining pattern (Peikert et al., 2008) . Multiple biopsy specimens taken from the outer margins of the lesion should be sent for histopathological investigation (Trimarchi et al., 2013). Peripheral tissue rather than the necrotic center of the disease should be always taken. There are no disease-specific histological characteristics of cocaine-induced palatal perforation lesions, as many of these features are also common to GPA. Hence GPA constitutes cocaine palatal lesion's primary histopathological differential diagnosis. The cocaine-induced palatal perforations share the following histological manifestations: leukocytoclastic vasculitis, mixed inflammatory infiltrates, microabscesses in vascular walls, perivenulitis, and vascular microthrombotic alterations (Trimarchi et al., 2001). The diagnosis of Cocaine-induced palatal perforation (CIPP) cannot be made with certainty by radiographic assessment of patients with CIPP. In severe cases, a computed tomography scan can reveal the extent of bone and cartilage damage as well as the centrifugal advance of the deterioration. Magnetic resonance imaging (MRI) with contrast medium provides more information in cocaine-induced palatal perforation lesions (Trimarchi et al., 2001 & Trimarchi et al., 2006). On MRI, affected nasal and paranasal mucosa might be seen as hypo intense areas on T2 and exhibit reduced or irregular enhancement (Trimarchi et al., 2003). Mapping the sites of soft tissue erosion with MRI is also helpful. Although none of the radiological findings presented are disease-specific, they do provide additional information that aids in defining the local extent of cocaine-induced palatal lesions and possible options for treatment (Trimarchi et al., 2013 & Trimarchi et al., 2003).

Management

Because complete abstinence from the substance is required before beginning therapy, it is necessary for the treatment to involve psychiatric assistance (Monasterio & Morovic, 2003). The majority of cocaine-related septal perforations are small, asymptomatic, and require no treatment (Cottrell et al., 1999). Treatment options for palatal perforations are numerous. Before beginning any reconstructive surgery, some surgeons suggest a drug-free period of at least a year that is confirmed by serum and urine toxicology testing (Di Cosola et al., 2007 & Rampi et al., 2021). Aleatory urine tests for metabolites may be advised to monitor the patient's progress because cocaine is excreted in the urine for 48 – 72 hours after consumption (Berman et al., 2016). Margin biopsies have also been suggested as a way to identify ongoing cocaine addiction signs like vasoactive effects, vasculitis, infarcted regions, and polymorphonuclear cell infiltrates (Silvestre et al., 2010). It is recommended to use a multidisciplinary and conservative management approach, which includes analgesics, antibiotic therapy, surgical debridement, saline solution lavage, and even the implantation of an obturator during the recovery phase (Rampi et al., 2021 & Villa, 1999). A removable palatal obturator is used in traditional management as a conservative approach. This incorporates a prosthetic sealing of the defect, which helps people deal with social problems and enhances quality of life by preventing solids and liquids from being regurgitated to the nose while swallowing and by reducing nasal speech. It is also the preferred course of treatment for people who reject surgical intervention or who are unable to give up the habit. Additionally, it can be periodically relined to enhance fitting in unstable-in-time defects (Barrientos et al., 2021). Any maxillary defect in the form of cleft that has an oronasal communication is comfortably restored with a tooth-supported modified partial denture with an obturator to close the opening. Existing teeth act as abutment and help to retain and support the obturator in place as a definitive treatment (Mattoo et al., 2014). Poor oral hygiene maintenance in young cocaine abusers can render them to an early stage of complete edentulousness. One drawback in such cases will be that edentulous patients will experience fitting and retention issues, and large obturators can recede sensory feedback from the covered mucosa, impairing speech, mastication, and deglutition (Di Cosola et al., 2007). Furthermore, an obturator cannot be worn continuously and may not be able to stop liquid and air leaks in all patients (Nord et al., 2012). There are numerous surgical methods that have been described. Only small perforations can be covered with local flaps. These include pediculated mucoperiosteal flaps, direct suture, cleft palate procedures, reverse mucosal flaps, and palatal flaps. Palatal tissue is directly influenced by factors like the underlying bone structure and perforation size. A small amount of tissue is provided by local flaps (Nord et al., 2012). For small to medium problems, regional flaps such as the tongue, buccinator, buccal fat pad, or temporal muscle flaps can be used ((Barrientos et al., 2021; Nord et al., 2012; Cintra et al., 2008). Regional pedicled flaps have limitations but are dependable. The temporalis muscle flap requires a maxillary tunnel and can affect aesthetic appearance, dorsal pedicled tongue flap requires three weeks of intermaxillary fixation, then a second stage of surgery to separate the flap from the tongue (Nord et al., 2012 & Jackson et al., 2009). Finally, distant flaps, and more frequently a radial forearm free flap, can be employed to repair the defect. These flaps can cover large areas, but they need a skin graft to cover the donor site, where any resulting cosmetic defects are crucial (Nord et al., 2012). In situations when there is insufficient blood supply to the oronasal mucosa, Pelo et al. developed a novel and efficient strategy for repairing small- and medium-sized oronasal connections. They discussed how to correct small- and medium-sized oronasal connections using the Le Fort I osteotomy surgical technique and a bilateral Bichat's fat pad flap. This procedure is simple to carry out and has little effect on the patient's aesthetic appearance (Pelo et al., 2008).

The general dentist should be alert for noticing palatal perforations and ascertain the etiology of these lesions. The clinicians

should always inquire about cocaine use in the patient history. Reconstructive surgery and a prosthesis obturator are the two treatment options for a cocaine-induced palate perforation. The management of these perforations is by a multidisciplinary team which includes a psychiatrist, medical physician, oral pathologists, maxillofacial surgeons, maxillofacial prosthodontists, speech therapists, and a psychologist.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Informed Consent: Informed consent was obtained from the patient who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

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