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Case Report

Oronasal and Orosinusal Lesions Associated with Intranasal Cocaine Use: Two Clinical Case Reports

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Abstract: Intranasal cocaine use may lead to a progressive spectrum of centrofacial destruction known as cocaine-induced midline destructive lesion (CIMDL), which may involve the hard palate and result in oronasal or orosinusal communication, with major functional and social repercussions. Because oral examination routinely exposes the palate, dentists may be the first clinicians to detect these lesions; however, diagnosis is often delayed because patients may initially omit illicit drug use and because the clinical presentation may overlap with vasculitic, infectious, and neoplastic disorders. This report describes two cases of cocaine-associated palatal destruction diagnosed at different stages of progression and discusses the main etiopathogenic, histopathological, diagnostic, and therapeutic aspects relevant to dental practice. The first case involved a woman with an established palatal perforation measuring approximately 3 cm, hypernasal speech, nasal regurgitation of food and liquids, and improvised self-occlusion of the defect with plastic material. The second case involved a 35-year-old man with a posterior palatal nodule, computed tomographic evidence of osteolysis affecting the floor of the nasal cavity, and incisional biopsy showing chronic granulomatous inflammation in the context of chronic intranasal cocaine use. Together, these cases illustrate that cocaine-related palatal destruction may be identified at markedly different points along the same pathological continuum, from pre-perforative osteolytic disease to extensive communication with severe functional impairment. Early recognition, structured differential diagnosis, cocaine cessation, and multidisciplinary rehabilitation are essential for appropriate management.

Keywords: Cocaine; Oronasal Communication; Palate; Midline Destructive Lesion; Dentistry.

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1. Introduction

Cocaine (benzoylmethylecgonine) is an alkaloid derived from the leaves of *Erythroxylum coca* and remains one of the most widely used illicit stimulants worldwide [1-3]. Among the different routes of administration, intranasal use is especially relevant for oral and maxillofacial clinicians because repeated deposition of cocaine powder on the nasal mucosa may trigger progressive ischemic and inflammatory injury involving the septum, turbinates, paranasal sinuses, and, in more severe cases, the hard and soft palate [4-8].

This destructive pattern is commonly referred to as cocaine-induced midline destructive lesion (CIMDL), a clinicopathological entity characterized by progressive osteocartilaginous and mucosal destruction of midfacial structures [8-10]. Nitro et al. proposed a four-stage classification according to anatomical extent, highlighting palatal involvement

as an advanced but well-recognized manifestation of the disease [9]. When the palate is affected, patients may develop oronasal or orosinusal communication with hypernasal speech, dysphagia, halitosis, nasal regurgitation of solids and liquids, and substantial impairment in quality of life [10,11].

The pathogenesis of CIMDL is multifactorial rather than purely ischemic. Cocaine-induced vasoconstriction plays a central role by reducing blood supply to the mucosa and septal perichondrium; however, direct chemical irritation, repeated mechanical trauma caused by crystalline insufflation, adulterants, recurrent bacterial infection, and immune-mediated mechanisms also contribute to tissue destruction [12-15]. Levamisole, a frequent cocaine adulterant, further complicates this scenario because it may induce a distinct vasculopathic or vasculitic syndrome with high p-ANCA anti-MPO titers, agranulocytosis, skin necrosis, and occasional renal involvement [16-18]. For that reason, cocaine-associated destructive lesions may mimic primary autoimmune disease both clinically and serologically.

The differential diagnosis is broad and must be handled rigorously. Granulomatosis with polyangiitis (GPA) is the main vasculitic mimic because it may present with sinonasal destruction, ulceration, and ANCA positivity [19, 21]. Other important considerations include granulomatous and infectious destructive diseases, extranodal NK/T-cell lymphoma, and malignant neoplasms of the upper aerodigestive tract or minor salivary glands [20-22]. Mucocutaneous leishmaniasis deserves particular attention in endemic or epidemiologically compatible settings because it may present as a midline destructive process and should be actively considered in the diagnostic workup of such lesions [20].

For dental clinicians, the practical relevance is obvious. Routine examination of the oral cavity allows early identification of suspicious palatal changes, including ulceration, tissue softening, bone exposure, perforation, and hypernasal speech. Yet diagnosis is frequently delayed because patients may initially deny or omit cocaine use, and because the lesions may be mistaken for common inflammatory, infectious, or neoplastic conditions [21-23]. The present article reports on two clinical cases of cocaine-associated palatal destruction at different stages of progression and discusses the main diagnostic and therapeutic implications for dental practice.

2. Case Report

2.1 Case 1

A 30-year-old woman sought care because of a progressively enlarging hole in the roof of the mouth associated with altered speech, difficulty chewing, and reflux of food and liquids into the nasal cavity (Figure 1). According to the patient, the lesion had been evolving for approximately two years. Her medical history included recurrent allergic rhinitis, frequent use of dipyrone for headache, and prior use of ranitidine for gastrointestinal symptoms. She also reported a long history of smoking and alcohol use. At her initial visit to a primary healthcare unit, the patient did not report illicit drug use. During later specialist assessment, however, she disclosed chronic intranasal cocaine use for approximately five years before the palatal defect became clinically evident. Extraoral examination showed deviation of the nasal septum to the right. Palpation of the neck revealed small, mobile, soft, non-tender lymph nodes in the right submandibular region, compatible with reactive inflammatory lymphadenopathy.

Intraoral examination revealed a round, well-defined perforation in the central hard palate measuring approximately 3 cm in diameter, establishing direct communication between the oral cavity and the nasal cavity. Nasal mucosa was visible through the defect. One of the most striking findings was the patient's use of a plastic bag as an improvised obturator during meals to reduce nasal reflux of food and liquids. Her speech was clearly hypernasal.

Figure 1. Clinical appearance of Case 1 showing the palatal perforation and improvised occlusion device.

The combination of midline palatal perforation, chronic rhinitis, septal abnormality, and subsequent confirmation of intranasal cocaine use strongly supported the diagnosis of CIMDL with established oronasal communication [8-10,21]. Because the lesion was already advanced and functionally disabling, the patient was referred to multidisciplinary management involving oral medicine, otorhinolaryngology, maxillofacial evaluation, and support for cocaine cessation. Conservative prosthetic obturation was considered essential for immediate functional improvement while longer-term reconstructive planning depended on clinical stabilization and sustained abstinence [24-26]. Two limitations of this case must be acknowledged: serological investigation for ANCA (anti-PR3 and anti-MPO) was not performed, precluding definitive exclusion of GPA or levamisole-induced vasculopathy; and no incisional biopsy was obtained, despite current recommendations mandating tissue sampling in all destructive midline lesions to exclude malignancy, granulomatous infections such as mucocutaneous leishmaniasis, and primary vasculitis.

2.2 Case 2

A 35-year-old man presented with a three-month history of a lump on the palate (Figure 2A). The lesion was initially asymptomatic but had become painful when blowing his nose in the weeks before consultation. He also described the sensation of expelling tissue fragments through the nose while blowing. His medical history included anxiety, depression, panic disorder, and dependence on cocaine and marijuana. He reported active intranasal cocaine use. His psychiatric comorbidities were under pharmacological treatment prescribed by his psychiatrist: quetiapine (atypical antipsychotic), levomepromazine (sedative), and paroxetine (selective serotonin reuptake inhibitor).

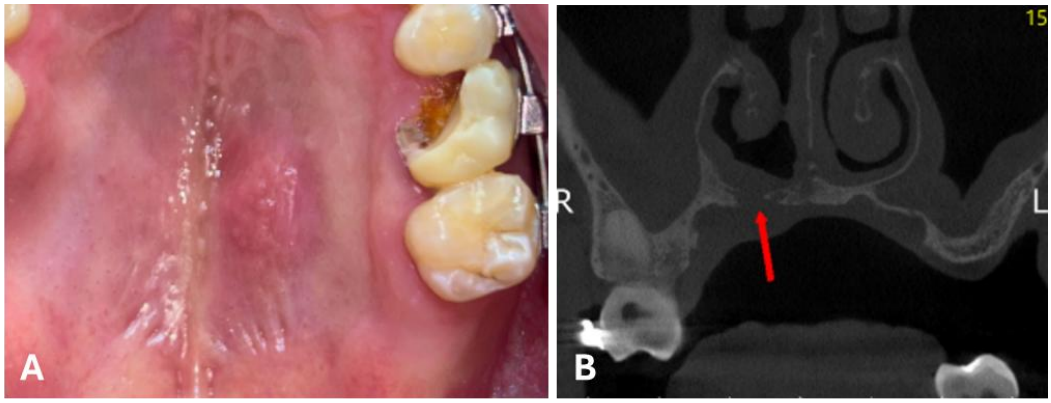
Intraoral examination showed a sessile erythematous nodule in the midline region of the posterior hard palate. The lesion had a smooth surface, poorly defined margins, and a softer consistency than the surrounding mucosa, with pain on palpation. No visible palatal perforation was present at that time. Computed tomography (Figure 2B) of the face and paranasal sinuses demonstrated a cavitary osteolytic lesion compromising the integrity of the floor of the nasal cavity, with mild soft-tissue swelling of the palate. The radiological report raised the possibility of a minor salivary gland neoplasm and recommended histopathological examination. Additional diffuse osteolytic changes were observed in the nasal septum and inferior turbinate's, interpreted as associated chronic inflammatory destruction.

An incisional biopsy was performed under local anesthesia. Histopathological examination showed granulation tissue with dense lymphoplasmacytic inflammatory infiltrate. Granulomatous formations composed of histiocytes, and multinucleated giant cells

were observed at the lesion periphery, together with numerous eosinophils and a neutrophilic abscess. Other areas displayed mucinous glandular tissue with degenerating acini. No evidence of malignancy was identified. When correlated with clinical history and imaging findings, the diagnosis was consistent with chronic granulomatous inflammation secondary to intranasal cocaine use.

This case represented a pre-perforative stage of CIMDL, in which the lesion was clinically subtle but structurally significant. The patient was informed that continued cocaine use could lead to progression toward frank palatal perforation with major functional impairment [10,11,25]. He was referred to as oral medicine follow-up, otorhinolaryngological assessment, psychiatric care, and chemical dependency treatment. Because this stage theoretically offered an opportunity to prevent more severe tissue loss, given that cessation of vasoconstriction may allow partial tissue recovery strict cocaine cessation was emphasized as the main therapeutic measure [8-10,24]. However, long-term follow-up data confirming radiological stabilization or regression of the osteolytic lesion in this specific patient are not available, and this must be acknowledged as a limitation of the present report.

Figure 2. A. Sessile erythematous nodule in the posterior hard palate at the midline in Case 2. B. Computed tomography showing cavitary osteolysis affecting the floor of the nasal cavity in Case 2.



3. Discussion

The two cases reported here illustrate different stages along the same destructive pathological continuum. In Case 2, the lesion was identified during an active granulomatous osteolytic phase, before visible perforation had developed. In Case 1, diagnosis occurred only after a large palatal defect had already formed, with clear oronasal communication and severe functional repercussions. This contrast is clinically important because it demonstrates that stage at diagnosis directly influences prognosis, treatment complexity, and the degree of irreversible structural loss.

CIMDL is not adequately explained by vasoconstriction alone. Although ischemia is central, the available literature supports a multifactorial pathogenesis involving direct chemical toxicity, mechanical trauma, recurrent bacterial colonization, epithelial apoptosis, and immune dysregulation [12-18]. Levamisole contamination further complicates the picture by producing an overlapping vasculopathy phenotype that may mimic primary autoimmune disease. This matters in real practice because serological findings, especially ANCA patterns, may be misleading when interpreted outside the broader clinical and histopathological context [16-18].

Serological findings differ between pure CIMDL (anti-HNE positivity), GPA (anti-PR3), and levamisole-induced vasculopathy (p-ANCA anti-MPO), but may be misleading when interpreted in isolation [16-18]. Neither patient underwent formal ANCA testing, which is a recognized limitation; clinicians evaluating similar cases should include a complete serological panel. Neither patient exhibited clinical features specifically attributable

to levamisole toxicity, such as cutaneous necrosis or agranulocytosis, and the levamisole discussion is therefore presented as epidemiological context rather than a direct finding in these cases.

Differential diagnosis remains one of the main clinical challenges. GPA is the classical mimic because both conditions may present with sinonasal destruction, ulceration, inflammatory histology, and ANCA positivity [19,21]. However, palatal perforation is substantially more associated with CIMDL than with GPA, making it a particularly useful clinical clue in the appropriate anatomical and historical setting [21]. The differential diagnosis must also include infectious and granulomatous destructive conditions, especially in endemic regions or compatible exposure settings. Mucocutaneous leishmaniasis is particularly relevant because it may clinically simulate other midline destructive lesions and should be actively investigated when epidemiological history supports that possibility [20]. Extranodal NK/T-cell lymphoma and malignant salivary or epithelial neoplasms must also be excluded by biopsy [20-22].

Case 2 underscores the indispensable role of histopathological examination. Computed tomography suggested a destructive lesion with the possibility of a minor salivary gland tumor, but biopsy redirected diagnostic reasoning toward chronic granulomatous inflammation associated with cocaine use. This is precisely why clinicians should avoid premature closure based solely on imaging or presumed substance history. Cocaine use increases the suspicion for CIMDL, but it does not eliminate the need to exclude infection, vasculitis, and malignancy in a structured manner.

Another practical point is that the visible mucosal lesion may underestimate the true extent of disease. In Case 2, a modest palatal nodule concealed cavitary osteolysis involving the floor of the nasal cavity. In cocaine users, clinically subtle lesions may already represent extensive deeper destruction. Therefore, computed tomography is not optional when midline palatal lesions are accompanied by pain, tissue softening, nasal symptoms, suspected bone loss, or any history compatible with intranasal drug use [8-10]. When imaging raises the possibility of a neoplastic process or perineural extension, as occurred in Case 2, magnetic resonance imaging (MRI) should be considered as a complementary modality because it provides superior soft tissue contrast resolution compared with CT. In Case 2, MRI was not obtained; the diagnostic uncertainty was ultimately resolved by histopathology, which remains the gold standard regardless of imaging findings.

The two-year delay between the onset of the palatal perforation and Case 1 seeking structured care warrants specific discussion. Several non-mutually exclusive factors likely contributed: (1) social stigma and fear of judgment associated with illicit drug use, evidenced by the patient's initial non-disclosure at the primary care visit; (2) barriers to healthcare access, including limited pathways from primary care to specialist services; (3) functional accommodation through the improvised plastic obturator, which may have reduced the perceived urgency of formal care; and (4) cognitive mechanisms of denial or minimization, common in substance use disorders. This pattern underscores that clinical detection alone is insufficient: integrated care pathways connecting dental practice, primary care, and addiction medicine services are necessary to ensure timely, non-stigmatizing access to treatment for patients with CIMDL [22, 23].

For dental clinicians, delayed disclosure of cocaine use is a recurring diagnostic obstacle [21-23]. Patients may withhold this information because of shame, stigma, fear of judgment, or fragmented prior care. That means the burden of early suspicion falls heavily on anatomy and pattern recognition. When a lesion is midline, destructive, poorly explained by common dental causes, and associated with hypernasality, halitosis, rhinitis, septal symptoms, or nasal regurgitation, the history must be explored directly and objectively. A nonjudgmental but explicit approach is clinically superior to vague questioning.

Treatment is fundamentally stage-dependent, but all successful strategies share one prerequisite: cocaine cessation [8-10,24]. Ongoing exposure perpetuates tissue injury and compromises both conservative and surgical rehabilitation. In early lesions, abstinence

may stabilize the process and prevent perforation. In advanced lesions, abstinence remains indispensable because reconstruction and long-term prosthetic management are unlikely to succeed if the destructive habit continues [24-26].

For established communications, prosthetic obturation remains the main conservative option because it offers immediate improvement in speech, swallowing, and daily function [25,26]. In Case 1, the improvised plastic barrier used by the patient is revealing. It demonstrates both the severity of her dysfunction and the prolonged absence of adequate rehabilitation. An obturator does not treat the underlying disease, but it can dramatically improve quality of life while broader management is organized. Prosthetic obturators for palatal defects of this type are typically fabricated in heat-cured acrylic resin as removable appliances, retained by existing teeth or implant-supported fixtures. In Case 1, the fabrication of a custom acrylic obturator was planned following clinical stabilization and reported cessation of cocaine use, replacing the patient's improvised plastic device.

Surgical reconstruction should be considered only in carefully selected patients with documented sustained abstinence, ideally confirmed by serial urinary benzoylecgonine measurements rather than self-reporting alone, which is demonstrably unreliable in this population [24,26]. It must be acknowledged that formal toxicological verification was not performed in either case reported here: in Case 1, abstinence was patient-reported at the time of referral; in Case 2, monitoring relied on inpatient rehabilitation supervision without documented post-discharge urinary screening. Clinicians should consider this limitation when applying these cases to surgical decision-making.

Surgical reconstruction in CIMDL carries substantially higher complication and flap failure rates than in non-cocaine users, even among those reporting sustained abstinence, due to irreversible endothelial dysfunction and microvascular remodeling induced by chronic use [24]. Realistic prognostic counselling is mandatory, and surgical teams should anticipate the need for revision procedures. Free microvascular flaps, such as the radial forearm free flap, are generally preferable to local pedicled options because adjacent tissues may also be compromised by the chronic microvascular injury [24].

Psychiatric and behavioral comorbidities must also be treated as central rather than peripheral issues. Case 2 involved anxiety, depression, panic disorder, and substance dependence, all of which directly affect adherence, follow-up, and relapse risk. A purely local treatment strategy is therefore insufficient. Effective management requires multidisciplinary coordination among dentistry, otorhinolaryngology, pathology, psychiatry, and addiction care. In practical terms, these lesions are anatomical, behavioral, and psychosocial at the same time. Taken together, these cases reinforce the point that dentists are often the first professionals able to recognize cocaine-related palatal destruction inside the mouth. Early identification, correct differential diagnosis, timely imaging and biopsy, and referral without stigma may prevent progression from granulomatous osteolysis to irreversible oronasal communication.

4. Conclusion

Cocaine-induced palatal destruction may present either as early granulomatous osteolytic disease without visible perforation or as advanced palatal loss with frank oronasal communication. The two cases reported here demonstrate that stage at diagnosis substantially influences prognosis, treatment burden, and functional outcome. Definitive diagnosis depends on integration of clinical history, intraoral examination, imaging, histopathology, and careful exclusion of infectious, vasculitis, and neoplastic mimics. For dental clinicians, the practical message is direct: suspicious midline palatal lesions should trigger structured investigation even when the patient initially omits cocaine use. Cocaine cessation remains the indispensable therapeutic foundation. In early lesions it may prevent progression, in advanced lesions it is required for successful prosthetic or surgical rehabilitation.

Early recognition and multidisciplinary management remain the key determinants of better outcomes. When a destructive midline palatal lesion is encountered, clinicians

should document the lesion and associated nasal signs, obtain a direct history including cocaine use, request CT and MRI when indicated, perform incisional biopsy, and ensure referral to addiction and psychiatric care.

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