Nasal cocaine abuse and centrofacial destructive process: Report of three cases including treatment

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We report 3 new cases of a centrofacial destructive process associated with chronic nasal abuse of cocaine. This complex first described in 1988 is a rare entity involving sinonasal tract necrosis after cocaine abuse. Of special interest in this report is a male patient with columella and lip involvement instead of the more usual rhinopalatal destruction. This cocaine abuse complex should be included in the differential diagnosis of centrofacial midline destructive processes in young patients as the first diagnostic possibility. We suggest a management strategy for these patients.

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Cocaine was first introduced to medicine in 1884 by Köller as a local anesthetic, but the earliest known use of cocaine was by sub-Andean Indians. They chewed leaves of the coca plant because of its stimulating properties and to help them tolerate working at higher altitudes. They were aware of the property of this substance to numb the tongue and oral mucosa.

Cocaine is an alkaloid prepared from the leaves of the *Erythroxylon coca* plant. For many years the illegal consumption of cocaine was based on a cocaine hydrochloride salt that could be administered by nasal inhalation or an intravenous route. The latter caused sudden and immediate euphoria, whereas nasal snorting provoked a less spectacular effect. The consumption of cocaine increased in the form of free-base (crack). Unlike the salt, crack could be smoked, giving an intense and rapid effect with less risk and inconvenience.

Social and economic consequences greatly changed cocaine abuse. It became a silent health problem because of the low incidence of medical problems and the socioeconomic level of its consumers. Its use in young people increased because of changes in its form of consumption and a decline in the price.

Cocaine blocks the reuptake of norepinephrine and dopamine of the sympathetic nervous system. It has a marked psychomotor stimulating effect that provokes euphoria, verbiage, motor activity, and an amplification of the sensation of well-being similar to the effect of amphetamines. With overdose, tremors and seizures may appear followed by respiratory depression with hypotension. Cocaine does not provoke physical dependence but tends to produce depression and dysphoria associated with anxiety for the drug. The pattern of dependence that starts with occasional consumption escalates to dependence that ends in massive abuse.

Well known are the systemic effects[1] of cocaine abuse (acute myocardial infarction, cardiac arrhythmias, rupture of the ascending aorta, cerebrovascular accidents) and also the deleterious local effects on the nasal cavity such as epistaxis, chronic rhinitis, diminished olfaction, and nasal septal perforation.[2]

Although a central midface destructive process can be caused by multiple conditions, it is less known that cocaine abuse can produce a severe destruction mimicking other aggressive pathologic conditions.[3,4] Although extremely rare, this condition should be included in the differential diagnosis of centrofacial destructive diseases in young patients.

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Fig 1. Central palatal fistula and lip splitting at the philtrum with lack of nasal tip support.

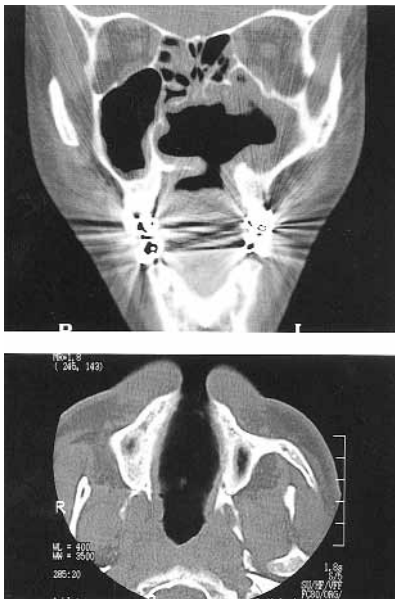

Fig 2. Computed tomography scan on coronal view showing the absence of vomer, nasal septum cartilage, left lateral nasal wall, and the palatal fistula. On sagittal view, the lip splitting.

CASE REPORTS

First case

Patient 1 was a 37-year-old man. He was first seen in a private office complaining of oronasal communication (ONC). On physical exploration, a defect of the labial philtrum, columella, vomer, septal cartilage, turbinates and a 1.5-cm ONC as evident (Fig 1). Crusting and ulcers were seen in this destructed nasal cavity.

The ONC had been present for 1 year. The defect of the labiocolumellar region had been present for 4 months as a result of the application of a topical substance prepared by a pharmacist. He also complained of severe facial pain and needed 6 g ibuprofen daily for analgesia. He was evaluated for anemia and chronic hepatitis C. A history of chronic abuse of cocaine was obtained from his relatives. Serologic test results for human immunodeficiency virus and syphilis were negative. Tests for rheumatoid factors as well as c–antineutrophil cytoplasm antibody (c-ANCA), anti-DNA, and antimitochondrial antibodies were done without pathologic results. Anemia was evident as well as a mild alteration of the hepatic function. A biopsy was performed, with the diagnosis of unspecific ulceration and chronic inflammation. A computed tomography (CT) scan was performed to investigate further the extent of this disease (Fig 2). In a literature search we could not find a similar case.

Treatment initially consisted of an oronasal obturator and lavages with saline solution. He started a program of dehabituation, and 6 months after completely stopping his use of cocaine, the labial defect was surgically corrected. He did extremely well with the palatal obturator but refused further reconstruction (Fig 3).

Second case

Patient 2 was a 35-year-old healthy woman with a history of chronic cocaine snorting who appeared for examination with a large ONC, loss of vomer and nasal cartilage, and a saddle nose deformity (Figs 4 and 5). The medical history revealed that she was treated surgically for a severe cervical and mediastinal nonodontogenic infection 1 year previously. Serologic test results showed an elevation of serum immunoglobulin A; immunoglobulin G anti-cytomegalovirus was highly positive; antinuclear antibody, c-ANCA, anti-DNA, antimitochondrial, luetic serology, and human immunodeficiency virus test results were negative. The biopsy specimen showed severe and chronic inflammation with reactive epithelial hyperplasia. A CT scan was performed (Fig 6). An obturator was constructed, and local measures were performed with lavage of saline solution distilled through her nose.

She was very conscious and concerned about her problem. She abandoned the cocaine addiction and was subsequently treated with an anterior-based tongue flap for closure of the oronasal fistula. A cantilevered calvarial bone graft was performed with a bicoronal approach to correct the saddle nose deformity. This graft was unable to restore the nasal tip prominence because of lack of internal nasal soft tissue coverage that precluded the amount of bone to be transferred. The healing of the tongue flap was uneventful, but two 1.5-mm fistulas remained anteriorly and posteriorly, which will be closed in future interventions (Fig 7).

Third case

Patient 3 was another healthy 30-year-old woman, seen in December 2000. She was seen in a private office, with a complaint of ONC due to a traumatic effect of a periodontal curetting by a dentist 1 year before. Obviously the lesion made by the periodontist was not as severe as her presentation, but the incident was thought to be a precipitating factor. On physical examination, a large 3-cm ONC was evident, with absence of the vomer and nasal cartilage (Fig 8). She had

Fig 3. Results of treatment of the same patient with a prosthetic obturator in place and direct closing with Z-plasty of the upper lip (4 months postoperatively).

Fig 5. Saddle nose deformity caused by the absence of nasal support on the same patient.

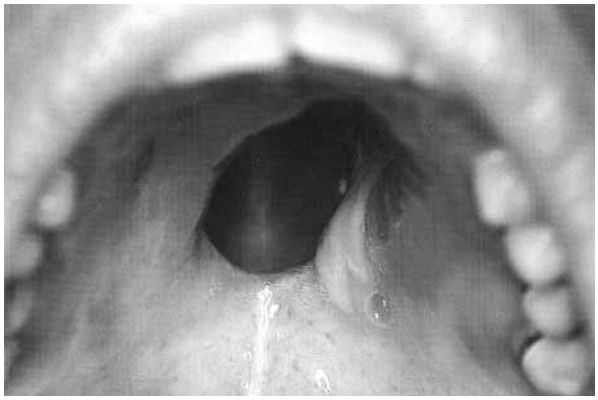

Fig 4. Large palatal fistula of the second patient.

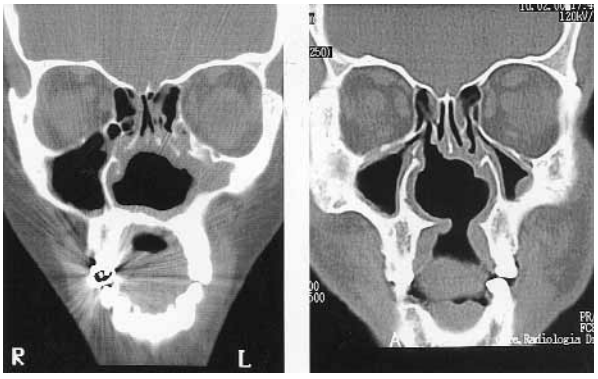

Fig 6. Computed tomography scan on coronal view showing the midline destruction.

a prior habit of snorting cocaine for 4 years (2 grams per weekend) and was a heavy cigarette smoker. The diagnosis of naso-oral destructive process as a result of cocaine abuse was determined. Serologic test results were normal, and CT was performed to assess the extent of the destruction. A biopsy was done, which yielded a diagnosis of chronic nonspecific inflammation. We fabricated a prosthetic obturator and recommended to her to stop her habit before any further surgical treatment was contemplated. The obturator fit extremely well and resolved her nasal speech problem. She did not cease her use of cocaine and subsequently was lost to follow-up.

DISCUSSION

Midline osteocartilaginous necrosis as a result of cocaine abuse was first reported in 1988.[5] Very few cases have been reported in the medical literature to date. In a recent article, Lancaster et al[6] reported a case of centrofacial osteocartilaginous necrosis with oronasal fistula and found 9 more cases in reviewing the medical literature.

Head and neck complications of cocaine abuse are well documented. Nasal septal perforation is the best known complication of nasal cocaine consumption and may be found in about 5% of cocaine snorters.[2] Extensive osteocartilaginous necrosis affecting the nasal cavity is an unusual local effect of cocaine abuse.[7-9] It is our impression that there may be many more cases not yet published because of the low socioeconomic level of these patients, which makes it difficult to get medical care, their avoidance of medical consultations, or because they are not concerned.

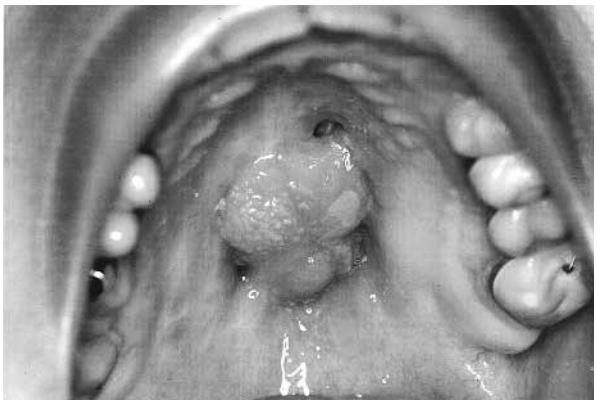

Fig 7. Tongue flap in place to close the palatal fistula. Two 1.5-mm fistulas remain.

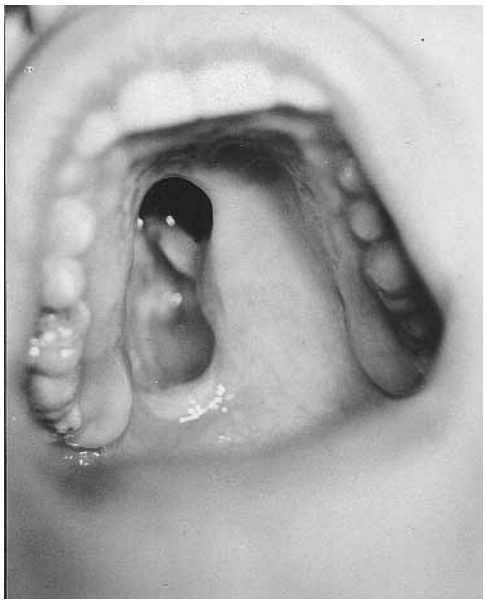

Fig 8. Large lateral palatal fistula of the third patient.

Necrosis in the nasal cavity is probably caused by the added ischemic effect of cocaine or topical decongestant, the caustic effect of the adulterants (talc, amphetamine, lidocaine), and the local anesthetic effect resulting in trauma or thermal injury leading to secondary infections. The systemic effects of the cocaine may further potentiate these local effects.

We recommend performing a local biopsy to rule out malignancy or other pathologic condition before making the diagnosis of cocaine abuse and to allow time for the users to try to cease their cocaine use.

The differential diagnosis should include Wegener granulomatosis.[10] In this disorder, an extremely high specificity of c-ANCA has been documented; characteristic histopathologic features such as epithelioid necrotizing granulomas are also seen. Facial squamous cell carcinoma with its characteristic evolution and histopathology is another possibility. Midline malignant reticulosis (T-cell lymphoma) with its rapid course and dense infiltrate of cells belonging to the lymphoid family, some of them showing moderate immaturity, with a tendency toward vascular orientation on histopathologic features should also be considered.[11] Finally, disorders such as idiopathic midline destructive disease[12] and chronic infections such as actinomycosis, tuberculosis, and syphilis should be ruled out.

The literature had little insight on the treatment of these patients. The management can be difficult because of their noncompliant lifestyles. Our experience could be summarized as follows. (1) Perform a thorough clinical examination and laboratory diagnostic tests to rule out other diagnoses. (2) When the diagnosis of necrosis as a result of cocaine abuse is made, we strongly recommend cessation of cocaine use, otherwise informing them the centrofacial necrosis will persist if they do not. (3) Initial treatment would consist of constructing a prosthetic obturator to improve speech and eating activities; subsequently, the nasal cavity will be irrigated with saline solution to alleviate crusting and mucosity. (4) After compliance with this recommendation and after a period of control ranging from 6 months to 1 year, the patient will then be reevaluated by an anesthesiologist to schedule the surgical correction. This evaluation is done to rule out any systemic condition that could be present in these patients. The primary surgical goals are to close the palatal fistula and to correct any esthetic deformity. This could be done during the same surgical procedure or could be staged, depending on clinical considerations. A good option to close this fistula is the tongue flap, as we demonstrated in our second patient. In other patients a prosthetic obturator may be sufficient. In large defects, such as in the third patient, a microvascular radial forearm flap is another option. Esthetic deformities such as a saddle nose or lip splitting are corrected accordingly. In our experience, if they cease their addiction and are highly motivated, these patients can achieve complete recovery.

REFERENCES

1. Cregler LL, Mark H. Medical complications of cocaine abuse. N Engl J Med 1986;315:1495-500.
2. Schwarz RH, Estroff T, Fairbanks DNF, Hoffman NG. Nasal symptoms associated with cocaine abuse during adolescence. Arch Otolaryngol Head Neck Surg 1989;115:63-4.
3. Deutsch HI, Millard DR Jr. A new cocaine abuse complex: involvement of nose septum, palate and pharynx. Arch Otolaryngol Head Neck Surg 1989;115:235-7.
4. Daggett RB, Haghighi P, Terkeltaub RA. Nasal cocaine abuse causing an aggressive midline intranasal and pharyngeal

destructive process mimicking midline reticulosis and limited Wegener granulomatosis. J Rheumatol 1990;17:838-40.
5. Becker GD, Hill S. Midline granuloma due to illicit cocaine use. Arch Otolaryngol Head Neck Surg 1988;114:90-1.
6. Lancaster J, Belloso A, Wilson A, Path MRC, McCormick M. Rare case of naso-oral fistula with extensive osteocartilaginous necrosis secondary to cocaine abuse: review of otorhinolaryngological presentations in cocaine addicts. J Laryngol Otol 2000;114:630-3.
7. Kurloff DB, Kimmelman CP. Osteocartilaginous necrosis of the sinonasal tract following cocaine abuse. Laryngoscope 1989;99:918-24.
8. Sittel C, Eckel HE. Nasal cocaine abuse presenting as a central facial destructive granuloma. Eur Arch Otorhinolaryngol 1998;255:446-7.
9. Caravaca A, Casas F, Mochón A, De Luna A, San Martín A, Ruiz A. Necrosis centrofacial secundaria a abuso de cocaína. Acta Otorrinolaringol Esp 1999;505:414-6.
10. Mc Donald TJ, De Reimee RA, Kern EB, Harrison EG Jr. Nasal manifestations of Wegener's granulomatosis. Laryngoscope 1974;84:2101-12.
11. Gaulard P, Henni T, Marolleau JP, Haioun C, Henni Z, Voisin MC, et al. Lethal midline granuloma (polymorphic reticulosis) and lymphomatous granulomatosis: evidence for a monoclonal T-cell lymphoma proliferative disorder. Cancer 1988;62:705-10.
12. Tsokos M, Fauci AS, Costa J. Idiopathic midline destructive disease (IMDD): a subgroup of patients with the "midline granuloma" syndrome. Am J Clin Pathol 1982;77:162-8.

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