Palatine perforation induced by cocaine

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ABSTRACT
Worldwide, the use of cocaine has an increased over the years, various secondary effects have been described. Here we present a 48 years old female with a 2-month evolution bucconasal ulcer in the hard palate induced by cocaine usage accompanied by swallow and phonation dysfunctions. Ethiopathogenesis, differential diagnoses and treatment are discussed.

Key words: Palatine perforation, recreational drug use, cocaine, palatine lesions.

RESUMEN
El consumo de cocaína va en aumento en la población mundial, los efectos del consumo de esta droga pueden ocasionar efectos secundarios. Se presenta un caso de paciente femenino de 48 años de edad que presenta una úlcera crónica buco-nasal de 2 meses de evolución, la cual le ocasiona problemas para la deglución y fonación.

Palabras clave: Perforación palatina, consumo de cocaína, uso de drogas, cocaína, destrucción palatina.
INTRODUCTION
Cocaine usage has a growing incidence worldwide. The most common route of administration is nasal that induces local vasoconstriction and irritation on the exposed tissue due to the effect of both the active substance and others added during its manufacture (1-2). The highest risk group are individuals between 18 to 30 years old regardless of gender, socioeconomic status or occupation (2-3). One of the described local complications is ischemic necrosis in the nasal tissue exposed to the drug, which can be followed by septum perforation and palatine perforation caused by necrosis in the bone and cartilage structure sometimes as soon as 3 weeks of intensive exposure (2-15). In a review by Seyer et al in 2002, 7 cases of palatine perforation were described with perforations in nasal septum or paranasal sinus which progressed into hard palate. In these patients usually there were fast destructive processes in the midfacial region, that could suggest other entities with similar lesions, like Wegener’s granulomatosis, NK/T cell lymphoma, infections and other neoplasias, that must be considered in the differential diagnosis (10, 11, 14, 15). Here we describe a case with palatine perforation, nasal septum and maxillary sinus medial wall destruction in a patient with cocaine abuse and discuss possible differential diagnosis.

CLINICAL CASE
48 year old female presents to Maxillo facial surgery service from the Hospital General de Occidente, Secretaria de Salud Jalisco, with a two-month of evolution bucconasal communication. Psychosocial interrogation: married 3 pregnancies. Toxic or recreative drug use denied. Surgeries, transfusions, allergies and hospitalizations denied. Physical examination reveals flattened facial features with wide and depressed nasal dorsum. Intraoral examination asymptomatic hard palate ulcer 15 x 17 mm without inflammation signs. The patient described it appear suddenly and has had an steady slow growth inducing swallowing difficulties due to food passage to the nasal cavities and phonation dysfunction (nasal voice) (Fig. 1). Nasal fossae examination revealed septum nasal of the destruction. Routine Laboratory test, within normal ranges. A head CT scan reported absence of nasal septum with destruction of the left maxillary medial wall (Fig. 2). An incisional biopsy was performed revealing necrotic areas with chronic inflammatory infiltration and squamous metaplasia in several minor salivary glands (Fig. 3 A y B). Based although on the previous data and suspecting a tissue destruction induced by cocaine the patient denied useful drugs, an antidopping test was requested upon sent approval of the patient revealed evidence of cocaine and marijuana usage; a new interrogatory was performed insisting on the use of recreational drugs the patient confirmed the consumption of alcohol intake since the age of 20, marijuana consumption for 10 years and cocaine usage for 1 year with 1 gr. consumption per day. Final diagnosis chronic ulcer due to cocaine intranasal exposure.

Fig. 1. Intraoral photograph showing the hard palate’s ulcer.

Fig. 2. Skull CAT scan, revealing the absence the left maxillary medial wall and destruction of the nasal medial septum.

Fig. 3. A) Photomicroscopic image showing necrotic areas with chronic inflammatory infiltration. B) Photomicroscopic image showing squamous metaplasia in several minor salivary glands.
The patient was treated with a palatine obturator and was
derived to psychological consultation. A surgical fistulæ
closure was advised but the patient did not return for
follow up.

**DISCUSSION**

Cocaine is derived from coca leaves. Its production includes
the transformation of the leaves into a paste and then into
cocaine clorhydrate using catalyst agents like ether, sulphuric
acid and gasoline (1).

Cocaine induces vasoconstriction and might cause necrosis
in the mucosae and surrounding tissues (cartilaginous and
osseous). Frequent contact induces nasal septal destruction,
choanæ, paranasal sinuæ walls and palatine (2).

Ulcerative lesions affecting the midface were considered
for differential diagnosis including traumatic, infectious
and neoplastic, such as Wegener’s granulomatosis, nasal
NK/T cell lymphoma and mucormycosis. Though unfreq-
uent rhinoscleroma and other lymphomas must be also
considerer (2,16)

Wegener granulomatosis causes a necrotic granulomatose
lesion, this is an unfrequent condition that shows respira-
tory tract should granulomatose ulcers, glomerulonephritis
patients develop and in some cases can be developed. This
disease stars with purulent nasal discharge, chronic pain in
maxillar sinu, fever, nasal congestion and nasal ulceration.
Middle ear and epistaxis are also described. 6% of the af-
ected patients have oral involvement. The characteristic
lesion is a granulomatose gingivitis with strawberry ap-
pearance, the tissue turns fragil and erythematous; osseous
destruction and displacement of teeth is also observed.

The diagnosis is base on the clinical and pathological
observations aided by laboratory studies like indirect im-
munoflourecence by the detection cytoplasmic antibodies
against neutrophiles (ANCAs), a useful serologic marker
in this diagnosis (3,9,11,12,16)

NK/T cell lymphoma must be considered when studying
this type of lesions; this lymphoma is common in Asian
population and in some Latinoamerican coountires like Peru,
Guatemala and Mexico, where it has been associated with
Epstein-Barr virus. The affected patients are adults that
develop midface destruction in the nasal area with pain and
epistaxis. The oral cavity shows a rapidly destructive ulcer
in the palate midline inducing a painful buccosinus fistulæ
with halitosis and general malaise. The diagnosis is achived
by accurate clinical and pathological correlation aided by
immunohistochemical studies (3,12).

Mucormycosis is another differential diagnosis. This
infection is caused by fungi in the class of Zygomycetes
which are opportunistic saprophytic fungi from the genres
Absidia, Mucor, Rhizomucor y Rhizopus, that can cause
rhinocerebral affection. This is most commonly observed
in uncontrolled insulin dependent diabetic patients with
ketoacidos and immunosupression.

The usual clinical signs included nasal obstruction, epistaxis,
facial pain, headaché, periorbital and facial swelling with

signs of orbital cellulites, visual, decrease and proptosis and
in some cases facial palsy can be observed. The progres-
sion of the infection without adequate treatment induces
lethargus, blindness, seizures and death. The maxillary si-

nus is commonly involved in alveolar processes, palatine or
both. diagnosis is confirmed by the histological finding of
characteristic non septe irregular hyphae up to 30 micra
in diameter.

We can conclude that intranasal cocaine abuse can induce
necrosis and focal ischemia that causes secondary mucosa
and midfacial bone destruction, and must be differentiated
from aggressive neoplasias, inflammatoty or infectious
processes.
REFERENCES