CASE STUDY

Orbital and Nasal Complications Secondary to Inhaled Cocaine Abuse

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Key words  Midfacial necrosis; Cocaine abuse; Subperiosteal abscess; Optic neuritis

Abstract The abuse of inhaled cocaine causes chemical sinus pathology by secondary midfacial destruction and necrosis. When midfacial necrosis is already established, other complications may occur related to the proximity of structures such as the orbit or optic nerve. We present the evolution of a young cocaine addict with midfacial destruction, who has had a subperiosteal abscess and optic neuritis over the course of the years. Differential diagnosis and management of these complications are also discussed.

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Complicaciones nasales y orbitarias secundarias al abuso de cocaína inhalada

Resumen El abuso de cocaína por vía intranasal provoca una sinusopatía química por destrucción y necrosis mediodfacial secundaria. Cuando esta necrosis mediodfacial ya está establecida, pueden aparecer otras complicaciones relacionadas con la proximidad de estructuras como la órbita o el nervio óptico. Presentamos la evolución de un joven cocalímano con destrucción mediodfacial, que en el transcurso de los años ha padecido un absceso subperióstico y una neuritis óptica. Planteamos el diagnóstico diferencial y el manejo de estas complicaciones.

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Introduction

Powder cocaine or cocaine hydrochloride is the second most consumed drug both in Spain and Europe. The annual prevalence of cocaine powder consumption has increased from 1.6% in 1999 to 3% in 2005 among the Spanish population aged between 15 and 64 years. The most common form of consumption is intranasally or snorted, with the use of crack cocaine being rare. Its use is sporadic.1

The contact of cocaine with mucous membranes causes vasoconstriction and secondary necrosis of the nasal mucosa and supporting tissues. When this contact is frequent and repeated, it can cause destruction and perforation of the nasal septum, choanae, walls of the sinuses and palate. Within the context of this chronic sinusopathy, there are other secondary complications such as those described in this clinical case.

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Clinical Case

We present the case of a 29-year-old male who attended consultation in 2005 due to repeated, self-limited epistaxis. His personal history included mild oligophrenia and snorted cocaine consumption for 17 years.

Nasal endoscopy found a large septal perforation, partial destruction of the inferior turbinates and abundant crusting in the bottom of the nasal fossae and cavum.

A CT scan of the paranasal sinuses conducted in 2006 revealed occupation of the frontal sinuses and ethmoid cells, with partial destruction of the septum, turbinates and medial wall of the maxillary sinuses, as well as mucosal thickening of the latter. There was no involvement of the ethmoid-sphenoid orbital walls. Biopsy of the mucosa in the free perforation edge showed necrotic tissue with acute inflammation and granulation tissue, as well as an absence of malignant cells.

In 2007, the patient attended the emergency service due to palpebral oedema and diplopia in extreme gaze, as well as proptosis, without involvement of ocular motility or visual acuity, of 3 weeks duration. He was admitted to hospital due to suspicion of subperiosteal orbital abscess. We conducted an axial and coronal CT scan of the nasal and paranasal sinuses which revealed a discontinuity in the lamina papyracea of the ethmoid at the level of the left medial orbit, along with the presence of a convex mass of heterogeneous density which included the medial rectus muscle (Fig. 1). Evolution was favourable with intravenous antibiotics and anti-inflammatory treatment, so surgical intervention was not required.

In 2008 the patient returned to the emergency service with symptoms of sudden blindness in the right eye and ocular pain upon its movement. He was hospitalised for empirical antibiotic treatment with cefuroxime sodium at a dose of 750 mg/8 h and steroid treatment with methylprednisolone at a dose of 80 mg/24 h, both intravenously. This time, a brain MRI scan showed total destruction of the septum and turbinates, forming a single nasal cavity. The right sphenoid wing presented erosion/destruction of the optical opening, with enhancement after administration of contrast, along with a change in signal intensity in the prechiasmal portion of the right optic nerve, compatible with retrobulbar optic neuritis (Fig. 2). We performed a nasal endoscopic examination under general anaesthesia in order to rule out a compressive infectious process of mucopyocele or mucocele type. This revealed a scab on the posterior wall of the sphenoid, corresponding to osteitic bone and infiltrate, with mucopurulent exudate (Fig. 3). We did not observe any abscesses which were susceptible of being drained. We maintained a conservative approach due to the risk of causing morbid haemorrhage and/or CSF fistula. The patient gradually recovered vision during postoperative hospital stay and with intravenous antibiotic treatment.

At present, the patient is in treatment for his addiction.

Figure 1  Coronal and axial CT scans showing a left subperiosteal abscess.

Figure 2  Cerebral MRI scan revealing a single nasal cavity.
Conclusions

Occasionally, a history of drug consumption is hidden from the physician. For this reason, a continued abuse of snorted cocaine should be included in the differential diagnosis of the 2 main processes which encompass what was previously known as "malignant granuloma of the midline", namely Wegener's granulomatosis and NK/T cell lymphoma.

On the other hand, it is important to be clear about the approach and treatment of complications of chronic sinusopathy due to cocaine consumption. In the case of orbital abscesses, drainage must take place early, especially if there is loss of vision. In the case of optic neuritis, endoscopic surgery is reserved for patients with compressive disease or those with a strong suspicion of suppurrative sinusitis.

Conflict of Interests

The authors have no conflicts of interest to declare.
Acknowledgements

The authors wish to thank the Head and Neck Unit of the Radiology Service at Hospital Universitario Virgen de la Victoria in Malaga, and in particular Dr. Rafael Aguilar.

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