CASE STUDY

Orbital and Nasal Complications Secondary to Inhaled Cocaine Abuse

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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 mediodifacial secundaria. Cuando esta necrosis mediodifacial 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 cocaínómano con destrucción mediodifacial, 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 intranasal 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.
Discussion

The differential diagnosis of midline destructive processes should include Wegener’s granulomatosis and nasal-type NK/T cell lymphoma.

Wegener’s granulomatosis is a multisystemic disease characterised by the formation of necrotising granulomas, as well as vasculitis of small arteries and veins in the upper and lower respiratory tract and the kidneys. The localised form is the most common, and often the first affected region is the sinonasal (60%–90%). The first sign is usually unilateral, chronic sinusitis with rhinorrhea, nasal obstruction and epistaxis. Later, erosive and crusted sinonasal lesions appear, along with septal perforation and nasal “saddle” deformity. Skin involvement only appears in advanced stages and involvement of the hard palate is rare. Only 50% of patients with localised involvement are ANCA positive, compared to 95% when involvement is systemic. Thus, the anatomopathological diagnosis is crucial, focusing on the presence of palisade granulomas, microabscesses and leukocytoclastic vasculitis.

NK/T cell lymphoma is the most common pathological entity associated with destructive processes of the midline. It begins as nonspecific, unilateral sinusitis which develops into ulcerated, infiltrative and necrotic nasofacial lesions. Destruction of the septum, sinuses and orbital walls, as well as the hard palate is typical in the early stages, unlike in Wegener’s granulomatosis, where it only appears in very advanced stages.

Regarding other destructive diseases of the midline, we must include specific granulomatosis, such as sarcoidosis, and those of infectious-type, such as tuberculosis, leprosy and syphilis. Rhinoscleroma, noma, mucocutaneous leishmaniasis and nasal fungal infections are less common. In our case, all these diseases were ruled out after nasal swab culture and serology.

The complications derived from chronic chemical sinusopathy, such as that caused by cocaine, are based on the anatomical proximity of the paranasal sinuses to structures like the orbit, optic nerve and carotid artery.

Subperiosteal orbital abscesses represent between 10% and 14% of orbital sinus complications. They are located between the medial, lateral, superior or inferior orbital walls and their periostium. In our case, the abscess was located between the medial orbital wall and its periostium. The point of entry originated from bone erosion and necrosis of the ethmoidal orbital plate. The CT scan enabled us to confirm this diagnosis. Medical treatment of subperiosteal abscesses must take place early. Surgical treatment consists in drainage of the abscess by endoscopy or, exceptionally, externally by rhinotomy. The surgical approach was not necessary in our case, since the response to medical treatment was satisfactory.

The most common cause of acute visual loss in young adults is idiopathic optic neuritis, whose pathogenesis is an inflammatory and demyelinating process of the optic nerve. The most common form regarding the location of inflammation is the retrobulbar, in which the appearance of the optical disc is normal, unlike in papillitis in which inflammation is anterior. However, in patients with a history of cocaine use, sinus infection or ethmoidal and/or sphenoid compression by mucocele or mucopyoceles should be ruled out.

There are several pathophysiological pathways whereby sinussitis can cause optic neuritis. The most obvious and most common is direct extension of sinus infection and inflammation to the optic nerve by the passage of pus through dehiscences of the posterior paranasal sinuses or through osteomyelitis in their walls. Another cause of optic neuropathy in cocaine users is severe vasoconstriction of the ophthalmic artery or one of its branches. In our case, the surgery did not reveal any cause of optic canal compression, so, given the presence of a discontinuity of the nasal cavity with the orbit, we accepted direct extension as the most likely pathophysiological mechanism. Medical treatment with antibiotics and a single cleaning of the nasal cavity under general anaesthesia resolved the process. Despite this and as a sequela, an MRI scan showed severe atrophy of the optic nerve.

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 suppurative 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.

References