

# SUBDURAL EMPYEMA AND INTRAORBITAL ABSCESS DUE TO INTRANASAL COCAINE ABUSE

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## ABSTRACT

Sinusitis as a complication of intranasal cocaine abuse is well-known. But concomitant intracranial and intraorbital infections due to intranasal cocaine abuse presented in this paper are not described before. Patient was treated surgically

and appropriate antibiotics were applied. He did well following treatment.

• **Key Words:** Cocaine, subdural empyema, intraorbital abscess *Nobel Med 2010; 6(1): 78-80*

## ÖZET

### BURUN İÇİ KOKAİN KULLANIMINA BAĞLI AMPİYEM VE İNTRAORBİTAL ABSE

Sinüzit, intranasal kokain kullanımına bağlı bir komplikasyon olarak iyi bilinmektedir. Ancak yazımızda belirtilen intranasal kokain kullanımına sekonder intra-

kranial ve intraorbital enfeksiyonların birlikteliği daha önce bildirilmemiştir.

Sunulan hastaya cerrahi drenaj ve uygun antibiyotik tedavisi uygulandı, problemsiz bir iyileşme dönemi gözlemlendi.

• **Anahtar Kelimeler:** Kokain, subdural ampiyem, intraorbital abse *Nobel Med 2010; 6(1): 78-80*

## INTRODUCTION

Intranasal cocaine abuse may cause significant local ischemia of bony and soft tissues around the nasal cavity. These may result in ulcerations and perforations. Orbital and periocular complications from intranasal cocaine abuse are uncommon. These usually occur following a sinus involvement. Contiguous inflammation or infection from the sinuses and destruction of bony

structures provides a conduit for spread of the process. Cases usually require surgical treatment followed by antibiotherapy.

## CASE REPORT

In this article we take the opportunity to report the very first case of concomitant subdural empyema and intraorbital abscess due to long term intranasal cocaine →



**Fig 1.** Bilateral ethmoidal cells with mucosal thickening and purulent collection; remarkable (Grade III) left eye proptosis.



**Fig 2.** Air-fluid level in left superior extraconal retroorbital region with highly dense purulent content and bone defect in the anterior frontoethmoidal recess with accompanying air in neighbouring subcutaneous tissue. Inflammation of cutaneous and subcutaneous tissue is evident.

buse. A 21-year-old man with a 12-year history of psychosis follow-up and 4-year history of cocaine abuse presented to emergency room with headache and loss of conscious. Physical examination was unremarkable except high fever (38,8 degrees C / 101,8 degrees F) and neurological examination showed left eye proptosis, neck stiffness and deterioration. Immediate computed tomography (CT) scans showed left retroorbital soft tissue density with Grade III proptosis, peripherally enhancing left frontotemporal collections, bone defect in the anterior frontoethmoidal recess with accompanying subcutaneous and peri orbital soft tissue edema (Figs 1, 2 and, 3). Patient was operated immediately via left frontotemporal craniotomy and left orbital wall was also partially removed.

Ethmoidal cells were filled with purulent exudate and superomedial orbital wall was destructed. Following dural opening purulent collections were drained and surgical site was irrigated with normal saline. Drainage material was kept for microbiological investigation. The histopathological evaluation of resected material revealed granulation tissue with acute and chronic inflammatory cells, fibrino-purulent exudates, and multinucleated foreign body giant cells. There was no evidence of granuloma formation, vasculitis, or malignancy. Empiric antibiotherapy was started with intravenous piperacillin and tazobactam and lasted 48 hours. Drainage material culture demonstrated growth of erythromycine resistant *Staphylococcus aureus*. Treatment was continued with oral amoxicillin and clavulanic acid for 10 days due to antibiogram profile. Intravenous steroids (methylprednisolone, 8 mg twice daily for 7 days) were also added. Postoperative course was uneventful. After resolving of all symptoms, patient was transferred to a drug abuse and addiction treatment centre.



**Fig 3.** Left frontotemporal peripherally enhancing subdural collections (white arrows) with minimal air content.

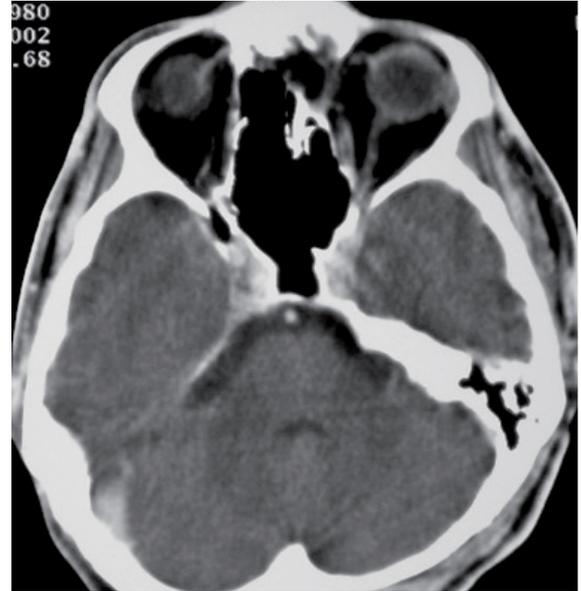
## DISCUSSION

Intranasal cocaine abuse may cause significant local ischemia of sinonasal and midfacial bones and soft tissues, and result in nasal septal perforation or necrosis, osteocartilaginous necrosis, hard palate perforation, and pharyngeal wall ulcerations.<sup>1, 2</sup> Orbital and periocular complications from intranasal cocaine abuse are uncommon. Brain abscess, preseptal cellulitis, limitations in extraocular muscle function, and optic neuropathy were previously reported.<sup>3-5</sup> The exact pathogenic mechanisms that resulted in are not clear; it is possible →

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**Fig 4.** Postoperative CT. Bone defect of medial orbital wall; air in the temporal tip secondary to operation with no subdural collections.



**Fig 5.** Postoperative CT. Proptosis is resolved, subdural collections are drained off ; cutaneous and subcutaneous edema are improved. Bone defect of medial orbital wall is remarkable.

that contiguous inflammation or infection from the sinuses and destruction of bone structures provided a conduit for intra-orbital spread of the inflammatory or infectious process.<sup>3</sup> In addition, vasoconstriction induced by cocaine can also be another factor of bone and soft tissue destruction that lead to intracranial spread of infection.

Cocaine abuse was shown to cause or worsen the present course of HIV infection causing encephalitis in animal studies.<sup>6</sup> This may explain a possible immune compromising effect of cocaine abuse. This is the first reported case of such complex intracranial infection due to intranasal cocaine abuse. Removal of superomedial

orbital wall is essential in such operations. This will ease the orbital approach. Generous irrigation of the operation site is also important to be sure of the total removal of infectious material.

## CONCLUSION

Intranasal cocaine abuse may lead to intraorbital even intracranial infections. Detailed medical history for unconscious patients should be obtained somehow and immediate therapeutic reaction is essential to avoid life threatening complications.

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