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Cocaine-associated Eustachian tube stenosis causing chronic 'glue ear': a rare cocaine-induced destructive lesion

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Abstract

Background. Cocaine is one of the most used recreational drugs. Whilst medical uses exist, chronic recreational nasal use of cocaine is associated with progressive destruction of the osseocartilaginous structures of the nose, sinuses and palate – termed cocaine-induced midline destructive lesions.

Case report. A 43-year-old male with a history of chronic cocaine use, presented with conductive hearing loss and unilateral middle-ear effusion. Examination under anaesthesia revealed a completely stenosed left Eustachian tube orifice with intra-nasal adhesions. The adhesions were divided and the hearing loss was treated conservatively with hearing aids. Whilst intra-nasal cocaine-induced midline destructive lesions are a well-described condition, this is the first known report of Eustachian tube stenosis associated with cocaine use.

Conclusion. This unique report highlights the importance of thorough history-taking, rhinological and otological examination, and audiometric testing when assessing patients with a history of chronic cocaine use. This paper demonstrates the complexity of managing hearing loss in such cases, with multiple conservative and surgical options available.

Introduction

Cocaine is a commonly used recreational drug across the world, with the most frequent method of administration being intra-nasal inhalation.[1,2] Because of its illicit, unregulated nature, it is often mixed with other chemical adulterants during production; these include levamisole, an anti-helminthic agent linked to vasculitides[3] and inflammatory leukoencephalopathy.[4]

Chronic use of recreational cocaine has been associated with progressive destruction of the osseocartilaginous structures of the nose, paranasal sinuses and palate,[1,2] also known as cocaine-induced midline destructive lesions.

We describe the first reported case of a patient with unilateral complete stenosis of the Eustachian tube orifice associated with cocaine use.

Case report

A 43-year-old man was referred to the otolaryngology clinic with a 5-year history of left-sided moderate conductive hearing loss, with a unilateral middle-ear effusion. He had a history of chronic cocaine use, with the last use being four years prior, and had previously been surgically treated for cocaine-induced midline lesions. These involved complete bilateral stenosis of the internal nasal valves and a septal perforation. Surgical treatments previously included the division of adhesions with temporary Silastic™ splinting, followed by a multistage nasovestibular reconstruction.

Otological examination revealed a unilateral left-sided middle-ear effusion. Upon rhinological examination, there was reduced airflow of the left nostril with partial stenosis, leftward caudal septal deviation, and an ongoing septal perforation. No lymphadenopathy was present. Examination of the post-nasal space was not possible in the clinic given the severity of the patient's nasal stenosis. Audiometry confirmed a left-sided moderate conductive hearing loss, with Jerger type B tympanometry.

Based on the patient's previous cocaine use, biochemistry samples were requested. The findings demonstrated a raised erythrocyte sedimentation rate of 20 mm/hour, with normal neutrophil cytoplasmic antibody results (antineutrophilic cytoplasmic antibody (ANCA) myeloperoxidase level of less than 0.3 kU/l, and ANCA serine proteinase 3 level of 1.3 kU/l). All other blood markers, including full blood count, complete blood count with differential, bone profile, thyroid function, renal function, and electrolytes, were within normal limits. Previous skin swabs showed no bacterial or fungal growth. Previous histology of nasal and paranasal tissue showed patchy chronic inflammation, with no evidence of malignancy. A review of previous non-contrast computed tomography scans of the sinuses confirmed left-sided otitis media with mastoid effusion, and ill-defined soft tissue lesions in the left middle, inferior and right middle meatuses, likely

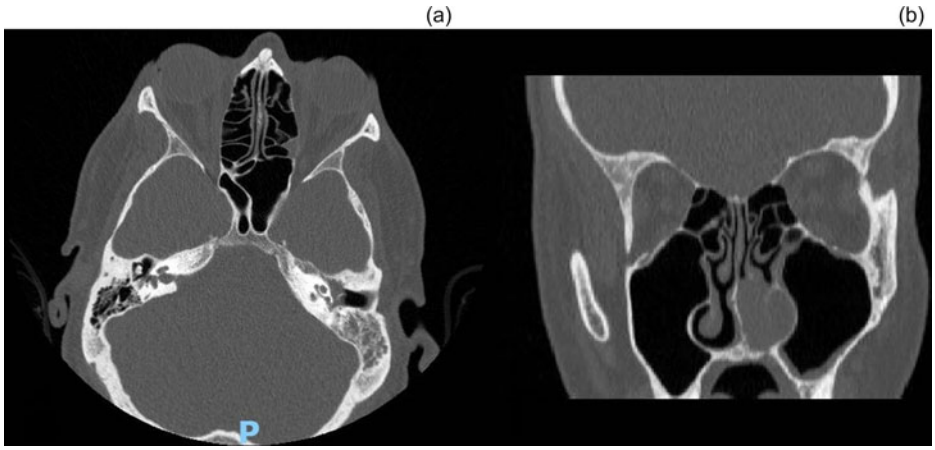

Figure 1. Computed tomography scans of the nose and paranasal sinuses: (a) left otitis media and mastoid air cell effusion, and (b) ill-defined soft tissue lesions of left nasal cavity. P = posterior

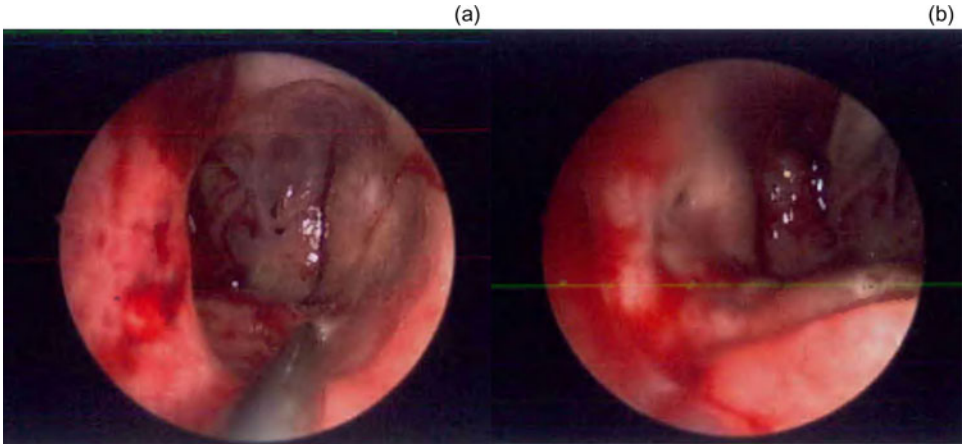

Figure 2. Nasal endoscopic images taken intra-operatively: (a) left Eustachian tube orifice with complete stenosis, and (b) right Eustachian tube orifice with significant narrowing.

secondary to mucus and scarring. No bony destruction was noted (Figure 1). Based on these findings, a surgical plan was made for the insertion of a left-sided ventilation tube and possible Eustachian tuboplasty, following examination under anaesthesia (EUA) of the nose with division of the adhesions.

The adhesions were successfully divided; however, EUA of the post-nasal space showed complete stenosis of the left nasal Eustachian tube orifice and a narrowed orifice on the right (Figure 2). Eustachian tuboplasty was not possible as no entry point was found into the orifice. Given the lack of a functioning Eustachian tube, concern was raised regarding the post-operative risk of developing a chronically discharging ear, if a ventilation tube was inserted. A decision was therefore made against myringotomy and ventilation tube insertion, to avoid this risk. Silastic splints were placed adjacent to the septum bilaterally (to prevent adhesions), and a conservative management plan for hearing aids to address the hearing loss was made post-operatively.

Discussion

Cocaine is an alkaloid extracted from the *Erythroxylum coca* plant leaves. It was first introduced for ophthalmic use in medicine, given its anaesthetic and vasoconstrictive actions, in 1884 by Carl Koller.[5] Cocaine is also commonly used as a topical preparation in nasal surgery, in combination with adrenaline, sodium bicarbonate and sodium chloride.[6] Known as Moffett's solution, this is an effective mucosal decongestant via vasoconstriction, as well as a topical anaesthetic.

Recreationally, its use is attributed mainly to the stimulating action on the central nervous system.[7] It is usually self-administered by intravenous injection, smoking, topical administration (orally via the gums), or most commonly, nasally via inhalation ('snorting').[8]

Given that the most common administration method for recreational cocaine use is via nasal inhalation, the pathophysiology of osseocartilaginous destruction associated with chronic cocaine use is thought to be multifactorial. Primarily, repeated vasoconstriction and trauma to the nasal mucosa by cocaine occurs, which can induce ischaemia and necrosis. There is also an irritant effect from chemical adulterants, as well as bacterial infections of disturbed mucosa and activation of the autoimmune system.[2,9,10] These may subsequently lead to damage of the cartilaginous structures of the nose, most frequently the cartilaginous part of the nasal septum, as well as the surrounding bony structures such as the hard palate, anterior skull base and maxilla.[11,12] This can cause deformities to the external nose and middle third of the face, due to loss of the facial midline support structures.[11,13,14]

This report focuses on a case of unilateral Eustachian tube stenosis associated with nasal cocaine use, something not previously reported in the published literature. A literature search was conducted on 18 September 2023, using the Web of Science, Medline, Cochrane Library and Clinicaltrials.gov databases, for Eustachian tube abnormalities associated with cocaine. Only one paper was found that directly associated a lesion of the Eustachian tube with cocaine use.[15] This was a 2018 case report showing multiple areas of cocaine-associated lytic destruction in the sinonasal complex, including 'erosion and widening of the

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cartilaginous and bony portions of the left Eustachian tube'; however, no stenosis was reported. This occurred in a patient presenting with oronasal reflux, rhinolalia, anosmia and bilateral otitis media.[15]

A 2022 systematic review of the distribution of cocaine-induced midline destructive lesions found that 99 per cent of reported cocaine-induced midline destructive lesion patients had a septal perforation.[16] The prevalence of damage to the inferior third of the sinonasal complex was lower, at 59 per cent in the nasal floor and 30 per cent in the inferolateral nasal wall. The middle third was damaged in 23 per cent (middle turbinate and ethmoid disease), and the lowest prevalence was expectedly seen in the skull base and lamina papyracea at 8 per cent. There were no direct reports of pathology to the Eustachian tubes in this review.[16]

At a microscopic level, cocaine-associated damage shows a vasculitis-like granulomatous pattern, with progressive loss of tissue through ischaemic and thrombotic events.[2] This can be histologically indistinguishable from diseases such as granulomatosis with polyangiitis, sarcoidosis, or non-Hodgkin's lymphoma.[17–19] Therefore, whilst a full investigation is required to rule out other aetiologies for nasal tissue loss, the clinical identification of a cocaine-induced midline destructive lesion is generally reported when a structural lesion of the sinonasal complex is seen in the context of confirmed chronic cocaine snorting.[2] Other diagnostic criteria have also been suggested.[20]

Consideration of managing conductive hearing loss in a case such as this is key, as it was the leading symptom in the patient's presentation. It is well recognised that Eustachian tube dysfunction has implications in the development of chronic otitis media with middle-ear effusion.[21] In this case, following the EUA, it became clear that the complete stenosis evident at the left Eustachian tube orifice was the principal cause of the middle-ear effusion and subsequent conductive hearing loss. Surgical aeration of the middle ear, in cases not responding to conservative or medical therapies, is often the treatment of choice, via myringotomy and ventilation tube insertion.[22] This may have improved this patient's conductive hearing loss; however, otorrhoea and persistent tympanic membrane perforation are known complications, even in routine cases.[23] Given that our patient no longer had a drainage pathway from the left middle ear into the nasal cavity, the risk of otological complications, primarily persistent tympanic membrane perforation with chronic discharge, were greatly increased. This creates a treatment dilemma of two options: ventilation tube insertion to improve hearing loss with the risk of chronic otorrhoea, or conservative treatment to avoid further otological complications but with hearing loss remaining. It is therefore suggested that patients with similar presentations are well-counselled on such risks, and that conservative treatment with hearing devices be considered in the first instance.

This case highlights the rare finding of Eustachian tube stenosis associated with cocaine use. We demonstrate the importance of thorough investigation when a cocaine-induced midline destructive lesion is suspected, including otological and post-nasal space examinations and audiometry, to screen for and diagnose Eustachian tube pathology. We also highlight the complexities associated with managing such cases, particularly when concurrent otological symptoms are present. Surgical decision-making is difficult and revision surgery is often necessary. Whilst this patient's hearing loss was treated medically, decisions must be made on a case-by-case basis, with other options including Eustachian tuboplasty or myringotomy and ventilation tubes.

- Chronic recreational cocaine use is associated with progressive destruction of the osseocartilaginous structures of the nose, paranasal sinuses and palate
- This paper describes a case of cocaine-induced midline destructive lesion causing complete stenosis of the Eustachian tube, with subsequent middle-ear effusion and conductive hearing loss
- Only one previous case report associating cocaine use to Eustachian tube widening was found, with no previous reports of Eustachian tube stenosis
- Pathophysiology of osseocartilaginous destruction is thought to be multifactorial; vasculitides have also been implicated
- The case report highlights the complexity of management in cocaine-induced midline destructive lesion cases
- Long-term otological complications should be considered when surgically treating middle-ear effusions with hearing loss in patients with non-functioning Eustachian tubes

Conclusion

Cocaine-associated Eustachian tube stenosis, as seen in this case, represents a previously unreported type of destructive lesion that can form part of the cocaine-induced midline destructive lesion clinical picture. This can have implications in terms of middle-ear effusions and conductive hearing loss. This highlights the importance of thorough history-taking, including drug history in these cases, as well as adequate assessment of the ears and the post-nasal space – both visually and by computed tomography – particularly in patients who may exhibit difficult-to-access nasal cavities due to scarring and adhesions.

Competing interest. None declared

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