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Acute angle closure glaucoma precipitated by intranasal application of cocaine

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Abstract
We describe a patient who developed acute angle-closure glaucoma following the application of topical intranasal cocaine. A 46-year-old woman underwent an elective antral washout under general anaesthesia and with local application of 25 per cent cocaine paste to the nasal mucosa. Twenty-four hours post-operatively the patient developed sudden painful blindness which was found to be due to acute glaucoma. Cocaine with its indirect sympathomimetic activity causes mydriasis, that can precipitate acute angle-closure glaucoma in predisposed individuals with a shallow anterior chamber. Although the incidence is rare, otolaryngologists need to be aware of this potential complication.

Key words: Cocaine; Anaesthesia, local; Glaucoma, angle-closure

Introduction

The clinical use of cocaine in medicine was reported in 1884 by Carl Koller (a colleague of Sigmund Freud), who introduced cocaine into ophthalmology as a local anaesthetic (Bailey, 1996). In 1947 Moffett described a method of providing local anaesthesia of nasal mucosa using a solution of cocaine hydrochloride, adrenaline and sodium bicarbonate (Moffett, 1947). Cocaine has been readily adopted for clinical use by otolaryngologists because of its rapid absorption and onset of action, excellent anaesthetic properties, and its added vasoconstrictive action decreasing blood loss. Today, cocaine remains the most popular topical anaesthetic for procedures in the nasal cavity. Cocaine is the only local anaesthetic that is known to interfere with the uptake of noradrenaline by adrenergic nerve terminals. The blockage of the uptake of noradrenaline leads to increased levels of circulating catecholamines and produces sympathetic nerve stimulation.

Most of the reported complications of cocaine are related to its illicit use. The systemic effects of cocaine are mainly central nervous system stimulation and the indirect activation of the sympathetic nervous system. Acute angle-closure glaucoma is a multifactorial disease for which the risk factors include genetic predisposition, female gender, shallow anterior chamber depth, increased lens thickness, smaller corneal diameter and increasing age (Fazio *et al.*, 1985). In a person with anatomically narrow angles, angle-closure glaucoma may be precipitated by certain drugs (e.g.: anticholinergics, sympathomimetics or antihistamines) or activities that stimulate the sympathetic nervous system (Moroi and Lichter, 1996). We report the case of a 46-year-old lady with a known shallow anterior chamber, who developed angle-closure glaucoma following the topical use of cocaine in the nose.

Case report

A 46-year-old Caucasian lady was admitted for elective right antral washout and submucous diathermy to the inferior turbinate as a day-case. Her main complaints were of foul smelling discharge from the right nostril and post-nasal drip. She had undergone right upper molar tooth extraction under general anaesthesia three months previously and had also suffered with ulcerative colitis, hiatus hernia and migraine in the past.

After induction with propofol and atracurium, isoflurane was used for maintenance. The nasal mucosa was painted with 25 per cent cocaine paste which was applied with a cotton applicator on to the inferior turbinate and the meatus. Approximately 200mg of cocaine hydrochloride was used for this. Antral washout was purulent and was sent for culture and sensitivity, which subsequently grew *Bacteroides* sp. The patient went home the same evening and was feeling well until 24 hours later, when she developed a severe headache which was mainly localized around the right eye, with haloes and blurring of vision on the same side and associated nausea and vomiting. This, she thought was an attack of migraine and she took her usual medication, Migraleve (a combination of buclizine hydrochloride, paracetamol and codeine phosphate). Although the medication relieved the pain to some extent, the blurring of vision and haloes persisted. The next day, when the patient woke up, she had lost the vision in the right eye almost completely.

The patient was readmitted and further examination of the right eye showed marked conjunctival congestion, corneal oedema, fixed mid-dilated pupil and vision reduced to counting fingers at two feet from the eye. There was no evidence of proptosis and the other eye was normal except for a sensation of pressure. An ophthalmologist's opinion was sought and who made a diagnosis of acute angle-closure glaucoma. Intraocular pressure on the

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right side was 50 mmHg and on the left side was 17 mmHg, the anterior chamber being very narrow. On close questioning it became clear that the migraine that she had suffered for many years was in fact subacute attacks of glaucoma. She admitted that the usual attack of migraine consisted of a sensation of pressure around the eyes with blurring of vision, that was relieved by analgesics and sleep. The patient was transferred to the ophthalmology ward and was treated with acetazolamide intravenously and pilocarpine eye drops. The intraocular pressure was lowered and the vision in the right eye improved subsequently. She later underwent Nd:YAG laser iridotomies on both eyes and has been doing well since. She has not suffered any 'migraine' attacks since.

Discussion

Toxic reactions to cocaine in a clinical setting are due to overdose (actual or relative) or allergy. Many references state that a safe maximal limit for cocaine is 200 milligrams, although the British National Formulary (BNF) recommends a dose of 1.5mg/kg body weight (BNF, 1998). Systemic absorption does occur when cocaine is applied to mucosal surfaces. Peak serum levels of 120–474 nanograms/ml occur 15–60 minutes after intranasal application of 1.5 mg/kg of cocaine, and may persist for four to six hours (Javaid *et al.*, 1978). Serum half life ($t_{1/2}$) is approximately 60 minutes (Wilkinson, 1980). The drug is no longer used in the eye because of clouding and possible ulceration of the sclera and the cornea.

The systemic toxicity of cocaine is due to adrenergic overstimulation of the central nervous system, the cardiovascular system and the respiratory system. Cocaine produces mydriasis by blocking the reuptake of norepinephrine at the adrenergic nerve terminals. Mitchell and Shwartz (1996) reported a case of acute angle-closure glaucoma associated with ipsilateral intranasal cocaine abuse. Our patient also developed glaucoma on the same side of cocaine application. It is possible that some of the cocaine would have travelled in a retrograde manner along the nasolacrimal duct. Orlick *et al.* (1990) found significant reflux along the nasolacrimal duct in their studies in patients who received aerosolized fluorescein intranasally. This route of absorption would have potentiated the effects produced by the systemic absorption of cocaine, leading to a greater degree of mydriasis on the right eye in our patient. Negative pressure caused by sniffing is another possible way of entry into nasolacrimal duct. Cocaine being a potent vasoconstrictor limits its own absorption. Cocaine is detectable in the nasal mucosa three hours after application (Wilkinson *et al.*, 1980), so late absorption is possible. The mydriasis caused appears to have precipitated the angle-closure event. One more reason for the selective affection of the right eye is the more narrow angle. Examination of our patient's eyes showed narrow angles on both sides, but worse on the right side. The fact that our patient was suffering from subacute attacks of glaucoma would have contributed to the sudden deterioration. The false impression of migraine by the patient caused a degree of delay in the diagnosis.

Acute angle-closure glaucoma is a sudden and serious ocular condition in which there is obstruction of the trabecular meshwork by the iris resulting in decreased aqueous outflow and elevated intraocular pressure. Exposure to dimlight, emotional upset, and use of pharmacological agents with anticholinergic or sympathomimetic properties all result in pupillary dilatation and potential closure of the anterior chamber angle (Skuta, 1994). Acute angle-closure glaucoma associated with surgical anaesthesia has been reported previously in the literature. Most cases are attributed to the usage of parasympatholytic agents, such as atropine which causes mydriasis by a direct effect. In our patient no such mydriatic drugs were used other than cocaine. Fazio *et al.* (1985) reported nine cases of angle-closure glaucoma occurring after spinal or general anaesthesia, among 913 records reviewed. Of these nine cases, two were bilateral and he suggested that drug induced mydriasis may have precipitated this complication.

Since angle-closure glaucoma is a serious and relatively unrecognized complication of surgical anaesthesia, we feel that that all surgeons and anaesthesiologists should be aware of this complication, as diagnostic delay may compromise the prognosis for vision.

Conclusion

Cocaine is routinely used in otolaryngological procedures and is considered a safe drug in this context. However, this does not mean that it is safe in all patients. A rare occurrence of acute angle-closure glaucoma secondary to intranasal application of cocaine is described. We emphasize that all head and neck surgeons should be aware of this potential complication so that the drug may be used with caution in high-risk patients.

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