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LOSS OF VISION FOLLOWING A DENTAL PROCEDURE- A CASE STUDY

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ABSTRACT

Nowadays intraoral local anaesthesia has been widely used to perform various dental procedures. The complications associated with it are very minimal though some of the commonly seen ocular complications include meiosis, nystagmus, diplopia, ptosis, amaurosis. In our case study a 30 year old female came to eye OPD with complaints of reduced vision. She had undergone a dental procedure which was done under intra oral local anaesthesia one week prior, following which her vision started deteriorating. Though various mechanisms have been suggested for the cause of diminution of vision post intra oral local anaesthesia, the exact aetiology still remains unknown. In our study intravascular injection of epinephrine which led to a cilioretinal artery occlusion could be the possible cause. The patient was followed up regularly and four weeks later she had recovered complete vision successfully.

KEYWORDS: Intraoral local anaesthesia, arterial occlusion.

INTRODUCTION

In order to perform painless dental procedures intraoral local anaesthesia was very widely used. Rarely the administration of a local anaesthesia intra orally can cause ocular complications. It is noted that complications like ptosis, meiosis, amaurosis, diplopia have occurred following therapeutic injections given around the nose, lips and eye.

The first case of blindness following intranasal injection of warm paraffin was noted in the early 90's and reported by Sir Von Bahr. Many cases of decreased vision have been reported following a maxillary nerve block. However in most of the cases the symptoms developed immediately after the administration of the drug and lasted for a few hours. A case of complete blindness following the injection is extremely rare. Though various theories have

been proposed to better understand the cause of ocular complications post intraoral local anaesthesia, the exact aetiology still remains unclear.

CASE REPORT

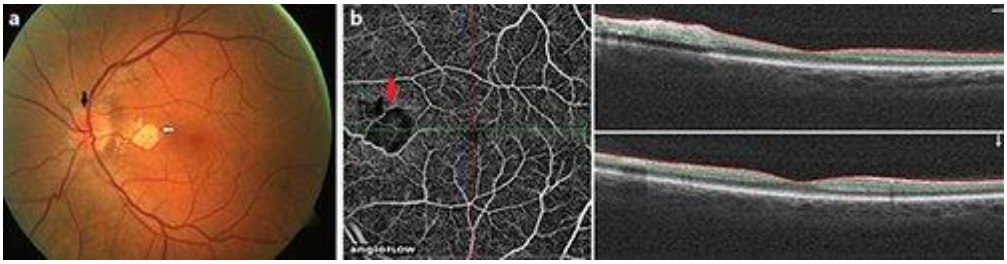
A 30 year old female came to eye OPD with complaints of reduced vision the left eye since 1 week. On further investigation it was found that she underwent a dental procedure of the left upper maxillary canine exactly one week ago. She was given intraoral local anaesthesia of lignocaine hydrochloride and epinephrine(1:100,000)prior to the procedure. Immediately after the procedure the patient started developing a headache and a gradual loss of vision. She also complained of seeing flashes of light occasionally. There was no history of nausea or photophobia and phonophobia. No significant medical history or surgical history or any previous ocular surgeries or trauma in the past.

On examination the patient's vision was 20/25(OS) and 20/20 (OD). The intraocular pressure was measured with applanation tonometer and was 12 mmHg in both eyes. Colour vision was slightly abnormal in the left eye. A relative afferent pupillary defect was noted in the left eye. The extra ocular movements were full in all directions with no evidence of internuclear ophthalmolegia. There were no signs of inflammation, no cells in the anterior chamber or vitreous.

The visual field test was done and it showed a relative paracentral scotoma in the left eye and normal field in the right eye. A dilated fundus examination was done and it showed swelling of the optic disc along with cotton wool spots, hard exudates near the papillomacular bundle. The right eye fundus was normal. Optical coherence tomography was done (OCT) and it showed hypo perfusion in the superficial and deep retinal capillary plexus in the affected areas.

Figure a: Fundus picture of the left eye showing a swollen optic disc with blurred disc margins along with cotton wool spots near the macular region.

Figure b: optical coherence angiogram showing capillary dropout along the superficial and deep retinal plexus in the macular region.



A complete blood examination was then done and it normal. The C reactive protein, glucose, liver function test, renal function test, cholesterol, ESR was well within the normal limits. No past history of any cardiovascular diseases.

A probable diagnosis of cilioretinal artery occlusion was made. The patient was started on 100 Mg of aspirin as a prophylactic measure and was asked to come for a follow up after 4 weeks. The patient showed a significant improvement and the vision improved to 20/20. Fundus examination showed a normal optic disc with very few hard exudates. OCT was also done and was normal except for a thinning of the outer retinal layer near the papillomacular bundle.

DISCUSSION

The cilioretinal artery arises from the short posterior ciliary artery. It is reported to be present in 50% of the eyes and can vary in size, number, distribution and point of origin. Isolated cilioretinal artery occlusion can occur but has a very good prognosis with 90% of the eyes returning to a 6/12 vision and over 60% returning to 6/6. In cases of retinal vascular occlusion, the presence of a cilioretinal artery can play a vital role as they supply the papillomacular bundle and macular region of the retina.

Epinephrine is very commonly used in dental procedures. Epinephrine causes vasoconstriction and provides a bloodless field for the procedure and also prolongs the duration of the anaesthetic effect. Epinephrine when it reaches the orbit can cause vasoconstriction of the ophthalmic and ciliary arteries. But the cilioretinal arteries are more vulnerable due to their small diameters.

The presence of hard exudates led us to a differential diagnosis of malignant hypertensive retinopathy. The clinical presentation of hypertensive retinopathy is usually bilateral and unilateral cases are very rare. However vessel changes such as arteriovenous nipping, silver

wiring typical of hypertensive retinopathy were absent. Clinically the blood pressure was well within the normal limits.

Presence of emboli was another differential. In such cases permanent visual loss can occur after an arterial occlusion due to emboli. In our case the patient started developing the symptoms around 5 to 7 minutes after the injection but she started improving gradually with 20/20 at the end of 4 weeks. A complete cardiovascular evaluation was also done and nothing significant was detected.

There was no history of previous attacks of migraine in the past or a similar family history. During migraine the vasospasm can also lead to a permanent retinal artery occlusion.

A number of theories were put forward to understand the mechanism by which the anaesthetic agent reaches the orbit. Some of them being diffusion, inadvertent needle penetration of the orbit, venous injection and retrograde arterial injection. One of the studies showed that retrograde flow of the epinephrine through the maxillary artery was one of the causes for reduced vision.

In our study it is considered that the inadvertent administration of epinephrine led to vasospasm and insufficient blood flow to the cilioretinal artery causing a transient loss of vision. The onset of action of epinephrine is between 1 to 3 minutes and our patient started developing the symptoms within 5 to 7 minutes.

CONCLUSION

Vasoconstriction of the cilioretinal artery following the injection of epinephrine is a very dreadful complication. In order to avoid such complication, it is important to aspirate before injection, or inject small quantities of the drug along with moving the needle while injecting to avoid accumulation of large quantities of epinephrine at one particular site. Hence it is important hat clinicians should be aware of this complication and take the necessary precautions.

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