Case Report

Unilateral Vision Loss after a Dental Visit

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Abstract
Intraoral local anesthetics are widely used for performing painless dental treatments; however, in some cases, they may cause ocular complications such as meiosis, diplopia, nystagmus, ophthamoplegia, ptosis, and amaurosis. Mostly, the symptoms disappear after several hours; rarely, they have a prolonged character. We describe the case of a 38-year-old young man who had reduced vision in the left eye 5 days after having received intraoral local anesthesia. A diagnosis of cilioretinal artery occlusion with optic disc swelling was made. Ten weeks later, the patient’s visual acuity had increased to 20/20, and the swelling of the optic disc had subsided. Although various possible mechanisms for ocular complications after intraoral local anesthetic administration were suggested in the literature, the exact etiology remains unclear. In this case, inadvertent intravascular injection is believed to be the cause.

Introduction

Intraoral local anesthetics have been used for performing painless dental treatments for a long time. Rarely, the administration of a local anesthetic can cause ocular complications
including meiosis, diplopia, nystagmus, ophthalmoplegia, ptosis, and amaurosis [1]. These complications may follow therapeutic injections around the eyes, nose, and lips. Von Bahr [2] reported the first case of blindness following an intranasal injection of warm paraffin in the early 1900s. Since then, many cases of ocular complications have been reported. The majority of the reported cases followed a maxillary nerve block [3]. In most reported cases, the symptoms developed immediately after local injection and persisted for less than several hours. The occurrence of total blindness after local anesthetic administration is extremely rare [4]. However, it is an alarming complication for both the patient and the physician. Although several cases of ocular complications after dental anesthesia have been reported, the mechanism remains unclear. This case may provide another hint about its etiology.

Case Report

A 38-year-old man was referred to the Department of Ophthalmology for consultation with reduced vision in the left eye 5 days after having received intraoral local anesthesia. The patient had received local infiltration anesthesia as part of treatment of dental caries at the upper left maxillary canine (cuspid). Aspiration was performed through a self-aspirating injection syringe (25 G, 0.6 × 25 mm) before the injection. Articaine hydrochloride and epinephrine 1:100,000 were injected into the left upper jaw. Shortly after the injection, the patient had a headache followed by a slight decrease in visual acuity, which gradually worsened after the procedure. The patient reported occasional flashing stars in the left visual field, but no other visual symptoms. He did not experience nausea or phonophobia. There was no periapical infection associated with the anesthetized tooth. The patient also had no significant medical history and no history of surgery or trauma. It is important to mention that it was not the first time that the patient received infiltration anesthesia. In July 2013, he had received infiltration anesthesia in the right upper jaw without any complications. The patient had not experienced any previous ophthalmic problems. His best corrected visual acuity 5 days after the dental treatment was 20/25 OS and 20/20 OD (normal value 20/20). The intraocular pressure on tonometry was 11 mm Hg in both eyes.

A relative afferent pupillary defect was present. The patient had no manifest or latent deviation. His ocular range of movement was normal with no evidence of internuclear ophthalmoplegia. The results of color plate testing were slightly abnormal in the left eye. The visual field test showed a relative paracentral scotoma in the left eye with a normal visual field in the right eye.

Ophthalmoscopic examination showed a swelling of the optic nerve (Fig. 1a, black arrow) as well as cotton wool spots (Fig. 1a, white arrow). Moreover, there were hard exudates in the papillomacular bundle. We did not find any pathology in the right eye. Optical coherence tomography (OCT) angiography showed hypoperfusion within the superficial and deep retinal capillary plexus in the affected area (Fig. 1b, red arrow). No preexisting cardiovascular disorders or any evidence of emboli were reported. The initial blood examination revealed a normal blood picture. There was no evidence of polycythemia or thrombocytosis. Also, the values of C-reactive protein, glucose, a liver function test, cholesterol, and the erythrocyte sedimentation rate were within normal limits.

A diagnosis of cilioretinal artery occlusion with optic disc swelling was made. Since the patient refused any invasive method of examination, a retinal angiography was not performed at the initial visit. We would predict some delayed filling and emptying of the artery.
on angiography. We started the therapy with 100 mg aspirin as prophylaxis and transferred the patient to an internist to evaluate any cardiovascular risk factors.

Ten weeks later, the patient’s visual acuity had increased to 20/20, and the swelling of the optic disc had subsided (Fig. 2a). The subsequent OCT angiogram was within normal limits as well. Although OCT showed thinning of the outer retinal layer in the papillomacular bundle (Fig. 2b, white arrow) and vitreous opacification, no retinal pigment epithelial changes were seen. There were no clinically visible signs of inflammation and no cells in the anterior chamber or vitreous. Because of the vitreous opacities on OCT 10 weeks after the incidence, we conducted fluorescein angiography, which showed no pathological changes. No hyperfluorescence was detected around the optic disc, nor any signs of retinal vasculitis.

Three months later, the patient was asymptomatic and had a regular visual field and full visual acuity of 20/20.

Discussion

Miller (1856) and Loring (1872) were the first to discover the cilioretinal arteries, and they described them in 3 cases. The cilioretinal arteries supply a specific and localized area of the retina. They were first demonstrated histologically by Nettleship (1877), who described them as communications between ciliary and retinal vessels [5]. The use of epinephrine in dental procedures is very common. Epinephrine counteracts the vasodilatory effects of a local anesthetic, reducing systemic absorption and toxicity, prolonging the duration of its effect, and providing a bloodless field for surgical procedures [6–8].

Epinephrine that reaches the orbit can induce vasoconstriction of the ophthalmic or ciliary arteries. Furthermore, cilioretinal arteries are more vulnerable to vasoconstriction by epinephrine than are ophthalmic arteries, because of their smaller diameter compared to the central retinal artery [9]. On the other hand, neurotoxicity has been reported as a side effect of articaine, caused by neural ischemia due to the vasoconstriction induced by articaine itself or by epinephrine [10].

Although it is unusual to find hard exudates in a case of cilioretinal artery occlusion, malignant hypertensive retinopathy should be considered as one of the differential diagnoses in this case. The clinical presentation of hypertensive retinopathy is usually bilateral. One case of unilateral malignant hypertensive retinopathy, which is a rare incidence, was described [11]. We did not see any vessel changes related to hypertensive retinopathy, such as arteriovenous nipping or silver wiring. In addition, the blood pressure was normal, and there was no history of hypertension. The majority of the case reports stated that the time of onset was within 5 min and the effect lasted no more than 6 h [3]. In this case, the onset of symptoms was noticed 3–5 min after the local injection and lasted for several days. However, we found no evidence of emboli – which is an important differential diagnosis in this condition, since permanent loss of vision usually occurs after arterial occlusion in a case of emboli. In 1 reported case with total blindness after a maxillary nerve block, the causative agent was attributed to embolization of the material injected [12]. In our case, the symptoms started to improve gradually, with complete recovery of vision at the end. No embolic risk factors were present in this case. Nevertheless, consulting an internist for the evaluation of cardiovascular risk factors is highly recommended to such patients.

Permanent retinal artery occlusion due to vasospasm during a migraine attack has already been documented [13–15]. Our patient denied any previous history of migraine attacks or a family history of similar conditions.
Many theories attempting to explain the possible mechanism allowing an anesthetic agent to reach the orbit have been proposed in the literature; these include diffusion, inadvertent needle penetration of the orbit, venous injection, hematoma formation due to trauma, and retrograde arterial injection [16]. Retrograde flow of epinephrine through the maxillary artery into the ophthalmic artery was also described as one of the leading causes of blindness [17].

In our case, we believe that inadvertent intravascular injection of epinephrine was the cause of vasospasm and blood flow insufficiency followed by transient visual loss. The presence of vitreous opacification 10 weeks after the incidence made uveitis an important differential diagnosis. However, the lack of symptoms, the absence of clinical signs in the anterior chamber, the lack of vitreous inflammation, and the normal fluorescein angiogram did not point to uveitis. We found no support for the diffusion theory, because the onset, course, and duration of the symptoms were close to those of epinephrine (the onset of action of epinephrine is between 1 and 3 min). In our case, the patient first presented to the ophthalmic clinic 5 days after this incidence. In this sense, gaining visual acuity in the acute phase would support our theory.

**Conclusion**

Vasoconstriction secondary to epinephrine may result in cilioretinal artery occlusion. It is of great importance to be aware of this complication. The following steps can be taken to avoid such complications, including aspiration before injection, slow injection of small quantities (if possible without epinephrine), and moving the needle during injection to avoid injecting a large bolus of epinephrine in one location. This case report expands the number of cases with visual loss after the administration of local anesthesia in the facial area. Clinicians should be aware of this severe ocular complication.

**Statement of Ethics**

This case report was conducted according to good clinical practice. The authors state that they have full control over all primary data and have no ethical conflicts to disclose.

**Disclosure Statement**

The authors have no financial disclosures and no commercial or proprietary interest in any materials discussed in this report.

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**Fig. 1.** 
**a** Fundus photograph of the left eye showing a swelling and blurred margin of the optic disc (black arrow) associated with cotton wool spots in the papillomacular bundle (white arrow). 
**b** Optical coherence tomography angiogram showing an area of capillary dropout within the superficial and deep retinal capillary plexus in the papillomacular bundle (red arrow).
Fig. 2. a Ten weeks later, a fundus photograph of the left eye shows a normal optic disc margin. b The optical coherence tomography scan demonstrates thinning of the outer retinal layer in the papillomacular bundle (white arrow).