A Case of Branch Retinal Artery Occlusion following Uneventful Phacoemulsification

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Key Words

Phacoemulsification · Sub-Tenon's anaesthesia · Branch retinal artery occlusion

Abstract

We would like to present a case of branch retinal artery occlusion following uneventful phacoemulsification, possibly caused by sub-Tenon's anaesthesia. There were no predisposing general health problems. There are two possible mechanisms: (1) mechanical effect of the bolus anaesthetic; (2) pharmacologically mediated changes in the vascular calibre. The latter mechanism is much more probable, because of the vasoconstrictive properties of both medications used. This is the first reported case of branch retinal artery occlusion after sub-Tenon's anaesthesia with preservative-free medications.

Case Report

A 74-year-old lady presented to our clinic complaining of progressive blurred vision more in the right eye (RE) compared to the left eye (LE), over the last 6 months.

There was no past ocular history of note. Past medical history showed transsphenoidal pituitary surgery (16 years ago), acromegaly, well-controlled hypertensive disease and hyperlipidaemia. On routine follow-ups and MRI scan after the neurosurgery, no recurrence or abnormalities in the visual field were noted. Her medical treatment was tab. perindopril erbumine 4 mg, tab. bisoprolol 2.5 mg, tab. oxybutynin 2.5 mg, tab. doxazosin 8 mg, tab. aspirin 75 mg, tab. simvastatin 40 mg. She is allergic to penicillin.

Ophthalmological examination showed best corrected visual acuity (BCVA) 0.5 (6/12; 20/40) in the RE and 0.7 (6/9; 20/30) in the LE. Pupillary reactions were normal, intraocular pressures were 19 mm Hg in both eyes. Anterior segment was normal with exception of

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nuclear cataracts in both eyes (BE), more pronounced in the RE compared to the LE. She had bilateral posterior vitreous detachment, but fundus examination was otherwise normal. The patient was booked for RE phacoemulsification under local anaesthesia.

Preoperatively g. tropicamide 1%, g. phenylephrine 2.5%, g. cyclopentolate 1% and g. oxybuprocaine 0.4% were instilled in the RE. Sub-Tenon's anaesthesia with lidocaine 2% (2 ml), bupivacaine 0.5% (3 ml) and hyalase was performed in the lower nasal quadrant of the RE. No Honan's balloon or digital massage was used. No proptosis or other signs of retrobulbar haemorrhage were noted. Uneventful phacoemulsification with implantation of posterior chamber intraocular lens was performed by an experienced surgeon (consultant grade) in less than 15 min. During the surgery the height of the irrigation bottle was 70 cm. Intracameral cefuroxime 1 mg and subconjunctival mixture of cefuroxime and Betnesol subconjunctivally were injected at the end of the procedure. The eye was left with normal intraocular pressure (IOP) after the closure of the wound. Postoperatively g. Pred Forte 1% q.d.s., g. ciprofloxacin 0.3% q.d.s. for 1 month and tab. acetazolamide 250 mg once in the night after the surgery were prescribed. During the procedure the highest level of blood pressure recorded was 135/95 mm Hg and all other parameters monitored were within normal limits (pulse, saturation, respiration).

On the 1st postoperative day visual acuity (VA) in the RE was counting fingers (CF). There were a few Descemet folds in the cornea, mostly around the incision site. No anterior chamber cells were noted. IOP in the RE was 22 mm Hg. Central scotoma in the RE was noted clinically. On the 6th postoperative day VA in the RE was still CF with no pinhole improvement. Anterior segment was normal with exception of the intraocular lens present. Both pupils had brisk reaction to light and no relative afferent papillary defect was noted. On standard slit lamp fundoscopy the fundus looked normal (fig. 1a). Optical coherence tomography scan of the RE showed increased reflectivity of the inner retinal layers and hyporeflexivity of the outer retinal layers (fig. 1d). These findings were consistent with retinal artery occlusion.

On the 9th postoperative day visual field investigation was performed and it showed central scotoma in the RE, occupying 5 degrees (fig. 1c). VA in the RE was unchanged. Standard slit lamp fundoscopy showed absence of foveal reflex, but no other major abnormalities. The same investigation performed with red-free filter showed pronounced macular nerve fiber layer oedema with relative foveal sparing in the RE (fig. 1b). On the fluorescein angiography performed on the same day, a small retinal artery branch, crossing the horizontal raphe, with very irregular lumen was noted. This same branch was supplying blood to the affected macula. No embolus was noted. Autorefractometry results showed $-0.50/+1.25/6^\circ$ D of postoperative refraction. Doppler ultrasound of the carotid arteries was normal.

Surprisingly the BCVA in the RE improved to 0.3 (6/18; 20/60) and 0.5 (6/12; 20/40) on the 2nd and 4th week after the surgery, respectively. A small central scotoma persisted.

Discussion

There are several case reports, describing central artery occlusion (CRAO) after cataract surgery, vitrectomy, pterygium excision and other ophthalmic procedures. All these procedures were performed under local anaesthesia, particularly retrobulbar, parabulbar or sub-Tenon's anaesthesia.

It is thought that the cause of these CRAO cases is the local anaesthetic. There are different possible mechanisms: (1) mechanical effect of the bolus anaesthetic; (2) pharmacologically mediated changes in the vascular calibre.

The mechanical effect of bolus anaesthetic may cause elevation of IOP or direct pressure over the vessels. Several studies done on the effect of sub-Tenon's anaesthesia on IOP show that there is no significant increase in the IOP [1–3]. This is thought to be one of the advantages over other types of peribulbar anaesthesia, where more pronounced IOP elevation was detected [1–3]. This may be because when sub-Tenon's anaesthesia is performed the incision in the conjunctiva may allow egress of fluid if the pressure is too high. This pressure valve effect is absent in peribulbar anaesthesia. The bolus pressure over the vessels is thought to be insignificant, because in the sub-Tenon's space a thin layer of anaesthetic is formed. This is additionally facilitated by the use of hyalase. In our case neither Honan's balloon, nor digital massage were used, in which cases the IOP may be increased significantly to a level sufficient to occlude the central retinal artery [4, 5]. The IOP may increase more significantly in patients with glaucoma [6] or other conditions, associated with deficient blood supply to the optic nerve.

The pharmacologically mediated changes in the vascular calibre are the more likely mechanism in which retinal artery occlusion can occur. Meyer et al. [7] have shown in a study performed on porcine ciliary artery that local anaesthetic agents of the amide type such as lidocaine, bupivacaine and mepivacaine interact with the vascular endothelium. This endothelial dysfunction may contribute to decrease ocular perfusion after local anaesthesia [7]. This effect is mediated via receptor-operated activity of the endothelial L-arginine/nitric oxide pathway [7]. Findl et al. [8] showed that after peribulbar anaesthesia pulsatile choroidal blood flow and retinal blood flow velocities were reduced. This effect was still present 5 min after the anaesthesia, when the IOP had returned to baseline [8]. This supports the pharmacologically mediated vasoconstriction as possible mechanism [8]. Pianka et al. [3] demonstrated that ocular pulse amplitude was significantly reduced when lidocaine was used either as sub-Tenon's or peribulbar anaesthesia. It is known that lidocaine in low concentration causes vasodilation, but in higher concentrations vasoconstriction can occur [3]. In the sub-Tenon's group of this study, because of the systemic absorption there was noted increased ocular pulse amplitude in the fellow eye in the 1st minute after the injection and reduced in the 10th [3]. This was explained with the lower dose early and the higher dose of systemic lidocaine reaching later on the other eye [7]. Because the amount of anaesthetic used in this study was relatively small (2 ml lidocaine) the mechanical effect on the ocular haemodynamics is unlikely [3].

Another possible cause for branch retinal artery occlusion (BRAO) after sub-Tenon's anaesthesia is presumed toxic effect from preservatives contained in the vials with anaesthetic drugs. There are 3 published cases of BRAO and 3 cases of CRAO after sub-Tenon's anaesthesia prior to vitrectomies with mepivacaine containing methyl- and propyl parahydroxybenzoate over a period of 3 months [9]. These vascular effects occurred between 2 and 14 days after the anaesthesia, which makes the toxic effect more probable in comparison with vasospastic effect [9]. In our case the loss of vision was noted immediately after removal of the dressing. Preservative-free medications were used for the anaesthesia. This mechanism is not relevant in our case.

It is believed that the cause of visual loss in this patient is spasm of a branch of the central retinal artery, which led to occlusion. Three cases of transient CRAO after sub-Tenon's anaesthesia were published by Feibel and Guyton [10]. CRAO is a possibility in our case as well, but the absence of relative afferent papillary defect, small central scotoma, localized

vascular changes in the macular area and the subsequent improvement in visual acuity, make BRAO more probable.

To our knowledge this is the first reported case of BRAO after sub-Tenon's anaesthesia with preservative-free medications.

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Fig. 1. **a** Fundus photo RE. **b** Red-free photo RE. **c** Visual field RE. **d** Optical coherence tomography RE.