

## Case report

# Central Retinal Arterial Occlusion (CRAO) after Phacoemulsification-A Rare Complication

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### Abstract

**Background:** While peribulbar anesthesia is generally safe, a remote risk of retinal vascular accident exists and its routine use should be done with caution. **Objective:** To report a case of central retinal artery occlusion (CRAO) that occurred within 24 hours of routine uneventful phacoemulsification cataract surgery using peribulbar anesthesia. We share our experience of a 45-year old man who underwent uneventful clear corneal temporal incision phacoemulsification cataract surgery using peribulbar lignocain injection with adrenaline. **Case:** A Patient who underwent routine phacoemulsification surgery of left eye for posterior sub-capsular cataract under peribulbar anesthesia developed central retinal artery occlusion in the immediate post-operative period. The surgery was uneventful. **Conclusion:** Central retinal artery occlusion is a rare but dreadful complication seen after uneventful phacoemulsification and the cause is mainly due to anesthesia related.

**Keywords:** Central retinal artery occlusion, peribulbar anesthesia, phacoemulsification

### Introduction

Effective anesthesia for major intraocular surgery in the past had been achieved using retro bulbar anesthesia. This technique, however, has an increased risk of direct damage to the optic nerve. Such damage includes central retinal artery occlusion (CRAO), combined CRAO and central retinal vein occlusion (CRVO) and anterior ischemic optic neuropathy (AION). These complications are thought to be due to direct needle penetration of the optic nerve, mechanical compression, drug toxicity, or from the effects of adjunct vasoconstrictor agents used in anesthetic solutions, among other causes (Klein ML et al, 1982). To reduce the incidence of such anesthesia-related complications, retro bulbar anesthesia has become largely replaced with other modalities such as topical, intracameral and peribulbar anesthesia. While these new modalities are much safer than retro

bulbar anesthesia, a handful of complications have been reported in the literature.

In this report, we present a case of CRAO in a 45-year-old male patient who underwent uneventful phacoemulsification cataract surgery under peribulbar anesthesia.

### Case report

A 45-year-old man presented with painless progressive diminution of vision in both eyes for 8 months. The best corrected visual acuities (BCVA) were 6/12 OD and counting finger close to face OS. Intraocular pressures (IOP) were 14 mmHg in each eye. Anterior segment evaluation revealed normal ocular findings except posterior sub-capsular cataract in both eyes. Fundus assessment and other ocular examination findings were all normal. His general medical history was unremarkable and the physical examination normal. A diagnosis of both eyes posterior sub-capsular cataract of grade I in OD and grade IV in OS was made. The patient

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was scheduled for OS phacoemulsification surgery. Before surgery, topical ciprofloxacin 0.3% was applied and a preparation of 4 ml lignocain 2 % in 1:100,000 adrenaline was injected through a single point in the lower eyelid immediately above the inferior orbital rim at the junction of the medial two thirds and lateral one third and running parallel to the orbital floor, 1cm into the peribulbar space, using a 25 G needle. The eye was compressed for 10 minutes using a pressure-reducing ‘pink ball’; the ocular surface was irrigated with 5 % povidone iodine and a universal wire speculum inserted. Phacoemulsification through clear corneal temporal incision of the left eye was performed. After the surgery 0.3 % ciprofloxacin ointment was applied and the eye lightly padded with eye shield ( a routine procedure for all surgical cases). At the first post-operative day, the patient complained of loss of vision in the operated eye. His BCVA was measured as hand motion (HM) close to face and the IOP 15 mmHg in the affected eye with relative afferent pupillary defect (RAPD). Funduscopy by indirect ophthalmoscope showed a whitened area of the entire retina most marked at the posterior pole, a classical cherry red spot and sluggish arterial blood flow. Scattered area of blot hemorrhage (figure 1) was also noticed. Fundus angiogram showed extensive area of capillary non-perfusion (figure 2). No peribulbar hemorrhage, lid hemorrhage nor proptosis was observed and extra ocular muscle movements were full. Tab acetazolamide 500 mg (250 mg x2) was given immediately with topical 0.5 % timolol maleate. The patient was put on routine topical steroid and antibiotic eye drops every two hours and tapered off gradually. The patient’s hemogram, fasting blood sugar and lipid profiles were all within normal limits. The systemic evaluation revealed no contributing factor. He was started on oral tablet of Aspirin 75 mg per day with antacid for one month and asked to be reviewed after 1 month.

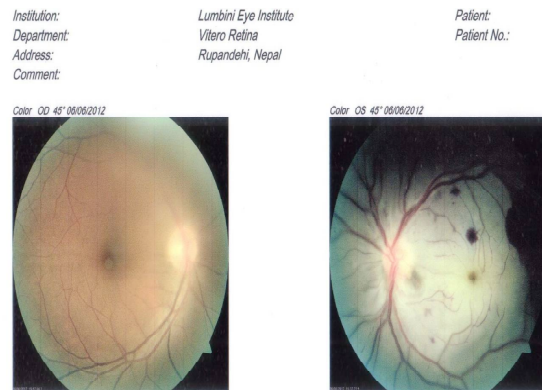


Figure 1

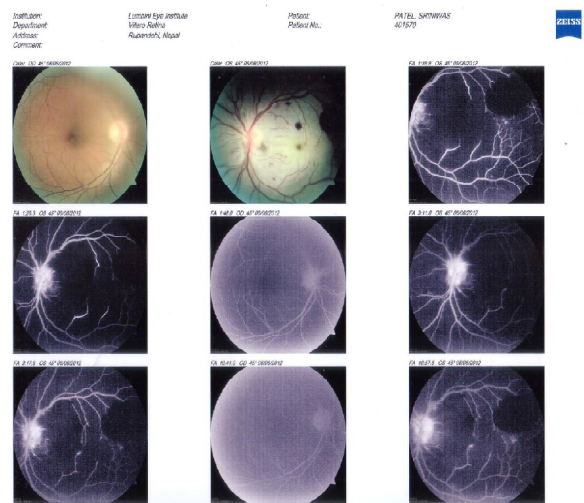


Figure 2

### Discussion

Peribulbar anesthesia is known to be safer but it still has the tendency to cause damage to the optic nerve through the remote effects of the anesthetic agent, amount injected, and speeds of injection and use of post-injection mechanical compression. Concurrent use of adrenaline in anesthetic agents is also known to cause vasoconstrictive effects that may lead to CRAO. Vinerovsky and co-workers suggested that while the event was likely to be caused by the vasospastic effects of adrenaline, it was also entirely possible to be caused by potential vasospasms in response to the anesthetic injection rather than the effects of the adrenaline. Such vasospastic effects of anesthetic agents used in local and regional blocks have been established in a number of other studies (Sullivan KL et al,

1983). Findl et al (1999) reported a decrease in retinal blood flow velocity by 10 to 15 %, one and five minutes respectively following peribulbar anesthesia without a vasoconstrictive agents like adrenaline (Klein ML et al, 1982). The group also established that such effect lasted between one to three days following a peribulbar injection for cataract surgery.

Occlusion of the central retinal artery may also be caused by increased IOP secondary to globe compression by the anesthetic agent and a subsequent weight placement on the globe. It is, however, known that extreme and prolonged increase in IOP (over the systolic arteriolar pressure) is needed to produce such retinal artery occlusion. Findl found no correlation between the high IOP and a decrease in retinal blood flow following peribulbar injection for cataract surgery (Findl et al, 1999). In this current report, the IOP remained within normal limits after completion of surgery as measured on the following day. In addition, the patient being reported on did not complain of postoperative pain which normally would accompany an acute rise in the IOP. Similar cases of retinal infarction with macular cherry red spot have been reported following intraocular injection of gentamycin and other amino glycosides aimed at preventing post-operative endophthalmitis. Many of such cases have had to do with either intravitreal injection or direct injection near areas of sclera thinning or laceration (Thomas et al, 2001). This scenario is unlikely as gentamycin was not used in this case.

The incidence of CRAO following peribulbar anesthesia suggests that damage to the optic nerve may occur even when the injection is away from the nerve. Immediate post-operative evaluation of retinal blood flow following peribulbar

injection may help early detection and prompt treatment of CRAO. This is, however, difficult in practice due to the high number of cases of peribulbar anesthesia/surgery undertaken in most centres and the rarity of such a damage.

### Conclusion

Central retinal artery occlusion after routine cataract surgery is unusual. We reviewed the literature on CRAO after routine intraocular procedures and proposed three hypotheses regarding the potential mechanisms involved. Although peribulbar anesthesia avoids direct optic-nerve injury, indirect injury presenting as CRAO may occur from vasospasm in response to the injection. A vasoconstrictive effect of the anesthetic agent on the central retinal artery, a rise in IOPs after anesthesia administration resulting in closure of the central retinal artery and a mechanical effect of the volume of anesthetic on the central retinal artery are considered as plausible mechanisms, with a mechanical effect being the favored hypothesis.

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