Commentary: Central retinal arterial occlusions after phacoemulsification: Our perspective

Vascular occlusions in the form of non-arteritic anterior ischemic optic neuropathy (NAION), and retinal arterial occlusions (RAO) like central retinal artery occlusion (CRAO) are devastating, sight-threatening complications after a routine cataract surgery. Although rare, CRAO has been described after phacoemulsification, more so when peribulbar and retrobulbar anaesthesia has been used. However, it has also been reported in patients undergoing phacoemulsification under topical anaesthesia, which highlights that there are multiple factors involved in causing a CRAO after a cataract surgery. Though, it cannot be denied that incidence of both cataract and CRAO is age dependent, there is some amount of ingrained bias when reporting CRAO with cataract surgery.

A report by Eichel and Goldberg suggested that most cataract surgeries these days are performed using peribulbar, sub-Tenon's and topical anaesthesia with >70% of cataract surgery being done using these modalities worldwide.[1] However, they are not without risks. A direct injury to the optic nerve or a retrobulbar hemorrhage causing local compression of optic nerve during retro- or peribulbar anaesthesia is known to cause vascular occlusion and subsequent visual loss. Peribulbar anaesthesia is considered to be safer, although it still has a tendency to cause vascular compression depending on the amount of drug injected, speed of injection and mechanical globe compression following injection. Another possibility is raised intraocular pressure (IOP) due to the volume of injected anesthetic drug causing globe compression. However, it is

to be noted that, an increase in IOP over the systolic arterial pressure for a prolonged period, is required to cause such a vascular occlusion. This factor may become important in patients having a compromised vasculature with subsequently reduced autoregulatory capacity of retinal vasculature such as patients with poor cardiac output or carotid stenosis.

A concurrent use of adrenaline in the anesthetic agent is also known to have vasoconstrictive effects and subsequent CRAO. It has also been suggested that injection of anesthetic drug without the use of adrenaline, can itself cause vasospasm.[2] Findl *et al*. reported a decrease in the retinal blood flow velocity by 10 and 15% at 1 and 5 minutes respectively, following peribulbar anesthesia without the use of adrenaline.[3] This suggests that the injection of anesthetic drug itself causes vasospastic effects. Relative vasoconstriction has been reported with lower concentrations of lignocaine and bupivacaine.[4] However, it is difficult to determine the clinical relevance of this observation, as the ultimate concentration of drug around the optic nerve following peribulbar injection, is unknown.

As compared to peribulbar anesthesia, IOP rise after sub-Tenon's anesthesia is lower.[5] A small amount of bolus is injected into the thin sub-Tenon's space. Consequently, the rise in IOP is insignificant. Also, the conjunctival incision during the surgery causes the anesthetic drug to egress, thereby lowering the pressure over vasculature. This effect is absent in peribulbar anesthesia. Despite this advantage, vascular occlusions have been reported following sub-Tenon's anesthesia.[6] A toxic effect of the preservatives used in the vial is presumed to be responsible for vasospastic effects in these cases.[7] A case of branch retinal artery occlusion (BRAO) has been reported following an uneventful phacoemulsification with use of preservative-free drug for sub-Tenon's anesthesia.[6]

The raised IOP leading to vascular occlusions may also occur due to intra-operative pressure fluctuations during phacoemulsification.[8] This factor may play a role in causing CRAO in patients operated under topical anesthesia. Intra-operative pressure fluctuations depend upon the aspiration pressure. Higher the aspiration pressure, higher is the IOP during surgery.

Apart from the local vascular compression, systemic factors also plays an important role in causing CRAO, as it has been stated earlier. Arterial hypertension, arteriosclerosis, cardiac arrhythmias, hypercholesterolemia, diabetes mellitus are some of the known risk factors causing CRAO in the age group undergoing cataract surgeries.

The study by Sen *et al*. published in this issue reports a cluster of CRAO after phacoemulsification. The authors studied a series of 185 cases of posterior capsular rent (PCR) that underwent anterior vitrectomy, and 14 of them had CRAO post-operatively. The causative factor has been highlighted as the vitrectomy probes that were sterilized using ethylene oxide (ETO). It is an interesting observation that changing the protocol of the ETO sterilization led to a cessation in the occurrence of further CRAO cases.[9] We would, however, like to highlight the fact that only 14 cases out of 185 cases of PCR developed a CRAO. Retinal arterial occlusions are multi-factorial in their causation and it is of prime importance to rule out all the other factors such as intra-operative IOP, use of adrenaline in anesthetic agents, vascular compromise due to systemic factors among many others.

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