Commentary: A cluster of central retinal artery occlusions following cataract surgery

Ocular vascular occlusions form a rare and often overlooked set of complications following intraocular surgeries which can have devastating sequelae. They can also be encountered following vitreoretinal interventions like scleral buckling, intravitreal antiVEGF injection, and vitrectomies with gas tamponade.[1] The spectrum can vary from central retinal artery occlusion (CRAO), branch retinal artery occlusion, to anterior and posterior ischemic optic neuropathy to rarely venous occlusions.

The technique and type of drug used to administer ocular anesthesia and elevation of intraocular pressure (IOP) intraoperatively may hamper the ocular perfusion leading to retinal vascular occlusion even after uncomplicated cataract surgery.[1,2] Systemic risk factors such as hypertension, diabetes mellitus, carotid artery stenosis, cardiac arrhythmias, and dyslipidemia can also exacerbate retinal vascular complications.[3]

Retrobulbar anaesthesia may predispose to CRAO and other complications by exerting direct mechanical compression over the central retinal artery (this risk increases with retrobulbar haemorrhage), direct injection into the optic nerve sheath, and by retinal artery vasospasm.[4,5] It has now largely been replaced by peribulbar or subtenon injection which have a minimal risk, but the risk still exists. Possible explanations include (1) diffusion of local anesthetic into artery causing vasospasm, (2) block-related rise of IOP, and (3) mechanical compression of the retinal artery due to a large volume of the agent.[2]

In the preoperative period, some surgeons prefer to use ocular compressive devices such as Honan balloon and other objects to aid in the diffusion of anesthetic agent locally to reduce positive vitreous pressure and associated complications during intraocular surgeries.[6] The duration of compression and the amount of pressure exerted can be variable. Therefore, injudicious pressure application over the globe can interrupt the retinal circulation when vitreous pressure exceeds central retinal artery pressure.

Yusuf *et al*. hypothesized about the transient retinal artery occlusion following uncomplicated cataract surgery as an idiosyncratic response.[7] The authors highlighted the indispensable role of optical coherence tomography (OCT) in these cases with unexplained visual loss in the immediate postoperative period. These can be easily missed on routine fundus fluorescein angiography as the findings are very subtle and thus difficult to appreciate. The present study also showed a normal retinal angiographic pattern in all patients but OCT revealed hyper-reflective inner retinal layers due to ischemia along these layers in the immediate postoperative period, which usually progress to inner retinal atrophy over a period of time.[8] In these cases, visual acuity may recover but the defect in colour vision, contrast and the central scotoma will persist. We must also be aware of hemorrhagic occlusive retinal vasculitis that has similar clinical symptoms. It is a type 3 hypersensitivity reaction to the vancomycin commonly used intracamerally as an antibiotic prophylaxis measure.

The present study elucidated the potential role of agents like lignocaine, adrenaline, and residual contaminants on instruments in the causation of vasospasm of retinal artery.[8] The causality of ethylene oxide for such complication can be better established by conducting pre-clinical studies. In addition, this study also highlights the need for standardizing sterilization protocols to avoid such complications.

To conclude, meticulous preoperative evaluation, judicious use of ocular anesthesia, optimal surgical intervention, and early postoperative recognition can drastically bring down such devastating complications. In addition, safer measures like topical anesthesia should be preferred in cases with pre-existing vascular compromises.

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