**Peri-operative Visual Loss in Non-Ocular Surgeries**

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 Visual loss after non ocular surgery/anaesthesia is a rare, but devastating injury. Every anaesthesiologist and surgeon should be aware of this entity so that preventive measures are taken as well as expert attention is provided to the patient on the slightest suspicion of the condition. Awareness about the condition can also help in avoiding unnecessary doubts on anaesthetic drugs used as well as possible litigation. Eye damage after ocular surgery is a separate entity well described in ophthalmology literature and is not discussed here.

**Causes and mechanism**: Peri-operative visual loss (POVL) is caused by 1. External Ocular injury 2.Central or branch Retinal Artery Occlusion (RAO), 3. Anterior and Posterior Ischaemic Optic Neuropathy (ION), 4. Cortical blindness and 5. Acute glaucoma etc.[1] Reduction in the retinal, optic nerve or cerebral perfusion due to raised intra ocular pressure, raised intra-orbital pressure, raised intracranial pressure, vascular emboli or thrombi, and reduced oxygen delivery due to severe anaemia or shock can cause POVL. Peri-operative visual loss is seen more frequently after cardiac, spinal and orthopedic joint surgeries. It can be complete or partial visual loss. It can be permanent in many cases.

 Transient visual loss may occur after transurethral resection of the prostate. Retinal vascular occlusion has been reported in patients who received Nitrous Oxide up to six weeks after a vitrectomy procedure. ION occurs approximately 1 in 60,000 to 1 in 125,000 anaesthetic procedures in over all surgical population.[2]

Largest study on the subject was done by Shan and associates who studied the prevalence of POVL in US data base for Eight most commonly performed surgeries excluding obstetrics & gynaecology.[3]

**Retinal Artery Occlusion:** Central Retinal Artery Occlusion causes ischemia to the entire retina, while occlusion of the branch of retinal artery affects only a portion of the retina. This is usually unilateral. Four causes identified are 1.External compression of the eye, 2.Decreased arterial supply to retina(embolism to retinal artery or decreased blood flow from systemic cause), 3.Impaired venous drainage of the retina and 4.Thrombosis of retinal artery. The most common cause of peri-operative retinal occlusion is improper patient positioning with external compression of the eye producing enough Intra-Ocular Pressure (IOP) to stop flow in the Central Retinal Artery. Altered facial anatomy and exophthalmos may predispose to damage by pressure of anaesthesia mask or head rests. Horse shoe head rest is a concern due to its shape and the narrow opening for the face. Improper head position or unintended head movement after positioning may place the eye in contact with the head rest. RAO mostly occurs during spine surgery done in prone position. IOP is increased further by head down position. Retro bulbar hemorrhage occurring due to vascular injuries during sinus or nasal surgery is another cause for it. Retinal micro-emboli are common during open heart surgery.[4] Paradoxical embolism originating from an operative site and reaching arterial circulation through a Patent foramen ovale has been reported to cause retinal artery occlusion rarely.[5] It is rarely seen in hypotensive anaesthetic procedures. Venous drainage of eye can be impaired after radical neck surgery due to jugular vein ligation .

The retinal blood supply is derived from the inner retinal and outer choroidal vessels.[6] So, some oxygen may still be supplied by diffusion from outer layers of retina even after Central Retinal Artery Occlusion(CRAO). Increased IOP from external compression of the eye is a more severe insult than CRAO because the former reduces both retinal and choroidal blood flow .

CLINICAL FEATURES:- Painless visual loss and abnormal pupillary reaction occur. Fundoscopy shows opacification or whitening of the ischemic retina. Narrowing of retinal artery may be visible. A cherry-red macula with a white background is a classic diagnostic sign in CRAO. Kumar & Colleagues in their review regarded unilateral loss of vision, usually with loss of light perception, afferent pupil defect, peri-orbital or lid oedema, chemosis, proptosis, ptosis, paresthesias of supra –orbital region, hazy or cloudy cornea and corneal abrasion, loss of eye movements, ecchymosis or other trauma near the eye.[7] Early orbital CT or MRI Scan has shown proptosis and extra –ocular muscle swelling.

ORBITAL COMPARTMENT SYNDROME: Incidence of orbital complications after endoscopic sinus surgery is about 0.12%. Orbital hemorrhage from blunt trauma during the procedure can result in orbital compartment syndrome with compression of the arterial and venous circulation & in CRAO and optic nerve injury. Indirect damage to CRA from intra-arterial injection of lignocaine with adrenaline has also been described, and is probably due to arterial spasm or embolism.

BRANCH RETINAL ARTERY OCCLUSION (BRAO): This usually leads to permanent ischemic retinal damage with partial visual field loss. Patient may not notice symptoms immediately if the visual field loss is peripheral or if the stoma is small, BRAO is primarily due to emboli, though vasospasm has been reported in some cases. Retinal flourescein angiography has demonstrated micro-emboli to the retina during Cardio Pulmonary Bypass. Bubble oxygenators used to cause micro-emboli in almost all patients while the incidence of retinal perfusion defects have reduced by half by using membrane oxygenators.

Multiple calcific emboli in CRA & its branches during surgeries like CABG causes visual field defects of varying size & location. Injection of drugs like steroids, chemotherapeutic agents, angiographic dyes, embolization materials etc injected into nasal mucosa have caused visual loss from RAO. Local anaesthetic infiltration with adrenaline for nasal septal surgery can cause partial or total visual loss defects attributable to RAO.[8] This could be due to accidental injection of the drug into the branches of external carotid artery. Fat injected into the orbit for cosmetic surgery can be a cause for RAO and loss of vision.

 TREATMENT: No satisfactory treatment is available at present and peri-operative RAO results in permanent loss of vision in most cases. Treatment with ocular massage to decrease IOP (avoid if glaucoma cannot be ruled out) and thereby dislodge an embolus if present to more peripheral route can be tried.[9] Intravenous acetazolamide can be given to reduce Intra Ocular Pressure. Inhalation of 5% CO2 in O2 may enhance vasodilatation, increase blood flow and increase O2 delivery from retinal and choroidal vessels. Thrombolysis may be tried, but is contra indicated after certain surgeries. Fibrinolysis through a catheter in the ophthalmic artery within 6-8 hours has shown to improve vision. Local application of hypothermia could be tried as it has shown to decrease injury in animal studies.[10]

PREVENTION: Avoid compression of the eye balls by anaesthetic masks, surgeon’s arms, head rests while prone position etc. Leaving the eye in open position in a sedated/paralyzed patient can cause drying of the cornea and opacification. Usual practice of applying lubricants & taping the eyes in closed position is helpful. The position of the head and eyes should be checked every 20 minutes or so by palpitation or visualization, placing the head in pin head holder and avoiding horse-shoe head rest for prone position surgery for cervical spine surgery etc. can be helpful. Use of goggles to cover eyes is not advised in prone position. Patient should be checked for signs of acutely elevated IOP after surgery and immediate ophthalmologic consultation should be made.

ISCHEMIC OPTIC NEUROPATHY (ION): It is a leading cause of sudden spontaneous loss of vision in patients 50 years or older. ASA Post-operative visual loss Registry is an important study on the subject.[11]

MECHANISM: Ischemia causes axonal destruction. ION may lead to neuronal injury by optic nerve cell death also. Optic disc edema and haemorrhage are seen with onset of symptoms in AION. Optic disc appears normal in PION even when patient has visual loss. Optic atrophy develops in weeks to months time. Can be unilateral or bilateral. MRI may show optic nerve oedema or perineural enlargement.

AN INTERESTING STUDY IN TRAUMA PATIENTS: Cullinane and co-workers did a retrospective study on 350 trauma patients who required massive volume resuscitation of 21 to 50 liters in first 24 hrs.[12] ION was found in 2.6% including bilateral blindness in 4 patients. The mean age was 34±13 years. Patients were acidotic. Mean lowest Hct was 7.5% to 28% (Mean 20 ± 8%). Blood product infused included 9 to 39 units of packed RBCs. All developed ARDS that required increased FIO2 & PEEP on ventilator (mean 29±9cm H2O). All patients had systemic inflammatory response syndrome and 66% had compartment syndrome at some location remote from eyes. Mean time until detection of visual loss was 36 days because patients needed prolonged mechanical ventilation and sedation and vision could be assessed only after they were out of ventilator.

ION affects the anterior portion of the optic nerve, proximal to the lamina cribrosi in AION and beyond the retro laminar region behind the eye in PION. Hypotension can reduce the perfusion pressure in the optic nerve and lead to ischemic injury. Fall in haemoglobin could also cause ischemia. In anaesthetized patients, the IOP was found to increase to 27 ±2mmhg in prone position compared to 13±1mmhg in supine position. After 5 hours in prone position, IOP increased to 40±2 mmhg in a study by Chang and others largest increase in IOP was observed more towards the time the patient was awakening.[13] When external pressure on the eye is the cause for ION, they will be associated retinal damage as well, because of the raised IOP affecting retinal and choroidal blood supply. Prone position was found to cause increase in diameter of optic nerve in healthy volunteers and has been explained to be due to venous hypertension. AION has been found in patients who used erectile dysfunction drugs and is advisable to discontinue this drugs 1 or 2 days before surgical procedures.

Retro-bulbar haematoma causes permanent blindness and hence the anaesthesiologist should be aware of its possibility so that early surgical decompression can be arranged.

Management and Prevention : Acetazolamide reduces IOP & may improve blood flow to the optic nerve head and retina. Diuretics like mannitol & frusemide can reduce oedema. Corticosteroids can reduce axonal swelling in acute phase, but should be used carefully as chance of infection may increase after surgery. Increasing ocular perfusion pressure and Hb concentration has to be considered. Head up position reduces ocular venous pressure and can be helpful if systemic BP is stable. Immediate decompression (lateral canthotomy) is to be done if the patient has visual loss from ocular compartment syndrome. Hyper baric oxygen has been tried to treat PION. Some patients recover vision after PION spontaneously. Head down position is to be avoided. Using some colloids during large volume resuscitation may be helpful. Avoid sudden haemodilution to Hct below 25. Minimally invasive surgical techniques in spinal surgery & fusion etc. can be considered.

CORTICAL BLINDNESS is bilateral visual loss due to damage to both left and right optical cortex. It is rare compared to partial cortical blindness. Here lid reflex response to threat is absent, while pupillary response, eye motility, retina & optic nerve are normal. Total cortical blindness is usually accompanied by signs of stroke in the parieto-occipital region. Often, vision improves over time. 55% of cortical blindness was found after CABG surgery and 23% after other thoraco-vascular operations.[14] It can be caused by global ischemia, cardiac arrest, hypoxemia, intracranial hypertension, exsanguinating hemorrhage, focal ischemia, vascular occlusion, thrombosis, intracranial hemorrhage , emboli & vasospasm. Visual recovery from cortical blindness can be prolonged, but previously healthy patients can have considerable recovery. Treatment should be directed towards preventing progressing of the stroke.

 ACUTE GLAUCOMA is a well known disease that can rarely manifest after anaesthesia. It is more common in women & elderly. Acute angle closure results in glaucomatous damage to the optic nerve and occurs when passage of aquous humor from the posterior to the anterior chamber is obstructed by apposition of the iris to the anterior surface of the lens. The pupil is mid-dilated, with an associated pupillary block. It should be suspected in a patient with a painful red eye and cloudy or blurred vision with headache, nausea & vomiting. Acute glaucoma is an ophthalmic emergency and may require peripheral iridectomy to create a permanent opening between anterior & posterior chambers.

 Visual defects after Transurethral Resection of Prostate happen due to excessive absorption of hypotonic irrigating fluid, usually 1.5% glycine. Visual changes in TURP syndrome can be detected during resection, or even on second post-operative day as the patient wakes up from coma. Preventing excessive absorption of irrigant solution is important .

Visual loss following anaesthesia after vitrectomy:- Perfluoro Carbon gas bubble instilled into the Vitreous during vitrectomy can remain in the eye for as long as six weeks and nitrous oxide administration to such patients can increase the size of the gas bubble by about 3 times, causing raised IOP and retinal vascular occlusion. Hence N20 should be avoided in such patients.

CONCLUSION: Visual loss can happen in the peri operative period from retinal artery occlusion, ION, cortical blindness, acute glaucoma, TURP syndrome & N2O induced gas bubble expansion in post vitrectomy patients. Anaesthesiologist and the surgical team should be aware of this, so that preventive measures can be taken whenever possible as well as treatment can be arranged at the earliest to help the patient to the maximum possible extent. Discussing this possible complication with the patient during preoperative visit and taking informed consent can avoid later legal problems. American Society of Anaesthesiologists 2012 task force summary on peri operative visual loss – Summary of Advisory Statements provides useful information on the subject.

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