Case Report

Visual loss after spine surgery

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Abstract

Incidence of perioperative visual loss ranges from 0.06% to 0.2% with the most common cause as ischemic optic neuropathy. We report one-year follow up of a 50-years-old hypertensive housewife who underwent lumbar decompression and fusion for degenerative scoliosis, but woke up with painless unilateral visual loss. Fundus examination was normal. Her visual acuity improved from initial finger counting close to face to finger counting at 3 m at 1 year. Identification of high risk patients may help in appropriate preoperative counselling, prevention and early recognition of this devastating complication.

Key words: Spine surgery, degenerative scoliosis, vision loss

Introduction

Perioperative visual loss (POVL) following spine surgery is a potentially devastating complication whose pathogenesis is often unclear in many cases. A recent population based, retrospective study conducted through the NIS (Nationwide inpatient sample), which is the largest all player inpatient database in the United States, reported an overall incidence of visual disturbance after spine surgeries as 0.094% among 4728815 cases operated between 1993 and 2002. Of these, 0.006% had ischemic optic neuropathy (ION) and 0.001% had central retinal artery occlusion (CRAO).[1] Another study which was also done evaluating the NIS data from 1996 to 2005 reported an incidence of POVL in various surgical procedures as follows: 8.64/10000 in cardiac surgeries, 3.09/10000 in cases of spinal fusion (5.36/10000 through the posterior approach), 1.08 and 1.86/10000 in knee and hip surgery respectively, and 0.12/10000 in appendicectomy.[2]

We present the case of a 50-years-old female operated for degenerative scoliosis with lumbar decompression and fusion. She had painless unilateral visual loss immediately after surgery, which recovered to finger counting at 3 metres at one year.

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Case Report

A 50-years-old woman disabled with back pain and lower limb claudication since four years on conservative treatment presented with further reduction in claudication distance and back pain enough to restrict activities of daily living. She weighed 69 kg and was 151 cm tall with a body mass index (BMI) of 30.26. She was a controlled diabetic and hypertensive with a preoperative blood pressure of 130/86 mm Hg and was on Tablet Losartan (80 mg) and Tablet Lisinopril (5 mg) once daily. She was found to have degenerative scoliosis with severe canal stenosis at L3-5 after investigations.

She underwent a lumbar decompression with fusion from L2-5 under general anesthesia. She was induced with Inj. Propofol (2 mg/kg) and Inj. Fentanyl (2 µm/kg). Inj. Atracurium (1 mg/kg) was used for neuromuscular relaxation. Anesthesia was maintained on Sevoflurane (MAC 2). Preoperative hemoglobin was 13.2 g%. Her blood pressure rose to 160/100 immediately after induction but it was later maintained at around 130/80 mm Hg during the surgery. An arterial line was used to monitor blood pressure till the end of surgery. She was operated in prone position with proper gel padding over bony prominences. A foam headrest with cut outs for eyes was used for the head and face. Total surgical time was 3.5 h with intraoperative blood loss of 900 ml. Intraoperatively and in the recovery room, 4 units of crystalloids and 2 units of colloids were infused. Postoperatively, her drain output was 750 ml over three days. She also received 2 units of packed red cells as on day 2 post surgery her hemoglobin dropped to 7.2 g%.

Immediately postoperatively, she had painless vision loss in the right eye without any local signs of swelling or bruising. Urgent ophthalmology consultation was made. The fundus examination was found to be normal; there was a relative afferent papillary defect (RAPD) and loss of visually evoked potential (VEP) on the right side. Clinically she had finger counting close to face only. A presumptive diagnosis of post operative visual loss (POVL) secondary to posterior ischemic optic neuropathy was made. She was treated with intravenous steroids for three days followed by oral steroids for two weeks followed by gradual tapering. On one year follow up, she had finger counting at 3 m and repeat fundoscopic examination showed pallor of the optic disc.

Discussion

Postoperative visual loss is a devastating complication after a non-ocular surgery. Most commonly, it presents as decreased visual acuity but visual field defects have been reported as well.[3-5] Among the nonocular surgeries, maximum incidence is found after cardiac and spine surgeries.[6] Spine surgeries being the second most common among the non ocular surgeries which can have such an unfortunate complication postoperatively. Various causes have been implicated in literature, such as ischemic optic neuropathy (ION), including anterior ischemic optic neuropathy (AION) and posterior ischemic neuropathy (PION), central retinal artery occlusion (CRAO), central retinal vein occlusion (CRVO), orbital lobe infarction and rare causes such as ischemic orbital compartment syndrome and pituitary apoplexy.[3-11] The overall outcome of POVL in general is very poor owing to limited treatment options and unclear pathogenesis; hence, all attempts must be made to prevent its occurrence.

Hypotension, peripheral vascular disease and anemia are commonly implicated in the development of ION, whereas excessive external pressure over the eye, uncontrolled hypertension, diabetes mellitus, morbid obesity peripheral vascular disease and blood transfusion have been associated with CRAO.[6-11] Other factors associated with ION include excessive blood loss, hemodilution, increased intraorbital/intraocular pressure, use of head low position during surgery and smoking. AION is more commonly reported after cardiac surgeries, while PION after prone positioned spine surgeries and radical neck dissection.[6] Visual loss has even been reported after anterior and lateral decubitus spine surgeries.[11]

The clinical presentation of both forms of ischemic neuropathies is the same, i.e. painless loss of vision or visual field defects with sluggish pupil.[3-10] On the other hand, orbital compartment syndrome is associated with painful loss of vision, acute periorbital edema, conjuctival chemosis and edema.[7] It probably occurs consequential to raised intra ocular pressure as a result of direct pressure on global and periorbital structures due to abnormal head position during prolonged prone position.[7] In the present case, the patient had painless loss of vision with no local signs, favoring the diagnosis of ischemic optic neuropathy. As she had a normal fundus examination, the patient was presumptively diagnosed as PION. AION, which is caused by occlusion or relative hypoperfusion of anterior optic nerve head by the posterior ciliary arteries, has diffuse or focal disc edema with or without peripapillary flame shaped hemorrhages or splinter hemorrhages at the optic disc margin.[1,6,9,10] PION, which is postulated to be caused by infarction of the intraorbital part of optic nerve, has a normal fundus examination in the beginning, with optic nerve pallor and atrophy ensuing only after 4-6 weeks.[6,10] PION shows gadolinium enhancement and/or restricted diffusion on MRI whereas in AION MRI is normal.[6] But on follow up, both AION and PION show disc pallor so cannot be differentiated based on fundoscopic examination.[6] Three types of PION have been recognized namely perioperative, arteritic and non arteritis.[6] In arteritic form of ischemic optic neuropathy, steroids have a definite role to play in management as it is an ophthalmologic emergency in which initially systemic steroids are given followed by oral steroids till the ESR and CRP are brought under control. It prevents any further visual loss in the same eye or both eyes.[12,13] But in other forms of ischemic optic neuropathies, use of steroids is controversial; but there are some cases reported which have shown improvement with steroids.[13]

On the other hand, CRAO also presents as postoperative sudden severe visual loss on awakening with absent or sluggish pupillary reflex and relative afferent pupillary defect. On fundoscopic examination, there are classical findings of retinal pallor with attenuated retinal vessels and the classical cherry red spot at the macula.[6]

Pituitary apoplexy is a rare complication due infarction or hemorrhage in an existing pituitary adenoma after a major surgery like coronary artery bypass grafting (CABG), due to intraoperative fluctuations in blood pressure, infusion of crystalloids, anticoagulants, excessive steroid secretion and transient increase in intracranial pressure. It results in rapid enlargement of the adenoma with compression of the parasellar structures with abrupt onset of headache, meningismus, ophthalmoplegia, partial/complete visual loss and/or stupor. It is diagnosed on an MRI and requires urgent sellar decompression.[6]

There are no definite clinical trials to show a direct causal relationship between some patient-related factors and intraoperative situations with the occurrence of POVL.

But on review of literature some factors may be associated with occurrence of POVL like preoperative anemia and vascular risk factors like hypertension, glaucoma, carotid artery disease, smoking, obesity and diabetes.[4,14] The literature also suggests association of perioperative visual loss with prolonged procedures, substantial blood loss or both.[1-14] Intraoperatively hypotension, blood loss, anemia, hemodilution, facial edema, pressure on the eye, use of vasopressors, prone and head down positions, substantial fluid resuscitation, increased venous pressures, and prolonged surgery have been proposed for postoperative visual loss; but of these prolonged surgical duration and substantial blood loss have been present in majority of the patients who have experienced postoperative visual loss.[4]

So despite postoperative visual loss being a well-known complication after non-ocular surgeries, there is dearth of level I evidence to directly relate some causal factors to its occurrence resulting in inability on our part to reduce its incidence and also managing such complications. But certain advisory groups like American Society of Anaesthesiologists have proposed some guidelines based on available literature and case reports to avoid such a complication like preoperative evaluation and preparation, intraoperative patient positioning, management of intravenous fluids, anemia, use of vasopressors, maintaining blood pressure during surgery and duration of surgery.[14] However in cases where post-operative visual loss occurs, urgent complete ophthalmologic evaluation, use of imaging techniques like MRI and prompt treatment are critical to the final outcome of the patient.

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