Case Report

Visual Loss Following Laproscopy with Contrast

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Case reports of serious operative complications resulting in permanent visual loss have appeared in the ophthalmology, anesthesialogy, and surgical literatures since the 1940's. Perioperative ischemic optic neuropathy (POION), unilateral or bilateral, is a common cause of visual loss in non-ophthalmologic surgery. It is associated with anemia, hypotension, long duration of surgery, excessive hydration (causing intra-operative and immediate postoperative anemia), or a combination of these factors. Bilateral visual loss in the presence of normal pupillary light reflexes places the causative lesion posterior to the initial synapse between the retinal ganglion cells and cells of the LGN (lateral geniculate nuclei of the thalamus).

CASE REPORT

I present two cases of complete visual loss after laparoscopy with contrast (methylene blue) complicated by accidental dye intravasation into the circulation. The procedure was done as one of the investigations for primary infertility.

CASE 1. Thirty three years old female presented with severe deterioration of vision both eyes one week after the procedure. Her vital signs were, Pulse - 80/min, blood pressure 110/70 mm of Hg, body temperature. 98.4^0F. On ocular examination visual acuity was hand movements (HM) both eyes. No affrent pupillary defect (APD) seen. Fundus revealed macular edema on both sides. On investigation, Hemoglobin % 11.4 mg/dl, Total Leucocyte Count $10.7x10^9$/l, Serum urea 4.9 mmol/l, Serum creatinine 1502 µmol/l, Sodium 126 mmol/l, Potassium 4.2 mmol/l, Serum Bilirubin 0.4 µmol/l, Serum ALT 49 U/l, Serum Alkaline phosphtase 316 U/l, Serum Total Protein 67 g/l. Ultrasonography abdomen revealed 40ml fluid (dye) in pelvis. After repeated haemodialysis renal function parameters stabilized and she was discharged. Poor prognosis regarding visual status was explained to the patient and her family.

Case 2: A 22 years old female developed anaphylactic shock followed by fluid overload and alveolar membrane diffusion defect after the procedure. On General physical examination, skin cold and clammy. Vital signs: Pulse 124/min, blood pressure 90/50 mm of Hg, Respiratory Rate 40/min. On ocular examination: visual acuity HM on both sides. No APD. Fundus revealed macular edema on both sides. Nasal margins of the optic disc were blurred. On systemic examination: chest auscultation revealed bilateral crackles more on right side. Blood oxygen saturation 68%. She was diagnosed as suffering from acute respiratory distress syndrome (ARDS) and managed with intra venous steroids, peripheral vasoconstrictors and oxygen inhalation. After 10 days she developed maculopapular rash on trunk and ecchymotic patches over left forearm. She was diagnosed with serum sickness and treated accordingly. Her visual status remained same except increased pallor of the optic disc and development of APD.

DISCUSSION

Postoperative visual loss is a rare but disastrous complication that has an estimated incidence of 0.01-1% after non-ocular surgery[1]. POION, most likely related to compromised blood flow to the optic nerve, is subdivided into an anterior type, with swelling of the optic disc, and a posterior or retro bulbar type, in which the optic disc initially appears normal. In both types, the optic disc becomes pale over time if there is irreversible damage to the nerve. Although no treatment has been proven to improve vision in POION, several groups recommend treatment with systemic corticosteroids, transfusion to a hematocrit above 30% and mean arterial pressure kept reasonably close to the patient's baseline[2]. Retro bulbar POION is the most likely diagnosis in Case 2 with the appearance of APD even though 10 days after the procedure.

Knox and associates[3] reported a variety of uremic optic neuropathy characterized by bilateral visual loss with disc swelling in patients with severe renal disease manifested by uremia, anemia and hypertension with improvement following haemodialysis. Bilateral Ischemic Optic Neuropathy (ION) was reported in a young woman with optic disc drusen and chronic hypotension while she was undergoing renal dialysis.[4] These risk factors, that is, pre-existing hypertension, anemia and uremia, taken collectively, interfere with vital auto regulation of arterial perfusion at the disc or retro-bulbar nerve in ways not yet completely understood. Frequently enough, both eyes are involved, and bilateral retro-bulbar infarcts with mild disc edema have been documented histologically.[5] Risk factors apparently include systemic hypertension, diabetes, coronary artery disease[6], pre-existing anemia and occasionally renal failure with uremia. Patients with acute non-surgical hypotensive episodes, including unduly rapid correction of malignant hypertension and during renal dialysis, had anterior ION with partial recovery on immediate reversal of hypotension, however pre-existing anemia was present (hematocrit range of 23% to 28%)[7,8]. One may conclude that intra operative hypotension, usually coupled with low hematocrit, is the single most common cause of genuine posterior ION. Cases of blood loss with visual loss have been reversed apparently by blood replacement[9].

Case 1 presented with bilateral visual loss with reactive pupils placing the causative lesion posterior to the site of the initial synapse between the retinal ganglion cells and cells of the LGN, the optic radiations, or the cerebral cortex of the occipital lobes. This is because pupillary reactivity requires intact function of the pupillomotor fibers, which diverge from the optic tracts anterior to the lateral geniculate nuclei. Bilateral occipital cortex lesions are more common than lesions of both thalami[10]. Cortical visual loss, decreased vision from involvement of the occipital cortex, is most commonly due to vascular lesions (infarction), but may also result from trauma, compressive lesions, and toxic agents. The visual loss may occur in association with other neurological deficits or in isolation. It should be noted that even with a complete homonymous hemianopia, visual acuity is preserved. This means that patients with reduced visual acuity from a cortical lesion must have bilateral cortical involvement. Bilateral occipital infarcts occur after involvement of both posterior cerebral circulations. Although simultaneous occipital cortical lesions may occur, a more common scenario is a sequential lesion. The initial lesion causes a homonymous hemianopia, which may go undetected. The second lesion, to the contra lateral occipital lobe, results in loss of visual acuity, which may be the first sign of trouble recognized by the patient[11]. The predominant CT finding is edema of the sub cortical white matter involving the parieto-occipital lobes. This predilection for the parieto-occipital lobes may be explained by the sparse sympathetic innervation of the vertebrobasilar system, resulting in increased

perfusion during periods of hypertension and breakthrough of auto regulation affecting these areas[12]. Could the visual loss have been due to Leber Hereditary Optic Neuropathy[13] but it presented coincidentally with the procedure performed, is a question, which remains to be answered.

CONCLUSION

Patients with bilateral visual loss and normal pupillary light reflexes should undergo neuro imaging focusing on the visual pathways posterior to the LGN[14]. Bilateral sudden visual loss is a great trauma for the patient and referral to the psychiatrist is a must to reduce the suicidal tendencies developing in such cases and also for the guidance they need to tackle with the daily life problems they would be facing in future.

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REFERENCES

1. **Kamming D, Clarke S.** Postoperative visual loss following prone spinal surgery. Br J Anaesth. 2005; 95: 257-60.
2. **Chang SH, Miller NR.** The incidence of vision loss due to perioperative ischemic optic neuropathy associated with spine surgery. The Johns Hopkins Hospital Experience. Spine. 2005; 11: 1299-1302.
3. **Knox DL, Hanneken AM, Hollows FC.** Uremic optic neuropathy. Arch Ophthalmol. 1988; 106: 50.
4. **Michaelson C, Behrens M, Odel J.** Bilateral anterior ischemic optic neuropathy associated with optic disc drusen and systemic hypotension. Br J Ophthalmol. 1989; 73: 767.
5. **Johnson MW, Kincaid MC, Trobe JD.** Bilateral retrobulbar optic nerve infarctions after blood loss and hypotension: a clinicopathologic case study. Ophthalmology 1987; 94: 1577.
6. **Slavin ML.** Ischemic optic neuropathy after cardiac arrest. Am J Ophthalmol. 1987; 104: 435.
7. **Connolly SE, Gordon KB, Horton JC.** Salvage of vision after hypotension-induced ischemic optic neuropathy. Am J Ophthalmol. 1994; 117: 235.
8. **Shapira OM, Kimmel WA, Lindsey PS.** Anterior ischemic optic neuropathy after open heart operations. Ann Thorac Surg 1996; 61: 660.
9. **Jaben SL, Glaser JS and Daily M.** Ischemic optic neuropathy following general surgical procedures. J Clin Neuro Ophthalmol. 1983; 3: 239.
10. Medscape General Medicine. 2005; 7: 58.
11. **Foroozan R, Coats DK.** Bilateral visual loss with normal pupillary light reaction. Medscape Ophthalmol. 2003; 4.
12. **Knower MT, Pethke SD, Valentine VG.** Reversible cortical blindness after lung transplantation. Southern Medical Journal Posted 07/01/2003.
13. **Kanski JJ.** Neuro-ophthalmology. In: Kanski JJ, editors. Clinical Ophthalmology. 5th ed. Philadelphia: Butterworth-Heinemann 2003; 607.
14. **Cardone SC, Coats DK.** An Occult Cause of Bilateral Visual Loss in a Child. Medscape Ophthalmology 2005; 6.