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Case Report

Transient Bilateral Visual Loss due to Posterior Ischemic Optic Neuropathy After Cardiac Surgery: A Very Rare Case

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ABSTRACT

Visual loss is a relatively rare but devastating and unpredictable complication of open heart surgery with cardiopulmonary bypass. The most common cause of postoperative visual loss following cardiac surgery is ischemic optic neuropathy (ION), which is generally categorized as anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION). PION is clinically differentiated from AION with a normal-appearing optic nerve head. PION is relatively more common in cases of spinal surgery and radical neck dissection, while AION appears to be more common than PION after cardiac surgery. We report a very rare case of transient bilateral visual loss due to PION in a 44-year-old man undergoing mitral valve replacement and coronary artery bypass graft surgery. ***(Iranian Heart Journal 2019; 20(1):67-71)***

KEYWORDS: Visual, Optic neuropathy, Cardiac surgery

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The chief causes of visual loss after non-ophthalmic surgery are ischemic optic neuropathy (ION) and central retinal artery occlusion. Other causes of postoperative visual loss (POVL) include cortical blindness and pituitary apoplexy.[1-4] POVL occurring in non-ophthalmic surgery is a disaster that typically occurs immediately after emerging from anesthesia, even though it may be delayed for several days.[5] The prognosis for visual improvement is usually poor.[6,7] Studies on 65 000 and 400 000 patients undergoing anesthesia for all types of surgery at 2 large academic institutions revealed a low prevalence rate of perioperative visual loss in the operations except for cardiac and spinal surgeries and the highest rates of POVL were in cardiac, spinal, and orthopedic surgeries.[8,9] The most common causes of PVOL following cardiac surgery are the 2 different forms of ischemic optic neuropathy (ION): anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION). Whereas PION is relatively more common with spinal surgery and radical neck dissection, AION appears to be more frequent after cardiac surgery.[8,10] According to large-scale reviews, the overall incidence of AION is 0.00079% (1/126 666) after spinal surgery and 0.024% (42/172 569) following cardiac surgery. The overall incidence rate of PION has been reported to be 0.005% (7/140 768) after spinal surgery and

0.0061% (10/164 282) following cardiac surgery.[4] Although the incidence of blindness due to PION after cardiac surgery is considered to be extremely low, avoiding this horrific complication is vitally important and requires the management of the perioperative factors associated with the development of PION after open-heart operations such as intraoperative hypotension, prolonged cardiopulmonary bypass (CPB) times, minimum anesthetic durations of 6 hours, blood loss of 1000 mL or greater and anemia, decreased blood oxygen-carrying capacity caused by anemia and intraoperative hemodilution, embolism, high lactate levels, and the use of vasopressors.[11,12]
We herein present an extremely rare case of PION occurring after combined mitral valve replacement and coronary artery bypass grafting with CPB and discuss the various factors associated with POVL.

Case Report

A 44-year-old man with severe mitral regurgitation, coronary artery disease (significant ostial stenosis of the ramus), and an ejection fraction of 55% was scheduled for elective coronary artery bypass graft surgery and mitral valve replacement using CPB under general anesthesia. In the preoperative visit, the patient did not have coexisting diseases such as hypertension, diabetes mellitus, renal disease, or endocrine or neurology diseases. All the preoperative blood tests and urinalyses were within the normal ranges. Before the induction of anesthesia, the patient was premedicated with 1 mg of intramuscular lorazepam and 0.1 mg/kg of morphine sulfate 1 hour before entering the operating room. The induction of anesthesia was performed under the monitoring of electrocardiography, pulse oximetry, capnography, cerebral oximetry, and invasive arterial blood pressure—with 0.2 mg/kg of etomidate, 2.5 µg/kg of sufentanil, and 0.2 mg/kg of cisatracurium. After the insertion of the central venous catheter, the maintenance of anesthesia was accomplished with a continuous infusion of midazolam, sufentanil, and atracurium. The patient's heart rate, blood pressure, arterial and cerebral blood oxygen saturation, and end-tidal CO_2 were recorded continuously. (At all times, they were all within the normal range.) Intraoperative transesophageal echocardiography was employed as a part of monitoring in this case. The administration of 3 mg/kg of heparin with a subsequent activated clotting time of 550 seconds was done just before the initiation of CPB. During CPB, the mean arterial pressure was maintained around 60 to 75 mm Hg, cerebral oximetry numbers were between 60% and 75%, and the body temperature was maintained at a moderately low degree of 30°C. Perioperative acid/base balance management was performed with an α-stat via the measurement of arterial blood gas, Na, Ca, K, Cl, lactate, blood sugar, and hemoglobin several times. All the parameters were within the normal range except the lactate level, which rose up to 5.3 mg/dL on rewarming and at the end of CPB (probably because of the prolonged CPB time and the initiation of an inotrope on rewarming). Preoperatively, the hemoglobin concentration was 11.3 g/dL, while intraoperatively, its range was 8.5 to 10.7 g/dL without the administration of packed cells. The urinary output during the surgery was 2100 mL (preoperative=300 cc, during CPB=1300 cc, and post CPB=500 cc). The total CPB time was 200 minutes, and the aortic cross-clamp time was 145 minutes. The patient was successfully weaned from CPB with epinephrine (0.1µg/kg/min) and milrinone (0.5µ/kg/min).
On admission to the open-heart intensive care unit (OPICU), the patient's mean blood pressure was 91 mm Hg with epinephrine (0.1µg/kg/min) and milrinone (0.5µ/kg/min). Because of tachycardia (heart rate=135 bpm), epinephrine was replaced with norepinephrine. The hemoglobin concentration immediately on admission to the OPICU was 8.7 g/dL, on account of which 1 unit of packed cells was administered. He was weaned from the

ventilator and extubated after the stability of his hemodynamics with a low dose of inotropes 12 hours after admission to the OPICU. Over the next 24 hours, he was weaned from all the inotropic medications.

On the morning of postoperative day 1, shortly after extubation and full recovery from anesthesia, the patient complained of profoundly decreased vision in both eyes (painless visual loss). He immediately underwent a magnetic resonance imaging of the brain, which documented no abnormalities. No acute infarct was identified within the brain parenchyma, and nor was there any evidence of progressive edema within the optic nerves (Fig. 1). An urgent ophthalmologist consultation was ordered, the findings of which were as follows: bilateral visual loss (blindness), without an alteration in the eye movements, a poor pupillary light reflex, and a normal-appearing optic disc without pallor or edema.

On the second postoperative day, complete visual recovery occurred, which was confirmed by the ophthalmologist. However, the patient was diagnosed with PION on account of this visual field deficit, normal funduscopic examination, and no clinical signs of cerebral impairment.

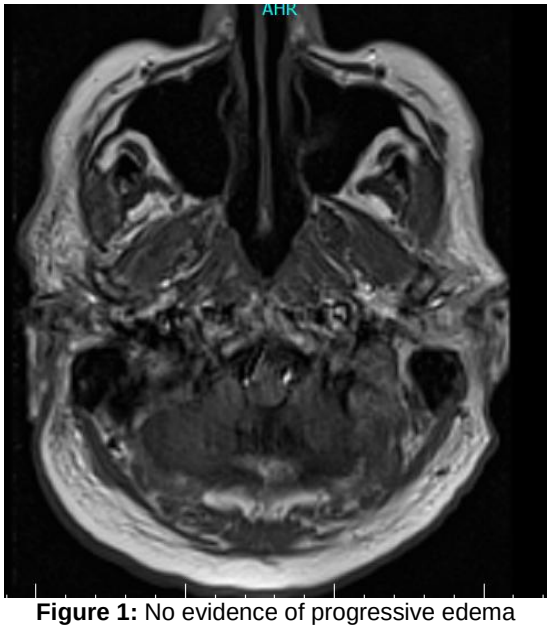

Figure 1: No evidence of progressive edema within the optic nerves was recognized.

DISCUSSION

Perioperative ocular injuries, including visual loss in non-ophthalmic surgery, are rare. Reports indicate that corneal abrasion is the most commonly reported injury and blindness is the least commonly reported injury.[4,8] Unfortunately, the real incidence of perioperative visual loss is not known and reported rates fluctuate significantly. The incidence of perioperative visual loss after non-ocular surgery ranges from 0.002% to 0.0008% of all surgeries to as high as 0.2% of cardiac and spinal surgeries. Nonetheless, after cardiac surgery, the incidence was estimated as high as 0.06% and 0.113% in 2 recent larger studies.[2,15]

The incidence of POVL after cardiac surgery has shown an increase in recent years, which is probably due to the heightened awareness of this complication.

The most common cause of PVOL following cardiac surgery is ION.[5,14] The incidence of ION after cardiac surgery varies significantly, but the incidence rates from large reviews are between 0.028 and 1.3%. [4,13] ION is an infarction of the optic nerve, affecting either the anterior part of the optic nerve (AION) or the posterior segment of the optic nerve (PION), and is caused by the disruption of the blood flow in the small arteries supplying the optic nerve. Both AION and PION are complications of a wide range of surgical procedures.

The type of ION differs based on the type of surgery performed: AION occurs most often after cardiac surgery, while PION occurs most often after spinal surgery in the prone position or radical neck dissection.[8,9] The incidence of PION after cardiac surgery is considered to be extremely low. Large-scale reviews show that the overall incidence rates of AION and PION following cardiac surgery are 0.024% (42/172 569) and 0.0061% (10/164 282), respectively.[4]

Our patient was a really rare case of PION occurring after combined mitral valve replacement and coronary artery bypass

grafting with CPB and we herein discuss the various factors associated with POVL.

Perioperative factors that have been associated with the development of PION after open-heart surgery include intraoperative hypotension, prolonged CPB times, minimum anesthetic durations of 6 hours, blood loss of 1000 mL or greater and anemia, decreased blood oxygen-carrying capacity caused by anemia and intraoperative hemodilution, embolism, the CPB technique, high end lactate levels, and the use of vasopressors.[11,12,14]

Our patient did not have preoperative risk factors for atherosclerotic vascular diseases like hypertension, dyslipidemia, and carotid atherosclerotic plaques; nevertheless, he had intraoperative risk factors such as a prolonged CPB time, an anesthetic duration of loner than 6 hours, hemodilution, a microemboli shower (probably because of the saline test of the mitral valve), cannulation and deairing techniques, the use of the membrane oxygenator for the CPB circuit, a high end lactate level, and the use of vasopressors on rewarming.

In the postoperative setting, since PION is a diagnosis of exclusion and there is no confirmatory diagnostic test for PION, an ophthalmologic examination is essential in all patients to exclude other causes of acute visual loss—including AION, central retinal artery occlusion, and retinal vein occlusion.[4] In our patient, we performed some diagnostic evaluations—including an assessment of giant cell arteritis, ophthalmologic examinations, and brain magnetic resonance imaging—with a view to excluding other causes of acute visual loss. We confirmed the PION diagnosis based on the patient's normal funduscopic examination and magnetic resonance imaging.

Although the incidence of blindness due to PION after cardiac surgery is considered to be extremely low, avoiding this horrific complication is of vital importance and requires the management of the perioperative factors associated with the development of PION following open-heart surgery.

There is a paucity of data regarding the treatment of perioperative PION. There are case reports outlining multiple treatment options—including systemic corticosteroids, intraocular pressure reduction, and the correction of hemodynamic instability.[7] The best option for treating perioperative PION currently is to maintain the intravascular volume by administering intravenous fluids and to replace blood so as to prevent hemodilution and reduction in the oxygen-carrying capacity of blood. Minimizing the use of vasopressor agents may help diminish the risk factors that determine the final visual outcome in PION.

In conclusion, cardiac anesthesiologists and cardiac surgeons should be aware of this rare complication and further investigations about the etiology, prevention, and management of postoperative visual disturbances are required.

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