Case Report

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An Unforeseeable Complication; Posterior Ischemic Optic Neuropathy after Penetrating Injury to the Heart

***Joseph J. Eid*[1]*, *Brian C. Cronin*[2], *Susan Seman*[3]**

[1]*Providence-Providence Park Hospital, Michigan State University College of Human Medicine, Southfield, Michigan, USA*
[2]*St. John Macomb-Oakland Hospital, Warren, Michigan, USA*
[3]*Sinai-Grace Hospital – Detroit Medical Center, Detroit, Michigan, USA*

***Corresponding author:** *Joseph J. Eid*
Address: Providence-Providence Park Hospital,
Michigan State University College of Human Medicine,
Southfield, Michigan, USA
e-mail: eidjose1@msu.edu

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ABSTRACT

Trauma surgeons are currently encountering unusual adverse events after traumatic injuries. Ischemic optic neuropathy is a rare complication that may occur in trauma and burn patients that present in extremis and require massive resuscitation. A 29-year-old male patient sustains a penetrating injury to the heart that required primary repair. He remained hemodynamically stable and required a limited amount of resuscitative fluids and products. Postoperatively, the patient developed acute painless bilateral loss of vision. These findings were consistent with posterior ischemic optic neuropathy. Ischemic optic neuropathy are uncommon entities that arise in trauma patients who require massive resuscitation. Given the limited treatment options, early diagnosis is key in limiting the hemodynamic insult to the optic nerve.

Keywords: Ischemic optic neuropathy; Posterior ischemic optic neuropathy penetrating wound; Postoperative complication.

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Introduction

After improvements in the management of life threatening traumatic injuries and surgical critical care, trauma surgeons are currently encountering rare complications not commonly seen in clinical practice. Ischemic optic neuropathy (ION) is one of the many rare complications arising in trauma and burn patients that require large volumes of resuscitative fluid and blood products [1-4]. Persistent hypotension is the main inciting event and clinical presentation is characterized by acute painless loss of vision [1, 2]. In this case report, we present a case of posterior ION that occurred in a hemodynamically stable trauma patient with limited fluid resuscitation.

Case Report

We present a case of a 29-year-old male patient

who was brought in to our emergency department triage unit after sustaining a penetrating injury to chest. The patient's past medical history was significant for untreated neurosyphilis and Human Immunodeficiency Virus (HIV) infection. The timing of presentation in relation to the initial insult was unknown.

The patient was immediately transferred to the trauma bay where initial vital signs showed a blood pressure of 76/49 mmHg and a pulse rate of 117 beats per minute. These initial findings were concerning for hemorrhagic shock. Primary and secondary surveys were conducted per the Advance Trauma Life Support protocol. During the primary survey the patient was found to have to a Glasgow Coma Scale Score of 7 for which an endotracheal tube was placed for airway protection. The stab wound entry site was at the right parasternal region along the 3rd intercostal space. Decreased breath sounds were noted over the right hemi-thorax, which required placement of a right thoracostomy tube that yielded 200 milliliters (ml) of sanguineous output. Intravenous crystalloid boluses were given, and the massive transfusion protocol was initiated which improved the patient's hemodynamic status with a blood pressure of 98/54 mmHg. A Focused Assessment with Sonography for Trauma (FAST) exam was performed at bedside, with the sub-xiphoid window revealing a significant amount of pericardial fluid. Given the patient's presenting hemodynamic lability, a pericardiocentesis was performed and 60 ml of fresh blood was aspirated. A concern for penetrating injury to the heart prompted an emergent transfer to the operating theater for a median sternotomy and exploration. At the conclusion of the primary and secondary survey the patient had received a total of 2 liters (L) of crystalloids, 2 units (u) of packed red blood cells, 2u of fresh frozen plasma, and 1u of platelets. This totals to 1400ml of blood products.

In the operating theater a median sternotomy was performed, and a significant amount of clotted blood was encountered upon entry into the pericardium. Examination of the heart revealed a single 1-centimeter laceration to the right ventricle that was repaired with a single pledgeted U-stitch polyprolene suture. The patient remained hemodynamically stable throughout the entirety of the procedure and did not require any additional blood products. This prompted discontinuation of the massive transfusion protocol. The patient was successfully extubated in the post-anesthesia care unit after 102 minutes of operative time. The patient required a total of 2 liters of intraoperative crystalloids and estimated blood loss was 300 ml.

Immediately after extubation, the patient complained of acute painless bilateral loss of vision. On ophthalmological examination, the patient was found to have lack light perception, normal intraocular pressures, 3-millimeter pupils with significantly delayed reaction times, and an unremarkable anterior exam. On posterior chamber examination the optic nerve was flat and pink optic with a cup:disc ration 0.3, normal vessels and flat peripheral retina. A CT scan of the head and neck did not visualize any intracranial bleeding, signs of cerebral ischemia, or vascular dissection of the branches of the internal carotid artery. Therein the patient was diagnosed with posterior ischemic optic neuropathy. The patient received supportive care and an alpha-2-adrenergic agonist eye drops (brimonidine). The patient had gradual regain of vision and light perception starting 48 hours after surgery. On postoperative day 3, the patient had full regain of visual acuity.

Discussion

Perioperative vision loss after non-ocular traumatic injury is a rare entity that arises due to relative ischemia to the optic nerve [1, 2]. Ischemic insults to the visual tract and the visual cortex may present similarly. Painless postoperative vision loss has been previously described in trauma [2, 3] and burn [4] patients requiring massive transfusion and resuscitation. It has also been reported in patients undergoing elective abdominal, orthopedic, spinal, and cardiac surgery [5-7].

Anatomically, the optic nerve consists of 4 portions: (1) intra-ocular (anterior), (2) intra-orbital (posterior), (3) intra-canalicular, and (4) intra-cranial. Ischemic optic neuropathies classically affect the intra-ocular and intra-orbital portions of the optic nerve: therefore, described as anterior (AION) or posterior ischemic optic neuropathy (PION). The watershed area in the anterior portion and pial plexus along the posterior portion of the optic nerve predisposes it to significant ischemic insult in the face of hemodynamic instability [8, 9]. Clinically patients report acute, unilateral or bilateral, partial or complete vision loss. On examination, patients with ION have afferent pupillary reflex deficit, visual field defects. While PION has no obvious findings on funduscopic examination, AION presents with pale disc edema.

In our case report, the patient reported painless loss of vision immediately after extubation. Fundoscopic examination was normal. A brain CT was performed which did not reveal any acute intra-cranial bleeding, dissection of the internal carotid artery, occlusion of the ophthalmic artery, or ischemia in the visual cortex. An echocardiogram with a bubble study essentially eliminated the possibility of an air embolus that may have migrate to the arterial circulation via a congenital patent foramen ovale or an acquired defect in the ventricular septum secondary to the penetrating injury. All these findings were consistent with PION. The patient did not have any previous visual deficits, however his history of neurosyphilis may have predisposed him to this ischemic insult.

AION occurs at a rate of 2.6% in trauma patients which may be secondary to prone position and massive resuscitation [2]. Previous reports of post-traumatic ION have been associated with severe prolonged hypotension, need large volume resuscitation, and prolonged ventilator days [1-4]. Kudo *et al*., [3] described two cases of blunt trauma requiring where one patient required 38.4 liters of crystalloid and 6 liters of blood product during the perioperative period. The second reported patient required even large volumes of resuscitative fluid up to 50 L and 10 L of blood products [3]. Similar findings are seen in patients with large burns requiring large volume of crystalloid resuscitation. In a case series by Medina *et al*., [4] patients with OIN had burn surface areas ranging 57-68%, requiring 20-47.6 L of crystalloids within the first 24 hours after the initial insult. However, in our patient the total volume of resuscitative fluid was 5.4 L which is merely a fraction of what is reported in the literature.

The best of our knowledge, this is the first report to describe PION in the setting of transient perioperative hypotension, relative hemodynamic stability, and limited resuscitative fluid intake. Given the limited treatment options, trauma surgeons should remain vigilant of this entity to avoid ongoing hemodynamic insult to the optic nerve.

Ethics approval and consent to participate: Institutional Review Board approval was not required for this case report.

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Conflicts of Interest: None declared.

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