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Post Appendectomy Blindness, Extremely rare complication after abdominal surgery

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ABSTRACT

Perioperative visual loss (POVL) is a drastic complication that can result after ocular and more commonly non-ocular surgery, mostly reported after spine and cardiac bypass procedures. Despite the rarity of such complication, it has been reported following the abdominal surgeries. Overall, the most common cause of POVL is ischemic optic neuropathy but any pathology to the optical system from the cornea to the occipital lobe can lead to this rare phenomenon. Here, we are reporting the second case in the literature of post-operative visual loss after laparoscopic appendectomy. A young female, with no underlying disease, underwent laparoscopic appendectomy after septic shock secondary to acute appendicitis. Postoperatively, patient complained of complete blindness after extubation. Neurologic examination revealed bilateral near complete blindness, and hemodynamic ischemic stroke in the occipital cortex.

Keywords: Appendectomy; Postoperative visual loss (POVL); Cortical blindness

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Introduction

Appendectomy is a one of the most common surgical procedure performed in emergency general surgery. Complications related to appendicitis are well known and usually recognized and managed by the surgical team. Post appendectomy visual loss is extremely rare complication following abdominal surgery and was only reported once before [1].

Case Description

A 32-year-old Indian female, presented to the Emergency Department due to abdominal pain of 2 days duration, with associated nausea, vomiting, fever and normal bowel habit. The patient had no underlying chronic medical problem. In emergency, she had tachycardia with heart rate of 130 beats per minute and hypotension with blood pressure of 80/50mmHg. Despite 2 liters of IV fluids, the patient was persistently hypotensive, signifying septic shock, and her hemoglobin was 9 g/dL, so a unit of Packed Red Blood cells was transfused during resuscitation. Her labs were significant for white cell count of 2.8, and lactate of 4.9.

She was continuously resuscitated with aggressive fluid and intravenous Tazocin, and diagnosis of appendicitis was made on computed tomography (CT), of the abdomen and the patient was shifted to operation room for Laparoscopic appendectomy.

Anesthesia was induced with a bolus injection of ketamine (2 mg/kg), propofol (1.5 mg/kg), and rocuronium (0.6 mg/kg) in combination with fentanyl infusion at 150µg. Anesthesia was maintained with sevoflurane and air/oxygen. Noradrenaline (NE) 0.07mcg/kg/min started in the operating room after successful central line insertion, no pre-operative vasopressor was given. The patient was placed in Trendelenburg position for the operation. Vital signs after induction were blood pressure 150/80 mmHg on noradrenaline, heart rate 100 beats/min and oxygen saturation 100%. Intraoperative vital signs were within normal range.

The laparoscopy showed severely inflamed gangrenous appendix with pus in the pelvis, healthy base was secured with PDS loop and appendectomy performed along with abdominal irrigation and suction of purulent fluid. The total anesthesia time was two and half hours and the surgery time was almost half of anesthesia. Post operatively, the patient was transferred to Surgical ICU, kept intubated and noradrenaline was continued.

The patient was successfully extubated and NE weaned off on her first post-operative day. Immediately after extubation, the patient was unable to see (black vision) and neurologic examination revealed that she can open her eyes on command, there was intact pupillary light reflex, normal extraocular muscle movements, intact macula and optic disc on fundoscopy.

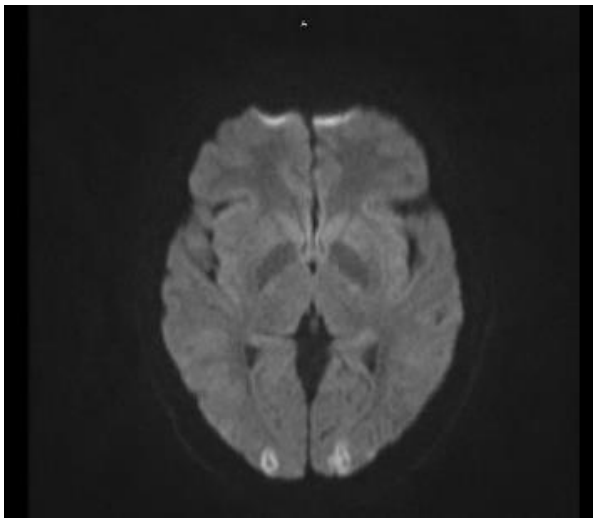
Due to suspected acute central blindness, CT head, CT angiogram and CT Brain perfusion study was obtained urgently which were normal. On second post-operative day 2, the patient vision improved as she was able to see blurred faces and MRI head and MRAngiography obtained with radiologic findings suggestive of bilateral occipital lobes cortical ischemic changes/ laminar necrosis.

Meanwhile, the patient was in stable condition, her bowel regained function and she started on soft diet. She was treated as central stroke, maintaining euvolemic status and aspirin was added.

The next day, the patient's vision was significantly improved bilaterally with minimal blurring and no hemianopia. The patient showed no pertinent features in the evaluation of possible underlying pathology including echocardiogram, MRA neck and hypercoagulable status. Her

symptoms were likely due to hemodynamic ischemic stroke in the occipital cortex, and the patient's vision was improved but did not return back to normal preoperative level.

The patient was transferred to the ward on her fifth post-operative day, her vision and visual fields were improved and she was discharged on sixth postoperative day. In outpatient follow up at the 17th postoperative day, her visual field was considerably improved as compared to the day she was discharged from the hospital. A new complain of headache started affecting her lifestyle, without any neurological symptoms, she was advised to maintain a headache diary until the next visit but the patient did not show up in the clinic.



[picture1: MRI head showing bilateral cortical infarction]

Discussion

Perioperative visual loss (POVL) after non-ocular surgery describes either unilateral or bilateral visual impairment that occurs after general surgery apart from ophthalmic operations. It occurs most likely after cardiac, lower spine, vascular or reconstructive surgeries. [2,3,4] Moreover, although rare, Chaudhry et al. reported that POVL can occur after a short period of abdominal surgery such as cholecystectomy or appendectomy [2].

The estimated number of POVL are usually retrieved from retrospective studies and case series. The prevalence of perioperative visual loss in the United States, over a 10 years period reported that the national estimate in cardiac surgery was 8.64/10,000 and 3.09/10,000 in spinal fusion. After searching PubMed and Google scholar, only one case of post appendectomy visual loss was reported from Korea, in 2014 [5].

Risk factors include hypotension and the use of vasopressor as with our patient, other including prone position, long duration of surgery, massive bleeding and increased venous pressure. The etiology of POVL remains incompletely understood and any portion of the visual system may be involved, from the cornea to the occipital lobe. Overall, the causes range from corneal abrasion, postoperative ischemic optic neuropathy, cerebral visual loss, and central retinal artery occlusion. [6]

The most common cause of ocular complaint postoperatively is the corneal abrasion, which is usually painful, benign, diagnosed with fluorescein stain and slit lamp exam and resolve within 24-48 hours using an eye lubricant. On the other hand, the most common cause of permanent

POVL in adults after non-ocular surgery is ischemic optic neuropathy, which can be arterial or non-arterial and of two types: anterior ischemic optic neuropathy (AION), affecting the optic disc; and posterior ischemic optic neuropathy (PION), which is retrobulbar or posterior to the lamina cribrosa. Distinguishing the two types can be accomplished after fundoscopic examination. [7,8,9]

Postoperative cortical blindness results from embolism, generalized cerebral under perfusion, or both. Profound hypotension to the watershed parieto-occipital region was the justification to the complete blindness that happened in our patient, in contrast to the previously reported case where cortical blindness was due to cerebral bleeding which was believed to be from high intracranial pressure resulting from Trendelenburg position and pneumoperitoneum. [10]

It has been suggested that repeated short episodes of extreme hypotension are more likely to result in cortical blindness than a longer one of moderate hypotension. [8] Symptoms may be immediate, identified in the first postoperative day or, as we had with this case, delayed to the second postoperative day onward.

Diagnosis of cortical blindness started by physical examination where pupillary light reflex and fundoscopic examination are usually normal, in contrary to the optic neuropathy. There might be unilateral or bilateral homonymous hemianopia, complete blindness, or other focal neurological pathologic deficits depending on the extent of injury to the cortex. Computed tomography or magnetic resonance imaging can help to identify the location of the lesion. [11]

The prognosis of postoperative parieto-occipital stroke ranges from total permanent blindness to brief periods of transient ischemic attacks with full recovery of visual acuity. Cortical blindness is generally seen as permanent after the window for spontaneous recovery has passed, which is thought to be several months post-lesion. [8] Our patient had near complete recovery.

Conclusion

POVL is rare but serious complication as visual loss might be permanent, the diagnosis can be established by thorough ocular examination and fundoscopy whereas CT and MRI brain can elucidate the central causes. The treatment is usually expectant and anticoagulant like aspirin can be advised in cases of ischemic stroke. The recovery is usually not up to the preoperative level as some visual loss is permanent.

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