A patient of terson’s syndrome for ocular surgery: Perianesthetic concerns

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ABSTRACT

Terson’s syndrome may be challenging for the anesthesiologist in view of its multisystem involvement including neurological, cardiovascular, and ophthalmological involvement. We describe anesthetic management of a 55-year-old male having Terson’s syndrome for pars plana viterctomy.

Key words: Anesthesia, intraocular hemorrhage, subarachnoid hemorrhage, Terson’s syndrome

INTRODUCTION

In 1881, Litten first described an intraretinal hemorrhage associated with subarachnoid hemorrhage in the German literature.[1] The syndrome of intra-vitreous bleeding in association with subarachnoid hemorrhage was first described by French ophthalmologist Albert Terson in 1900.[1] Terson syndrome now encompasses any intraocular hemorrhage associated with intracranial hemorrhage and elevated intracranial pressures. It has been reported that such intraocular hemorrhage in patients with subarachnoid hemorrhage leads to increased mortality.[1] Vitreous hemorrhage is an adverse prognostic finding in patients with subarachnoid hemorrhage.[1] The presence of Terson syndrome has been reported to be predictive of poor clinical outcome in terms of recovery from the intracranial hemorrhage.[2] Such patients not only have intracranial pathology but also have intraocular lesion and thus mandating careful management with some peculiar concerns in the perioperative period. Such patients may be scheduled for one of these surgical procedures but care needs to be taken to prevent exacerbation of pathology in the other site. Though the surgical concerns and its management have been reported in the literature but perioperative concerns related to anesthetic management has not been reported for the Terson’s Syndrome.

CASE REPORT

A 55-years-old male weighing 60 kg was diagnosed with bilateral vitreous hemorrhage and scheduled for pars plana vitrectomy. He was known hypertensive since 1 year and was managed with oral amlodipine (5 mg) twice a day and losartan (50 mg) once a day. Since 4 months, patient started having progressively increasing loss of vision which was followed by sudden onset headache associated with vomiting and subsequently loss of consciousness. Pupils of left eye were dilated and sluggishly reacting while right eye was of normal size and sluggishly reacting. Magnetic resonance imaging (MRI) of head revealed left frontal intracranial hemorrhage with intraventricular extension. Diagnosis of stroke with left frontal bleed was made. He was kept on conservative management with continuation of antihypertensive drugs. The patient’s consciousness gradually improved. GCS was E4 M6 V5, motor power was 5/5 with down going planters. He had a full recovery after 1 month. The vision did not improved and there was progressively increasing loss of vision. On further evaluation, it revealed hypertensive bleed in the eye and magnetic resonance angiography was
done which could not rule out aneurysmal bleed. He was planned for pars plana vitrectomy.

Preoperatively, patient was conscious and maintaining vitals. His routine investigations including 12 lead electrocardiogram, chest X-ray, hemogram, liver, and kidney function test were normal. His antihypertensive drugs were continued and was premedicated with oral alprazolam (0.5 mg) night before the surgery. He was also administered oral ranitidine (150 mg) night before the surgery and in the morning after the surgery. A fasting status of 6 h for solid food and 2 h of clear fluid was advised.

In the operating room, monitors including 5 lead electrocardiogram, non invasive automated blood pressure, and pulse oximeter were attached. After securing intravenous access, intravenous midazolam (1 mg) and fentanyl (120 µg) were administered. Anesthesia was induced with propofol (120 mg) and lungs were ventilated with 3% sevoflurane in nitrous oxide and oxygen (50:50). After achieving neuromuscular blockade with rocuronium (40 mg) and intravenous administration of preservative lidocaine (60 mg), airway was secured with proseal laryngeal mask airway (PLMA). Anesthesia was maintained with sevoflurane in nitrous oxide and oxygen (MAC 1) and boluses of rocuronium (10 mg) and fentanyl (20 µg). After the surgery, the residual neuromuscular blockade was reversed with neostigmine (2.5 mg), glycopyrollate (0.4 mg) and PLMA was removed after adequate respiratory efforts were present. The patient had an uneventful recovery and discharged 3 days later for further follow up in ophthalmology and neurology clinics.

**DISCUSSION**

Surgical intervention in patient with Terson syndrome is challenging for anesthesiologist. Papilledema and unconsciousness are both positively correlated with Terson syndrome. The pathogenesis of Terson syndrome has been controversial. The earliest reports assumed that the intracerebral blood directly connected with the intraocular space through the lamina cribrosa. While electron microscopy of the optic nerve anatomy has not demonstrated a communication between the two spaces but bilateral optic nerve sheath hemorrages following rupture of an anterior choroidal artery aneurysm resulting in a Terson syndrome has been described. Pathological specimens have not shown any blood in the optic nerve sheath within 3 mm of the globe. The most commonly cited mechanism is that elevated intracranial pressure resulting from subarachnoid hemorrhage is transmitted within the optic nerve sheath and obstructs intraocular venous drainage causing distension and rupture of peripapillary and retinal capillaries resulting in significant hemorrhage in the subhyaloid space or vitreous cavity. Because of these assumptions regarding the pathogenesis of the syndrome, the definition of Terson syndrome now includes any intraocular hemorrhage associated with intracranial bleeding and acutely increased intracranial pressure. The neurologic symptoms are related to intracranial bleeding.

Though the chance of associated intracranial aneurysm is controversial but its occurrence has been reported with increased mortality. So it becomes prudent whenever patient for ocular surgery with suspected Terson’s syndrome needs to rule out the presence of intracranial aneurysm which may get ruptured during anesthetic management if adequate blunting of stress response is not provided. Retinal hemorrhage has been described in association with valsalva maneuver, such as forceful vomiting or coughing. So care should be taken during anesthetic management as to avoid any such increase in intrathoracic pressures.

Terson syndrome is a relatively common complication of subarachnoid hemorrhage that may require early surgical attention to prevent long-term vision loss and hasten recovery. One patient had progressively increasing vision loss mandating intraocular surgical intervention (pars plena vitrectomy).

Preanesthetic evaluation should determine whether rupture had occurred and signs of intracranial hypertension should be sought. Generally, most patients have normal intracranial pressure by the time they come for surgery. A small group of patients, however, may have persistent elevation in intracranial pressure. Hydrocephalus develops in these patients as a result of interference with absorption of CSF and is usually evidenced by ventricular enlargement on the CT scan. The possibility of Terson’s syndrome should be considered in every patient with subarachnoid hemorrhage. In case patient is undergoing some intracranial procedures, then any visual loss postoperatively may also occur because of the mechanism involved in Terson’ syndrome in addition to other common causes of visual loss after anesthesia (general/regional). A variant of Terson’s syndrome has also been proposed where in visual loss after anesthesia (general/regional).

In addition to neurological findings, evaluation should include a search for coexisting diseases that may modify the use of elective hypotension intraoperatively. Preexisting hypertension and renal, cardiac, or ischemic cerebrovascular disease are relative contraindications to controlled hypotension. Electrocardiographic abnormalities are commonly seen in patients with subarachnoid hemorrhage.
but do not necessarily reflect underlying heart disease. Sudden increases in blood pressure with tracheal intubation or surgical stimulation should be avoided. Patients with persistent elevation in intracranial pressure should receive little or no premedication to avoid hypercapnia. Treatment of hypertension from pain and anxiety needs to be adequately managed. Intravenous lidocaine during endotracheal intubation could be one of the option to obtund hypertensive response. Short-acting hypotensive drugs (esmolol, labetolol, or nitroprusside) for control of labile hypertension or transient hypertension should be considered. Elevated head positioning with bed rest and avoidance of anticoagulation medications (e.g., aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), warfarin) may be helpful.

CONCLUSION

Terson’s syndrome may be related to acute elevation of intracranial pressure, independent of its causes, and may occur with similar incidence in patients with severe brain injury and those with subarachnoid hemorrhage. Because recognition and treatment of Terson’s syndrome may prevent visual impairment and associated secondary damage to the eye, increased awareness of this entity in all patients with acute raised intracranial hypertension is recommended.

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