

Case Report

Penetrating Injury of the Oropharynx with Tonsillar Prolapse in a Patient with Hepatic Cirrhosis and Portal Hypertension: Anesthetic Management

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Abstract

A 52-year-old man, a known case of hepatic cirrhosis and portal hypertension, presented with a bleeding lacerated wound of his pharyngeal wall and tongue with prolapse of the tonsil. He underwent emergency surgical repair of the laceration. We discuss the airway management and other anesthetic implications of this case.

Key words: Airway management, hepatic cirrhosis, neck trauma, pharyngeal lacerations, portal hypertension

INTRODUCTION

Trauma to the neck poses a considerable challenge to the anesthesiologist. Penetrating trauma to the pharynx is associated with danger to the airway as well as injury to important structures such as large blood vessels, nerves, and cervical spine in the vicinity of the pharynx. Violation of the retropharyngeal and the parapharyngeal spaces can cause complications such as dissecting emphysema, mediastinitis, and major vascular bleed. During surgical repair of such pharyngeal lacerations, airway management mandates special importance as the anesthesiologist has to share the surgical field with the surgeon. In such patients, comorbid illnesses like hepatic cirrhosis and portal venous hypertension demand special attention during general anesthesia. We discuss the successful management of a patient who presented for emergency surgical repair of his pharyngeal wall laceration and who was also a known case of hepatic cirrhosis with portal venous hypertension.

CASE REPORT

History

A 52-year-old man presented to the emergency department (ED) of a tertiary care hospital with profuse bleeding from his oral cavity, following alleged history of trauma to his throat with a flashlight during a fall at home. He was an alcoholic and

a diagnosed case of hepatic cirrhosis with portal venous hypertension. He had undergone banding of the bleeding esophageal varices 3 months prior to this. He was on treatment with oral propranolol for variceal bleed prophylaxis. There was no history of alcohol consumption immediately prior to the occurrence of the event. He had no other significant comorbid illnesses.

Physical examination

The patient was conscious, oriented, and cooperative. He weighed 60 kg and was pale. His pulse rate was 60 beats per minute that was regular in rhythm, blood pressure (BP) was 110/70 mmHg, and respiratory rate was 18 breaths per minute.

His abdominal examination revealed mild ascites and no hepatomegaly. Signs of hepatic failure such as spider naevi, scanty axillary hair, and parotid swelling were present. Other systemic examination did not reveal any abnormality.

Examination of the oral cavity revealed a profusely bleeding lacerated wound that was 5 cm long in the right lateral pharyngeal wall extending through the tonsillar pillars, resulting in incomplete avulsion of the right palatine tonsil into the oral cavity [Figure 1]. There was also a 2-cm-long laceration on the lateral border of the tongue in its posterior one-third.

Investigations

Hemoglobin : 8 g/dL

Total white blood cell (WBC) count : 9000/ μ L

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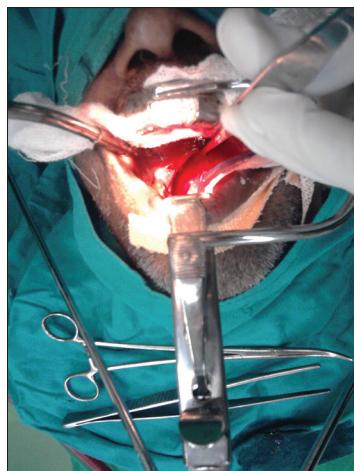


Figure 1: View of the pharynx during surgery

Differential count: Neutrophils - 72%

Lymphocytes : 24%

Monocytes : 2%

Eosinophils : 2%

Packed cell volume : 25%

Platelet count : 1.1 lakh/ μ L

Blood urea : 32 mg/dL

Serum creatinine : 1.1 mg/dL

Random blood sugar : 112 mg/dL

Serum sodium : 132 mEq/L

Serum potassium : 4.0 mEq/L

Serum chloride : 101 mEq/L

Serum bilirubin (total) : 1.5 mg/dL

Serum bilirubin (direct) : 0.8 mg/dL

Serum glutamic oxaloacetic transaminase (SGOT) : 85U/L

Serum glutamic pyruvic transaminase (SGPT) : 70U/L

Serum alkaline phosphatase : 98 IU/L

Serum albumin : 3.1 g/dL

Albumin: Globulin : 1.9

Prothrombin time/international normalized ratio (PT/INR) : 18 s/1.2

Electrocardiogram (ECG) showed poor R wave progression from lead V1 to lead V3.

X-ray of his chest did not reveal any abnormality.

X-ray scan of his cervical spine did not reveal any abnormality. Computed tomography (CT) scan of his head and neck also did not reveal any abnormality other than partial avulsion of the right palatine tonsil. Indirect laryngoscopy done by the

ear, nose, and throat (ENT) surgeon did not reveal laryngeal edema, distortion of the laryngeal anatomy, or mucosal tear.

Anesthetic management

The patient was adequately nil by mouth. He was moved into the operating room and monitors including noninvasive BP cuff, peripheral capillary oxygen saturation (SpO_2), ECG, and end-tidal CO_2 ($EtCO_2$) were attached. The following baseline vitals were noted: Heart rate 60 bpm, BP 110/70 mmHg, and SpO_2 100% on breathing ambient air. He was premedicated with intravenous (IV) metoclopramide 10 mg, IV glycopyrrolate of dosage 0.2 mg, IV ranitidine 50 mg, IV midazolam 1 mg, and IV fentanyl 2 μ g/kg. Difficult airway cart was kept ready.

After adequate preoxygenation, rapid sequence induction (RSI) was done with IV 2.5% thiopentone sodium 300 mg, IV succinylcholine 100 mg and cricothyroid pressure. Laryngoscopy was performed carefully and orotracheal intubation was done with a 8.0-size cuffed portex endotracheal tube and the position of the tube was confirmed. Maintenance of anesthesia was achieved using O_2 , N_2O and sevoflurane. Muscle relaxation was maintained with IV atracurium 0.5 mg/kg initial bolus and then with two subsequent maintenance doses. Further, two doses of fentanyl 1 μ g/kg were administered hourly to maintain analgesia.

Intraoperative heart rate, BP, SpO_2 , $EtCO_2$, and urine output of the patient were monitored. His vitals remained stable throughout the surgery that lasted for 2 hours. After repair of the laceration, the ENT surgeon carefully inserted a nasogastric tube and decompressed the stomach.

Reversal of neuromuscular blockade was achieved with IV neostigmine 0.05 mg/kg and IV glycopyrrolate 0.01 mg/kg. The patient was extubated when he was fully awake after thorough and gentle oropharyngeal suctioning and after ensuring positive "cuff-leak test."

After the surgery, the patient was shifted to the intensive care unit (ICU) for monitoring and observation. His head was elevated to 30° and humidified oxygen was administered. After an uneventful stay in the ICU, he was shifted to the general ward. He was monitored for 24 hours in the general ward for neurological deficit that fortunately did not develop in this patient.

DISCUSSION

Oropharyngeal injuries

Oropharyngeal impalement injuries are potentially life-threatening injuries with variable manifestations. They commonly occur in children, especially toddlers, due to their propensity to fall easily while carrying objects in their mouths.^[1,2] Not much literature is available on oropharyngeal injuries in adult patients. The most common areas of injury are superior to the tonsil and the posterior pharyngeal wall, with other common sites including the dorsum of the tongue and the palate. Penetrating trauma to the pharynx is associated with danger to the airway as well as injury to important structures such as large blood vessels, nerves, and cervical spine in the vicinity of the pharynx.

Soft tissue injuries of the oropharynx include abrasions, contusions, and lacerations of the tongue, palate, pharynx, cheek, parotid gland, and facial nerve. Airway management in oropharyngeal injury is challenging due to multiple concurrent factors such as:

- Hemorrhage and debris causing impaired laryngoscopy and visualization during intubation
- Aspiration due to swallowing of large quantities of blood
- Clot inhalation causing airway obstruction
- Soft tissue edema and hematomas.

The immediate priorities in the management are the following:^[3]

- Assess and monitor for signs of airway obstruction
- Clear airway to assist in breathing
- Definitive airway intervention.

Figure 2 shows an airway management algorithm for maxillofacial trauma. Massive airway edema may evolve over 24-48 hours and can cause gross distortion of the soft tissue structures. An initially unobstructed airway may become compromised during this period. Careful close monitoring in an ICU or high dependency unit (HDU) is recommended. Head elevation to 30° and administration of humidified oxygen can ameliorate the likelihood of delayed airway compromise. Sudden airway obstruction may occur with clot dislodgement and inhalation. The signs of partial airway obstruction include noisy breathing, stridor, intercostal retraction, supraclavicular tug and restlessness.

One of the dreaded complications of oropharyngeal injuries is intravascular thrombosis of the internal carotid artery, which has devastating neurologic manifestations.^[4] The majority of injuries resulting in THROMBOSIS of the internal carotid artery occur in the peritonsillar region. The symptoms of this complication are usually delayed. There may be life-threatening sequelae, which include occult internal carotid artery damage with aphasia, hemiplegia, and even death.^[5] The proposed mechanism of injury is compression of the internal carotid artery between the penetrating object and the transverse process of an upper cervical vertebra. This leads to a tear in the intima of the vessel and thrombus formation that dislodges and embolizes to the brain. "Lucid interval" is the time interval during which a thrombus is formed and propagated to the cerebral vessels and it usually ranges 3-60 hours after the injury.^[6]

Oropharyngeal penetrating injuries can result in breach of the retropharyngeal and the parapharyngeal spaces, leading to complications such as dissecting emphysema, mediastinitis, and major vascular bleed. Other non-neurologic sequelae may occur such as retropharyngeal abscess, facial cellulitis, velopharyngeal insufficiency, and pneumomediastinum.

Diagnosis can be radiologically done using plain lateral neck radiographs as well as contrast-enhanced CT angiography.^[7] In cases with pharyngeal perforation, plain lateral soft tissue radiographs can aid in diagnosing the presence of retropharyngeal air. CT angiography is a quick and convenient method for promptly diagnosing internal carotid artery injury.^[8]

When presented with a case of oropharyngeal injury, it is important to get a thorough history and perform physical examination with detailed neurological evaluation. Mask ventilation, laryngoscopy, and endotracheal intubation could be impossible due to the presence of a protruding foreign body. Dislodgment of the object before intubation could cause bleeding and compromise airway. In this particular case, there was no protruding foreign body and there were no signs of airway obstruction. The indication for emergency surgical repair was bleeding. An indirect laryngoscopy, if feasible, will give us an idea about laryngeal anatomy, distortion, or mucosal edema that will further help in planning the airway management.

Even though these patients may be adequately nil by mouth, we have to consider them as full stomach, taking into consideration the fact that a considerable amount of blood might have been swallowed. Hence, RSI is preferred.

In this case, the ideal method of securing the airway would have been endotracheal intubation using fiber-optic laryngoscopy awake endotracheal intubation. As a fiber-optic laryngoscope was not available in our emergency operating room, the ENT surgeon performed an indirect laryngoscopy that did not reveal any anatomical distortion, mucosal tear, or laryngeal edema. CT scan of the patient's head and neck also supported the same findings. We proceeded with direct laryngoscopy and orotracheal intubation. Laryngoscopy has to be done carefully and gently, and is to be preferably performed by an experienced anesthesiologist. Nasogastric tube has to be passed for gastric decompression and also for the purpose of postoperative feeding. These patients have to be extubated only after confirming a "positive cuff-leak test."

Postoperatively, they have to be monitored in an ICU or an HDU. Head-end elevation to about 30° is preferred. It is preferable to monitor these patients for at least 48 hours for complications such as airway edema, rebleed and internal carotid artery thrombosis.

Cirrhosis

Patients with liver disease are at a particularly high risk for morbidity and mortality in the postoperative period due to both the stress of the surgery and the effects of general anesthesia. Furthermore, decompensated liver disease increases the risk of postoperative complications. Assessing risk in these patients is a challenging but important endeavor. The liver is vital for protein synthesis, coagulation homeostasis, glucose homeostasis, bilirubin excretion, drug metabolism, and toxin removal. Secondary to loss of hepatic reserve capacity and other systemic derangements that are the result of liver dysfunction, patients with liver disease exhibit an inappropriate response to surgical stress. These individuals are accordingly at an increased risk of bleeding, infection and postoperative hepatic decompensation including hepatic coma or death.

Assessing the risk in these patients depends upon the type of surgery and severity of the liver disease. Cirrhotic patients have decreased plasma-binding proteins and

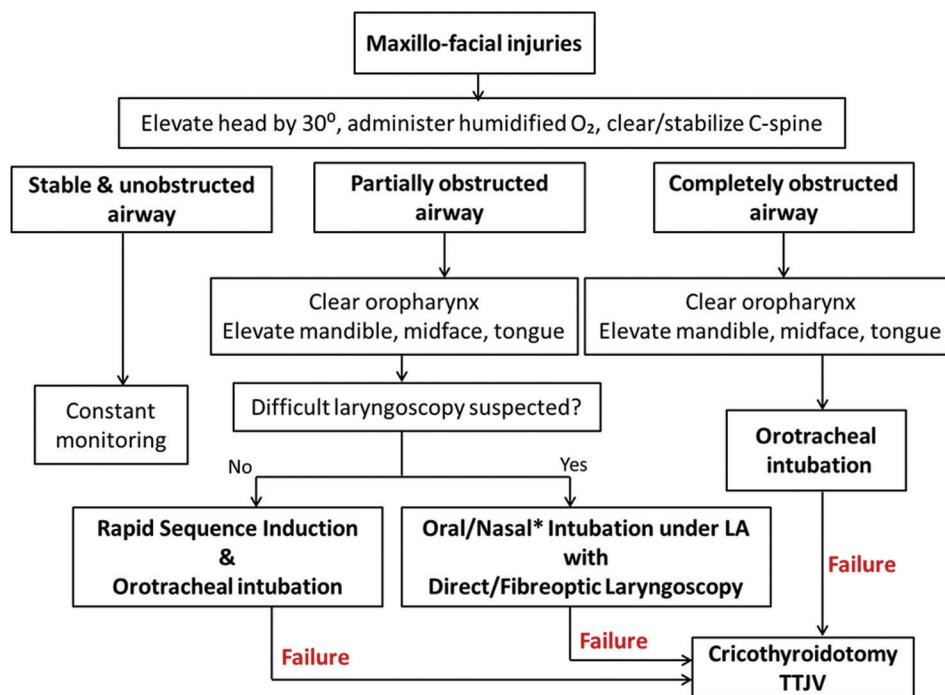


Figure 2: Algorithm for airway management in maxillofacial and oropharyngeal injuries

hypoalbuminemia impairs drug-binding. Impaired drug metabolism, detoxification, and excretion by the liver prolong drug half-life. Thus, absorption, distribution, metabolism, and excretion of the anesthetics, muscle relaxants, analgesics, and sedatives may be affected.^[9]

The analgesic used in our patient was fentanyl as it is the best available and preferred narcotic in such cases. Thiopentone sodium was carefully used in titrated doses as it depends on the liver for metabolism but it does not cause much hemodynamic instability. In contrast, propofol, as an inducing agent, could cause hemodynamic instability and hence, was not used in this patient.

The drug effects of neuromuscular blocking agents may be prolonged in patients with liver disease due to impaired biliary excretion. Atracurium is the agent of choice as it is excreted by Hofmann elimination and so does not depend on the kidney or the liver for elimination. Among the inhalational anesthetics, isoflurane has fewer effects on hepatic blood flow and less hepatic metabolism and hence, preferred and was used in this patient. Normocarbia and normothermia were maintained in the patient. After the surgery, the patient was monitored in the general ward for any untoward complication.

CONCLUSION

Surgery in a patient with penetrating pharyngeal trauma and cirrhosis poses a formidable challenge. Clinical signs might not be very evident; therefore, a high index of suspicion is necessary so that the life-threatening complications are not neglected. Knowledge of airway management in trauma, effects of the

drugs used in anesthesia in cirrhotic patients, and anticipation of the complications will help to reduce perioperative morbidity.

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