Anesthesia for Traumatic Diaphragmatic Hernia Associated with Corneal Laceration

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Conflict of interest: None declared

Patient: Male, 37
Final Diagnosis: Diaphragmatic hernia
Symptoms: Dyspnea
Medication: —
Clinical Procedure: CT-scan
Specialty: Anesthesiology

Objective: Rare co-existence of disease or pathology
Background: Diaphragmatic rupture can be seen in up to 5% of car accidents, and 80%-100% of diaphragmatic hernias are associated with other vital organ injuries. Brain, pelvis, long bones, liver, spleen, and aorta are some other organs that can be severely damaged and need different anesthetic managements.
Case Report: A 37-year-old male victim of a head-on collision who was suffering diaphragmatic rupture and corneal laceration was prepared for an emergency operation 11 hours after the car accident. Gastric decompression, pre-oxygenation, rapid sequence induction with succinylcholine, immediate use of non-depolarizing muscle relaxant, and mechanical ventilation with low tidal volume after intubation were used in anesthetic management of the patient.
Conclusions: Because of the high prevalence of coexisting pathologies with traumatic diaphragmatic hernia, anesthetic management must be tailored to the associated pathologies.

MeSH Keywords: Anesthesia • Corneal Diseases • Hernia, Diaphragmatic

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/897908
**Background**

Diaphragmatic rupture can be seen in up to 5% of car accidents. In majority of these cases, the left side of thorax is involved [1–5]. Most (80–100%) of diaphragmatic hernias are associated with other vital organ injuries. Brain, pelvis, long bones, liver, spleen, and aorta are some other organs that can be severely damaged [6–8] and need different anesthetic managements. Most of the proposed anesthetic management strategies in the literature did not consider the influences of these associated injuries on our practice, and there are also some controversies about the pathophysiologic aspects of the disorder.

**Case Report**

Five hours after a car accident, a 37-year-old male victim of a head-on collision was admitted to Hazrat Rasol Medical Complex (Tehran, Iran). In the first assessment the patient was in a sitting position, was alert (Glasgow Coma Scale [GCS]=15), and did not have shortness of breath. Vital signs were as follows: blood pressure (BP)=130/100 mm Hg; heart rate (HR)=100; respiratory rate (RR)=9; temperature 37°C, and O₂ saturation 90% (breathing room air). Head and neck exam revealed laceration of left cornea. There was no sign of neck vein engorgement, deviation of trachea, subcutaneous emphysema, rhinorrhagia, otorrhagia, or cerebrospinal fluid (CSF) leakage. Decreased breathing sound was detected in the left lung. Tenderness in left upper quadrant (LUQ) was recorded. Computed tomography (CT) scan and chest X-ray indicated gastric herniation into left thoracic space (Figures 1, 2). Patient lab data were as follows: hemoglobin (Hb)=12.1; hematocrit (Hct)=41.4; prothrombin time (PT)=13; partial thromboplastin time (PTT)=30 international normalized ratio (INR)+1; blood urea nitrogen (BUN)=18; creatinine=1.3; Na=145, K=4; blood sugar (BS)=193; pH=7.27; PCO₂=5; Po₂=132.

Two hours after admission, patient felt dyspnea in the sitting position. No changes in hemodynamic indices were detected, and O₂ saturation was 97% with oxygen mask. On ophthalmologic exam, the patient found to have a left corneal full thickness laceration, iris prolapse, and lens dislocation.

About 11 hours after the accident, the patient was sent to the operating room. His vital signs were stable. Peripheral capillary oxygen saturation (SpO₂) was 86% (room air). Volume replacement was done with 1000 mL of Lactated Ringer's solution. A nasogastric (NG) tube was already fixed in the emergency room. Three large-bore IV lines were inserted. Standard monitoring and arterial line were installed. After complete readiness of the surgical team, 10 mg of metoclopramide, 150 µg of fentanyl, 2 mg of midazolam, and 5 mg of atracurium were administered as premedication, and pre-oxygenation was performed without any pressure on the patient’s globe. Propofol 160 mg and succinylcholine 110 mg were used for induction. The patient was intubated with a single lumen endotracheal tube. Atracurium 40 mg was administered before return of
Diaphragmatic hernia can compromise cardiorespiratory function by different mechanisms [6,9,10]. Multiple associated injuries may complicate the patient’s condition and delay the diagnosis. Paraclinical diagnostic methods are usually not conclusive. In 30–69% [1,11] of patients, the diagnosis is made during surgery. In 4.5–15% of cases, the diagnosis remained undiscovered in the postoperative period [1,6]. Peritoneal lavage, thoracoscopy, and laparoscopy [12] are also used to confirm the diagnosis of diaphragmatic hernia. A high degree of suspicious is always the key for rapid diagnosis. Severity of coexisting injuries is the major determinant of the high mortality rate (13–25%) [2,4], and urgent surgery is usually indicated.

Although different anesthetic methods have been proposed for management of these patients [13], the usual recommended methods of anesthesia are awake intubation, crash induction, NG tube insertion [14], avoidance of mask ventilation, administration of nitrous oxide for maintaining spontaneous ventilation after induction of anesthesia [15], and one lung ventilation [10,12].

Awake intubation and maintaining spontaneous ventilation after induction are very dangerous in patients with traumatic brain injuries, penetrating eye traumas, and neck traumas; these patients benefit from smooth induction. Among blunt trauma patients, 32% have associated head injury [1]. Traumatic head injury is also the leading cause of death (25%) in patients with diaphragmatic hernia. The role of increased visceral volume in the thoracic cavity is prominent in the pathophysiology of the disease [15], and the stomach is the most common herniated viscera; therefore, decompression of the stomach and avoiding bag mask ventilation (rapid sequence induction with succinylcholine) are crucial [1,10]. In contrast, avoiding succinylcholine administration in patients with high intracranial pressure [16], open globe [17], and penetrating neck wounds is not strictly necessary. If there are no other major hemodynamic compromises like hemorrhage or heart contusion, it would be better to use high doses of hypnotic drugs.

Lobb and Butlin reported a diaphragmatic hernia patient with basal skull fracture who was intubated in an awake state and then spontaneously ventilated with a mixture of nitrous oxide [2]. Basal skull fracture seemed not to be a primary concern for the authors; their greater priority was hemodynamic stability. Loehning et al. found a chance for viscera to be pushed into the thoracic cavity by the contralateral diaphragm via relaxed dilated hole of the perforated diaphragm during mechanical ventilation [14]. Spontaneous ventilation always carries the risk of cough. Some authors believe that positive pressure ventilation can prevent viscera from entering the thoracic cavity. This is supported by some clinical observations, i.e., the less suggestive or diagnostic chest x-rays (CXRs) of diaphragmatic hernia in intubated patients [18] or progressive herniation of abdominal viscera after termination of ventilatory support [19]. Some authors [6] have considered a prophylactic effect for higher intrathoracic pressure with diaphragmatic hernia. The role of increased visceral volume in the pathophysiology of the disease [15], and the stomach is the most common herniated viscera; therefore, decompression of the stomach and avoiding bag mask ventilation (rapid sequence induction with succinylcholine) are crucial [1,10]. In contrast, avoiding succinylcholine administration in patients with high intracranial pressure [16], open globe [17], and penetrating neck wounds is not strictly necessary. If there are no other major hemodynamic compromises like hemorrhage or heart contusion, it would be better to use high doses of hypnotic drugs.

Figure 3. Postoperative chest X-ray (day 2).
viscerothorax or gastrothorax and enlarging the diaphragmatic hole [13]. The case reported by Yoshidome et al., in which the patient’s lung was herniated into the abdominal cavity, could also indicate that the pressure gradient could prevent abdominal viscera from entering the thoracic cavity during mechanical ventilation [19]. Diaphragmatic rupture was diagnosed in a great percentage of cases intraoperatively (26–63%) [1,2,4].

Usual intubation and ventilation methods for trauma patients are probably applied in these undiagnosed cases, and if positive pressure ventilation was so dangerous, a higher number of circulatory collapses would be reported in literature. These observations may lead us to conclude that there is no need to insist on spontaneous ventilation in patient with diaphragmatic hernias during induction of anesthesia.

Williams et al. [10] recommended double lumen tubes and one lung ventilation for patients with diaphragmatic hernia. Double lumen tube insertion is time consuming and is associated with a higher failure rate [13,21]. They did not succeed in inserting such a tube and changed it to an endobronchial single lumen tube, which kept the other bronchus unprotected during the whole length of surgery. Air trapping, pneumothorax, ventilation failure caused by tube misplacement, and airway trauma are other disadvantages of double lumen tubes.

**Conclusions**

Recommended anesthetic management for patients with traumatic diaphragmatic hernia is not applicable when there is associated brain trauma, open globe, or penetrating neck trauma. Complete decompression of the upper gastrointestinal (GI) tract by NG tube and complete readiness of the surgical team before induction of anesthesia are necessary. Invasive monitoring of arterial line and central venous pressures before induction in a calm and cooperative patient is helpful. For this group of patients, we recommend rapid sequence induction with the use of succinylcholine, complete dose of induction agents (if there are no hemodynamic compromises), and avoidance of mask ventilation before intubation. A single lumen tracheal tube expedites the process, and immediate non-depolarizing muscle relaxant administration prevents patient strain after induction of anesthesia. Mechanical ventilation with low tidal volume and rapid start of the surgical procedure may prevent the negative effects of involved pathophysiologies. We do not recommend awake intubation and spontaneous ventilation in these patients. Nitrous oxide can also be detrimental, and the efficacy of the double lumen tube is undetermined. Given that brain injury is the leading cause of mortality and some cases of traumatic brain injuries are still undiagnosed at the time of emergency surgery, our recommendations could be applicable for the majority of traumatic diaphragmatic hernias.

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