neuromuscular transmission. Other conditions may exist where evoked twitch tension and EMG yield contradictory results.

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64:281–283, 1986

Esophageal Perforation Associated with Endotracheal Intubation

KENT G. JOHNSON, M.D.,* DAVID D. HOOD, M.D.*

Esophageal perforation, associated with tracheal intubation, is more likely to occur when emergency intubations are performed by inexperienced individuals.[1] However, esophageal injury has occurred in more controlled settings. Subcutaneous emphysema, pneumothorax, cyanosis, throat pain, fever, mediastinitis, empyema, pericarditis, and death can occur as untoward sequelae.[1,2] Prompt diagnosis and surgical treatment reliably prevent the delayed complications.[1]

We describe a case of esophageal perforation during induction of anesthesia and tracheal intubation which resulted in an immediate, life-threatening airway emergency, subcutaneous emphysema, and bilateral pneumothoraces. Immediate diagnosis and surgical treatment facilitated a successful outcome.

Report of a Case

A 62-year-old woman was admitted for treatment of a small bowel obstruction. Her past medical history revealed no other significant abnormalities. She refused surgical intervention initially and received nasogastric suction. The surgical team instituted parenteral nutrition through a left subclavian catheter on the 7th hospital day.

The patient consented subsequently to abdominal exploration 10 days following admission. Before surgery, she complained of a severe sore throat and left ear pain. Left otitis media was diagnosed. Hematocrit and serum electrolyte values, as well as chest radiograph and ECG were within normal limits.

Prior to induction of anesthesia, we removed the nasogastric tube while applying suction. The patient received d-tubocurarine, 3 mg iv, and breathed oxygen for 5 min. Anesthesia was induced using thiopental, 4 mg/kg iv, and succinylcholine 1.5 mg/kg iv. Cricoid pressure was continuously applied. A laryngoscopy was performed with difficulty; however, an endotracheal tube was inserted eventually with the help of a malleable copper stylet. The tip of the stylet did not protrude beyond the tip of the endotracheal tube. After inflating the cuff, an esophageal intubation was recognized immediately during attempted positive pressure ventilation. The endotracheal tube was removed. Cyanosis developed and an attempt at ventilation *via* a mask resulted in extensive facial and cervical subcutaneous emphysema. A second laryngoscopy revealed blood in the pharynx and pharyngeal edema. Prompt endotracheal intubation and ventilation relieved the cyanosis. Anesthesia was maintained with enflurane, morphine, and oxygen. Pancuronium provided skeletal muscle relaxation.

Auscultation of the chest revealed equal but somewhat distant breath sounds bilaterally with some scattered inspiratory rales. Subcutaneous emphysema made interpretation of chest percussion difficult. An intraoperative A-P chest radiograph revealed extensive subcutaneous emphysema, which obscured the lung fields. Mediastinal emphysema was evident. A radial arterial catheter was inserted for monitoring.

Esophagoscopy using a flexible fiberoptic endoscope demonstrated a 1.5-cm, posterior, longitudinal, perforation of the esophagus 16 cm from the teeth. This was irrigated and drained externally through a neck incision. Subsequently, an abdominal exploration and lysis of adhesions were performed without incident. Ventilation was controlled throughout the procedure. The initial arterial blood gas values were: PA_{O_2} 183 mmHg, PA_{CO_2} 53 mmHg, and pH_a 7.33 with an FI_{O_2} of 1.0. Addition of a 10-cm H_2O peep valve and increased minute ventilation

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* Staff Anesthesiologist.

Received from Wilford Hall U. S. Air Force Hospital in San Antonio, Texas. Accepted for publication August 15, 1985.

Address reprint requests to Dr. D. Hood: Wilford Hall Medical Center/SGHSA, Lackland AFB, San Antonio, Texas 78236.

Key words: Intubation, endotracheal: Complications. Complications: Pneumothorax.

resulted in a $P_{A_{O_2}}$ of 362 mmHg, $P_{A_{CO_2}}$ of 40 mmHg, and a pH_a of 7.34. Peak airway pressure was 35 cm H_2O immediately following tracheal intubation, and did not change throughout the operation.

After surgery, ventilation was controlled. A repeat chest radiograph showed resolution of most of the subcutaneous emphysema, but also revealed bilateral 30% pneumothoraces. No mediastinal shift, hypotension, or jugular venous distention resulted. Bilateral chest tube thoracostomies were performed. Controlled ventilation was discontinued on the first postoperative day. The chest tubes were removed on the 7th postoperative day.

Insertion of methylene blue through a nasopharyngeal tube on the 8th postoperative day demonstrated no evidence of an esophageal leak. The patient was discharged on the 10th postoperative day.

Discussion

Boerhaave was credited with the first description of esophageal rupture in 1724.[3] Perforation of the cervical esophagus occurs most often where the esophagus is narrowest, at the level of the cricopharyngeus muscle. When the neck is extended during laryngoscopy, the posterior esophageal wall is compressed by the body of the 6th or 7th cervical vertebra.[3] Full-thickness esophageal perforations can occur from foreign bodies that cause pressure necrosis.[4]

Several factors may have contributed to esophageal perforation in this patient. The presence of a nasogastric tube for 10 days may have weakened or perforated the esophagus. Edema of the glottic opening and pharynx caused by the nasogastric tube contributed to the difficulty encountered by the inexperienced laryngoscopist who inserted the entire length of the Miller 3® blade into the pharynx. We feel that perforation of the weakened esophagus occurred by pinching it between the 6th cervical vertebra and the Miller 3® blade. The stylet or endotracheal tube also could have caused or exacerbated the esophageal injury, which was not apparent until positive-pressure ventilation was begun *via* a face mask.

Between the esophagus and the vertebrae, a fascial plane extends from the base of the skull to the diaphragm.[2] Spread of pharyngeal material in this hypovascular space accounts for much of the late morbidity and mortality associated with cervical esophageal perforations, including mediastinitis, empyema, and pericarditis.[5]

In this patient, positive-pressure ventilation using a mask in the presence of the esophageal perforation caused rapid spread of oxygen between fascial planes to the mediastinum and subcutaneous tissues. From the mediastinum, rupture into the pleural space is the most likely mechanism for the development of the pneumothoraces.[6] Following successful tracheal intubation, the endotracheal tube bypassed the esophageal tear. Thus, positive-pressure was no longer applied to the mediastinum and tension pneumothoraces, which otherwise might have developed, did not occur.

Definitive diagnosis of an esophageal tear is made either by endoscopy or by radiographic techniques using water soluble contrast media.[7] Radiographic techniques have demonstrated hypopharyngeal tears when endoscopy failed to do so.[8]

Esophageal perforation should be suspected in a patient with cervical subcutaneous emphysema, fever, dysphagia, and a history of a difficult intubation. Adequate treatment requires prompt surgical intervention. Delay in diagnosis results in a high mortality rate from mediastinitis and related complications.

In the largest series, Dubost reported an 85% mortality (6 out of 7) in patients when the diagnosis was delayed for 1 to 14 days, while all patients (5 out of 5) treated with surgical drainage within 12 hours of injury made a prompt and complete recovery.[1] In 38 of the 61 reported cases for which adequate information is available, a 50% mortality occurs if surgical intervention is delayed for more than 12 hours; overall mortality is 38%. Even among survivors, morbidity is substantial when the diagnosis is delayed.[1] Mediastinitis nearly always develops, necessitating prolonged antibiotic therapy and surgical drainage. Respiratory failure requiring extended ventilatory support is a common sequela to mediastinal sepsis. Only seven of the 38 described cases were surgically treated within 12 hours of injury. This group experienced minor morbidity and no mortality.

Patients who develop subcutaneous emphysema or cyanosis immediately following esophageal perforation may suffer other complications. Massive subcutaneous emphysema complicated the evaluation of the relatively high peak inspiratory airway pressure (35 cm H_2O) and widened alveolar to arterial oxygen gradient intraoperatively. The auscultatory findings were confusing in the presence of the subcutaneous emphysema. Subcutaneous emphysema obscured the intraoperative radiographic appearance of pneumothoraces. However, they were apparent postoperatively after partial resolution of the subcutaneous emphysema.

Following successful tracheal intubation, we could ventilate and oxygenate this patient adequately. The widened alveolar to arterial oxygen gradient and relatively high peak airway pressures demonstrated the presence of pulmonary pathology. These abnormalities did not progress during the evaluation and surgical treatment of the esophageal tear. The operating surgeon felt the patient's condition was emergent and required relief of the small bowel obstruction. If aspiration was the problem, we had already demonstrated that we could maintain satisfactory ventilation and oxygenation. If the patient had pneumothoraces, we felt the risk of intraoperative progression was minimal because the endotracheal tube bypassed the esophageal injury and positive-pressure was no longer forcing air along fascial planes into the pleural space.

We did not employ nitrous oxide for two reasons: 1) this patient had a small bowel obstruction, and 2) the pos-

sible occurrence of pneumothorax in patients with esophageal perforation contraindicates its use.

In summary, we have presented a case of esophageal perforation associated with anesthetic induction and endotracheal intubation, delineated the associated complications, and outlined appropriate management.

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Anesthesiology
64:283–287, 1986

Autonomic Neuropathy in a Diabetic Patient with Renal Failure

LAWRENCE L. CICCARELLI, M.D.,* CHRISTOPHER M. FORD, M.D.,* KENTARO TSUEDA, M.D.†

Abnormalities of cardiovascular innervation in diabetes mellitus[1–4] and chronic renal failure[5] have been described. The prevalence of cardiac autonomic neuropathy in unselected diabetic patients is 20–40%, depending on the sensitivity of tests employed, indicating that the autonomic damage is more common than previously believed.[6,7] Once it develops, the prognosis is poor with the mortality rate approaching 56% over a 5-yr period.[3] A high proportion of these reported deaths were sudden and unexpected.[3,8,9] In diabetes mellitus, autonomic damage appears to occur at various sites in the reflex arc, but the precise location of damage remains controversial. In chronic renal failure, abnormalities are consistently located in the baroreceptor area.

This report concerns three episodes of rapidly progressing bradycardia and hypotension that occurred in a diabetic patient who had renal failure. These episodes occurred suddenly and unexpectedly, and were unresponsive to iv atropine or usual doses of ephedrine, ultimately requiring epinephrine and/or external cardiac massage for resuscitation.

* Instructor.
† Professor.

Received from the Department of Anesthesiology, Humana Hospital-University, University of Louisville, Louisville, KY 40292. Accepted for publication September 19, 1985.

Address reprint requests to Dr. Kentaro Tseuda.

Key words: Metabolism: diabetes. Complications: autonomic neuropathy.

The cardiovascular reflexes of the patient were examined after discharge: beat-to-beat heart rate variation was minimal during both deep breathing and the Valsalva maneuver; the hand grip test was abnormal; and marked postural hypotension was present.

Report of a Case

A 33-year-old woman, 152 cm and 65 kg, was admitted for insertion of an Ash® catheter for peritoneal dialysis. She had become increasingly intolerant of hemodialysis for the past 3 weeks, with hypotension and severe nausea during the procedure. The patient was a known juvenile diabetic of approximately 20 yr duration. She had been receiving hemodialysis for the past 14 months for end-stage disease secondary to diabetes mellitus. In addition, she had a long-standing history of peripheral neuropathy, retinopathy, and peripheral vascular disease. More recently, the patient had developed various symptoms suggestive of autonomic nervous dysfunction (*i.e.*, frequent esophageal spasm for 2 years, dizziness on standing for 1 year, and intermittent diarrhea that developed without warning two to three times a week for 6 months). Approximately 2 weeks prior to her current admission, blood sugar decreased to 2.3 mM (43 mg%) and 1.2 mM (22 mg%) in two successive days without any subjective symptoms. The patient had not received general anesthetics in the past, and there was no family history of anesthetic problems. Current medications were NPH insulin, 26 units, and regular insulin, 8 units twice a day.

Pertinent findings in the physical examination included bilateral retinal scars due to laser surgery and advanced peripheral somatic neuropathy involving forearms, as well as lower thoracic and lumbosacral distribution. Pain sensation, tactile discrimination, vibratory sensation, and proprioception all were impaired. Deep tendon reflexes were weak at the knees and absent at the ankles. Arterial blood pressure was 130/80 mmHg and the heart rate was 98 beats/min. The hemoglobin was 7.7 g/dl and the serum potassium value was 5.1 mM on