

ANESTHETIC MANAGEMENT OF IATROGENIC ESOPHAGEAL PERFORATION

- Case Report and Literature Review -

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Abstract

The incidence of esophageal perforation may be increasing as endoscopic procedures are becoming a standard diagnostic technique. Other situations such as vomiting, infection, malignancies may also be associated with esophageal rupture. Presenting symptoms may be non specific but delay in accurate diagnosis carries a high morbidity and mortality. Anesthetic management is critical to survival. A case of esophageal rupture and its management is presented along with a literature review.

Key words: Esophageal perforation, complications, pneumothorax, respiratory failure, shock.

Case Report

A 69-year-old Saudi male presented to the Emergency Room complaining that a piece of meat had stuck in his throat following lunch a few hours previously. He had tried to induce vomiting to clear the obstruction to no effect. He complained of retrosternal chest pain, which he attributed to his retching efforts.

Examination revealed an elderly patient in no apparent distress, with stable vital signs except for tachycardia 135 beats per minute. On

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auscultation of the lungs, basal rhonchi were heard on the right side. Chest X-ray was normal. Flexible gastroesophagoduodenoscopy (GED) was performed under intravenous sedation by the gastroenterologist. The meat was located in the lower esophagus and was extracted after multiple attempts. Further examination of the upper gastrointestinal tract (GI) revealed grade 3 esophagitis with moniliasis and a large amount of fluid in the stomach consistent with gastroparesis.

Four hours later the patient was noted to be diaphoretic, tachypneic (40 breaths per minute), and tachycardic (147 beats per minutes). Pulse oximetry recorded 80%. The patient was breathing oxygen at high flow through a non-rebreathing mask. Reduced breath sounds were heard throughout the left side. Chest x-ray showed a left pneumothorax with left sided pleural collection, right mediastinal shift, and slight tracheal deviation. A left-sided chest tube was immediately inserted and air together with 300 cc of brown fluid and other gastric contents were released. Oxygen saturation increased to 100%. A possible iatrogenic esophageal rupture was suspected. A large bore cannula was placed in the left subclavian vein and fluid resuscitation started. Esophagogram with Gastrografin® swallow demonstrated perforation at the distal esophagus near the gastroesophageal junction with extravasation into the left pleural cavity. Infectious disease consult was obtained to consider the consequences of grade 3 esophagitis with moniliasis and esophageal tear. Mediastinitis and subsequent sepsis were considered. Antibiotic coverage included piperacillin and tazobactam 2.25 grams, metronidazole 500 milligrams, fluconazole 150 milligrams and vancomycin 1 gram. Emergency left thoracotomy and repair of the esophageal perforation were planned.

On preanesthetic assessment, he was noted to be obese (115 kg), with no chipped or loose teeth and a Mallampati classification of III. He was assessed as ASA IV-E secondary to long-standing hypertension, uncontrolled diabetes (blood sugar 221 mg/dl), esophageal perforation with pneumothorax and tracheal deviation, and esophageal moniliasis with mediastinitis.

After routine monitors were attached, an epidural catheter was

inserted in the sitting position for postoperative pain management. A thorough airway examination was performed. Despite Mallampati classification III, the patient had good mouth opening, (thyromental distance >4cm), compliant mandibular space and adequate neck mobility. Following appropriate oxygenation, rapid sequence induction included etomidate and suxamethonium. Endotracheal intubation was performed with a 41 French double lumen tube (DLT). Correct position was confirmed by the differential inflation/deflation/auscultation method. Fiberoptic confirmation, although perhaps not a “standard of care”, is strongly recommended with DLTs to ensure correct placement. An arterial cannula was inserted for continuous arterial blood pressure monitoring and arterial blood gas sampling. Central venous pressure was transduced. Hourly blood glucose monitoring was performed. Intravenous insulin was administered to maintain the blood glucose level below 130mg%. Anesthesia was maintained using oxygen in isoflurane with intermittent doses of fentanyl for analgesia and rocuronium for muscle relaxation. Intermittent doses of 0.25% bupivacaine were used via the epidural catheter to supplement the general anesthesia.

Surgery, which consisted of left thoracotomy with repair of esophageal perforation, feeding jejunostomy, decompression gastrostomy, and replacement of chest tube, was performed uneventfully. Thereafter the patient was placed supine and blood gas analysis showed a large A-ADO₂ difference (PaO₂ 104 mmHg on FiO₂ of 1). Postoperatively, the decision was made to continue endotracheal intubation and ventilation. The double lumen tube was changed to a single lumen tube and the patient transferred to the intensive care unit for further care.

The postoperative course was turbulent due to the development of respiratory failure and the need for inotropic support. He made a gradual recovery and was discharged home on postoperative day 27.

Discussion

The most common cause of esophageal perforation is iatrogenic¹. It can follow routine endoscopy with or without esophageal dilation,

mechanical trauma from insertion of endotracheal tubes, esophageal bougies, nasopharyngeal tubes, and after both regional and general anesthesia secondary to vomiting^{2,3}. Morbidity is high and mortality can reach 50%, especially if the diagnosis is delayed⁴.

Few articles discuss the anesthetic management of esophageal perforation⁵. In this case report we consider the etiology, pathophysiology, clinical presentation, and anesthetic management of this condition with alternative methods of airway management.

Iatrogenic esophageal perforations have become more common due to the rapid increase and development of upper gastrointestinal tract endoscopies and now account for 63.9% of perforations. A further 16.7% can be attributed to foreign bodies, 13.9% to external trauma, and 5.5% to spontaneous rupture (Boerhaave's Syndrome)⁶. The incidence of esophageal perforation from rigid esophagoscopy is 0.11% while that associated with fiberoptic examination ranges from 0.018% to 0.03%^{6,7}.

Therapeutic endoscopy, for example, for removal of a foreign body is associated with a much higher incidence of 1 to 10%^{8,9}. Our patient presented with a foreign body in his esophagus. He underwent difficult removal of the foreign body via fiberoptic esophagoscopy and esophageal perforation was diagnosed. The exact cause of the rupture is unknown and could be iatrogenic from the endoscopy or a spontaneous tear from retching and attempted vomiting after the food bolus was lodged in his throat. While the tear probably antedated the endoscopic examination, the procedure might have "reactivated" the lesion. The endoscopist should try to prevent retching by applying local anesthesia to the oropharynx and administering adequate sedation before starting the procedure. Support from an anesthesiologist is most helpful.

The diagnosis of esophageal perforation can be difficult because often classic symptoms (pain, fever, rhonchi) are not present and delay in presentation for medical care is common. Approximately one third of all cases are atypical. The presentation is frequently non-specific and emulates other disorders such as reflux esophagitis, peptic ulcer disease, myocardial infarction, gastritis, hiatal hernia, and esophageal varices¹⁰. Pressure-induced rupture accounts for 30-40% of all cases of perforation

and is the most sinister cause¹¹.

Esophageal rupture may occur in all age groups, from neonates to elderly individuals, but is most frequent in middle-aged males¹². The syndrome usually results from an increase in intraabdominal pressure, frequently associated with excessive consumption of food, followed by vomiting. The sudden rise in the intraluminal esophageal pressure tears the distal, left lateral esophageal wall 3 to 5 cm above the gastroesophageal junction. The tear usually involves the full thickness of the esophagus and communicates with the left pleural cavity in 80% of cases¹³. Prompt recognition of this potentially lethal condition is vital to ensure appropriate treatment. Mediastinitis, sepsis and shock frequently develop late in the course of the illness, which further confuses the diagnostic picture. A reported mortality estimate is approximately 35%, making it the most lethal perforation of the GI tract. Best outcomes are associated with early diagnosis and definitive surgical management within 12 hours of rupture. If intervention is delayed longer than 24 hours, the mortality rate (even with surgical intervention) exceeds 50% and is 90% after 48 hours¹⁴.

Iatrogenic esophageal perforation usually presents within 6 hours of instrumentation and is generally diagnosed earlier because of a high index of suspicion in patients undergoing esophageal instrumentation. Factors that increase the risk of iatrogenic esophageal injuries include foreign body removal, coexisting esophageal disease, i.e. malignancy, esophageal stenosis and the use of rigid rather than fiberoptic esophagoscopes⁶.

The clinical picture depends on the level of the perforation and the time interval from rupture to presentation. The mid esophagus lies next to the right pleura while the lower esophagus abuts the left pleura. Once a perforation occurs, saliva, retained gastric contents, bile and acid enter the mediastinum, resulting in mediastinitis, pneumomediastinum and pleural collections. Initially, the patient presents with pain at the site of perforation usually in the neck, chest, epigastrium, or upper abdomen¹. The first sign may be subcutaneous emphysema^{15,16}. Pneumomediastinum may cause a cracking sound upon chest auscultation, known as the Hamman crunch. Other early symptoms include dyspnea, fever, and

dysphagia. The Mackler triad defines the classic presentation. It consists of vomiting, lower thoracic pain, and subcutaneous emphysema. Tachycardia, diaphoresis, fever, and hypotension are common, particularly as the illness progresses. However, these findings are nonspecific and are due to sepsis from the mediastinitis and pleural collections. Gastric contents and fluids are drawn into the pleural space by the negative intrathoracic pressure and result in inflammation and fluid sequestration leading to hypovolemia, hypotension, tachycardia, and a systemic inflammatory response⁵. Due to the nonspecific nature of its presentation, esophageal perforation may be confused with myocardial infarction, peptic ulcer disease, acute pancreatitis, or pneumonia. In our patient, the clinical features were those of respiratory distress and tachycardia. Respiratory distress was probably secondary to the leak of gastric contents into the pleural space with accompanying inflammation and pleural effusion. His tachycardia was most likely due to early mediastinitis or developing shock.

Laboratory findings are often nonspecific. Patient may present with leukocytosis and a left shift (increased number of immature neutrophils (band forms) which suggests acute inflammation) As many as 50% of patients have a hematocrit value that approaches 50%, usually due to intravascular dehydration caused by fluid loss into pleural spaces and tissues. Serum albumin is normal but may be low, while the globulin fraction is normal or slightly elevated.

Posteroanterior and lateral upright chest radiographs are useful to identify any cervical or mediastinal emphysema, mediastinal widening, mediastinal air-fluid levels, pneumothorax, pneumopericardium, or pleural effusion¹. In the initial diagnosis, most patients reveal an abnormal finding after perforation, most commonly, a left unilateral effusion. However, the chest x-ray may be normal in 12 to 33% of patients^{17,18}. If the clinical picture continues to suggest an esophageal perforation, a chest CT and or esophagogram should be performed^{1,19}. The CT may show air in the soft tissues of the mediastinum and around the aorta, abscess formation, and pleural collections. An esophagogram helps confirm the diagnosis and typically shows extravasation of contrast

into the pleural cavity. The use of a water-soluble contrast such as Gastrografin® is advocated if esophagotracheal perforation into the lung is suspected. Esophagoscopy may be negative in up to 10% of patients¹⁷.

In patients that present with pleural effusions, chest tube thoracentesis should be performed if the effusions are significant. Large collections interfere with ventilation and contribute to the development of sepsis due to contamination of the pleural space, as the fluid is a good culture medium for polymicrobials. Undigested food particles and squamous cells from saliva may be identified¹⁷. The pH of the pleural fluid is usually less than 6, and the amylase content is elevated^{18,20}.

Endoscopy is not commonly used as a diagnostic aid. It carries the additional risks of increasing the size and extent of the original perforation and forcing air through the perforation into the mediastinum or pleural cavity. Endoscopy may be useful when a perforation is suspected but not proven, especially when trauma (e.g., ingested foreign body) is known or suspected to be present.

Management of esophageal perforation is either surgical or medical. Surgery has been the most common treatment, but in selected cases medical management may give better results^{21,22}. Selection criteria for medical treatment reported by Cameron et al.²² were disruption contained within the mediastinum, and minimal symptoms and signs of sepsis. A poor clinical state may also be a reason for medical treatment¹⁴. Medical management consists of the following: intravenous fluids, broad-spectrum antibiotics (for example imipenem/cilastatin (Primaxin®)), nasogastric suction, no oral intake, and adequate drainage with tube thoracostomy. Early use of nutritional supplementation is helpful.

Barrett described the first successful surgical repair of the esophagus in 1947²³. Surgical repair within 12 to 24 hours is associated with a mortality of 34% while a delay of 24 hours is associated with 64% mortality²⁴. Direct repair of the rupture and adequate drainage of the mediastinum and pleural cavity provide the best survival rates. Surgery in these patients usually consists of initial rapid resuscitation followed by primary repair of the esophageal tear with interrupted resorbable sutures and reinforcement by an intercostal flap. Thoracic esophageal perforation

repair can be reinforced by an autologous pleural flap or by pedicle muscle flaps from the intercostal muscles, chest wall musculature, diaphragm, or a mobilized pedicle of omentum^{25,26}. Reinforcement with vascularized tissue decreases fistula formation (13%) and mortality (6%), compared with unreinforced primary repair (39% fistula formation, 25% mortality)²⁶. Either a left or a right thoracotomy, depending on the site of the lesion, followed by pleural and mediastinal lavage with debridement of necrotic tissue, and insertion of pleural and mediastinal drains is usually performed. A mini laparotomy allows for placement of a decompressive gastrostomy and a feeding jejunostomy²⁷. In patients operated within 24 hours of rupture, primary closure and wide drainage of the mediastinum is effective and recommended as the gold standard treatment^{21,28}. Alternative surgical techniques include insertion of a metallic stent, esophagectomy, and controlled fistula with a T-tube, well-vascularized pedicle tissue flap using omentum, fundoplasty, and esophageal exclusion^{21,27,29}.

A Medline® search did not reveal any literature on specific anesthetic management of these patients. Anesthesia for the patient with esophageal rupture must consider the critical condition of these patients. Morbidity and mortality increase exponentially with delay. Rapid resuscitation with the use of large bore cannulae and intravenous warm fluids must be started prior to induction. Broad-spectrum antibiotics should be administered as soon as possible.

Induction should take into consideration the high risk for aspiration. Any increase in intra-abdominal pressure should be avoided to prevent further contamination through the esophageal rupture. Induction should be smooth. Coughing and straining should be avoided as they may increase the chances of further tearing in tissue that is already inflamed and friable. Insertion of a nasogastric tube is not recommended as any instrumentation can aggravate the injury to the esophagus, or pass through the hole. Rapid sequence induction is mandatory. Securing the airway during the awake state is a possible alternative but it should be attempted cautiously because retching, coughing or straining during the procedure may aggravate the esophageal tear.

Patients are often volume depleted, in the early stages of septicemic shock and may become hemodynamically unstable with any reduction in afterload. Etomidate and ketamine are suitable induction agents. Even so, hypotension may still occur and hydration prior to induction of anesthesia should always be performed. Intravenous inotropic agents should be readily available³⁰.

The choice of muscle relaxants for intubation is between suxamethonium, a depolarizing muscle relaxant, and a rapidly acting non-depolarizing agent. A theoretical disadvantage of suxamethonium is that it may cause an increase in intra-abdominal pressure due to fasciculations and increase the risk of reflux through the esophageal perforation. A defasciculating dose of a non-depolarizing drug may not reliably attenuate this rise in intra-abdominal pressure. Rocuronium provides excellent intubating conditions within 25-35 seconds and is a reasonable alternative. Onset of maximum single twitch depression after the administration of 3-4x ED₉₅ of rocuronium resembles the onset of action of suxamethonium, 1 mg/kg IV. However, if there is any doubt about the patient's airway, or if the airway has to be secured as fast as possible, suxamethonium is still preferred. Cricoid pressure (Sellick's maneuver) has been associated with esophageal rupture³¹, and its use during rapid sequence induction and intubation in patients with esophageal rupture is controversial²³. The risk of worsening the contamination through the esophageal perforation if reflux occurs should be weighed against the risk of contamination of the lungs in a patient who already has at least one lung damaged.

Most esophageal repairs involve a thoracotomy. Lung separation is usually required to facilitate surgical access and can be performed by the use of a DLT, or a single lumen tube with a bronchial blocker. The choice of endotracheal tube depends on the patient's airway anatomy and the anesthesiologist's preference. The choice is less important than the skill of the operator. Proper confirmation of placement should be performed using a fiberoptic bronchoscope³².

Monitoring should consist of standard American Society of Anesthesiologists monitors, cannulation of an artery, both for continuous

blood pressure monitoring and for frequent blood gas analysis, and insertion of a central venous catheter to aid assessment of fluid status and for infusion of inotropic agents. A urinary catheter is essential to monitor urinary output and a warming blanket, fluid warmer and a heat moisture exchange should be used to maintain body temperature. Special attention is required during retrosternal manipulation, because direct pressure on the heart may produce arrhythmias or hypotension. Major blood loss is also expected. Serial hemoglobin and hematocrit measurements allow assessment of blood loss.

Appropriate measures should be taken to correct the blood volume by infusion of colloids, crystalloids, and blood components. Various factors including prolonged surgery, lung congestion, large fluid shifts, hypothermia, a long surgical incision, and pain may lead to post-operative hypoventilation, hypoxia, and atelectasis. Elective post-operative mechanical ventilation is suggested.

Post operatively, these patients are usually monitored in the intensive care unit, because they are highly prone to cardiorespiratory complications. If there is any doubt about the adequacy of ventilation postoperatively, continued intubation and mechanical ventilation is necessary until status improves. Postoperative care consists of pain control with epidural local anesthetics and or epidural or intravenous narcotics, meticulous care with intravenous fluids, continuation of antibiotics, and on going drainage of the pleural and mediastinal spaces. Close attention to nutrition is also important, either parenterally or enterally through a jejunostomy tube.

Conclusion

We present a case of a patient with a ruptured esophagus. In the absence of clear guidelines and based on our experience in this patient, emphasis must be placed on early diagnosis. We suggest that anesthetic considerations should include avoidance of aspiration pneumonia and further aggravation of the esophageal tear, and early and continued resuscitation from a morbid inflammatory condition.

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