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Perioperative Airway Management and Septic Shock Resuscitation in Patients with Esophageal Rupture Complicated by Pyothorax Undergoing Thoracotomy: A Case Report

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Abstract

Introduction: Esophageal rupture with pyothorax and septic shock is a life-threatening emergency. Patients undergoing emergency thoracotomy face the dual challenges of airway management and hemodynamic support during one-lung ventilation (OLV).

Case Presentation: A 71-year-old man presented with esophageal rupture, right-sided pyothorax, and fistulas. Rapid sequence induction (RSI) was chosen to avoid positive-pressure ventilation. During OLV, hypoxemia was managed with positive end-expiratory pressure (PEEP) and intermittent two-lung ventilation. Hypotension required norepinephrine; however, intraoperative lactate levels remained within the normal range. The patient recovered without major complications.

Conclusion: Successful perioperative management hinges on RSI to avoid positive-pressure ventilation, a stepwise approach to OLV-induced hypoxemia, and early vasopressor support, with recognition that hypotension is primarily driven by septic shock. Normal lactate levels can serve as a reassuring marker of adequate resuscitation.

Keywords

Esophageal Rupture, Pyothorax, Septic Shock, One-Lung Ventilation, Rapid Sequence Induction, Norepinephrine, Airway Management

Abbreviations

OLV: One-Lung Ventilation; PEEP: Positive End-Expiratory Pressure; RSI: Rapid Sequence Induction

Introduction

Esophageal perforation is a rare but highly lethal condition. Esophageal rupture can be iatrogenic (e.g., following endoscopy or esophageal dilation) or non-iatrogenic, including spontaneous Boerhaave syndrome, tumors, and trauma, with an overall mortality rate exceeding 10% [1]. Patients typically present with progressive dyspnea and worsening infection. Emergency thoracotomy in patients with associated pleural or mediastinal infection is particularly

challenging.

Pulmonary infection and impaired respiratory function make intraoperative ventilation considerably more difficult, especially when one-lung ventilation (OLV) is required. In addition, these patients often have severe infection or septic shock, and prolonged preoperative fasting further increases the risk of circulatory failure and other vital organ dysfunction.

This case report describes the perioperative management of a patient undergoing emergency thoracotomy for esophageal rupture, with a focus on preoperative risk assessment and key management considerations.

Case Presentation

Patient Information:

A 71-year-old man (152 cm, 50 kg) presented to another hospital with chest pain and dyspnea after vomiting. He was diagnosed with esophageal rupture and received antibiotic therapy and chest tube drainage. Upon transfer to our hospital, computed tomography (CT) confirmed esophageal rupture, right-sided pyothorax, and right-sided pneumothorax. Emergency thoracotomy was scheduled. He had previously undergone bladder cancer surgery.

Admission Assessment:

The patient appeared acutely ill and was in a semi-recumbent position. Breath sounds were diminished bilaterally, with crackles, and dullness to percussion was present bilaterally.

Vital signs: HR 79 bpm, BP 138/86 mmHg, RR 20/min, SpO_2 92% on room air, temperature 36.5°C.

Airway assessment: In the semi-recumbent position, the chest tube was patent. Mouth opening was adequate, and airway evaluation revealed Mallampati class II with normal cervical spine mobility.

Ancillary Investigations:

Arterial blood gas (nasal cannula at 3 L/min): pH 7.569, PaO_2 66.3 mmHg, $PaCO_2$ 30.2 mmHg, lactate (Lac) 1.3 mmol/L, base excess (BE) 5.3 mmol/L, bicarbonate 28.8 mmol/L, potassium 2.8 mmol/L, calcium 0.89 mmol/L.

Laboratory and CT findings: Hb 110 g/L, PLT 357×10^9/L, WBC 5.09×10^9/L, PT 13.7 s, APTT 31.9 s, albumin 26.8 g/L, procalcitonin 0.079 ng/mL, interleukin-6 33.72 pg/mL, and C-reactive protein 28.4 mg/L. CT of the chest showed esophageal rupture with associated esophagopleural and esophagomediastinal fistulas, right-sided hydropneumothorax, and scattered bilateral pulmonary infiltrates (**Fig-1**).

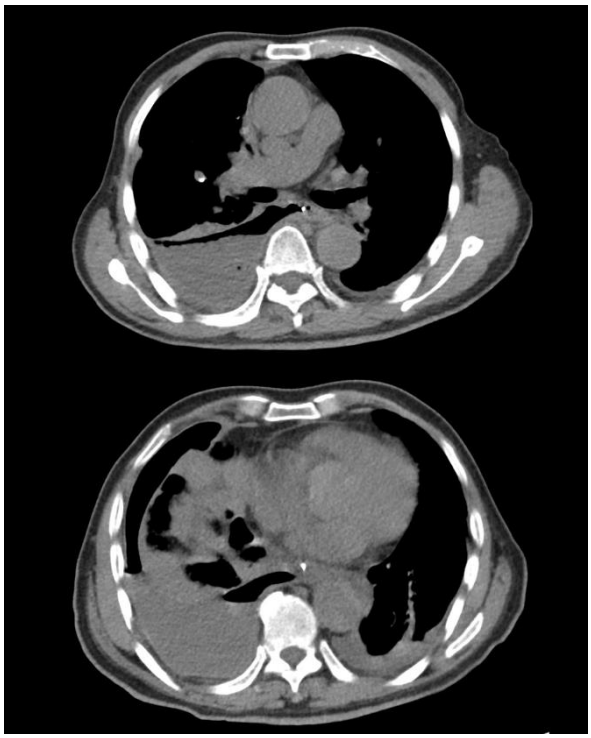

Fig-1:

Intraoperative Management:

We suctioned gastric fluid via an orogastric tube. Preoperative CT showed no gastric retention. We chose rapid sequence induction (RSI) primarily to avoid positive-pressure ventilation, which could force air through the fistulous tract into the chest and mediastinum. Awake intubation was not feasible because of severe pain.

Induction: After adequate preoxygenation, we administered propofol 90 mg and rocuronium 65 mg. A 35F left-sided double-lumen tube was inserted smoothly, and its position was confirmed. Immediately after intubation, we administered sufentanil 20 μg and methylprednisolone 40 mg. Anesthesia was maintained

with sevoflurane and remifentanil, targeting a BIS of 40-60. Radial artery and central venous catheters (CVCs) were placed.

One-lung ventilation (OLV) lasted approximately 65 minutes. One hundred percent oxygen was used throughout the procedure. Initial OLV settings were a tidal volume of 6 mL/kg, RR of 14 breaths/min, and an inspiratory-to-expiratory ratio of 1:2. During OLV, SpO_2 fluctuated between 89% and 93%. Arterial blood gas analysis showed that PaO_2 fell to a nadir of 63.6 mmHg from a baseline of 281.5 mmHg (while breathing 100% oxygen before incision), and $PaCO_2$ rose to a peak of 63.6 mmHg from 45.9 mmHg. To manage hypoxemia, we applied positive end-expiratory pressure (PEEP) of 6 cmH_2O to the dependent lung and intermittently resumed two-lung ventilation when SpO_2 dropped below 90%. After two-lung ventilation was resumed at the end of surgery, PaO_2 increased to 88.1 mmHg and $PaCO_2$ decreased to 51.9 mmHg.

Hypotension developed shortly after OLV began. Invasive blood pressure (IBP) monitoring was established. Norepinephrine was started at 0.03 μg/kg/min and titrated up to 0.2 μg/kg/min. While receiving norepinephrine, IBP ranged from 85 to 100 mmHg systolic and 40 to 60 mmHg diastolic. After OLV was discontinued, the patient still required norepinephrine at 0.1 μg/kg/min to maintain IBP.

Intraoperative arterial Lac remained normal throughout (1.3 → 1.2 → 1.4 mmol/L). Total fluid administered was 2000 mL (balanced crystalloids plus 20 g albumin), along with 2 units of packed red blood cells. Hemoglobin decreased from 115.7 g/L to 93.5 g/L. Hypokalemia and hypocalcemia were continuously monitored and corrected. Urine output during surgery was 900 mL. The operation lasted approximately 3 hours. About 400 mL of purulent fluid was drained from the pleural space, debris was removed from the mediastinum, and the esophageal rupture was repaired.

Postoperative Outcome:

The patient was transferred to the intensive care unit (ICU) after surgery and remained there for 10 days. He was extubated on postoperative day 2. Electrolyte disturbances were corrected gradually, and no major complications or anastomotic leakage occurred. He was discharged to a primary hospital for further rehabilitation on postoperative day 51.

Discussion

Core Challenges in This Case:

This patient with esophageal rupture, right-sided pyothorax, esophagopleural and esophagomediastinal fistulas, and septic shock undergoing emergency thoracotomy presented two major perioperative challenges: first, how to safely secure the airway and manage OLV; second, how to maintain hemodynamic stability and metabolic homeostasis in the setting of septic shock.

Perioperative Airway Management Strategies:

In our patient, preoperative CT showed no gastric retention. We chose RSI primarily to avoid positive-pressure ventilation rather than because of aspiration risk, since positive pressure could force air through the fistulous tract into the chest and mediastinum, worsening pneumomediastinum or causing tension pneumothorax [2].

OLV lasted about 65 minutes. On 100% oxygen, PaO_2 fell from 281.5 mmHg to a nadir of 63.6 mmHg, and SpO_2 fluctuated between 89% and 93%. This marked hypoxemia reflects two concurrent mechanisms: severe ventilation/perfusion mismatch induced by OLV itself, and ongoing lung injury from systemic inflammation [3]. Sepsis-related systemic inflammatory response syndrome (SIRS) can further aggravate oxygenation during OLV, as the lung is a primary target organ.

Our management included applying PEEP of 6 cmH_2O to the dependent lung and intermittently resuming two-lung ventilation when SpO_2 dropped below 90%. After two-lung ventilation was resumed, PaO_2 increased to 88.1 mmHg, confirming the effectiveness of this strategy [4]. This stepwise approach is consistent with the standard management of OLV-induced hypoxemia in thoracic surgery.

During OLV, the patient's $PaCO_2$ rose to a peak of 63.6 mmHg. This suggests either relative inadequacy of

minute ventilation or increased dead-space ventilation due to inflammation and pyothorax. After two-lung ventilation was resumed, $PaCO_2$ decreased to 51.9 mmHg, supporting this interpretation. Accordingly, end-tidal CO_2 and arterial blood gases should be closely monitored during OLV, with timely adjustments to ventilatory parameters.

Perioperative Circulatory and Metabolic Management in Septic Shock:

The patient exhibited clinical features of severe infection/sepsis preoperatively, as reflected by markedly elevated inflammatory markers (IL-6 33.72 pg/mL, CRP 28.4 mg/L). Hemodynamically, however, vital signs were stable on admission (HR 79 bpm, BP 138/86 mmHg). Hypotension requiring vasopressor support developed during the intraoperative period, shortly after the initiation of OLV. The underlying pathophysiology involves two interrelated mechanisms: (1) relative hypovolemia, resulting from prolonged fasting, fever-induced losses, increased vascular permeability, and hypoalbuminemia; and (2) vasoplegia driven by inflammatory mediators. The timing and strategy of vasopressor initiation, rather than just the choice of agent, are critical in septic shock resuscitation [5].

A clinically meaningful observation was that hypotension developed after the initiation of OLV, but the patient still required norepinephrine at 0.1 μg/kg/min to maintain IBP after OLV was discontinued. This temporal relationship suggests that increased intrathoracic pressure and reduced preload from OLV may have acutely exacerbated hypotension, but septic shock remained the underlying driver.

Despite intraoperative hypotension requiring norepinephrine (up to 0.2 μg/kg/min), arterial Lac remained normal throughout (1.3-1.4 mmol/L). In addition, urine output was adequate (900 mL). These reassuring findings indicate that tissue perfusion and oxygen delivery were effectively maintained through norepinephrine support and goal-directed fluid management.

Fluid resuscitation consisted of balanced crystalloids plus 20 g albumin, with a total fluid volume of 2000 mL, along with 2 units of packed red blood cells. Albumin was administered for preoperative hypoalbuminemia (26.8 g/L) to maintain colloid osmotic pressure. Hypokalemia and hypocalcemia are common in septic shock, and both were continuously monitored and corrected.

Broad-spectrum antibiotics were administered preoperatively. We chose methylprednisolone 40 mg. Although septic shock guidelines favor hydrocortisone for its combined glucocorticoid and mineralocorticoid activity, our use of exogenous corticosteroids still reflects clinical practice aimed at circulatory support in severe stress states.

Monitoring and Limitations:

Advanced hemodynamic monitoring, such as Pulse Index Continuous Cardiac Output (PiCCO), was not available in this emergency setting; it might offer more precise resuscitation guidance in more critically ill patients. Central venous pressure (CVP) was also not measured. This is a single retrospective case report, and long-term functional outcomes were not followed up.

Conclusion

This case highlights the dual perioperative challenges of airway management and hemodynamic support in patients with esophageal rupture, pyothorax, fistulas, and septic shock undergoing emergency thoracotomy. A successful outcome relies on recognizing that hypotension may be exacerbated by OLV but is primarily driven by septic shock, and that normal intraoperative lactate levels can serve as a reassuring marker of adequate resuscitation.

Conflict of Interest

The author has read and approved the final version of the manuscript, and the author declares no conflicts of interest.

References

[1] Sdralis EIK, Petousis S, Rashid F, Lorenzi B, Charalabopoulos A. Epidemiology, diagnosis, and management of esophageal perforations: systematic review. Dis Esophagus. 2017 Aug 1;30(8):1-6. [**PMID**: 28575240]

[2] Oh MK, Jeon WJ, Cho SY, Kwon YD, Kim KH.

Development of bilateral tension pneumothorax under anesthesia in a Boerhaave's syndrome patient: a case report. Korean J Anesthesiol. 2016 Apr;69(2):175-80. [**PMID**: 27066209]

[3] Merchant N, Liu C. Thoracic empyema: aetiology, diagnosis, treatment, and prevention. Curr Opin Pulm Med. 2024 May 1;30(3):204-209. [**PMID**: 38323933]

[4] Zajac JC, Arrigo RT, McCarthy DP. Intraoperative Ventilation and Anesthesia Issues During Thoracic Surgery. Thorac Surg Clin. 2025 Aug;35(3):365-75. [**PMID**: 40619184]

[5] Wieruszewski PM, Jabaley CS. It's Time to Consider How We Should Use Vasopressors, Rather Than Just Which We Should Use. Crit Care Explor. 2025 Sep 9;7(9):e1317. [**PMID**: 40924922]

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