# Critical care management of iatrogenic gastric perforation following chest tube insertion in traumatic diaphragmatic rupture: A case report

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**Abstract**

**Iatrogenic gastric perforation after chest tube implantation is rare but devastating. This can cause widespread thoracoabdominal contamination, fulminant sepsis, and acute respiratory distress syndrome, requiring immediate surgery and intensive care. For suspected tension pneumothorax, a 44-year-old man with traumatic thoracoabdominal injuries underwent urgent chest tube installation. Through an undiscovered traumatic diaphragmatic rupture, the surgery caused gastric perforation. In trauma, diaphragmatic injuries must be detected early, and anesthesiologists and intensivists must manage complex postoperative consequences using multidisciplinary, evidence-based critical care.**

**Keywords**: Diaphragmatic rupture, iatrogenic gastric perforation, ventilator-associated pneumonia, critical care multiple traumas.

## Introduction

Iatrogenic gastric perforation, a rare but life-threatening surgical emergency, involves inadvertent full-thickness gastric wall damage during medical procedures. It occurs most often after endoscopic interventions (0.03%–0.5%) but also during nasogastric tube insertion and chest tube thoracostomy. Visceral injury during chest tube insertion is estimated at 1%–2%, but an undiagnosed diaphragmatic defect can cause intrathoracic contamination, chemical pleuritis, and fulminant sepsis. (1,2)

Traumatic diaphragmatic rupture (TDR) is rare but catastrophic after high-energy blunt or penetrating thoracoabdominal trauma, with an incidence ranging from 0.8%–1.6% in blunt trauma and up to 15% in penetrating injuries. (3) The main clinical risk is abdominal viscera herniation into the thoracic cavity, which may lead to rapid cardiac compromise.

When chest tube placement is performed for suspected tension pneumothorax in this setting, inadvertent perforation of the herniated gastrointestinal tract creates a complex scenario of dual-cavity contamination, severe sepsis, and multi-organ failure requiring prompt surgical repair and multidisciplinary critical care.

## Case presentation

After a high-energy traumatic fall, a regional hospital referred a 44-year-old man (60 kg, 168 cm). He arrived at the Emergency Department of Dr. Soetomo General Hospital with acute respiratory distress. A patent airway, protected cervical spine, and 40 breaths/min respiratory rate, rightward tracheal deviation, and absent breath sounds over a hyperres-

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onant left hemithorax were found during primary survey. The referring hospital's chest X-ray confirmed left-sided tension pneumothorax. Later imaging followed needle thoracocentesis stabilization. Chest X-ray (**Figure 1**) confirmed a large left-sided pneumothorax with fractures of the 9th–11th left posterior ribs. No evidence of subcutaneous emphysema or pulmonary contusion was noted. Abdominal focused assessment with sonography in trauma (FAST) scan was negative for intra-peritoneal fluid in the Morrison's pouch, splenorenal space, or pelvic cul-de-sac.

The left 5th intercostal space was drained with a 24 Fr chest tube. Following air release, 550 ml of blackish serous fluid was unexpectedly discharged, necessitating rapid reassessment. Repeat chest radiography revealed that the chest tube tip was improperly positioned at the 12th thoracic vertebra (**Figure 2**). Radiographs and unusual tube output showed severe diaphragmatic rupture with intestine herniation and iatrogenic gastric perforation. The patient had emergency surgery immediately.

**Surgical exploration and findings**

Emergency surgery revealed a 7 cm left diaphragm rupture and stomach herniation into the thorax. The chest tube caused a grade II American Association for the Surgery of Trauma (AAST) gastric body perforation of 1 cm. Approximately 500 ml of gastric content was present in the peritoneal cavity. Exploratory laparotomy included primary gastric repair, diaphragmatic reconstruction, and thoracic and abdominal lavage.

Intraoperative thoracotomy revealed significant gastric contamination of the pleural space. A 1 cm laceration on the left lung's inferior lobe was repaired. A new 28 Fr chest tube was inserted.

**Intensive Care Unit (ICU) course and deterioration**

After surgery, the patient was mechanically ventilated in the ICU. On day 2, he was hemodynamically stable, with blood pressure (BP) 110/78 mmHg and heart rate (HR) 76 bpm, and was extubated to a simple mask. Hypoalbuminemia (albumin 2.68 g/dl) and anemia (Hb 7.9 g/dl) indicated hemorrhagic loss and systemic inflammation. His fluid balance was +1519 ml/24h. Intravenous (IV) crystalloid, parenteral nutrition (Intralipid® 20%), albumin 20% infusions, packed red cell transfusion, broad-spectrum antibiotics (ceftriaxone 1g q12h, metronidazole 500 mg q8h), epidural analgesic (Naropin 0.2%), and IV tramadol were the initial ICU treatments.

Reintubation was needed on day 4 due to fever and respiratory distress. The patient had mild acute respiratory distress syndrome (ARDS), with a temperature of 38.2 °C, a heart rate of 115 bpm, an arterial oxygen partial pressure (PaO2) of 68 mmHg, and a ratio of arterial oxygen partial pressure (PaO2) over the fraction of inspired oxygen (FiO2) (P/F ratio) of 136. Mechanical ventilation was initiated in pressure-synchronized intermittent mandatory ventilation (PSIMV) mode (peak pressure = 25 cmH2O), suggesting low lung compliance. A left-sided fluidopneumothorax and fresh left-sided pulmonary infiltrates on chest radiography (**Figure 3**) were suggestive of ventilator-associated pneumonia.

On day 7, severe sepsis was verified with leukocytosis (19,000/µl) and elevated procalcitonin (6.80 ng/ml). Fluid overload and septic shock were indicated by increasing pulmonary inflammation and an enlarged heart profile on chest X-ray (**Figure 4**).

Endotracheal tube (ETT) aspirates showed Acinetobacter spp. (resistant to ceftriaxone, susceptible to meropenem) and Candida tropicalis (fluconazole-sensitive). Then, empirical antibiotics were changed to IV meropenem. The ventilator strategy now prioritizes lung protection. The goal was to maintain oxygenation and reduce ventilator-induced lung damage. On day 7, the P/F ratio reached 200, and the fever subsided. On day 9, leukocyte count peaked at 25,710/µl, whereas procalcitonin decreased to 4.08 ng/ml, but anemia and hypoalbuminemia persisted.

**Recovery and weaning**

On day 10, with a SpO2 of 98%, the patient was successfully extubated to a nasal cannula. By day 11, vital signs were steady, afebrile, and fully alert. Resolution of pulmonary infiltrates and reduction in pleural effusion were two signs of radiologic improvement. The fluid balance became negative (-340 ml/24 hours), indicating resolution of sepsis. A clear indication of a successful course of treatment was the clearance of the lung areas (**Figure 5**). By day 14, the patient had fully recovered clinically and was released from the ICU.

**Discussion**

TDR is a rare but severe polytrauma injury that occurs in 0.8% to 1.6% of blunt thoracoabdominal trauma and more often in penetrating injuries. (1,2) Diagnostic ambiguity and high morbidity impact the condition. Beyond mechanical diaphragm disruption, abdominal viscera herniation into the thoracic cavity can cause cardiac compromise, bowel strangulation, and multi-organ failure. Sepsis and respiratory failure with late diagnosis cause 17%–21% deaths. (3)

TDH commonly resembles hemopneumothorax or pulmonary contusion. Radiographic signs such as a raised hemidiaphragm, mediastinal shift, and intrathoracic gas are commonly missed. (3) Tracheal deviation and unilateral hyperresonance indicated tension pneumothorax; thus, an emergent chest tube was placed, which caused gastric perforation due to diaphragm gastric herniation. (3)

Advanced imaging, such as computerized tomography (CT), may be impractical in unstable trauma patients. The Advanced Trauma Life Support (ATLS) guidelines suggest applying readily available diagnostics to identify suspected diaphragmatic injury after high-energy thoracoabdominal trauma. (5) The principal modality is chest radiography, which can show hemidiaphragm elevation, blurred borders, or intrathoracic air-fluid levels. Nasogastric tube coiling into the thoracic cavity is pathognomonic. FAST has low sensitivity, but competent operators can detect diaphragmatic discontinuity, particularly on the left side.

Early complications of chest tube implantation for suspected tension pneumothorax include gastric perforation. When undetected TDR allows gastric herniation into the thoracic cavity, this special condition is well characterized. (6) The herniated stomach is immediately injured by the thoracostomy tube. Occult TDR is dangerous, but emergent decompression saves lives in true tension pneumothorax. Içöz et al. suggested CT or targeted bedside imaging to check anatomy and evaluate diaphragmatic rupture in atypical presentations before invasive intervention. (7)

The patient had a "two-hit" paradigm with trauma followed by a fulminant septic cascade. Severe infection was verified by serial laboratory results, including leukocytosis (>25,000/μl) and peak procalcitonin (6.80 ng/ml). Acinetobacter spp. culture showed multidrug resistance, requiring a switch from empirical ceftriaxone to targeted meropenem.

Initial resuscitation stabilized hemodynamics but caused fluid overload, possibly causing pulmonary interstitial edema and cardiac strain. Reintubation and lung-protective ventilation were required to prevent moderate ARDS (P/F ratio 136) in accordance with ARDS.Net and international ventilator-induced lung injury prevention guidelines. The P/F ratio is crucial for managing FiO2 and PEEP levels. Reintubation with deep sedation optimized oxygen delivery by reducing metabolic demand and breathing work. This “lung rest” method allowed focused sepsis therapy and pulmonary recovery. (8) Extubation after five days of invasive ventilation was crucial to beating ventilator-associated pneumonia (VAP) and recovering respiratory function.

Early laparotomy was performed to control gastric perforation, correct a diaphragmatic defect, and perform thorough lavage to reduce contamination. Early culture and personalized treatment regimens, as guided by the Surviving Sepsis Campaign guidelines, guided antimicrobial therapy. (9) VAP prevention bundles limited pulmonary injury. A diagnostic bronchoscopy confirmed the presence of microorganisms, and therapeutic lavage was performed.

Nutritional optimization was crucial. Diaphragmatic instability precludes enteral feeding; therefore, the European Society for Clinical Nutrition and Metabolism recommends early parenteral nutrition (24–48 hours). The aim of 1.3 g/kg/day of protein and 25%–40% of non-protein calories from lipids was to minimize hyperglycemia and carbon dioxide production, with careful monitoring for refeeding syndrome. (10) Comprehensive pain management with thoracic epidural anesthetic and systemic medicines reduced opioid-induced respiratory depression. (3)

**Conclusion**

Despite iatrogenic gastric perforation and ventilator-associated pneumonia, this polytrauma patient was successfully managed in the critical care unit. Early identification of problems, surgical source management, and evidence-based intensive care led to a good recovery. Individualized ventilatory adjustments based on dynamic oxygenation indices (P/F ratio) and immediate pathogen-specific antibiotic therapy based on microbiologic data were crucial. Multidisciplinary collaboration, clinical vigilance, and modern critical care protocols are essential for managing trauma, iatrogenic injury, and nosocomial infection.

**Figure 1**. Chest X-ray on arrival

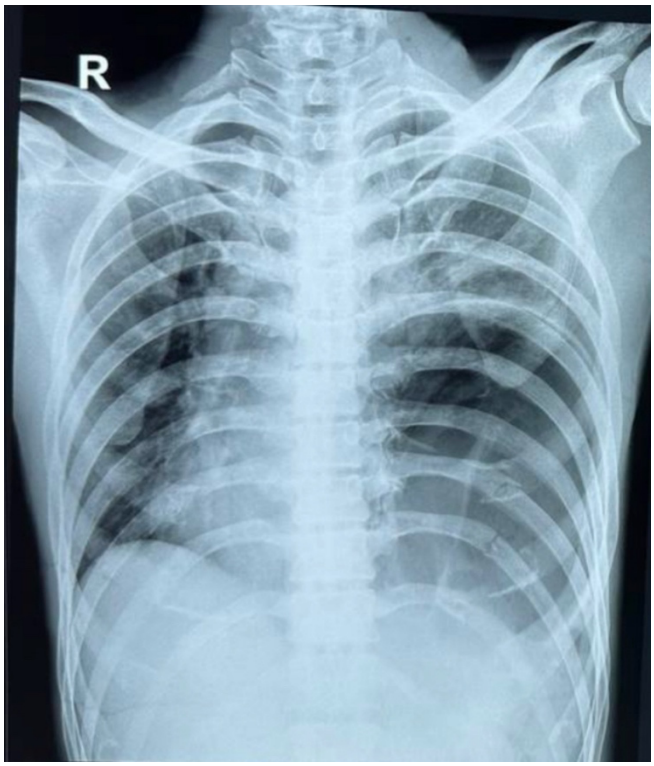



**Figure 2**. Post-initial chest tube placement

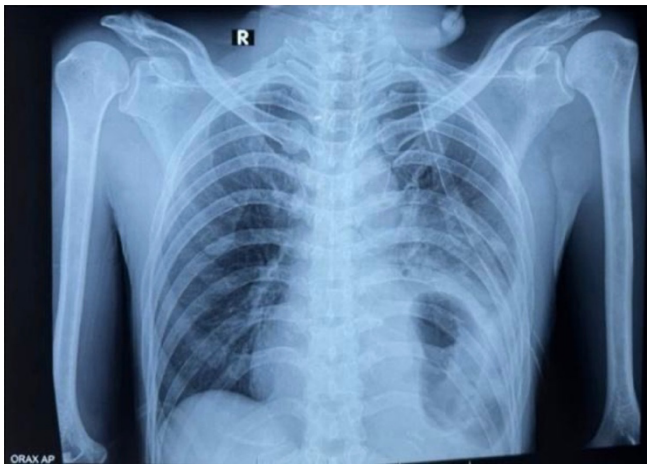


**Figure 3**. Day 4 chest X-ray

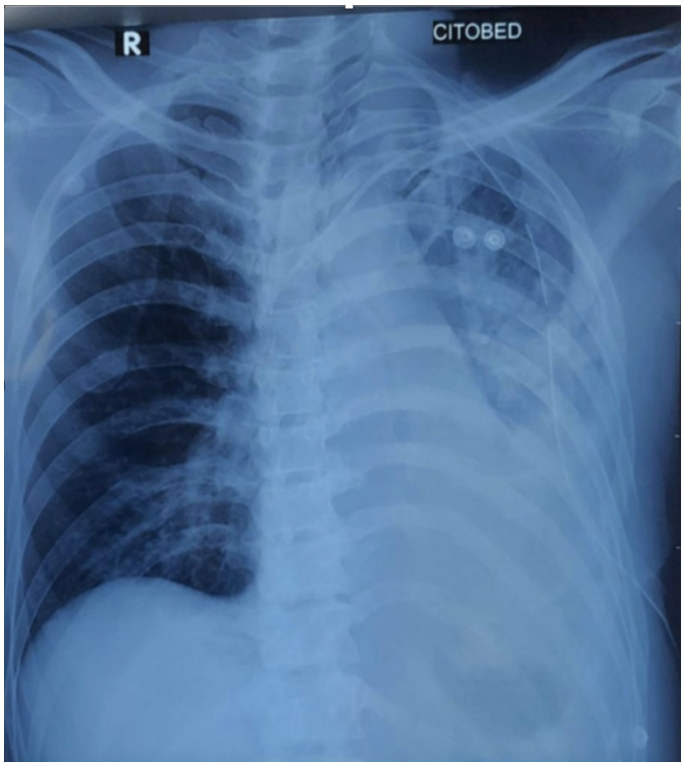



**Figure 4**. Pulmonary inflammation with fluid overload

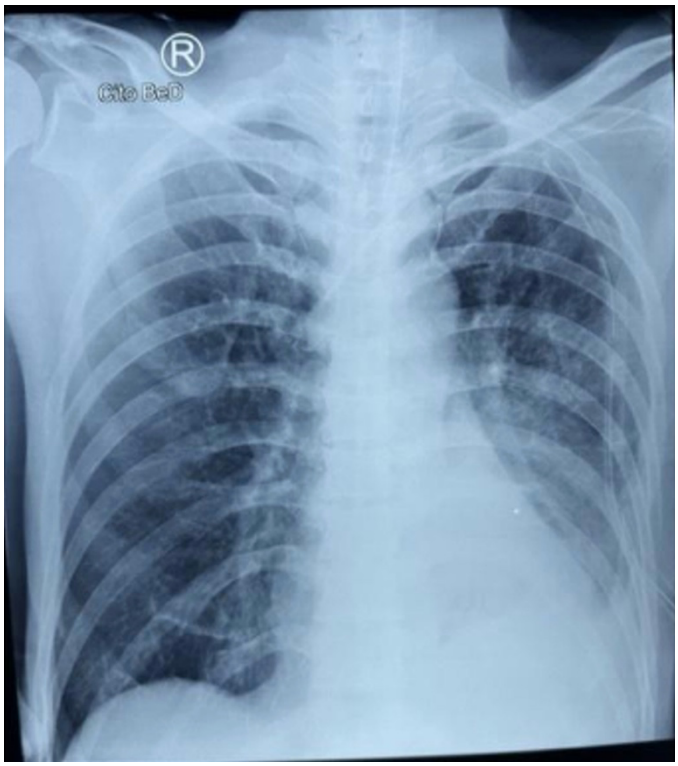
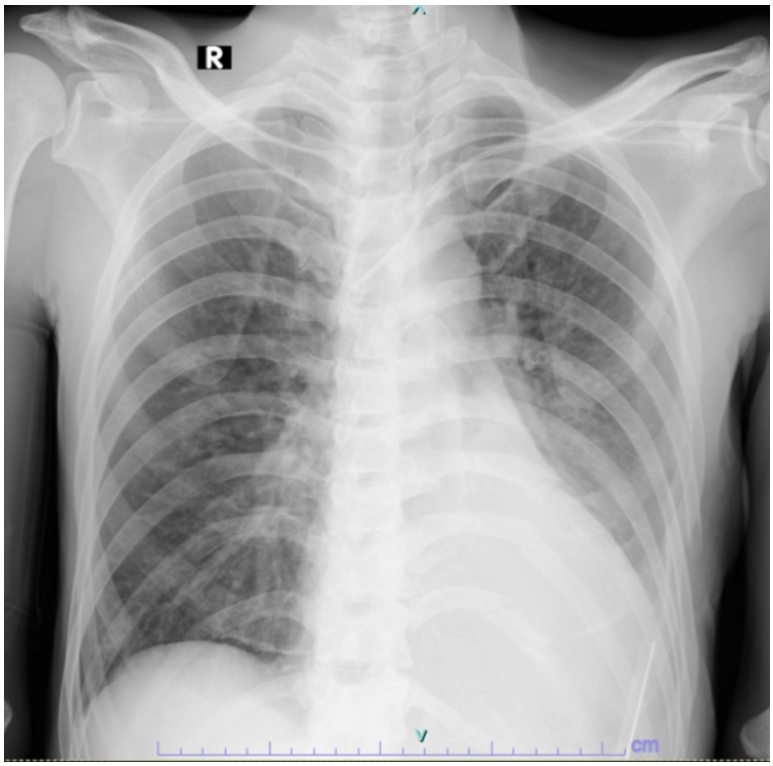


**Figure 5**. Improved lung fields and resolved pleural effusion after therapy



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