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Silent Impact: Acute Respiratory Distress Syndrome Induced Delayed Pulmonary Contusion Without External Trauma Signs

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ABSTRACT

Pulmonary contusion is the most common parenchymal injury following blunt thoracic trauma. It is defined as damage to the alveolar–capillary membrane, leading to alveolar hemorrhage and interstitial edema without macroscopic laceration of the lung tissue. Pulmonary contusion should be suspected in any patient with blunt chest trauma who develops unexplained hypoxemia or respiratory distress. We report a case of a patient who sustained high-energy blunt trauma without external bruising or radiologic evidence of pulmonary contusion, yet developed acute respiratory distress syndrome (ARDS) secondary to pulmonary contusion within 48 hours, despite an uncomplicated surgical course. This case highlights the importance of early recognition and close monitoring of ARDS secondary to pulmonary contusion to reduce mortality. Delayed onset pulmonary contusion often poses a diagnostic challenge, as initial imaging may appear normal while microvascular injury continues to evolve leads to eventual respiratory failure. Therefore, continuous clinical assessment and serial imaging are essential, particularly in patients with high-energy trauma, even in the absence of overt thoracic findings. Early diagnosis and timely intervention remain key determinants of patient outcomes.

KEYWORDS: Pulmonary Contusion, Acute Respiratory Distress Syndrome, Blunt Thoracic Trauma

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INTRODUCTION

Although the overall incidence of trauma-associated ARDS has declined over time, the mortality rate among trauma patients who develop ARDS has paradoxically increased. Hospital mortality among trauma patients with ARDS rose from 21% to 28%, yielding an overall mortality rate of 22%. Identified risk factors for the development of ARDS included the presence of direct thoracic trauma, advanced age, male sex, higher Injury Severity Scores (ISS), and lower Glasgow Coma Scale (GCS) scores [1]. Early identification of delayed pulmonary contusion is important because effective management may reduce the risk of progression to severe respiratory compromise or acute respiratory distress syndrome (ARDS) [2]. This case illustrates a young male polytrauma patient who developed delayed-onset pulmonary contusion leading to acute respiratory failure following an initially stable postoperative course. This case highlights the importance of vigilant respiratory monitoring and repeated imaging in high- energy trauma patients, as pulmonary contusion may not be immediately apparent on initial assessment but can progress rapidly, necessitating timely ventilatory support and critical care management.

Case Presentation

A 20-year-old male was brought to the Emergency Department of Dr. Soetomo General Hospital after sustaining high-energy blunt trauma from a head-on collision with a truck while riding a motorcycle. At the scene, the patient was conscious and groaning in pain, with open wounds noted on the frontal region and right thigh. Upon arrival at the emergency department, airway assessment revealed a patent airway with spontaneous breathing and no adventitious sounds. Vital signs showed blood pressure of 85/55 mmHg, heart rate of 134 beats per minute, capillary refill time greater than two seconds, and cold extremities, consistent with hypovolemic shock. Physical examination of the thoracoabdominal region revealed no external bruising, and chest auscultation showed no added sounds. Cardiac examination was unremarkable without gallop or murmur. Initial urine output through a Foley catheter was 30 mL, suggesting inadequate renal perfusion. Neurological evaluation revealed a Glasgow Coma Scale (GCS) of E4V5M6 with isocoric pupils measuring 3 mm bilaterally and positive light reflexes. An initial chest X-ray demonstrated no evidence of infiltration or pulmonary contusion.

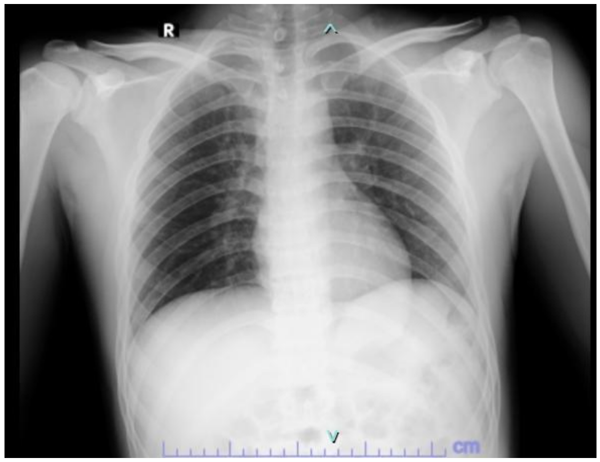


Figure 1: Chest X-ray admission

Resuscitation was initiated with Ringer's lactate 500 mL over one hour, followed by 500 mL of Gelofusine. After fluid administration, the patient's hemodynamics improved, with blood pressure increasing to 112/60 mmHg (MAP 77 mmHg), heart rate decreasing to 112 beats per minute, and capillary refill time improving to less than two seconds. The extremities became warm, oxygen saturation reached 99% on a simple mask at 6 L/min, and urine output increased to 100 mL/hour. The working diagnosis included unstable pelvic fracture, multiple fractures of the bilateral lower extremities, open wound of the frontal region, blunt abdominal trauma, and hypovolemic shock responsive to fluid resuscitation. The patient subsequently underwent emergency surgery consisting of wound debridement, pelvic external fixation, bilateral femoral external fixation, and right tibial external fixation. Intraoperative fluid input consisted of 1500 mL of crystalloids, 1000 mL of colloids, and 800 mL of packed red blood cells, with an estimated blood loss of 800 mL and urine output of 800 mL. The surgical procedure proceeded uneventfully.

Following the operation, the patient was successfully extubated with adequate spontaneous breathing, a respiratory rate of 20 breaths per minute, and oxygen saturation of 99% on a nasal cannula at 3 L/min. Hemodynamics were stable with blood pressure of 125/75 mmHg (MAP 92 mmHg) and heart rate of 86 beats per minute. The patient was conscious with GCS 4-5-6, mild pain (NRS 1–2), warm extremities, CRT less than two seconds, and urine output of 300 mL over three hours (approximately 1.6 mL/kg/hour). The abdomen was soft without tenderness or bleeding from the surgical site, and body temperature was 36.7°C.

On postoperative day one, the patient experienced sudden desaturation accompanied by increased work of breathing. Respiratory rate ranged between 24 and 28 breaths per minute, and oxygen saturation dropped to 80% despite oxygen supplementation via a simple mask at 6 L/min. Physical examination revealed decreased breath sounds on the left lung field with basal rhonchi. Hemodynamics remained stable with blood pressure of 107/59 mmHg (MAP 75 mmHg) and heart rate of 118 beats per minute. Chest X-ray evaluation showed left lung infiltrate. These findings raised the suspicion of left-sided pulmonary contusion causing acute respiratory failure. The patient was intubated and placed on mechanical ventilation to decrease the work of breathing and oxygen demand. Ventilator was set in pressure-controlled mode with inspiratory pressure of 12 cmH_2O, PEEP of 8 cmH_2O, FiO_2 of 60%, respiratory rate of 18 breaths per minute, and tidal volume of 468 mL. Sedation was maintained with RASS score -4. Urine output remained adequate at 400 mL over five hours (approximately 1.3 mL/kg/hour).

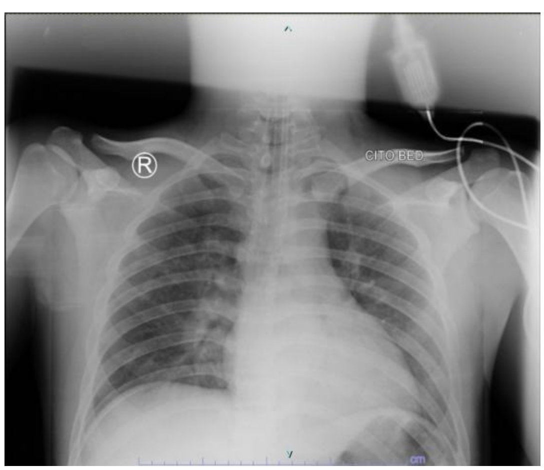


Figure 2: Chest X-ray D+1 Post Operation

Serial arterial blood gas (ABG) analyses and CXR showed progressive changes. Preoperatively, ABG demonstrated pH 7.34, pCO_2 37 mmHg, and pO_2 130 mmHg on nasal cannula 3 L/min, with a PF ratio of 433. On postoperative day one, prior to intubation, pO_2 dropped to 49 mmHg (PF ratio 82) on simple mask 6 L/min. After intubation, pO_2 improved to 148 mmHg (PF ratio 246) on FiO_2 60%, though pCO_2 rose to 71 mmHg, indicating respiratory acidosis. Subsequent evaluations on postoperative days two and three demonstrated compensated respiratory acidosis with stable oxygenation (PF ratio 233–220). However, by postoperative day four, severe hypercapnic failure developed with a PF ratio of 120 and deteriorated to a PF ratio of 64 by day five, consistent with progressive type II respiratory failure. Serial Chest X-ray revealed progressively prominent infiltrates with each subsequent day. These radiographic changes were clinically correlated with arterial blood gas (ABG) analysis results, which showed a progressive decline in oxygenation parameters consistent with the worsening pulmonary findings.

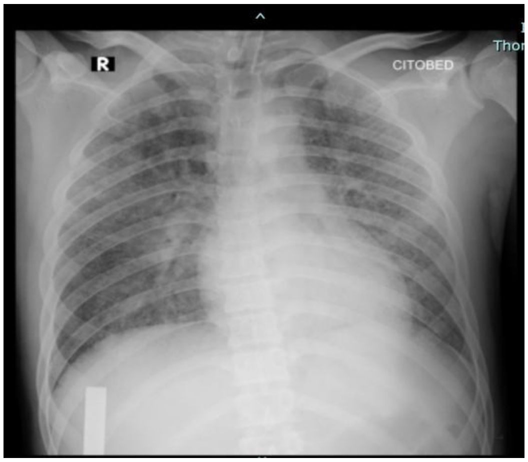
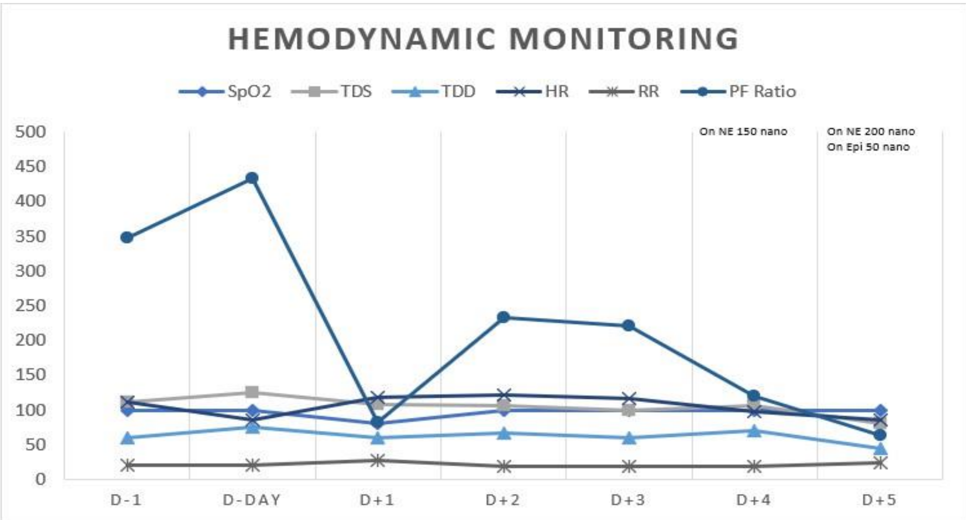


Figure 3: Chest X-ray D+4 Post Operation

Additional data:



*NE : Norephynephrine; Epi : Epinephrine

Figure 4: Hemodynamic Monitoring

DISCUSSION

This case highlights the dynamic progression of pulmonary complications following high- energy blunt trauma, even in the absence of initial physical and radiologic findings. Despite early hemodynamic stabilization and uneventful surgery, the patient developed delayed pulmonary contusion and progressive respiratory failure. Early recognition of delayed pulmonary contusion is crucial because early and appropriate management can mitigate progression toward severe respiratory failure or acute respiratory distress syndrome (ARDS), which carries a high morbidity and mortality risk in trauma patients [2].

Pulmonary contusion represents the most common parenchymal injury following blunt thoracic trauma. It is defined as damage to the alveolar-capillary membrane resulting in alveolar hemorrhage and interstitial edema without macroscopic laceration of the lung tissue [3,4]. The injury leads to impaired gas exchange, reduced pulmonary compliance, and increased pulmonary shunt fraction.

While most contusions are visible on the initial chest imaging, a subset of patients develops pulmonary opacities in a delayed time typically within 24 to 48 hours after trauma, referred to as delayed pulmonary contusion [2].

The delayed presentation can be attributed to progressive microvascular injury, endothelial activation, and inflammatory mediator release that compromise the integrity of the alveolar- capillary barrier over time [5]. Initially, the patient may appear hemodynamically stable with normal oxygenation and unremarkable imaging findings like the patient in this case, even without bruise in thoracoabdominal area. However, as inflammatory cascades evolve, alveolar flooding and ventilation-perfusion mismatch ensue, manifesting as hypoxemia and respiratory distress. This delayed onset poses diagnostic challenges, often leading clinicians to misinterpret the findings as aspiration, atelectasis, or early pneumonia rather than evolving contusion [5].

Clinically, pulmonary contusion should be suspected in any patient with blunt chest trauma who develops unexplained hypoxemia or respiratory distress. The physiologic abnormality is hypoxemia that appears disproportionate to the degree of radiologic abnormality. The mechanism involves alveolar collapse, intrapulmonary shunting, and decreased lung compliance due to alveolar flooding.

Radiographic findings typically appear within the first 6 hours but may progress over 24 to 48 hours. On chest radiography, the contusion appears as patchy, non-segmental alveolar infiltrates, often located adjacent to the area of impact and not confined to lobar or vascular boundaries. The opacities do not respect anatomical divisions because the injury follows the distribution of the mechanical force rather than the bronchial tree. Computed tomography (CT) is more sensitive, revealing ground-glass opacities, consolidation, and interstitial thickening that correlate with the degree of parenchymal injury [6].

From a physiologic standpoint, blood gas analysis typically demonstrates a decreased PaO_2/FiO_2 ratio, and lung compliance measurements show reduced distensibility. Clinically, patients may present with tachypnea, increased work of breathing, and occasionally hemoptysis. However, the absence of these findings does not exclude significant injury, particularly in the delayed form. The progressive nature of the process underscores the need for continuous monitoring, even when initial assessments are normal.

Lung ultrasonography (LUS) has revolutionized bedside pulmonary evaluation in both emergency and critical care settings. It offers a rapid, non-invasive, and radiation-free tool for the detection and follow-up of pulmonary contusions. The ultrasonographic hallmark of contusion includes the presence of multiple, confluent B-lines representing interstitial edema, subpleural consolidations indicating alveolar filling, and irregular or fragmented pleural lines that reflect underlying parenchymal disruption [7].

In delayed pulmonary contusion, these findings may appear before radiographic changes, making LUS an excellent tool for early detection. Serial ultrasonographic examinations allow clinicians to monitor disease evolution dynamically, assess response to therapy, and identify complications such as atelectasis or pleural effusion. Compared with CT, which remains the gold standard for diagnosis, LUS offers portability and the advantage of real-time bedside assessment, which is particularly beneficial in unstable or postoperative trauma patients where transport carries significant risk [8].

Moreover, LUS is valuable for guiding ventilatory management decisions. For instance, assessing reaeration patterns helps determine the effectiveness of positive end-expiratory pressure (PEEP) adjustments. The integration of lung ultrasound into trauma care protocols, such as the extended Focused Assessment with Sonography for Trauma (eFAST), has improved early recognition of evolving pulmonary injuries and reduced dependence on repeated radiation-based imaging [8].

Initial management of trauma in these patients follows the Advanced Trauma Life Support (ATLS) principles: securing the airway, ensuring adequate breathing, and maintaining circulation. In patients with pulmonary contusion, special attention must be paid to oxygenation and fluid management [9]. Although hypovolemia remains a primary concern in trauma management, excessive fluid resuscitation may aggravate pulmonary interstitial edema and compromise gas exchange secondary to increased capillary permeability [10]. Nonetheless, in the absence of overt thoracic injury or radiologic abnormalities, this potential complication is frequently underestimated. However, in the absence of hypotension or vasopressor requirements, a fluid-conservative strategy is recommended [11].

The pathophysiology of trauma-induced ARDS often involves a “two-hit” model. The first hit is the direct parenchymal injury from the contusion, and the second hit arises from systemic inflammatory responses triggered by surgery, transfusion, infection, or ischemia-reperfusion injury. The activation of neutrophils and cytokines, such as interleukin-6 and tumor necrosis factor-alpha, amplifies the inflammatory cascade, resulting in diffuse alveolar damage, surfactant dysfunction, and loss of alveolar integrity [12].

In postoperative settings, the risk of worsening pulmonary function increases due to several factors: prolonged mechanical ventilation, high inspired oxygen concentrations, fluid overload, and impaired mucociliary clearance. Residual anesthetic effects may further depress respiratory drive and airway protection, increasing the likelihood of atelectasis and infection. Together, these processes may precipitate ARDS in a patient whose lungs were initially injured by contusion but subsequently destabilized by secondary insults [13].

Noninvasive positive pressure ventilation (NIPPV) may be effective in avoiding intubation in selected trauma patients with pulmonary contusions and ARDS when combined with multimodal analgesia, including epidural and systemic analgesics. Nevertheless, patients with frank respiratory failure, airway compromise, hemodynamic instability, or significant agitation should be promptly intubated. Following landmark studies supporting lung-protective ventilation, low tidal volume and low plateau pressure ventilation have become standard in ARDS management, including in trauma-related cases [14]. While PEEP assists alveolar recruitment, its optimal level remains uncertain, excessive PEEP may worsen ventilation which perfusion mismatch in unilateral lung injury [15].

Neuromuscular blocking agents may improve ventilator synchrony and outcomes in severe ARDS, awhere a short course of paralysis improved 90-day survival and ventilator-free days [16]. Meanwhile pharmacologic interventions such as prostaglandin E_1, N-acetylcysteine, early high-dose corticosteroids, and surfactant lack sufficient evidence for benefit in ALI or ARDS [16].

Adjunctive strategies like prone positioning improve oxygenation but only confer a survival benefit in patients with severe ARDS (PaO_2/FiO_2 <100 mmHg). Extracorporeal membrane oxygenation (ECMO) may serve as a life-saving intervention for refractory hypoxemia, with newer studies showing improved survival in trauma-related ARDS and the advantage of heparin-bonded circuits allowing delayed systemic anticoagulation [17].

Overall, trauma patients with pulmonary contusion should be managed using an individualized, evidence-informed strategy emphasizing conservative fluid therapy, lung-protective ventilation, optimal analgesia, and judicious use of adjunctive modalities such as NIPPV, ECMO, or surgical fixation when indicated [14].

CONCLUSION

Delayed pulmonary contusion is a dynamic and evolving manifestation of blunt thoracic trauma. Early imaging may underestimate the severity of lung injury, and deterioration can occur hours to days later due to progressive inflammatory processes. Lung ultrasound serves as an invaluable modality for early detection and continuous monitoring. A meticulous balance resuscitation and lung-protective ventilatory support remains the cornerstone of management. Understanding the transition from contusion to ARDS, especially in postoperative or multiply injured patients, enables clinicians to implement timely interventions and reduce mortality.

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