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CASE REPORT

Silent Lung Injury: Acute Respiratory Distress Syndrome without Chest Trauma Following a High-Level Fall

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Abstract: Acute respiratory distress syndrome (ARDS) is a life-threatening form of non-cardiogenic respiratory failure that can occur after severe systemic or traumatic insults. We report a rare case of ARDS in a 17-year-old female who sustained a burst fracture at the second lumbar vertebra and bilateral calcaneal fractures after jumping from a height of six meters. She underwent uneventful spinal decompression and instrumentation six hours post-injury, with stable hemodynamics and balanced fluid administration. Initial chest CT showed no lung abnormalities. On postoperative day 2, she developed respiratory distress preceded by a metallic, blood-like taste during breathing. Follow-up CT revealed new bilateral diffuse lung consolidations. Bronchoscopy showed diffuse pulmonary hemorrhage without active bleeding, suggesting lung contusion. Echocardiography excluded cardiac causes, and extensive microbiological testing, including bronchoalveolar lavage and viral panels, yielded no infectious pathogens. Although broad-spectrum antibiotics were used in response to perioperative fever and elevated inflammatory level, no definitive infection was identified, and inflammatory markers normalized with ventilatory support. This case illustrates a potential underrecognized mechanism of ARDS: pulmonary contusion from high-energy vertical deceleration in the absence of external chest trauma. To our knowledge, no prior literature has reported ARDS following spinal fractures due to vertical falls without chest wall injury. We propose that sudden deceleration during impact can transmit force through the thoracic cage, causing occult microvascular lung injury. Early recognition and close respiratory monitoring are essential in similar trauma scenarios, even when initial imaging is unremarkable.

Plain Language Summary: A 17-year-old girl fell from a 6-meter-high bridge, breaking her spine and heels. Although her chest looked normal at first, she later developed serious breathing problems. Doctors believe the strong impact from the fall caused hidden lung damage through a "deceleration force", a sudden stop that injured lung tissues without breaking ribs. This led to a rare condition called ARDS. The case shows that even without chest injury, dangerous lung problems can still happen after big falls. Careful follow-up is important.

Keywords: acute respiratory distress syndrome, pulmonary contusion, occult lung injury, vertical fall, postoperative respiratory failure

Introduction

Acute respiratory distress syndrome (ARDS) is a severe manifestation of acute lung injury characterized by hypoxemia, bilateral pulmonary infiltrates, and respiratory failure.[1,2] Common causes include sepsis, pneumonia, shock, pancreatitis, chest trauma, and transfusion-related acute lung injury (TRALI).[3–5] Blunt chest trauma from high-energy mechanisms such as motor vehicle collisions is a well-established trigger of ARDS, typically through the development of lung contusion.[6,7] Lung contusion involves disruption of alveolar-capillary membranes, leading to hemorrhage, inflammation, and impaired gas exchange.[6] Trauma-related ARDS occurs in approximately 10–30% of critically injured patients, often presenting with delayed onset 24 to 48 hours post-injury.[8,9] While pulmonary contusion is typically associated with chest

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trauma, it can occur without visible thoracic injury, especially in high-velocity vertical deceleration events. In such mechanisms, the rapid deceleration force can be transmitted through the spine and thorax, causing microvascular pulmonary injury without rib fractures or external chest signs.

To our knowledge, ARDS following spinal fractures from high-level falls in the absence of overt chest trauma and initial radiographic abnormalities has been rarely described. The novelty of this case lies not in the timing of ARDS onset, which occurred within the expected post-trauma window, but in the absence of clinically apparent chest injury and the presence of direct bronchoscopic evidence of pulmonary microvascular hemorrhage. In this case, a 17-year-old female developed ARDS two days after a high-energy fall, with bronchoscopic evidence of microvascular hemorrhage despite an initially normal chest computer tomography (CT). This case highlights the need to consider deceleration-induced pulmonary contusion in the differential diagnosis of ARDS, even when early clinical and radiographic evaluations appear unremarkable.

Case Report

A 17-year-old female sustained a high-energy vertical fall from a 6-meter pedestrian overpass after an accidental fall in a public setting during the early morning hours. Upon arrival at the emergency department, she was alert, afebrile, and complained of severe pain in the thoracolumbar region and both heels. Her body mass index was approximately 24.2. She had no significant past medical history and no known respiratory diseases. No external signs of head or chest trauma were observed. Her Glasgow Coma Scale score was 15. Neurological examination revealed a Frankel Grade D spinal cord injury, characterized by preserved motor function with mild sensory impairment.

Initial imaging, including head and abdominal CT scans, showed no evidence of intracranial or intra-abdominal hemorrhage. Chest CT demonstrated clear lung fields without pneumothorax, hemothorax, or pulmonary contusion.[7] However, a burst fracture of the second lumbar vertebra (L2) was identified, with retropulsed bone fragments compressing the spinal cord (Figure 1). The patient underwent emergency posterior spinal decompression and instrumentation six hours after arrival. (Figure 2). The procedure lasted 4.5 hours and was completed with stable intraoperative hemodynamics and balanced fluid administration.[10]

Approximately 51 hours after trauma, the patient developed acute respiratory distress characterized by dyspnea, agitation, and hypoxemia (Figure 3). Notably, on postoperative day 2, the deterioration was preceded by an unusual symptom, as the patient reported a metallic, blood-like taste during breathing. Arterial blood gas analysis revealed a PaO_2 /FiO_2 ratio consistent with moderate to severe ARDS as defined by the Berlin criteria (Table 1).[11]

Follow-up chest CT demonstrated new-onset bilateral pulmonary infiltrates not present on admission imaging (Figure 4). Transthoracic echocardiography showed normal left ventricular function, with a left ventricular ejection

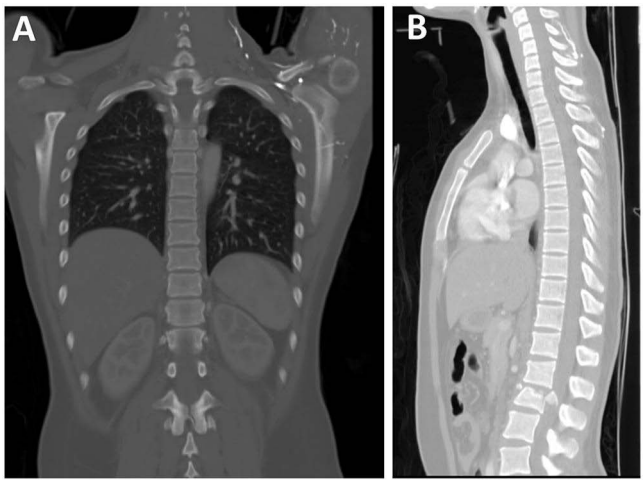

Figure 1 Initial chest CT scan in the emergency department. (**A**) Coronal view showing clear lung fields without infiltrates or opacities. (**B**) Sagittal view revealing an L2 burst fracture with retropulsed bone fragments compressing the spinal cord.

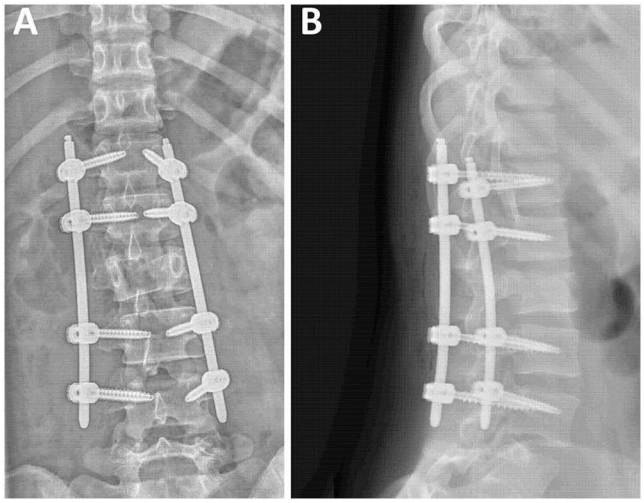

Figure 2 Postoperative thoracolumbar spine radiographs. (**A**) Anteroposterior view. (**B**) Lateral view demonstrating L2 fracture fixation and decompression.

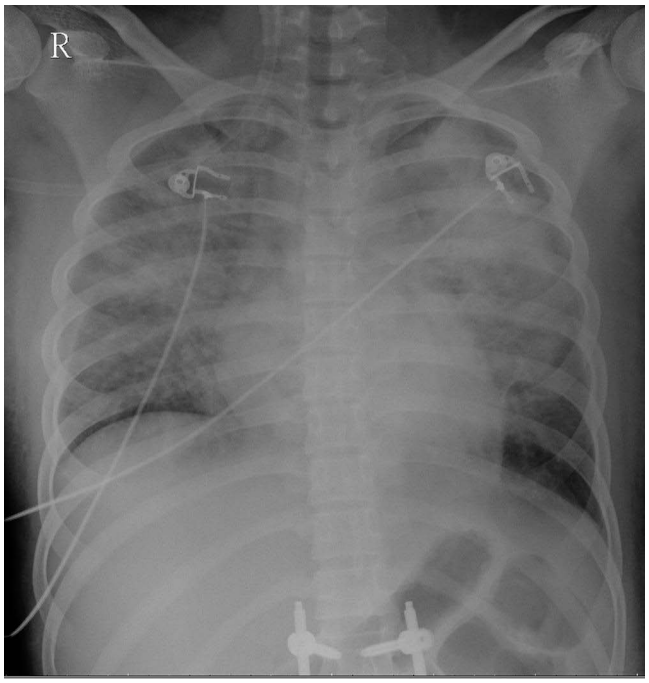

Figure 3 Chest X-ray obtained 51 hours post-injury, showing extensive bilateral diffuse opacities, raising suspicion for ARDS.
Abbreviation: R, Right.

fraction of 78%, effectively ruling out cardiogenic pulmonary edema. Although an elevated N-terminal pro–B-type natriuretic peptide level (999 pg/mL) was noted during the acute respiratory deterioration, the echocardiographic findings were not consistent with cardiogenic pulmonary edema. Bronchoscopy revealed diffuse microvascular hemorrhage throughout the airways without active bleeding, findings consistent with pulmonary contusion.

An extensive microbiological evaluation was conducted to investigate potential infectious etiologies. At presentation, the patient was afebrile and had no respiratory symptoms. Postoperatively, she developed transient high-grade fever up to 39 °C on the day of surgery and postoperative day 1, prompting initiation of empirical antibiotic therapy. On postoperative day 2, inflammatory markers were elevated, with a C-reactive protein (CRP) level of 23.05 mg/L, and broad-spectrum antibiotics were therefore continued. Despite these measures, cultures of blood, sputum, urine, and

Table 1 Arterial Blood Gas Parameters During Acute Respiratory Failure and After Initiation of Mechanical Ventilation

Test Item	Report Item	Intubation	10 Hours Post-Intubation
Blood gas analysis	pH	7.394	7.489
Blood gas analysis	$PaCO_2$	27.1 mmHg	26.4 mmHg
Blood gas analysis	PaO_2	107.2 mmHg	59.5 mmHg
Blood gas analysis	Base Excess	−6.2mmol/L	−1.5 mmol/L
Blood gas analysis	HCO_3^-	16.7 mmol/L	20.3 mmol/L
Blood gas analysis	O_2 Saturation	98.5%	92.7%
Blood gas analysis	FiO_2	100%	60%

Abbreviations: pH, potential of hydrogen; $PaCO_2$, partial pressure of arterial carbon dioxide; PaO_2, partial pressure of arterial oxygen; HCO_3^-, Hogen carbonate ion; FiO_2, fraction of inspired oxygen.

bronchoalveolar lavage fluid remained negative, and viral testing, including influenza A and B, was unremarkable. No microbiological evidence of infection was identified throughout the clinical course. The patient's respiratory status improved with ventilatory support, and inflammatory markers subsequently normalized without further escalation of antimicrobial therapy.

These findings support a diagnosis of ARDS secondary to pulmonary contusion rather than infectious or cardiogenic etiologies, suggesting a mechanism of occult lung injury related to high-energy vertical deceleration without direct chest wall impact.

Discussion

This case describes a rare instance of ARDS in a 17-year-old female who sustained a vertical fall from a 6-meter pedestrian overpass, resulting in spinal and bilateral calcaneal fractures, but no overt chest trauma. Despite initially stable hemodynamics, no fever on arrival, and normal preoperative chest imaging, the patient developed acute hypoxemic respiratory failure approximately 51 hours post-injury, meeting the Berlin criteria for moderate to severe ARDS under appropriate positive end-expiratory pressure support.[11] Chest CT at the time demonstrated new-onset bilateral pulmonary infiltrates,[6] and bronchoscopy confirmed diffuse alveolar hemorrhage consistent with pulmonary contusion.[12,13] Transthoracic echocardiography demonstrated preserved left ventricular systolic function,[14] and extensive microbiological testing yielded no infectious etiology.

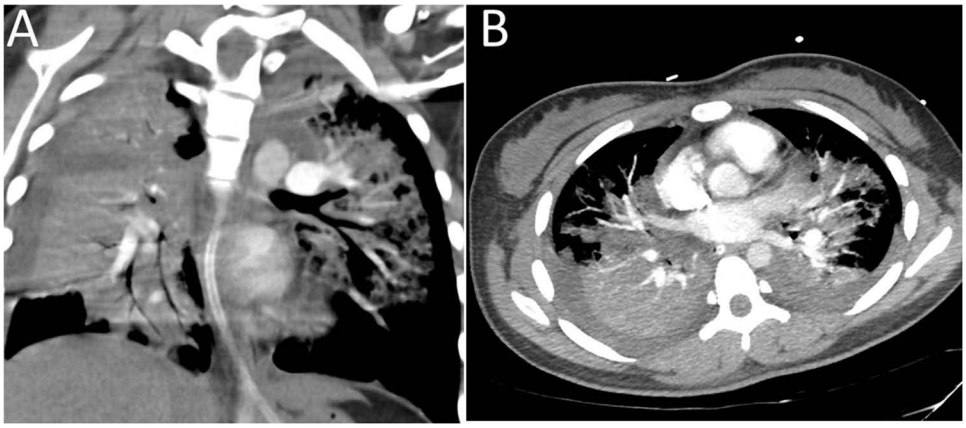

Figure 4 Follow-up chest CT scan one day after intubation. (**A**) Coronal view. (**B**) Axial view. Both images show bilateral lung opacities consistent with ARDS.

In evaluating the cause of ARDS in this patient, several alternative etiologies were carefully considered and subsequently excluded. Cardiogenic pulmonary edema was initially suspected due to an elevated NT-proBNP level during the acute phase. However, transthoracic echocardiography demonstrated preserved systolic function, making cardiogenic pulmonary edema unlikely.[14] Infectious etiologies such as pneumonia or sepsis were unlikely, as there were no clinical signs of infection and cultures from blood, urine, sputum, and bronchoalveolar lavage remained negative throughout the hospital course.[1,2] Additionally, both CRP levels and fever resolved without further escalation of antimicrobial therapy, further arguing against an infectious cause. Fat embolism syndrome was considered given the presence of vertebral and bilateral calcaneal fractures; however, the patient lacked characteristic signs such as petechial rash, neurological symptoms, or pulmonary vascular filling defects on CT, rendering this diagnosis improbable.[15,16] TRALI was also unlikely, as respiratory deterioration occurred more than 24 hours after transfusion, well beyond the 6-hour window typically associated with TRALI, and there were no signs of hypotension or leukocyte-mediated response.[4]

Importantly, the significance of this case does not lie in an unexpected temporal pattern of ARDS development, as the onset occurred within the expected timeframe following trauma. Rather, the clinical relevance resides in the development of ARDS in the absence of overt chest trauma or early radiographic abnormalities, supported by direct bronchoscopic evidence of pulmonary microvascular hemorrhage. In addition, intraoperative records documented stable hemodynamics, normovolemia, and limited transfusion during spinal surgery, further reducing the likelihood of transfusion-related lung injury as a contributing factor.[10]

The most plausible explanation for this patient's ARDS is pulmonary contusion resulting from high-energy vertical deceleration. Although the patient presented without external signs of chest trauma and initial imaging was unremarkable, the substantial downward force during the fall likely generated intrathoracic shearing stress sufficient to disrupt the alveolar-capillary membrane.[6,7] Similar injury mechanisms have been observed in motor vehicle collisions, where significant pulmonary injury may occur in the absence of chest wall bruising or rib fractures.[7,13] In this case, the first clinical clue was a metallic, blood-like taste during respiration on postoperative day 2, which may have reflected early alveolar hemorrhage preceding over hypoxemia. As is typical in pulmonary contusion, radiographic abnormalities became apparent within 24–72 hours after trauma and subsequently progressed to diffuse bilateral infiltrates consistent with ARDS.[6,9] Bronchoscopic findings further corroborated the diagnosis by revealing diffuse alveolar bleeding.[12,13]

This case underscores the importance of maintaining clinical vigilance for ARDS even in trauma patients without apparent chest injury. High-energy vertical deceleration can result in occult pulmonary contusion, which may not be radiographically evident in the early phase but can evolve rapidly. Serial chest imaging, early bronchoscopy, and close respiratory monitoring are critical for early identification and intervention. Furthermore, this case illustrates the importance of integrating clinical, radiologic, and laboratory data to guide diagnosis and avoid premature attribution of symptoms to cardiac, infectious, or transfusion-related etiologies. Awareness of subtle prodromal symptoms, such as altered respiratory sensations, may facilitate earlier identification of evolving pulmonary injury before progression to overt respiratory failure.

Conclusion

This case highlights that ARDS may develop within the expected post-trauma period even when no overt chest injury is clinically apparent and initial chest imaging is normal. The combination of a negative infectious and cardiogenic workup and direct bronchoscopic evidence of pulmonary microvascular hemorrhage supports occult traumatic lung injury as a plausible underlying mechanism of respiratory failure. Although pulmonary contusion can result from both direct and indirect mechanisms, this case underscores the importance of clinical vigilance, repeat imaging, and close respiratory monitoring in trauma patients exposed to high-energy mechanisms, as early recognition and supportive management may help mitigate progression to severe respiratory compromise.

Data Sharing Statement

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethical Approval and Informed Consent

The study protocol was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board of Changhua Christian Hospital, Taiwan (IRB approval number: 250419; approval date: May 20, 2025). According to institutional policy, publication of the case report was included in the approved protocol and did not require additional separate approval for publication. The patient provided written informed consent for the publication of this case report, including clinical information and accompanying images. The patient reviewed the final version of the manuscript and understands that identifiable details will be published with efforts made to ensure anonymity. A copy of the signed consent form is available from the corresponding author upon reasonable request.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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