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*Case report*

# Trapped Lung and Pneumothorax ex Vacuo Presenting as Refractory Hypercapnic Respiratory Failure in a Hemodialysis Patient: A Case Report

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**Abstract**
Trapped lung is a form of non-expandable lung caused by fibrotic restriction of the visceral pleura, failing lung re-expansion despite adequate pleural drainage. Pneumothorax ex vacuo may occur following fluid evacuation and should be distinguished from conventional pneumothorax. We report a 69-year-old man with end-stage renal disease on maintenance hemodialysis, diabetes mellitus, peripheral vascular disease, and multiple comorbidities who presented with progressive dyspnea, altered mental status, and severe hypercapnic respiratory failure. Arterial blood gas analysis showed a pH of 7.15, $PaCO_2$ of 81 mmHg, and $HCO_3^-$ of 13 mmol/L. BiPAP resulted in initial improvement in hypercapnia and mental status; however, the patient remained ventilator-dependent. A right-sided chest tube subsequently drained approximately 2300 mL of transudative pleural fluid, without significant lung re-expansion. Chest CT demonstrated pneumothorax ex vacuo, thickened visceral pleura, and chronically collapsed right lung, supporting the diagnosis of trapped lung. Despite continued ventilatory support, the patient developed progressive respiratory fatigue and required invasive mechanical ventilation. Thoracic surgery deemed decortication inappropriate because of his poor physiological reserve and advanced comorbidities. The patient subsequently developed refractory cardiorespiratory failure and died. This case highlights trapped lung with pneumothorax ex vacuo as an under-recognized cause of refractory hypercapnic respiratory failure. It emphasizes that failure of lung re-expansion after pleural drainage should prompt consideration of non-expandable lung physiology, even when the pleural effusion is transudative.
**Keywords:** Trapped Lung, Non-Expandable Lung, Pneumothorax Ex Vacuo, Hypercapnic Respiratory Failure.

## Introduction

Trapped lung is a subtype of non-expandable lung caused by formation of a fibrotic visceral pleural peel that mechanically restricts normal pulmonary expansion [1,5]. The condition usually develops following chronic pleural inflammation secondary to infection, malignancy, hemothorax, thoracic surgery, or recurrent pleural effusions [1,2]. Patients commonly present with dyspnea, recurrent pleural effusions, and persistent pleural space following thoracentesis or chest tube drainage [2,5].

An important associated phenomenon is pneumothorax ex vacuo, which develops when pleural fluid evacuation is followed by failure of lung re-expansion [2,5]. In this setting, persistent negative intrapleural pressure results in passive air accumulation within the pleural space. Unlike conventional pneumothorax, pneumothorax ex vacuo does not result from visceral pleural rupture or bronchopleural fistula and therefore does not represent a true persistent air leak [2,5].

Recognition of trapped lung is clinically important because repeated pleural drainage procedures frequently fail to improve pulmonary mechanics and may expose patients to unnecessary invasive interventions [1,2,5]. Although dyspnea is common, severe hypercapnic respiratory failure requiring escalation from non-invasive to invasive mechanical ventilation has been infrequently described [6]. We report a case of trapped lung with pneumothorax ex vacuo in a patient with end-stage renal disease on maintenance hemodialysis who developed refractory hypercapnic respiratory failure despite pleural drainage and escalating ventilatory support.

## *Case Presentation*

A 69-year-old man with hypertension, long-standing diabetes mellitus complicated by advanced diabetic retinopathy and glaucoma, peripheral vascular disease with prior below-knee amputation, and end-stage renal disease on maintenance hemodialysis presented with progressive respiratory distress and altered mental status. According to family members, the patient had experienced progressive excessive daytime sleepiness, generalized weakness, and fluctuating level of consciousness for several days before admission. Progressive dyspnea and reduced functional capacity were also noted. On examination, the patient appeared drowsy with evidence of increased work of breathing. Respiratory rate was 24 breaths/min, oxygen saturation was 75% on room air, blood pressure was 130/90 mmHg, and pulse rate was 107 beats/min. Oxygen saturation improved following initiation of non-invasive ventilatory support. Chest examination revealed markedly decreased air entry over the right hemithorax, predominantly involving the mid and basal zones, with associated bronchial breathing. Approximately one month before the current presentation, the patient had been hospitalized because of worsening dyspnea and orthopnea. At that time, pleural effusion had been identified, and therapeutic pleural drainage was performed with temporary symptomatic improvement. However, the patient subsequently left the hospital against medical advice shortly after the procedure, and therefore further post-drainage evaluation, follow-up imaging, and assessment of


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lung re-expansion were not completed. Initial arterial blood gas analysis demonstrated severe acidemia and marked hypercapnia with pH 7.15, $PaCO_2$ 81 mmHg, and $HCO_3^-$ 13 mmol/L, consistent with acute hypercapnic respiratory failure associated with mixed respiratory and metabolic acidosis. Laboratory investigations demonstrated elevated inflammatory markers, with CRP increasing from 46.4 mg/L to 113.7 mg/L during hospitalization. Renal profile was consistent with end-stage renal disease, with serum creatinine ranging from 6.0 to 7.0 mg/dL and serum urea ranging from 143 to 168 mg/dL despite maintenance hemodialysis. Serum LDH was 275 U/L. Electrolytes remained relatively stable throughout admission. Pleural fluid analysis was performed bilaterally and demonstrated low protein and LDH levels consistent with transudative pleural effusion according to Light's criteria [7,8]. Right-sided pleural fluid protein was 2.2 g/dL with LDH of 96 U/L, while left-sided pleural fluid protein was 2.6 g/dL with LDH of 68 U/L. Pleural fluid pH was 7.38. Microbiological investigations, including pleural fluid cultures and GeneXpert testing for tuberculosis, were negative.

Complete blood count demonstrated neutrophilic leukocytosis and chronic normocytic anemia with hemoglobin of 8.0 g/dL. Troponin-T and CK-MB levels were within normal limits. The patient was commenced on non-invasive ventilation using BiPAP mode, resulting in marked initial improvement in carbon dioxide clearance and mental status. However, despite successful early correction of hypercapnia, he remained dependent on BiPAP support and was unable to maintain adequate spontaneous ventilation independently. Empirical broad-spectrum antibiotics were initiated because infection could not initially be excluded in the setting of respiratory failure and pleural disease. Hemodialysis was also performed as part of supportive management. Radiological evaluation demonstrated significant right-sided pleural pathology with incomplete expansion of the right lung. (see figure 1, 2 x ray and CT chest respectively).

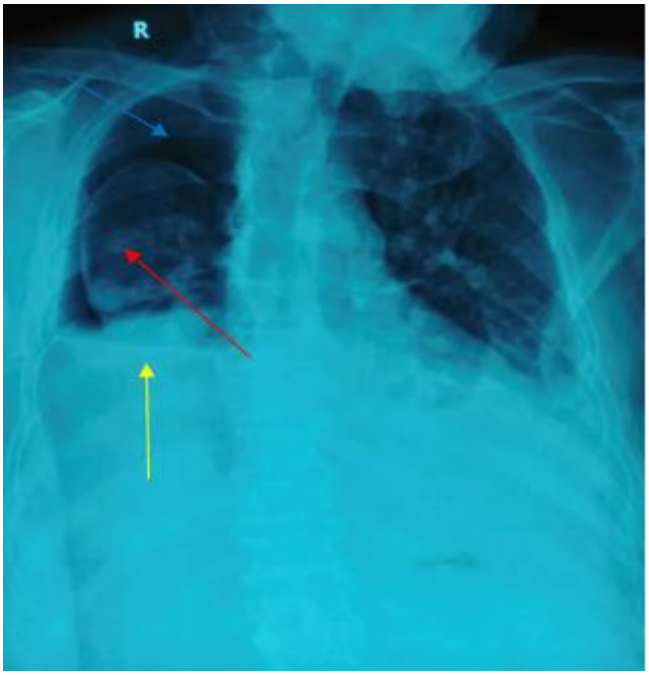


***Figure 1. Chest radiograph at presentation demonstrating right-sided pleural effusion (yellow arrow), collapsed right lung (red arrow), and associated pneumothorax ex vacuo (blue arrow)***


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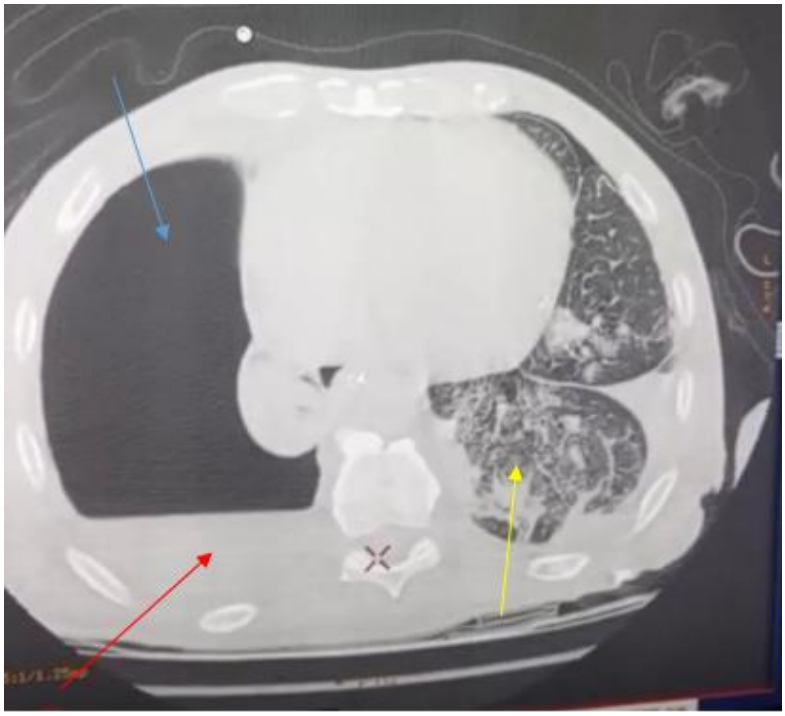

***Figure 2. CT chest image demonstrating large right-sided pneumothorax ex vacuo (blue arrow), residual pleural effusion (red arrow), and left-sided pulmonary consolidation (yellow arrow)***

On the second hospital day, a right-sided chest tube was inserted for both diagnostic and therapeutic purposes, resulting in drainage of approximately 2300 mL of pleural fluid. Despite large-volume pleural drainage, no significant clinical or radiological improvement in lung expansion was achieved. Computed tomography of the chest demonstrated right-sided pneumothorax ex vacuo with thickened visceral pleura and chronically collapsed lung, findings strongly supportive of trapped lung physiology [2,5] (see figures 3 and 4).

***Figure 3. CT chest image demonstrating pneumothorax ex vacuo (blue star), thickened visceral pleura consistent with trapped lung (red arrow), and right-sided chest tube in situ (yellow arrow).***

During hospitalization, the patient developed progressive respiratory fatigue with increasing dependence on non-invasive ventilation despite initial biochemical improvement. By the fifth hospital day, worsening hypercapnic respiratory failure necessitated endotracheal intubation and initiation of invasive mechanical ventilation. Thoracic surgical consultation was obtained for consideration of surgical intervention such as decortication. However, the patient was considered unsuitable for operative management because of poor general condition, advanced comorbidities, and severe respiratory failure. Despite escalation to full ventilatory support and maximal supportive therapy, the patient continued to deteriorate clinically and eventually died from refractory cardiorespiratory failure


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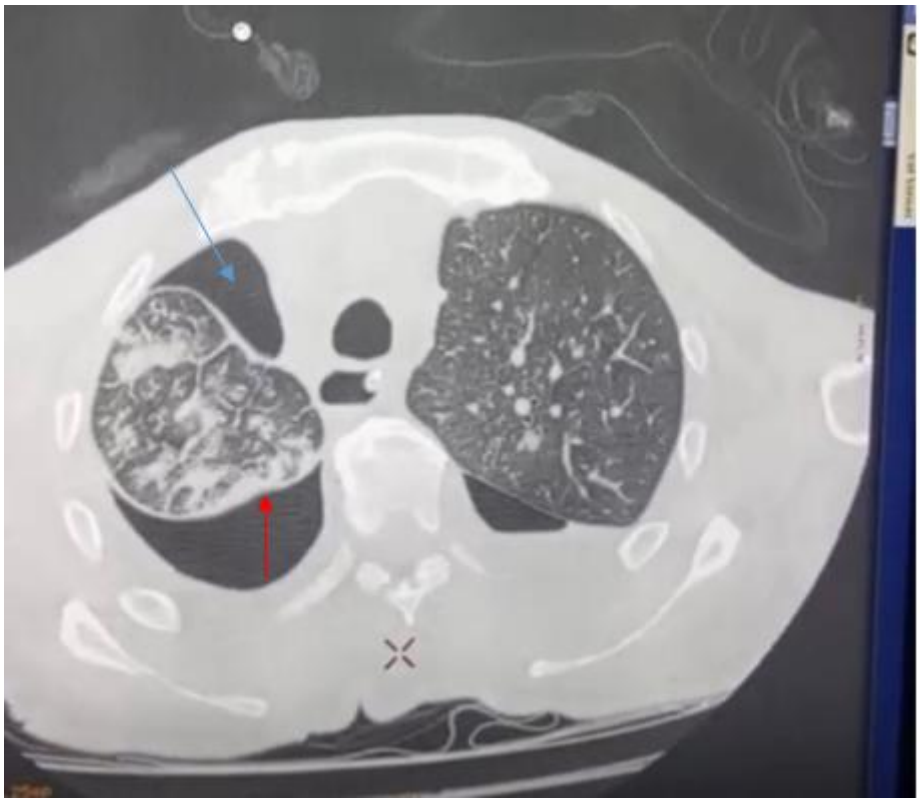

***Figure 4. CT chest image demonstrating chronically collapsed, thickened right lung (red arrow) with persistent pneumothorax ex vacuo (blue arrow)***

**Discussion**

Trapped lung is characterized by failure of lung expansion due to fibrotic restriction of the visceral pleura, resulting in persistent pleural space despite adequate drainage [1,5]. The condition develops as a consequence of prior pleural inflammation and may remain unrecognized until thoracentesis or chest tube drainage reveals failure of pulmonary re-expansion [2,5]. In the present case, persistent right-sided residual pleural space following large-volume drainage strongly supported the diagnosis of trapped lung, a classic radiological feature of non-expandable lung physiology [2,5]. The previous episode of pleural effusion one month before admission may have contributed to chronic pleural inflammation and progressive fibrotic remodeling of the visceral pleura, ultimately resulting in non-expandable lung physiology [1,6]. Furthermore, the patient's discharge against medical advice following the initial pleural drainage likely contributed to delayed recognition of the underlying pathology because no reassessment of lung expansion was performed. An important associated phenomenon in this setting is pneumothorax ex vacuo. This entity develops following pleural fluid evacuation when the lung fails to re-expand, resulting in passive air accumulation within the pleural space because of persistent negative intrapleural pressure [2,5]. Unlike traditional pneumothorax, pneumothorax ex vacuo is not caused by visceral pleural rupture or bronchopleural fistula, but rather develops secondary to persistent negative pleural pressure following failure of lung re-expansion [2,5].

Recognition of this phenomenon is clinically important because misinterpretation may lead to unnecessary procedures and prolonged chest tube management [5]. Failure of lung re-expansion despite adequate chest tube drainage reflects the underlying mechanical restriction imposed by the fibrotic visceral pleural peel [1,5]. In trapped lung physiology, removal of pleural fluid alone is insufficient to restore normal lung expansion because the chronically fibrosed visceral pleura prevents re-expansion of the underlying lung parenchyma [1,2]. Therefore, persistent pleural space after apparently successful drainage should not automatically be interpreted as inadequate tube position or persistent air leak, but may instead indicate underlying non-expandable lung physiology [2,5]. An unusual feature of this case was the presence of transudative pleural effusion despite imaging findings suggestive of chronic pleural fibrosis. Trapped lung is more commonly associated with exudative effusions because of chronic pleural inflammation [1,7]. However, transudative effusions may occasionally occur in trapped lung physiology because of altered pleural fluid dynamics and chronic pleural pressure abnormalities [1,8]. In the present patient, end-stage renal disease, chronic fluid dysregulation related to hemodialysis, and altered pleural fluid dynamics associated with chronic pleural fibrosis may have contributed to the transudative pleural profile despite established non-expandable lung physiology. The patient developed severe hypercapnic respiratory failure with altered mental status suggestive of hypercapnic encephalopathy. Severe ventilatory impairment in trapped lung may result from marked restriction of functional lung expansion and impaired respiratory mechanics [2,6]. The hypercapnia in the present case was likely multifactorial, predominantly resulting from impaired ventilatory mechanics due to non-expandable lung physiology. The markedly reduced expandable lung volume likely increased the work of breathing and impaired effective


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alveolar ventilation. This was further compounded by limited physiological reserve, metabolic derangements, end-stage renal disease, and multiple advanced comorbidities.

An important clinical feature was the patient's initial improvement with BiPAP ventilation, indicating partial reversibility of carbon dioxide retention during the early phase of respiratory support. However, despite biochemical improvement, the patient remained unable to sustain spontaneous ventilation independently and gradually developed progressive ventilatory dependence. The subsequent requirement for invasive mechanical ventilation underscores the dominant role of irreversible mechanical restriction in driving progressive respiratory failure. Previous reports have primarily focused on dyspnea and recurrent pleural effusion as the major manifestations of trapped lung [1,6]. In contrast, the present case demonstrated progressive refractory hypercapnic respiratory failure requiring escalation from non-invasive ventilation to invasive mechanical ventilation, highlighting the potentially severe ventilatory consequences of advanced non-expandable lung physiology in medically fragile patients. Thoracic surgical intervention such as decortication may be considered in selected patients with trapped lung [1,5]. However, surgical management was considered inappropriate in the present patient because of advanced comorbidities, poor physiological reserve, and severe respiratory failure.

Although the outcome was fatal, this case provides several important educational messages. First, persistent pleural space after drainage should prompt early consideration of trapped lung and pneumothorax ex vacuo [2,5]. Second, failure of lung re-expansion after large-volume drainage is an important diagnostic clue to non-expandable lung physiology [1,5]. Third, severe hypercapnic respiratory failure may occur even in unilateral pleural disease when ventilatory mechanics are significantly impaired. Finally, early recognition of trapped lung may help avoid repeated ineffective pleural interventions and guide more appropriate management strategies in medically fragile patients [1,2,5]. Pleural manometry and histopathological confirmation were not available in this case, and the diagnosis was primarily based on radiological findings, persistent non-expansion following drainage, and the overall clinical presentation.

### *Learning Points*

Persistent non-expansion of the lung after pleural drainage should raise suspicion for trapped lung [1,2]. In this setting, pneumothorax ex vacuo is a consequence of non-expandable lung physiology and must not be mistaken for a persistent air leak [2,5]. Trapped lung may present with severe hypercapnic respiratory failure accompanied by altered mental status, highlighting its potential clinical severity. Importantly, the presence of a transudative pleural effusion does not exclude trapped lung physiology [1,8]. Failure of lung re-expansion after large-volume pleural drainage remains an important diagnostic clue [2,5]. Moreover, initial improvement with non-invasive ventilation such as BiPAP does not preclude progressive ventilatory failure in advanced trapped lung physiology. Early recognition of trapped lung is crucial, as it may help avoid repeated and ineffective pleural interventions [1,2,5].

### *Ethics Statement*

### *Consent*

Written informed consent for publication of this case report and accompanying images was obtained from the patient's next of kin. A copy of the written consent is available for review by the Editor-in-Chief upon reasonable request.

### *Conflict of Interest*

The authors declare no conflicts of interest.

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### *Data Availability*

All relevant data supporting the findings of this case report are included within the article. Additional information is available from the corresponding author upon reasonable request.

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