Non-expandable lung: an underappreciated cause of post-thoracentesis basilar pneumothorax

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DESCRIPTION

A 67-year-old woman with a history of ischaemic cardiomyopathy and chronic kidney disease presented to the hospital with worsening anasarca and shortness of breath for a few weeks' duration. She was tachypnoeic and hypoxic, requiring oxygen supplementation. Chest radiography revealed significant bilateral pleural effusion and right middle lobe atelectasis (figure 1). She was diagnosed with acute decompensated heart failure and received intravenous furosemide. Despite adequate diuresis, there was no significant improvement of the pleural effusion. The patient continued to be hypoxic and dyspnoeic. An ultrasound (US) revealed a large right-sided pleural effusion without any complexity. Thoracentesis was performed, and 1400 cm³ of straw-coloured fluid was drained. The pleural fluid analysis is shown in table 1. The pleural fluid analysis was consistent with lymphocyte predominant transudative pleural effusion.

The patient had chest discomfort throughout the procedure. After fluid removal, air was aspirated into the negative suction bottle. The postprocedure chest X-ray is shown in figure 2A revealing a basilar pneumothorax. Serial chest X-rays were performed at 4 and 12-hour intervals after procedure and are shown in figure 2B,C, showing reaccumulation of the pleural fluid.

Initially, the patient was diagnosed with iatrogenic pneumothorax; however, the evolution of subsequent chest X-rays is consistent with the diagnosis of pneumothorax ex vacuo and non-expandable lung (NEL).¹

NEL is defined as the inability of the lung to expand to the chest wall allowing for the apposition of the visceral and parietal pleura at the end of pleural drainage.² There are many causes for NEL and can be subdivided into airway, lung and pleural aetiologies.² The pleural aetiologies can be separated into lung entrapment and trapped lung.³ Both lung entrapment and trapped lung are associated with the development of pleural effusion; however, the pathophysiology of these two conditions is different. Lung entrapment occurs in the setting of an active pleuropulmonary process, such as pneumonia, pleurisy or pleural malignancy. The inflammation restricts the lung from expanding. The pleural effusion associated with lung entrapment is exudative in nature. In case of trapped lung, a thick fibrous peel over the visceral pleura prevents pleural expansion and development of NEL. In most cases, trapped lung develops in patients resulting from lung entrapment in the past. As their inflammation resolves, the effusion in

Figure 1 (A) Anteroposterior chest X-ray showing non-free-flowing bilateral pleural effusion with atelectasis of the right middle lobe. (B) Axial CT scan of the chest confirming large right-sided effusion with atelectatic right middle lobe.

trapped lung results in a transudative or protein-discordant exudative effusion. The differential cell count should show a lymphocytic predominance.⁴ The use of pleural manometry can further support the diagnosis of NEL. NEL physiology is associated with low pleural compliance or high pleural elastance. A pleural elastance (change in pleural pressure divided by pleural fluid removed) greater than 14.5 cm of H₂O/L is considered diagnostic of trapped lung.¹ In the absence of pleural manometry, several radiological findings can provide a clue towards a diagnosis of trapped lung. These include pleural thickening, pleural effusion with ipsilateral mediastinal shift and/or volume loss, and development of post-thoracentesis basilar pneumothorax.⁴ In addition, vague mediastinal pain during pleural fluid drainage can also hint at the underlying physiology.⁵ Our patient developed vague chest pain during the procedure and post-thoracentesis pneumothorax that was more conspicuous in the basilar area. Performance of air-contrast CT scans may have helped in the identification of a likely thickened visceral pleura.⁴ The use of bedside US for evaluation of the pleural space has revolutionised modern medicine. US is a non-invasive and relatively inexpensive technique that can reliably identify pleural space pathology and guide clinicians in the appropriate management of malignant and non-malignant pleural diseases.⁶ Additionally, the US might assist

Figure 2 Anteroposterior chest X-ray showing collapsed lung margin in the right lower lung zone (A). Rapid accumulation of right-sided effusion after 4 hours (B) and 12 hours (C) post-thoracentesis.

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Table 1 Pleural fluid analysis

Parameters	Value (normal)
Cell count	
White cell	834×10 ⁹ /L
Neutrophil	38%
Lymphocyte	62%
Eosinophil	0%
Chemistry	
pH	7.3 (6.5–7.5)
LDH	74 U/L
Protein	1.5 g/dL
Albumin	0.9 g/dL
Glucose	116 mg/dL
Serum studies	
LDH	157 U/L
Serum protein	5.4 (6.6–8.7 g/dL)
Serum albumin	3.1 (3.5–5.2 g/dL)

LDH, lactate dehydrogenase.

in prognostication by predicting the possibility of NEL even before performing pleural drainage, especially in patients with malignant pleural effusion.⁷ Researchers have used the motion mode ultrasonography to assess the degree of lung movement due to heartbeats during a breath-hold manoeuvre and found that NEL was associated with reduced lung movement compared with lungs that expanded fully following pleural drainage. They also assessed 'lung deformation' by speckle tracking function of the echocardiography and concluded that NEL was associated with less deformation. Interestingly, the combination of 'lung movement' and 'lung deformation' measurements provided a better sensitivity than pleural manometry. This evolving technique needs further research and maybe extremely valuable for the management of NEL as pleural manometry is often cumbersome.

Drainage of pleural effusion due to trapped lung might result in hydropneumothorax or pneumothorax ex vacuo.¹ Physiologically, pneumothorax can be divided into two types: (A) pressure independent and (B) pressure dependent. Pressure-independent pneumothorax is caused by bronchopleural or alveolopleural fistula that demonstrates a ball-valve action leading to unidirectional flow of air into the pleural space and progressive accumulation of air and development of at least atmospheric or even supra-atmospheric pressure, which can lead to tension pneumothorax and lung collapse.⁸ In pressure-dependent pneumothorax, as the pleural effusion is drained it creates a lung size and thoracic cage mismatch causing localised lung deformation opening alveolopleural fistula. This causes the leakage of air into the pleural space until the deformation is relieved, resulting in a pneumothorax.¹ Development of pneumothorax following thoracentesis in this condition has also been attributed to the suction of atmospheric air along the catheter insertion tract due to an extreme reduction in intrapleural pressure. However, this mechanism is unlikely to play an important role in the development of the pneumothorax. Pneumothorax ex vacuo remains

remarkably stable following removal of the negative suction and is rapidly replaced by fluid as was the case in our patient.

Knowledge of this entity is crucial for clinicians as many of these patients would be unnecessarily managed with chest tube insertions for the pneumothorax. Furthermore, application of negative suction will result in persistent air leak resulting in prolonged hospitalisation or even surgical correction of suspected non-healing bronchopleural fistula which is completely unnecessary.^{1 9 10}

Learning points

- ▶ Post-thoracentesis basilar pneumothorax due to non-expandable lung is not an emergency and does not require an emergent evacuation of the pleural space.
- ▶ Non-expandable is common in clinical practice and should be considered when chest radiology reveals pleural thickening, pleural effusion with ipsilateral mediastinal shift and/or volume loss, and development of post-thoracentesis basilar pneumothorax. Assessment of 'lung movement' and 'lung deformation' due to heartbeats by bedside ultrasound is an evolving technique and might prove to be highly valuable in the prediction of non-expandable lung in the future.
- ▶ Pleural manometry might assist in the diagnosis of non-expandable lung by demonstrating the development of pressure-dependent pneumothorax.

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