Characteristics of Trapped Lung*

Pleural Fluid Analysis, Manometry, and Air-Contrast Chest CT

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Study objectives: To review the pleural fluid characteristics, pleural manometry, and radiographic data of patients who received a diagnosis of trapped lung in our pleural diseases service. Design: Retrospective case series. Methods: The procedure records of 247 consecutive patients who underwent pleural manometry at the Medical University of South Carolina between October 2002 and November 2005 were reviewed. Eleven patients in whom a diagnostic pneumothorax was introduced were identified. Manometry data, radiographic findings, pleural fluid analysis, final clinical diagnosis, and information regarding the initial pleural insult were retrieved from the medical record. Results: All 11 patients had a clinical diagnosis of trapped lung. The causes of trapped lung were attributed to coronary artery bypass graft surgery, uremia, thoracic radiation, pericardiectomy, spontaneous bacterial pleuritis and repeated thoracentesis, and complicated parapneumonic effusion. Mean pleural fluid pH was 7.30, pleural fluid lactate dehydrogenase (LDH) was 124 IU/L, and pleural fluid total protein was 2.9 g/dL. Pleural fluid was paucicellular with mononuclear cell predominance. Pleural space elastance was increased in all cases and ranged from 19 to 149 cm H2O/L of pleural fluid removed. All demonstrated abnormal visceral pleural thickness on air-contrast chest CT. Conclusions: Trapped lung is a clinical entity characterized by the presence of a restrictive visceral pleural peel that was first described in 1967. The pleural fluid is paucicellular, LDH is low, and protein may be in the exudative range. The elevated total pleural fluid protein may be related to factors other than active pleural inflammation or malignancy and does not exclude the diagnosis. (CHEST 2007; 131:206–213)

Key words: lung entrapment; pleural elastance; pleural manometry; trapped

Abbreviations: CAVG = coronary artery bypass graft surgery; CHF = congestive heart failure; LDH = lactate dehydrogenase

The cause of a pleural effusion can usually be determined by the medical history, physical examination, and pleural fluid analysis. However, some pleural effusions defy diagnosis by such means, and even biopsy may not yield a specific diagnosis. In our clinical experience, some patients present with a chronic pleural effusion associated with an inability of the lung to fully expand and fill the thoracic cavity after drainage. Although most cases of unexpandable lung are diagnosed readily and are associated with infection or malignancy, in a small number of patients the cause of the effusion remains unclear and appears to be associated with visceral pleural restric-

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tion in the absence of active pleural disease.\textsuperscript{1–5} Similar cases were first described by Moore and Thomas\textsuperscript{3} in 1967 and referred to as trapped lung. Trapped lung has not been well characterized using current routine pleural fluid analysis, radiographic imaging, and pleural manometry.\textsuperscript{6–7} Therefore, we instituted a clinical protocol in 2001 to identify possible cases of trapped lung early in the diagnostic sequence. The pleural fluid characteristics, pleural manometry, and air-contrasted chest CT of 11 patients who received a diagnosis of trapped lung are reported.

**Materials and Methods**

We performed a retrospective review of 247 consecutive patients referred for therapeutic thoracentesis in our database at the Medical University of South Carolina between October 2002 and November 2005. We identified 11 patients in whom a diagnostic pneumothorax was performed in accordance with the clinical protocol that we established in 2001.

The clinical protocol we use routinely during pleural manometry requires a diagnostic pneumothorax to be performed when all of the following criteria are met: (1) development of excessively negative pleural liquid pressure (defined by a mean pleural liquid pressure $\leq 25$ cm H$_2$O); and (2) prior thoracentesis with complete pleural fluid analysis and a clinical history that excludes pleural malignancy or active pleural space inflammation. In patients in whom diagnostic pneumothorax is performed, the introduction of air allows for safe removal of all pleural fluid and alleviates chest pain when excessively negative pleural pressures are encountered. We entrain enough air to raise the intrapleural pressure to a normal physiologically range (mean pleural pressure of $\approx 5$ cm H$_2$O). Air-contrast chest CT is performed to evaluate the thickness of the visceral pleural surface. A complete pleural fluid analysis is also obtained.

The instrumentation and technique of pleural manometry that was employed was identical to that described by Doelken and colleagues.\textsuperscript{8} With the introduction of air into the pleural space, only the electronic manometric method can be used. This research was approved by the Institution Review Board of the Medical University of South Carolina.

**Results**

Eleven patients with a clinical diagnosis of trapped lung were identified in this series. The causes of trapped lung were attributed to coronary artery bypass graft surgery (CABG) in four patients, uremia in three patients, thoracic radiation in one patient, spontaneous bacterial pleuritis and multiple thoracenteses in one patient, pericardiomyotomy in one patient, and complicated parapneumonic effusion in one patient. Mean pleural fluid pH was 7.37 (range, 7.26 to 7.46). Mean pleural fluid lactate dehydrogenase (LDH) was 124 IU/L (range, 57 to 170 IU/L). Mean pleural fluid total protein was 2.9 g/dL (range, 2.0 to 4.2 g/dL). A pleural fluid total protein value $> 3.0$ g/dL was observed in 5 of 11 patients. Serum protein values were not available. The mean pleural fluid nucleated cell count was 416 cells/$\mu$L (range, 21 to 1,837 cells/$\mu$L). The differential of the nucleated cells showed mononuclear cell predominance in all cases (Table 1). Patient 11 had a decortication; the visceral pleural peel showed a nonspecific fibrous pleuritis. The other 10 patients were not symptomatic from their trapped lung, and the patients were reassured about the benign nature of the condition. They were informed that further diagnostic evaluation was not necessary provided the effusion remained stable.

Five of the 11 patients had an initial negative mean pleural liquid pressure. Pleural space elastance was increased in all patients and ranged from 19 to 149 cm H$_2$O/L of fluid removed. In 8 of the 11 patients, the only cause of the pleural effusion was attributed to a trapped lung. The pressure/volume curves of these eight cases are shown in Figure 1. In three patients, a concomitant diagnosis of congestive heart failure (CHF) was made in addition to trapped lung. The curves of these cases are shown in Figure 2. Dyspnea resolved in all three patients after therapy for CHF was instituted.

Air-contrast chest CT showed abnormal visceral pleural thickness in all 11 patients. Visceral pleural thickness was $< 3$ mm in all 11 patients (Fig 3).

**Discussion**

**Background**

At our pleural disease service, we are frequently asked to evaluate patients with chronic, persistent pleural effusions that have defied diagnosis and have undergone multiple thoracenteses. In our database, there were patients in whom a diagnostic pneumothorax was induced that provided a confident clinical diagnosis of trapped lung. The detection of a trapped lung was a direct consequence of the clinical protocol we introduced in 2001 that was designed for the evaluation of pleural effusion. Protocol development evolved from the clinical necessity and recognition that some patients with a pleural effusion remain without a diagnosis after initial evaluation with complete pleural fluid analysis and radiography. Some of these undiagnosed cases were similar in presentation following thoracentesis and could be characterized by the following: (1) development of an unexpected pneumothorax; (2) inability to fully expand the lung; or (3) inability to completely drain the effusion due to the development of chest pain. Although the failure of the lung to expand following thoracentesis is not infrequent, in most cases, the cause for the pleural effusion, malignancy, or infection dominates the presentation. Our protocol prompts an air-con-
Table 1—Manometry and Pleural Fluid Characteristics in Trapped Lung (n = 11)*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Case 5</th>
<th>Case 6</th>
<th>Case 7</th>
<th>Case 8</th>
<th>Case 9</th>
<th>Case 10</th>
<th>Case 11</th>
<th>Mean (Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosis</td>
<td>Peri</td>
<td>Uremia</td>
<td>CABG</td>
<td>CABG</td>
<td>Uremia and CHF</td>
<td>CABG Multiple thoracentesis and SBPL</td>
<td>Uremia and CHF</td>
<td>CABG and CHF</td>
<td>Rad</td>
<td>CPE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.44</td>
<td>7.42</td>
<td>7.26</td>
<td>7.33</td>
<td>7.38†</td>
<td>7.39</td>
<td>7.40</td>
<td>7.33</td>
<td>7.31</td>
<td>7.37 (7.26–7.46)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total protein, g/dL</td>
<td>4.2</td>
<td>2.2</td>
<td>3.6</td>
<td>3.6</td>
<td>4.1</td>
<td>2.0</td>
<td>2.0</td>
<td>2.0</td>
<td>2.0</td>
<td>3.2</td>
<td>2.9 (2.0–4.2)</td>
<td></td>
</tr>
<tr>
<td>LDH, IU/L</td>
<td>99</td>
<td>114</td>
<td>100</td>
<td>153</td>
<td>170</td>
<td>132</td>
<td>107</td>
<td>155</td>
<td>57</td>
<td>115</td>
<td>124 (99–170)</td>
<td></td>
</tr>
<tr>
<td>Nucleated cells, /μL</td>
<td>214</td>
<td>192</td>
<td>1837</td>
<td>148</td>
<td>315</td>
<td>235</td>
<td>538</td>
<td>21</td>
<td>352</td>
<td>213</td>
<td>415 (21–1,837)</td>
<td></td>
</tr>
<tr>
<td>Differential, %</td>
<td>Mø 64, L 22</td>
<td>L 94</td>
<td>L 96</td>
<td>Mø 42, L 39</td>
<td>L 67</td>
<td>L 63</td>
<td>L 70</td>
<td>Mø 74</td>
<td>L 76</td>
<td>L 69</td>
<td>L 80</td>
<td>Mono 78 (63–96)</td>
</tr>
<tr>
<td>Volume, mL</td>
<td>400</td>
<td>180</td>
<td>310</td>
<td>950</td>
<td>100</td>
<td>650</td>
<td>850</td>
<td>800</td>
<td>1,650</td>
<td>140</td>
<td>660</td>
<td>611 (140–1,680)</td>
</tr>
<tr>
<td>Initial mean pleural liquid pressure, cm H₂O</td>
<td>-2.7</td>
<td>-8.0</td>
<td>-34.0</td>
<td>+6.6</td>
<td>-1.0</td>
<td>+3.0</td>
<td>+4</td>
<td>+2.2</td>
<td>+4.5</td>
<td>-1.2</td>
<td>-2.0</td>
<td>-2.6 (–34–+6.6)</td>
</tr>
<tr>
<td>Terminal mean pleural liquid pressure (cm H₂O)</td>
<td>-12.3</td>
<td>-30.2</td>
<td>-63.2</td>
<td>-11.1</td>
<td>-27.4</td>
<td>-24</td>
<td>-15.3</td>
<td>-30.2</td>
<td>-19.5</td>
<td>-30.4</td>
<td>-40.3</td>
<td>(-11.1–63.2)</td>
</tr>
<tr>
<td>Pleural space elastance, cm H₂O/L</td>
<td>24</td>
<td>149</td>
<td>86</td>
<td>19</td>
<td>45</td>
<td>42</td>
<td>22</td>
<td>65</td>
<td>38</td>
<td>89</td>
<td>35</td>
<td>(19–149)</td>
</tr>
<tr>
<td>Time of diagnosis from pleural injury, mo‡</td>
<td>30</td>
<td>60</td>
<td>23</td>
<td>27</td>
<td>42</td>
<td>12</td>
<td>NA</td>
<td>16</td>
<td>144</td>
<td>20</td>
<td>14</td>
<td>39 (12–144)</td>
</tr>
</tbody>
</table>

*CPE = complicated parapneumonic effusion; L = lymphocyte; Mono = mononuclear cells; Mø = macrophage; NA = not available; Peri = pericardiotomy; Rad = radiation; SBPL = spontaneous bacterial pleuritis.
†Laboratory error on pH measurement.
‡Calculated from the time of surgery, start of radiation therapy, first documented thoracentesis or pleural space infection, and when hemodialysis was first initiated.
contrast CT if excessively negative pressure or chest pain is encountered during thoracentesis while fluid is still present. A diagnostic pneumothorax is only induced if the initial clinical evaluation did not reveal malignancy or active infection as the most likely cause of the pleural effusion.

In order to distinguish noninflammatory visceral pleural restriction leading to an unexpandable lung following thoracentesis from active pleural disease, we adopted the narrow definition of trapped lung of Moore and Thomas, which excluded patients with evidence of active pleural disease. We consider a visceral pleural peel that results in an unexpandable lung immediately following pleural space drainage from an active pleural process a complicating factor and define this condition as lung entrapment. The recognition of trapped lung as a clinical entity has consequences for the management of affected patients. For example, a patient with an asymptomatic trapped lung may be subjected to repeated diagnostic evaluations due to the persistent radiographic finding. Of note, in such cases even thoracoscopic biopsy does not provide a diagnosis because only nonspecific fibrous pleuritis will be found, as in patient 11. Furthermore, patients with an asymptomatic trapped lung are not immune from other pleural diseases, and the management of these patients may be complicated by the preexisting trapped lung. Finally, patients symptomatic from an extensive trapped lung will usually require decortication.

Manometry

The hallmark of trapped lung is an inability of the lung to conform to the shape of the chest wall with the application of negative pressure in the physiologic range. Therefore, pleural manometry is a central modality in the evaluation of trapped lung in our laboratory. The upper limit of the normal range of pleural space elastance has recently been estimated as 14.5 cm H₂O/L, with any value > 15.5 cm H₂O/L not being compatible with overall respiratory system mechanics. Thus, a pleural space elastance > 14.5 cm H₂O/L is highly likely to represent a local mechanical abnormality in the pleural space. All patients reported in our series had a pleural elastance > 14.5 cm H₂O/L. The elastance value of interest is the terminal slope of the pressure volume curve as pleural apposition occurs during drainage of the last remaining fluid.

Pleural manometry is a valuable tool to document abnormal pleural space mechanics but in isolation is not diagnostic of trapped lung. Elevated elastance may also be observed in malignant or inflammatory lung entrapment. Pleural manometry is also used to
establish negative pleural pressure in the physiologic range when a diagnostic pneumothorax for the purpose of air-contrast CT or radiography is induced.

**Air-Contrast CT**

All patients in our series have documented visceral pleural thickening by air-contrast CT. The visceral pleural peel in our patients was thin (<3 mm) and is unlikely to be detected when not outlined by air. The pressure in the pneumothorax was adjusted to slightly negative values (approximately $-5\, \text{cm H}_2\text{O}$ at end-expiration) according to our clinical protocol. In our opinion, the demonstration of the visceral pleural peel, the persistence of pleural air under negative pleural pressure conditions, and abnormal elastance in the absence of other causes of unexpandable lung (such as endobronchial obstruction) establish the presence of a mechanical condition consistent, but not diagnostic, for trapped lung.

**Pleural Fluid Protein**

With the diagnosis of trapped lung requiring the absence of an active pleural inflammatory or malignant process, the pleural fluid may be expected to be a transudate with low protein and LDH levels. However, in five of our patients pleural fluid protein was elevated into the exudative range. This finding may be explained by the following considerations.

The pleural fluid protein concentration in pleural effusions of vascular origin depends on plasma protein concentration, the protein reflection coefficient, solvent filtration into the pleural space (wash down), and bulk flow of fluid via pleural lymphatics. The clinical utility of the distinction between transudates and exudates by protein and LDH criteria is undisputed. The finding of a transudate by these criteria in effusions of vascular origin almost always indicates a normal protein reflection coefficient but also increased filtration and increased lymphatic bulk flow conditions, narrowing the differential diagnosis considerably. Although the pleural fluid of trapped lung is by definition of vascular origin and the absence of inflammation and malignancy implies a normal protein reflection coefficient, solvent filtration may not be increased and lymphatic bulk flow may be impaired. Based on these considerations, we do not interpret isolated increases of pleural fluid protein concentration as unequivocal evidence of an abnormal protein reflection coefficient, solvent filtration may not be increased and lymphatic bulk flow may be impaired. Based on these considerations, we do not interpret isolated increases of pleural fluid protein concentration as unequivocal evidence of an abnormal protein reflection coefficient, solvent filtration may not be increased and lymphatic bulk flow may be impaired. Based on these considerations, we do not interpret isolated increases of pleural fluid protein concentration as unequivocal evidence of an abnormal protein reflection coefficient, solvent filtration may not be increased and lymphatic bulk flow may be impaired. Based on these considerations, we do not interpret isolated increases of pleural fluid protein concentration as unequivocal evidence of an abnormal protein reflection coefficient, solvent filtration may not be increased and lymphatic bulk flow may be impaired. Based on these considerations, we do not interpret isolated increases of pleural fluid protein concentration as unequivocal evidence of an abnormal protein reflection coefficient, solvent filtration may not be increased and lymphatic bulk flow may be impaired. Based on these considerations, we do not interpret isolated increases of pleural fluid protein concentration as unequivocal evidence of an abnormal protein reflection coefficient, solvent filtration may not be increased and lymphatic bulk flow may be impaired. Based on these considerations, we do not interpret isolated increases of pleural fluid protein concentration as unequivocal evidence of an abnormal protein reflection coefficient, solvent filtration may not be increased and lymphatic bulk flow may be impaired.

An alternative explanation for the elevated pleural fluid protein concentration may relate to the timing of the thoracentesis. All trapped lungs begin as a form of lung entrapment, which is defined by the presence of an unexpandable lung with concomitant active pleural inflammation or malignancy. By defi-

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**Figure 3.** Top, A: Prethoracentesis posteroanterior chest radiograph in a woman who received prior thoracic radiation. The chest radiograph shows a right pleural effusion of moderate size without mediastinal shift and volume loss. There is an absence of a normal meniscus sign suggesting loculation. Bottom, B: Air-contrast chest CT shows a right-sided hydropneumothorax and subpleural atelectasis. Abnormal visceral pleural thickening is seen. The normal thickness of the visceral pleura should be minimally visible.
nition, the effusion from lung entrapment is exudative. While most cases of lung entrapment resolve with resolution of the inflammatory process, in others resolution is incomplete resulting in a trapped lung. Therefore, a trapped lung and lung entrapment represent a continuum of the same disease process, and the timing of the thoracentesis is, therefore, critical whether an exudate or transudate is found on pleural fluid analysis (Fig 4, 5).

LDH

In contrast to pleural fluid protein concentration, LDH concentration in the pleural fluid does not follow microvascular exchange dynamics. The finding of elevated LDH in the pleural fluid is an important indicator for the presence of active pleural disease, and the diagnosis of trapped lung should be questioned accordingly. However, given that trapped lung is merely the ultimate outcome of an inflammatory process, and thus represents the end point of a continuum of decreasing inflammation, it should be expected that occasionally mildly elevated LDH concentrations may be found in patients meeting all other criteria for the diagnosis of trapped lung described here. Indeed, in one of our patients we found an LDH value of 170 IU/L, which is in the exudative range (upper limit of normal serum LDH in our laboratory of 238 IU/L resulting in an upper limit of transudate LDH of 159 IU/L). However, in a recent receiver operating characteristic curve analysis, the LDH value was clearly in the transudative range. We interpret slight elevations of LDH in this situation as evidence that the inflammatory insult has not totally resolved; the decision for further investigation or observation has to be made on a case-by-case basis.

Pleural Fluid Cellularity

The pleural fluid is paucicellular with a mononuclear predominance. The mean nucleated cell count was 415 cells/μL. Only in one patient was the nucleated cell count >1,000 cells/μL. Polymorphonuclear cell predominance or pleural fluid eosinophilia are not findings consistent with trapped lung, and their presence suggests an active pleural process. If an elevated mononuclear cell count is found, we apply considerations similar to those in the case of an increased LDH.

Causes and Natural History of Trapped Lung

The diagnosis of trapped lung requires chronicity and stability over time. The diagnosis of trapped lung is often delayed, which may be due to failure to consider the diagnosis or may simply reflect the time required for the resolution of the initial insult. In
our series, the time of diagnosis from initial pleural insult ranged from 12 to 144 months (mean, 39 months).

Conditions reported to result in trapped lung include CABG, post-cardiac injury syndrome, empyema, uremic pleuritis, hemothorax, rheumatoid pleurisy, tuberculous pleurisy and, historically, pneumothorax therapy for pulmonary tuberculosis.1,2,5–10 It is not surprising that cardiac surgery was the most common cause of trapped lung in our series, given that > 600,000 cardiac surgeries are performed per year in the United States.13

Management of Trapped Lung

The majority of patients with underlying trapped lung are asymptomatic or have minimal dyspnea with exertion. In our series, one patient had clinically significant dyspnea related to a trapped lung and was successfully treated with decortication. Three patients had concomitant CHF, and their dyspnea responded to treatment for CHF. It is important to exclude other causes of dyspnea prior to recommending an unnecessary decortication. In our opinion, reexpansion with prolonged tube thoracostomy should only be attempted in symptomatic patients with trapped lung who are poor surgical candidates.

CONCLUSIONS

Trapped lung represents the end stage of dysfunctional healing of pleural injury that begins as a form of lung entrapment that results in the formation of a visceral pleural peel and a persistent pleural effusion. Trapped lung should be included in the differential diagnosis of patients with a remote pleural injury and in whom a chronic, radiographically stable pleural effusion without obvious cause is encountered. A high index of suspicion must be maintained in order to avoid repeated diagnostic procedures that may cause inflammation and make the establishment of the diagnosis more problematic. We recommend the use of pleural manometry and air-contrast CT during the initial evaluation of these patients. If used in conjunction with manometry and a thorough history, the routine use of air-contrast CT in patients meeting the above criteria will not lead to an excessive number of diagnostic pneumothoraces in patients with a diagnosis other than trapped lung. Indeed all 11 patients who underwent diagnostic pneumothorax induction in our series had the diagnosis of trapped lung confirmed.

The absence or paucity of inflammatory parameters on the pleural fluid analysis in patients with the appropriate history, pleural space restriction by manometry, and a visceral pleural peel, in the absence of other causes of an unexpandable lung, allows the clinician to establish the diagnosis of trapped lung. The pleural pressure/volume curve in some patients with trapped lung may reveal initially normal elastance, but the elastance increases when more fluid is withdrawn. In these cases, a secondary cause such as CHF, is contributing to pleural fluid formation. Obviously, the patient with trapped lung is not
protected from other pleural disease, and development of an exudative process in a patient with underlying trapped lung may pose diagnostic and management challenges.

**References**