Ultrasound assessment of lung consolidation and reaeration after pleural effusion drainage in patients with Acute Respiratory Distress Syndrome: a pilot study

B. Chinardet, H. Brisson, C. Arbelot, O. Langeron, J.-J. Rouby and Q. Lu

Abstract: Purpose: The aim of the pilot study was to assess by ultrasound changes in dimensions of lung consolidation and reaeration after drainage of large pleural effusion in patients with acute respiratory distress syndrome (ARDS).

Methods: Lung ultrasound and blood gas were performed before, 2 hours (H2) and 24 hours (H24) after drainage of pleural effusion. Lung ultrasound aeration score was calculated. Cephalocaudal dimension and diaphragmatic transversal area of lung consolidation were measured.

Results: Ten patients were studied. Median volume of drained effusion was 675 ml at H2 and 895 at H24. Two hours after drainage, dimension of cephalocaudal consolidation and diaphragmatic transversal area decreased significantly. Lung reaeration after drainage occurred mainly in latero-inferior and postero-superior regions. PaO2/FiO2 increased significantly at H24.

Conclusions: Ultrasound is a useful method to assess lung consolidation after pleural effusion drainage. Drainage of pleural effusion may lead to a decrease of lung consolidation and improvement of lung reaeration.

Key words: Pleural effusion; lung ultrasound; Acute Respiratory Distress Syndrome; consolidation; aeration.

Pleural effusion is regularly observed in critically ill patients. Experimental and clinical data have provided evidence that pleural effusion induces significantly loss of end-expiratory lung volume with consequent hypoxemia (1-3).

The loss of lung volume resulting from pleural effusion is mainly explained by compression of adjacent lung parenchyma (4). In mechanically ventilated patients, however, other factors can also contribute to lung collapse, such as sedation-induced diaphragm paralysis, heart and abdominal compression of lower lobes (5-7). On the other hand, in patients with acute respiratory distress syndrome (ARDS) or pneumonia, a significant amount of lung parenchyma cannot be compressed by pleural effusion due to the presence of alveolar edema and/or lung inflammation. Therefore, less improvement in oxygenation after pleural effusion drainage has been reported in these patients (2, 8). It is still unknown, however, how the dimension of adjacent pulmonary consolidation changes after drainage of large pleural effusion in mechanically ventilated patients with pneumonia or ARDS.

Lung ultrasound is increasingly used to manage critically ill patients (9). It can predict with accuracy volume of pleural effusion (10-13). The relevance of lung ultrasound in diagnosis of acute respiratory failure has been also demonstrated (14). In addition, a lung ultrasound aeration score (LUS) has been validated to assess at bedside lung reaeration in PEEP-induced lung recruitment, antibiotic-induced pulmonary reaeration in ventilator-associated pneumonia and lung aeration loss during a weaning trial (15-18). Thus, lung reaeration after pleural effusion drainage can be adequately evaluated at the bedside. The consolidation of lower lobes can be easily visualized by lung ultrasound (19, 20); its transversal area and cephalocaudal dimension can be measured.

The aim of the pilot study was to assess the potential of lung ultrasound to estimate the changes in dimensions of pulmonary consolidation and lung reaeration after drainage of large pleural effusion in mechanically ventilation patients with ARDS.

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Lung ultrasound aeration score

Twelve lung areas were examined in right and left lung: antero-superior, antero-inferior, latero-superior, latero-inferior, postero-superior and postero-inferior. A number is attributed to each area according to the most severe lung ultrasound pattern detected in corresponding intercostal spaces: 0 = normal aeration (N): presence of lung sliding with A lines or fewer than two isolated B lines; 1 = moderate loss of lung aeration: multiple well-defined B lines (B1 lines) issued either from the pleural line or from small juxtapleural consolidations; 2 = severe loss of lung aeration: multiple coalescent B lines (B2 lines) issued either from the pleural line or from small juxtapleural consolidations; and 3 = lung consolidation (C), presence of a tissue pattern characterized by static or dynamic air bronchograms. LUS was calculated as the sum of points (17, 23, 24).

Dimension of lung consolidation

The probe was positioned on the posterior axillary line just above the diaphragm and a transversal view allowed measuring lung consolidation area (Fig. 1). Then the cephalocaudal dimension of lung consolidation was measured by slipping the probe to each adjacent intercostal space from the base to the top of the lung. The lower and upper limits of intercostal spaces where lung consolidation was visualized were drawn on the patient’s skin and the distance between markings was measured.

Pleural effusion drainage

A pleural catheter (Cook Medical, Bloomington USA or Smiths Medical International, UK) was inserted at the posterior axillary line level after ultrasound tracking. A negative pressure of -20 cmH₂O was applied for active suctioning. The volume of drained pleural effusion was quantified at H₂, then every 3 hours by the nurse, and at H₂₄.

Statistical analysis

All values are expressed as median and 25%-75% interquartile range. Comparisons of lung ultrasound measurements or respiratory parameters at H₀, H₂ and H₂₄ after pleural effusion drainage were made by Friedman Repeated Measures Analysis of Variance on Ranks followed by a Turkey test for pairwise comparisons. The volume of drainage between H₂ and H₂₄ after drainage of pleural effusion was compared by Wilcoxon test. The statistical significance level was fixed at 0.05.

Materials and Methods

This is a prospective, observational and pilot study conducted during a period of 10 months in two multidisciplinary intensive care unit of a university hospital. The study was approved by French ethics committee (le Comité de Protection des Personnes, Île de France – VI), who considered the study as an integral part of care provided to the patients and waived need for patients to give written informed consent.

Inclusion criteria were: 1) mechanical ventilated patients with PaO₂/FiO₂ < 300 due to ARDS according to the Berlin definition (21); 2) age ≥ 18 years and 3) presence of large pleural effusion > 500 ml as assessed by lung ultrasonography with indication for drainage. Exclusion criteria were: 1) Traumatic hemothorax; 2) loculated pleural effusion; and 3) severe coagulation troubles with high bleeding risk.

Study protocol

In each patient, lung ultrasound was performed before (H₀), 2 hours (H₂) and 24 hours (H₂₄) after drainage of pleural effusion. A Siemens Acuson CV70 and a 2 to 4-MHz probe were used. The volume of pleural effusion was measured using the technique described by Remerand et al. (11). LUS (17, 22) was calculated and dimension of cephalocaudal lung consolidation and diaphragmatic transversal area of lung consolidation were measured at H₀, H₂ and H₂₄ respectively. Blood gases were sampled and respiratory parameters including clinical signs and ventilator settings were recorded at H₀, H₂ and H₂₄.

Lung ultrasound assessment

Measurement of pleural effusion volume

The volume of pleural effusion was quantified by ultrasound. The patient was lying in supine position. The pleural effusion volume was explored on transversal views by positioning the probe in each paravertebral intercostal space. The lower and upper limits of intercostal spaces where pleural effusion was detected were drawn on the patient’s skin and the distance between these two points was measured. The volume of pleural effusion was calculated by multiplying the paravertebral pleural effusion height by its transversal area measured at half the distance between the apical and caudal limits (11).
measured. As shown in table 2 and figure 2, following drainage of pleural effusion, the transversal area and cephalocaudal dimension of lung consolidation decreased significantly at H2. These changes were associated with a significant lung reaeration as attested by the significant decrease in LUS. LUS and cephalocaudal dimension of lung consolidation remained significantly low at H24 compared to H0. The transversal area of lung consolidation, however, was not significantly different between H24 and H0. Lung reaeration occurred mainly in latero-inferior and postero-superior lung regions (Fig. 3).

Pleural effusion drainage resulted in a significant increase in arterial oxygenation at H24. Other respiratory parameters were not modified at H2 and H24 after drainage of pleural effusion (Table 3).

**Discussion**

This pilot study shows that lung ultrasound allows measuring changes in dimensions of lung consolidation after large pleural effusion drainage. In mechanically ventilated patients with ARDS, drainage of large pleural effusion induces a significant decrease in dimensions of lung consolidation that is associated with improvement of lung aeration and oxygenation. The diaphragmatic transversal area is reduced immediately after drainage but returns almost to its baseline level after 24 hours.

Drainage of pleural effusion is frequently performed in critically ill patients when it is large enough to impair lung mechanics and oxygenation, aimed at releasing its compression on adjacent lung
immobile and the transpulmonary pressure does not increase during inspiration resulting in passive atelectasis of lower lobes (27-29). Therefore, it is not surprising that some studies reported that, in patients with ARDS, pleural effusion led to a greater chest wall expansion than lung volume reduction (30) and that change in lung volume after pleural effusion drainage was weakly correlated with oxygenation improvement (2). To our knowledge, the consequences of large pleural effusion drainage on lung consolidation dimension have never been reported in patients with ARDS or pneumonia (31).

In the present study, among 10 patients with ARDS, 6 had pneumonia. Our preliminary results showed that drainage of large pleural effusion induced an immediate decrease in cephalocaudal dimension and diaphragmatic transversal area of lung consolidation associated with a concomitant improvement of lung aeration. These results indicate that, in patients with ARDS and pneumonia, a significant part of the lung parenchyma that is compressed by pleural effusion can be reaerated by lung reaeration as attested by the decrease in LUS. In a previously published study, a significant correlation between improvement in PaO₂/FiO₂ ratio from baseline to H24 and increase in end-expiratory lung volume during the same time frame has been demonstrated (2).

In mechanically ventilated patients with ARDS or pneumonia, a part of adjacent lobe is not able to be compressed by pleural effusion due to the presence of edema and/or inflammation within the alveolar space. In addition, during sedation and paralysis, the diaphragm does not contract anymore and moves passively with cyclic increases in intrathoracic pressure. In the supine position, nondependent parts of the diaphragm move in the caudal direction increasing regional lung volume (26). In posterior lung regions, the diaphragm remains immobile and the transpulmonary pressure does not increase during inspiration resulting in passive atelectasis of lower lobes (27-29). Therefore, it is not surprising that some studies reported that, in patients with ARDS, pleural effusion led to a greater chest wall expansion than lung volume reduction (30) and that change in lung volume after pleural effusion drainage was weakly correlated with oxygenation improvement (2). To our knowledge, the consequences of large pleural effusion drainage on lung consolidation dimension have never been reported in patients with ARDS or pneumonia (31).

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**Table 2**

<table>
<thead>
<tr>
<th></th>
<th>H0</th>
<th>H2</th>
<th>H24</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume of drainage</td>
<td>675 (505-825)</td>
<td>895 (645-1255)</td>
<td>&lt; 0.001</td>
<td></td>
</tr>
<tr>
<td>Total LUS</td>
<td>15 (13-17)</td>
<td>10 (9-12)*</td>
<td>8 (5-10) #</td>
<td>0.001</td>
</tr>
<tr>
<td>Diaphragmatic transversal area</td>
<td>29 (25-50)</td>
<td>16 (0-25) *</td>
<td>23 (8-40)</td>
<td>0.028</td>
</tr>
<tr>
<td>Cephalocaudal extension (cm)</td>
<td>9 (7-10)</td>
<td>2 (0-5) *</td>
<td>1 (1-1.5) #</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Definition of abbreviation : H0 = before drainage of pleural effusion ; H2 = 2 hours after drainage of pleural effusion ; H24 = 24 hours after drainage of pleural effusion. LUS = lung ultrasound aeration score. Data are expressed as median (25-75% interquartile). * p < 0.05  H2 vs. H0, # p < 0.05 H24 vs. H0.
pleural effusion drainage. The present study shows an unexpected result: 24 hours following pleural drainage, the diaphragmatic transversal cross-sectional area of lung consolidation returned to pre-drainage values, although its cephalocaudal dimension remained significantly reduced. The likely hypothesis to explain this paradoxical result is a caudal shift of the nonaerated part of the lower lobe resulting from additional pleural drainage occurring between H2 and H 24. As demonstrated previously (11), the location of the maximum transversal dimension of pleural effusion varies from patient to patient: at the lung base in some patients and at mid-thorax in some others. The maximum transversal dimension of lung consolidation varies in the opposite direction. Therefore it is possible that with additional effusion removal and despite significant re-aeration, there is a caudal shift of nonaerated part of the lower lobe, explaining the lack of decrease in its diaphragmatic transversal area at H24. It should be pointed out that this finding is obtained from a pilot study in 10 patients. Further study including more patients is needed to confirm this intriguing result, which could have practical consequence: the reduction of lung consolidation volume after pleural drainage is more easily evidenced by assessing its cephalocaudal dimension than its diaphragmatic transversal cross-sectional area. In the present study, the dimension of chest wall was not measured before and after drainage.

Chiurillo et al. reported that in patients with ARDS, moderate pleural effusion led to a greater chest wall expansion (30).

We found that lung reaeration following pleural effusion drainage occurred predominantly in latero-inferior and postero-superior parts of lung, whereas lung consolidation persisted in postero-inferior lung regions (Fig. 3). In mechanically ventilated patients, tidal volume distributes differently: the posterior pleural effusion drainage. The present study shows an unexpected result: 24 hours following pleural drainage, the diaphragmatic transversal cross-sectional area of lung consolidation returned to pre-drainage values, although its cephalocaudal dimension remained significantly reduced. The likely hypothesis to explain this paradoxical result is a caudal shift of the nonaerated part of the lower lobe resulting from additional pleural drainage occurring between H2 and H 24. As demonstrated previously (11), the location of the maximum transversal dimension of pleural effusion varies from patient to patient: at the lung base in some patients and at mid-thorax in some others. The maximum transversal dimension of lung consolidation varies in the opposite direction. Therefore it is possible that with additional effusion removal and despite significant re-aeration, there is a caudal shift of nonaerated part of the lower lobe, explaining the lack of decrease in its diaphragmatic transversal area at H24. It should be pointed out that this finding is obtained from a pilot study in 10 patients. Further study including more patients is needed to confirm this intriguing result, which could have practical consequence: the reduction of lung consolidation volume after pleural drainage is more easily evidenced by assessing its cephalocaudal dimension than its diaphragmatic transversal cross-sectional area. In the present study, the dimension of chest wall was not measured before and after drainage.

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muscular sections of the diaphragm move more than anterior tendon plate and the dorsal regions are normally ventilated. As a result, the drainage of pleural effusion compressing posterior part of the lung may increase ventilation efficiency. Further study is required to assess dimension of cephalocaudal and diaphragmatic transversal area of lung consolidation in spontaneously breathing patients with large pleural effusion.

Quantitative assessment of lung consolidation area has been reported in patients with community-acquired pneumonia (20, 32). These authors showed a significant decrease in area of pneumonia lesion following antibiotic therapy. Compared with the biplane chest radiography, however, pneumonic lesion appeared smaller when it was measured by ultrasound. Moreover, differences in cephalocaudal and ventrodorsal extensions between the 2 techniques were observed (20). It is well known that measurement by lung ultrasound is operator dependent. Many factors can influence the measurement of lung consolidation, such as position of probe on the thorax, location of the consolidation and echogenicity. In the present study, intra and interobserver agreements were not assessed and the ultrasound technique used for measurement was not compared with a reference method. Furthermore, the limited number of patients and the fact that the investigators could not be blinded to the study conditions due to the presence of chest tube after drainage may introduce additional bias into interpretation of the results.

In conclusion, ultrasound is a useful method to assess lung consolidation after pleural effusion drainage. Drainage of large pleural effusion may lead to a decrease of adjacent lung consolidation dimension, improvement of lung aeration and oxygenation in mechanically ventilated patients with ARDS. Using bedside lung ultrasound, this beneficial effect may be better assessed by measuring cephalocaudal dimension of lung consolidation than its diaphragmatic transversal cross-sectional area. Further studies including more patients are required to confirm these results and to assess whether such benefit is obtained in spontaneously breathing patients without ARDS.

References


