Bedside Lung Ultrasound in Critical Care Units

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Abstract

The accurate etiological diagnosis of lung opacities on a bedside chest radiograph in critically ill patients is a real challenge. Acute respiratory failure patients is one of the most distressing conditions that are not ideal for immediate diagnosis, which sometimes compromises the outcome. The aim of this study was evaluation of chest ultrasound as a bedside method to assess lung morphology in hypoxemic patients in critical care practice. This study was conducted in 34 beds in ICU. The study included 130 dyspnic patients with a definite diagnosis. The chest ultrasonography results were compared with the final diagnoses. Those 130 patients represented 34 patients with cardiogenic pulmonary edema, 22 pneumonia patients, COPD were 32 cases, bronchial asthma in exacerbation were 26 patients, 9 cases of pulmonary embolism, and 7 patients diagnosed as pneumothorax. The ultrasonography findings were grouped under profiles A, A', B, B', A/B+, C, and PLAPS to avoid repetitive descriptions. 5 Mhz microconvex probe was used. The results revealed B profile in pulmonary edema with 97% sensitivity and 95% specificity. Normal profile and A' profile without PLAPS in COPD and asthma with sensitivity of 93% and specificity of 97%. Ultrasound in pulmonary embolism when accompanied by deep vein thrombosis showing predominant A lines have 89% sensitivity and 99% specificity. The presence of lung point and absent lung sliding represented 100% sensitivity and specificity in pneumothorax cases. Pneumonia yields numerous signs B', AB, and C profiles that highlighted distinctions between pneumonia and pulmonary edema. Ultrasound in pneumonia cases has sensitivity of 88% and specificity of 94%. The conclusion was that lung ultrasound saves time and decreases the need for CT whose drawbacks include delayed care implementation, irradiation, cost, and required supine position. Lung ultrasound may appear complex at first but simply requires a change in thinking. Once the process has been learned, a step-by-step use will make it routine.

Key Words: Lung – Ultrasound – Critical- Care units – Bedside – Clinical

Introduction

MANAGEMENT of critically ill patients requires imaging techniques, which are essential for optimizing diagnostic and therapeutic procedures [1]. The accurate causal diagnosis of lung opacities on a bedside chest radiograph in patients who are critically ill is a frequent challenge [2]. Acute respiratory failure is one of the most distressing situations for the patient. Emergency cases do not always present in conditions that are ideal for immediate diagnosis, which sometimes compromises outcome [3]. Physical examination and bedside radiography are imperfect, resulting in a need for sophisticated test results that delay management [4]. To date, chest imaging has relied on bedside chest radiography and lung computed tomography (CT). These are representing beside the bedside echocardiography the imaging techniques for the diagnosis and drainage of localized pneumothorax, pleural effusion, and empyema. The assessment of lung over-inflation and the evaluation of areation loss and its distribution require direct visualization of the lungs [1]. The diagnosis and monitoring of pulmonary congestion in acute decompensated heart failure (ADHF) was another challenge inspite of the role of echocardiography as a bedside diagnostic tool. General and cardiac ultrasound can be easily performed at the bedside by physicians working in the intensive care units (ICU) and may provide accurate information with diagnostic and therapeutic relevance. Furthermore, ultrasound is relatively inexpensive and does not utilize ionizing radiation [5,6].

Recently, chest ultrasound has become an attractive new tool for assessing lung status in critically ill patients. There is a growing trend to be performed by physicians practicing in chest, inten-
sive care, and emergency medicine. The aim of this study was to evaluate that chest ultrasound can be used easily at the bedside to assess lung morphology in hypoxemic patients and can be easily repeated to monitor progress and effect to therapy in critical care practice.

Patients and Methods

This study was conducted in Mouwasat General Hospital, Dammam, Eastern Region, Saudi Arabia. The ICU has 34 beds capacity including eight beds for coronary care units. This study started on April 2009 and was planed to continue for two years. Over the last 11 months till February 2010 included in the study 130 dyspneic patients with a definite diagnosis. We performed lung ultrasonography on patients admitted to the ICU with acute respiratory failure. The ultrasonography results on initial presentation were compared with the final diagnosis by the ICU team using standardized tests and not including lung ultrasound data. We excluded uncertain and rare diagnoses and patients who never received a definite diagnosis or those who have several official diagnoses.

Acute respiratory failure was defined based on the classical clinical and biological criteria for requiring ICU admission. All patients had lung ultrasound test by investigators who did not participate in the patient management. Those patients were treated by ICU member blinded to ultrasound results. The ultrasound was performed within twenty minutes of ICU admission without interrupting management and the procedure lasted less than 3 minutes. The study team and the hospital board approved this study and waived the requirement for informed consent.

The sings observed in each disease were methodically collected in each patient; then the ultrasound data were compared with the diagnosis established by the ICU team.

Ultrasound approach:

Ultrasound was performed with a 5-MHZ microconvex probe (Hitachi-405; Hitachi Medical; Tokyo, Japan). Patients were investigated in a semirecumbent position, or were supine if intubated (11 cases).

The normal lung displays lung sliding; that is a movement in rhythm with respiration at the pleural line. It indicates sliding of the visceral pleura against the parietal pleura [7].

A lines are repetitive horizontal artifacts arising from the pleural line generated by subpleural air, which either intra-alveolar or pure (Pneumothorax) blocks the ultrasound waves. Normal interlobular speta are not detected. The ultrasound investigators concentrate to assess three signs:

Artifact analysis:

A or B lines:

The B line is an artifact with seven features: A hydroaemic comet-tail artifact; arising from the pleural line; hyperechoic; well defined; spreading up indefinitely; erasing A lines; and moving with lung sliding when lung sliding is present. It reflects the coexistence of elements such as fluid and air (major acoustic impedance gradient elements). Septal oedema fulfills this condition as it is fluid at the subpleural interlobular septum surrounded by air-filled alveoli.

B+ lines indicates the subpleural part of interstitial syndrome. B+ are three or more B lines in a single view.

The A lines describe the horizontal lines arising from the pleural line are separated by regular intervals that are equal to the distance between the skin and the pleural line. A lines are usually large but can be shorter which has no clinical significance.

Lung sliding:

Present or Abolished: Abolition occurs when the visceral pleura does not slide against parietal pleura (e.g. inflammatory adherences, loss of lung expansion, atelectasis, apnea, chronic symphysis) or is separated as in pneumothorax and pneumonec- tomy. If abolished lung sliding is associated with A lines, the search for pneumothorax is mandatory. The sign of alternating lung sliding and abolished lung sliding plus A lines at the same location is a specific sign of pneumothorax. It is known as the lung point [8].

Alveolar consolidation and/or pleural effusion (absent or present):

Alveolar consolidation results in fluid filled alveoli-The alveolar-interstitial interfaces generate reflections yielding a tissue pattern, absence of the lung line, absence of the sinusoid sign. These signs confirm alveolar consolidation with ultrasound sensitivity of 90%, and specificity of 98% [10]. The lung line represents the visceral pleura. It is the roughly rectangular shape with a regular lower border. The sinusoid sign is the inspiratory
shift of the lung line toward the pleural line. Pleural effusion yields an anechoic-dependent pattern. The sensitivity of these signs is 92% and specificity is 97% [9].

N.B. Deep venous thrombosis was sought using the same probe [11]. Visualization of anatomic echoic intraluminal thrombosis or absence of compressibility was considered as positive finding. An examination combined an anterior approach (analyzing artifacts, lung sliding, and alveolar consolidation), a lateral subposterior search for posterolateral alveolar and/or pleural syndrome (PLAPS) and venous analysis.

Table (1): Study plan.

<table>
<thead>
<tr>
<th>Patients diagnosis</th>
<th>Methods of diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• For all patients</td>
<td>History and physical examination, Radiography (read by radiologist), clinical progression under treatment and:</td>
</tr>
<tr>
<td>• Cardiogenic pulmonary oedema (referred as pulmonary oedema) (n=34)</td>
<td>Evaluation of cardiac function using echocardiography and American Heart Association recommendations.</td>
</tr>
<tr>
<td>• Pneumonia (n=22)</td>
<td>Chronic respiratory disease and beginning ARDS. Infectious profile, microorganisms isolation, recovery with antibiotics and radiological asymmetry. These groups included community acquired pneumonia of hospital acquired pneumonia, aspirationpneumonia, pneumonia complicating.</td>
</tr>
<tr>
<td>• Chronic obstructive pulmonary disease (COPD) (n=32)</td>
<td>This condition considered as exacerbation of chronic obstructive pulmonary disease without pneumonia, pulmonary embolism, pulmonaryoedema, or pneumothorax. COPD was confirmed by functional tests (from medical record). COPD patients with pneumonia were counted under pneumonia.</td>
</tr>
<tr>
<td>• Bronchial asthma (n=26)</td>
<td>History, responds to bronchodilator treatments.</td>
</tr>
<tr>
<td>• Pulmonary embolism (n=9)</td>
<td>Helical CT.</td>
</tr>
<tr>
<td>• Pneumothorax (n=7)</td>
<td>Radiography (CT if necessary).</td>
</tr>
</tbody>
</table>

• n = Number of patients.
• ARDS = Adult Respiratory Distress Syndrome.
• CT = Computed tomography.

Results

This study included 130 patients with a definite diagnosis: A 72 men and 58 women (mean age was 66 years; range 24 to 90 years; SD = 14 years).

Table (2) shows the signs observed and the collected data.

Ultrasound accuracy:

We retained ultrasound characteristic combinations of signs that produced specificities >90%. We adopted the practical nomenclature that avoids repetitive descriptions. These descriptions were grouped under the coming profiles (Table 3).
Table (2): Diagnosis and signs observed in the studies cases.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Signs observed</th>
</tr>
</thead>
</table>
| Pulmonary oedema   | • Pulmonary oedema was observed in 34 patients. Anterior-predominate bilateral B+ lines were observed in 33 cases:  
|                    |   - Anterior-predominate A lines were seen only in one case.  
|                    |   - Anterior lung sliding was preserved in all 34 cases.  
|                    |   - PLAPS was detectable in 32 cases.  
| Pneumonia          | • Pneumonia cases were 22 patients:  
|                    |   - PLAPS was present in 21 patients.  
|                    |   - Anterior predominant B+ pattern was associated with lung sliding in 3 cases, and lung sliding was abolished in 4 cases.  
|                    |   - Anterior-predominate B+ lines in one lung coexisted with predominant A lines in contralateral lung in 4 cases.  
|                    |   - Anterior predominant A pattern with lung sliding with PLAPS was in 13 cases.  
|                    |   - Lung sliding was abolished in 9 cases.  
| COPD               | • This group included 32 patients:  
|                    |   - Anterior-predominant bilateral A lines with lung sliding and no PLAPS was observed in 30 cases.  
|                    |   - Abolished lung sliding was present in one patient.  
|                    |   - PLAPS was seen in two cases.  
| Bronchial Asthma (Status asthmaticus) | • This was observed in 26 patients:  
|                    |   - Anterior-predominant A lines with lung sliding in all cases.  
|                    |   - Calf thrombosis in one patient.  
| Pulmonary embolism | • P.E was observed in 9 patients:  
|                    |   - Anterior predominant A lines with lung sliding in all cases.  
|                    |   - PLAPS was found in 5 cases.  
|                    |   - Eight patients had venous thrombosis.  
| Pneumothorax       | • Pneumothorax was observed in 7 cases:  
|                    |   - Anterior predominant A lines with abolished anterior lung sliding in all cases.  
|                    |   - PLAPS was found in 2 cases.  

Table (3): Ultrasound profiles (adopted in this study).

<table>
<thead>
<tr>
<th>The profile</th>
<th>Characteristic Combinations</th>
<th>Diagnosis relevant to profile</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Profile</td>
<td>• Predominant A lines plus lung sliding at the anterior surface (on semi-setting patients).</td>
<td>• This suggest COPD embolism some posterior pneumonia.</td>
</tr>
<tr>
<td>A’ Profile</td>
<td>• Profile with abolished lung sliding and without lung point.</td>
<td>• This rule out pulmonary edema.</td>
</tr>
<tr>
<td>B Profile</td>
<td>• Predominant B+ lines associated with lung sliding.</td>
<td>• This suggests cardiogenic pulmonary edema.</td>
</tr>
<tr>
<td>B’ Profile</td>
<td>• It is a B profile with abolished lung sliding.</td>
<td>• It nearly rules out COPD out pulmonary embolism and pneumothorax.</td>
</tr>
<tr>
<td>A/B+ Profile</td>
<td>• Massive B lines at the left lung and A lines at the right lungs.</td>
<td>• This is usually associated with pneumonia.</td>
</tr>
<tr>
<td>C Profile</td>
<td>• Designates anterior alveolar consolidation.</td>
<td>• This is usually associated with pneumonia.</td>
</tr>
<tr>
<td>PLAPS</td>
<td>• Profile described in conjunction with other profiles.</td>
<td></td>
</tr>
</tbody>
</table>

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Table (4): Combined results of ultrasound profiles for the cases, included in this study.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>A profile + PLAPS</th>
<th>Normal Profile &amp; A profile without PLAPS</th>
<th>B profile</th>
<th>B' profile</th>
<th>C profile</th>
<th>A/B profile</th>
<th>Lung point</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic pulmonary oedema (34)</td>
<td>1</td>
<td>0</td>
<td>33</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pneumonia (17)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>COPD</td>
<td>1</td>
<td>31</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Status asthmatics</td>
<td>1</td>
<td>24</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>5</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
</tbody>
</table>

Table (5): Accuracy of ultrasound profiles (relevant to each diagnosis).

<table>
<thead>
<tr>
<th>Disease</th>
<th>Ultrasound signs &amp; Profile</th>
<th>Sensitivity %</th>
<th>Specificity %</th>
<th>Positive predictive value %</th>
<th>Negative predictive value %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic pulmonary oedema (34)</td>
<td>B Profile</td>
<td>97</td>
<td>95</td>
<td>88</td>
<td>99</td>
</tr>
<tr>
<td>COPD &amp; asthma (32+26) (58)</td>
<td>Normal profile and A' profile without PLAPS</td>
<td>93</td>
<td>97</td>
<td>93</td>
<td>98</td>
</tr>
<tr>
<td>Pulmonary embolism (9)</td>
<td>Predominant bilateral A lines + deep vein thrombosis</td>
<td>89</td>
<td>99</td>
<td>100</td>
<td>99</td>
</tr>
<tr>
<td>Pneumothorax (7)</td>
<td>Lung point, Absent anterior A lines &amp; absent lung sliding</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>99</td>
</tr>
<tr>
<td></td>
<td>B' Profile</td>
<td>10.5</td>
<td>100</td>
<td>100</td>
<td>70</td>
</tr>
<tr>
<td></td>
<td>A/B Profile</td>
<td>14.5%</td>
<td>100</td>
<td>100</td>
<td>73</td>
</tr>
<tr>
<td>Pneumonia (22)</td>
<td>C Profile</td>
<td>16.5%</td>
<td>99</td>
<td>90</td>
<td>75.5</td>
</tr>
<tr>
<td></td>
<td>Anterior alveolar consolidation</td>
<td>40</td>
<td>96</td>
<td>83</td>
<td>77</td>
</tr>
<tr>
<td></td>
<td>A Profile + PLAPS</td>
<td>40</td>
<td>96</td>
<td>83</td>
<td>77</td>
</tr>
<tr>
<td></td>
<td>A/B or C Profile</td>
<td>88</td>
<td>94</td>
<td>88</td>
<td>95</td>
</tr>
</tbody>
</table>

Discussion

Our results confirmed that, the B profile (anterior interstitial syndrome with lung sliding) indicated pulmonary edema. The B' profile (lung sliding abolished) indicated pneumonia. The A/B profile (asymmetric anterior interstitial syndrome) and the C profile (anterior consolidation) indicated pneumonia, as did the A profile plus PLAPS. The A profile plus venous thrombosis indicated pulmonary embolism. COPD/asthma revealed A normal profile. These results correspond to physiopathologic patterns, particularly echoed by ultrasound artifacts, that have been in clinical use since 1994 [12]. The pleural line is superficial: Most acute disorders reach it; acute interstitial changes involve deep as well as subpleural areas; most (98.5%) cases of acute alveolar consolidation abut the pleura [10]. Pneumothorax and pleural effusion are always about the wall [11]. The high acoustic impedance gradient between air and fluids generates artifacts. Air stops ultrasounds, and fluid facilitates their transmission. The air-fluid ratio is 1 in pneumothorax; roughly 0.98 in asthma, COPD and normal lungs [13]; roughly 0.95 in interstitial syndrome [14]; near zero in alveolar consolidation; and zero
in pleural effusion [11]. In pulmonary oedema, the transuded under pressure is pushed along interlobular septa against gravity, up to the anterior wall, explaining the quasiconstant anterior, symmetric interstitial patterns (indicating anterior Kerly Lines). Oedema of the interlobular septa is constant and early [15]. The B profile (with or without PLAPS due to gravitational filling of dependent alveoli) characterizes pulmonary oedema with high accuracy. Pulmonary oedema produces transudate which is not supposed to generate inflammatory adherences. The adhesion is a factor that may hinder lung sliding. Volpicalli et al., [16] concluded that lung ultrasound is a reliable tool to assess and monitor pulmonary congestion. In acute decompensated heart failure, increased lung arterial pressure causes fluid extravasation into the pulmonary interstitium and alveoli. Not only can this be heard at auscultation, and seen at CXR but can also be observed at lung ultrasound as vertical B lines or comet tail artifacts, a fairly new finding. Searching for such sonographic artifacts is a simple approach that can be proposed as a bedside method to monitor clinical effects of medical therapy in acute decompensated heart failure. It has many advantages and could replace other traditional tools.

COPD and asthma are bronchial diseases assumed to yield a normal lung surface. This explains the ability of ultrasound to distinguish these entities from pulmonary oedema. Volicalli et al. [17] focused on differential diagnosis of pulmonary edema and exacerbation of COPD. This is possible using simple unit and easy-to-acquire technique performed by radiologists and clinicians. Major advantages include bedside availability, absence of radiation, high feasibility and reproducibility, and cost efficiency. The presence of diffuse alveolar interstitial syndrome, which is often a sign of acute pulmonary edema rules out exacerbation of COPD as the main cause of an acute dyspnea.

Pneumonia yields numerous signs. The frequent abolition of lung sliding (B profile) is explained by inflammatory adherences due to exudate. Pneumonia can be found in a wide variety of locations, which explains the asymmetric patterns (AB profile), anterior consolidation (C Profile), or lack of anterior interstitial patterns (A profiles) Briefly, ultrasound highlighted distinctions between pneumonia and pulmonary edema [18]. Partamento et al. [19] confirmed the feasibility of lung ultrasound (U/S) in an ED (emergency department) setting. This study suggested significant role of lung U/S in the diagnostic workup of pneumonia in ED as it is bedside, reliable, rapid, and noninvasive technique. This conclusion considered even if neither sensitivity nor specificity can be inferred from Partamento et al. [19] study because the real gold standard is CT, which could not be performed in all patients.

Pulmonary embolism does not yield interstitial change. A normal anterior lung surface was usually seen as previously reported. The search for venous thrombosis should be associated with lung analysis. Mohn and colleagues [20] found some disappointing results about the accuracy of lung ultrasound in excluding or confirming pulmonary embolism. They concluded that lung sonography could be useful as a bedside method when combined with other non-invasive tests.

Pneumothorax is ruled out by lung ultrasound if there is lung sliding, and lung point. Although usually considered a hindrance, the ultrasound artifacts generated by the lung can lead to an accurate interpretation. The possibility of immediately obtaining basic information, at the bedside, on this vital organ can make ultrasound a genuine visual stethoscope. Lung ultrasound can reduce the need for CT in diagnosing pneumothorax [21].

Conclusions:

Lung ultrasound generates standardized, reproducible patterns, explaining the high interobserver agreement, feasibility is high. Using lung ultrasound saves time and decreases the need for CT whose drawbacks include delayed care implementation, irradiation, cost and required supine position. Lung ultrasound is nearly equivalent to CT in detecting most disorders, can be repeated as well, and provides additional information. Lung ultrasound immediately provided diagnosis of acute respiratory failure in 90.5% of cases. It can be added to the armamentarium of critical care. The additional value of saving time should provide prompter relief for these severely dyspneic patients.

References


