Milky Lungs: Ultrasonographic Findings in Pediatric Acute Respiratory Distress Syndrome

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Abstract: Diagnosis and following up the dynamics of Pediatric Acute Respiratory Distress Syndrome demand a more feasible, non-invasive and bedside tool, such as lung ultrasound, for monitoring the damaged lungs. We report on a 6-month-old child admitted in our Pediatric Surgical Intensive Care Unit with a clinical presentation of ileus and concomitant community acquired pneumonia. Lung ultrasound (LUS) examinations according to the BLUE Protocol were done several times during the hospital stay. A-lines were seen at admission in the upper segments, but 2–3 B-lines were present in the posterolateral segments bilaterally. Later on, separated and coalescent B-lines were seen. White lung parenchyma or milky lungs with a thickened pleural line were seen, while the worst gas exchange according to the results of Arterial Blood Gases (ABGs) has been detected. According to the findings, as many B-lines will be detected, as the severeness of lung damage and gas exchange impairment. The improvement of the gas exchange with the disappearance of the coalescent B-lines was seen later on, after ventilating the child in a prone position. Bedsides, LUS is a feasible and non-invasive point of care method that could be used for diagnosing Pediatric Acute Respiratory Distress Syndrome (PARDS) but in guiding therapy of the damaged lungs, also. The finding of diffuse, coalescent and homogenous B-lines interpreted as “Milky lungs” is consistent with the diagnosis of PARDS.

Keywords: lung ultrasound; pediatric acute respiratory distress syndrome; point of care ultrasonography; milky lungs


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Introduction

According to the Berlin Definition made by the American–European Consensus Conference, any condition manifested with severe hypoxemia and $\text{PaO}_2/\text{FiO}_2 \leq 300 \text{ mmHg}$ should be considered as Acute Respiratory Distress Syndrome (ARDS) in adults [1]. While the Pediatric Acute Lung Injury Consensus Conference (PAILCC) has considered that the value of Oxygenation Index (OI) and Oxygenation Saturation Index (OSI) should be taken as the baseline criteria for diagnosing pediatric Acute Respiratory Distress Syndrome (PARDS) [2]. Despite the fact that Pediatric Acute Respiratory Distress Syndrome (PARDS) is a rare condition among the pediatric population, it still has a high mortality rate. According to the severity of the condition, patients are unstable to do CT scans for diagnosis and following up the dynamics of PARDS, demanding a more feasible, non-invasive and bedside tool, such as the lung ultrasound, for monitoring the damaged lungs.

Case Report

We report on a 6-month-old male child admitted at University Clinical Center Mother Theresa in Skopje in our Pediatric Surgical Intensive Care Unit with a clinical presentation of Ileus with history of previous surgical interventions made after birth because of anal atresia. The child was admitted with concomitant community acquired pneumonia previously treated in a regional hospital. At admission, we made a chest X-ray that showed bilateral perihilar infiltrations followed by severe leukocytosis ($46.7 \times 10^9/\text{L}$) and a severely elevated CRP of 183 mg/L. The child was tachypnoic and febrile with a temperature of 38.9 ºC. We’ve made an initial lung ultrasound examination according to the BLUE Protocol. Anterior, lateral and posterior upper and lower segments (in total 12 segments), were examined bilaterally and were scored according to the well-known lung ultrasound (LUS) score with 0, 1, 2 or 3 points by segments when A-lines, separated B-lines, coalescent B-lines or consolidation were identified, respectively.

Due to the first examination, at admission, A-lines were seen in the upper segments, but 2–3 B-lines were present in the posterior and lateral segments bilaterally (LUS score 6). The next day, the child was treated surgically and, because of low oxygen saturation after the ending of surgical treatment, was transferred back to the Pediatric Surgical ICU already intubated. Because of respiratory failure, the child was sedated and mechanically ventilated using Volume Controlled Ventilation (VCV).

On the third day of admission, we performed a new lung ultrasound examination where we found 4–5 B-lines in the anterior, lateral and posterior lower segments, while, in the upper segments, 1–2 B-lines were found (LUS score 10) (Figure 1). According to the arterial blood gas analysis, the child was experiencing respiratory failure with pO$_2$ of 57 mmHg and pCO$_2$ of 54 mmHg at FiO$_2$ of 0.5 while SpO$_2$ was 88%. The child was mechanically ventilated with VCV mode, while we used a tidal volume (Vt) of 8 mL/kg, a Positive End Expiratory Pressure (PEEP) of 8 cmH$_2$O and a driving pressure of 16 mBar. The infant had an OI of 9 and an OSI of 5.8. The mechanical ventilation was set up according to the Arterial Blood Gas (ABG) analysis findings (Chart 1, Table 1). Serological blood tests for viruses and atypical microorganisms (Pneumoslide) were made and were negative.
**Figure 1.** Lung ultrasound (LUS) finding of 4–5 separated B-lines (3rd day of admission).

**Table 1.** Summary of laboratory findings, Arterial Blood Gas (ABG) findings, calculated Oxygenation Index (OI) and Oxygenation Saturation Index as well as Lung Ultrasound findings.

<table>
<thead>
<tr>
<th></th>
<th>Admission</th>
<th>3rd Day</th>
<th>7th Day</th>
<th>11th Day</th>
<th>15th Day</th>
<th>20th Day</th>
<th>25th Day</th>
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<tbody>
<tr>
<td>WBC</td>
<td>46.7</td>
<td>31.4</td>
<td>22.7</td>
<td>25.2</td>
<td>18</td>
<td>14.1</td>
<td>12.9</td>
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<tr>
<td>CRP</td>
<td>183</td>
<td>165</td>
<td>102</td>
<td>98</td>
<td>154</td>
<td>83</td>
<td>56</td>
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<tr>
<td>PaO₂</td>
<td>57 mmHg</td>
<td>50 mmHg</td>
<td>27 mmHg</td>
<td>37 mmHg</td>
<td>75 mmHg</td>
<td>79 mmHg</td>
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<tr>
<td>PaCO₂</td>
<td>54 mmHg</td>
<td>87 mmHg</td>
<td>109 mmHg</td>
<td>98 mmHg</td>
<td>62 mmHg</td>
<td>56 mmHg</td>
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<tr>
<td>SpO₂</td>
<td>94%</td>
<td>88%</td>
<td>83%</td>
<td>50%</td>
<td>67%</td>
<td>92%</td>
<td>94%</td>
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<tr>
<td>PEEP</td>
<td>8</td>
<td>10</td>
<td>12</td>
<td>13</td>
<td>8</td>
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<td>PIP</td>
<td>22</td>
<td>26</td>
<td>29</td>
<td>26</td>
<td>21</td>
<td>21</td>
<td>18</td>
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<tr>
<td>Driving pressure</td>
<td>16</td>
<td>16</td>
<td>17</td>
<td>13</td>
<td>13</td>
<td>13</td>
<td>11</td>
</tr>
<tr>
<td>MpaW</td>
<td>10.3</td>
<td>13.7</td>
<td>16.7</td>
<td>17</td>
<td>10.8</td>
<td>9</td>
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<tr>
<td>VT</td>
<td>8 mL/kg</td>
<td>7 mL/kg</td>
<td>6 mL/kg</td>
<td>6 mL/kg</td>
<td>7 mL/kg</td>
<td>7 mL/kg</td>
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<tr>
<td>FiO₂</td>
<td>0.5</td>
<td>0.7</td>
<td>1.0</td>
<td>1.0</td>
<td>0.7</td>
<td>0.5</td>
<td></td>
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<tr>
<td>RR</td>
<td>40</td>
<td>50</td>
<td>65</td>
<td>70</td>
<td>50</td>
<td>40</td>
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<tr>
<td>OI</td>
<td>9</td>
<td>19.1</td>
<td>61.8</td>
<td>45.9</td>
<td>10.08</td>
<td>5.6</td>
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<td>OSI</td>
<td>5.85</td>
<td>11.55</td>
<td>33.4</td>
<td>25.3</td>
<td>8.2</td>
<td>4.7</td>
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<tr>
<td>PaO₂/FiO₂</td>
<td>114</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>158</td>
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<tr>
<td>Mode of ventilation</td>
<td>VCV</td>
<td>Bilevel</td>
<td>Bilevel</td>
<td>Bilevel</td>
<td>SIMV</td>
<td>SIMV</td>
<td></td>
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<tr>
<td>Noradrenaline infusion rate (μg/kg/min)</td>
<td></td>
<td></td>
<td>0.1</td>
<td>0.15</td>
<td>0.2</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>Dopamine infusion rate (μg/kg/min)</td>
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<td></td>
<td></td>
<td>10</td>
<td>5</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>LUS score</td>
<td>6</td>
<td>10</td>
<td>18</td>
<td>24</td>
<td>24</td>
<td>16</td>
<td>12</td>
</tr>
<tr>
<td>LUS finding description</td>
<td>A-lines in the upper segments and 2–3 B-lines in the posterolateral lower segments</td>
<td>1–2 B-lines in the upper segments &amp; 4–5 B-lines in the lower segments</td>
<td>Multiple B-lines in the upper segments &amp; coalescent B-lines in lower segments bilaterally</td>
<td>Bilateral coalescent B-lines (milky lungs)</td>
<td>Bilateral milky lung parenchyma with a thickened pleural line</td>
<td>Thickened pleural line and 3–5 B-lines bilaterally</td>
<td>Bilateral 3–4 B-lines</td>
</tr>
</tbody>
</table>


On the 7th day of admission, we made a new chest X-ray where we found the existence of bilaterally present consolidated white lungs, while the child was still on mechanical ventilation using Bilevel Mode with a PEEP of 10 cmH₂O, an Respiratory Rate (RR) of 50 breaths per minute, driving pressure of 16 mBar and a Vt of 7 mL/kg. The findings of the ABGs with pO₂ of 62 mmHg and pCO₂ of 87 mmHg correlated with the chest X-ray finding of PARDS, while OI was 19.1 and OSI was 11.5. We made another bedside ultrasound examination where multiple B-lines were seen bilaterally that were becoming coalescent in the lower postolateral segments (LUS score of 18). Because of previously developed hemodynamic instability, continuous infusion of Noradrenaline with an infusion rate of 0.1 mcg/kg/min was initiated (Figure 2).

On the 11th day of admission, we made another bedside lung ultrasound exam where we found bilateral coalescent B-lines in all the segments that were examined appearing as homogenous “milky” white colored lung parenchyma as a typical finding in ARDS (LUS score of 24) (Figure 3). Furthermore, the ABGs were showing severe respiratory failure with findings of pO₂ of 27 mmHg and pCO₂ of 109 mmHg and SpO₂ from 50% to 56%, while OI was 61 and OSI 33. White Blood Cells (WBC) was 25.2 × 10⁹/L and CRP was 98 mg/L. The CT scan was showing, bilaterally, the presence of “ground glass” opacifications confirming the diagnosis.
of ARDS. We were treating the child with triple antibiotic therapy with Colistin, Vancomycin and Imipenem while continuous infusions of Noradrenaline in a dose of 0.1 mcg/kg/min and Dopamine 5 mcg/kg/min were given because of hemodynamic instability. The child was sedated, placed in a prone position and mechanically ventilated with a respiratory rate of 50 to 80 breaths per minute and PEEP 10–13 cm H₂O, which were adjusted according to the findings of the ABGs. Acinetobacter species was isolated into the tracheal specimens and therapy with Colistin was started.

Figure 3. LUS finding of Coalescent B-lines (11th day of admission).

On the 15th day of hospitalization, the ultrasound examination was showing, bilaterally, milky white lung parenchyma with a thickened pleural line (LUS score of 24) (Figure 4). ABGs were showing persistence of respiratory failure with pO₂ of 37 mmHg, pCO₂ of 98 mmHg and SpO₂ of 67% while the count of leukocytes was 18 × 10⁹/L and CRP was 154 mg/L. The OI was 45.9 while OSI was 25.3. The child was ventilated with a PEEP of 13 cmH₂O, a driving pressure of 13 mBar, a Vt of 6 mL/kg and a respiratory rate of 70.

Figure 4. LUS finding of a “Milky” lung (15th day of admission).

On day 20th of hospital admission, the bedside lung ultrasound showed persistence of a thick pleural line bilaterally and persistence of 3–5 B-lines bilaterally in all segments examined (LUS score of 16), while pO₂ was 75 mmHg and pCO₂ of 62 mmHg, CRP was 83 mg/L and WBC count was 14.1 × 10⁹/L. OI was 10 and OSI was 8.2. We ventilated the child with a PEEP of 8 cm H₂O, a Vt of 7 mL/kg and driving pressure of 13 mBar. The child was paced in
a prone position for 12 h per day since third day of admission and lasting to the 20th day
of admission.

At day 25th of hospital admission, the lung US was showing presence of 3–4 B-lines
in the lower segments and 1–2 B-lines in the upper segments (LUS score of 12), while
ABGs were showing pO$_2$ of 79 mmHg, pCO$_2$ of 57 mmHg and SpO$_2$ of 94%. WBC count
was 12.9 $\times$ 10$^9$/L and CRP was 56 mg/L while OI and OSI were 5.6 and 4.7, respectively.
The child was ventilated on SIMV mode with a Vt of 7 mL/kg, PEEP of 7 cmH$_2$O and a driving
pressure of 11 mBar.

Two days later, the child developed a new hemodynamic instability with ultrasonographycal
findings of absence of left-sided pleural sliding and presence of the Barcodesign in M-mode, suggesting the existence of pneumothorax. Chest radiography
was made immediately, where left-sided pneumothorax and pneumomediastinum were
confirmed. Left thoracic drainage was established immediately. Because of persistence of
the pneumomediastinum, jugulotomy was considered for a suitable surgical intervention but
the child developed a new hemodynamical instability and unfortunately died on the 30th day
of hospital admission.

Discussion

In 2015, the Pediatric Acute Lung Injury ConesnsusConference (PAILCC) established changes
of the criteria for diagnosing PARDS. According to PAILCC, when diagnosing PARDS, the
simple equitation of PaO$_2$/FiO$_2$ is not enough, but rather new infiltrates on the chest X-ray
have to be found, which should not be bilateral obligatory, and the child has to have an
OI higher than 4 and an OSI higher than 5. OI is measured according to the following
equitation: $\text{OI} = (\text{FiO}_2 \times \text{mean airway pressure} \times 100)/\text{PaO}_2$, while OSI is measured as:
$\text{OSI} = (\text{FiO}_2 \times \text{mean airway pressure} \times 100)/\text{SpO}_2$. [2].

The child that we presented above had bilateral infiltrates on the chest X-ray, suggesting
pulmonary infection, an OI of 9 and an OSI of 5.8, which means that the above-mentioned
criteria for PARDS were fulfilled. The chest X-ray showed bilaterally white lungs, while the
chest CT scan showed the existence of bilateral “ground glass” opacifications, all confirming
the diagnosis of PARDS.

The usage of the bedside Lung Ultrasound (LUS) in the ICU was previously reported and
described as a feasible and non-invasive and radiation-free method due to monitoring the
lungs and providing valuable data in a mechanically ventilated critically ill adult patients [2–6].
Even LUS is still not well established as a diagnostic tool among children in Pediatric ICUs, we
were performing a repeated LUS examinations in our patient in order to monitor the evolution
of the disease, the aeration, deaeration and reaeration of the lungs as well as looking for
improvement or worsening of the condition while using mechanical ventilation.

The examination was performed according to the BLUE Protocol established by
Lichtenstein et al. Due to each examination, we have examined 12 pulmonary segments
or six segments by lung. The upper anterior, lateral and posterior as well lower anterior,
lateral and posterior segments of each side were examined and the findings were scored
according to the LUS score, where the total scoring of 0 means complete aeration of the lungs,
while the worst total score possible showing bilateral consolidation and deaeration would be 36 [3,4]. We were performing ABGs routinely as well and the findings were previously
mentioned already.

Due to the first examination of the child, we found the presence of A-lines in the upper
segments and 2–3 B-lines bilaterally in the posterolateral segments of the lungs (LUS Score of 6).
The finding of A-lines has shown presence of normal, non-affected and well-aerated
lung parenchyma, while the finding of 2–3 B-lines has shown affection of the lower lung segments, especially posterior and lateral, while the anterior segments were still preserved.

The finding of a 3 B-lines in one intercostals acoustic window is a sign of interstitial-alveolar syndrome existence which means loosing of the lung aeration because of extravascular lung water presence [3–9]. The LUS is the most accurate method for diagnosing the interstitial-alveolar syndrome in patients with ARDS with an accuracy of 95% according to the study of Lichtenstein D. et al., where the diagnostic accuracy of chest auscultation, chest X-ray and LUS were compared [10].

Due to the second LUS scan, we found existence of 1–2 B-lines in the upper segments bilaterally and 4 to 5 separated B-lines in the lower lung segments (LUS Score of 10), while the findings of the ABGs has shown existence of acute respiratory failure manifested with hypoxemia and hypercapnia. The finding of the second LUS exam in comparison with the first examination is showing worsening of the lung aeration bilaterally, even in the upper lung segments, which is compatible with the ABGs finding of impaired gas exchange.

On the 7th day of hospitalization, we made a third LUS exam, which has showed the presence of multiple separated B-lines in the upper segments and coalescent B-lines in the lower segments, respectively, with an LUS score of 18. ABGs were showing worsening of the hypoxemia and severe hypercapnia.

On the 11th day of hospitalization, the ABGs were showing further worsening of the gas exchange with very low paO\textsubscript{2} and extremely high values for paCO\textsubscript{2}, while LUS has shown bilaterally present coalescent B-lines with an LUS Score of 24. Coalescent B-lines seen bilaterally were giving to us an appearance of a “Milky” white lung parenchyma under the thickened pleural line when LUS was made at the 15th day of admission, showing the worse LUS finding in our patient. Coalescent B-lines, seen as a homogenous “milky” white appearance, are suggesting filling the alveolar space with edema or, in other words, further loss of the lung aeration [2–7], which will lead to more dramatic gas exchange impairment. Coalescent B-lines as a sign of lung deaeration present bilaterally accompanied with pleural involvement and/or presence of subpleural consolidations with presence of small spared zones in the upper segments are the typical LUS findings of PARDS [3,5,6,8].

On the day 20th of admission, when improvement of the gas exchange was seen by the ABGs findings, we made a new LUS examination, which showed the finding of a thickened pleural line and the presence of 3–5 separated B-lines with a total LUS Score of 16.

On the 25th day of admission, only 3–4 B-lines were seen while further improvement of the gas exchange followed showing better lung function and possible further reaeration and improvement. Unfortunately, a few days later, due to new hemodynamic instability, we found no lung sliding and a left-sided “Barcode” sign after we made a chest X-ray confirming the existence of left-sided pneumothorax and pneumomediastinum. Even beside the usage of protective ventilation strategies with low tidal volumes and the highest PEEP used of 13 cmH\textsubscript{2}O, the child has developed a pneumothorax and pneumomediastinum, which, eventually, can be counted as a complication of the mechanical ventilation due to ventilating patients with PARDS which could be a possible etiology in our case, as well.

As the disease had been progressing in the first two weeks, progressive deaeration with the loss of the A-lines and showing up of B-lines instead, was also noted. According to the findings, B-lines were detected due to lung de-aeration and gas exchange impairment. From the LUS examination results discussed above, we can see that coalescent B-lines as a worst seen finding in our patient due to LUS examination quantified with an LUS Score of 24 correlated with the worst-seen ABGs findings, where the retention of the CO\textsubscript{2} was the highest, showing significant impairment of the gas exchange and alveolocapilar insufficiency. Improvement of the gas exchange was seen later on, accompanied by the disappearance
of the coalescent B-lines and detecting 3–5 separated B-lines instead, which suggests lung reaeration and improvement of the ventilation settings.

Performing LUS on a daily basis in a patient with PARDS based on the previous experience from the usage of the LUS in adults could be of a tremendous help in monitoring the injured lung, while identifying B-lines is crucial and following their evolution in a time framework is more than essential.

**Conclusions**

As a point of care method, performing an LUS in patients with PARDS could be used due to the monitoring of the injured lung and following the evolution of the disease as well as monitoring the aeration of the lung and its impairment or improvement in the function of time. Repeated LUS scans give us a significant insight into the pathophysiologic events concerning ventilation in critically ill children with PARDS, providing the data needed to adjust and guide therapy. Separated B-lines are the mainstay of diagnosis of alveolo-interstitial syndrome, while their evolution into coalescent ones with the aspect of a “milky” white lung parenchyma is typical for more severe lung damage, as is seen in PARDS.

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**Conflicts of Interest:** The authors declare no conflict of interest.

**References**