Pseudocyst in Pulmonary Acute Respiratory Distress Syndrome (ARDS)

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ABSTRACT

Childhood ARDS is mostly caused by pneumonia. Pulmonary pseudocysts are reported in adults recovering from ARDS, usually in non-dependent lung regions. The authors present a 1.5-year-old boy, who survived severe pulmonary ARDS with development of pulmonary giant pseudocysts and other structural abnormalities in dependent lung region. To the best of authors knowledge, it is the first follow up report of pulmonary abnormality in a toddler with ARDS of extreme severity.

Key words: Pneumonia; ARDS; Pulmonary pseudocyst; Children

Progress in pathological understanding of ARDS led to evolution of concepts of ‘barotrauma,’ ‘volutrauma’ and now ‘cellular biotrauma’ to explain air-leaks with high pressure ventilation. Air leaks usually present as pneumothorax and pneumomediastinum in patients with severe ARDS. Of late, covert injury as pseudocyst formation has been reported on CT scans. We report development of giant pseudocyst in a young toddler, who survived severe protracted ARDS.

REPORT OF CASE

A 1.5-yr-old boy presented with fever for 7 days, recurrent vomiting and 3 brief tonic seizure episodes on day 6-7 of illness with loss of sensorium. He developed fast breathing after first episode of seizure. Examination revealed tachycardia (114/min), tachypnea (44/min), hypotension (60/44 mm Hg), petechiae and ecchymosis, palpable splenomegaly (2cm), hepatomegaly (span: 10cm), altered sensorium (GCS score-6; E 1M4V1), neck rigidity, exaggerated tendon jerks, extensor plantars, normal pupils and fundus. PRISM III score was 5. He was intubated for airway instability. Chest radiograph showed bilateral non-homogeneous alveolar opacities. He had anemia (Hb 71g/L), relative neutrophilia (TLC, 13X10^9/L), thrombocytopenia (42X10^9/L), coagulopathy (PTI 53%, PTTK 36%[control 30%], negative D-dimer), normal serum electrolytes and renal functions. CSF analysis showed 20 cells (50% PMNs), elevated protein (60 mg/dL) and hypoglycorrhachia (CSF sugar, 60mg/dL; blood sugar, 134mg/dL). Initial cultures were sterile. He was managed with antibiotics (Ceftriaxone, Amikacin and Metronidazole), CVP-guided fluid, inotropes and other supportive care as per our Pediatric Intensive Care Unit (PICU)’s protocol.

On day 3 of admission, he was mechanically ventilated for rising PaCO₂ with initial settings of Positive End-Expiratory Pressure (PEEP) 5 cmH₂O, ΔP of 10 cmH₂O, FiO₂ 0.4 and tidal volume 6-8 ml/Kg. At the outset, PaO₂/FiO₂ ratio and oxygenation index were 282 and 2.8 respectively. Over next 2 days, there was gradual worsening of radiological opacities, PaO₂/FiO₂ ratio and oxygenation index were 282 and 2.8 respectively. Next 2 days, there was gradual worsening of radiological opacities, PaO₂/FiO₂ ratio and oxygenation index were 282 and 2.8 respectively. Next 2 days, there was gradual worsening of radiological opacities, PaO₂/FiO₂ ratio and oxygenation index were 282 and 2.8 respectively. Next 2 days, there was gradual worsening of radiological opacities, PaO₂/FiO₂ ratio and oxygenation index were 282 and 2.8 respectively. Next 2 days, there was gradual worsening of radiological opacities, PaO₂/FiO₂ ratio and oxygenation index were 282 and 2.8 respectively. Next 2 days, there was gradual worsening of radiological opacities, PaO₂/FiO₂ ratio and oxygenation index were 282 and 2.8 respectively.

During acute phase, high PEEP (maximum; 13 cmH₂O) and high ΔP (maximum; 22-23 cmH₂O) were needed to achieve the goals. During recovery, ventilatory support was gradually reduced; by day 57, he was on ΔP of 7 cmH₂O and PEEP

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of 5 cmH\textsubscript{2}O. Subsequent weaning was slow for
development of critical illness neuropathy. CPAP could
be initiated on 95\textsuperscript{th} day and was required for another 36
days. Ventilatory and blood gas parameters at 0800
everyday for initial 60 days are shown in fig. 1. His PICU
stay was complicated by polymicrobial sepsis and
nosocomial pneumonia. Growth of coagulase-negative
\textit{Staphylococcus} (blood, day 13), \textit{Candida tropicalis} (blood,
day 9 and urine, day 15) and \textit{Klebsiella pneumoniae} (blood,
day 37 and 55) were obtained at different points of PICU-
stay. The clinically significant cultures were treated as per
sensitivity. His total hospital stay was 5 months.

On day 24 of PICU admission, chest radiograph
revealed pneumomediastinum and pneumopericardium,
and next day he developed subcutaneous emphysema,
which improved with conservative management. Chest
radiograph obtained on day 73 revealed a large thin
walled pneumocyst on left side. CT chest on day 112
revealed patchy areas of consolidation and ground glass
densities in both the lungs. Multiple small thin walled
pneumocystic lesions were seen in left lower lobe, largest
measuring 49mm × 38mm (Fig. 2a). At 8 months of ARDS
onset, there was persistence of the large thin walled left
sided pneumocyst on chest radiograph (Fig. 2c); and of
consolidation, ground glass densities and thin walled
multiple cysts were persisting on CT chest (Fig. 2c).
Patchy areas of fibrosis had appeared bilaterally.

During follow up (fortnightly visits for initial 3 months
and then monthly visits), he continued to have cough,
tachypnea (RR, 35-40/min), nasal flaring, chest
retractions, bilateral fine crepts, right sided bronchial
breathing, and reduced air entry in left hemithorax.
His critical illness neuropathy gradually improved and he
became ambulatory by 10 months. At 10 months of ARDS
onset, he was prescribed high dose oral ambroxol (12 mg/
Kg/day for one month) for its potential mucolytic
expectorant effects. With ambroxol, cough had
disappeared within 2 weeks. He was last seen at 18
months with slow but continued improvement in exercise
tolerance. Chest examination revealed improved but
reduced air entry and fine crepts at infrascapular and
inferior axillary areas in left hemithorax. The repeat chest
radiograph revealed bilateral multiple thin-walled cysts
(Fig. 2d).

\textbf{DISCUSSION}

Acute respiratory tract infections (ARTIs) are the
commonest cause of ARDS in Pediatric Intensive Care
Units of developed economies,\textsuperscript{4} and should not be
different in developing economies where ARTIs continue
to cause alarming childhood mortality. The published
data on changes in lung parenchyma during recovery
from ARDS are scanty\textsuperscript{2,3} and almost non-existent in
children. Presence of fast breathing and bilateral non-
homogeneous alveolar opacities in the index case at
admission suggested direct acute lung injury, presumably
caused either by community-acquired pneumonia or by
aspiration pneumonitis, which might have been
aggravated by septic shock. The patient progressed to
severe protracted ARDS and later developed large
pseudocyst in dependent lung region. Pneumatoceles/
pseudocyst formation is commonly seen in staphylococcal

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure1.png}
\caption{Line diagram showing daily (0800) ventilatory and oxygenation parameters.}
\end{figure}