

## MEDICINE

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### SEVERE BRONCHOPULMONARY DYSPLASIA AND PEDIATRIC ACUTE RESPIRATORY DISTRESS SYNDROME. RESEMBLANCE AND CONTRAST

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#### Abstract

The leading causes of death among children under five still stay a respiratory insufficiency, preterm birth complications, pneumonia, intrapartum-related complications, diarrhea, and congenital abnormalities. Acute respiratory distress syndrome (ARDS) is the most dramatic complication of lung system failure. Similar clinical manifestation has a severe bronchopulmonary dysplasia (BPD). Both of these pathologies lead to severe respiratory insufficiency and require intensive therapy, respiratory support in paediatric intensive care unit (PICU). Manifestation of severe BPD similar to ARDS, but it is not the same and management has some nuances. In this article we had tried to analyze resemblance between BPD and PARDS and determine contrasts.

**Keywords:** *BPD. ARDS. Chronic lung disease. Intensive therapy. Respiratory support.*

**Introduction:** Bronchopulmonary dysplasia (BPD) was known since 1967 year as a chronic lung disease most commonly seen in premature infants who required mechanical ventilation and oxygen therapy for acute respiratory distress but can also occur in neonates that had a less severe respiratory course [1,2]. BPD is the most common chronic respiratory disease

in infants. BPD is the leading cause of chronic lung disease in children [3]. Main causes of BPD these are lung tissue immaturity, mechanical ventilation, oxygen toxicity, infection and inflammation, genetic predisposition. Mortality at children with severe BPD until 2 years old about 25%[4].

**Materials and methods:** We had provided literature review of up to dated information about BPD and PARDS, further more comparing analysis of collected data was done. Our own observing study was provided in the City hospital #1 Astana, Kazakhstan. We had provided observation of clinical manifestations and laboratory data collection at 18 children. The groups were balanced by age they were studied during hospitalisation at PICU. Age of the patients from 28 days till 3 years old, 7 of them with PRDS and 11 with severe BPD in paediatric intensive care unit. All patients had a respiratory insufficiency of the 3rd degree, due to pneumonia. They were on invasive ventilation, orotracheal intubation, with the same modes of ventilation. Management of all patients was equal includes antibacterial therapy, fluid management, cardiovascular support, diuretics and artificial lung ventilation.

ARDS represents the most severe form of acute lung injury (ALI) and is characterized by alveolar leukocyte infiltration and protein-rich pulmonary oedema [5]. Mortality from paediatric acute respiratory distress syndrome (PARDS) varies widely from 15% and up to 45% [6,7].

**Results:** The same Biomarkers in BPD and PARDS were detected in numerous investigations. Various biomarkers detected in different biological fluids have been proposed for early identification of BPD predisposed newborns (8). Among these that have been implicated both in BPD and PARDS (*vide infra*) are the following. KL-6 (a lung injury marker), interleukin (IL)-6, interleukin-8, sICAM-1, angiopoietin-2, and matrix metalloproteinase-8,9. [9-13].

Pathogenesis of BPD and PARDS. BPD is caused due to an interaction between genetic and environmental factors (hyperoxia, invasive mechanical ventilation, and sepsis [14]. Immature lung tissue impacted by external factors: infections, high concentration of oxygen, long time ventilation, barotrauma, volutrauma, or atelectotrauma, which initiates a cascade of inflammation reaction involving cytokines. This activates the cell death pathways. Damage of immature lungs is followed by resolution of injury to close to normal lung architecture or repair, and leads to fibrosis [15]. On the other hand PARDS pathogenesis consists of cascade mechanism after direct pulmonary tissue damage resulting into the destruction of alveolar-capillary unit. Damage of alveolar-capillary barrier resulted in increased permeability of big molecules such as protein rich oedema fluid into the alveolar lumen, dysfunction of surfactant production, and impaired fluid clearance from alveolar cells. These changes are ARDS pathophysiology chain and accompanied by dysregulated inflammation from dysfunctional leukocytes and influx of cytokines [13].

Pathogenesis of BPD and PARDS has a lot of resembling features and contrast points. But one pathology cannot exclude another one. Moreover, children with BPD are at risk for development ARDS. [16].

Data of patients with PARDS and BPD. Tab.1.

	PARDS	BPD
Total amount	7	11
Mean day of PICU hospitalisation	27	31
Type of respiratory support	Invasive ventilation	Invasive ventilation
Regiment of ventilation	SIMV	SIMV
Mean days on ventilation.	12.3(±4.2)	21.4(± 6.1)
Mean days on noninvasive respiratory support.	6.4(±3.8)	7.8(±7.2)
Dead	1(14.3%)	3 (27.3%)

Findings indicate that children with bronchopulmonary dysplasia have longer duration of hospitalisation in PICU than children with respiratory distress syndrome. Quantity of days on respiratory support in BPD group was higher. Furthermore, in the group of patients with severe BPD mortality rate was higher than in group with ARDS. At the same moment management was consimilar. During observing study of patients with severe respiratory insufficiency we emphasised what patients with BPD had high titre of cytomegalovirus infection, but this issue was not included in our study, potentially it could have impact to results. This item should be examined more carefully.

**Conclusion:** despite of numerous resembling points in aetiology, pathogenesis and biomarkers diagnostics PARDS and BPD is not the same, because of outcomes are divers. Group of patients with respiratory insufficiency on the background of BPD had worse results than previously well fitted patients with ARDS. Variability in reported data presents challenges in tactics of intensive care of patients with BPD. Patients with severe BPD should be managed in another way. And issues at management of severe respiratory failure in the background of BPD in PICU should be more enlightened.

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**12th International Scientific Conference «Science and Society»  
London, 24-29 November 2017**

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cases of severe pediatric acute respiratory distress syndrome (ARDS) improves oxygenation. A chronic lung disease such as bronchopulmonary dysplasia or cystic fibrosis, participation in other clinical studies except treatment protocols and studies for oncological diseases. Neonatal acute respiratory distress syndrome (NARDS) reflects pulmonary surfactant dysfunction, and the usage of bovine surfactant (Calsurf) supplement may therefore be beneficial. To determine whether bovine surfactant given in NARDS can improve oxygenation and survival rate, we conducted a multicenter, randomized trial between January 2018 and June 2019, and we compared Calsurf treatment to controls in neonates with pneumonia accompanied by NARDS. Bronchopulmonary dysplasia (BPD) is a chronic inflammatory lung disease of very-low-birth-weight (VLBW) preterm infants, associated with arrested lung development and a need for supplemental oxygen. Over the past few decades, the incidence of BPD has significantly raised as a result of improved survival of VLBW infants requiring mechanical ventilation. In this review, we provide an overview of biomarkers for pediatric BPD and PH that have been identified in clinical studies using various biological fluids. We also present a brief summary of the information available on current strategies and guidelines to prevent and diagnose BPD and PH, as well as their pathophysiology, risk factors, and experimental therapies.

Acute respiratory distress syndrome (ARDS) is a type of respiratory failure characterized by rapid onset of widespread inflammation in the lungs.[1] Symptoms include shortness of breath, rapid breathing, and bluish skin coloration.[1] Among those who survive, a decreased quality of life is relatively common.[1]. ARDS is the severe form of acute lung injury (ALI) and of transfusion-related acute lung injury (TRALI). The Berlin definition included ALI as a mild form of ARDS.[4] However, the criteria for the diagnosis of ARDS in the Berlin definition excludes many children and a new definition for children was called pediatric acute respiratory distress syndrome (PARDS). This is known as the PALLIC definition.[5][6].

Bronchopulmonary dysplasia (BPD), also known as neonatal chronic lung disease (CLD), is an important cause of respiratory illness in preterm newborns. The pathophysiology of BPD is complex. It has been challenging to maintain a consistent definition of BPD because of changes in the population at risk (ie, greater number of patients at earlier gestational ages) and advances in neonatal management (ie, surfactant and antenatal glucocorticoid therapy and less aggressive mechanical ventilation) have altered the pathology and clinical course of BPD and led to revisions in its definition (table 2).

Bronchopulmonary dysplasia (BPD) is a form of chronic lung disease that develops in preterm neonates treated with oxygen and positive-pressure ventilation (PPV). Northway et al reported clinical, radiographic, and histologic changes in the lungs of preterm infants who had respiratory distress syndrome (RDS) and were treated with oxygen. Many infants born with bronchopulmonary dysplasia exhibit signs and symptoms of respiratory distress syndrome, including the following: Tachypnea. Tachycardia. Increased respiratory effort (with retractions, nasal flaring, and grunting). Frequent desaturations. These infants are often extremely immature, have a very low birth weight, and have significant weight loss during the first 10 days of life.