

# 11<sup>TH</sup> WORLD PEDIATRIC CONGRESS

AUGUST 16-17, 2018 SINGAPORE

## A case report of pulmonary vascular air embolism in ELBW premature neonate

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**Introduction:** Pulmonary vascular air embolism is a rare and almost fatal complication of positive pressure ventilation in premature neonates with respiratory distress syndrome.

**Case Report:** Male premature baby born at 26 weeks of gestation, by caesarean section weight 940 g because of chorioamnionitis, with Apgar score 3 and 4 at 5 minutes was transferred from another hospital at the age of 10 hours to our NICU, with the diagnose of pulmonary air leak syndrome. He was treated with surfactant, high mechanical ventilatory support, antibiotic and insulin therapy on vasopressors. At admission he was intubated, ventilated mechanically with positive pressure, FiO<sub>2</sub> 100%, with no spontaneous respirations, hypotensive, oliguric, generalized edematous with multiple bruises on scalp, anterior chest wall and legs, hypothermic 33.5 °C, initial oxygen saturation 70%, heart rate 120/min and weak pulse. Breath sounds on right side were decreased, no heart murmur. Umbilical vein catheter was inserted. On chest X-ray were signs of respiratory distress syndrome and right lung pneumonia, with normal cardiac appearance. He was treated with SIPPV+VG ventilation, PEEP 6 cm H<sub>2</sub>O, second dose of surfactant, dopamine, dobutamine and infusion of 10% glucose with insulin. His respiratory and circulatory condition started to improve, satisfactory urine output, better laboratory and chest radiography findings. Suddenly 10 hours later his clinical condition deteriorated with drop in oxygen saturation to 44% and hypotension. Chest X-ray had no signs of pulmonary air leak syndrome (pneumothorax, pneumomediastinum). Parameters of mechanical ventilation were raised, PEEP 8, PIP to 34 cm H<sub>2</sub>O for 70/min, as well as supportive therapy, but poor result (saturated O<sub>2</sub> to 70%). He was generalized cyanotic, but suddenly two hours later his lower half of the body from the umbilicus became completely pale. In few minutes revascularization started, but heart sounds were silent, with bizarre ECG irregularities, flat pulse. Chest radiography showed air in heart chambers and in the major vessels of the neck and abdomen. Echocardiography could not be performed because of air. As a consequence, he became progressively bradycardic and expired shortly thereafter. Postmortem examination revealed hyaline membrane disease with diffuse alveolar hemorrhage, diffuse hypoxic multiorgan damage. However, post-mortem examination was unable to disclose the cause of this massive fatal air embolism.

**Discussion:** The exact mechanism of the air leaking in the vascular space is less likely to be clinically explained. Two preconditions must exist: Direct communication between a source of air and the vasculature and a pressure gradient favoring the passage of air into circulation. Rupture of alveoli into pulmonary capillaries due to barotraumas through alveolar-capillary or bronchovenous fistulae is thought to be the main cause of massive air embolism in premature neonates which puts a substantial strain on the right ventricle, especially if this results in a significant rise in Pulmonary Artery (PA) pressure. The increase in PA pressure can lead to right ventricular outflow obstruction and further compromise pulmonary vascular return to the left heart. The diminished pulmonary venous return will lead to decreased left ventricular preload with resultant decreased cardiac output and systemic cardiovascular collapse.

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