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PERSISTANT PULMONARY HYPERTENSION OF THE NEWBORN AND PULMONARY VASCULAR REMODELING

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Annotation

Persistent pulmonary hypertension of the newborn (PPHN) represents continued high pulmonary vascular resistance (PVR) after birth resulting in extrapulmonary shunting of the blood from pulmonary to systemic circulation, leading to hypoxemia. The disorder can be idiopathic or primary or could complicate respiratory, neurologic, and cardiovascular morbidities. Perinatal asphyxia with meconium aspiration syndrome or pneumonia can result in impaired lung recruitment, suboptimal oxygenation, and pulmonary vasoconstriction.

Keywords: Persistent pulmonary hypertension of the newborn (PPHN), pulmonary oligoemia, hypoxemia, pulmonary vasodilatators, early surfactant, ductus areteriosus, foramen ovale, prostacyclin (PGI2).

Persistent pulmonary hypertension of the newborn (PPHN) is estimated to affect 1.9 per 1000 live births and is characterized by a failure of transition from fetal to newborn circulation. PPHN is secondary to the persistence of high pulmonary vascular resistance (PVR), resulting in extrapulmonary shunting of the blood from pulmonary to systemic circulation and leading to hypoxemia. It is often secondary to respiratory disease, although occasionally, PPHN can present as primary or idiopathic, often associated with “black lungs” on a chest x-ray due to absence of lung disease and pulmonary oligemia. PPHN could complicate the course of a sick newborn infant, leading to respiratory, neurologic, and cardiovascular morbidities and mortality. Commonly identified in term infants, increasing evidence supports

the presence of PPHN in preterm neonates. Neonates with PPHN are often managed using positive pressure ventilation (PPV), oxygen to correct hypoxemia, surfactant replacement therapy, vasopressors to maintain systemic pressures, pulmonary vasodilators, and adequate sedation with minimal stimulation. According to the Extracorporeal Life Support Organization, 20% of neonates with PPHN required extracorporeal membrane support (ECMO), although early surfactant and inhaled nitric oxide (iNO) can potentially further reduce the need for ECMO.

Interference in the mechanism of transition during birth could lead to persistent elevation of pulmonary arterial pressures, leading to PPHN. PPHN is characterized by labile systemic arterial hypoxemia secondary to elevated PVR in relation to systemic vascular resistance (SVR), with resultant right-to-left (pulmonary to systemic circulation) shunting through persistent fetal channels such as the ductus arteriosus and foramen ovale, bypassing the lungs.

The transition from fetal to newborn circulation occurs at birth with aeration of the lungs, which drops PVR and increases blood flow to the lungs by 8- to 10-fold. With clamping of the umbilical cord, there is an increase in SVR, increasing left ventricular afterload, and switching of the shunts at the foramen ovale and ductus arteriosus from right-to-left to left-to-right.

Idiopathic PPHN secondary to pulmonary vascular remodeling in the absence of lung pathology could present with histopathological features of smooth-muscle hypertrophy and extension of the muscular layer to more peripheral vasculature such as the preacinar arterioles. During fetal life, high pulmonary vascular pressures and resistance are maintained by humoral mediators such as endothelin 1, leukotrienes, and thromboxanes along with decreased levels of pulmonary vasodilators such as nitric oxide (NO) and prostacyclin (PGI2). NO exhibits its effect on pulmonary vasodilation by increasing cyclic guanosine monophosphate (cGMP) in the smooth muscles, which causes relaxation. Higher endothelin 1 and lower cGMP could contribute to abnormal pulmonary vasculature, in turn contributing to the pathology. Urea cycle defects leading to low levels of arginine, the precursor of NO, could contribute to PPHN. Maternal ingestion of cyclooxygenase inhibitors such as aspirin during late pregnancy may be associated with ductal closure and lead to PPHN, although this association has been recently questioned. Maternal intake of selective

serotonin reuptake inhibitors has also been associated with PPHN, possibly due to pulmonary vasoconstrictive effects of serotonin.

In conclusion, Neonates with PPHN are often managed using positive pressure ventilation, oxygen to correct hypoxemia, surfactant replacement therapy, vasopressors to maintain systemic pressures, pulmonary vasodilators, and adequate sedation with minimal stimulation. Regardless of treatment intervention, neonates with PPHN have 10% mortality along with poor pulmonary and neurodevelopmental outcomes. In neonates randomized to early inhaled NO with PPHN, 25% had impaired neurodevelopmental outcome and 22% had hearing impairment.

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