Abnormal Respiratory Conditions in the Newborn

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Meconium Aspiration Syndrome (MAS)

Definition: This is neonatal respiratory distress in a newborn in the context of meconium-stained amniotic fluid (MSAF) when respiratory symptoms cannot be attributed to another etiology (Sayad and Silva-Carmona, 2023). Aspiration of meconium, an irritant that produces inflammation, creates an obstruction ball-valve effect, and interferes with surfactant activity. It occurs in 2–10% of term and post-term infants exposed to meconium-stained amniotic fluid (MSAF), and about 5–30% of term and post-term births occur with MSAF. There is a 4-40% mortality rate (Snell and Gardner, 2017).

Causes: MAS is due to the aspiration of MSAF. MSAF is not always associated with MAS. Increased peristalsis, anal sphincter relaxation, and changes in vagal and sympathetic tones in the context of fetal distress and hypoxia can all lead to meconium passage. Fetal breathing usually leads to amniotic fluid moving in and out of the lungs. When amniotic fluid is stained with meconium, the fetus is at risk of aspiration. This is especially true with hypoxia, which can trigger the fetus to increase gasping. As meconium is thick and the fetal airways are small in diameter, the presence of meconium in the airways can cause obstruction. The meconium plug can cause complete or partial obstruction, leading to lung collapse distally, atelectasis, and ball valve effect with increased air trapping, thus increasing the risk of air leak syndromes and pneumothorax. Material that constitutes meconium has been shown to trigger inflammatory processes that further contribute to the development of respiratory distress in MAS. Airway inflammation results in a form of chemical pneumonitis. Inflammation and hydrolysis can alter and inactivate surfactants. This leads to increased surface tension, poor compliance, and impaired oxygenation. All these factors combined contribute to the severity of MAS (Sayad and Silva-Carmona, 2023).

Signs & Symptoms: Progressive disorder with symptoms increasing from inflammation to inability to oxygenate/ventilate. Tachypnea, grunting, flaring, retracting, hypoxemia, acidosis, low Apgar's, cyanosis, or hypotonic (Sayad and Silva-Carmona, 2023) and (UCSF Benioff Children's Hospitals, n.d.). Increased risk for pneumothorax and persistent pulmonary hypertension.

Management/Treatments: Management requires an interprofessional team approach, including the obstetrician, midwife, neonatologist, respiratory therapist, nurse, pediatric pulmonologist, and pediatric cardiologist. Treatments for meconium aspiration depend on the amount and thickness of the meconium, the length of time your baby was exposed to it, and the severity of the respiratory problems your baby is experiencing. Initial steps would be Suctioning the baby's upper airways, including the nose, mouth, and throat, and giving the baby supplemental oxygen by hood or mechanical ventilator. Tapping on the baby's chest to loosen secretions, a technique known as chest physiotherapy, antibiotics to treat infection, and radiant warmer to maintain the

baby's body temperature (UCSF Benioff Children's Hospitals, n.d.). Supplemental oxygen is often needed in MAS with a goal of oxygen saturation > 90% to prevent tissue hypoxia and improve oxygenation. Ventilatory support is indicated with refractory hypoxemia despite oxygen therapy, carbon dioxide retention, and increased respiratory distress. Oxygenation monitoring and serial blood gasses to help optimize oxygenation and ventilation are key. In severe cases with refractory hypoxemia, the newborn might need extracorporeal membrane oxygenation (ECMO) for cardiorespiratory support. Using surfactant in MAS is not standard of care, but may be helpful in some cases. Inhaled nitric oxide is a pulmonary vasodilator that has a role in pulmonary hypertension and PPHN (Sayad and Silva-Carmona, 2023).

Risk Factors: Post-term pregnancy, Pre-Eclampsia, Maternal Diabetes, Maternal Hypertension, Difficult Delivery, Fetal Distress, and Intra-uterine Hypoxia (UCSF Benioff Children's Hospitals, n.d.).

Infant Respiratory Distress Syndrome (RDS)

Definition: Insufficient surfactant, a phospholipid that lowers alveolar surface tension, prevents alveolar collapse at expiration, and maintains functional residual capacity (Snell and Gardner, 2017). Neonatal respiratory distress syndrome, or RDS, is a common cause of respiratory distress in a newborn, presenting within hours after birth, most often immediately after delivery. RDS primarily affects preterm neonates, and, infrequently, term infants. The incidence of RDS is inversely proportional to the gestational age of the infant, with more severe disease in the smaller and more premature neonates (Yadav et al., 2023).

Causes: Neonatal respiratory distress syndrome (RDS) occurs from a deficiency of surfactant, due to either inadequate surfactant production, or surfactant inactivation in the context of immature lungs. Prematurity affects both these factors, thereby directly contributing to RDS (Yadav et al., 2023).

Signs & Symptoms: The newborn may present with decreased breath sounds, diminished peripheral pulses, increased work of breathing, including tachypnea, expiratory grunting, nasal flaring, retractions (subcostal, subxiphoid, intercostal, and suprasternal), and use of accessory muscles, as well as cyanosis and poor peripheral perfusion. In untreated RDS, the symptoms will progressively worsen over 48 to 72 hours towards respiratory failure, and the infant may become lethargic and apneic (Yadav et al., 2023)

Management/Treatments: Diagnosis can be done by identifying perinatal risk factors, clinical presentation, radiographic findings, and evidence of hypoxemia on blood gas analysis. An echocardiogram or a complete blood count (CBC) may also be indicated for diagnosis. The goals of optimal management of neonatal respiratory distress syndrome include decreasing incidence and severity using antenatal corticosteroids, followed by optimal management using respiratory support, surfactant therapy, and overall care of the premature infant. This includes antenatal corticosteroids, monitoring oxygenation and ventilation, assisted ventilation of the neonate

exogenous surfactant therapy, and supportive care, including thermoregulation, nutritional support, fluid and electrolyte management, antibiotic therapy, etc. (Yadav et al., 2023).

Risk Factors: Preterm <35 weeks, siblings that had RDS, twin or multiple births, cesarean delivery, maternal GDM, infection, baby that is sick at the time of delivery, cold, stress, or hypothermia (Nationwide Children's, 2022)

Transient Tachypnea of the Newborn (TTN)

Definition: Failure to clear lung fluid by usual mechanisms and inadequate sodium transport at the cellular level (Snell and Gardner, 2017). TTN is a benign, self-limited condition that can present in infants of any gestational age shortly after birth. It affects approximately 10% of infants delivered between 33 and 34 weeks, approximately 5% between 35 and 36 weeks, and less than 1% in term infants (Jha et al., 2023).

Causes: It is caused by a delay in the clearance of fetal lung fluid after birth, which leads to ineffective gas exchange, respiratory distress, and tachypnea (Jha et al., 2023)

Signs & Symptoms: Typical presentation includes Tachypnea (respiratory rate greater than 60 per minute), Nasal flaring, Grunting, Intercostal/subcostal/suprasternal retractions, Crackles, diminished or normal breath sounds on auscultation. Occasionally, symptoms may include Tachycardia, Cyanosis, and a Barrel-shaped chest because of hyperinflation (Jha et al., 2023)

Management/Treatments: Diagnosis can be done with preductal and postductal saturations to rule out differential cyanosis, a CBC, blood culture, C-reactive protein (CRP), and lactate to rule out neonatal sepsis. ABG analysis may show hypoxemia and hypocapnia due to tachypnea, and hypercapnia is a sign of fatigue or air leak. A chest x-ray may show hyperinflation, prominent perihilar vascular markings, edema of interlobar septae, or fluid in the fissures. Additional diagnosis measures may be an ammonia level in the setting of lethargy and metabolic acidosis to rule out inborn errors of metabolism or an echocardiography to rule out congenital cardiac defects in patients with differential cyanosis or persistent tachypnea for over 4 to 5 days. Supportive care is the main treatment. Two hours after the onset of respiratory distress, if an infant's condition has not improved or has worsened, or if FiO2 required is more than 0.4 or the chest x-ray is abnormal, consider transferring the infant to a NICU. Routine NICU care includes continuous cardiopulmonary monitoring, maintenance of the neutral thermal environment, securing intravenous (IV) access, blood glucose checks, and observation for sepsis (Jha et al., 2023).

Risk Factors: Maternal risk factors include delivery before completion of 39 weeks gestation, a cesarean section without labor, gestational diabetes, and maternal asthma. Fetal risk factors include male gender, perinatal asphyxia, prematurity, small for gestational age, and large for gestational age infants (Jha et al., 2023).

Persistent Pulmonary Hypertension (PPHN)

Definition: Failure to relax the pulmonary vasculature after breathing oxygen at birth; pulmonary vasoconstriction due to hypoxia after birth, resulting in elevated PVR and decreased SVR (Snell and Gardner, 2017). The placenta functions as the primary organ for air exchange in the fetus. The lungs have to assume this role rapidly after birth. Persistent pulmonary hypertension occurs due to failure of normal transition from intrauterine circulation (Nandula and Shah, 2023). The overall incidence in newborns is 1.8 per 1000 live births. The incidence is higher in late preterm infants at 5.4 per 1000 live births. In term infants, the incidence is 1.6 per 1000 live births. Mortality ranges from 7.6 to 10.7%, depending on the severity of the condition. Boys had a higher risk than girls, and African-American babies had the highest risk, followed closely by Hispanic and Asian infants (Nandula and Shah, 2023).

Persistent pulmonary hypertension in the newborn is categorized into three types:

- 1. **Maladaptation:** abnormal pulmonary vascular response in lung parenchymal disorders such as meconium aspiration syndrome.
- 2. **Underdeveloped vasculature:** decreased pulmonary vasculature as seen in small for gestational age or oligohydramnios.
- 3. **Idiopathic persistent pulmonary hypertension in the newborn,** likely due to excessive pulmonary vascular smooth muscle thickness (Nandula and Shah, 2023).

Causes: Persistent pulmonary hypertension in the newborn can occur due to several reasons. Lung parenchymal conditions such as meconium aspiration syndrome (MAS), pneumonia, respiratory distress syndrome (RDS), and sepsis (Nandula and Shah, 2023).

Signs & Symptoms: Tachypnea, Tachycardia, respiratory distress, including signs such as flaring nostrils and grunting, cyanosis, heart murmur, low oxygen saturation levels (UCSF Benioff Children's Hospitals, n.d.a).

Management/Treatments: Management of persistent pulmonary hypertension includes maintaining temperature, glucose, cardiovascular support, and intravascular volume. Treatment includes inotropes, oxygen therapy and mechanical ventilation, nitric oxide, high-frequency oscillatory ventilation, lung recruitment, and vasopressor support, along with pulmonary vasodilator therapy (Nandula and Shah, 2023).

Risk Factors: Risk factors are oligohydramnios, pulmonary hypoplasia, in utero closure of ductus arteriosus, small and large for gestational age status. Maternal risk factors such as obesity, diabetes, pre-eclampsia, chorioamnionitis, smoking, selective serotonin reuptake inhibitors (SSRI), and NSAID use during pregnancy can also contribute. Congenital anomalies such as transposition of great arteries (TGA) and congenital diaphragmatic hernia (CDH) are also associated with persistent fetal circulation in the immediate neonatal period (Nandula and Shah, 2023).

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