Respiratory Distress in the Newborn

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• Health disparities requirement: there may be language barriers when talking with non-English speaking parents about their newborns with respiratory distress. Information must be communicated to them effectively, through an interpreter, in a culturally sensitive manner.
Case #1

You are called to assess an infant on the Mother-Baby Unit due to tachypnea. The infant is a 36 week GA male born an hour ago via elective C-section due to maternal preeclampsia and breech positioning. Mother also had gestational diabetes.

On exam, the infant is well-appearing, but has a RR of 80 bpm and very mild intercostal retractions. His O₂ saturation is 88%, but increases to 95% with blowby oxygen (FiO₂ 30%).
Transient Tachypnea of the Newborn (TTN)

• “Retained fetal lung fluid” syndrome
• Pathogenesis
  – Inadequate or delayed clearance of fetal lung fluid
  – In fetal life, the lung epithelium secretes chloride and fluid into the air spaces. In late gestation, the lung epithelium switches to reabsorbing sodium and liquid
    • Increased expression and activity of ENaC channels
      – This process is enhanced by labor (catecholamines and endogenous steroids)
    • Mechanical force of birth canal squeeze is a minor contributor in fluid resorption
  – Extra fluid causes decreased pulmonary compliance
Transient Tachypnea of the Newborn (TTN)

• Clinical Presentation
  – Risk factors include preterm or elective C-section delivery without labor
    • Also more common in infants born to mothers with diabetes and asthma
  – Main feature is tachypnea
  – May have increased work of breathing (nasal flaring, mild retractions, grunting), cyanosis (usually need <40% FiO2) and/or mild respiratory acidosis
  – Usually occurs within 2 hours after delivery, often resolves in 24-72 hours (although can last a little longer)
Transient Tachypnea of the Newborn (TTN)

• **Diagnosis**
  – Chest x-ray
    • Bilateral perihilar streaking
    • Fluid in fissure line
    • Can also have patchy infiltrates that clear in 24-48 hours (can be hard to differentiate from pneumonia)

• **Management**
  – Supportive
  – May need supplemental oxygen (usually <40%)
  – May need CPAP (unusual)

• **Prognosis**
  – Good; self-limiting condition
  – Some data that infants are at increased risk of wheezing in the future
Case #2

You are called to an emergent C-section delivery for a patient who was just admitted to L&D. The mother is a South Asian G1 at 41 6/7 weeks gestation who had attempted a home birth, but was not successful (failure to progress, meconium-stained amniotic fluid). The fetal heart tracing shows a fetal heart rate in the 80’s.

The infant is born cyanotic, apneic, and meconium-stained. He does not respond to routine resuscitative measures, including bulb suctioning. You give him PPV, and after ~5 minutes, he starts breathing. He is grunting and has significant subcostal and intercostal retractions. His lung exam reveals a barrel-chest and coarse breath sounds throughout. He is requiring CPAP 6, FiO$_2$ 60% to get his O$_2$ saturations to 90%.
Meconium Aspiration Syndrome (MAS)

• “Respiratory distress in an infant born through meconium stained amniotic fluid whose symptoms cannot be otherwise explained”
  – Meconium-stained amniotic fluid occurs in 5-24% of deliveries
    • Of these, 2-10% have MAS
• Pathogenesis
  – Meconium passage in utero
    • Intrauterine event (stress from fetal hypoxia, vagal input from cord compression) → peristalsis and relaxation of the anal sphincter → in utero meconium passage
  – Aspiration can occur in utero (prolonged hypoxia can cause fetal gasping) or immediately after birth when infant gasps
Meconium Aspiration Syndrome (MAS)

• Risk factors
  – >41 weeks GA
  – SGA
  – Fetal distress
  – Compromised in-utero conditions (ex: placental insufficiency, cord compression), non-reassuring fetal heart tracing, low 5-minute APGAR, instrumented delivery, emergency C-section
  – African American or South Asian race
  – Planned home birth
Meconium Aspiration Syndrome (MAS)

- Clinical
  - Respiratory distress, barrel-shaped chest, rales, rhonchi
  - Chest x-ray- streaky with diffuse parenchymal infiltrates
    - Over time, areas of hyperinflation with patchy areas of atelectasis and infiltrate

- Management
  - Supportive
  - May need supplemental O₂, CPAP, mechanical ventilation, high frequency ventilation
  - Can give exogenous surfactant in severe cases (decreases need for ECMO, no difference in mortality)
  - Evaluate for and manage associated conditions
    - Pulmonary hypertension- occurs in ~1/3
    - Pulmonary air leaks (pneumothorax, pneumomediastinum)- occurs in 15-30%
    - Infection- meconium is sterile, but is a growth medium for gram-negative organisms
      - Blood culture, ideally tracheal culture, antibiotics
  - ECMO in severe cases
NRP

• 7th edition came out on May 6, 2016

• New guidelines supposed to be in use by January 1, 2017

• Non-vigorous infants born through meconium-stained amniotic fluid do not require routine intubation and tracheal suctioning

• Meconium-stained amniotic fluid requires the presence of one team member with full resuscitative skills
Case 3

• A 33 week gestational age infant is delivered precipitously. You initially stabilize the infant with CPAP, but over the next 24 hours, he becomes more tachypneic with increasing FiO$_2$ needs.
Respiratory Distress Syndrome (RDS)

• Formerly called hyaline membrane disease

• Pathogenesis
  – Surfactant deficiency
    • Surfactant reduces alveolar surface tension, which lowers the pressure needed to keep alveoli inflated
    • Surfactant deficiency → low compliance, microatelectasis and low lung volumes → hypoxemia (intrapulmonary shunting)
  – Developmental regulation of surfactant, which the risk increases with decreasing GA
    • Can also (rarely) be due to genetic mutations (SP-B, SP-C, ABCA3) - can occur in term infants
Respiratory Distress Syndrome (RDS)

• Clinical Presentation
  – Respiratory distress shortly after birth- tachypnea, labored breathing, cyanosis, grunting, nasal flaring, retractions
  – Progresses over the first 48 to 72 hours, then improves (correlates with diuresis phase and when infant produces endogenous surfactant)
  – More common in infants of diabetic mothers
  – Chest x-ray- diffuse, reticulogranular, ground-glass air bronchograms, low lung volumes
Respiratory Distress Syndrome (RDS)

- **Management**
  - CPAP/nasal ventilation/mechanical ventilation
  - Exogenous surfactant
    - Standard is through endotracheal tube
    - Non-invasive methods being investigated

- **Prognosis**
  - Risk for BPD

- **Prevention**
  - Antenatal glucocorticoid therapy
Case #4

- You are taking care of a 2 day old 34 week GA infant with RDS, on CPAP 6, FiO$_2$ 30%. You are called urgently to the infant’s bedside he suddenly decompensated. His FiO$_2$ requirements are now 80% and he is working hard to breathe. His heart rate is in the 70’s and his BP is 35/20.
Pneumothorax

• Epidemiology
  – Occurs more often in the newborn period than any other time of life
    • Estimated incidence is 1-2%
    • Higher risk in VLBW infants- estimated incidence is 4.1%

• Pathogenesis
  – Rupture of an overdistended alveolus → air escapes from the lung into the pleural space
  – Conditions that predispose to pneumothorax: RDS, MAS, pulmonary hypoplasia, pneumonia, TTN, any condition that needs mechanical ventilation
Pneumothorax

• Clinical Presentation
  – Infants with small pneumothoraces may be asymptomatic
  – Infants may have respiratory distress- tachypnea, grunting, pallor, cyanosis
    • May be sudden onset
  – Can have chest asymmetry (enlargement of the affected side), decreased breath sounds on the affected side, shift of the cardiac PMI away from the affected side
  – Tension pneumothorax can increase the intrathoracic pressure → decrease venous return → decreased cardiac output → hypotension, bradycardia, hypoxemia
Pneumothorax

• **Diagnosis**
  – Transillumination
  – Chest x-ray

• **Management**
  – Close observation if infant is asymptomatic and not on mechanical ventilation
    • Typically resolve in 1-2 days
    • Strategy may also work in infants on low ventilatory settings
  – If symptomatic
    • Oxygen, as needed (no “nitrogen washout”)
    • Thoracentesis
    • Chest tube placement

• **Prognosis**
  – Many resolve spontaneously
  – Pneumothorax in RDS is associated with increased risk of IVH, CLD, and death
Case #5

You are called to attend the delivery of a 40 6/7 weeks gestation female fetus due to maternal PROM x 24 hours and fetal tachycardia.

At birth, the infant is cyanotic and apneic. You administer PPV and the infant starts spontaneously breathing, but still has significant retractions, nasal flaring, and grunting. You transfer the infant to the IMN on CPAP.
Neonatal Pneumonia

• Epidemiology
  – Causes significant morbidity and mortality in developing countries
  – In developed countries, estimated at 1% in full-term infants
    • Incidence may be closer to 10% in ill LBW infants
  – Only discussing early-onset pneumonia (onset within 3 days of birth)

• Pathogenesis
  – Congenital pneumonia (subset of early-onset pneumonia)
    • Acquired in utero
      – Aspiration of infected amniotic fluid
      – Ascending infection through intact or ruptured membranes
      – Hematogenous spread through the placenta
    • Presents immediately after delivery
  – Early-onset pneumonia can also be acquired during labor
    • Aspiration of infected amniotic fluid or bacteria colonizing the vaginal canal
  – Most early-onset pneumonia is caused by aerobic bacteria (usually GBS); may also be due to other bacteria, viruses and other infectious organisms
  – Infants are more susceptible due to an immature immune system, underdeveloped respiratory cilia, and decreased pulmonary macrophages (decreased clearance of pathogens)
Neonatal pneumonia

- **Risk factors**
  - Prolonged rupture of membranes (>18 hours), maternal infection, prematurity, fetal tachycardia

- **Clinical symptoms**
  - Respiratory distress soon after birth (increased work of breathing, oxygen requirement)
  - May have associated signs of sepsis: lethargy, apnea, tachycardia, poor perfusion
  - Can develop pulmonary hypertension

- **Diagnosis**
  - Chest x-ray- diffuse parenchymal infiltrates with air bronchograms or lobar consolidation; might have pleural effusions
  - Obtain CBC, CRP, blood cultures
    - Consider trach culture, CSF culture, viral cultures (if suspected)

- **Treatment**
  - Antibiotics if concern about bacterial infection (usually ampicillin and gentamicin)
  - Respiratory support as needed (CPAP, mechanical ventilation) + supplemental oxygen
Case #6

A woman comes into the hospital and delivers precipitously. She had no prenatal care. The infant appears term.

He cries at delivery, then develops nasal flaring, severe retractions, and cyanosis. You provide PPV with a mask, with no improvement. You intubate the infant, after which the infant’s O₂ saturations stabilize with 50% FiO₂.

The physical exam is notable for decreased breath sounds on the left side of the chest, heart sounds on the right side of the chest, and a flat abdomen.
Congenital Diaphragmatic Hernia (CDH)

- **Pathogenesis**
  - Diaphragmatic defect that allows abdominal viscera to herniate into the chest
    - Bochdalek - posterolateral (95%)
    - Morgagni - anterior
    - 80-85% cases occur on the left, 10-15% occur on right, 2% are bilateral
  - Lung compression causes less bronchial branching, less alveoli, and persistent muscular hypertrophy in the pulmonary arterioles
    - Get pulmonary insufficiency and persistent pulmonary hypertension
  - Often detected on prenatal ultrasound
    - Other anomalies are detected in 30-50% of cases
Congenital Diaphragmatic Hernia (CDH)

• Clinical symptoms
  – Respiratory distress within hours of life (severity depends on degree of lung hypoplasia and PPHN)
  – Absent breath sounds on ipsilateral side
  – Heartbeat on right side (if left CDH with mediastinal shift)
  – Barrel-shaped chest
  – Scaphoid-appearing abdomen

• Diagnosis
  – Chest x-ray- herniated abdominal contents (usually bowel) into hemithorax.
    • May have displacement of mediastinal structures
Congenital Diaphragmatic Hernia (CDH)

- Management
  - Intubation ASAP (if prenatally diagnosed, intubate in delivery room)
    - Decreases gastric distention and associated lung compression
  - NG tube to decompress abdominal contents
  - Gentle ventilation (low PIPs, permissive hypercapnia), avoid acidosis, target pre-ductal saturations ≥85%
  - Central line and arterial line placement
  - Echo-evaluate for associated cardiac anomalies and the presence/severity of PPHN
  - BP support (maintain mean BP >= 50 to minimize shunting)
  - Surgery after respiratory status is stabilized and pulmonary hypertension improves
    - Primary closure, patch repair
  - May need high-frequency ventilation, ECMO

- Surfactant does not improve outcomes
- iNO does not have long-term benefits, but is often tried
Congenital Diaphragmatic Hernia (CDH)

- Prognosis
  - Recent survival rates of 70-92% (infants born at or transferred to tertiary care centers)
    - Decreased survival in premature infants, congenital heart disease (41% vs 70% in one study), persistent and severe PPHN, outborn from tertiary care center, low preductal O2/high CO2, large defects
  - Post-repair complications
    - PPHN, hemorrhage, chylothorax, patch infection
  - Long-term risks
    - Respiratory infections, chronic lung disease
    - Recurrence of diaphragmatic hernia (2-22%)
      - Presents with respiratory or GI symptoms
    - Pectus excavatum, pectus carinatum, thoracic scoliosis
    - GERD (chronic problem)
      - About 10% have intestinal obstruction due to adhesions
    - Failure to thrive (some need NG/g-tube feedings and extra calories)
    - Neurodevelopmental impairment (30-80% of patients), including hearing loss in 30-50%
Case #7

You are called urgently to a L&D room 5 minutes after the delivery of a term baby girl. Mom had an uncomplicated prenatal course and delivery.

The infant cried well after birth, but is now cyanotic with O₂ saturations of 70%. She has moderate retractions. She cries when you examine her, and her O₂ saturations increase to 90%. However, when you give her a pacifier to suck on, her O₂ saturations drop again.
Choanal Atresia

• **Pathogenesis**
  – Congenital blockage of the posterior nares
    • Can be bony or membranous
      – 70-90% have a bony component

• **Clinical presentation**
  – **Unilateral** (~2/3 of cases)
    • Often present in childhood with unilateral nasal discharge or obstruction
  – **Bilateral**
    • Respiratory distress (upper airway obstruction, noisy breathing, cyanosis)
      – Infants are preferential nose breathers for the first 4-6 weeks
      – Symptoms worsen with feeding
      – Symptoms improve with crying
Choanal Atresia

- Diagnosis
  - Inability to pass a catheter into the nasopharynx
  - Confirm with CT
Choanal Atresia

• Management
  – Secure airway
    • Infants may need oral airway or intubation
  – Initiate gavage feedings
  – Bilateral atresia is usually surgically repaired within days of birth
    • Usually endoscopically, transnasally

• Prognosis
  – Associated anomalies in 20-60% of individuals
    • Associated with syndromes including CHARGE & Treacher Collins
  – Most do well, although may need dilations to maintain choanal patency
References

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• Up To Date