Respiratory distress in a newborn

Mohamed Ahmed Fouad
Pediatrics Lecturer
Jazan faculty of medicine
Objectives

• Evaluate and diagnose the most common causes of respiratory distress in the newborn period.

• Differentiate between the normal results of a newborn chest radiograph and the radiographic patterns that reflect RDS, MAS, and neonatal pneumonia.

• Recognize subglottic stenosis as a complication of endotracheal intubation.

• Distinguish between pulmonary disease and cyanotic congenital heart disease as a cause of hypoxemia and acidosis in the neonate.

• Discuss common complications of various respiratory disorders (such as meconium aspiration syndrome)
Neonatal respiratory disorders account for most admissions to intensive care units in the immediate newborn period.

Newborns in respiratory distress must be evaluated promptly and accurately; occasionally, neonatal respiratory distress is life-threatening and requires immediate intervention.

The causes of respiratory distress in the newborn are numerous and are due to pulmonary or non-pulmonary processes.
Respiratory distress in newborns can be assessed by the presence of the following signs:

- Nasal Flaring & Tachypnoea
- Retractions
- Cyanosis
- Grunting
Etiologies

Neurological Disorder
- Birth trauma
- Intraventricular hemorrhage
- Meningitis
- Hypoxic ischemic encephalopathy

Cardiac Diseases

A. Cyanotic
- Transposition of great arteries
- Total anomalous pulmonary venous return
- Ebstein’s anomaly
- Tricuspid atresia
- Pulmonic stenosis
- Tetralogy of Fallot
- Severe congestive heart failure

B. Acyanotic
- Hypoplastic left heart syndrome
- Interrupted aortic arch
- Critical aortic coarctation
- Patent ductus arteriosus
A. Airway Obstructions
- Nasal stenosis
- Choanal atresia
- Laryngeal stenosis or atresia
- Hemangioma
- Tracheobronchial stenosis
- Pierre Robin's sequence
- Vocal cord paralysis
- Vascular rings

B. Disorders of the Chest Wall and Diaphragm
- Disorders of the chest wall
- Congenital diaphragmatic hernia

C. Malformation of the Mediastinum and Lung Parenchyma
- Congenital cystic adenomatoid malformation
- Congenital lobar emphysema
- Pulmonary arteriovenous malformations
- Congenital pulmonary cyst
- Neoplasms (teratomas, mediastinal, neuroblastoma)
- Bronchopulmonary sequestrations

D. Air Leak Syndromes
- Pulmonary interstitial emphysema
- Pneumomediastinum
- Pneumopericardium
- Pneumoperitoneum
- Pneumothorax

E. Pulmonary Parenchymal and Vascular Diseases
- Lung parenchymal diseases
  - Transient tachypnea of newborn
  - Meconium aspiration syndrome
  - Hyaline membrane disease
  - Pneumonia
  - Congenital alveolar proteinosis
  - Pulmonary edema
- Persistent pulmonary hypertension of the newborn
Other Miscellaneous Diseases

- Sepsis
- Anemia or polycythemia
- Hypo or hyperthermia
- Hypo or hypernatremia
- Hypoglycemia
- Inborn errors of metabolism
- Maternal medication (magnesium sulfate, opiates) or drug abuse
Term Baby
- TTN
- MAS
- Congenital Pneumonia
- Dev Anomalies

Preterm Baby
- RDS
- Congenital Pneumonia
- TTN

Respiratory Distress at birth
later after a period of normal function

Possible causes
- Acquired/Nosocomial Pneumonia
- Dev anomalies
- CHD
- IEM
- Metabolic (Met acidosis/electrolytes)
Transient tachypnea of the newborn

- (TTN) occurs in approximately 1–2% of all newborn infants and is due to respiratory maladaptation at birth causing retention of fluids in the lungs (which are normally replaced by air by this time).

- Tachypnea is generally the outstanding feature.

- TTN is usually benign and self-limiting, with symptoms rarely persisting beyond 48 h.
Pathogenesis and risk factors

- Respiratory Distress
- Retained lung liquid
- Elective cesarean section without labor
- Perinatal epinephrine surge
- Lung liquid absorption and mechanical drainage
- Labor
- Vaginal delivery
- Transient Tachypnea of the Newborn (TTN)

Other Risk Factors:
- Maternal diabetes
- Macrosomia
- Cesarean section
- Male sex
- Maternal asthma
- Delivery at a lower gestational age (including late preterm)

Normal Transition
• **Diagnosis**

• The diagnosis of TTN is confirmed by chest radiograph and the clinical course.

• Resolution of the respiratory distress within 48 h confirms the clinical diagnosis retrospectively.

• Chest radiograph shows streakiness caused by interstitial fluid, fluid in the lung fissures and perihilar cuffing.
Treatment

• TTN does not usually require respiratory support, other than extra inspired oxygen.

• Regular blood-gas measurements should be performed in the early stages of the illness. In more severe cases CPAP may aid resolution.

• If the blood gases deteriorate, the diagnosis should be reconsidered or complications such as pulmonary hypertension or pneumothorax may have developed.
Respiratory distress syndrome

- RDS is a specific clinical entity occurring predominantly but not exclusively in preterm infants owing to a lack of surfactant (a surface tension-lowering agent) in the alveoli.

- **Predisposing factors to RDS**
  - Prematurity
  - Infant of a diabetic mother
  - Antepartum haemorrhage
  - Second twin
  - Hypoxia, acidosis, shock
Etiology and pathogenesis

- The major hallmark of RDS is a deficiency of surfactant which leads to higher surface tension at the alveolar surface and interferes with the normal exchange of respiratory gases.

- The higher surface tension requires greater distending pressure to inflate the alveoli.
Clinical features

• The signs of RDS start immediately after birth or become obvious in the first 6h of life.

• In the absence of surfactant each breath the infant takes is like the first breath in an effort to expand the alveoli.

• Fatigue contributes to the respiratory failure.

• The clinical course is usually associated with worsening of the symptoms, with a peak severity at 48–72 h
As the disease progresses the infant shows a need for increasing oxygen, the expiratory grunt may diminish and prolonged apnoea may occur.

Without intervention, recurrent and worsening apnoea superimposed on tachypnoea would indicate impending respiratory failure and the need for mechanical ventilation.

The breath sounds are decreased
Complications

- Pneumothorax
- Patent ductus arteriosus (PDA)
- Necrotising enterocolitis (NEC)
- Subglottic stenosis (causing stridor)
- Chronic lung disease (CLD) and bronchopulmonary dysplasia (BPD)
- Intraventricular-periventricular haemorrhage (PVH-IVH)
- Retinopathy of prematurity (ROP)
Investigations

- Blood gas analysis
- Sepsis work-up (CBC with differential, CRP, and blood culture) to rule out early-onset sepsis.

Chest x-ray: findings can be graded according to the severity

- Grade 1 (mild cases): slight reticular (slight granular) decrease in transparency of the lung, no certain difference to normal findings.
- Grade 2: Soft decrease in transparency with an aerobronchogram, which overlaps the heart.
- Grade 3: like stage 2, but with gradual stronger decrease in transparency, as well as a blurry diaphragm and heart.
- Grade 4: complete white lung fields with obscuring of the cardiac shadow.
Treatment

• **Prevention**

  • Antenatal corticosteroid therapy (dexamethasone 6 mg/dose IM for 4 doses, 12 hrs apart) for pregnant women 24-34 wks' gestation at high risk of preterm delivery within the next 7 days.

• Prophylactic surfactant therapy in preterm infants <27 wks’ gestation.

• Early CPAP administration in the delivery room.
Treatment postnatally

- Administer oxygen (depending on the severity of illness).
- Initiate CPAP as early as possible in infants with mild RDS.
- MV in CPAP failure, severe frequent apnea.
- Administer surfactant therapy: early rescue therapy within 2 hrs after birth is better than late rescue treatment when the full picture of RDS is evident.
Meconium Aspiration Syndrome (MAS)

- Meconium staining of the amniotic fluid indicates fetal distress.

- **Risk Factors**
  - Post-term pregnancy, pre-eclampsia, eclampsia, maternal hypertension, maternal diabetes mellitus, IUGR, and evidences of fetal distress (e.g., abnormal biophysical profile)
pathogenesis
Clinical features

• There is a wide spectrum of presentations of MAS, ranging from severe birth asphyxia requiring active resuscitation through early onset of respiratory distress to a vigorous baby with no major problems.

• Typically the infant is born covered in meconium stained liquor and has meconium staining of the umbilical cord, skin and nails.

• The chest appears to be hyperinflated and there may be a prominent sternum (barrel shaped chest).

• Respiratory distress may be mild initially, becoming rapidly more severe after several hours. If asphyxia has occurred, the baby may also show signs of early-onset encephalopathy.
• Areas of hyperexpansion mixed with patchy densities and atelectasis

• patchy infiltrates, coarse streaking of both lung fields
• Diffuse “ropey” densities
• Patchy areas of atelectasis and emphysema from air-trapping
• Hyperinflation of lungs
• No air bronchograms
Management in the delivery room

- Meconium is present
  - Baby is vigorous or not?
    - Vigorous*: Continue initial steps of resuscitation:
      - Position, clear airway (as necessary)
      - Dry, Stimulate, Reposition
    - Not vigorous: Rapidly Insert an endotracheal tube and suction trachea
In the NICU

• Empty stomach contents to avoid further aspiration.

• Suction frequently & perform chest physiotherapy.

• Maintain an antibiotic coverage (ampicillin & gentamicin).

• Supplemental oxygen

• Consider CPAP, if FiO2 requirements >0.4; however CPAP may aggravate air trapping and must be used cautiously.

• Mechanical ventilation: in severe cases

• Manage PPHN, if present
Pneumonia

• Etiology
• Common: GBS, gram–ve organisms (e.g. *E.Coli, Klebsiella, Pseudomonas*, *Staph. aureus, Staph. epidermidis & Candida*).
• LESS common: acquired viral infections (e.g., HSV, CMV).

• Clinical Manifestations

• prior to delivery (e.g., fetal distress, tachycardia), at delivery (e.g., perinatal asphyxia) or after a few hours (e.g., respiratory distress, shock).

• Early manifestations may be nonspecific (e.g., poor feeding, lethargy, irritability, cyanosis, temperature instability).
• Respiratory distress, cyanosis, apnea & progressive respiratory failure may become evident.

• In preterm infants, these signs may be superimposed upon RDS or BPD.

• Signs of pneumonia (dullness to percussion, change in breath sounds, rales or rhonchi) are difficult to appreciate.
Investigation

- Chest x-rays: infiltrates or effusion
- Work-up for sepsis: CBC with differential & CRP
- Tracheal aspiration & blood culture

Management
- Follow general management rules.
- Initiate ampicillin and gentamicin IV; modify according to culture results and continue therapy for 14 days.
- If there is a fungal infection, an antifungal agent is used.
PNEUMOTHORAX

- presence of air or gas in the pleural cavity between the visceral and parietal pleura, which results in violation of the pleural space.

- May occur spontaneously during delivery, Most common when receiving positive pressure

- Space occupying lesion within the chest displacing lung, and if under tension, compromising venous return

- Frequency: 1-2% in term births
  6% in premature births
Pneumothorax: Clinical Findings

- Presents with non-specific signs of respiratory distress
  - Grunting
  - Flaring
  - Retracting
- Unequal, decreased breath sounds
Treatment

• O2 as needed
  – Nitrogen washout (pneumo contains 21% O2, >75% nitrogen, if lung has 100% O2, nitrogen will diffuse out of pneumothorax)

• Try to avoid positive pressure

• Evacuate as needed by thoracentesis or chest tube
Diaphragmatic hernia

- is when there is an absence of a child's diaphragm, or a hole in the diaphragm.

- This can occur on either the left or right side, but is most common on the left.

- The contents the child's abdomen, including stomach, intestines, liver and spleen may go through the hole and into the chest.

- This prevents the normal development of the lung on that side, and may affect the growth of the other lung.
Clinical presentation

- Signs of respiratory distress
- Abnormal chest development, with one side being larger than the other
- Abdomen that appears caved in (scaphoid)
Treatment

• In the delivery room, if the infant is known or suspected to have congenital diaphragmatic hernia, immediately place a vented orogastric tube and connect it to continuous suction to prevent bowel distension and further lung compression.

• avoid mask ventilation and immediately intubate the trachea.

• Mechanical ventilation strategies are targeted at avoiding high peak inspiratory pressures

• Surgery - When the baby's condition has improved, the diaphragmatic hernia will be repaired with an operation.
Persistent Pulmonary Hypertension of the Newborn

- defined as a failure of normal pulmonary vasculature relaxation at or shortly after birth

- Resulting in impedance to pulmonary blood flow which exceeds systemic vascular resistance that unoxygenated blood is shunted to systemic circulation

- Data suggest that 2-6 cases of PPHN occur per 1000 live births.
Following delivery pulmonary vascular resistance \textbf{DECREASE}, in order to establish pulmonary blood flow.

In kids with PPHN, they never drop their resistance, leading in the worst case to a right to left shunt at the ductal level.
CAUSES

pulmonary vasoconstriction
- MAS
- RDS
- Pneumonia

Pulmonary hypoplasia
- Diaphragmatic hernia

Idiopathic pulmonary hypertension
- Hypoxia
- Cold stress
- Sepsis
- Hypoglycemia
• Onset: 6-12 hrs after birth, may also represent late.

• Cyanosis and respiratory difficulties.

• Hypoxemia out of proportion to degree of distress

• Prominent right ventricle, single and loud S2, and soft regurgitant systolic murmur of tricuspid regurgitation & systemic hypotension
Investigations

- Chest x-ray films: usually normal or demonstrate pulmonary parenchymal disease.
- Echocardiography

- **difference between postductal and preductal oxygenation levels:**

  In PPHN, postductal oxygenation is especially low in contrast to preductal oxygenation.

  A PaO2 difference of 10–20 mm Hg or greater measured with arterial blood gas sampling, or an SaO2 difference of 10% or greater measured with a pulse oximeter, suggests a diagnosis of PPHN.
Treatment

- **Oxygen** -- 100 percent supplemental oxygen may be given to your baby through a mask or plastic hood.

- **Nitric Oxide** -- Research has shown that this gas is effective in treating PPHN because it relaxes contracted lung blood vessels and improves blood flow to the lungs. It is given through the ventilator.

- **High Frequency Oscillatory Ventilation** -- This type of ventilation may improve the oxygen level in the blood if other types of ventilation are not effective.

- **Extracorporeal Membrane Oxygenation** -- In addition, an extracorporeal membrane oxygenation (ECMO) machine may be used for patients who are experiencing serious heart or lung failure. It delivers oxygen to the brain and body as temporary support while the PPHN resolves.
Hyperoxia test

- Obtain ABG → then place the patient on 100% O2 for 10 minutes then repeat the ABG, if the cyanosis is pulmonary PaO2 should be increased by 30 mmHg. If the cause is cardiac there will be minimal improvement of PaO2