Respiratory disorders in neonates **Dr Aliaa Mohammed Radhi**

Learning objectives

- ✓ Important differential diagnosis of respiratory distress in neonatal period
- ✓ Clinical presentation of Respiratory distress
- ✓ Management and possible preventive measure

Case study

1 hr. old male neonate deliver to prim mother at 34 week of gestation present with chest retraction and tachypnea

What is the provisional diagnosis ?

What is the DDx?

What is the line of management ?

Respiratory disorders are the most frequent cause of admission for

neonatal intensive care in both term and preterm infants.

Signs and symptoms of respiratory distress

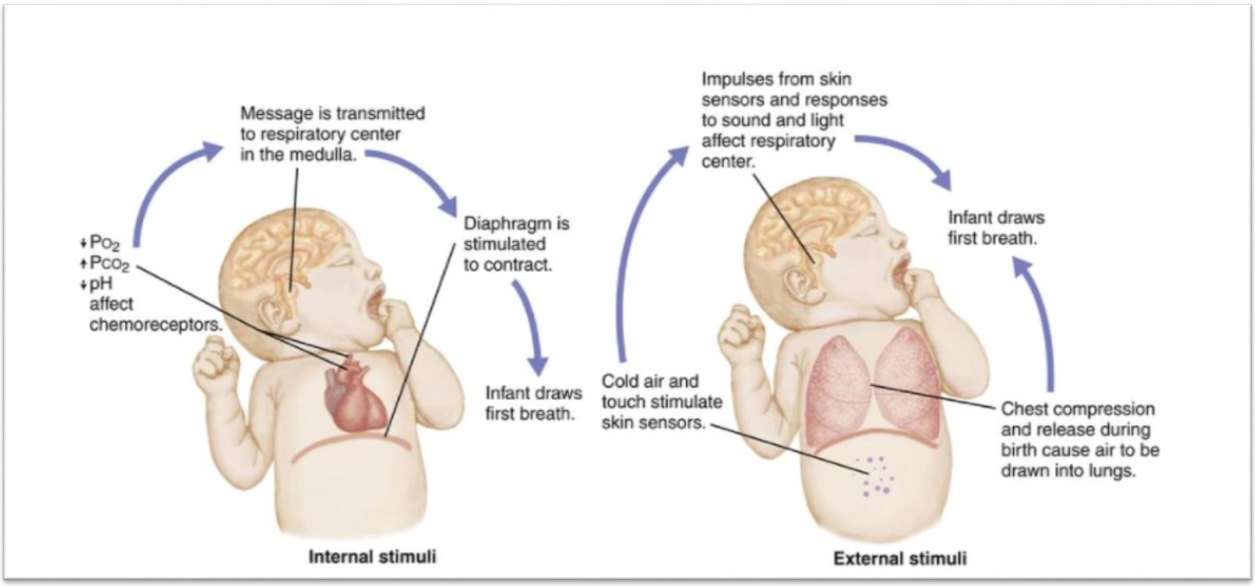
- cyanosis
- grunting
- nasal flaring
- retractions, tachypnea
- decreased breath +-crackles or rhonchi
- Pallor

THE FIRST BREATH

- During vaginal delivery, intermittent compression of the thorax facilitates removal of lung fluid.
- Surfactant lining the alveoli reduce the surface tension, thereby lowering the pressure required to open alveoli.
- infants requiring positive-pressure ventilation at birth need an
- opening pressure of 13-32 cm H2O

- Air entry into the lungs displaces fluid, decreases hydrostatic

pressure in the pulmonary vasculature, and increases pulmonary blood flow.



<u>Fluid removal from the may be impaired</u>

- after cesarean section
- surfactant deficiency,
- Endothelial cell damage,
- Hypoalbuminemia
- high pulmonary venous pressure
- neonatal sedation.

Respiratory Distress Syndrome (Hyaline Membrane Disease)

- Respiratory distress syndrome occurs primarily in premature infants;
- its incidence is inversely related to gestational age and birthweight.
- It occurs in 60-80% of infants <28 wk. of gestational age,
- in 15-30% of those between 32 and 36 wk. of gestational age
- rarely in those >37 wk. of gestational age

Risk factors for RDS

- maternal diabetes
- multiple births
- cesarean delivery
- Precipitous delivery
- asphyxia
- cold stress
- preterm male ,white

Risk of RDS Reduce in

- pregnancies with chronic or pregnancy-associated hypertension
- Prolonged rupture of membranes
- antenatal corticosteroid prophylaxis
- maternal heroin use

ETIOLOGY AND PATHOPHYSIOLOGY

- Surfactant deficiency (decreased production and secretion) is the primary cause of RDS.
- The failure to attain an adequate FRC
- high surface tension which make the lung had a tendency to collapse
- Surfactant is present in high concentrations in fetal lung homogenates by 20 wk of gestation, but it does not reach the surface of the lung until later. It appears in amniotic fluid between 28 and 32 wk of gestation.
- Mature levels of pulmonary surfactant are present usually after 35 wk of gestation.

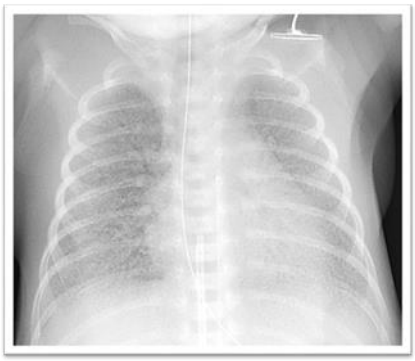
CLINICAL MANIFESTATIONS

- Signs of RDS usually appear within minutes to several hours after birth
- tachypnea, prominent (often audible) grunting,
- Intercostal and subcostal retractions
- nasal flaring, and cyanosis are noted.

- Breath sounds may be normal or diminished with a harsh tubular quality
- on deep inspiration, fine crackles may be heard
- In most cases, the signs reach a peak within 3 then there is gradual improvement

DIAGNOSIS

- **Chest X rays :**
- **initially may be normal finding**
- **then the lung may have a fine reticular granularity of the parenchyma**
- **air bronchograms, which are often more prominent early in the left lower lobe**



Lab finding

- Feature of hypoxemia
- Then , hypercapnia
- variable metabolic acidosis.

PREVENTION

- Avoidance of unnecessary or poorly timed early cesarean section (<39 wk) or induction of labor,
- appropriate management of high-risk pregnancy and labor (including administration of antenatal corticosteroids(Betamethasone and dexamethasone) to the women before 34 week of gestation
- Steroid administration is recommended for all women in preterm labor who are likely to deliver a fetus within 1 wk.

TREATMENT

1) Treatment of infants with RDS is best carried out in the neonatal ICU with monitoring of vital sign and blood gass
2) To avoid hypothermia the infant should be placed in an incubator or radiant warmer,and core temperature maintained between 36.5 and 37°C
3) Calories and fluids should initially be provided intravenously. For the 1st 24 hr, 10% glucose solution with additional amino acids in extremely premature infants, should be infused through a peripheral vein at a rate of 65-75 mL/ kg/24 hr.
4) Warm humidified oxygen should be provided at a concentration initially sufficient to keep arterial oxygen pressure between 50 and70 mm Hg (91-95% saturation)

- CPAP at pressure 5-10 cm H2O if the O2 saturation can not maintain above 90%
- Assisted ventilation and surfactant therapy indicated if

❖ the O2 saturation can not maintained above 90% with CPAP

❖ Infants with respiratory failure or persistent apnea

Feature of respiratory failure

- arterial blood pH <7.20,
- arterial blood Pco2 of 60 mm Hg or higher

- oxygen saturation <90% at oxygen concentrations of40-70% and CPAP of 5-10 cm H2O.

5) Surfactant therapy
   - Surfactant therapy gave by endotracheal tube to neonate who fail CPAP .
   - Repeated dosing is given every 6-12 hr for a total of 2 to 4 doses, depending on the preparation
6) Treat the metabolic acidosis with sodium bicarbonate
7) Antibiotic (ampicillin and gentamicin)

Complications

- complications of **tracheal intubation** are pneumothorax and other air leaks
- complication related to arterial catheterization
- increased risks for development of PDA(patent ductus arteriosus)
- development of BPD (bronchopulmonary dysplasia

Differential diagnosis

- **Transient tachypnea of newborn**
- **early-onset sepsis**
- **Cyanotic heart disease (total anomalous pulmonary)**
- **venous return**
- **pneumonia,**
- **Meconium aspiration syndrome**
- **congenital anomalies (diaphragmatic hernia,**
- **spontaneous pneumothorax**

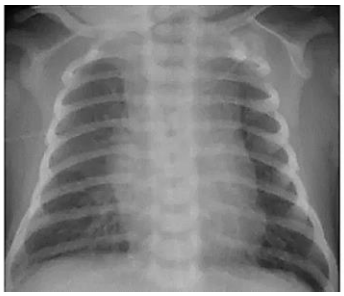
Transient Tachypnea of the Newborn

- Transient tachypnea is most common after term cesarean delivery.
- Clinical feature
- early onset of tachypnea with retraction or expiratory grunting

- occasionally, cyanosis that is relieved by minimal oxygen supplementation (<40%).
- Most infants recover rapidly, usually within 3 days.

Diagnosis

- **chest radiograph shows**
- **prominent pulmonary vascular markings,**
- **fluid in the intralobar fissures,**
- **Hyperinflation**
- **flat diaphragms**
- **rarely, small pleural effusions**



Fetal Aspiration Syndrome

- With fetal distress, infants often initiate vigorous respiratory movements in utero and then the infant may aspirate

❖ amniotic fluid containing vernix caseosa,

❖ epithelial cells

❖ meconium

❖ blood, or material from the birth canal

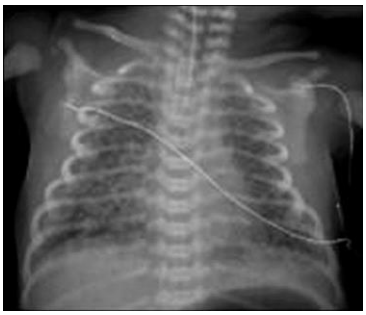
which may block the smallest airways and interfere with alveolar exchange of oxygen and carbon dioxide.

Meconium Aspiration

- Meconium-stained amniotic fluid is found in 10-15% of births

- Meconium aspiration syndrome (MAS) develops in 5% of such infants
- fetal distress and hypoxia occur before the passage of meconium into amniotic fluid
- The infants are meconium stained and may be depressed and require resuscitation at birth.
- Either in utero or with the first breath, thick, particulate meconium is
- aspirated into the lungs and lead to respiratory distress soon after delivery

CXR shows patchy infiltrates, coarse streaking of both lung fields, increased anteroposterior diameter, and flattening of the diaphragm.



Treatment

- Treatment of the MAS includes supportive care and standard management for respiratory distress
- **Prevention**
- rapid identification of fetal distress and initiation of prompt delivery

PROGNOSIS

- The mortality rate of meconium-stained infants is considerably higher than that of nonstained infants
- Residual lung problems are rare, but include symptomatic cough, wheezing, and persistent hyperinflation for up to 5-10 yr.