Neonatology
Respiratory distress in newborn

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Respiratory distress in newborn

- Tachypnea , sternal & intercostal retraction and grunting .
- **Pulmonary disorders**
- **Common** **less common**
- 1 – RDS 1 , TTN
  2 – MAS , Pneumonia
  3 –Pneumothorax .

  1 – Pulmonary hypoplasia
  2 – Upper airway obstruction .
  3 – Rib congenital anomalies .
  4-Diaphragmatic hernia .
  5-Pulmonary hemorrhage .
  6-Immature lung .

Extra-pulmonary disorders

- **Vascular**
- 1 – Persistent pulmonary hypertension.
  2 – Congenital heart diseases .
  3 – Hypovolemia , anemia .
  4 – Polycythemia .

- **Metabolic**
- 1 – Acidosis .
  2 – Hypoglycemia .
  3 – Hypothermia .

- **Neuromuscular**
- 1 – Cerebral hypertension.
  2 – Cerebral hemorrhage .
  3 – Muscular or NMD .
  4 – Spinal cord .
  5 – Phrenic nerve palsy .
  6 – Drugs → morphine , phenobarbitone .

Hyaline membrane disease (HMD)

- **RDS- Type 1** ; it is respiratory disorders that primarily affects preterm infants who are born before the biochemical maturation .Which is persist or progress over the first 48-90 hrs of the life . 30 % of all neonatal deaths are from HMD or its complications .Primarily, it 's the disease of prematurity .

- **Incidence**
- 60-80% < 28 weeks gestation .
  15-30% 32-36 weeks gestation
  5% beyond 37 weeks .

- ……. ↑ in : infant of diabetic mother , delivery before 37 weeks , multi-fetal pregnancy , Cs , precipitous delivery , asphyxia , cold stress , history of prior affected infants , pre-term white male and acidosis .
- ……. ↓ in : chronic hypertension , maternal opiate addiction , prolong rupture of membrane and antenatal corticosteroids .

Etiology ;

- surfactants are synthesized and stored in type ii (2) alveolar cells. Surfactant deficiency is primarily cause of HMD .Failure to develop FRC & tendency of lung to become atlectatic correlates with high surface tension and absence of pulmonary surfactant .

Pathophysiology :

- ** Surfactant is surface active agent that release into alveoli reducing surface tension and maintaining alveolar stability by preventing collapse at the end expiratory phase .
- It is appear in amniotic fluid between 28-32weeks .Mature level of pulmonary surfactants are usually present after 35weeks .
- Lecithin : Sphingomyelin L:S ratio is 2:1 or greater by 35 weeks *

- **↓ synthesis and release of surfactant with small respiratory unit & complaint chest wall produces atelectasis causing perfused not ventilated alveoli→ hypoxia
- ** ↓ lung compliance , ↑ work of breathing and insufficient alveolar ventilation→hypercapnia .
- ** hypoxia , hypercapnia & acidosis → ↑ Rt → lf shunt through foramen ovale and ductusus arteriosus .
- ** ↓ pulmonary blood flow → ischemia → injury to alveolar cells & to vascular bed result in an effusion of proteinous material into alveolar space which is stained typically as Eosinophilic (HMD) → vicious circle .

Prematurity

C.S

Intra-partum asphyxia

familial predisposition

acidosis

↓surfactant

Impaired cellular metabolism

progressive atelectasis

Alveolar hypo perfusion

hypo ventilation

Pulmonary vasoconstriction

↑Pco2 , ↓Po2 , ↓PH

T.T
Neonatal asphyxia
Hypothermia
Apnea

(shock)

Hypotension

Hypovolemia

Factors in the pathogenesis of HMD →Vicious cycle .

- Abnormalities of surfactant & protein genes → lethal familial RDS 1. ***
- *** Other familial respiratory distress not (RDS) : alveolar capillary dysplasia , pulmonary lymphangectasia and mucopolysaccharidosis .

Clinical manifestation

- _ Signs appear within minutes although many not be recognized for several hours in large premature .
- _ Rapid , shallow breathing RR > 60 /min , tachypnea and prominent grunting , inter-costal and sub-costal retraction & nasal flaring & duskiness are noted → cyanosis .
- Breath sound →normal or ↓ , on deep inspiration ; fine rales over lung bases . _
- _ Natural course →progressive cyanosis & dyspnea .If no treated well →↓Bp , ↓ temperature →fatigue- cyanosis , pallor and grunting ↓ or disappears as condition worsens .

- Apnea and irregular respiration are ominous sings .Mixed respiratory and metabolic acidosis.
- Signs of asphyxia due to apnea and respiratory failure . _
- Signs and symptoms peak at 3 days after that will be gradually improved . _
- Death between 2-7 days →air leakage , pulmonary hemorrhage & IVH . _
- Mortality may be delayed weeks or months if CLD develops in infant with sever HMD .

Diagnosis

- CXR : fine reticular granularity →micro atelectasis , Air bronchogram .
- Blood gases & acid base value .
- Hb , serum glucose , sepsis work up , electrolyte Sr. Ca , Echo .

- **D/D**
- 1 – Group B strep sepsis (pneumonia)
- 2 – Cyanotic heart diseases .
- 3 – P.Pulmonary hypertension .
- 4 – Aspiration syndrome .
- 5 – Pneumothorax (spontaneous).
- 6 – Congenital anomalies like : diaphragmatic hernia , lobar emphysema .
- 7 – Transient tachypnea.
- 8 – Diaphragmatic eventration .

Prevention

- **1 – Prevention of prematurity** like avoidance of unnecessary or poorly timed C.s including intra-partum & intra-uterine monitoring to decrease the risk of asphyxia .
- **2 – Administration of Dexamethasone** 48 hr before the delivery of fetus 24-34 weeks .
- **3 – Administration of the first dose of surfactant** into the trachea of symptomatic premature infant immediately after birth or during 1st 24 hrs .

Treatment

- 1 – Temperature maintained between 36.5-37 c .
- 2 –Calories and fluid should be provided intravenously and fluid volume ↑ gradually .
- 3 – Warm humidified oxygen to keep arterial level between 55-70 mmHg (> 90% saturation) through CPAP .If CPAP can't maintain an arterial O2 tension above 50 mmHg while breathing : 70-100% oxygen , assisted ventilation is required .
- 4 – Multi-dose endotracheal instillation of exogenous surfactant to LBW, requiring 30% oxygen and mechanical ventilation

Pharmacological therapies:

- 1 – Vitamin A to infant < 1000 g to ↓ death , BPD, nosocomial sepsis and retinopathy of prematurity .
- 2 – Systemic corticosteroid to infant require continuous respiratory support & reduce the need for re-intubation .
- 3 – Inhaled steroid during 1st 2 weeks without adverse effects of steroid.
- 4 – Inhaled NO for hypoxemic respiratory failure .

- 5 - Sodium bicarbonate depending on the result of acid-base analysis .
- 6 – Dopamine for hypotension and low flow in SVC or using systemic steroids for ↓ blood pressure due to transient adrenal insufficiency .
- 7 – Empirical antibiotics → ampicillin + aminoglycosides .Because difficult to differentiate between group B strep pneumonia (and) from RDS 1 until results of blood cultures are available .

Complications

- 1 – Complications of tracheal intubation →asphyxia , sub-glottic stenosis , edema of larynx .
- 2 – Umbilical arterial catheterization : embolism , thrombosis , spasm and perforation .
- 3 – Umbilical vein catheterization :cardiac perforation , pericardial tomponade .
- 4 – Air leaking disorders .
- 5 – PDA .
- 6 – Chronic lung diseases (BPD)
- 7 – Necrotizing enterocolitis , IVH , Retinopathy of prematurity .

- **Prognosis of HMD :**
- Mortality ↑ with ↓ gestational age .Survivors of sever neonatal respiratory failure may significantly have pulmonary and neuro developmental impairment .

- **** Transient tachypnea 1 newborn :**
- Type 2 RDS – due to ↓ lymphatic absorption of fetal lung fluid .It follows uneventful normal preterm or term vaginally delivered or C.s .

- Early tachypnea with retraction & expiratory grunting and occasionally cyanosis that relived by minimal O2 < 40% .Patient recovered rapidly within 3 days.
- Risk factors of T.T : C.s , male , macrosomia , excessive maternal sedation , prolonged labour , negative amniotic fluid phosphtidyl glyceride , low Apgar score < 7 at 1 minute .

Clinically :

- The baby not severely ill .
- Radiologically : absence of reticulo-glandular pattern or air bronchogram .
- CXR : show prominent perihilar streaking ↑ lung volume (over inflation) fluid in the minor fissure and perhaps in the pleural space .
- Refractory hypoxia → need ECMO due to pulmonary hypertension ; called malignant TTN.Hypoxemia , hypercapnia ,and acidosis are uncommon