PERINATAL ASPHYXIA

Perinatal asphyxia

Hypoxic Ischemic Encephalopathy is a condition of impaired blood gas exchange during intrapartum period that if persists leads to progressive hypoxemia, Hypercapnea and metabolic acidosis

It is one of the causes of

Neonatal Encephalopathy, A disturbance of neurological function demonstrated by

- difficulty in maintaining respiration.
- hypotonia
- altered level of consciousness,
- poor reflexes and feeding
- seizures

HIE is an important cause of permanent damage to neuronal tissues and neuronal death resulting in

- Neonatal death 15-20%
- Permanent sequelae (Cerebral Palsy and Mental Retardation) 25-30%

Etiology

- **Interruption of Umbilical circulation** (tight nuchal cord, cord prolapse)
- **Impaired placental perfusion**: Maternal Hypotension, Hypertension, abruptio placentae, abnormal uterine contractions
- **Impaired maternal oxygenation**: anemia, CardioPulmonary disease
- **Impaired fetal perfusion**: fetomaternal Hmg
- **Maternal infection**

Etiology

- Placental insufficiency---chronic hypoxia---IUGR---uterine contractions further depress circulation ---asphyxia

After birth

- Failure of oxygenation due to CHD or Pulmonary disease
- Severe anemia : Hemorrhage or hemolysis
- Shock due to sepsis blood loss intracranial or adrenal hmg

Pathophysiology

- Hypoxia and Ischemia-----anaerobic metabolism----accumulation of lactate and inorganic phosphates
- Excitatory and toxic amino acids particularly glutamate accumulate in the damaged tissues
- Increased intracellular Na and Ca result in tissue swelling, cell injury and cerebral edema
- Production of free radicals and nitric oxide

Pathophysiology

- The initial circulatory response is increased shunting through ductus venosus, ductus arteriosus and foramen ovale----with
- transient maintenance of circulation in brain, heart and adrenals in preference to lungs, liver, kidneys and intestine

Pathophysiology

Prolonged intrapartum hypoxia leads to

- PVL,
- Pulm. Hypertension,
- meconium aspiration,
- cortical neuronal damage, (cortical atrophy) multiple infarcts (seizures, focal deficits), status marmoratus of basal ganglia

Clinical Manifestations

CNS (NN Encephalopathy) Seizures, abnormal respiratory pattern, apnea, posturing and movement disorders and jitteriness

- Stage I: Hyperalertness and strong moros reflexes, clonus,no seizures normal EEG lasting less than 24 h
- Stage II: Obtundation, hypotonia, decreased body movements or seizures. EEG Low voltage changing to seizure activity
- Stage III: flaccid, stuporous, seizures, poor brain stem reflexes. EEG burst suppression or isoelectric

Clinical Manifestations

Multi-organ system involvement

- **CVS: Myocardial ischemia Cardiac arrest, tricuspid insufficiency hypotension shock**
- **Pulmonary:RDS, Hmg, Pulm Hypertension**
- **RenalOliguria, ATN, Cortical necrosis renal failure**
- **GIT: Hmg, ulceration perforation NEC**

Clinical Manifestations

- **Adrenal: Hemmorhage**
- **Metabolic: SIADH, Hypoglycemia, hypocalcemia, myoglobinuria**
- **Integument: subcutaneous fat necrosis**
- **Hematological: DIC**
- **Hepatic: Increased gama GT, indirect bilirubin**

Diagnosis

Identification of fetal Hypoxia

- IUGR and increased vascular resistance
- Fetal bradycardia, and loss of beat to beat variation
- Type II decelerations
- Fetal scalp blood analysis; pH<7.0
- Meconium stained liqour

At Birth:

- Failure to breath spontaneously and depressed

Diagnosis

- **CT scan brain**: Focal hemorrhagic lesions, cortical and basal ganglial injury
- **MRI**
- **Amplitude integrated EEG:** used to determin which infants are at a highest risk of significant brain injury. Identifies subclinical seizure activity
- **Cranial USG**

Management

- Prevention
- Immediate resuscitation
- Adequate ventilation; assisted ventilation may be used to maintain physiological levels of PCO2
- Adequate oxygenation Po2 >40 in preterm >50 in term
- Perfusion ; Inotropic support, volume expanders

- Correct Metabolic acidosis
- Correct hypoglycemia
- Control of seizures Phenobarbital, diazepam, lorazepam, phenytoin
- Prevent Cerebral edema: Avoid fluid overload 60ml/kg

Potential new therapies

Aimed at preventing delayed neuronal death following asphyxial insult. A widow period of 6-12 hrs during which neuroprotective strategies could reduce brain damage

- Resuscitation in room air
- Cerebral cooling
- Magnesium
- Allopurinol, inhibitors of nitric oxide production, Calcium channel blockers and neurotropins


THANKYOU !