

Birth injury

1

DR.MOHAMMAD MAHIR

Birth injury

2

- It is avoidable and non-avoidable injury occur during labor and/or delivery it is either hypoxic ischemic injury or mechanical injury .
- Predisposing factors including : macrosomia , prematurity , cephalopelvic disproportion , dystocia , prolonged labor and breech presentation .

Hypoxic – Ischemic injury

3

- Hypoxia is decrease of arterial oxygen concentration ; anoxia is a term used to indicate the consequences of complete lack of O₂ , while ischemia is a decrease of blood supply to the cell or organ .
- Asphyxia is a clinical condition consider if fetal acidosis (ph 7.0) , 5 min apgar score 0-3 with hypoxic ischemic encephalopathy (HIE) & other multiple organ signs .

- HIE is an important cause of preminent damage to CNS tissue that may result in neonatal death or manifest later as CP or developmental delay .

Etiology :

5

- Fetal hypoxia :
- 1- maternal hypoxia* : respiratory and heart disease , anesthesia etc ..
- 2- low maternal BP e.g spinal anesthesia , gravid uterus pressure on blood supply .
- 3- excessive uterine contraction by oxytocin .
- 4- cocaine use* causing vasoconstriction of uterine vessels .
- 5- placental insufficiency* .
- 6- Premature separation of placenta .
- 7- cord prolapse or knotting .

- After delivery :
- 1- breathing problems e.g narcotic use , sever pulmonary disease .
- 2-anemia or shock : bleeding , hemolysis , sepsis
- *these may cause acute fetal distress at birth or chronic fetal distress in utero .

Pathophysiology :

7

- Within minutes of the onset of total hypoxia there is increase of catecholamine secretion leading to transient increase of HR & BP then decrease ; while the RR is decreasing from the start as a response to hypoxia , the metabolic and respiratory acidosis will develop , there will be shunting of the blood to the vital organs (brain , heart and adrenal) with transient maintenance of perfusion in preference to the lung , liver , kidneys , intestine & skin .

- On pathology many organs will be affected by early congestion , fluid leak due to increase vascular permeability and endothelial cell swelling then may lead to necrosis and cell death .
- Increase anaerobic metabolism will increase lactate and inorganic phosphate , excitatory toxic aminoacid e.g. glutamate accumulate in tissue
- Increase Na and Ca intracellular leading to tissue swelling and cerebral edema also increase nitric oxide and free radicals in tissue

- CNS : encephalopathy , hemorrhage , infection and edema .
- Heart : cardiomyopathy and infarction .
- Respiratory : PHT , RDS and hemorrhage .
- GIT : ulcer and bleeding .
- Renal : tubular and cortical necrosis .
- Adrenal : hemorrhage .
- Hematology : DIC .
- Metabolic : hypocalcaemia , hypoglycemia and SIADH .
- If fetal distress producing gasping , amniotic fluid content (Meconium , lanugo ...) are aspirated to the trachea or lung .

Clinical manifestation :

10

- IUGR with increase vascular resistance may be the first indication of chronic fetal hypoxia , although IUGR may develop in chronically hypoxic fetuses without traditional signs of fetal distress .
- During labor , the fetal HR slows , and beat-beat variability decline and fetal scalp blood analysis may show a PH < 7.20 . particularly in infant near term , these signs should lead to the administration of high concentration of O₂ to the mother and immediate delivery to avoid fetal death or CNS damage .

- At delivery , the presence of yellow , meconium stained amniotic fluid is evidence that fetal distress has occurred .
- At birth the baby frequently appears depressed with failure to response to stimuli , failure to take breathing , pallor , cyanosis bradycardia with hypotonia (which changes later to hypertonia or normal tone) cerebral edema will be followed within 24 hr leading to more brain stem depression , during this time seizure activity may occur , it may be sever and refractory to usual doses of anticonvulsants .

- In addition to CNS dysfunction , other systems problems may be associated secondary to inadequate perfusion (as mentioned above) .
 - After delivery , the severity of neonatal encephalopathy depend on duration and timing of injury . symptoms develop over a series of days , make it important to perform serial neurological examination . during initial hours after the insult , infant have depressed level of consciousness with apnea or bradycardia ; seizures are common with extensive injury . hypotonia is also common as an early manifestation .

Diagnosis

13

- Diffusion –weighted MRI will show loss of signal in posterior limb of internal capsule and increase signal of basal ganglia and thalamus .
- Ultrasonography preferred modality in evaluation of the preterm infant .

Treatment

14

- Supportive measures :
- If the baby has meconium staining , you must aspirate all meconium material from the lung (by intubation) & trachea before any stimulation of breathing as soon as possible , it is matter of few minutes .
- Start resuscitation by ABC .

- Control of fit : phenobarbital is the drug of choice , is given with an IV loading dose (20 mg/kg) additional dose 5-10 mg/kg (up to 40-50mg/kg total) may be needed . phenytoin (20 mg/kg loading dose) or lorazepam (0.1 mg/kg) may be needed for refractor seizures . though most often the result of the hypoxic – ischemic encephalopathy , seizures in asphyxiated neoborn may also be due to hypocalcaemia , hypoglycemia or infection.

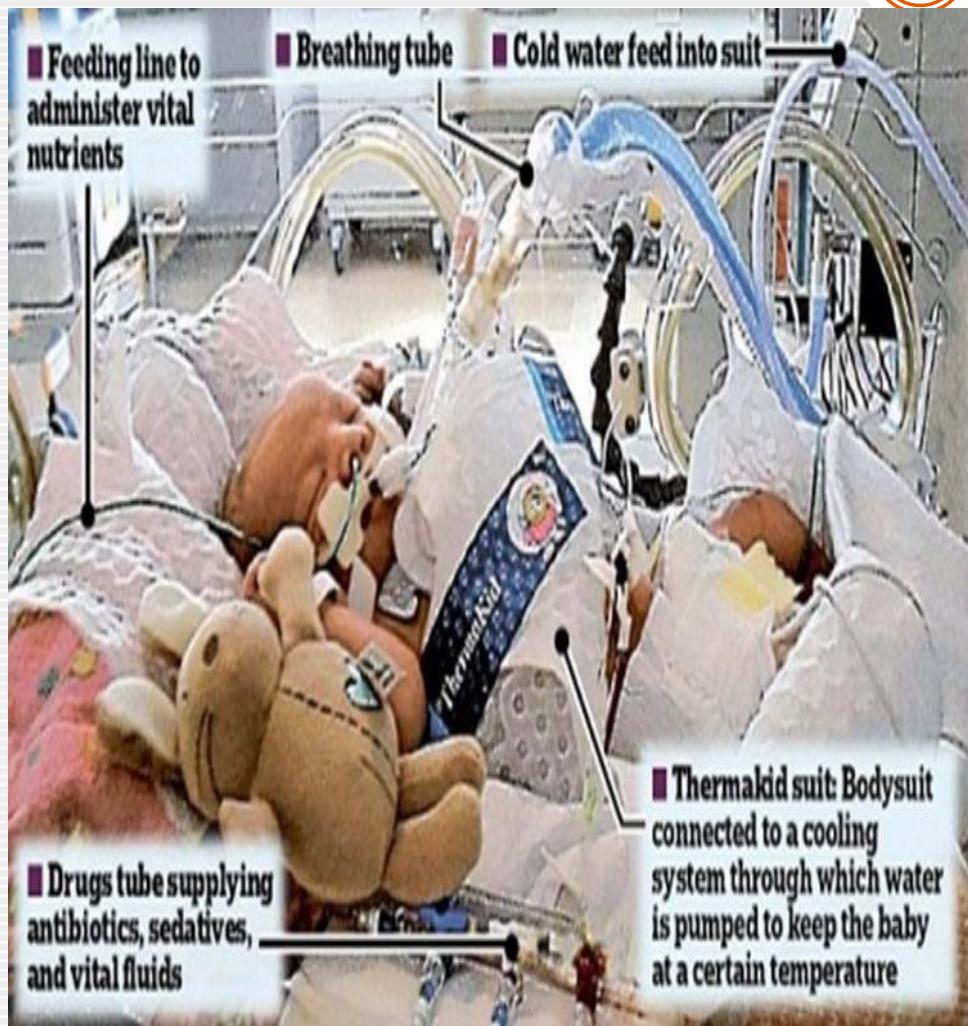
- Treat other associated problems accordingly :
- Selective cerebral or whole body therapeutic hypothermia reduce mortality and major neurodevelopmental impairment.
- Cerebral or systemic cooling to a core temp. Of 35.5°C with in the 1st six hrs .
- Hypothermia will decrease apoptoss and suppress production of mediator

Prognosis :

17

- The prognosis varies depending on whether the metabolic and cardiopulmonary complications are treated , the infant's gestational age (outcome is poorest if the infant is preterm) and the severity of the encephalopathy . sever encephalopathy characterized by flaccid coma , apnea , absent of oculocephalic reflexes , and refractory seizure is associated with poor prognosis .

- fifteen to 20% on infant with hypoxic – ischemic encephalopathy (HIE) die in the neonatal period , and 25-30% of survivor are left with permanent neurodevelopmental abnormalities (cerebral palsy , MR) .
- The combination of chronic fetal hypoxia and acute hypoxic ischemic injury after birth results in gestational age-specific neuropathy . term infants demonstrate neuronal necrosis of the cortex (later , cortical atrophy) while pre-term infants demonstrate PVL-periventricular leukomalacia (later spastic diplegia) and IVH-intraventricular hemorrhage .



Mechanical birth injuries (birth trauma)

Mechanical birth injuries (birth trauma)

21

- **Cranial injuries :**
- **Subconjunctival , retinal hemorrhage** and petechiae of the skin of the head and neck are probably secondary to sudden increase in the intrathoracic pressure during the passage of the chest through the birth canal .
- Parent should be assured that these hemorrhages are temporary and the result of normal event of delivery . they resolve within the 1st 2 Wk of life .

• Caput seccedeneum

22

- It is a diffuse , sometimes ecchymotic , edematous swelling of the soft tissues of the scalp involving the area presenting during the vertex delivery . it may extends across suture lines . the edema disappears within the first few days of life . molding of the head and overriding of the parietal bones are frequently associated . no specific treatment is needed .

• Cephalohematoma

23

- It is a subperiosteal hemorrhage , hence always limited to the surface of one bone , doesn't cross the suture & midline . no discoloration of the overlying scalp occurs and swelling is not usually visible for several hr after birth because subperiosteal bleeding is a slow process . the lesion becomes a firm tense mass with a palpable rim . an underlying skull fracture usually linear and not depressed , coagulopathy and intracranial hemorrhage may be associated . most cephalohematomas are resorbed within 2 wk – 3 months .

- depending on their size . they may begin to calcify and remain for many years as bony protuberance cephalohematomas require no treatment , although phototherapy may be necessary to treat hyperbilirubinemia . incision and drainage are contraindicated because the risk of introducing infection in a benign condition . massive hematoma may need blood transfusion . cranial meningocele may be differentiated from cephalohematoma by pulsation , increase pressure on crying and roentgenographic evidence of bony defect .

Peripheral nerve injury :

25

- **Brachial palsy**
- Brachial pexus injury is a common problem , may cause paralysis of the upper part of the arm (Erb-Duchenne paralysis) with or without paralysis of the forearm or hand (Klumpke paralysis) or more commonly paralysis of the entire arm . these injuries occur in macrosomic infants and when lateral traction is exerted to the head and neck during delivery of the shoulder in a vertex presentation , when the arms are extended over the head in breech presentation or when excessive traction is placed on the shoulders .

Erb-Duchenne paralysis

26

- The injury is limited to the 5th and 6th cervical nerves . the infant loses the power to abduct the arm from the shoulder , rotate the arm externally and supinate the forearm . the characteristic position consist of adduction and internal rotation of the arm and pronation of the forearm (this position is called policeman hand) power to extend the forearm is retained but the biceps reflex is absent , the Moro reflex is absent on the affected side . power in the forearm and the hand grasp is preserved unless the lower part of the plexus is also injured . the presence of the hand grasp is favorable prognostic sign . When the injury include the phrenic nerve , alteration in the diaphragmatic excursion may be observed fluoroscopically .

Klumpke paralysis

27

- It is a rare form of the brachial palsy , injury to the 7th and 8th cervical nerves and the 1st thoracic nerve produces a paralyzed hand (with loss of grasp reflex , while the moro reflex is maintained and ipsilateral ptosis and miosis (Horner syndrome) if the sympathetic fibers of the 1st thoracic root are also injured .

- **Differential diagnosis**
- Differentiation of the brachial palsy at birth must be made from cerebral injury , from fracture , dislocation and epiphyseal separation of the humerus , and from fracture of the clavicle . MRI demonstrate nerve root rupture or avulsion .

•prognosis

- full recovery occurs in most patients , the prognosis depending on whether the nerve was compressed , merely injured , or was lacerated .
- if the paralysis was due to edema and hemorrhage about the nerve fibers , function should return within few months ; in due to laceration , permanent damage may result . in general , paralysis of the upper part of the arm has a better prognosis than paralysis of the lower part .

- **Treatment :**
- It consist of : (1) partial immobilization and appropriate positioning to prevent the development of contractures . in upper arm paralysis , the arm should be abducted 90 degrees with external rotation at the shoulder , full supination of the forearm and slight extention at the wrist with the palm turn on the face . this position may be achieved with a brace or splint during 1st 1-2 wk . immobilization should be intermittent through the day while the infant is asleep and between feeding .

- in lower arm and hand paralysis , the wrist should be splinted in the neutral position and padding placed in the fist . when the entire are is paralyzed , the same treatment principle should be followed . (2) gentle massage and range-of-motion exercises may be started by 7-10 days of age . infants should be closely monitored with active and passive corrective exercise .

- If the paralysis persists without improvement for 3-6 mon with such above therapy , a lacerated nerve should be suspected , so neuroplast , end-end anastomosis and nerve grafting for partial recovery .

Facial palsy

33

- It is usually peripheral paralysis that result from pressure over the facial nerve inutero , from effort during labor or from forceps used during delivery .
- Rarely it may result from nuclear agenesis of facial nerve (non-obstetric central cause) .

- Peripheral paralysis is flaccid and when complete involve the entire side of the face including the forehead . on the affected side the forehead is smooth , the eye can not be closed , the nasolabial fold is absent and the corner of the mouth droops when the infant cries , movement occur on the non-paralysed side of the face and the mouth drawn to that side . while in the central paralysis the forehead wrinkles on the affected side because only the lower of the face is involved and the infant also usually has other manifestations of intracranial injury , most commonly 6th nerve palsy .

- The prognosis of the peripheral palsy depend on whether the nerve was injured by pressure or whether the nerve fibers were torn . improvement occur within a few weeks in the former instance . care of the exposed eye is essential . neuroplasty may be indicated when the paralysis is persistent .
- Facial palsy may be confused with absence of depressor muscles of the mouth which is a benign problem .
- Other peripheral nerves are seldom injured in utero or at birth except when they are involved in fractures or hemorrhage .