Chapter 9

POST - RESUSCITATION MANAGEMENT OF AN ASPHYXIATED NEONATE

Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal morbidity and mortality. Perinatal asphyxia is an insult to the fetus or the newborn due to lack of oxygen (hypoxia) and/or a lack of perfusion (ischemia) to various organs. It ranks as the second most important cause of neonatal death after infections, accounting for around 30 % mortality worldwide. In India, between 250,000 to 350,000 infants die each year due to birth asphyxia, mostly within the first three days of life.

Learning objectives
After completion of this module the participant should be able to -
- Perform initial stabilization and management of an asphyxiated neonate
- Monitor an asphyxiated neonate
- Recognize poor prognostic factors in asphyxia

Clinical presentation
- Perinatal asphyxia may result in adverse effects on all major body systems including the kidney, brain, heart and lungs. The clinical features in asphyxiated babies range from mild to severe impairment.
- The extent of multi-organ dysfunction determines the early outcome of an asphyxiated neonate.
- The most severely affected babies may manifest with stupor or coma, periodic breathing or irregular respiration, hypotonia and loss of complex reflexes like Moro’s and sucking.
- About 50 % of the moderate to severely asphyxiated babies may have seizures.
- Severely affected babies may have progressive deterioration of the CNS function in terms of decreasing tone, increasing degree of coma and prolonged apneas over the next 48 hours. These neonates would eventually die or have permanent neurologic sequelae.

Initial stabilization and management
The management consists of supportive care to maintain temperature, perfusion, ventilation and a normal metabolic state including glucose, calcium and acid-base balance. Early detection by clinical and biochemical monitoring and prompt management of complications must be done to prevent extension of cerebral injury.
- **Temperature:** Baby should be placed under a radiant warmer. The temperature should be maintained in the normal range of 36.5 – 37.5 °C as hypothermia imposes additional stress to the baby by increasing the metabolic needs. This may
lead to acidosis, myocardial depression, hypotension, bleeding tendency and pulmonary hemorrhage. Hyperthermia is detrimental and should be avoided.

- **Oxygenation** should be kept in the normal range by monitoring oxygen saturation by pulse oximetry, if facilities exist. SpO2 should be maintained between 88-92 %. Hypoxia should be treated with O2 and if required ventilation. Hyperoxia should always be avoided.

- **Blood glucose** level should be kept at 75-100 mg/dl to provide adequate substrate for the brain. If the baby is hypoglycemic, treat appropriately. The baby should be on 60mL/kg/day of 10% Dextrose.

- **Calcium** level should be kept in the normal range and serum calcium should be maintained between 9-11 mg/dl. Hypocalcaemia is a common metabolic alteration and should be monitored within the first 24 hours. If hypocalcemic (<9mg/dL), give IV 10% Calcium gluconate @ 2mL/kg diluted 1:1 with 5% Dextrose under heart rate monitoring. Infuse at a rate of 1ml/min. Withhold infusion if HR <100/min.

- **Vitamin K** 1mg IM must be administered to all those babies who have not received Vit K at birth.

- **Blood Pressure**: It is important to maintain cerebral perfusion pressure (CPP) within a narrow range to optimally perfuse the brain. Cerebral perfusion entirely reflects systemic BP in a pressure passive fashion. Hence, to maintain cerebral perfusion, a systemic mean arterial BP of at least
  - 45 to 50 mm of Hg is desirable for term infants,
  - 35-40 mm for infants weighing 1000-2000 g and
  - 30-35 mm for infants <1000 g.

If blood pressure cannot be measured, assess CFT and pulse volume. Pressor agents like dopamine should be used if there is evidence of poor perfusion. Frequent boluses of fluids should be minimized. Volume replacement should be done slowly. If the infant is well hydrated and urine output is normal (>1 ml/kg/hour) intravenous fluids should be provided as per the day’s requirement. (Table 6.1)

- **Seizures**: Seizures should be controlled with phenobarbitone in a loading dose 20 mg/kg which should be given slowly at the rate of 1 mg/kg/min intravenously. The bolus should be followed by maintenance dose of 3-4 mg/kg/day. Additional boluses of 10 mg/ kg (maximum two) may be administered if seizures continue or recur. One should always be vigilant for respiratory depression and/or cardiovascular compromise with hypotension during administration of the drug. If seizures persist, phenytoin may be administered slowly as a second drug (20 mg/kg intravenously as the loading dose followed by 4 to 8 mg/kg/day as a
maintenance dose). Before starting anticonvulsants one should ascertain that metabolic derangements that may complicate asphyxia and cause seizures (hypoglycemia, hypocalcaemia, hyponatremia) have been managed. (Refer to chapter on seizures).

- **Feeding:** As soon as the baby is hemodynamically stable, there is no abdominal distension and the baby has passed meconium, start enteral feeds with expressed breast milk (EBM) @ 30ml/kg and increase daily by 20-30 ml/kg/day or more as the baby tolerates.

**Monitoring**

**Clinical monitoring**
- All neonates who have suffered asphyxia must be closely monitored clinically as well as by performing certain bedside tests.
- The respiratory status must be monitored by meticulous record of the respiratory score every 2-3 hours.
- The CVS status assessment should include HR, color, CFT, Pulse oximetry and Non invasive blood pressure (NIBP).
- The abdominal circumference should be recorded to rule out any ileus due to gut ischemia.
- The urine output should be measured as it is a direct indicator of the state of perfusion. Moreover, this entity is also used as a prognostic sign and the outcome is uniformly poor if the output remains <1ml/kg/hr beyond 36 hrs of life.

**Biochemical monitoring**
- The biochemical monitoring should aim at measuring the blood sugar by Dextrostix, the hematocrit, serum electrolytes (Na, K), serum calcium and blood urea and creatinine.

**Poor prognostic factors**
The presence of one or more of the following features has been found to be a predictor of poor neurodevelopmental outcome in the long term. These are:

1. Failure to establish respiration by 5 minutes of life
2. Apgar score of 3 or less at 5 minutes
3. Onset of seizures with in 12 hours
4. Refractory seizures
5. Severe HIE (See Levene Chart in Annexure )
6. Persistent oliguria ( <1 ml/kg/hr) for the first 36 hrs of life
7. Inability to establish oral feeds by 1 wk

**Prevention of asphyxia**
- Some cases of asphyxia can be prevented by better antenatal and intranatal management of high risk cases. The approach should be regular antenatal check ups to detect high risk cases and adoption of an ‘at risk approach’ to anticipate complications so that timely intervention in terms of emergency LSCS can be
instituted. An alternative strategy for the not so well equipped centers can be appropriate timely referral of the pregnant woman to a well equipped neonatal and obstetric centre with active surveillance and timely delivery. Prompt and efficient resuscitation followed by management of neonatal complications in a level III NICU should go a long way in preventing some of the morbidity and mortality related to asphyxia.
EVALUATION

1. Outline steps of initial stabilization and management of an asphyxiated neonate

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2. Enumerate steps of monitoring of an asphyxiated neonate

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3. Write management of seizures in an asphyxiated neonate

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4. Enumerate poor prognostic factors in asphyxia

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5. Mention the strategies for prevention of perinatal asphyxia.

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