INTRODUCTION

Neonatal encephalopathy is characterized by difficult in initiating and maintaining respirations, depression of reflexes, altered levels of consciousness and often seizures ***(Shah et al., 2004).***

The world Federation of Neurology Group defines asphyxia as 'a condition of impaired blood gas exchange leading, if it persists, to progressive hypoxemia and hypercapnia ***(Michelle et al., 2006).***

Perinatal hypoxic-ischemic encephalopathy 'HIE' occurs as a result of hypoxia and/or ischemia results during labour and delivery. The reported incidence of HIE in full term infants is 2.9 – 9.0 for 1000 deliveries. The syndrome leaves significant handicaps in 100% of survivors of severe HIE and in 20% of survivors of moderate HIE ***(Hiroyuki Ichiba et al., 2002).***

Perinatal asphyxia is one of the leading causes of perinatal death and a recognized cause of neuromotor disability ***(Gathwala et al., 2006).***

Severe perinatal hypoxia and ischemia lead to brain damage of the newborn and remains a frequent cause of death and adverse outcome ***(Charlotte et al., 2006).***

Early identification of neonates with perinatal asphyxia who are at risk for hypoxic ischemic encephalopathy (HIE) is crucial because of the cascade of biochemical events (i.,e energy failure acidosis, free radical formation, calcium accumulation, lipid peroxidation, and neurotoxic

reactions to glutamate and nitric oxide) that eventually may lead to neurotoxic reactions to glutamate and nitric oxide) that eventually may lead to neuronal necrosis and/or apoptosis ***(Mari et al., 2002)***.

Asphyxia was diagnosed if they met at least three of the following criteria:-

- Signs of intrauterine asphyxia, as indicated by late decelerations on fetal monitoring or meconium stained liquor.
- Arterial cord blood pH < 7.10.
- Delayed onset of spontaneous respiration.
- Apgar score of < 5 at five minutes.
- Multiorgan failure

(Van Rooij et al., 2005).

HIC is classified as, mild, moderate or severe HIE according to the criteria of Sarnat and Sarnat, ***(Sarnat and Sarnat, 1976)***. Mild HIE is characterized by hyper-alertness and irritability, normal muscle tone, normal or hyperactive reflexes, ankle clonus and no seizures. Moderate HIE includes lethargy, decreased spontaneous movements, proximal muscular weakness, depressed primitive reflexes and seizures. Severe HIE includes stupor or coma, markedly reduced muscle tone or flaccidity and absent primitive reflexes. Seizures are often frequent and may be difficult to control, but may also be totally absent.

The financial and social burden of hypoxic-ischaemic brain injury in the newborn is huge because of the large number of quality life years lost and the severity of disability ***(Marianne and Andrew, 2005).***

To develop effective preventive measure, early identification of babies at high risk for brain injury is necessary. Although several methods (scoring systems, markers, EEG, cerebral function monitoring, etc.) are developed for early identification of neonates who may benefit from intervention, these indexes are reported to have a limited predictive value for death or survival with abnormal neurodevelopmental outcome ***(Greisen et al., 1997).***

Reactions involving free radical toxicity in neonates have a high potential for tissue damage, and particularly brain damage because fast-growing tissues are especially sensitive to free radicals ***(Buonocore et al., 2002a).***

Free radicals may be generated by several sources including phagocyte activation catecholamine metabolism, mitochondrial dysfunction, arachidonic acid cascade and Fenton's reaction driven by non protein bound iron (NPBI). Indirect markers of increased free radical release and perinatal brain injury have recently emerged with reports of increased NPBI in erythrocytes and plasma of hypoxic newborns ***(Buonocore et al., 2001).***

Recent studies have demonstrated the key role of NPBI and free radicals in the development of hypoxic-ischaemic brain damage ***(Giuspeepe Buonocore et al., 2003).***

Some studies have demonstrated a correlation between the degree of acidosis and the neonatal neurological outcome. So measurement of lactate and base defect is variable in the diagnosis and short term prognosis of intrapartum asphyxia in term neonates ***(Silva et al., 2000).***

AIM OF THE WORK

To study the level of non protein bound iron, lactate and pH in serum of asphyxiated neonates, correlating the level with degree of encephalopathy to predict the outcome of HIE cases.