

## **SUMMARY AND CONCLUSION**

perinatal asphyxia continues to be a major cause of neonatal morbidity, and neurodevelopmental disabilities.

neonatal encephalopathy is characterized by difficulty in initiating and maintaining respiration, depression of reflexes, altered level of consciousness and often seizures.

the early prediction of outcome is necessary to identify infants with a higher risk for brain damage for neuroprotective interventions aiming to limit the extent of brain injury at high risk for brain injury is necessary.

Although several methods “scoring system 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 outcomes.

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.

Indirect markers of increased free radical release and perinatal brain injury have recently emerged with reports of increased NPBI in plasma of hypoxic newborns.

There is a correlation between the degree of acidosis and the neurological outcome so measurements of lactate is variable in the diagnoses and short term prognosis of intrapartum asphyxia in term neonates.

Our study included 25 term neonates who met the flowing criteria of perinatal asphyxia; evidence of fetal distress “fetal heart rate abnormalities and / or meconium staining”, arterial blood cord PH values  $< 7.2$ , apgar score after 5 minutes  $< 5$ .

We evaluate the frequency and spectrum of seyerity of multisystem dysfunction after perinatal asphyxia. we found that C.N.S was the system most frequently affected and that sever C.N.S involvement was asociated with moderate or sever involvement of other organs mainly the lung and kidneys. C.N.S was affected in (25) (100%) cases. the stage of encephalopathy was assessed according to Sarnat and Sarnat stages of hypoxic ischemic encephalopathy. 10 cases had stage I, 7 cases had stage II, 8 cases had sage III.

In our work we recognized the lower the level of apgar score at I and 5 minutes, the higher the incidence of seizure, mental retardation.

Also in our work we found an association between metabolic acidosis and increase the risk of complication in C.N.S, C.V.S, R.S and kidneys.

Also in our work we found an association between serum lactate level and the degree of asphyxia and handicaps

In our study we measured the level of NPBI in the asphyxiated (25 cases) and healthy control (25 cases) and found that levels were significantly elevated in asphyxiated newborns compared with healthy newborns.

We found a negative correlation coefficient between level of NPBI and pH, Na<sup>+</sup>, Ca<sup>+</sup>, apgar at 1 minute, apgar at 5 minute and PO<sub>2</sub> as increase level of NPBI is associated with decrease the level of them. We found a positive correlation coefficient between the level NPBI and Hco<sub>3</sub>, urea, creatinin. PCO<sub>2</sub>, lactate and K as increase the level of NPBI is associated with increase the level of them.

In our study we found that all cases with elevated NPBI had delayed outcome according to Denver developmental scoring test (DDST).

We conclude that NPBI is a positive predictor marker to diagnose the degree of asphyxia and to diagnose abnormal outcome of the asphyxiated cases. Also there is a correlation between presence of low apgar score at birth and presences of seizure, high lactate level, acidosis and the outcome of asphyxiated newborns.