Summary and Conclusion

Perinatal asphyxia is a major cause of neonatal mortality and irreversible damage to the brain. Severe asphyxia may induce major deficit shortly after birth, while mild to moderate asphyxial episodes may result in cognitive and attentive disorders later on in development.

There is an urgent need to better understand its pathophysiology and to identify as early as possible reliable indices of brain injury in the asphyxic newborns to apply potential therapeutic interventions at the optimal time and to identify those infants at high risk for developmental delays and disabilities.

The syndrome of HIE is considered a reflection of a cascade of biochemical events leading to Ca^{+2} influx via (NMDA) channel increasing the intracellular Ca^{+2} concentration which is the key mechanism of cell death in asphyxiated infants.

(Mg^{+2}) ions as one of the inotropic receptors of this channel, it gates the channel, thus prevents Ca^{+2} influx and therefore prevents cell damage. So it is considered to be neuroprotective against perinatal hypoxic – ischemic injury. Also the use of NMDA receptor channel antagonist or Ca channel antagonists as blockers for Ca influx acts to prevent neuronal injury.

The aim of our study was to investigate the total serum Mg level and the ionized serum Ca^{+2} level in term infants with HIE correlating the results with the severity of HIE and asphyxia (Sarnat and Sarnat staging).

Our study was conducted on 50 term babies with hypoxic – ischemic encephalopathy and 30 healthy term babies in their first two

days of life were enrolled in the study as control group. All were subjected to full clinical examination and laboratory investigations:

-Blood gases, CBC, ALT, AST, Urea, creat, Na^{+}, K^{+}

-Ionized serum Ca^{+2} and total serum Mg.

The neonates in the present study were classified into two groups:-

-Group I (patient group):

This group included fifty term babies with perinatal Asphyxia and they were (26 Males and 24 Females).

With mean gestational age (38±5) weeks, and mean birth weight (3.3±0.5) Kg.

And this group further classified as grade I, grade II and grade III according severity of the disease.

-Group II (control group):

This group included thirty apparently healthy term babies (15 male and 15 female) with mean gestational age (39± 1) weeks and mean birth weight (3.3 ± 0.4) kg.

Among both groups, there were no statistical significant differences as regard sex, weight and gestational age.

We discovered that incidence of perinatal risk factors e.g antipartum haemorrage, cord prolapse, contracted pelvis occurs more in patient group.

Also we found that intrauterine fetal distress, intauterine growth retardation and meconium aspiration syndrome are major risks for HIE.

We measured ionized serum Ca^{+2} level and total serum mg levels in infants with HIE and the controls (healthy infants).

We found decreased levels of ionized serum Ca^{+2} and total serum Mg in infants with HIE as compared to the controls.

Also we found an observed decrease in serum ionized calcium and total serum magnesium levels in infants with moderate and severe HIE compared to those with mild HIE and in severe HIE compared to moderate HIE.

Both hypomagnesemia and hypocalcemia were assoicated with unfavorable outcome.

Recommendations

1. More extended study is indicated to emphasize over the role of serum ionized calcium and total serum magnesium as diagnostic and prognostic markers of HIE.

2. Cord blood samples and follow up serum samples for ionized calcium and total magnesium in infants with HIE to highlight the changes that occurs in the first 2 days of life.

3. Research is advised over the therapeutic role of $MgSO_4$ and NMDA receptor channel antagonists (NM-801) and Ca channel antagonists for infants with HIE and correlating the results of serum ionized calcium and total serum magnesium in those who received the treatment to the disease outcome.