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A clinical study of hepatic dysfunction in severe birth asphyxia neonates

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Abstract
Background: The effect of asphyxia on the liver and the hepatic functions of the neonate is unexplored. This study was done to see the status of SGOT, SGPT, Alkaline phosphatase and Serum bilirubin in asphyxiated babies and to know the correlation existing between hepatic dysfunction and severity of perinatal asphyxia.
Objectives: This study was undertaken to assess the liver function in healthy newborns and in severe birth asphyxiated babies and compare with the controls. It was also done to collect the information about severity of liver damage and their prognostic value.
Methods: A prospective cohort study was done in Krishna Institute of Medical Sciences, Karad involving 30 severely birth asphyxiated neonates and 11 full term normal neonates were taken as controls. Liver enzymes that is SGOT, SGPT, Alkaline phosphatase, and Serum Bilirubin were estimated in serum of neonates included in this study.
Results: SGOT, SGPT, Alkaline Phosphatase were significantly higher in neonates of study group as compared to neonates of control group. Among the asphyxiated newborns SGOT, SGPT, Alkaline Phosphatase were significantly raised in neonates who succumbed to asphyxia than those who survived. Levels of Serum Bilirubin were also raised in expired than who survived.
Interpretation and conclusion: Severe birth asphyxia causes liver dysfunction. Approximately 76% of severely asphyxiated neonates suffered from deranged hepatic function. Enzyme derangement is significantly more in those neonates who succumbed to asphyxia than those who survived.

Keywords: birth asphyxia, serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT)

Introduction

Birth asphyxia occurs when a baby does not receive enough oxygen before, during or after birth. It is an insult to the fetus or new born due to lack of oxygen and or lack of perfusion to various organs. Fetus totally depends for its oxygen supply and other nutrients on the blood supplied through placenta. In any case, if blood supplied through placenta is hampered, it leads to asphyxial injury. The incidence of perinatal asphyxia is about 1 to 1.5 percent in most centres and is usually related to gestational age and birth weight. It occurs in 9 percent of infants less than 36 weeks of gestation and 0.5 percent of infants of more than 36 weeks gestation, accounting for 20 percent of perinatal deaths or as high as 50 percent of deaths if still borns are included.

Ninety percent of asphyxia insult occurs in antepartum/ intrapartum period as a result of placental insufficiency. During normal labour there is reduced blood flow to placenta, hence decreased oxygen delivery to the fetus. Target organs of perinatal asphyxia are the brain, heart, lungs, kidneys, gut and bone marrow. The most frequent abnormalities involving kidneys (50%) followed by CNS (28%), cardiovascular (25%) and pulmonary system (23%).Thus there is evidence of multiorgan system dysfunction in the immediate neonatal period. The effects of asphyxia on the liver and the hepatic functions of the neonate is a relatively unexplored avenue. This study is being carried out to assess the effect of birth anoxia on hepatic functions of a neonate as also the ultimate outcome of these cases.

The liver plays a central role in the synthesis, degeneration and regulatory metabolism. Due to asphyxia the liver may be so damaged ("Shock liver") that it may not provide its basic functions. Therefore, during perinatal hypoxia which causes a reversible increase in cellular membrane permeability, there is a release into the blood stream of cytoplasmic enzymes (SGOT, SGPT, and LDH). In the presence of liver cell necrosis both the cytoplasmic and mitochondrial enzymes are increased. Since SGOT is also present in myocardium, kidney and RBC while SGPT is primarily released from the liver therefore, this enzyme is more specific for liver damage or injury. Alkaline phosphatase also rises in liver damage but it is less specific and less sensitive.

The present study was conducted in birth asphyxia neonates at KIMSDU, Karad from June 2017 to March 2018.

Methodology

The present study was carried out in Department of Pediatrics in Krishna Institute of Medical Sciences, Karad from July 2017 to October 2017.The cases included in the study were divided into two groups that is study and control groups.This study was approved by the hospital ethical committee.

Selection cases

A) Control group - 22 normal full term neonates appropriate for gestational age were included in this group. These neonates did not have perinatal asphyxia or evidence of any liver disease.

B) Study group- 60 Full term neonates with severe birth asphyxia were selected for study group.

Inclusion criteria
- Full term neonates of appropriate gestational age.
- Apgar score of 3 at 1 minute and 5 at 5 minutes

Exclusion criteria
- Babies having congenital anomalies, septicemia, and history of leaking for more than 18 hours, preterm neonates and low birth weight neonates.

Blood samples were withdrawn after 48-72 hours of birth and the blood collected was allowed to clot and then centrifuged to obtain serum for estimation of biochemical parameters like Serum Bilirubin
SGOT
SGPT
Alkaline phosphatase.

Statistical analysis
Values have been expressed as Mean±SD. The data were compiled and analyzed using descriptive statistics using students 't' test. P<0.05 was considered as significant.

Results
Out of total of 82 full term neonates during the study period at Krishna Institute of Medical Sciences, Karad total of 60 were included in study group and 22 were included in control group.

Table 1: General characteristics of neonates of both the groups.

Characteristics	Control group (n=22)	Study group (n=60)	p value
Sex: Male	14(63.63%)	36(60%)	>0.05
Female	8 (36.36%)	24(40%)	>0.05
Gestational age (weeks)	38.90±0.83	39.50±1.04	>0.0
Mean±SD Range	38 to 40	39 to 41	5
Weight (GMS) Mean±SD	2790±181	2798±206	>0.0
Range	2500 to 3000	2500 to 3100	5

The difference between the two groups were statistically insignificant (p>0.05).

Out of 60 neonates of study group, 36 (60%) were males and remaining 24(40%) were females, while in control group out of 22 neonates,14(63.63%) were males and rest 8(36.36%) were females. The difference was statistically insignificant.

Table 2: Mean±SD and range of liver enzymes in study and control group cases

Liver enzymes	Expired neonates (n=28)	Control group (n=22)	P value
SGOT(IU/L)	157.71±42.94 (94 to 234)	48.73±16.08 (24 to 76)	<0.001
SGPT(IU/L)	72.27±41.49(38 to 154)	20.54±6.99(12 to30)	<0.001
Alkaline Phosphatase (kAU/dl)	18.81±2.71 (16.61to 21.3)	14.45±1.53 (11.8 to 17)	<0.001
Serum Bilirubin(mg/dl)	6.91±2.58 (3.4 to 12.2)	5.32±2.49 (1.6 to 8.2)	>0.05

These values were statistically significant as p<0.05.

Table 3: Outcome of asphyxiated neonates with Apgar score ≤3 or >3 at 5 minutes.

Apgar score at 5 minutes	Expired	Survived	Total
≤3	18(90%)	2(10%)	20
>3	10(25%)	30(75%)	40

Table 4: Outcome of neonates in study group as per liver enzymes value

Liver enzyme	Expired	Survived	P Value
Deranged(n=46)	28(60.87%)	18(39.13%)	<0.001
Normal(n=14)	-	14(100%)	-
Total(n=60)	28(46.67%)	32(53.33%)	

The values were highly significant statistically (p<0.001).All the 14 neonates having normal values of liver enzymes survived.

Table 5: Mean ± SD, range values of liver enzymes studied in expired neonates of study and control groups

Liver enzymes	Expired neonates (n=28)	Control group (n=22)	P value
SGOT(IU/L)	157.71±42.94 (94 to 234)	48.73±16.08 (24 to 76)	<0.001
SGPT(IU/L)	72.27±41.49 (38 to 154)	20.54±6.99 (12 to30)	<0.001
Alkaline Phosphatase (kAU/dl)	18.81±2.71(16.61to 21.3)	14.45±1.53 (11.8 to 17)	<0.001
Serum Bilirubin(mg/dl)	6.91±2.58 (3.4 to 12.2)	5.32±2.49 (1.6 to 8.2)	>0.05

The levels of SGOT,SGPT and Alkaline phosphatase in expired neonates of study group and that of control group were statistically highly significant (p<0.001).
The values of serum bilirubin in expired neonates of study group and their controls were statistically insignificant (p>0.05).

Discussion

Birth asphyxia is a multisystem disorder. The liver too exhibits biochemical and histopathological changes. Birth asphyxia in newborn infants can cause hepatic hypoxic injury. Hypoxia, Hypercapnia and acidosis represent at birth the main symptoms of fetal distress, which manifests clinically in utero

as varying fetal heart rate and after delivery as low apgar score.

The serum activity of SGOT and SGPT is one of the more specific parameters of liver cell injury both in adults and pediatric age group. According to some authors rise in transaminases indicative of liver cell dysfunction is either due to hepatocyte necrosis or due to changes in cell permeability.

Goldberg *et al* showed SGOT ranged from 446-3050 IU/L in asphyxiated babies (13). Elevated SGPT of more than 40 IU/L was observed by other authors.

Sail *et.al* 1990 in his study found that the serum levels of transaminases (SGOT, SGPT) and alkaline phosphatase in non-survivors were significantly higher than those of survivors.

Islam MT *et.al*. 2011 in his study found that mean SGOT, SGPT and ALP of the asphyxiated babies were 76.27±37.44, 82.16±48.08 & 369.59±123.05 U/L and that of normal babies were 23.46±8.45, 26.54±7.76 & 208.20±46.95 U/L respectively and these rise were statistically significant ($p<0.001$). The levels of SGOT, SGPT and ALP were positively correlated with the severity of asphyxia and these correlations were also statistically significant ($p<0.001$).

Conclusion

Severe birth asphyxia causes liver dysfunction as evident by deranged SGOT, SGPT and alkaline phosphatase. Deranged liver enzymes could be detected in first 48 hours of life. Approximately 76.66% of severely asphyxiated neonates suffered from liver derangement. Enzyme derangement is significantly more in those neonates who succumbed to asphyxia than those who survived. SGPT levels were significantly deranged than SGOT levels among asphyxiated neonates.

In asphyxiated neonates, those who had apgar score of ≤3 at 5 minutes had significant higher value of SGOT, SGPT and Alkaline phosphatase and 90% of them expired than those who had apgar score of >3 at 5 minutes.

Birth asphyxia is still common, more so in developing countries where obstetric and newborn resuscitation facilities are not universally available yet. Combination of dehydration, sepsis, shock and nephrotoxic drugs is not an uncommon situation in NICU.

These lead to high incidences of neonatal failure. They are often reversible if identified and managed in time.

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