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Paediatrics Section

# Liver Dysfunction in Perinatal Asphyxia

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## ABSTRACT

**Introduction:** Liver dysfunction in perinatal asphyxia may be manifested by elevation of hepatocellular enzymes. It affects the outcome of perinatal asphyxia in newborn.

**Aim:** To study the effect of perinatal asphyxia on liver function.

**Materials and Methods:** This study was conducted on 100 newborns with perinatal asphyxia and 50 healthy newborns were taken as control group. Baby with APGAR score <7 at 1 minutes, fetal heart variation and meconium passage in utero were considered to have perinatal asphyxia. Qualitative data was analysed statistically by Person Chi-Square test. Numerical analysis was done by mean, SD and independent t-test.

**Results:** The study included 59 male babies and 41 female babies in the case group and 25 male babies and 25 female babies in control group. Mean gestational age in case group was 37.29±2.1 weeks and in control group was 37.06±2.25 weeks. The difference between perinatal asphyxia and control group was highly significant for Serum Glutamate Oxaloacetic Transaminase (SGOT), Serum Glutamate Pyruvic Transaminase (SGPT) and Alkaline Phosphatase (ALP) ($p<0.001$) and significant for Total Serum Bilirubiun (TSB) ($p<0.025$). The difference between fetal asphyxia alone and control group was highly significant ($p<0.001$) for SGOT, SGPT and significant for ALP ($p=0.002$) and TSB ($p=0.009$) respectively. In birth asphyxia alone group, the difference was highly significant for SGOT, SGPT, ALP ($p<0.001$) and significant for TSB ($p=0.034$) as compared to control group.

**Conclusion:** Early detection of hepatic dysfunction helps to predict the complication of hepatic dysfunction and their early treatment.

Keywords: Hepatic dysfunction, Liver Enzymes, Newborn

## INTRODUCTION

In under developed countries, perinatal asphyxia remains a major cause of death and disability [1]. According to WHO between four to nine million newborns develop birth asphyxia each year. Of these, an estimated 1.2 million die and at least the same number develop severe consequences, such as cerebral palsy, epilepsy, and developmental delay [2]. With time, studies were conducted and it was explored that liver function was compromised in perinatal asphyxia in newborn [3].

As a defense in hypoxic ischaemia, the cardiac output is centralised to organs such as brain, heart and adrenals at the expense of less important organs such as liver, lungs, skin, kidney, muscles [4]. Liver dysfunction may be manifested by isolated elevation of hepatocellular enzymes. More extensive damage may occur, leading to DIC, inadequate glycogen stores with resultant hypoglycaemia, altered metabolism, or elimination of drugs [5]. Knowledge of the behaviour of the AST and ALT activity may have an important implications in the diagnosis and early treatment of perinatal asphyxia [6].

This study adds to assessment of liver dysfunction (SGOT, SGPT and ALP) in the babies who had fetal/birth asphyxia alone or combined with some other diseases which was not available in existing literature. The existing literature had data on perinatal asphyxia alone. Thus, the aim was to study the effect of perinatal asphyxia on liver function.

## MATERIALS AND METHODS

This was a cross-sectional hospital based prospective non randomized study, conducted in neonatalology section of Department of Paediatrics, Government Medical College/ Rajindra Hospital, Patiala, Punjab, India. The study period was from July 2012 to June 2013.The study included 100 newborns with perinatal asphyxia. Fifty healthy newborns were taken as control group.

Sample size was calculated based on prior observation of babies born with birth asphyxia (160-180/year) taking into consideration attrition of participants and ability to draw appropriate statistical conclusions.

Ethical committee approval was taken vide letter number BFUHS/2k13/p-TH/9180. Parental consent was taken from each case prior to enrollment in the study.

### Inclusion Criteria

All newborns whether Appropriate for Gestational Age (AGA), Small for Gestational Age (SGA), Large for Gestational Age (LGA) or Term, Postterm, Preterm were included in this study who developed perinatal asphyxia. A newborn was included in perinatal asphyxia group if baby had:

1. Apgar score ≤7 at 1 minute after birth (birth asphyxia).

2. Abnormal fetal monitoring, indicated by prolonged fetal bradycardia or tachycardia or irregular heart rate (fetal asphyxia).

3. Meconium stained Amniotic fluid [7]. (fetal asphyxia).

### Exclusion Criteria

All newborns with proven septicaemia; congenital malformation; inborn error of metabolism; shock; and cardiac failure.

Liver dysfunction was evaluated by ALT, AST, ALP and serum bilirubin. A 2 mL venous blood sample was collected within 24-72 hours of birth. Test for statistical analysis of qualitative data was done by Person Chi-Square. For numerical analysis, mean, SD and independent t-test were used.

## RESULTS

Mean weight of babies in case group (perinatal asphyxia) was 2647±604.6 gm and in control group was 2602±537 gm. Mean gestational age for case group was 37.29±2.10 weeks and for control group was 37.06±2.25 weeks. Out of 100 babies in case group, 41 were female and 59 were male (p=0.421) whereas in control group, out of 50 babies, there were 25 male and 25 female babies (p=0.421).

Difference between perinatal asphyxia and control group were statistically highly significant for SGOT, SGPT and ALP and significant for TSB [Table/Fig-1]. Difference between fetal asphyxia alone group and control group were statistically significant [Table/Fig-2]. Difference between birth asphyxia alone group and control group were statistically significant [Table/Fig-3].

| Group | Case group No. of cases 100 | Control group No. of cases 50 | Statistical analysis | | |
|---|---|---|---|---|---|
| | | | Case v/s Control group | | |
| | | | T | p | S |
| SGOT (IU/L) Mean±SD | 139.64±81.20 | 53.58±19.48 | 7.37 | <0.001 | HS |
| SGPT (IU/L) Mean±SD | 76.38±69.74 | 17.90±5.46 | 5.91 | <0.001 | HS |
| ALP (IU/L) Mean±SD | 178.52±109.8 | 119.40±40.23 | 3.68 | <0.001 | HS |
| TSB (mg/dL) Mean±SD | 8.92±5.73 | 6.63±4.60 | 2.45 | 0.015 | S |

**[Table/Fig-1]:** SGOT, SGPT, ALP, TSB in both groups.

| Group | Case group No. of cases 30 | Control group No. of cases 50 | Statistical analysis | | |
|---|---|---|---|---|---|
| | | | Case v/s Control group | | |
| | | | T | P | S |
| SGOT (IU/lt) Mean±SD | 127.37±73 | 53.58±19.48 | 6.78 | <0.001 | HS |
| SGPT (IU/lt) Mean±SD | 39.87±28.22 | 17.90±5.45 | 5.36 | <0.001 | HS |
| ALP (IU/lt) Mean±SD | 167.20±94.72 | 119.40±40.23 | 3.17 | 0.002 | S |
| TSB (mg/dL) Mean±SD | 9.68±5.43 | 6.63±4.60 | 2.68 | 0.009 | S |

**[Table/Fig-2]:** SGOT, SGPT, ALP, TSB in fetal asphyxia alone and control group.

| Group | Case group No. of cases 51 | Control group No. of cases 50 | Statistical analysis | | |
|---|---|---|---|---|---|
| | | | Case v/s control group | | |
| | | | T | p | S |
| SGOT (IU/L) Mean±SD | 144.94±90.79 | 53.58±19.48 | 6.95 | <0.001 | HS |
| SGPT (IU/L) Mean±SD | 92.82±81.86 | 17.90±5.45 | 6.45 | <0.001 | HS |
| ALP (IU/L) Mean±SD | 187.12±115.74 | 119.40±40.23 | 3.91 | <0.001 | HS |
| TSB (mg/dL) Mean±SD | 8.94±6.05 | 6.63±4.60 | 2.15 | 0.034 | S |

**[Table/Fig-3]:** SGOT, SGPT, ALP, TSB in birth asphyxia alone and control group.

## DISCUSSION

In the present study, difference between case group and control group was statistically highly significant (p<0.001) for SGOT, SGPT and ALP and significant for TSB (p<0.05) [Table/Fig-1]. Similar observation was noted by Islam M et al., where mean value of SGOT, SGPT, TSB and ALP in asphyxiated babies were 76.3±37.4 IU/L; 82.2 ± 48.08 IU/L; 5.5±2.01 mg/dl; 369±123.05 IU/L respectively while of normal babies were 23.5±6.5 IU/L; 26.5±7.8 IU/L; 4.5±1.2 mg/dl; 208.2+46.9 IU/L respectively and these difference were statistically significant (p-value<0.001) [8]. Similarly in a study by Saili A et al., mean value of SGOT, SGPT, ALP in newborns with perinatal asphyxia were 97.84±119.42 IU/L, 44.09±61.94 IU/L, 176.4±123.0 IU/L and in control group were 54.83±48.86 IU/L, 22.11±32.96 IU/L, 143.6±90.6 IU/L (p-value <0.05). They also found increased value of TSB in perinatal asphyxia group [3]. In a study by Vajro P et al., found significantly raised level of SGOT, SGPT, ALP, and TSB in babies with perinatal asphyxia [9].

Various authors [10-14] in their studies have found increased levels of SGOT and SGPT in babies with perinatal asphyxia

compared to control group. Talati AJ et al., found SGOT and SGPT increased in 43% of babies with perinatal asphyxia [14].

## LIMITATION

The statistical difference in mean SGOT, SGPT, ALP and TSB for 30, 31, 41 week of gestation between perinatal asphyxia and control group could not be evaluated because of small sample size.

## CONCLUSION

This study suggests that hepatic dysfunction occurs in perinatal asphyxia. Estimation of hepatic enzymes can be used as a diagnostic tool as well as to detect the severity of perinatal asphyxia. Early detection of hepatic dysfunction helps to predict the complication of hepatic dysfunction and their early treatment.

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