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Hepatic Profile in Asphyxia Neonatorum

S.V. Godambe
R.H. Udani
Sushma Malik
B.M. Kandalkar

Perinatal asphyxia is one of the leading causes of neonatal mortality in India. In addition to hypoxic-ischemic brain injury and neurological deficits, there is evidence of multi-system insult (l). Hepatic dysfunction is caused by redistributing cardiac output away from nonvital viscera to the heart, brain and adrenal glands (2). The present study was carried out to determine the extent of liver dysfunction following asphyxia.

Subjects and Methods

This observational investigation comprised a study group of 70 newborns with an Apgar score of ≤ 7 at 1 minute. Fifty babies with an Apgar score of > 7 at 1 minute, matched for gestation, birth-weight and sex, comprised the control group. Severity of asphyxia was graded as mild if Apgar score was 5-7, moderate if 3-4, and severe if Apgar was < 3(3). Venous blood samples of the neonates were collected within 24 hours of birth. The estimations included SGPT, prothrombin time and serum proteins. All biochemical tests were carried .out by standard methods (4). Elevated SGPT of more than 40 IU/L or more than twice the control group, reduction in prothrombin index of less than 85% and reduction in serum proteins of less than 4.5 g/dl were considered abnormal. Prothrombin time could be done only in 44 babies due to non availability of investigations during odd hours. Postmortem liver biopsy was done in all the 32 patients who died (4 had mild, 9 had moderate and 19 had severe asphyxia). The histopathological hepatic changes were categorized as follows (5):

(a) Mild-Central vein dilated and congested, mild dilatation of the sinusoids with mild congestion, portal triad congested with mononuclear cell infiltration and foci of extramedullary hemopoiesis in the sinusoids.

(b) Moderate-Along with above changes, hepatocytes showed diffuse microvesicular fatty changes.

(c) Severe—Along with all above changes, centrilobular hepatocytes showed moderate to severe fatty change.

Results and Discussion

Out of 70 newborns, 9 (12%) were mild-

From the Departments of Pediatrics and Pathology Topiwala National Medical College and B.Y.L. Nair Ch. Hospital, Mumbai 400 008.

Reprint requests: Dr. R.H. Udani, C 1/5, 358, Municipal Tenements, Khan Abdul Gaffarkhan Road, Worli, Mumbai 400 018.

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ly asphyxiated, 31 (44.3%) were moderately and 30 (42.9%) were severely asphyxiated. There were 15 (21.4%) preterms and 55 (78.6%) full terms. Almost 67% preterm and low birth weight babies were severely asphyxiated as compared to 36.4% of term babies. Severe asphyxia was noted in 31% of normally delivered babies and in 64% of those delivered by LSCS or assisted deliveries. *Table I* summarizes the biochemical and histopathologic changes.

SGPT levels were significantly elevated in asphyxiated neonates compared with the controls. Raised SGPT was documented in 75% of asphyxiated babies who expired. Similarly, reduced prothrombin index (PI) was noted in a significantly larger proportion of asphyxiated neonates compared to controls. Histopathologic changes in liver were noted to significantly increase with the severity of asphyxia.

Birth asphyxia is a multisystem disorder. The liver too exhibits biochemical and histopathologic changes. The mean SGPT levels in this study were noted to increase from 35.3 ± 28.8 IU/L in mild asphyxia to 65.6 ± 33.2 IU/L in severe asphyxia. Similar results were observed by other workers who noted a rise from 44 ± 61.9 IU/L in mild to 59.5 ± 108 IU/L in severe asphyxia(6-8). The rise in SGPT indicates liver cell dysfunction either due to hepatocyte necrosis or due to changes in the cell permeability.

Prothrombin index (PI) was reduced in all grades of asphyxia and similar observations of altered liver dependent coagulation parameters were observed by others (9-11). A steady fall of PI with increasing severity of asphyxia could be due to reduced capacity of liver to produce coagulation factors with increasing hypoxic damage. Hypo-proteinemia was noted in 34% asphyxiated babies in the present study and 44% prevalence has been reported by others(8). Hypoproteinemia is an imprecise index of the severity of liver damage due to the long life of serum proteins (12).

Hepatic histopathological changes were compared with elevated SGPT and reduced prothrombin index and hypoproteinemia. All these parameters were maximally affected in severe asphyxia which was also associated with a high mortality. The clinical grading of asphyxia did no correlate with histopathological gradings but all those who died had mild to severe liver biopsy changes.

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TABLE I–*Biochemical Parameters and Histopathological Grading of Liver Biopsy Changes in Relation to Asphyxia*

Severity of asphyxia	SGPT (IU/L) Mean ± SD (n=70)	Reduction in prothrombin index (%) (n = 44)	Reduction in total serum protein (%) (n=68)	Histopathological liver biopsy changes				
				Mild (n=10)	Moderate (n=10)	Severe (n=12)	Total (n=32)	%
Mild (n=9)	35.3* ± 28.8	5 (62.5)* (n=8)	2 (25.0) (n=8)	2	2	-	4	12.5
Moderate (n=31)	38.1* ± 23.2	16 (84.2)* (n=19)	7 (23.3)* (n=30)	5	3	1	9	28.1"
Severe (n=30)	65.5* ± 33.2	15 (88.2) (n=17)	14 (46.7)* (n=30)	3	5	11	19	59.4*
Control	14.0 ± 12.8	6 (12) (n=50)	0 (n=50)					

* $p < 0.001$

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