ORIGINAL RESEARCH PAPER
Volume - 10 | Issue - 04 | April - 2021 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A STUDY ON ETIOLOGY, CLINICAL FEATURES, MANAGEMENT AND OUTCOME OF ACUTE LIVER FAILURE IN PAEDIATRIC INTENSIVE CARE UNIT OF BURDWAN MEDICAL COLLEGE

Paediatric

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ABSTRACT

Introduction: In the present study, etiology, important clinical features, management and outcome of acute liver failure in children admitted in Pediatric Intensive Care Unit (PICU) of **BURDWAN MEDICAL COLLEGE**, WB are studied.

Aims and objectives: To study different clinical features among those children having acute liver failure.

I. To study the management and outcome of those children with acute liver failure in the Pediatric Intensive Care Unit.

Material and methods: Pediatric Intensive Care Unit of **BURDWAN MEDICAL COLLEGE**, WB**.** 1 Year [December 2019 to November 2020]**.** Patients from 3months of age to 12years of age are eligible for enrolment if they meet the following criteria.

Conclusion: Present study showed that cerebral edema was significantly associated with mortality whereas AKI and spontaneous bacterial peritonitis were not significantly related with mortality. Mean TSB and Prothrobin Time were higher but mean serum albumin was lower in those cases that had died.

KEYWORDS

Prothrombin, Encephalopathy, Ornithine transcarbamylase deficiency and Galactosemia

INTRODUCTION

Acute Liver Failure (ALF) is a clinical syndrome resulting from severe functional impairment of hepatocytes[1]. Synthetic, excretory and detoxifying capacities of the liver are all severely impaired1. In adults, hepatic encephalopathy has been an essential diagnostic feature[1]. However, in paediatrics, this narrow definition may be problematic because early hepatic encephalopathy can be difficult to detect in infants and children[1].

Definition of paediatric acute liver failure includes biochemical evidence of acute liver injury (usually < 8 weeks duration); no evidence of chronic liver disease; and hepatic based coagulopathy defined as a prothrombin time (PT) > 15 sec or international normalised ratio (INR) > 1.5 not corrected by vitamin K in the presence of clinical hepatic encephalopathy, or a PT > 20 sec or INR > 2 regardless of the presence of clinical hepatic encephalopathy. [1]

Acute liver failure (ALF) is a rare but challenging clinical syndrome with multiple causes. Sometimes, specific etiology cannot be identified in 15% of adult and 50% of pediatric cases despite thorough investigations and research. [2] Etiology of acute liver failure varies with age and geographical location. [3] Most cases of acute liver failure in India are due to infectious causes, predominantly viral hepatitis though a significant group with indeterminate causation remains.[3] The etiology of acute liver failure in infants is largely metabolic. [3] Galactosemia, mitochondrial DNA depletion syndrome (MDS), ornithine transcarbamylase deficiency, tyrosinemia type 1, long-chain 3-hydroxyacyl-CoA dehydrogenase deficiency, hereditary fructose intolerance (HFI) are some common inherited metabolic diseases that can cause severe life threatening acute liver failure.[4] Etiology also differs from developing countries to developed countries. In developed nations drug toxicity like acute acetaminophen toxicity predominates among identified etiologies of paediatric acute liver failure.[5]

Acute liver failure in childhood is characterized by jaundice, encephalopathy and coagulopathy leading to multiorgan failure in a patient with no prior history of liver disease.[6] Vomiting, failure to thrive, hypotonia and developmental delay are generally seen in cases of acute liver failure due to some inherited metabolic diseases.[7] Commonly associated disorders that require initial recognition and treatment include energy production defects manifested as hypoglycaemia, coagulation abnormalities, immune system dysfunctions, encephalopathy, intracranial hypertension and cerebral edema.[8]

The course of paediatric ALF is variable and the mortality rate is high. Liver transplantation is the only therapy of proven benefit, but the rapidity of progression and the variable course of ALF limit its use.[2, 3] Excellent intensive care is critical in management of patients with ALF. The mainstay of management is supportive care in an intensive care unit. Monitoring of clinical and biochemical parameters at frequent intervals is necessary until the patient becomes stable.[2, 3] Routine correction of coagulopathy is not recommended but FFP and cryoprecipitates are recommended in patients with clinically significant bleeding and during invasive procedures. As infection remains one of the major causes of death in patients with ALF, administration of antibiotics must be considered. [9], Complications must be taken care of with prompt and justified managements.[2]

The outcome of ALF varies by etiology; favorable prognoses being found with acetaminophen overdose, hepatitis A and ischemia and poor prognoses with drug-induced ALF, hepatitis B and indeterminate cases.[2] Mortality is predominantly due to raised intracranial pressure, infections and multi-organ failure.[1] Children with grade 3 and grade 4 encephalopathy are generally having highest mortality. Various prognostic indicators and predictors of mortality have been identified through worldwide studies.[6,9]

Future possible therapeutic approaches of paediatric ALF include N-acetylcysteine, hypothermia, liver assist devices, and hepatocyte transplantation. Advances in stem cell research may allow provision of cells for bioartificial liver support in future.[2] Multicentric research is necessary for therapeutic advancement.

- To study the etiology of acute liver failure of children admitted in Paediatric Intensive Care Unit in **BURDWAN MEDICAL COLLEGE**, WB.
- To study different clinical features among those children having acute liver failure.
- To study the management and outcome of those children with acute liver failure in the Pediatric Intensive Care Unit.

MATERIAL AND METHODS

STUDY SETTINGS: Pediatric Intensive Care Unit of **BURDWAN MEDICAL COLLEGE**, WB

STUDY DURATION: 1 Year [December 2019 to November 2020]

STUDY SAMPLE:

A. INCLUSION CRITERIA: Patients from 3months of age to 12years of age are eligible for enrolment if they meet the following criterias:

1. Children with no known evidence of chronic liver disease.
2. Biochemical evidence of Acute Liver Injury : Hepatic based coagulopathy defined as Prothrombin Time (PT) >/=15seconds or International Normalized Ratio (INR) >/=1.5 not corrected by Vitamin K in the presence of clinical Hepatic Encephalopathy or Prothrombin Time (PT)>/=20seconds or INR >/=2.0 regardless of the presence or absence of clinical Hepatic Encephalopathy.
3. Patients whose parents gave consent to include their child in the study.

B. EXCLUSION CRITERIA:

1. Patients below 3months of age are excluded from the study group.
2. Patients above 12 years of age are also excluded from the study.
3. Patients whose parents don't give consent to include their child in the study.
4. Children with known evidence of Chronic Liver Disease

RESULT AND DISCUSSION

Total 36 patients with pediatric acute liver failure (PALF) were studied and study results and analysis are already given. Now clinical features, etiology, management and outcome of PALF were discussed here.

The mean age of the present study population is 7.9 ± 2.5 years (range 2 years to 11.5 years). This finding is almost similar with the mean age of the previous study done in Kolkata by Samanta et al.[10] (mean age was 7.12 ± 0.37).

The mean age of this study is considerably higher than what has been reported in other previous studies (5.8 years by Poddar et al [11], 5.5 years by Bhowmick et al. [12],

The male: female ratio of this study is 1.12: 1 A very slight male preponderance is seen in the present study with a share of 52.8 % of the study population (19 are male out of 36 patients). This study finding has similarity with the gender distribution of the study by Yasmin et al.[13] (53.2 % were male; 33 were male out of total 62 patients).

In this study 'decreased' liver span is found in 36.1 % (13 out of 36 cases) of study population. This finding is almost similar with the finding of Samanta et al [10] (decreased liver span is seen in 31.11% of study population; 14 out of 45 cases). 'Normal' and 'increased' liver span are seen in 19.4 % and 44.4 % of study population, respectively in the present study.

According to Yasmin et al. [13], ascites was higher in frequency in their study perhaps because of large number of children were suffering from Wilson's disease and hepatitis A virus infection.

The frequency of encephalopathy in this study is higher than the study findings of Kaur et al. [14] (New Delhi, India) and Yasmin et al.[13] (Bangladesh)(83.7 % and 75.8% of study population had suffered from hepatic encephalopathy respectively in their study). Though the study of Kulkarni et al.[15] 2015 in US reported only 38.6 % of study population to have hepatic encephalopathy.

The present study assesses the etiology of ALF at one center in Kolkata, India. The etiology was detected and verified in 28 out of the 36 cases (77.8 %). Samanta et al. [10] (Kolkata) also detected specific etiology in 77.7 % of study population which is similar with the present study.

Mean serum albumin of the present study is 2.75 ± 0.8 g/dl (range 1.5 – 3.9 g/dl). Serum albumin value of 2.5 g/dl is seen in 16 cases (44.4% of study population) and severe hypoalbuminemia (2g/dl) is seen in 9 cases (25 % of study population). Present study is corroborating with the study of Samanta et al.[10] where hypoalbuminemia (2.5g/dl) was found in 42.2% (19 out of 45 total cases) of study population.

Table: Distribution of mean lowest platelet count: outcome

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Highest value of TSB	Death	18	32.9333	9.9294	6.6000	45.0000	33.9500	<0.0001
	Survived	18	11.0111	4.2240	6.6000	20.2000	9.2000	
Highest value of Prothrobin Time	Death	18	86.8889	24.4514	29.0000	120.0000	90.0000	<0.0001
	Survived	18	26.6111	5.9125	18.0000	38.0000	26.5000	
Serum Albumin	Death	18	2.0944	.4856	1.5000	3.5000	1.9500	<0.0001
	Survived	18	3.4111	.4057	2.4000	3.9000	3.6000	
Lowest platelet count	Death	18	111472.2222	65492.8088	16000.0000	200000.0000	140000.0000	0.0002
	Survived	18	193333.3333	52650.9036	120000.0000	321000.0000	194000.0000	

CONCLUSION

We found that mean age of children with acute liver failure who had died was lower and Viral Hepatitis was more common in our study.

It was found that decreased liver span and ascites were significantly associated with mortality.

A higher stage of encephalopathy was more associated with death which was statistically significant.

We found that children with bleeding manifestation were significantly associated with mortality.

Hypoglycemic episode was significantly associated with mortality but Blood culture; Urine Culture and Chest X Ray findings were not significantly associated with mortality.

Present study showed that cerebral edema was significantly associated with mortality whereas AKI and spontaneous bacterial peritonitis were not significantly related with mortality.

Mean TSB and Prothrobin Time were higher but mean serum albumin was lower in those cases that had died.

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