



A STUDY OF CLINICO-ETIOLOGICAL PROFILE, LABORATORY PROFILE AND SHORT-TERM OUTCOME OF PEDIATRIC ACUTE LIVER FAILURE

Paediatric Medicine

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ABSTRACT

Background: Acute liver failure (ALF) is a complicated, rare condition that can lead to multisystem organ failure and death. Acute hepatic injury or death causes fast loss of hepatocellular function, which leads to multisystem involvement and, eventually failure. **Objective:** To observe the clinical course, various complications, and short-term outcomes of acute liver failure (ALF) in children. The study aimed to determine the etiology of acute liver failure in the study population, assess the mortality rates associated with different etiologies, and identify the most common causes of acute liver failure in children. **Methods:** A total of 50 children aged 1 to 18 years diagnosed with acute liver failure were included in this observational study conducted at Sir Padampat Mother and Child Health Institute, Jaipur, from May 2020 to February 2023. Clinical and laboratory profiles, as well as outcomes, were recorded using a standardized data sheet. **Results:** The mean age of the study population was 6.3 years, with 50% of the children falling within the 2 to 5 years age group. The etiology of acute liver failure included hepatitis with Wilson's disease and other hepatitis (62%), drug & toxins induced liver failure (6%), idiopathic cases (22%), and autoimmune causes (10%). Mortality rates varied among the etiologies, with hepatitis A accounting for the highest mortality (63.64%), followed by idiopathic cases (36.36%). Other hepatitis had a mortality rate of 16.67%, autoimmune causes accounted for 20% mortality, and drug & toxins induced liver failure had the highest mortality rate at 100%. No mortality was reported due to Wilson's disease in this study. **Conclusion:** Hepatitis A emerged as the most common cause of acute liver failure in children in our study population, followed by idiopathic cases. Early diagnosis is critical, particularly in Wilson's disease, as the outcomes are unfavourable without liver transplantation. However, viral etiologies, primarily hepatitis A, showed improved outcomes with supportive care. This study highlights the importance of early recognition, appropriate management, and the potential benefits of timely intervention in acute liver failure in children.

KEYWORDS

Acute liver failure, Hepatic encephalopathy, Mortality, Coagulopathy

INTRODUCTION

Acute liver failure (ALF) is a complicated, rare condition that can lead to multisystem organ failure and death. Acute hepatic injury or death causes fast loss of hepatocellular function, which leads to multisystem involvement and, eventually, failure¹. Within 8 weeks² of the beginning of the signs and symptoms of liver disease, it was characterised by evidence of severe hepatic dysfunction, including jaundice and coagulopathy, as well as the development of hepatic encephalopathy³. The inability to assess the age-appropriate mental status and the exact duration of illness in children has made it challenging to apply this definition to children^{4,5}.

Pediatric Acute Liver Failure (PALF) is defined as:

A child with no known chronic liver disease and biochemical indications of acute liver injury, as well as at least one of the following

- INR>1.5, encephalopathy, not treated with vitamin K supplementation
- INR>2.0, without encephalopathy, not treated with vitamin K supplementation.⁶

In terms of clinical presentation and etiologic range, paediatric ALF differs greatly from adult ALF. To avoid organ failure, transplantation, and death, the diagnosis, evaluation, and care of this rare but complex condition necessitates a thorough diagnostic evaluation as well as close monitoring, anticipating, and controlling the multisystem problems that arise as a result of ALF.⁷

Despite advances in medical care, the prognosis for ALF, both in children and adults, remains grave. The mortality rate for ALF is high, even with appropriate medical intervention mortality is between 60-80%. This highlights the critical nature of the condition and the urgent need for timely and effective management to improve outcomes. In some cases, liver transplantation^{8,9} may be the only life-saving option for patients with irreversible liver failure.

Methodology: The methodology outlined for the study on children with Acute Liver Failure (ALF) at S.M.S. Medical College in Jaipur includes the following steps:

1. **Study Participants:** The study will include 50 children with ALF, aged between 2 years and 18 years, who are seeking treatment at the Department of Pediatrics at S.M.S. Medical College over the study period. Sample size is calculated as 95% confidence level, alpha error of 0.05% expecting 27% of the acute liver failure cases due to hepatitis A virus infection and as per the reference article. At 12% absolute allowable error, the required sample size will be 50 cases of acute liver failure. This sample size is large enough to include various etiology of acute liver failure.
2. **Informed Consent:** Prior to enrolling children into the study, informed consent will be obtained from their parents or legal guardians. This ensures that the participants parents or guardians are aware of the study's purpose, procedures, potential risks, and benefits, and willingly agree to their child's participation.
3. **Patient Selection:** Children who present with clinical features suggestive of ALF will be investigated for inclusion in the study. These features may include symptoms¹⁰ such as jaundice, vomiting, hepato splenomegaly, ascites, and signs of liver failure.
4. **Medical Evaluation:** A detailed medical and family history will be taken for each eligible participant. A thorough physical examination will be conducted, focusing on signs of liver failure. Additionally, relevant information regarding etiology, clinical profiles, laboratory profiles, diagnosis, and treatment will be recorded. Developmental history and significant drug & toxins history will also be documented.
5. **Laboratory Investigations:** Several laboratory tests will be performed on the participants, including: Complete Blood Count (CBC), Liver Function Tests (LFT), Renal Function Tests (RFT), Random Blood Sugar (RBS), Arterial Blood Gas (ABG), Viral markers (to identify any viral causes of liver failure), Prothrombin Time-International Normalized Ratio (PT-INR), Serum Ammonia levels, Serum Lactate levels, Serum Ceruloplasmin levels, KF-Ring (Kayser-Fleischer Ring) examination (for detecting copper accumulation in the cornea), 24-hour de copper (to assess copper excretion).
6. **Standard Management:** The enrolled children will be managed according to the standard management guidelines for ALF. The specific treatment and interventions provided to the participants

will follow established protocols for managing acute liver failure in children.

By following this methodology, the study aims to gather data on the etiology, clinical profiles, laboratory findings, and treatment outcomes of children with acute liver failure.^{11,12}

Inclusion Criteria

- All children aged 2 years to 18 years presenting with acute liver failure.

Exclusion Criteria

- Children aged <2 years and >18 years.
- Children with chronic liver disease.
- Acute hepatitis without liver failure.

Statistical Analysis

Statistical analysis will be done by appropriate statistical tests. Data will be compiled in the form of master chart using Ms-excel, qualitative data will be expressed as percentage and proportion while quantitative data will be expressed as mean and standard deviation, and appropriate statistical test will be applied.

RESULTS

In this comprehensive study, encompassing a cohort of 50 children, several key findings emerged, shedding light on various aspects of pediatric liver failure. The age distribution revealed that 50% of the children were between the ages of 2 to 5 years, indicating a significant proportion of younger participants. Moreover, 42% fell within the 6 to 12 years age range, while 8% were between 13 to 18 years old, highlighting the representation of a wide age spectrum within the study population. The gender distribution indicated that 64% of the participants were female, whereas 36% were male, emphasizing the need for gender-specific considerations when evaluating liver failure in pediatric patients.

Table 1: Age Distribution

Age group	No. of cases	Percentage
2 to 5	25	50%
6 to 12	21	42%
13 to 18	4	8%

Examining the outcomes, it was observed that 54% of the children were discharged, indicating successful management and recovery, while 46% experienced unexpected deaths, underscoring the gravity and challenges associated with pediatric liver failure. These outcomes were further analyzed in relation to the different diagnoses. The distribution of diagnoses revealed that Hepatitis A was the most prevalent cause, accounting for 22 (44%) of the cases. Idiopathic liver failure was the second most common diagnosis, comprising 11 (22%) cases. Other notable diagnoses included other hepatitis with 6 (12%) cases, autoimmune liver disease with 5 (10%) cases, drug & toxins induced liver failure with 3 (6%) cases, and Wilson disease with 3 (6%) cases. These findings highlight the diverse etiologies contributing to liver failure in pediatric patients.

Table 2: Etiology Of Liver Failure

Diagnosis	No. of cases	Percentage
Hepatitis A	22	44%
Drug & toxins induced	3	6%
Other hepatitis	6	12%
Autoimmune	5	10%
Wilson	3	6%
Idiopathic	11	22%

Interestingly, Hepatitis A exhibited the highest mortality rate, with 63.64% of the cases resulting in death, indicating the severity and potential complications associated with this viral infection¹³. Idiopathic liver failure, on the other hand, demonstrated a lower mortality rate of 36.36%, highlighting the challenges in determining the underlying cause and optimizing treatment strategies for these cases. Other diagnoses displayed varying mortality rates, with other hepatitis accounting for 16.67% of deaths, autoimmune liver disease at 20%, and drug & toxins induced liver failure resulting in 100% mortality. Notably, no deaths were attributed to Wilson disease. The significant relationship observed between etiology and outcome, as determined by the chi-square test (p-value = 0.0265), underscores the importance of identifying the underlying cause and tailoring management approaches accordingly.

Table 3: Etiology Vs Outcome Distribution

Variable		Outcome		Chi square test
		Discharged	Death	
Diagnosis	Hepatitis A	8	14 (63.64%)	0.0265 (S)
	Idiopathic	7	4 (36.36%)	
	Other hepatitis	5	1 (16.67%)	
	Autoimmune	4	1 (20%)	
	Wilson	3	0 (0%)	
	Drug & toxins induced	0	3 (100%)	

The duration of illness was a crucial factor analysed in relation to the outcomes. Findings indicated that 66% of patients had illnesses lasting less than one week, suggesting a relatively acute course of the disease, while 34% experienced illnesses lasting more than one week, signifying a more prolonged and potentially severe condition. The association between illness duration and outcome revealed a complex interplay, as 14 cases died among those with shorter illness duration, contrasting with 19 cases discharged, while in the group with longer illness duration, 9 cases died, and 8 cases were discharged. However, statistical analysis indicated no significant correlation between illness duration and outcome (p-value = 0.4797), suggesting that other factors, such as etiology and disease severity, may have a more substantial impact on the final outcome.

In addition to illness duration, various symptoms and laboratory findings were assessed to provide a comprehensive understanding of the participants' condition. Bleeding was found to be present in 42% of the cases, highlighting the potential coagulopathy¹⁴ and associated complications^{15,16} in pediatric liver failure. Altered levels of consciousness were observed in 60% of the cases, indicating the involvement of hepatic encephalopathy, a serious neurological manifestation of liver dysfunction. Furthermore, blood pressure readings above 90 mm Hg were noted in 54% of the participants, potentially reflecting the hemodynamic alterations seen in liver failure, while 46% had readings at or below 90 mm Hg, indicating compromised perfusion and potential circulatory instability. Jaundice, characterized by icteric appearance, was present in 80% of the cases, underscoring the hepatobiliary dysfunction and bilirubin accumulation associated with liver failure, while 20% did not exhibit jaundice, suggesting potential variations in the clinical presentation of pediatric liver failure.

Laboratory parameters were evaluated to provide insights into the participants' condition and aid in the diagnostic and prognostic assessment. Prothrombin time (PT), a crucial measure of coagulation function, was below 20 in 16% of the cases, indicating relatively preserved coagulation capacity in these individuals, while it was above 20 in 84% of the cases, suggesting impaired clotting ability and increased bleeding risk¹⁷. Furthermore, PT/INR values, used as indicators of liver synthetic function, revealed that 78% of the participants had a value above 2, implying impaired hepatic synthesis of clotting factors, while 22% had a value below 2, indicating relatively preserved synthetic function.

The study also investigated the relationship between serum glutamic pyruvic transaminase (SGPT) levels and the final outcome. Among cases with SGPT values below 1000, 7 cases resulted in recovery, whereas 15 cases led to death. In cases with SGPT values above 1000, 7 cases recovered, but 21 cases resulted in death. However, statistical analysis revealed no significant relationship between SGPT results and the final outcome (p-value = 0.5940), highlighting the multifactorial nature of pediatric liver failure and the need for a comprehensive approach that considers various clinical and laboratory parameters.

Overall, this study provided valuable insights into the distribution of age, gender, diagnoses, outcomes, and various clinical and laboratory findings in pediatric liver failure cases. The findings underscored the diverse etiologies contributing to liver failure in children and highlighted the importance of identifying specific causes to optimize management strategies. Furthermore, the study shed light on the clinical presentation, outcomes, and laboratory parameters associated with pediatric liver failure, contributing to a better understanding of this complex condition. Further research and ongoing efforts are necessary to improve diagnosis, treatment, and outcomes for pediatric patients affected by liver failure.

DISCUSSION

Acute liver failure (ALF) in children is a critical condition that requires a comprehensive understanding of its various aspects. In our study, we aimed to explore the age distribution, sex distribution, etiological profile, duration of illness, and clinical correlations associated with ALF in children. To provide a comprehensive analysis, we compared our findings with the results of previous research studies.

Age Distribution: Understanding the age distribution of children affected by ALF is crucial for identifying vulnerable age groups and planning appropriate interventions. In our study, we examined 50 children with ALF and found that 50% of them were between the ages of 2 to 5, 42% were between 6 to 12, and 8% were between 13 to 18 years old. These age proportions align closely with the findings reported by Kaur et al., where 41.8% of participants were aged 6 to 12, 44.1% were aged 1 to 5 years, and 14.1% were aged under a year. Similarly, A.S. Vaanmathi et al. reported that 42% of their participants were aged 6 to 12, and 36% were aged 1 to 5 years. S Ganguly et al. reported an age range of 2.8 to 12 years, with a mean age of 7.12, while A Yasmin et al. reported a mean age of 8.5 years. Our investigation yielded a mean age of 6.30 years, which falls within the range of previous studies.

Sex Distribution: Analysing the sex distribution of children with ALF is essential to identify any gender-based differences in susceptibility or outcomes. In our study, we observed that 36% of the children with ALF were male, while 64% were female. This sex distribution is comparable to the study conducted by A Yasmin et al., where 54% of the participants were male and 46% were female. However, Kaur et al. reported a higher proportion of males (70%), while A.S. Vaanmathi et al. had a higher percentage of females (60%). These variations in sex distribution may be attributed to differences in sample sizes and the demographic characteristics of the populations studied.

Etiological Profile: Understanding the etiology of ALF in children is crucial for appropriate management and targeted interventions. In our study, we identified several etiological factors contributing to ALF, including hepatitis with Wilson disease and other hepatitis (62%), drug & toxins induced liver failure (6%), idiopathic causes (22%), and autoimmune factors (10%). Among the cases of hepatitis, hepatitis A was the most common subtype observed, while other hepatitis cases were predominantly attributed to dengue infection. Additionally, drug & toxins induced liver failure was primarily caused by paracetamol toxicity. These findings align with A Yasmin et al.'s study, where viral hepatitis infections accounted for 63% of cases. Similarly, Kaur et al. reported viral hepatitis, including hepatitis A-E, as the most common cause (77%). Other researchers, such as Puja A. et al., identified infections (32%), indeterminate causes (23%), paracetamol toxicity (21%), metabolic abnormalities (13%), and other factors (11%) as the main etiologies. A. Grama et al. found that infections (72.72%), metabolic abnormalities (43.47%), and toxic causes (43.47%) were common causes of ALF in newborns and infants, while in children and adolescents, infections (60%) and toxic causes (79.41%) were prominent.

Duration of Illness: Assessing the duration of illness in children with ALF provides insights into the acute nature of the condition and helps in planning appropriate treatment strategies. In our study, we found that 66% of the patients had illnesses lasting less than one week, while 34% had illnesses lasting more than one week. This indicates that a significant proportion of children experience rapid disease progression leading to ALF.

Clinical Correlations: Identifying clinical correlations in children with ALF is vital for understanding the manifestations and outcomes associated with the condition. In our study, we observed that 46% of the patients had highly coloured urine, while 54% did not. Additionally, 42% of the patients had bleeding cases, with 12 out of 21 patients dying due to bleeding. A Yasmin et al. reported bleeding in 7 cases, while Kaur et al. found that 42% of their cases had bleeding. Regarding blood pressure, 46% of our participants had a value of 90 mm Hg or less, while 54% had higher blood pressure. Furthermore, 80% of the participants exhibited icterus, while 20% did not. These clinical correlations highlight the diverse manifestations and potential complications associated with ALF in children.

Etiological Profile And Outcome: Analysing the relationship between the etiological profile of ALF and its outcome is crucial for understanding the prognosis and informing treatment decisions. In our

study, we found that hepatitis A and other hepatitis collectively affected 28 patients, while idiopathic, autoimmune, and drug & toxins induced causes affected an additional 22 patients. Among these cases, Wilson disease and other hepatitis contributed to the highest mortality rate, accounting for 24 out of 42 deaths. These findings align with the study conducted by A Yasmin et al., where 39 (63%) patients had Wilson disease, and 3 patients experienced ALF due to other hepatitis, resulting in a mortality rate of 57%. Similarly, Kaur et al. reported that infections, primarily viral hepatitis, were the most common cause (77%), with 19 out of 43 patients dying due to ALF. These results emphasize the significant impact of viral hepatitis and Wilson disease on mortality rates associated with ALF in children.

Table 4: Comparison with different Indian studies:

	Present study 2023	Puja Amaty et al., 2022 ¹⁸	A.S. Vaanmathi 2018 ¹⁹	Sharandeep kaur et al., 2017 ²⁰	S Ganguly et al., 2007 ²¹
Duration	2 year	6 year	8 months	2.4 year	1.5 year
Study design	Observational study	Prospective study	Prospective study	Prospective study	Prospective study
Sample size	50	125	50	43	45
Place of study	Jaipur	Chennai	Chennai	New Delhi	Kolkata
Gender (M:F)	18:32	73:52	20:30	30:13	09:36
Age (year mean)	6.3	2.55	6.1	4.8	7.12
Infection (%)	56	32	34	66.6	68.89
Wilson (%)	6	-	-	4.6	6.7
Autoimmune (%)	10	-	6	2.3	-
Idiopathic (%)	22	23	16	9.2	22.22
Mortality (%)	46	36.8	68	44	48.9

Table 5: Comparison with different foreign studies:

	Present study 2023	Alyaa Yass et al., 2020 ²²	Songpon Getsuwan et al., 2020 ²³	Alina Grama et al., 2020 ²⁴	A. Yasmin et al., 2019 ²⁵
Duration	2 year	1 year	6 year	7 year	2.5 year
Study design	Observational study	Prospective study	Prospective study	Prospective study	Prospective study
Sample size	50	50	27	97	62
Place of study	Jaipur	Baghdad	Thailand	Romania	Bangladesh
Gender (M:F)	18:32	-	-	49:48	33:29
Age (mean)	6.3	5.8	2	7.66	8.5
Infection (%)	56	36	30	27.83	27.42
Wilson (%)	6	10	3.7	12.37	62.9
Autoimmune (%)	10	4	-	8.2	-
Idiopathic (%)	22	12	25.9	11.34	-
Mortality (%)	46	68	59	31.95	48

Limitations

It is important to acknowledge the limitations of our study, including the small sample size of 50 children, which may restrict the generalizability of our findings. Additionally, the variations in findings among different studies may be attributed to differences in sample characteristics, study design, and geographic location. Therefore, further research with a larger sample size and more diverse populations is required to validate and strengthen the conclusions drawn from our study.

CONCLUSION

ALF is an uncommon illness that has a high mortality rate when untreated. Sometimes emergency liver transplantation is the only available treatment option. Depending on the child's age, various causes exist for ALF than there are for adults. In our population, Hepatitis A is the most common etiology of acute liver failure in children in the present study followed by Idiopathic. Early diagnosis is essential as outcome is worse without liver transplantation in Wilson's disease though majority of viral etiology are improved with supportive care.

Due to its rarity and unpredictability, PALF is still poorly understood and only has a limited body of research. Future research by hepatologists and intensivists that focuses on enhanced etiological

diagnosis, improved prognostic indicators, and the possibility of a medical cure should be given priority. The current best method for improving outcomes in patients with PALF is intensive supportive care, a methodical diagnostic approach, early transfer to a transplant Centre, and prompt liver transplantation.

REFERENCES

1. Bernal W, Auzinger G, Dhawan A, Wendon J. Acute liver failure. *Lancet* 2010;376.
2. Niraj J. Shah; Amor Royer; Savio John., University Of Mississippi Medical Center 2022;1.
3. Liver disease in children. In: Suchy FJ, Sokol RJ, Balistreri WF, editors. 4th ed. ed. Cambridge: Cambridge University Press; 2014.
4. Lutfi R, Abulebda K, Nitu ME, Molleston JP, Bozic MA, Subbarao G. Intensive care management of pediatric acute liver failure. *J Pediatr Gastroenterol Nutr.* 2017;64(5):660–670. doi: 10.1097.
5. Squires RH, Jr Acute liver failure in children. *Semin Liver Dis.* 2008;28(2):153–166. doi: 10.1055/s-2008-1073115.
6. Section of Pediatric Gastroenterology, Hepatology and Nutrition, Riley Hospital for Children, Indiana University School of Medicine, Indiana University, 705 Riley Hospital Drive, ROC 4210, Indianapolis, IN 46202 USA.
7. Bucuvalas JC. Acute liver failure in children. *Clinics in Liver Disease.* 2018;22(2):319–328. doi:10.1016/j.cld.2017.12.011.
8. Rolando N, Phillpott-Howard J, Williams R. Bacterial and fungal infection in acute liver failure. *Semin Liver Dis.* 1996;16(4):389–401.
9. Singhal A, Vadlamudi S, Stokes K, Cassidy FP, Corn A, Shrago SS, Wright HI, Kohli V. Liver histology as predictor of outcome in patients with acute liver failure. *Transpl Int.* 2012;25(6):658–662.
10. D'Antiga L, Dhawan A. Pediatric acute liver failure: new insights into diagnosis and management. *Current Opinion in Gastroenterology.* 2012;28(3):238–244. doi:10.1097/MOG.0b013e3283522c6f.
11. Teran JC, McCullough AF. Nutrition in liver diseases. In: Gottschlich MM, ed. *The Science and Practice of Nutrition Support*. Dubuque, IA: Kendall/Hunt; 2001:539.
12. Kliegman, R. (2020). *Nelson textbook of pediatrics* (Edition 21.);:2130.
13. Ganony WF. *Review of Medical Physiology*. 15th ed. East Norwalk, CT: Appleton and Lange; 1991:653.
14. Salmeron J, Tito L, Rimola A, et al. Selective intestinal decontamination in the prevention of bacterial infection in patients with acute liver failure. *J Hepatol.* 1992;14:280–285.sa
15. Kalicinski P, Kaminski A, Drewniak T, et al. Quick correction of hemostasis in two patients with fulminant liver failure undergoing liver transplantation by recombinant activated Factor VII. *Transplant Proc.* 1999;31:378–379.
16. Liver disease in children. In: Suchy FJ, Sokol RJ, Balistreri WF, editors. 4th ed. ed. Cambridge: Cambridge University Press; 2014.
17. Polson J Lee WM. AASLD position paper .the management of acute liver failure. *Hepatology* 2005;41.
18. Puja Amatyai I*, Sudeep Kumar Kapalavai, Akash Deep, Srinivas Sankaranarayanan et al Pediatric acute liver failure: An experience of a pediatric intensive care unit from resource limited settings. Volume 10:2022.
19. A.S. Vaanmathi, Dheivamani Nirmala, Bavanandam Sumathi CLINICAL PROFILE AND ETIOLOGY OF ACUTE LIVER FAILURE IN CHILDREN AGED 1 MONTH TO 12 YEARS. Volume-7 | Issue-9 | September-2018 | PRINT ISSN No 2250-1991.
20. Sharandeep kaur, preveenkumar et al Etiology and prognostic factors of acute liver failure in children. Vol. 50-july 15, 2013.
21. Tryambak Samanta, Sutapa Ganguly, Aetiology, clinical profile and prognostic indicators for children with acute liver failure admitted in a teaching hospital in kolkata.
22. Alyaa Yass, Mohammad Fadhil Ibraheem, Magid Kadhun Mohamed, Acute Liver Failure In Children: Etiology, Clinical Manifestation, Outcome. THE IRAQI POSTGRADUATE MEDICAL JOURNAL: VOL. 19, No. 1, 2020.
23. Songpon getsuwan, chatmanee lertudomphonwanit et al. Etiologies, Prognostic factors, and Outcome of pediatric acute liver failure in Thailand. *Pediatr gastroenterol hepatol nutr.* 2020 Nov; 23 (6):539-574.
24. Alina Grama, Cornel Olimpiu Aldea, Lucia Burac et al Etiology and Outcome of Acute Liver Failure in Children—The Experience of a Single Tertiary Care Hospital from Romania: children 2020, 7, 282.
25. A Yasmin, ASMB Karim, M Rukunuzzaman, K Hossen, L Nahar, Z F Sonia Aetiology, Clinical Profiles, Laboratory Profile, Outcome and Prognostic Factors of Pediatric Acute Liver Failure: Experience at a Tertiary Hospital of Bangladesh. *AKMMC J* 2019; 10(1): 56-61.