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Original Research

Assessment of cases of acute liver failure in children

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***ABSTRACT*:**
Background: Acute liver failure (ALF) is not a diagnosis but a clinical syndrome. The present study was conducted to assess cases of acute liver failure (ALF) in children. **Materials & Methods:** 60 children with acute liver failure of both genders were recruited. Laboratory parameters (transaminases, bilirubin, gammaglutamyl transferase, alkaline phosphatase, albumin, prothrombin time and INR, glycemia, complete blood count, and blood pH), and outcome of children with ALF was recorded. **Results:** Age group 0-12 months had 6 boys and 3 girls, 1-5 years had 10 boys and 12 girls, 6-18 years had 12 boys and 11 girls and >18 years had 2 boys and 4 girls. The etiology found to be drug-induced liver injury in 22, hereditary fructose intolerance in 5, mushroom poisoning in 7, herpes simplex virus type 1 infection in 10, hepatitis B virus infection in 8, type I autoimmune hepatitis in 4 and type II autoimmune hepatitis in 2 cases. The difference was significant (P< 0.05). **Conclusion:** Most common etiology was drug-induced liver injury, mushroom poisoning and herpes simplex virus type 1 infection.
Key words: Acute liver injury, drug-induced liver injury, mushroom poisoning

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INTRODUCTION

Acute liver failure (ALF) is a rare condition in pediatric pathology, with increased mortality (50%) despite optimal medical therapy in the absence of emergency liver transplantation.[1] ALF is a syndrome characterized by hepatic coagulopathy not-corrected by parenteral administration of vitamin K and the value of the INR (International Normalized Ratio) of 1.5–1.9 in the presence of hepatic encephalopathy or INR > 2.0 in the absence of hepatic encephalopathy in a child without the pre-existing liver disease.[2]

Acute liver failure (ALF) is not a diagnosis but a clinical syndrome. ALF was initially characterized in adults with biochemical evidence of severe hepatic dysfunction (e.g., jaundice and coagulopathy) complicated by hepatic encephalopathy that develops within 8 weeks of the onset of the signs and symptoms of liver disease.[3] Initial studies in children utilized the adult definition of ALF.[4] However, recognition of hepatic encephalopathy in children is difficult and may not be clinically apparent until the terminal stages of the disease process.[5] Thus, more recent single site reviews of ALF have included children without clinical encephalopathy. The Pediatric Acute Liver Failure (PALF) Study Group was formed in 2000 as a multisite, multinational consortium to prospectively study ALF in children from birth up to 18 years of age.[6] The present study was conducted to assess cases of acute liver failure (ALF) in children.

MATERIALS & METHODS

The present study comprised of 60 children with acute liver failure of both genders. Criteria for pediatric acute liver failure (PALF) were uncorrectable coagulopathy with INR > 1.5 in the presence of encephalopathy, or INR > 2 in the absence of hepatic encephalopathy. Parents of all neonates were informed regarding the study and their consent was obtained.

Data such as name, age, gender etc. was recorded. A thorough examination was done. Laboratory parameters

(transaminases, bilirubin, gammaglutamyl transferase, alkaline phosphatase, albumin, prothrombin time and INR, glycemia, complete blood count, and blood pH), and outcome of children with ALF was recorded. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Age group (years)	Boys	Girls	P value
0-12 months	6	3	0.05
1- 5 years	10	12	
6-18 years	12	11	
>18 years	2	4	

Table I shows that age group 0-12 months had 6 boys and 3 girls, 1-5 years had 10 boys and 12 girls, 6-18 years had 12 boys and 11 girls and >18 years had 2 boys and 4 girls. The difference was significant (P< 0.05).

Graph I Distribution of patients

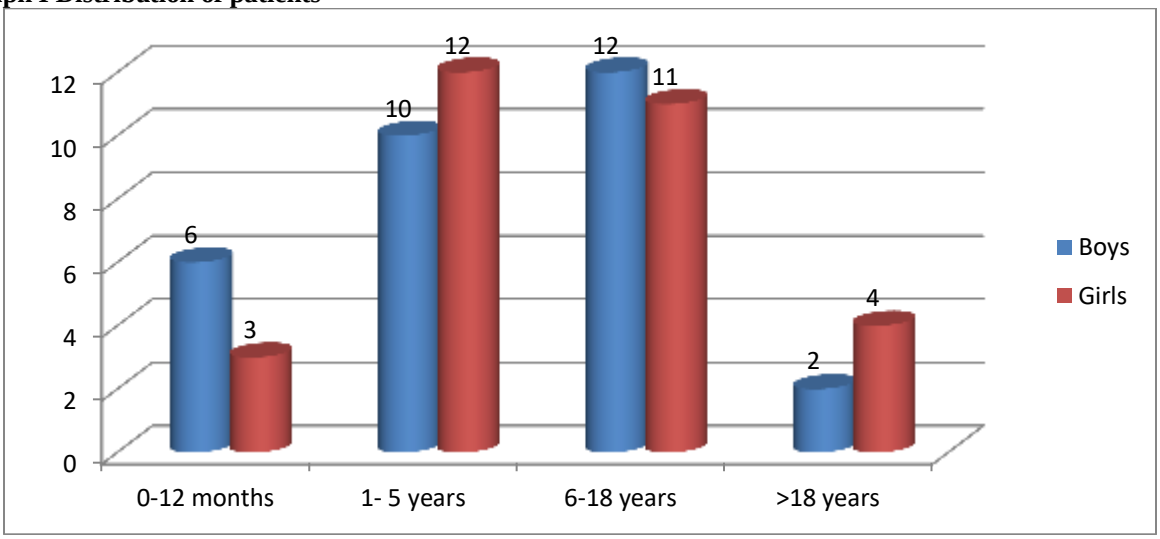
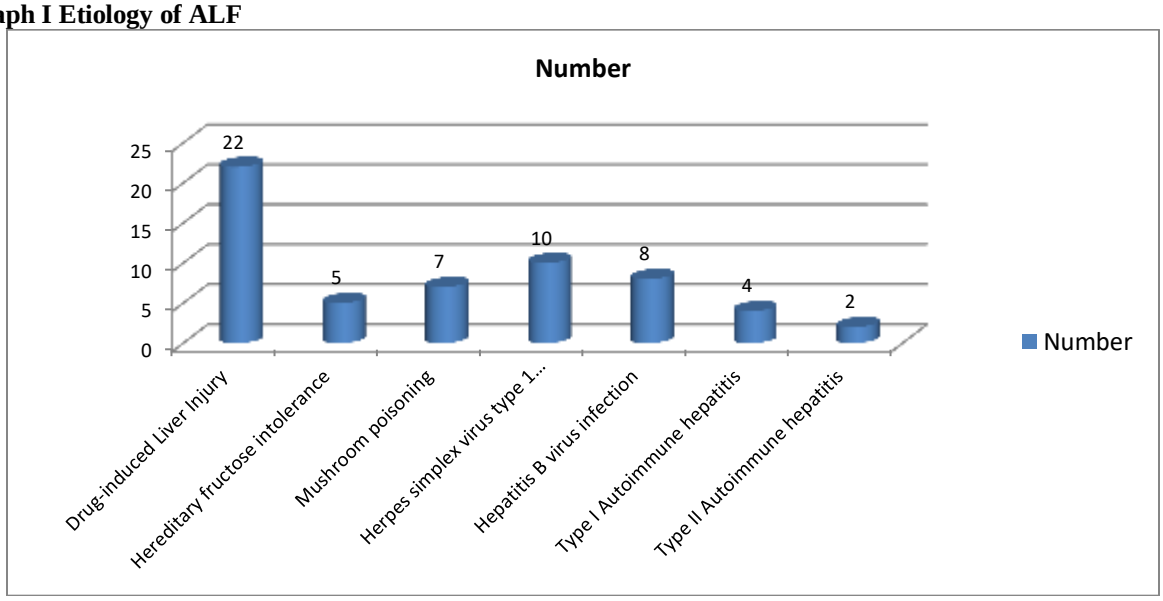


Table II Etiology of ALF

Etiology	Number	P value
Drug-induced Liver Injury	22	0.03
Hereditary fructose intolerance	5	
Mushroom poisoning	7	
Herpes simplex virus type 1 infection	10	
Hepatitis B virus infection	8	
Type I Autoimmune hepatitis	4	
Type II Autoimmune hepatitis	2	

Table II, graph II shows that etiology found to be drug-induced liver injury in 22, hereditary fructose intolerance in 5, mushroom poisoning in 7, herpes simplex virus type 1 infection in 10, hepatitis B virus infection in 8, type I autoimmune hepatitis in 4 and type II autoimmune hepatitis in 2 cases. The difference was significant (P< 0.05).

Graph I Etiology of ALF



DISCUSSION

Acute liver failure (ALF) is a rare disease, associated with high mortality, despite optimal medical therapy without emergency liver transplantation.[7] Knowing the possible cause of ALF plays a vital role in the management, as the child could benefit from effective specific therapies in emergencies.[8]

A consensus reached by 21 PALF investigators defined entry criteria of the study: no evidence of a known chronic liver disease; hepatic-based coagulopathy that is not corrected by parenteral administration of vitamin K; hepatic encephalopathy must be present if the uncorrected prothrombin time (PT) or international normalized ratio (INR) was between 15 and 19.9 seconds or 1.5 to 1.9, respectively; and hepatic encephalopathy was not required if the PT or INR was greater than or equal to 2.0 seconds or 2.0, respectively.[9,10] The present study was conducted to assess cases of acute liver failure (ALF) in children.

In present study, age group 0-12 months had 6 boys and 3 girls, 1-5 years had 10 boys and 12 girls, 6-18 years had 12 boys and 11 girls and >18 years had 2 boys and 4 girls. Sundaram et al[11] analyzed the etiology and outcome of ALF in children. The patients were grouped into different age categories: neonates (0–1 month), infants (1–12 months), children (1–14 years), and teenagers (14–18 years). 97 children (46 males, 47.42%, the mean age of 7.66 ± 8.18 years) were admitted with ALF. The most important causes of ALF were in neonates and infants, infections (72.72%), and metabolic disorders (43.47%), in children and adolescents were the toxic causes (60% and 79.41%). The mortality rate was 31.95% (31 patients), mainly in ALF due to infections or metabolic disorders. In neonates and infants, the main causes of ALF were infections and metabolic diseases, while in older children and teenagers, were toxin-induced liver injuries. The mortality among neonates and infants was significantly higher than in other ages. Early recognition and immediate therapeutic intervention could improve the outcome of these patients.

We found that etiology found to be drug-induced liver injury in 22, hereditary fructose intolerance in 5, mushroom poisoning in 7, herpes simplex virus type 1 infectionin 10, hepatitis B virus infection in 8, type I autoimmune hepatitis in 4 and type II autoimmune hepatitis in 2 cases. Alam et al[12] in their study the etiological spectrum of acute liver failure in infants and young children and to identify clinical and biochemical markers for metabolic liver disease (MLD). Comparison analysis (MLD vs. non MLD) of the clinical and biochemical parameters was done. There were 30 children under 3 y of age with acute liver failure (ALF) with median age of 12.5 mo. Fifteen children were less than12 mo. MLD (33%)and hemophagocytic lymphohistiocytosis (HLH) (17 %) together accounted for half of the cases of ALF in children below 3 y of age. The other co mon etiologies were drug induced liver injury and acute viral hepatitis A. Etiology remained indeterminate in 3 cases (10 %). Comparative analysis of the clinical and biochemical parameters between MLD and non MLD group showed significant difference between the two groups in the median values of age ($p = 0.014$), bilirubin ($p = 0.017$), jaundice to encephalopathy (JE) interval ($p = 0.039$) and blood sugar ($p = 0.001$). Suggestive family history, developmental delay, presence of diarrhea/vomiting in the history and presence of urinary non glucose reducing substance (NGRS) were also significantly associated with MLD group. Only 40 % children

survived with native liver. MLD and HLH account for majority of ALF in infants. About 10 % of cases remain indeterminate. Viral hepatitis is more common in young children. Apart from clinical indicators, young age, high bilirubin, synthetic dysfunction, low sugar and NGRS in urine indicate MLD as a cause. Survival with native liver is low.

The shortcoming of the study is small sample size.

CONCLUSION

Most common etiology for acute liver failure was drug-induced liver injury, mushroom poisoning and herpes simplex virus type 1 infection.

REFERENCES

1. Devictor, D.; Tissieres, P.; Afanetti, M.; Debray, D. Acute liver failure in children. In Clinics and Research in Hepatology and Gastroenterology; Poupon, R., Arrive, L., Ballet, F., Eds.; Elsevier: Paris, France, 2011; 35: 430–437.
2. Squires, S.R. Acute Liver Failure in Children. Semin. Liver Dis. 2008; 28, 153–166. 3. Grama, A.; Burac, L.; Cainap, S.S.; Aldea, C.; Delean, D.; Bulata, B.; Pacurar, D.; Bodea, L.; Sirbe, C.; Pop, T.L. Acute liver failure in children: Aetiology and evolution. Arch. Dis. Child. 2018; 104, A1–A428.
3. Withington, F.P.; Alonso, M.W. Fulminant Hepatitis and Acute Liver Failure. In Diseases of the Liver and Biliary System in Children, 3rd ed.; Deirdre, K., Ed.; Wiley-Blackwell: Oxford, UK, 2008; pp. 92–123.
4. Grama, A.; Aldea, C.; Burac, L.; Delean, D.; Boghitoiu, D.; Bulata, B.; Nitescu, V.; Ulmeanu, C.; Pop, T. Acute liver failure secondary to toxic exposure in children. Arch. Med. Sci. 2019; 15. 5. Dhawan, A. Etiology and Prognosis of Acute Liver Failure in Children. Liver Transplant. 2008; 14, S80–S84.
5. Góra-G ¸ebka, M.; Liberek, A.; Sikorska-Wi´sniewska, G.; Rytlewska, M.; Bako, W.; Korzon, M. Acute liver failure and acquired aplastic anaemia in children-life-threatening clinical entities of common etiopathogenesis? Med. Sci. Monit. 2003; 9: 64–67.
6. Colleti, J., Jr.; Azevedo, R.T.; de Calvalho, W.B. Pediatric Acute Liver Failure: Current Perspectives. Liver Res. 2017; 2: 14–15.
7. Dhawan, A. Acute Liver Failure in children and adolescents. Clin. Res. Hepatol. Gastroenterol. 2012; 36: 278–283.
8. Bitar, R.; Thwaites, R.; Davison, S.; Rajwal, S.; McClean, P. Liver Failure in Early Infancy: Aetiology, Presentation, and Outcome. J. Pediatr. Gastroenterol. Nutr. 2017; 64: 70–75.
9. Squires, R.H., Jr.; Shneider, B.L.; Bucuvalas, J.; Alonso, E.; Sokol, R.J.; Narkewicz, M.R.; Dhawan, A.; Rosenthal, P.; Rodriguez-Baez, N.; Murray, K.F.; et al. Acute liver failure in children: The first 348 patients in the paediatric acute liver failure study group. J. Pediatr. 2006; 148: 652–658.
10. Sundaram, S.S.; Alonso, E.M.; Narkewicz, M.R.; Zhang, S.; Squires, R.H. Characterization and outcomes of young infants with acute liver failure. J. Pediatr. 2011; 159: 813–818.
11. Alam S, Lal BB, Khanna R, Sood V, Rawat D. Acute liver failure in infants and young children in a specialized pediatric liver centre in India. The Indian Journal of Pediatrics. 2015 Oct;82(10):879-83.