DS (Child) H J 2024; 40(1): 30-34

ORIGINAL ARTICLE

Etiological and Clinical Profile of Acute Hepatitis in Children: Experience of A Tertiary Care Hospital in Bangladesh

Emdadul Haque[1], Ahmed Rashidul Hasan[2], Tanzila Farhana[2], Salahuddin Mahmud[3]

Abstract

Background: *Acute hepatitis is a major public health concern, affecting millions of children worldwide . The four most frequent viruses that cause infection are hepatitis A, B, C, and E. The etiology of acute viral hepatitis varies depending upon several epidemiological and individual factors.*

Objective: *The study aimed to determine the etiology and clinical characteristics of acute hepatitis in pediatric patients in a tertiary care hospital.*

Methods: *This cross-sectional study was conducted in the Department of Pediatric Gastroenterology, Hepatology, and Nutrition, Bangladesh Shishu Hospital & Institute (BSH&I) from January 2023 to January 2024. A total of 50 children aged 1 to 12 years, presenting with acute hepatitis of various etiologies, were included. Acute hepatitis was diagnosed based on clinical manifestation (jaundice lasting <3 months without any stigmata of chronic liver disease) and biochemical evidence of elevated alanine aminotransferase (ALT) and aspartate transaminase (AST). Specific serological markers were used to determine the etiology of acute hepatitis. Data were analyzed by using SPSS version 25*

Results: *Acute hepatitis was common in children aged 6- to 10-year-old age group (52%), with a mean age of 8.9±3.3 years. The majority of affected children were male (62%), while females accounted for 38%. Common clinical features were jaundice (92%), nausea and vomiting (88%), abdominal pain (70%), hepatomegaly (80%), fever (64%), dark urine (62%), yellow eyes (58%), and decreased appetite (24%).*

The Etiological profile showed:Hepatitis A virus is 35 (70%) had hepatitis A virus, 5 (10%) had hepatitis E virus, 2 (4%) had hepatitis B virus, 3 (6%) had Salmonella hepatitis, 2 (4%) had Wilson's disease, and 1 (2%) had hepatitis C virus, hepatitis A+E virus, anti-TB, and valproic acid each.

Conclusion: *The most prevalent etio-logical agent was hepatitis A, followed by hepatitis B and hepatitis E. Additionally, a mixed hepatitis A and E infection was observed . Common clinical features were jaundice, nausea & vomiting, abdominal pain, hepatomegaly, fever, dark-colored urine, yellow eyes, and decreased appetite.*

Keyword: *Acute hepatitis, clinical feature, etiological profile.*

1 Associate Professor (Current Charge), Department of Paediatric Gastroenterology, Hepatology & Nutrition, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh.

2 Assistant Professor, Department of Paediatric Gastroenterology, Hepatology & Nutrition, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh.

3 Professor, Department of Paediatric Gastroenterology, Hepatology & Nutrition, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh.

Correspondence to: Dr. Emdadul Haque, Associate Professor (Current Charge), Department of Paediatric Gastroenterology Hepatology and Nutrition, Bangladesh Shishu Hospital & Institute, Sher-E-Bangla Nagar, Dhaka-1207, Bangladesh. Cell: 01711884176, E-mail: emdadulhaque25012018@gmail.com

Received: 5 February 2024; **Accepted**: 2 May 2024

Introduction

Acute hepatitis is a significant public health problem throughout the world, affecting millions of children.[1] The incidence of acute hepatitis in developing countries remains high despite the availability of vaccines, preventive measures, and improved sanitation.[2] Hepatitis is a systemic infection affecting the liver predominantly and causing its inflammation. It may be acute or chronic. It is caused by infection with one of the five known hepatotropic viruses, which are named as hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis D virus (HDV), and hepatitis E virus (HEV).[3] HDV is considered to be a subviral satellite, as it cannot produce infection without concurrent hepatitis B infection.[4,5] The etiology of acute hepatitis varies depending upon several epidemiological and individual factors.[6,7]

The clinical manifestations of acute hepatitis range from a completely subclinical and invisible infection to fulminant and rapidly progressive liver failure. [2] The majority of symptoms described in the weeks leading up to hospital admissions were abdominal pain, vomiting, and diarrhea.[6] Together with jaundice, very high levels of liver enzymes (ALT and AST) were found. High levels of serum aminotransferases, which exceed 500 IU/L, are a remarkable feature.[8,9]

In the pediatric age group, acute liver failure is defined as the presence of biochemical signs of liver damage and coagulopathy that are not corrected by a single dose of parenteral vitamin K with an International Normalized Ratio (INR) > 1.5 in the presence of encephalopathy or INR > 2 without signs of encephalopathy within 8 weeks of onset of liver injury, without prior known liver disease.[6] The consequences of acute liver failure also vary depending on the etiology: survival is better in different etiologies such as hepatitis A or paracetamol intoxication, while in metabolic diseases it is poor and requires liver transplantation in a specialized center. [10,11] This study aims to identify the etiology and clinical presentations of acute hepatitis in pediatric patients in a tertiary care hospital.

Materials and Methods

This cross-sectional study was conducted in the Department of Pediatric Gastroenterology, Hepatology, and Nutrition, Bangladesh Shishu Hospital & Institute (BSH&I) from January 2023 to January 2024. A total of 50 children, aged 1 to 12 years, presenting with acute hepatitis of various etiologies were included in the study. Acute hepatitis was diagnosed based on clinical manifestations (jaundice <3 months without any stigmata of chronic liver disease) and elevated biochemical markers, including of alanine aminotransferase (ALT) and aspartate transaminase (AST). Acute hepatitis A and E were diagnosed by positive anti-HAV IgM and anti-HEV IgM, respectively. Acute hepatitis B was diagnosed by positive anti-HBc IgM and HBsAg. Acute hepatitis C was diagnosed by negative anti-HCV and HCV-RNA. Acute hepatitis due to HSV was diagnosed by positive anti-HSV IgM. Salmonella hepatitis, or enteric fever, was diagnosed as a positive blood culture for S. typhi/S. para-typhi and very high titers of antibodies to Salmonella O antigen and negative serological markers for viral hepatitis. Drug-induced liver injury was diagnosed by elevated levels of ALT, AST, and ALP with positive drug exposure and exclusion of other causes of hepatitis. Non-A-E hepatitis was diagnosed by exclusion of viral etiology with enteric fever, malaria, dengue, hemolytic anaemia , Wilson disease, drug-induced liver injury, and wasp bite also included in the study. The relevant clinical findings of these patients were evaluated. Data analysis was performed using the Statistical Package for Social Science (SPSS) version 25.0 for Windows.

Results

Acute hepatitis was common among children aged 6-to 10-year-old age group (52%), with a mean age of 8.9±3.3 years. The majority of affected children were male (62%), while females accounted for 38% (Table I).

Table I
Demographic characteristics of the study patients (n=50)

	Frequency	Percentage
Age (years)		
≤5.0	13	26.0
6.0-10.0	26	52.0
11.0-12.0	11	22.0
Mean ±SD	8.9±3.3	
Range (min-max)	1.0-12.0	
Sex		
Male	31	62.0
Female	19	38.0

Common clinical features were jaundice, nausea & vomiting, hepatomegaly, abdominal pain, fever, dark-colored urine, yellow eyes, and decreased appetite.

46 (92%), 44 (88%), 40 (80%), 35 (70%), 32 (64%), 31 (62%), 29 (58%), and 12 (24%), respectively (Table II).

Table II
Clinical feature of the study patients (n=50)

	Frequency	Percentage
Jaundice	46	92.0
Nausea & vomiting	44	88.0
Abdominal pain	35	70.0
Fever	32	64.0
Dark colored urine	31	62.0
Yellow eyes	29	58.0
Decreased appetite	12	24.0
Pale stool	8	16.0
Pruritis	3	6.0
Constipation	1	2.0
Hepatomegaly	40	80.0
Splenomegaly	8	16.0
Ascites	3	6.0

Total bilirubin 5-10 mf/dl 22(44%), ALT 500-1500 U/L 32(64%), AST 500-1500 U/L 35(70%), Prothrombin time (sec)>15sec12(24%) and Serum albumin (gm/dl) <3.5gm/dl were 4(8%) respectively (Table III).

Table III
Laboratory parameters of the study patients (n=50)

	Frequency	Percentage
Total serum bilirubin (mg/dl)		
<5	26	52.0
5-10	22	44.0
>10	2	4.0
ALT (U/L)		
<500	15	30.0
500-1500	32	64.0
>1500	3	6.0
AST (U/L)		
<500	15	30.0
500-1500	35	70.0
Prothrombin time (sec)		
≤15	38	76.0
>15	12	24.0
Serum albumin (gm/dl)		
>3.5	46	92.0
<3.5	4	8.0

Regarding vaccination status, 32 (64%) were vaccinated against hepatitis B, and none were vaccinated against hepatitis A (Table IV).

Table IV
Vaccination status of the study patients (n=50)

Vaccination status	Frequency	Percentage
Taken hepatitis B	32	64.0
Taken hepatitis A	0	0.0
Not taken vaccine	18	36.0

The etiological profile of acute hepatitis revealed that 35 patients (70%) had hepatitis A virus, 5(10%) had hepatitis E virus, 2(4%) had hepatitis B virus, 3(6%) had Salmonella hepatitis, and 2(4%) had Wilson's disease. Additionally, one patient (2%) each had hepatitis C virus, co-infection with hepatitis A and E viruses, anti-tubercular drug-induced hepatitis, and valproic acid-induced hepatitis (Table V).

Table V
Etiology profile of acute hepatitis of the study patients (n=50)

Vaccination status	Frequency	Percentage
Hepatitis A virus	35	70.0
Hepatitis E virus	5	10.0
Hepatitis B virus	2	4.0
Hepatitis C virus	1	2.0
Hepatitis A+ E virus	1	2.0
Salmonella hepatitis	3	6.0
Wilson's disease	2	4.0
Anti-TB and valproic acid	1	2.0

Discussion

This study observed a mean age was 8.9±3.3 years, with the majority were male (62%). Acute hepatitis was common in the 6-10 year age group (52%), followed by children younger than 5 year age group (26%).

Tasneem et al[12] reported similar finding's with acute hepatitis occurring more frequently in children aged 5-10-year (56.6%) age group, followed by those aged 10-15-year (34.5%) with 66.0% were male. The mean age of presentation was 8.08±3.27 years. Girish et al[13], Das et al[14], and Kamath et al[15] also reported similar results. Mahmud et al[16] from Bangladesh

also found that 70.9% of children were exposed to HAV within 5 years of age, 70.9% were exposed within 10 years of age, and 92.6% were exposed within 15 years of age.

Common clinical features were jaundice (92%), nausea & vomiting (88%), abdominal pain (70%), hepatomegaly (80%), fever (64%), dark-colored urine (62%), yellow eyes (58%), and decreased appetite (24%). Tasneem et al[12] reported many of the children had jaundice (100%); more bacterial cases (100%) had fever than viral cases (52.1%); and abdominal pain was more common in bacterial (83%) cases than viral (50.1%) cases. Hepatomegaly was present in the majority (94.3%) of cases, and splenomegaly was present in a few (27.7%) cases. In 4.6% of cases, signs of ascites were present with the evidence of fluid thrill and shifting dullness. Patel et al[1] reported the most common symptoms at presentation were fever (94%), abdominal pain (78%), vomiting (76%), dark-colored urine (76%), yellow eyes (73%), and decreased appetite observed in 61% of patients. About 98% of patients had jaundice at the time of presentation. About 75% had hepatomegaly, and 18% had hepatosplenomegaly. Pallor was found in 24%, and liver tenderness was present in 20% of cases. Bradycardia (12%) and ascites (10%) were less common.

Total bilirubin level was found to be 5-10 mg/dl in 44%, ALT 500-1500 U/L in 64%, AST 500-1500 U/L in 70%, prothrombin time (sec) >15 sec in 24%, and serum albumin (gm/dl) 5 gm/dl in (8%), according to the current study. Compared with Patel et al[1], they reported the highest total serum bilirubin noted was 16.1 mg/dL. Bilirubin more than 10 mg/dL was seen in 3 children, in which one had prolonged cholestatic viral hepatitis A while another one had mixed infection with viral hepatitis A+E. More than 1000 U/L of ALT and AST were seen in 41% of viral hepatitis A. Maximum ALT and AST were observed in one 13-year-old female with viral hepatitis A, 4164.5 U/L and 6065.5 U/L, respectively.

This study showed that 64% of participant were vaccinated against hepatitis B, while none had received vaccination against hepatitis A. Similarly, Patel et al[1] reported about 88% of admitted patients were vaccinated against hepatitis B, while none of the study population was vaccinated against hepatitis A.

In this study, the hepatitis A virus was found to be the most common cause of acute hepatitis, accounting for 70% of cases, followed by the hepatitis E virus (10%), Salmonella hepatitis (6%), the hepatitis B virus (4%), Wilson's disease (4%), and the hepatitis C virus (2%). Additionally, 2% of cases were associated with co-infection of hepatitis A and E viruses, as well as with anti-tuberculosis (anti-TB) drugs and valproic acid. Similarly, Tasneem et al[12] reported that among acute hepatitis cases, the majority (71.1%) tested positive for anti-HAV IgM, followed by 6.6% for hepatitis E. Co-infections were observed in 3.3% of cases, including combined HAV and HEV infections and salmonella hepatitis. One case involved a combination of HAV, CMV, and Wilson's disease. Notably, 12.2% of cases in their study had an undetermined etiology. Sarmin et al[2] reported the most common cause of acute hepatitis was hepatitis A virus, followed by hepatitis E virus, non-A and non-B, and hepatitis B virus. HAV with HEV co-infection was found in 2.5% of cases. Other than viral hepatitis salmonella-positive (6%) cases, drug-induced hepatitis due to anti-TB and valproic acid (2.5%) cases, Wilson's disease (2.5%) cases, and wasp bites (1.7%). Sarker et al[6] from Bangladesh, Sudhamshu et al[17] from Nepal, and Behera et al[7] & Poddar et al[18] from India also found similar results.

Conclusion

The current study found hepatitis A is the most common etiological agent, followed by hepatitis E and hepatitis B. Mixed infection with hepatitis A and E were also observed. Common clinical features were jaundice, nausea & vomiting, abdominal pain, hepatomegaly, fever, dark-colored urine, yellow discoloration of the eyes, and decreased appetite.

References

1. Patel MB, Sivanandan S, Prajapati BS. Study of profile of acute viral hepatitis in hospitalized children at a tertiary care center in Gujarat. *National Journal of Physiology, Pharmacy and Pharmacology* 2024;**14**:390-94.

2. Sarmin Z, Sultana K, Shamsad IA, Glory P, Hena H, Jahan SL, Islam MM, Hossen K. Clinical Presentation and Etiology of Acute Hepatitis In Children: An Experience From A Tertiary Centre of Bangladesh. *Journal of Dhaka Medical College* 2023;**32**:32-37.

3. National Centre for Disease Control. Viral Hepatitis: The Silent Disease- Prevention, Control and

Treatment Guidelines. New Delhi: National Centre for Disease Control; 2016. Available from: guideline_hep20158117187417.

4. World Health Organization. Regional Strategy for the Prevention and Control of Viral Hepatitis: WHO Regional Office for South-East Asia. Geneva: World Health Organization; 2013. Available from: https://iris.who.int/handle/10665/205846.
5. Kliegman R, Geme S, Shah B. Nelson Textbook of Pediatrics. 21st ed. Philadelphia, PA: Elsevier; 2020. p.2106-18.
6. Sarker NR, Saha SK, Ghosh DK, Adhikary A, Mridha A, et al. Seropositivity of viral markers in icteric children. *Bangladesh Med J* 2014;**43**:26-29.
7. Behera MR, Patnaik L. Clinico-biochemical profile and etiology of acute viral hepatitis in hospitalized children: A study from Eastern India. *Indian J Child Health* 2016;**3**: 317-320.
8. Zhang LY, Huang LS, Yue YH, Fawaz R, Lim JK, Fan JG. Acute hepatitis of unknown origin in children: early observations from the 2022 outbreak. *Journal of clinical and translational Hepatology* 2022;**10**:522-30.
9. Marsh K, Tayler R, Pollock L, Roy K, Lakha F, Ho A. et al. Investigation into cases of hepatitis of unknown etiology among young children. *Eurosurveillance*. 2022;**27**: 2200318. DOI: 10.2807/1560-7917.ES. 2022.27. 15.2200318.
10. Nandi GCB, Hadimani MP, Arunachalam CR, Ganjoo RK.Spectrum of acute viral hepatitis in Southern India. *Med J Arm for India* 2008;**65**:7-9.
11. Jain P, Prakash S, Gupta S, Singh KP, Shrivastava S, Singh DD, et al. Prevalence of hepatitis A virus, hepatitis B virus, hepatitis C virus, hepatitis D virus and hepatitis E virus as causes of acute viral hepatitis in North India: A hospital based study. *Indian Journal of Medical Microbiology* 2013;**31**:261-65.
12. Tasneem F, Imteaz SM, Farhana T, Hasan AR, Haque E, Mahmud S. Clinico-pathological profile of acute hepatitis in children: a tertiary center experience from Bangladesh. *Gastroenterol Hepatol Open Access* 2025;**16**:35-40.
13. Girish N, Sunil B, Devaranavadagi RA. A clinical study of viral hepatitis in children: A prospective hospital-based study. *Int J Contemp Pediatr* 2018;**5**:563-68.
14. Das S, Deka A, Biswas T. Clinical profile of acute viral hepatitis in children - in Southern Assam. *J Med Sci Clin Res* 2021;**9**:111-18.
15. Kamath SR, Sathiyasekaran M, Raja TE, Sudha L**.** Profile of viral hepatitis A in Chennai. *Indian Pediatr* 2009;**46**:642-43.
16. Mahmud S, Ahmed SS, Hussain M, Afroz M, Tasneem F. Recent spectrum of acute viral hepatitis in children: An experience in a tertiary centre of Bangladesh. *Adv Res Gastroenterol Hepatol* 2017; 6(3):555686. DOI: 10.19080/argh.2017.06. 555686.
17. Sudhamshu KC, Dilip Sharma DS, Poudyal N, Basnet BK. Acute viral hepatitis in pediatric age groups. *J Nepal Med Assoc* 2014;**52**:687-91.
18. Poddar U, Thapa BR, Prasad A, Singh K. Changing spectrum of sporadic acute viral hepatitis in Indian children. *Journal of Tropical Pediatrics* 2002;**48**: 210-13.