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Original Research Article

Clinical profile of hepatitis A virus infection in children in a tertiary care hospital

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ABSTRACT

Background: Hepatitis A virus (HAV) infection is a frequent form of hepatitis primarily affects the liver and is commonly observed in children particularly in areas with limited access to clean water and sanitation. The disease is characterized by a variety of clinical manifestations including jaundice, fever, and abdominal pain. This study aims to evaluate the clinical, and biochemical of HAV infection in children in a tertiary care hospital.
Methods: This prospective observational study was conducted at Bangladesh Shishu hospital and institute, Dhaka, Bangladesh from January 2023 to December 2023. The study aimed to investigate the clinical and biochemical spectrum of HAV infection in children at the department of paediatric gastroenterology, hepatology and nutrition. A total of 200 children with confirmed HAV infection were enrolled in the study. Data were analyzed using SPSS version 26 (IBM Corp., Armonk, NY). Descriptive statistics were used to summarize the clinical and biochemical characteristics of the study population.
Results: Clinical features were dominated by jaundice and hepatomegaly (100%), followed by fever (98.5%), nausea (98%), anorexia (98%), and abdominal pain (92.5%). Cholestasis was the most common complication (9.5%), followed by acute liver failure (4.5%). Laboratory findings showed that 86.5% had total serum bilirubin levels ≤10 mg/dl, 67.5% had serum ALT levels between 500-1500 IU/L, and 4.5% had an INR >1.5.
Conclusions: This study provides a comprehensive analysis of the clinical and biochemical spectrum of HAV infection in children at a tertiary care hospital. The findings highlight the typical presentation of HAV in children, including symptoms such as jaundice, fever, vomiting, and abdominal pain, alongside elevated serum ALT and bilirubin levels. Despite the generally self-limiting nature of the disease, complications such as cholestasis and acute liver failure were observed, underlining the importance of early diagnosis and appropriate management and emphasize the necessity of including hepatitis A vaccination in routine immunization programme.

Keywords: Clinical features, Biochemical spectrum, Hepatitis A virus, Jaundice

INTRODUCTION

Hepatitis A is an infectious viral disease caused by the HAV, a member of the *Picornaviridae* family. It is primarily transmitted via the fecal-oral route, typically through contaminated water, food, or close person-to-person contact. While hepatitis A is generally self-limiting, it can lead to severe complications, particularly in children and individuals with pre-existing liver conditions. Despite the availability of an effective

vaccine, hepatitis A remains a significant public health concern in many parts of the world, particularly in low- and middle-income countries (LMICs), where sanitation and hygiene practices may be inadequate. In these regions, hepatitis A poses a considerable burden on healthcare systems, leading to hospitalization, morbidity, and in some cases, mortality.[1,2] The clinical manifestation of hepatitis A in children is variable, ranging from asymptomatic infection to severe acute liver failure. Children are often more likely to develop asymptomatic or mild cases of hepatitis A, which contrasts with adults who typically experience more severe disease. However, the clinical spectrum can vary depending on the child's age, nutritional status, and immune response. Children may present with nonspecific symptoms such as fatigue, nausea, abdominal pain, and jaundice. Therefore, clinical evaluation supported by laboratory investigations is crucial for accurate diagnosis and management.[3,4] Biochemically, hepatitis A infection is characterized by an elevation in liver enzymes, specifically alanine aminotransferase (ALT) and aspartate aminotransferase (AST), along with the presence of specific serological markers. The detection of anti-HAV IgM antibodies indicates recent infection, while the presence of anti-HAV IgG antibodies suggests past exposure or vaccination. The rise in liver enzymes during acute infection reflects hepatocellular damage, and the pattern of enzyme elevation can provide insight into the severity of liver injury. In some cases, biochemical markers may also indicate a risk of developing complications, such as fulminant hepatic failure, though this is rare in children.[5] A key aspect of managing hepatitis A in children is timely diagnosis and supportive care, as there is no specific antiviral therapy available. Most children recover completely with appropriate symptomatic management, including adequate hydration, rest, and nutritional support. However, early identification of at-risk individuals, such as those with chronic liver disease, can help prevent severe outcomes.[6] Additionally, the introduction of the hepatitis A vaccine has been a crucial measure in reducing the incidence of new infections, particularly in areas where vaccination is part of the national immunization program.[7] In Bangladesh, hepatitis A is endemic, with periodic outbreaks occurring in various regions, particularly in areas with poor sanitation and limited access to clean drinking water. The prevalence of HAV infection among children in Bangladesh has been reported to be high, with several studies indicating that most children are exposed to the virus at an early age. A study conducted by Khan et al. found that the seroprevalence of anti-HAV antibodies was 79% among children in rural areas of Bangladesh.[8] Other studies have demonstrated that the incidence of hepatitis A infection is highest in children between the ages of 5 to 14 years.[7] Furthermore, the clinical outcomes of hepatitis A in Bangladeshi children have been reported to be generally favourable, with most cases resolving without complications.[8] Despite the overall favourable prognosis, complications such as acute liver failure, though rare, can occur, particularly in children with co-existing conditions such as malnutrition, underlying chronic liver disease, or immunocompromised status.[9] Therefore, it is essential to establish effective surveillance systems to monitor the prevalence and clinical outcomes of hepatitis A infections among children in Bangladesh. Furthermore, while the hepatitis A vaccine is available, its uptake remains limited in some regions due to challenges related to vaccine accessibility and awareness. Increased public health efforts are required to improve vaccination coverage and reduce the burden of hepatitis A in the paediatric population.

METHODS

This prospective observational study was conducted at Bangladesh Shishu hospital and institute, Dhaka, Bangladesh from January 2023 to December 2023. The study aimed to investigate the clinical and biochemical spectrum of HAV infection in children at the department of paediatric gastroenterology, hepatology and nutrition. Children aged 1 to 15 years who were diagnosed with acute hepatitis A during the study period were eligible for inclusion. A total of 200 children with confirmed HAV infection were enrolled in the study. A detailed demographic profile was taken from the caregivers, including the child's age, gender, socio-economic status, place of residence (urban or rural), and family history. Clinical data including the presenting symptoms and physical examination findings were collected. Common symptoms such as fever, vomiting, jaundice, nausea, anorexia, abdominal pain, and constipation were documented. The physical signs noted on examination included jaundice, hepatomegaly, abdominal tenderness, ascites, splenomegaly, and elevated body temperature. Total and direct serum bilirubin (mg/dl), serum ALT (IU/L), serum AST (IU/L), serum albumin (g/dl), prothrombin time (PT) and International Normalized Ratio (INR) were measured. Data were analyzed using SPSS version 26 (IBM Corp., Armonk, NY). Descriptive statistics were used to summarize the clinical and biochemical characteristics of the study population. Categorical variables, such as gender, symptoms, and complications, were presented as frequencies and percentages. Parental or guardian consent was obtained for all participants, and patient confidentiality was maintained throughout the study. Personal identifiers were removed from the data to ensure anonymity.

Inclusion criteria

Children eligible for inclusion in the study were those aged 1 to 15 years who had been diagnosed with acute hepatitis A. The diagnosis was confirmed by the presence of anti-HAV IgM antibodies in serum, detected through enzyme-linked immunosorbent assay (ELISA). Additionally, only children who had experienced the onset of symptoms within the last two weeks were considered. Written informed consent was obtained from the parents or guardians before participation in the study.

Exclusion criteria

Children were excluded from the study if they had pre-existing chronic liver diseases such as chronic hepatitis B or C, cirrhosis, or biliary atresia. Those with co-infections involving other hepatitis viruses, including hepatitis B, C, or E, were also not eligible. Additionally, children with immunocompromised conditions, such as HIV or those receiving immunosuppressive therapy were excluded. Participation was further restricted to children who were willing to take part in study, with consent obtained from their guardians. Lastly, children with other significant comorbidities, such as malignancies/metabolic disorders that could affect liver function, were excluded.

RESULTS

Table 1 presents the demographic distribution of the study population (n=200). The majority of patients (53.5%) were between 5-10 years old, with a nearly equal distribution in the <5 years (22.5%) and >10 years (24%) age groups. Males comprised 55.5% of the participants, while females accounted for 44.5%. The study population was almost evenly split between urban (53.5%) and rural (46.3%) areas. In terms of socio-economic status, the largest group belonged to the low-middle class (47.5%), followed by the upper-middle class (28.5%), low-class (17.5%), and high-class (6.5%). Tap water was primary drinking source (39.5%), followed by underground water (25.5%), boiled water (20.5%), and unboiled water (14.5%). A positive family history was noted in 13.5% of cases, and while 21.5% consumed only homemade food, 78.5% had a mixed diet of homemade and outside food.

Table 2 summarizes the clinical features of the study population (n=200), including both symptoms and signs. The most common symptoms were yellowish discoloration of the skin and sclera (100%), fever (98.5%), nausea (98%), anorexia (98%), and abdominal pain (92.5%). Vomiting was reported in 95.5% of cases, while constipation (10.5%) and pruritus (2.5%) were less frequent. Among clinical signs, jaundice and hepatomegaly were present in all patients (100%), while elevated temperature was observed in 98.5%. Abdominal tenderness was noted in 86% of cases, with less frequent findings of splenomegaly (6%) and ascites (2.5%).

Table 3 outlines the frequency of complications observed in the study population (n=200). Cholestasis was the most common complication, affecting 9.5% of patients, followed by acute liver failure in 4.5%. Less frequent complications included ascites (2.5%), relapse (2%), and cholangitis (1.5%). Mortality was recorded in 1% of case.

Table 4 presents laboratory findings of study population (n=200). The majority of patients (86.5%) had total serum bilirubin levels up to 10 mg/dl, while 10.5% had levels between 10-20 mg/dl, and 3% had levels above 20 mg/dl. Direct bilirubin was ≤2 mg/dl in 85% of cases, with only 1% exceeding 10 mg/dl. Serum ALT levels were elevated in most patients, with 67.5% having values between 500-1500 IU/L and 12% exceeding 1500 IU/L. INR was ≤1.3 in 78% of patients, 4.5% had INR >1.5, indicating impaired coagulation. Serum albumin was >3 g/dl in 79.5% of cases, with only 3.5% showing levels <2 g/dl, reflecting varying degrees of liver dysfunction.

Table 1: Demographic distribution of study population, (n=200).

Category	N	Percentage (%)
Age group (in years)		
<5	45	22.5
5-10	107	53.5
>10	48	24.0
Gender		
Male	111	55.5
Female	89	44.5
Area of residence		
Rural	93	46.3
Urban	107	53.5
Socio-economic status		
Low	35	17.5
Low middle class	95	47.5
Upper middle class	57	28.5
High	13	6.5
Source of drinking water		
Tap water	79	39.5
Underground water	51	25.5
Boiled	41	20.5
Unboiled	29	14.5
Family history		
Positive family history	27	13.5
Homemade food only	43	21.5
Both homemade and outside food	157	78.5

Table 2: Clinical features of study population, (n=200).

Clinical feature	N	Percentage (%)
Symptoms		
Fever	197	98.5
Vomiting	191	95.5
Yellowish discoloration of skin and sclera	200	100.0
Nausea	196	98.0
Anorexia	196	98.0
Abdominal pain	185	92.5
Constipation	21	10.5
Pruritus	5	2.5
Signs		
Elevated temperature	197	98.5
Jaundice	200	100.0
Hepatomegaly	200	100.0
Abdominal tenderness	172	86.0
Ascites	5	2.5
Splenomegaly	12	6.0

Table 3: Frequency of complications in study population, (n=200).

Complications	N	Percentage (%)
Cholestasis	19	9.5
Acute liver failure	9	4.5
Ascites	5	2.5
Relapse	4	2.0
Cholangitis	3	1.5
Death	2	1.0

Table 4: Laboratory findings of study population, (n=200).

Parameters	Range	N	Percentage (%)
S. bilirubin (mg/dl)-total	Up to 10	173	86.5
	10-20	21	10.5
	>20	6	3.0
S. bilirubin (mg/dl)-direct	Up to 2	170	85.0
	2-5	17	8.5
	5-10	11	5.5
	>10	2	1.0
Serum ALT (IU/L)	Up to 500	51	25.5
	500-1500	135	67.5
	>1500	24	12.0
INR	Up to 1.3	156	78.0
	1.3-1.5	35	17.5
	>1.5	9	4.5
S. albumin (gm/dl)	>3	159	79.5
	3-2	34	17.0
	<2	7	3.5

DISCUSSION

The majority of the patients in our study were between the ages of 5 to 10 years (53.5%), followed by those under 5 years (22.5%) and above 10 years (24%). This finding is consistent with previous studies that have reported a higher incidence of HAV infections in children under the age of 10.[10] In terms of socio-economic status, most of the children belonged to the low-middle class (47.5%), followed by the upper-middle class (28.5%). Studies have shown that socio-economic factors play a significant role in the transmission and prevalence of HAV, with children from lower socio-economic backgrounds being more susceptible to HAV infection.[11] The 39.5% used tap water and 25.5% used underground water for drinking purpose. Only 20.5% used boiled water and 14.5% used unboiled water. Majority 78.5% ate both homemade and outside food. They are infected while they are taking food or beverage from roadside that have been made with contaminated water. Positive family history was present in 13.5% cases.[12] The clinical presentation of HAV infection in this study was characterized by fever (98.5%), vomiting (95.5%), yellowish discoloration of the skin and sclera (100%), nausea (98%), anorexia (98%), and abdominal pain (92.5%). These symptoms are typical of acute hepatitis A and have been reported in numerous studies.[13,14] The hallmark signs of HAV infection, jaundice, and hepatomegaly, were observed in all patients (100%), which corroborates findings from other studies that emphasize the high prevalence of these signs in children with hepatitis A.[15,16] Elevated body temperature was found in 98.5% of cases, which is indicative of the acute inflammatory response to the virus.. In terms of complications, cholestasis was the most common, affecting 9.5% of the study population, followed by acute liver failure (4.5%). Although acute liver failure in HAV infection is uncommon, it is a recognized complication, particularly in children, and can be life-threatening.[17] Ascites and relapse occurred in 2.5% and 2% of patients, respectively.[18] Mortality was observed in 1% of the cases, which highlights the potential severity of acute HAV infection. While mortality in HAV is rare, it is more likely in patients who progress to acute liver failure.[19] The laboratory findings from this study demonstrated elevated serum bilirubin levels, particularly total bilirubin, with 86.5% of patients having levels up to 10 mg/dl. This finding is in agreement with previous studies where total bilirubin is commonly elevated in acute hepatitis A, reflecting liver dysfunction.[20] Direct bilirubin was elevated in 15% of cases, which indicates a degree of cholestasis, which is consistent with the clinical finding of cholestasis observed in 9.5% of the patients. Serum ALT levels were markedly elevated, with 67.5% of patients showing values between 500-1500 IU/L, which is typical for acute viral hepatitis, as ALT is a marker of hepatocellular injury.[21] Elevated ALT levels in HAV infection have been consistently reported, and their degree of elevation often correlates with the severity of the disease.[22] The INR was elevated in 22% of patients, with 4.5% showing an INR >1.5, indicating impaired coagulation, which is a sign of liver dysfunction and indicates the severity of the hepatic injury. Serum albumin levels were decreased in 21% of the patients, with 3.5% showing levels below 2 g/dl, which reflects a reduction in liver synthetic function. Our findings are largely consistent with previous reports from both high- and low-income countries, where hepatitis A predominantly affects children and presents with similar symptoms and biochemical abnormalities.[23,24] However, the prevalence of complications such as acute liver failure and cholestasis observed in our study appears to be somewhat higher compared to other studies, which may reflect the severity of the cases treated at a tertiary care centre.

Limitations

The study was conducted in a single hospital with a small sample size. So, the results may not represent the whole community.

CONCLUSION

This study showed the typical presentation of HAV in children, including anorexia, nausea, vomiting, jaundice,

fever, and abdominal pain, alongside with elevated serum ALT and bilirubin levels. Despite the generally self-limiting nature of the disease, complications such as cholestasis and acute liver failure were observed, underlining the importance of early diagnosis and appropriate management and emphasize the necessity of including hepatitis A vaccination in routine immunization programme.

Recommendation

It is recommended that early diagnosis and close monitoring of children with hepatitis A be prioritized to detect potential complications such as cholestasis and acute liver failure. Universal immunization against hepatitis A and raising social awareness against viral hepatitis may effectively decrease morbidity and mortality from acute hepatitis and acute liver failure from hepatitis A in the pediatric age group of Bangladesh.

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REFERENCES

1. Thomas HC, Lok AS, Locarnini SA, Zuckerman AJ. Viral hepatitis. John Wiley and Sons. 2013.
2. Hernandez-Suarez G, Saha D, Lodroño K, Boonmahittisut P, Taniwijaya S, Saha A, et al. Seroprevalence and incidence of hepatitis A in Southeast Asia: A systematic review. Plos One. 2021;16(12):e0258659.
3. Matheny SC, Kingery JE. Hepatitis a. Am Fam Phys. 2012;86(11):1027-34.
4. Gholizadeh O, Akbarzadeh S, Ghazanfari Hashemi M, Gholami M, Amini P, Yekanipour Z, et al. Hepatitis A: Viral Structure, Classification, Life Cycle, Clinical Symptoms, Diagnosis Error, and Vaccination. Batra L, editor. Canad J Infect Dis Med Microbiol. 2023;2023:1-17.
5. Mahmud S, Ahmed SS, Hussain M, Afroz M, Tasneem F. Recent spectrum of acute viral hepatitis in children: an experience in a tertiary center of Bangladesh. Adv Res Gastroenterol Hepatol. 2017;6(3):555686.
6. Migueres M, Lhomme S, Izopet J. Hepatitis A: epidemiology, high-risk groups, prevention and research on antiviral treatment. Viruses. 2021;13(10):1900.
7. Wasley A, Fiore A, Bell BP. Hepatitis A in the era of vaccination. Epidemiologic Rev. 2006;28(1):101-11.
8. Khan AI, Salimuzzaman M, Islam MT, Afrad MH, Shirin T, Jony MHK, et al. Nationwide hospital-based seroprevalence of hepatitis A and hepatitis E virus in Bangladesh. Ann Global Health. 2020;86(1):29.
9. Siddique AB, Nafisa T, Akram A, Resma TI, Akter T. Co-Infection of Hepatitis A and Hepatitis E Viruses in Outpatients in Dhaka, Bangladesh: A Case Series. East West Med College J. 2025;13(1):65-8.
10. Nelson NP. Prevention of hepatitis A virus infection in the United States: recommendations of the Advisory Committee on Immunization Practices, 2020. MMWR Recommendations and Reports, vol 69, 2020. Available from: https://www.cdc.gov/mmwr/volumes/69/rr/rr6905a1.htm. Accessed on 8 February 2025.
11. Franco E, Meleleo C, Serino L, Sorbara D, Zaratti L. Hepatitis A: Epidemiology and prevention in developing countries. World J Hepatol. 2012;4(3):68.
12. Yeasmin S, Rukunuzzaman M, Islam F, Sultana K, Chowdhury AS, Zaman R, et al. Clinical and Biochemical Profile of Hepatitis A Virus infection in Children. Ann Int Med Dental Res. 2022;8(4):263-9.
13. Lai M, Chopra S. Hepatitis A virus infection in adults: Epidemiology, clinical manifestations, and diagnosis. U: UpToDate, Baron EL ed UpToDate Waltham, MA: UpToDate. 2019 Available at: https://medicowise.com/wp-content/uploads/2023/10/Hepatitis-A-virus-infection-in-adults_-Epidemiology-clinical-manifestations-and-diagnosis-UpToDate.pdf. Accessed on 8 February 2025.
14. Murphy TV. Progress toward eliminating hepatitis A disease in the United States. MMWR supplements, Vol 65, 2016. Available at: https://www.cdc.gov/mmwr/volumes/65/su/su6501a6.htm. Accessed on 8 February 2025.
15. Howard CM, Handzel T, Hill VR, Grytdal SP, Blanton C, Kamili S, et al. Novel risk factors associated with hepatitis E virus infection in a large outbreak in northern Uganda: results from a case-control study and environmental analysis. Am J Trop Med Hyg. 2010;83(5):1170.
16. Abdel-Ghaffar TY, Sira MM, Sira AM, Salem TA, El-Sharawy AA, El Naghi S. Serological markers of autoimmunity in children with hepatitis A: relation to acute and fulminant presentation. Eur J Gastroenterol Hepatol. 2015;27(10):1161-9.
17. Kumar KJ, Kumar HCK, Manjunath VG, Anitha C, Mamatha S. Hepatitis A in Children- Clinical Course, Complications and Laboratory Profile. Indian J Pediatr. 2014;81(1):15-9.
18. Surender Kumar Yachha for the Pediatric Gastroenterology Chapter of Indian Academy of Pediatrics, Bhatia V, Bavdekar A. Management of acute liver failure in infants and children: Consensus statement of the pediatric gastroenterology chapter, Indian Academy of Pediatrics. Indian Pediatr. 2013;50(5):477-82.
19. Manka P, Verheyen J, Gerken G, Canbay A. Liver failure due to acute viral hepatitis (AE). Visceral Med. 2016;32(2):80-5.
20. Radha Krishna Y, Saraswat VA, Das K, Himanshu G, Yachha SK, Aggarwal R, et al. Clinical features and predictors of outcome in acute hepatitis A and

hepatitis E virus hepatitis on cirrhosis. Liver International. 2009;29(3):392-8.
21. Cheong JY, Kim DJ, Hwang SG, Yang JM, Kim YB, Park YN, et al. Serum markers for necroinflammatory activity in patients with chronic viral hepatitis and normal or mildly elevated aminotransferase levels. Liver Int. 2011;31(9):1352-8.
22. Fujiwara K, Kojima H, Yasui S, Okitsu K, Yonemitsu Y, Omata M, et al. Hepatitis A viral load in relation to severity of the infection. J Med Virol. 2011;83(2):201-7.
23. Grover M, Gupta E, Samal J, Prasad M, Prabhakar T, Chhabra R, et al. Rising trend of symptomatic infections due to Hepatitis A virus infection in adolescent and adult age group: An observational study from a tertiary care liver institute in India. Indian J Med Microbiol. 2024;50:100653.
24. Oettinger R, Brunnberg A, Gerner P, Wintermeyer P, Jenke A, Wirth S. Clinical features and biochemical data of Caucasian children at diagnosis of autoimmune hepatitis. J Autoimmunity. 2005;24(1):79-84.

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