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Original Research Article

Clinical Profile and Outcomes of Acute Viral Hepatitis in Children: Correlation with Laboratory Parameters

Rutika Kumari[1], Suman Kumari[2], Hemant Kumar[3]

[1]Senior Resident, Department of Pediatrics, Patna Medical College and Hospital, Patna, Bihar, India.
[2]Senior Resident, Department of Pediatrics, Patna Medical College and Hospital, Patna, Bihar, India.
[3]Professor and HOD, Department of Pediatrics, Patna Medical College and Hospital, Patna, Bihar, India.

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Corresponding Author: Dr. Suman Kumari
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Abstract:

Background: Acute viral hepatitis is an important cause of morbidity and hospital admission among children, with a variable clinical spectrum ranging from uncomplicated jaundice to severe hepatic dysfunction and acute liver failure. Correlation of clinical manifestations with laboratory abnormalities may help identify children at risk of complications.

Aim: To evaluate the clinical profile, laboratory characteristics, complications, and outcomes of acute viral hepatitis in children and assess their relationship with disease severity.

Methodology: A prospective observational study was conducted over one year in the Department of Pediatrics, Patna Medical College and Hospital, Bihar, India. Forty-seven children aged >1 month to <12 years with laboratory-confirmed acute viral hepatitis were evaluated. Clinical findings, hematological and liver function parameters, coagulation profile, viral markers, ultrasonography findings, complications, and hospital outcomes were documented and analyzed.

Result: Hepatitis A was the predominant etiology (27; 57.45%), followed by hepatitis B (9; 19.15%), C (7; 14.89%), and E (4; 8.51%). Most children were aged 5–10 years (68.09%) and were male (72.34%). Icterus (95.74%), fever (76.60%), pallor (76.60%), and hepatomegaly (72.34%) were common. Total bilirubin of 5–10 mg/dL and SGPT of 500–1000 U/L were each observed in 40.43% of children. Most had PT-INR <1.5 (72.34%). Hepatomegaly was the most common USG finding (59.57%). Overall, 95.74% were discharged and 4.26% died; serious complications occurred in 12.77%.

Conclusion: Acute viral hepatitis in children predominantly presents with jaundice and biochemical evidence of hepatocellular injury. Early recognition and monitoring of laboratory abnormalities, particularly bilirubin, transaminases, and coagulation parameters, are important for identifying severe disease and improving outcomes.

Keywords: Acute viral hepatitis; Children; Hepatitis A; Hepatitis B; Liver function tests; SGPT; Bilirubin; Clinical outcome.

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Introduction

Hepatitis, or inflammation of the liver, can occur due to a variety of infectious and non-infectious causes, among which viral infections remain one of the most important causes of acute liver disease and contribute substantially to morbidity and mortality.[1] Viral hepatitis is a contagious disease caused by different hepatotropic viruses, primarily hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), hepatitis D virus (HDV), and hepatitis E virus (HEV), which can produce temporary or, in some cases, persistent damage to hepatocytes.[2] The clinical spectrum of viral hepatitis is broad, ranging from asymptomatic infection and mild constitutional symptoms to clinically apparent jaundice, acute hepatitis, hepatic dysfunction, acute liver failure, and, in selected viral infections, progression to chronic liver disease. The burden and the pattern of viral hepatitis is quite different depending on the age group, geographical location, socioeconomic status, sanitation, vaccination status, and the way of transmission.

Viral hepatitis is still a significant global public health issue. In 2015, the number of deaths due to viral hepatitis was estimated to be around 1.34 million, equivalent to the number of deaths for tuberculosis and more than for HIV infection. In children, acute viral hepatitis is especially significant because its clinical picture can be quite confused and reminiscent of a number of other febrile or gastrointestinal causes. Hepatitis A and hepatitis E spread primarily via the fecal–oral route and are linked to poor

hygienic practices, unsafe drinking water and lack of sanitation. Hence these infections are of special importance in developing countries like India. Available literature indicates that HAV is responsible for approximately 10–30% of cases of acute hepatitis and 5–15% of acute liver failure cases in India. HEV is believed to cause acute hepatitis in about 10-40% of cases, and acute liver failure in 15-45% of cases. So, HAV and HEV are significant causes of acute viral hepatitis and acute liver failure (ALF) among Indians [3].

Despite the fact that HAV and HEV are the leading causes of acute hepatitis, HBV and HCV remain important due to their ability to cause chronic infection and chronic liver disease. In the general Indian population, the prevalence of hepatitis B surface antigen (HBsAg) positivity has been reported to be 1.1% to 12.2% with an average prevalence of about 3-4%. According to regional studies, the estimated number of people suffering from chronic HBV infection is 40 million and from chronic HCV infection is 6–12 million in India. Chronic HBV infection is a major cause of hepatocellular carcinoma (HCC) and cirrhosis, accounting for some 40% of HCC and 20–30% of cirrhosis in India. There is also a significant number of HCC and cirrhosis cases associated with chronic HCV infection. Clinical signs of acute infection may be mild in some children, but it is important to recognize that the clinical course and risk of chronic liver disease varies with the different viral infections.

Childhood hepatitis is an important cause of childhood morbidity and hospitalization in India. Overall, pediatric liver disease occurs at a frequency of 1:8,000 around the world, and one U.S. study estimated nearly 15,000 hospital admissions each year from pediatric liver disease in the United States. The nature of liver disease in children has dramatically altered over the past few decades. Acute infectious hepatitis, on the other hand, remains common in the paediatric clinical practice and in areas where environmental and socio-economic conditions are conducive to the spread of enteric viruses like HAV and HEV [4].

Acute viral hepatitis in children has variable clinical presentations. Children may have fevers, lack of appetite, nausea, vomiting, tummy pain, feeling unwell, enlarged liver, dark urine, light stools, and yellowing of skin. Clinical diagnosis alone is difficult as some children are either asymptomatic or have only nonspecific constitutional symptoms. Laboratory investigations are therefore of major importance in establishing hepato involvement and the severity of the disease. During acute hepatocellular injury, the serum aminotransferases, specifically alanine aminotransferase (ALT) and aspartate aminotransferase (AST), are usually elevated and bilirubin and alkaline phosphatase levels help to determine the severity and type of liver dysfunction. Some biochemical parameters such as serum albumin, prothrombin time/international normalized ratio (PT/INR) may be helpful to evaluate hepatic synthetic function as well as to identify children at risk of developing severe disease or acute liver failure.

It is important to note the relationship between clinical manifestations and laboratory abnormalities in pediatric patients as illness severity does not necessarily directly correlate with biochemical derangement. Children with subsequent complete recovery may have more marked transaminase elevations, while coagulation abnormalities or demonstration of decreased liver synthetic function may suggest more severe illness. In the same way, the length of duration of jaundice, hepatomegaly, vomiting, altered sensorium, bleeding manifestations or other complications might affect clinical management and outcome. A correlation of laboratory parameters with clinical findings can therefore help to better understand the disease severity and to identify children who need to be more closely monitored and timely interventions.

Acute viral hepatitis is clinically important in Bihar, India, due to the ongoing importance of infections via contaminated food and water as well as the general importance of hepatitis B and C infections. The clinical spectrum, laboratory picture, complications and outcome of acute viral hepatitis in children, however, can be different in different regions and hospitals. Data on children with acute viral hepatitis obtained in local hospitals are thus useful in understanding the nature of children who present with acute viral hepatitis and the laboratory data that are associated with their clinical course.

The present study was carried out to assess the clinical profile and outcome of children with acute viral hepatitis and to look for any correlation between laboratory parameters and clinical outcome in a tertiary care centre at Bihar, India. The study's primary objectives are to specifically address the presenting clinical features, biochemical abnormalities, complications and outcomes of affected children with emphasis to see if biochemical parameters can be used to characterize disease severity and predict the clinical course of the disease. This information can help to better assess, stratify risk, monitor, and manage children with acute viral hepatitis in the general pediatric setting.

Methodology

Study Design: This study was designed as a prospective observational study to evaluate the clinical profile, laboratory parameters, and outcomes of children with acute viral hepatitis. The study focused on the relationship between clinical manifestations, biochemical and hematological parameters, viral markers, and clinical outcomes among hospitalized pediatric patients.

Study Area: The study was conducted in the Department of Pediatrics, Patna Medical College and Hospital, Patna, Bihar, India.

Study Duration: The study was conducted over a period of 1 year from July 2025 to June 2026.

Sample Size: A total of 47 children (N = 47) fulfilling the predefined inclusion and exclusion criteria were enrolled during the study period.

Study Population: The study population comprised children aged >1 month and <12 years who were admitted to the pediatric ward with clinical features suggestive of acute viral hepatitis and in whom the diagnosis was subsequently supported by appropriate laboratory investigations.

Data Collection: Children presenting with suspected acute viral hepatitis were evaluated after initial stabilization. A detailed history was obtained regarding presenting complaints, duration of illness, fever, anorexia, nausea, vomiting, abdominal pain, jaundice, dark-colored urine, pale stools, pruritus, and other relevant symptoms. Information regarding possible sources of infection and relevant epidemiological exposure was also recorded.

A detailed general and systemic examination was performed, with particular attention to the presence and severity of jaundice, hepatomegaly, splenomegaly, dehydration, bleeding manifestations, altered sensorium, and clinical features suggestive of hepatic encephalopathy. Relevant demographic and clinical information, investigation findings, diagnosis, treatment, and outcome were systematically documented in a predesigned data-collection proforma.

Laboratory investigations included complete blood count, liver function tests, serum bilirubin, liver enzymes including serum alanine aminotransferase (ALT/SGPT), and coagulation profile including prothrombin time (PT) and international normalized ratio (INR). Urine examination for bile salts and bile pigments was performed where clinically indicated. Ultrasonography (USG) of the abdomen and pelvis was performed to assess hepatobiliary and abdominal findings. Viral serological investigations for hepatitis A, B, C, D, and E were performed as clinically appropriate. Additional investigations, such as renal function tests and chest radiography, were undertaken when clinically indicated.

Acute viral hepatitis was defined in accordance with the clinical and biochemical criteria provided in the supplied methodology, namely an acute illness with discrete onset of symptoms such as nausea, anorexia, fever, malaise, or abdominal pain, accompanied by either serum total bilirubin ≥2 mg/dL or ALT ≥2 times the upper limit of normal, in the absence of an underlying chronic liver disease.

Inclusion Criteria

Children were included if they fulfilled the following criteria:

- Age >1 month and <12 years.
- Clinical features suggestive of acute viral hepatitis.
- Evidence of acute hepatic involvement based on biochemical investigations, including elevated serum bilirubin and/or ALT.
- Laboratory confirmation of viral hepatitis through appropriate viral markers.
- Children admitted to the pediatric ward during the study period.
- Written informed consent obtained from the parent or legally authorized guardian.

Exclusion Criteria

The following children were excluded:

- Children with pre-existing chronic liver disease.
- Children with known metabolic or inherited disorders affecting the liver.
- Children receiving drug therapy associated with hepatic injury or with suspected drug-induced hepatitis.
- Children with a surgical or obstructive cause of jaundice/hepatitis.
- Children whose clinical or laboratory evaluation did not support acute viral hepatitis.

Procedure: Initially, children suspected of having acute viral hepatitis were assessed for their general clinical condition and the presence of complications, particularly features of hepatic encephalopathy. Immediate management was provided when required, with stabilization of airway, breathing, and circulation. Children requiring intensive monitoring or management of complications were treated in the pediatric intensive care unit, while clinically stable children were managed in the pediatric ward.

After stabilization, a detailed history and clinical examination were performed. Relevant demographic characteristics, presenting symptoms, possible source of infection, and clinical findings were documented systematically. Blood and other appropriate samples were collected for hematological, biochemical, coagulation, and virological investigations. Imaging studies were performed according to clinical requirements.

The etiological diagnosis was established using appropriate viral markers. Acute hepatitis A was diagnosed by the presence of serum anti-HAV IgM. Acute hepatitis B was diagnosed by the presence of IgM antibody against hepatitis B core antigen (IgM anti-HBc), with or without HBsAg. Acute hepatitis E was diagnosed by detection of anti-HEV IgM, while hepatitis C was diagnosed by detection of anti-HCV antibody. Testing for hepatitis D and other viral markers was performed when clinically indicated.

All children received appropriate symptomatic and supportive treatment according to their clinical condition. Children were monitored during hospitalization for clinical progression, changes in liver function and coagulation parameters, development of complications, and response to treatment. The primary clinical outcome was recorded as discharge or death.

Statistical Analysis: All collected data were systematically entered into a Microsoft Excel spreadsheet and subsequently analyzed using licensed OpenEpi software along with Microsoft Office 365. Categorical variables were summarized using frequencies and percentages, while continuous variables were summarized using appropriate measures of central tendency and dispersion. The relationship between relevant clinical characteristics, laboratory parameters, viral etiology, and clinical outcomes was assessed using appropriate statistical methods. Multivariate analysis was performed to evaluate associations between selected explanatory variables and outcome variables. Statistical significance was assessed using an appropriate level of significance, with $p < 0.05$ considered statistically significant".

Result

Table 1 presents the socio-demographic profile of the 47 children diagnosed with viral hepatitis. The majority of children were aged 5–10 years, comprising 32 (68.09%) of the total cases, followed by those aged <5 years (10; 21.28%) and >10 years (5; 10.64%). Within the individual hepatitis groups, the 5–10-year age group was predominant in Hepatitis A (17; 62.96%), Hepatitis B (7; 77.78%), Hepatitis C (4; 57.14%), and Hepatitis E (4; 100%). There was a male predominance, with 34 (72.34%) males and 13 (27.66%) females. Male predominance was observed across all groups, being highest in Hepatitis A (21; 77.78%) and Hepatitis E (3; 75%). Regarding place of residence, 42 children (89.36%) were from urban areas, while only 5 (10.64%) were from rural areas. Urban residence predominated in Hepatitis A and B (24; 88.89% and 8; 88.89%, respectively), Hepatitis C (7; 100%), and Hepatitis E (3; 75%). However, there were no statistically significant differences among the hepatitis groups with respect to age (p=0.413), gender (p=0.713), or place of residence (p=0.635).

Table 1: Socio-demographic profile

Parameters	Hep A (n=27)	Hep B (n=9)	Hep C (n=7)	Hep E (n=4)	Total (%)	p-value
Age (years)						
<5	8 (29.63%)	1 (11.11%)	1 (14.29%)	0 (0%)	10 (21.28%)	0.413
5 to 10	17 (62.96%)	7 (77.78%)	4 (57.14%)	4 (100%)	32 (68.09%)	
>10	2 (7.41%)	1 (11.11%)	2 (28.57%)	0 (0%)	5 (10.64%)	
Gender						
Female	6 (22.22%)	3 (33.33%)	3 (42.86%)	1 (25%)	13 (27.66%)	0.713
Male	21 (77.78%)	6 (66.67%)	4 (57.14%)	3 (75%)	34 (72.34%)	
Place of Residence						
Urban	24 (88.89%)	8 (88.89%)	7 (100%)	3 (75%)	42 (89.36%)	0.635
Rural	3 (11.11%)	1 (11.11%)	0 (0%)	1 (25%)	5 (10.64%)	

Table 2 presents the distribution of clinical symptoms according to the type of viral hepatitis among 47 children. Yellow sclera was the most frequently reported symptom, occurring in 45 children (95.74%), including 100% of Hepatitis A, B, and E cases and 5 (71.43%) cases of Hepatitis C. Fever was reported in 36 children (76.60%), being most common in Hepatitis A (24; 88.89%) and least common in Hepatitis C (3; 42.86%). Yellow urine was reported in 22 children (46.81%), with the highest proportion in Hepatitis E (3; 75%) followed by Hepatitis B (6; 66.67%). Vomiting was present in 15 children (31.91%), while abdominal pain was reported in 9 (19.15%). Diarrhoea occurred in 2 children (4.26%), both with Hepatitis A. Convulsions were reported in 2 children (4.26%), both belonging to the Hepatitis B group. Other symptoms were documented in 6 children (12.77%), with the highest proportion observed among Hepatitis C cases (3; 42.86%). Overall, differences in symptom distribution were observed across the viral hepatitis groups, but the association was not statistically significant (χ^2=23.22, df=21, p=0.332).

Table 2: Distribution of viral hepatitis according to clinical symptoms					
Symptoms	**Hepatitis A (n=27)**	**Hepatitis B (n=9)**	**Hepatitis C (n=7)**	**Hepatitis E (n=4)**	**Total (%)**
Fever	24 (88.89%)	6 (66.67%)	3 (42.86%)	3 (75%)	36 (76.60%)
Yellow sclera	27 (100%)	9 (100%)	5 (71.43%)	4 (100%)	45 (95.74%)
Yellow urine	11 (40.74%)	6 (66.67%)	2 (28.57%)	3 (75%)	22 (46.81%)
Vomiting	10 (37.04%)	2 (22.22%)	2 (28.57%)	1 (25%)	15 (31.91%)
Abdominal pain	6 (22.22%)	1 (11.11%)	1 (14.29%)	1 (25%)	9 (19.15%)
Diarrhoea	2 (7.41%)	0 (0%)	0 (0%)	0 (0%)	2 (4.26%)
Convulsion	0 (0%)	2 (22.22%)	0 (0%)	0 (0%)	2 (4.26%)
Others	1 (3.70%)	2 (22.22%)	3 (42.86%)	0 (0%)	6 (12.77%)

Chi Square = 23.22, Df = 21, p = 0.332

Table 3 presents the distribution of clinical signs according to the type of viral hepatitis among 47 children. Icterus was the most common clinical finding, present in 45 children (95.74%), including all cases of Hepatitis A, B, and E, and 5 (71.43%) cases of Hepatitis C. Pallor was observed in 36 children (76.60%), being most frequent in Hepatitis C (6; 85.71%), followed by Hepatitis A (21; 77.78%), Hepatitis E (3; 75%), and Hepatitis B (6; 66.67%). Hepatomegaly was present in 34 children (72.34%), with the highest proportion among Hepatitis B cases (8; 88.89%). Splenomegaly was documented in 3 children (6.38%), while hepatosplenomegaly was present in 3 (6.38%). Ascites was observed in 3 children (6.38%), occurring in Hepatitis A in none, Hepatitis B in 1 (11.11%), Hepatitis C in 1 (14.29%), and Hepatitis E in 1 (25%). Altered sensorium was documented in 2 children (4.26%), both belonging to the Hepatitis B group (2; 22.22%). Although variations in clinical signs were observed across the different types of viral hepatitis, the association was not statistically significant (χ^2=20.02, df=18, p=0.331).

Table 3: Distribution of viral hepatitis according to clinical signs					
Clinical sign	**Hepatitis A (n=27)**	**Hepatitis B (n=9)**	**Hepatitis C (n=7)**	**Hepatitis E (n=4)**	**Total (%)**
Icterus	27 (100%)	9 (100%)	5 (71.43%)	4 (100%)	45 (95.74%)
Pallor	21 (77.78%)	6 (66.67%)	6 (85.71%)	3 (75%)	36 (76.60%)
Hepatomegaly	18 (66.67%)	8 (88.89%)	5 (71.43%)	3 (75%)	34 (72.34%)
Splenomegaly	2 (7.41%)	0 (0%)	1 (14.29%)	0 (0%)	3 (6.38%)
Hepatosplenomegaly	0 (0%)	2 (22.22%)	1 (14.29%)	0 (0%)	3 (6.38%)
Ascites	0 (0%)	1 (11.11%)	1 (14.29%)	1 (25%)	3 (6.38%)
Altered sensorium	0 (0%)	2 (22.22%)	0 (0%)	0 (0%)	2 (4.26%)

Chi Square = 20.02, Df = 18, p = 0.331

Table 4 presents the haematological profile of the 47 children according to the type of viral hepatitis. Regarding haemoglobin, 22 children (46.81%) had levels of 5–10 g/dL and an equal number, 22 (46.81%), had levels >10 g/dL, while 3 (6.38%) had severe anaemia with haemoglobin <5 g/dL. Haemoglobin >10 g/dL was most common in Hepatitis A (16; 59.26%), whereas haemoglobin of 5–10 g/dL predominated in Hepatitis C (5; 71.43%) and Hepatitis E (3; 75%). For WBC count, the majority, 28 (59.57%), had counts within the range of 4000–11000/cumm, while 11 (23.40%) had counts >11000/cumm and 8 (17.02%) had counts <4000/cumm. Leukocytosis was particularly frequent in Hepatitis B, affecting 5 (55.56%) children. Regarding platelet count, most children had counts >1.5 lakh/cumm (38; 80.85%), while 9 (19.15%) had counts <1.5 lakh/cumm. The proportion with thrombocytopenia was highest in Hepatitis C (2; 28.57%). PT-INR was <1.5 in 34 children (72.34%), 1.5–2.5 in 10 (21.28%), and >2.5 in 3 (6.38%). Elevated PT-INR >2.5 was observed in 1 (11.11%) child with Hepatitis B and 2 (50%) children with Hepatitis E. Overall, most children had relatively preserved haemoglobin, platelet counts, and coagulation parameters, although abnormalities were observed in a subset, particularly among Hepatitis B and Hepatitis E cases.

Table 4: Distribution of viral hepatitis according haematological profile					
Parameter	**Hepatitis A (n=27)**	**Hepatitis B (n=9)**	**Hepatitis C (n=7)**	**Hepatitis E (n=4)**	**Total (%)**
Haemoglobin (g/dl)					
<5	1 (3.70%)	2 (22.22%)	0 (0%)	0 (0%)	3 (6.38%)
5 to 10	10 (37.04%)	4 (44.44%)	5 (71.43%)	3 (75%)	22 (46.81%)
>10	16 (59.26%)	3 (33.33%)	2 (28.57%)	1 (25%)	22 (46.81%)
WBC count (per cumm)					
<4000	4 (14.81%)	2 (22.22%)	1 (14.29%)	1 (25%)	8 (17.02%)
4000–11000	19 (70.37%)	2 (22.22%)	5 (71.43%)	2 (50%)	28 (59.57%)
>11000	4 (14.81%)	5 (55.56%)	1 (14.29%)	1 (25%)	11 (23.40%)
Platelet count (per cumm)					
<1.5 lakh	4 (14.81%)	2 (22.22%)	2 (28.57%)	1 (25%)	9 (19.15%)
>1.5 lakh	23 (85.19%)	7 (77.78%)	5 (71.43%)	3 (75%)	38 (80.85%)
PT-INR					
<1.5	21 (77.78%)	5 (55.56%)	6 (85.71%)	2 (50%)	34 (72.34%)
1.5 to 2.5	6 (22.22%)	3 (33.33%)	1 (14.29%)	0 (0%)	10 (21.28%)
>2.5	0 (0%)	1 (11.11%)	0 (0%)	2 (50%)	3 (6.38%)

Table 5 presents the distribution of liver function test parameters among 47 children with viral hepatitis. Regarding total bilirubin, levels of 5–10 mg/dL were most frequent overall, occurring in 22 children (46.81%), followed by levels <5 mg/dL in 17 (36.17%) and >10 mg/dL in 8 (17.02%). In Hepatitis A, 18 (66.67%) had total bilirubin of 5–10 mg/dL, whereas Hepatitis B showed a relatively higher proportion with bilirubin >10 mg/dL (3; 33.33%), and Hepatitis E had 3 (75%) with levels >10 mg/dL. Direct bilirubin was <10 mg/dL in 43 children (91.49%), while 4 (8.51%) had levels ≥10 mg/dL; the latter included 1 (3.70%) case of Hepatitis A and 3 (33.33%) cases of Hepatitis B. Similarly, indirect bilirubin was <5 mg/dL in 43 children (91.49%) and ≥5 mg/dL in 4 (8.51%). Regarding SGPT, 19 children (40.43%) had levels <500 U/L and another 19 (40.43%) had levels of 500–1000 U/L, while 9 (19.15%) had levels >1000 U/L. Hepatitis A showed the highest proportion with SGPT of 500–1000 U/L (15; 55.56%), whereas all Hepatitis C cases had SGPT <500 U/L (7; 100%). SGPT >1000 U/L was observed in 7 (25.93%) children with Hepatitis A, 1 (11.11%) with Hepatitis B, and 1 (25%) with Hepatitis E, with none among Hepatitis C.

Table 5: Distribution of viral hepatitis according to liver function test					
Parameters	**Hepatitis A (n=27)**	**Hepatitis B (n=9)**	**Hepatitis C (n=7)**	**Hepatitis E (n=4)**	**Total (%)**
Total bilirubin					
<5	7 (25.93%)	4 (44.44%)	5 (71.43%)	1 (25%)	17 (36.17%)
5 to 10	18 (66.67%)	2 (22.22%)	2 (28.57%)	0 (0%)	22 (46.81%)
>10	2 (7.41%)	3 (33.33%)	0 (0%)	3 (75%)	8 (17.02%)
Direct bilirubin					
<10	26 (96.30%)	6 (66.67%)	7 (100%)	4 (100%)	43 (91.49%)
≥10	1 (3.70%)	3 (33.33%)	0 (0%)	0 (0%)	4 (8.51%)
Indirect bilirubin					
<5	26 (96.30%)	7 (77.78%)	7 (100%)	3 (75%)	43 (91.49%)
≥5	1 (3.70%)	2 (22.22%)	0 (0%)	1 (25%)	4 (8.51%)
SGPT					
<500	5 (18.52%)	5 (55.56%)	7 (100%)	2 (50%)	19 (40.43%)
500–1000	15 (55.56%)	3 (33.33%)	0 (0%)	1 (25%)	19 (40.43%)
>1000	7 (25.93%)	1 (11.11%)	0 (0%)	1 (25%)	9 (19.15%)

Table 6 presents the ultrasonography (USG) findings among the 47 children with viral hepatitis. Hepatomegaly alone was the most common finding, observed in 28 children (59.57%). Hepatosplenomegaly was detected in 3 children (6.38%), while splenomegaly alone was observed in 1 child (2.13%). Ascites was present in 3 children (6.38%). The USG examination was normal in 12 children (25.53%). Overall, hepatomegaly was the predominant ultrasonographic abnormality, whereas a quarter of the children had no detectable abnormality on USG.

Table 6: Distribution according to USG finding		
USG finding	**Frequency**	**Percentage**
Hepatomegaly only	28	59.57%
Hepatosplenomegaly	3	6.38%
Splenomegaly only	1	2.13%
Ascites	3	6.38%
Normal	12	25.53%
Total	**47**	**100.00%**

Table 7 presents the distribution of outcomes according to the type of viral hepatitis among 47 children. Overall, 45 children (95.74%) were discharged, while 2 (4.26%) expired. All children with Hepatitis A (27; 100%) and Hepatitis E (4; 100%) were discharged. Among children with Hepatitis B, 8 (88.89%) were discharged and 1 (11.11%) expired, whereas among those with Hepatitis C, 6 (85.71%) were discharged and 1 (14.29%) expired. Thus, mortality was observed only among children with Hepatitis B and Hepatitis C, while no deaths were recorded among those with Hepatitis A or Hepatitis E.

Table 7: Distribution of outcome according to viral hepatitis					
Outcome	**Hepatitis A (n=27)**	**Hepatitis B (n=9)**	**Hepatitis C (n=7)**	**Hepatitis E (n=4)**	**Total (%)**
Discharge	27 (100%)	8 (88.89%)	6 (85.71%)	4 (100%)	45 (95.74%)
Expired	0 (0%)	1 (11.11%)	1 (14.29%)	0 (0%)	2 (4.26%)
Total	**27 (100%)**	**9 (100%)**	**7 (100%)**	**4 (100%)**	**47 (100%)**

Table 8 shows the distribution of complications according to the type of viral hepatitis. A total of 6 complication events (12.77%) were documented. Hepatitis B accounted for the highest number of complications, with 3 events, including 2 cases of hepatic encephalopathy and 1 case of acute renal failure. Hepatitis E was associated with 2 cases of acute hepatic failure, while Hepatitis C had 1 case of acute hepatic failure. No complications were reported among children with Hepatitis A. Overall, acute hepatic failure was the most frequent complication, occurring in 3 cases (6.38%), followed by hepatic encephalopathy in 2 cases (4.26%) and acute renal failure in 1 case (2.13%).

Table 8: Distribution of complications according to viral hepatitis					
Complication	**Hepatitis A**	**Hepatitis B**	**Hepatitis C**	**Hepatitis E**	**Total**
Hepatic encephalopathy	0	2	0	0	2 (4.26%)
Acute renal failure	0	1	0	0	1 (2.13%)
Acute hepatic failure	0	0	1	2	3 (6.38%)
Total complication events	**0**	**3**	**1**	**2**	**6 (12.77%)**

Discussion

A total of 47 children with laboratory confirmed acute viral hepatitis were included in the present study, with the highest percentage of children being acute Hepatitis A followed by acute Hepatitis B, acute Hepatitis C and acute Hepatitis E. Hepatitis A is the chief cause of acute viral hepatitis in children was in agreement with other Indian and neighbouring studies that have shown hepatitis A to be a major cause of acute viral hepatitis in children. The predominance of hepatitis A in present study supports the continued relevance of enterically transmitted viral hepatitis in children as observed by Behera and Patnaik in Eastern India [5] and Mahmud in Bangladesh [6] and Poddar et al [7] in India".

Most children in the present study were aged 5–10 years, accounting for 32 (68.09%) cases, while 10 (21.28%) were below 5 years and only 5 (10.64%) were above 10 years. The age group 5-10 years had a higher proportion of cases of hepatitis A (62.96%) and all 4 cases of hepatitis E were in this age group. This age distribution contrasts with the data of Behera and Patnaik and Mahmud who found that acute viral hepatitis was significantly prevalent in children of school age group. [5,6] On the other hand, in the study conducted by Poddar et al and Dalal et al, higher proportion of children below 5 years were observed respectively. [7,8] This difference may be understood as differences in local epidemiology, sanitation, exposure patterns, referral characteristics, and vaccination or immunity profile. The high proportion of school-aged children in the current study may also be associated with exposure to contaminated food or water outside of school. The age distribution was not significantly different between the virus groups (p=0.413) although there were a slight proportion of children aged >10 years with hepatitis C.

There was a male predominance with male 34 (72.34%) and female 13 (27.66%). In the present study, males accounted for 77.78% of cases of hepatitis A, 66.67% of hepatitis B, 57.14% of hepatitis C and 75% of hepatitis E cases, but this difference was not significant (p=0.713). Among the study population 42 (89.36%) children were from urban areas. This finding contrasts with studies carried out in mostly rural populations but is consistent with predominance in urban areas reported by Dalal et al., [8]. The urban predominance may be partly due to referral patterns and easy access to tertiary healthcare, and not necessarily due to a difference in incidence.

Yellow sclera was the most common presenting symptom (95.74%) followed by fever (76.60%) and yellow urine (46.81%) in the children clinically. The majority of our cases presented with scleral icterus and fever as the common presenting features which is similar to what is observed by Deore, Patel et al., Das et al., and Singh et al., who have all reported fever and jaundice related manifestations as common presenting features in acute viral hepatitis. Vomit, abdominal pain and diarrhoea were less common in the current study [10,12-14]. Two children had convulsions, which is much less than the 22.7% reported by Singh et al. [14] In selected cases, these neurological signs could indicate severe liver involvement. Of those examined, 45 (95.74%) had icterus and 34 (72.34%) had hepatomegaly. This is similar to the findings of previous paediatric studies in which icterus and hepatomegaly were key clinical features. This difference may be due to the difference in nutritional status, background anaemia and study population characteristics.

Biochemical and haematological differences were noted based on viral aetiology in the laboratory results. Anaemia was also found in significant number of children, 22 (46.81%) had haemoglobin range from 5-10 g/dl and three (6.38%) were severely anaemic (less than 5 g/dl). Thrombocytopenia was relatively more common among cases with hepatitis C, while leukocytosis was more common with hepatitis B. Coagulation abnormalities were also noted where 10 (21.28%) children had a PT-INR of between 1.5 and 2.5 and three (6.38%) >2.5. The highest abnormality was seen in the selected hepatitis B and E cases. Das et al., too, highlighted the deranged coagulation parameters in paediatric acute hepatitis, especially in severe cases.[15]

Biochemical features included hyper-bilirubinaemia and elevated transaminases. Of the children in the present study, 22 (46.81%) had total bilirubin levels of 5-10 mg/dL and 8 (17.02%) children had levels of >10 mg/dL. Hepatitis E had a relatively higher percentage with bilirubin levels >10 mg/dl (75%). SGPT was between 500 and 1000 IU/L in 19 (40.43%) children, while 9 (19.15%) had levels above 1000 IU/L. This study shows a higher prevalence of the 500–1000 IU/l range compared with Das et al., [13] who found a higher proportion at SGPT level below 500 IU/l and Girish et al., [11] who found a higher proportion at a level of above 1000 IU/l. These variations suggest that the extent of transaminase elevation could vary depending on the viral aetiology, time of presentation and severity of hepatic injury.

The ultrasonographic examination revealed hepatomegaly in 28 (59.57%) children and was normal in 12 (25.53%). Hepatosplenomegaly, splenomegaly and ascites were less common. The incidence of hepatomegaly was generally similar to that reported by Sudhamshu et al., who found hepatomegaly to be an important clinical and imaging feature of paediatric viral hepatitis [9] and Sharma et al., who emphasized the value of ultrasonography in the assessment of children with acute viral hepatitis.[17] However, in one-fourth of the children, the ultrasound examination was normal, suggesting that biochemical evidence of hepatic injury does not always correlate with imaging findings.

Overall short-term results were good as 45 (95.74%) children were discharged and two (4.26%) died. All children with hepatitis A and E survived, whereas one child each with B and C died; importantly, no complications were reported in those with hepatitis A, but there was hepatic encephalopathy in B, acute renal failure in E and acute hepatic failure in C and E. These findings generally corroborate the positive result found by Dalal et al. and Das et al. [8,13] The current pattern, however, is different from that of Dalal et al. where significant complications were reported in cases of hepatitis A such as acute hepatic failure and hepatic encephalopathy. [8] This difference may be attributed to difference in sample size and degree of severity of the referral and case selection. Overall, the present study shows that most children with AVPH had good short-term outcomes but biochemical and clinical parameters like marked hyperbilirubinaemia, high transaminase level and raised PTINR can help identify children who need close monitoring, especially in the non-HAV infections.

Conclusion

This study shows that clinical pictures of acute viral hepatitis in children is predominantly icteric with fever, pallor, hepatomegaly, vomiting and abdominal discomfort. Hepatitis A was the most common viral etiology and there was some variation in the clinical and laboratory features of hepatitis B, C, and E. Laboratory profile showed varying degree of hepatic injury, bilirubin and SGPT were the consistent abnormalities whereas abnormal platelet count, WBC count and coagulation parameters were observed in lesser number of children. Hepatomegaly was frequently seen on ultrasonography, corroborating clinical findings. While majority of the children who

were discharged had a good short-term outcome, a few children developed acute hepatic failure, acute renal failure and hepatic encephalopathy, especially in non-hepatitis A infections. In general, the results underline the need of early diagnosis of clinical signs, proper viral diagnosis, and careful interpretation of liver function and haematological parameters to identify children at risk of severe illness and poor outcomes. Close monitoring and management are always necessary to minimize complications and maximize recovery in paediatric acute viral hepatitis.

References

1. Kumar V, Das S, Jameel S. The biology and pathogenesis of hepatitis viruses. Current science. 2010 Feb 10:312-25.
2. Pambuk CI, Muhammad FM, Ali MR. Viral hepatitis: implication of viral types from A to E. Am J Biomed Sci Res. 2019;4(6):442-4.
3. Acharya SK, Madan K, Dattagupta S, Panda SK. Viral hepatitis in India. The National medical journal of India. 2006 Jul 1;19(4):203-17.
4. Rewatkar SS, Shendre SD, Agarwal P, Loni R, Ray M. Etiology of hepatitis in children. International Journal of Contemporary Pediatrics. 2017 Nov;6(4):2130-5.
5. 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(4):317-20.
6. 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.
7. Poddar U, Thapa BR, Prasad A, Singh K. Changing spectrum of sporadic acute viral hepatitis in Indian children. Journal of tropical pediatrics. 2002 Aug 1;48(4):210-3.
8. Dalal EA, Vishal G, Shah JM. Outcome of Acute Viral Hepatitis in Children Admitted in Tertiary Care Hospital of Ahmedabad, Gujarat. National Journal of Community Medicine. 2017 Mar 31;8(03):131-4.
9. Sudhamshu KC, Dilip Sharma DS, Poudyal N, Basnet BK. Acute viral hepatitis in pediatric age groups.
10. Deore JR. Assessment of clinical profile of acute viral hepatitis among paediatric age group patients at tertiary care centre.
11. 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(2):563-8.
12. Patel A, Kumar B. A study on recent spectrum of acute viral hepatitis in children in a tertiary care centre in Eastern Bihar. Indian J Appl Res. 2020 Feb;10(2):41-43.
13. Das M, Mili MK. Spectrum of Acute Hepatitis in Children at a Tertiary Care Hospital. IOSR J Dental and Med Sci. 2019;18(5):73-5.
14. Singh B, Kshatri JS, Das S, Das L, Tripathy RM. Clinical profile of acute viral hepatitis in a tertiary care hospital of Odisha. Int J Curr Res. 2017;9(05):47467-9
15. Das S, Deka A, Biswas T. Clinical profile of acute viral hepatitis in children-in Southern Assam. J Med Sci Clin Res. 2021;9:111-8.
16. Sarker NR, Saha SK, Ghosh DK, Adhikary A, Mridha A, Alam MR. Seropositivity of hepatitis viral markers in icteric children. Bangladesh Medical Journal. 2014 Dec 30;43(1):26-9.
17. Sharma CM, Gupta S, Aggarwal B, Chaudhary P. Acute viral hepatitis in children: a prospective hospital based study. International Journal of Contemporary Pediatrics. 2020 Jul 22;7(8):1681.