Original Research Article

Journal of College of Medical Sciences-Nepal, Vol-21, No.1, Jan-Mar 2025
ISSN: 2091-0657 (Print); 2091-0673 (Online) Open Access
DOI: 10.3126/jcmsn.v20i2.54738

Clinical and Biochemical Characteristics of Children with Hepatitis A Infection

Harsh Vardhan Sharma[1], **Anmol Khajuria**[1], **Ravinder Kumar Gupta**[1]

[1]Department of Pediatrics, Acharya Shri Chander College of Medical Sciences, Jammu, India.

ABSTRACT

Background

Hepatitis A is the most common cause of infectious hepatitis among children in India. It consists of 3 phases- Prodromal phase, Icteric phase, and Recovery phase. Hepatitis A virus (HAV) infection is widespread accounting for high incidence in our community. The current study intends to assess different clinical and biochemical parameters of HAV infection in our community.

Methods

A single-centered cross-sectional study was conducted among 50 patients attending the outpatient department and in-patient department at ASCOMS and Hospital Jammu, with symptoms suggestive of Acute Viral Hepatitis, was done from April 2024 to August 2024.

Results

HAV infection was more common in males (64%) and mostly among the age group of 12-15 years (28%). The most common symptom was fever (92%) and the biochemical parameter was raised serum bilirubin (94%). Ultrasonography findings were gall bladder edema, pericholecystic edema, minimal free fluid, and minimal right-sided pleural effusion, either of which were seen in only 46% of patients.

Conclusions

Hepatitis A is a major health problem due to bad hygiene and sanitary practices and lack of immunization. Clinically, the most common symptom of Hepatitis A infection was fever (92%). In terms of biochemical parameters, the most consistently raised bilirubin (>1mg/dl) is seen in 94% of the cases.

Keywords: hepatic encephalopathy; hepatitis A; jaundice; viruses; vomiting.

INTRODUCTION

Hepatitis A is the most common etiology among hepatitis infections in children in India. Hepatitis A virus (HAV) is a member of the Hepatovirus genus of the family Picornaviridae and is a nonenveloped single-stranded RNA virus.[1] A typical presentation of hepatitis A infection includes prodromal symptoms such as fever, malaise, anorexia, vomiting, and muscle and joint pains. With the onset of the icteric phase, there may be a decrease in these prodromal symptoms, but weakness may persist. The peak infectivity precedes jaundice and the elevation of liver enzymes. After that, the infectivity declines with the appearance of jaundice.[2-4] In 2016, 7134 persons died from hepatitis A worldwide (accounting for 0.5% of the mortality due to viral hepatitis).[5] The proportion of overall Acute Viral Hepatitis (AVH), acute liver failure, and acute-on-chronic liver failure cases attributed to HAV infection is around 70-85%, 40-60%, and 10-40% in India. In USA, rate of hep A per lakh screened population were 0.1 in age groups both 0-9 years and 10-19 years.[6]

METHODS

A cross-sectional study from April 2024 to August 2024 was conducted in ASCOMS and Hospital Jammu on diagnosed cases of children of acute viral hepatitis A. ethical clearance was taken from the institutional review committee (Ref no. ASCOMS/

Correspondence: Dr. Ravinder Kumar Gupta, Department of Pediatrics, Acharya Shri Chander College of Medical Sciences, Jammu, India. Email: urvigupta00@gmail.com, Phone: +91-7006438884. **Article received**: 2024-11-29. **Article accepted**: 2025-02-15. **Article published**: 2025-03-31.

IEC/2024/Meeting-II/FM/11). The data from 50 patients attending the Out-Patient Department and In-Patient Department with symptoms suggestive of Acute Viral Hepatitis (AVH) was collected, tabulated, and statistically analyzed in terms of clinical and biochemical parameters.

Patients of age group 1-19 years and Patients with recent onset of jaundice, conjugated hyperbilirubinemia or mixed hyperbilirubinemia, positive serum report of immunoglobulin M [IgM] HAV were included in this study while All patients with ages <1 year and >19 years, Patient with hepatitis caused by other hepatotropic and non-hepatotropic viruses, Patient with underlying chronic liver disease, negative serological test for HAV, USG suggestive of cirrhosis of the liver, Patients with hepatitis due to metabolic disease and sepsis-induced multiorgan failure and Patient with drug-induced or autoimmune hepatitis were excluded from this study.

Acute Viral hepatitis is defined as an acute illness consisting of prodromal symptoms (e.g., nausea, anorexia, fever, malaise, or abdominal pain) with an increase in total serum bilirubin (≥2 mg/dl) or elevation of serum alanine aminotransferase (ALT; ≥twice the upper limit of normal) at any point in the course of the disease in the absence of underlying chronic liver disease.[7] Acute liver failure (ALF) is defined as the presence of biochemical evidence of acute liver injury (<8 weeks duration); no evidence of chronic liver disease; and liver-based coagulopathy defined as a prothrombin time (PT) >15 s or international normalized ratio (INR) >1.5 not corrected by vitamin K in the presence of clinical hepatic encephalopathy, or a PT>20 sec or INR >2 regardless of the presence of clinical hepatic encephalopathy.[8] Diagnosis of AVH-A was elicited by the presence of anti-HAV IgM in the serum. Viral titer estimation was performed by fully automated bidirectionally interfaced chemiluminescent immunoassay method using HAV Ab-IgM Reagent kit, values more than 1.2 are considered significant for our study's purpose.

RESULTS

A total of 50 patients diagnosed with AVH caused by Hepatitis A were considered for this study. It was seen that Males 32 (64%) were more commonly affected than females. The age of patients ranged from 1-19 years out of which the most common age group affected was 12-15 years 14 (28%) followed by the 8-11 years group 13(26%) (Table 1).

Table 1. Age and gender distribution of study patients. (n= 50)

Age Group	Male n(%)	Female n(%)
1-3 years	4 (67%)	2 (33%)
4-7 years	8 (67%)	4 (33%)
8-11 years	8 (62%)	5 (38%)
12-15 years	8 (57%)	6 (43%)
16-19 years	4 (80%)	1(20%)

The most common clinical symptoms at presentation were fever 46 (92%), vomiting 40 (80%), and Jaundice 40 (80%). Other symptoms were loss of appetite 37 (74%), pain in the abdomen 26 (52%), and hepatic encephalopathy 2(4%) (Table 2).

Table 2. Clinical Symptoms of patients seen at the time of presentation. (n=50)

Clinical symptoms	Frequency (%)
Fever	46 (92%)
Vomiting	40 (80%)
Jaundice	40 (80%)
Decreased Appetite	37 (74%)
Pain Abdomen	26 (52%)
Hepatic Encephalopathy	2 (4%)

Serum alanine aminotransferases (SGPT) and serum aspartate aminotransferase (SGOT) were significantly raised (>1000 u/l) in 26 (52%) and 28 (56%) of patients respectively. Serum bilirubin (>1mg/dl1) was seen in 47 (94%) of patients. A significant number of patients had a prolonged PT and INR 36 (72%) whereas a single patient (2%) reported hypoalbuminemia (<3mg/dl) (Table 3).

Ultrasonographic findings seen in patients were gall bladder edema 7 (14%), pericholecystic edema 6 (12%), minimal free fluid 6 (12%), minimal right-sided pleural effusion 4 (8%) whereas there were no significant findings in 27 patients (54%) (Table 4).

DISCUSSION

Hepatitis A infection is a leading cause of hepatitis globally. Our study indicates the most common age group affected being 12-15 years (28%) in contrast to

Table 3. Lab findings of enrolled patients.	
Biochemical Parameters	**Patients with Hep A (%)**
Total serum bilirubin	
Normal (<1.0mg/dl)	3(6%)
1-5mg/dl	35(70%)
>5mg/dl	12(24%)
Serum SGPT (u/L)	
Normal	3 (6%)
<500	6 (12%)
500-1000	15 (30%)
>1000	26 (52%)
Serum SGOT (u/L)	
Normal	1 (2%)
<500	13 (26%)
500-1000	7 (14%)
>1000	28 (56%)
Serum Albumin	
> 3mg/dl	49 (98%)
<3mg/dl	1 (2%)
PT and INR values	
Normal	14 (28%)
Prolonged	36 (72%)

Table 4. Ultrasonographic findings seen in patients. (n=50)	
USG Findings	**No. of Patients (%)**
Gall bladder edema	7 (14%)
Pericholecystic edema	6 (12%)
Minimal free fluid	6 (12%)
Minimal right sided pleural effusion	4 (8%)
No significant findings	27 (54%)

a similar survey by Behera et al.[9], and Kamath et al.[10] where the most common age group was 5-10 years, 62.5% and 61.6% respectively. The male-female ratio in our study is 2:1 which is consistent with the same study by Behera et al.[9] where the M: F ratio was 2.2:1. Common presenting clinical symptoms were fever (92%), jaundice (80%), and vomiting (80%) which is consistent with the findings in other similar studies like Parekh Z et al.[11] and Das S et al[12] Only 2 patients (4%) in our study had signs of hepatic encephalopathy and only 1 patient had hypoalbuminemia. Lab biochemical tests provide details regarding the status of liver function. Total serum bilirubin and hepatic enzymes (SGOT and SGPT) were elevated in a maximum number of cases in our study. We have found that cases with elevated liver enzymes (SGOT & SGPT >1000U/L), bilirubin of more than 5mg/dl, and INR of more than 1.5 had suffered a more severe clinical course and took longer duration to recover.

Almost half of the study participants (46%) have reliable USG findings on admission like gall bladder edema, pericholecystic edema, minimal free fluid and minimal pleural effusion. It is comparable to findings seen in the case series published by Koehler J et al[13]. The merit of this study is that it also includes USG findings which is not done in most of the similar studies on AVH.

CONCLUSIONS

Hepatitis A is a major health problem due to bad hygiene and sanitary practices and lack of immunization. Clinically, the most common symptom of Hepatitis A infection was fever (92%) followed by jaundice and vomiting seen in 80% of cases. In terms of biochemical parameters, the most consistently raised bilirubin (>1mg/dl) is seen in 94% of the cases, followed by a significant number of patients having prolonged PT and INR (72%).

Limitations

The limitation of the study is that it was done on a handful of patients and thus cannot be generalized to the whole community. Larger community-based studies on AVH are the need of the hour.

ACKNOWLEDGEMENTS

I would like to pay my gratitude towards our Director Principal Dr. Pavan Malhotra and Medical Superintendent Dr. Rabinder Rattanpal for providing me with all the resources of the hospital to carry out my research work.

Conflict of interest: None

Funding: None

REFERENCES

1. Cuthbert JA. Hepatitis A: old and new. Clin Microbiol Rev. 2001 Jan;14(1):38-58. [DOI].
2. Locarnini S, Chen DS, Shibuya K: No more excuses: viral hepatitis can be eliminated. Lancet. 2016, 387:1703-4. 10.1016/S0140-6736(16)30295-1.[PubMed]
3. Satsangi S, Chawla YK: Viral hepatitis: Indian

scenario. Med J Armed Forces India. 2016, 72:204-10. [PubMed]
4. Franco E, Meleleo C, Serino L, Sorbara D, Zaratti L: Hepatitis A: epidemiology and prevention in developing countries. World J Hepatol. 2012, 4:68-73. [Link]
5. Centers for Disease Control and Prevention. Viral Hepatitis Surveillance Report – United States, 2022. [Link]
6. Wasley A, Miller JT, Finelli L; Centers for Disease Control and Prevention (CDC). Surveillance for acute viral hepatitis-United States, 2005. MMWR Surveill Summ. 2007;56(3):1-24. [Link]
7. Suchy FJ. Fulminant hepatic failure in children. Saudi J Gastroenterol. 1996 Jan;2(1):39-43. [PMID].
8. 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):317-320 [DOI]
9. Kamath SR, Sathiyasekaran M, Raja TE, Sudha L. Profile of viral hepatitis A in Chennai. Indian Pediatr. 2009 Jul;46(7):642-3. [PubMed]
10. Parekh Z, Modi R, Banker D. Clinical study of hepatitis in children with special reference to viral markers. NHL J Med Sci. 2013;2(1):23-7. [DOI]
11. Das S, Deka A, Biswas T. Clinical Profile of Acute Viral Hepatitis in Children – In Southern Assam. JMSCR. 2021;9(3):111-18. [DOI]
12. Koehler J, Stolz L,Minges P. Sonographic findings of acute hepatitisin the emergency department. Clin Case Rep.2022;10:e06046. [DOI] [PubMed]

Citation: Sharma HV, Khajuria A, Gupta RK. Clinical and Biochemical Characteristics of Children with Hepatitis A Infection. JCMS Nepal. 2025; 21(1): 29-32.