

Original Research Article

CLINICAL AND BIOCHEMICAL PROFILE OF PEDIATRIC PATIENTS WITH ACUTE VIRAL HEPATITIS: A RETROSPECTIVE HOSPITAL-BASED STUDY

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ABSTRACT

Background: Objectives: To characterize the demographic, clinical, biochemical and hematological profiles of pediatric patients documented with acute viral hepatitis in a hospital-based cohort and to identify limitations in routine clinical documentation relevant to future pediatric hepatitis surveillance.

Materials and Methods: We conducted a retrospective observational study of pediatric clinical records documented between 1 July 2018 and 31 July 2019. Records in which acute viral hepatitis was documented as the principal, suspected, or follow-up/recovery diagnosis were included. Demographic characteristics, presenting complaints, biochemical and hematological parameters, coagulation data, severe manifestations, and available viral-marker results were extracted. Continuous variables were summarized using mean±SD and median (IQR), and categorical variables using frequencies and percentages. Missing observations were not imputed or interpreted as negative.

Results. The dataset comprised 133 clinical records. Females accounted for 72 (54.1%) and males for 61 (45.9%) records. Mean age was 7.98±4.35 years and median age was 8.0 years (range, 0.12–16 years); 47 (35.3%) records were from children aged 10–<15 years and 42 (31.6%) from those aged 5–<10 years. Jaundice/yellowish discoloration was documented in 87 (65.4%), fever in 49 (36.8%), vomiting in 30 (22.6%), and lethargy/weakness in 19 (14.3%). Among 68 records with biochemical measurements, median ALT was 1423 U/L (IQR 706–2038), AST 1105 U/L (IQR 333–1174), total bilirubin 7.1 mg/dL (IQR 5.8–7.1), and alkaline phosphatase 333 U/L (IQR 176–572). INR was available in 30 records and had a median of 1.19 (IQR 1.19–1.70). Six records documented hepatic encephalopathy in association with hepatitis A.

Conclusions. This hospital-based pediatric cohort was characterized by frequent clinically apparent jaundice and substantial aminotransferase elevation, with a smaller subgroup showing documented hepatic encephalopathy. The concentration of records among school-aged children and adolescents is clinically relevant in the context of changing HAV susceptibility in India, but the present data cannot establish an epidemiological shift or estimate HAV/HEV prevalence because etiological testing was incomplete. Prospective studies using standardized case definitions, systematic viral testing, serial assessment of synthetic function, and longitudinal outcomes are warranted.

Keywords: Acute viral hepatitis; children; hepatitis A; hepatitis E; jaundice; alanine aminotransferase; bilirubin; acute liver injury; India.

INTRODUCTION

Acute viral hepatitis in children represents a clinical syndrome rather than a single etiological entity. Hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), and hepatitis E virus (HEV) are the principal hepatotropic viruses of public-health importance, while a range of non-hepatotropic infections and non-infectious disorders may produce a similar biochemical and clinical phenotype. The presentation ranges from asymptomatic or nonspecific illness to fever, anorexia, nausea, vomiting, abdominal discomfort, dark urine, jaundice and hepatomegaly; a small proportion of children develop severe coagulopathy, encephalopathy and acute liver failure. Because clinical manifestations overlap substantially between etiologies, etiological classification requires appropriate laboratory testing rather than clinical impression alone.^[1-6]

HAV and HEV are particularly relevant to South Asian pediatric populations because transmission is closely linked to fecal contamination, water and sanitation, although their epidemiology and routes of transmission are not identical. In endemic settings, HAV is predominantly transmitted by the fecal-oral route, whereas HEV genotype 1 transmission is strongly associated with contaminated water and outbreaks. HEV is also increasingly recognized as a globally distributed infection with diverse clinical and epidemiological patterns.^[1,2,6-8,30]

India presents a particularly informative setting for studying pediatric acute hepatitis because HAV epidemiology is heterogeneous and has changed over time. Earlier Indian serological studies documented high childhood exposure in several populations, whereas subsequent studies reported lower seroprevalence among younger children and a larger susceptible population at older ages. Recent multicentric and national serological investigations continue to demonstrate substantial geographic and age-related heterogeneity rather than a uniform national pattern.^[9-13] This heterogeneity has implications for the age at which symptomatic HAV infection may be encountered and for interpretation of hospital-based pediatric cohorts.

Published Indian pediatric studies also demonstrate that HAV and HEV account for a substantial proportion of laboratory-confirmed acute hepatitis, but the relative contribution of individual viruses varies by location, study period, case definition and testing strategy. Hospital studies have reported HAV as a major cause of pediatric acute hepatitis, while HEV, dengue and other infections may contribute appreciably to the syndrome.^[14-18] Importantly, older North Indian studies of acute liver failure demonstrated that multiple viral etiologies, including HAV and HEV, may coexist or produce severe disease.^[19,20]

Biochemical assessment is central to clinical characterization. Acute viral hepatitis commonly produces pronounced aminotransferase elevation and variable hyperbilirubinemia; however, the magnitude of ALT or AST elevation should not be equated with hepatic synthetic failure. Prothrombin time/INR and neurological status are particularly important when severe acute liver injury or pediatric acute liver failure is suspected. Contemporary Indian consensus recommendations emphasize structured etiological evaluation, serial assessment of coagulation and neurological status, and early recognition of children who may require specialist or transplant-center care.^[21-24]

Against this background, we analyzed a one-year hospital-based pediatric clinical-record dataset to describe the demographic distribution, documented clinical presentation, biochemical and hematological abnormalities, available viral-marker findings, and severe manifestations of acute viral hepatitis. A secondary objective was to identify limitations in routine documentation that are relevant to the design of future prospective pediatric hepatitis surveillance.

MATERIALS AND METHODS

2.1 Study design and data source

This retrospective observational study was based on routinely recorded pediatric clinical data covering 1 July 2018 through 31 July 2019. The study was conducted at Department of Pediatrics, District Hospital - Autonomous State Medical College Hardoi, Uttar Pradesh. The source dataset contained 133 records and included demographic information, recorded diagnosis, presenting complaints, investigation results, prescribed medicines and visit dates. Reporting was structured in accordance with the principles of the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement.^[28,29]

2.2 Study population and analytical unit

All available records in which acute viral hepatitis was recorded as the principal diagnosis, a suspected diagnosis, or a follow-up/recovery diagnosis were included in the descriptive analysis. Because the source dataset contained both acute presentations and follow-up/recovery records, the unit of analysis was a clinical record rather than a strictly incident episode. Accordingly, the results are reported as characteristics of the recorded cohort and should not be interpreted as population incidence or prevalence.

2.3 Variables and data extraction

Age and sex were extracted from the age/sex field. Age values recorded in months were converted to fractional years. Presenting complaints were extracted from the clinical complaint field. Laboratory variables included ALT, AST, alkaline phosphatase (ALP), total bilirubin, hemoglobin, total leukocyte count, platelet count and INR when available. Viral-marker documentation included HBsAg and HCV testing. Diagnostic labels such as

hepatitis A, non-B/non-C acute viral hepatitis, recovery phase, and hepatic encephalopathy were retained as recorded and were not used to infer an etiological diagnosis in the absence of confirmatory serology.

2.4 Statistical analysis

Continuous variables were summarized as mean±standard deviation (SD) and median with interquartile range (IQR), together with observed ranges where available. Categorical variables were summarized as frequencies and percentages. Analyses used the available denominator for each variable; missing laboratory or viral-marker observations were not imputed and were not treated as negative results. Given the retrospective design, heterogeneous documentation and incomplete

etiological testing, the primary analysis was descriptive and no causal or predictive inference was attempted.

2.5 Definition and interpretation of severe disease

Documented hepatic encephalopathy was considered a clinically important marker of severe hepatic dysfunction. However, a diagnosis of pediatric acute liver failure (PALF) was not assigned solely on the basis of encephalopathy because standardized contemporaneous INR measurements and timing relative to neurological deterioration were not available for every affected record. This conservative approach is consistent with modern recommendations that emphasize both coagulopathy and clinical context in PALF assessment.^[21-24]

RESULTS

3.1 Demographic characteristics

The dataset contained 133 pediatric clinical records. Females accounted for 72 (54.1%) records and males for 61 (45.9%). Mean age was 7.98±4.35 years, with a median of 8.0 years (range, 0.12–16 years). The largest age category was 10–<15 years (47; 35.3%), followed by 5–<10 years (42; 31.6%). Together, these two school-aged groups accounted for 89 (66.9%) of the records.

Table 1: Age and sex distribution of pediatric acute viral hepatitis records. n= number, percentage is indicated inside bracket

Age group	Female, n (%)	Male, n (%)	Total, n (%)
<5 years	21 (15.8)	13 (9.8)	34 (25.6)
5–<10 years	28 (21.1)	14 (10.5)	42 (31.6)
10–<15 years	19 (14.3)	28 (21.1)	47 (35.3)
≥15 years	4 (3.0)	5 (3.8)	9 (6.8)
Total	72 (54.1)	61 (45.9)	133 (100)

3.2 Clinical presentation

Jaundice or yellowish discoloration of the eyes was the most frequently documented presenting feature, occurring in 87 (65.4%) records. Fever was documented in 49 (36.8%), vomiting in 30 (22.6%), and lethargy/weakness in 19 (14.3%).

Pruritus/itching was documented in 2 (1.5%). Because the dataset was retrospective and based on routine clinical documentation, these figures represent documented symptoms rather than complete symptom prevalence.

Table 2: Documented presenting complaints. n= number, %= percentage

Clinical feature	n	%
Jaundice/yellowish discoloration	87	65.4
Fever	49	36.8
Vomiting	30	22.6
Lethargy/weakness	19	14.3
Pruritus/itching	2	1.5

3.3 Diagnostic categories and severe manifestations

Most records were coded as acute viral hepatitis. Six records (4.5%) specifically documented follow-up of hepatitis A with hepatic encephalopathy. Two records were coded as acute viral hepatitis non-B/non-C, three were explicitly identified as being in the recovery phase, and four documented follow-up of acute viral hepatitis with microcephaly and severe wasting. These labels were retained as recorded and were not used to infer viral etiology where confirmatory serology was unavailable.

The six records with hepatic encephalopathy represent a clinically important subgroup. Because

INR was available in only 30 records and the timing of INR relative to encephalopathy was not standardized, the dataset does not permit definitive retrospective classification of all six records as PALF. The finding nevertheless highlights the need for early assessment of coagulation and neurological status in children with severe acute hepatitis.

3.4 Biochemical and hematological profile

Biochemical measurements of ALT, AST, ALP and total bilirubin were available in 68 records. ALT and AST showed substantial elevations, with median values of 1423 U/L and 1105 U/L, respectively. Median total bilirubin was 7.10 mg/dL and median ALP was 333 U/L. INR was available in 30 records

and had a median of 1.19. Hematological measurements were available in 57 records.

Table 3: Available biochemical and hematological parameters. SD, standard deviation; IQR, interquartile range; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; INR, international normalized ratio; WBC, white blood cell count

Parameter	Available n	Mean $\pm$ SD	Median (IQR)	Range
ALT (U/L)	68	1299 $\pm$ 952	1423 (706–2038)	25–4430
AST (U/L)	68	800 $\pm$ 605	1105 (333–1174)	27–2944
Total bilirubin (mg/dL)	68	7.77 $\pm$ 4.74	7.10 (5.80–7.10)	0.30–16.10
ALP (U/L)	68	375 $\pm$ 167	333 (176–572)	112–572
INR	30	1.33 $\pm$ 0.27	1.19 (1.19–1.70)	0.99–1.70
Hemoglobin (g/dL)	57	11.62 $\pm$ 1.84	11.70 (10.70–12.60)	6.80–15.30
WBC (cells/mm ³)	57	9325 $\pm$ 3109	9500 (8000–10000)	5000–23000
Platelets (lakh/mm ³)	57	4 $\pm$ 1	4 (2–6)	2–6

3.5 Viral-marker findings

HBsAg testing was documented in 18 records and was non-reactive in all available tested records. HCV testing was documented in 17 records and was non-reactive in all available tested records. In contrast, the source dataset did not contain sufficiently documented anti-HAV IgM or anti-HEV IgM results to estimate the etiological contribution of HAV or HEV. Accordingly, the non-reactive HBV/HCV results apply only to the tested subset and cannot be generalized to the complete cohort.

DISCUSSION

The present study describes a one-year hospital-based cohort of 133 pediatric records associated with acute viral hepatitis or its follow-up. Three findings are particularly relevant: first, two-thirds of records were from school-aged children and adolescents; second, clinically apparent jaundice was common and was accompanied by marked aminotransferase elevation among records with available biochemical data; and third, a smaller subgroup had documented hepatic encephalopathy. These findings provide a clinically coherent picture of acute hepatitis encountered in hospital practice while also demonstrating the limitations of routine retrospective documentation.

The age distribution is noteworthy. Children aged 10–<15 years represented the largest single age group (35.3%), while 5–<10-year-olds accounted for a further 31.6%. This pattern differs from some contemporary HAV-specific hospital cohorts in which younger children predominate, but it is broadly compatible with the heterogeneity of pediatric hepatitis epidemiology across Indian regions and study designs. Studies of laboratory-confirmed HAV and acute viral hepatitis in India have reported substantial representation of school-aged children, although the exact distribution varies according to whether the cohort is clinically suspected, serologically confirmed, hospitalized, or population-based.^[14–18]

An important contextual issue is the changing age distribution of HAV susceptibility in India. Earlier serological studies from Delhi, Hyderabad and other regions documented high childhood exposure, whereas later investigations demonstrated lower

anti-HAV seroprevalence among younger children and substantial susceptibility among older children and adolescents.^[9–13] Recent national and multicentric surveys have confirmed marked geographical variation and an overall intermediate or high-intermediate epidemiological pattern, depending on the population sampled.^[10–12] The relatively large school-aged component of the present cohort may therefore be epidemiologically interesting; however, the present dataset contains neither anti-HAV IgG measurements nor population denominators and cannot itself demonstrate a secular shift in HAV endemicity.

Jaundice was the dominant documented presenting feature, occurring in 65.4% of records. This is consistent with the clinical phenotype expected in hospital-based cohorts, because children with anicteric or very mild infection are less likely to reach tertiary or secondary care. Indian studies of serologically confirmed hepatitis A have reported similarly high frequencies of icterus, although fever, abdominal symptoms and vomiting have varied considerably across cohorts.^[14,15,17] The lower symptom frequencies in the present dataset should therefore be interpreted as documentation frequencies rather than evidence that these symptoms were biologically uncommon.

The biochemical profile provides objective support for substantial hepatocellular injury. Median ALT and AST were 1423 U/L and 1105 U/L, respectively, and median total bilirubin was 7.1 mg/dL. Comparable marked aminotransferase elevations have been described in pediatric HAV and acute viral hepatitis cohorts.^[15,17,18] The relatively high bilirubin concentration is also concordant with the high frequency of clinically visible jaundice. Importantly, aminotransferase magnitude should not be interpreted as a direct surrogate of hepatic synthetic failure. ALT and AST primarily reflect hepatocellular injury, whereas INR is more informative regarding impaired hepatic synthetic function in the setting of severe acute liver injury.^[21–24]

The six records documenting hepatic encephalopathy in association with hepatitis A warrant particular attention. Encephalopathy is a clinically important marker of severe hepatic dysfunction, but the diagnosis of PALF requires

appropriate assessment of coagulopathy and exclusion of chronic liver disease, with the precise framework depending on the guideline applied.^[21-24] Because only 30 records had INR measurements and the temporal relationship between INR and encephalopathy was not consistently available, it would be inappropriate to label all six records definitively as PALF. Nevertheless, the finding reinforces the importance of serial INR assessment, neurological monitoring and timely escalation of care in children with severe acute hepatitis.

The viral-marker findings highlight the central methodological limitation of this dataset. HBsAg and HCV testing was non-reactive in the tested records, but only 18 and 17 records, respectively, had documented testing. More importantly, systematic anti-HAV IgM and anti-HEV IgM results were unavailable. Clinical phenotype alone cannot reliably discriminate HAV from HEV, HBV, HCV or non-viral mimics. Contemporary pediatric studies using systematic serology have demonstrated that HAV may predominate, but HEV and other infectious etiologies remain important.^[16-20] Therefore, the present study should be interpreted as a clinical characterization of records labelled as acute viral hepatitis rather than as a study of the etiological prevalence of individual hepatitis viruses.

The incomplete etiological classification is particularly relevant in India because the spectrum of acute hepatitis is broader than HAV alone. Earlier Indian pediatric studies documented HAV, HEV, HBV and mixed infections, while more recent hospital studies have also identified dengue and other infections among children presenting with acute hepatitis.^[16-20] This argues for a standardized diagnostic panel in prospective studies rather than relying on a syndromic diagnosis. At minimum, systematic testing should include anti-HAV IgM, anti-HEV IgM, HBsAg with age- and context-appropriate additional HBV markers, and anti-HCV with confirmatory testing where indicated. Depending on local epidemiology, testing for dengue, malaria, enteric fever and relevant non-infectious causes should also be considered.

The findings have implications for the interpretation of hospital-based pediatric hepatitis surveillance. Routine clinical records can capture large numbers of patients and provide valuable information on age distribution, symptom burden and biochemical abnormalities without the resource requirements of prospective recruitment. However, missingness is unlikely to be random: viral testing and INR are more likely to be obtained in selected patients, while mild or anicteric cases may be underrepresented in hospital records. Inclusion of follow-up/recovery records further means that the record count cannot be interpreted as the number of unique incident cases. These issues can produce selection and information bias and limit causal or prevalence-based inference.

The study also points to a practical opportunity for strengthening future surveillance. A prospective pediatric hepatitis registry should use a predefined case definition, distinguish new episodes from follow-up visits, record symptom onset and duration, vaccination status, water and sanitation exposures, nutritional status, relevant medications, serial bilirubin and INR measurements, etiological serology, complications, duration of hospitalization and final outcome. Such a design would allow future investigators to move beyond descriptive clinical profiling toward identification of predictors of severe disease and more reliable assessment of the changing etiological spectrum.

CONCLUSION

In this one-year retrospective hospital-based pediatric cohort, acute viral hepatitis was predominantly characterized by clinically apparent jaundice and marked biochemical evidence of hepatocellular injury. School-aged children and adolescents constituted a substantial proportion of the recorded cohort. Six records documented hepatic encephalopathy in association with hepatitis A, underscoring the importance of early recognition of severe hepatic dysfunction and assessment of synthetic liver function. Although available HBsAg and HCV results were non-reactive, incomplete HAV and HEV serology prevented reliable etiological classification. The findings support prospective, standardized pediatric hepatitis surveillance incorporating systematic viral testing, serial severity assessment, exposure characterization and longitudinal outcomes.

7. Declarations

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Competing interests

The authors declare there are no competing interests.

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