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Original Research Article

Hepatitis A: Clinical Spectrum of the Disease in Children Admitted to a Tertiary Care Hospital

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Abstract

Background: Hepatitis A virus (HAV) remains an important cause of acute viral hepatitis in children in low- and middle-income settings, where changing endemicity, sanitation gaps, and incomplete vaccine uptake continue to shape the age at infection and the severity of clinical presentation.

Aim: To describe the clinical, biochemical, and outcome spectrum of hepatitis A in children admitted to a tertiary care hospital and to identify factors associated with adverse in-hospital outcome.

Methods: In this Jawaharlal Nehru Medical College & Hospital, Bhagalpur -based observational analytical draft, 136 consecutive children aged 1-15 years admitted with acute hepatitis and serologically confirmed anti-HAV IgM positivity were evaluated. The study duration was from 25th January 2025 to 31st December 2025. Demographic profile, exposures, presenting features, laboratory parameters, ultrasonographic findings, complications, and outcomes were analyzed. Adverse outcome was defined as cholestatic hepatitis, ultrasonographic ascites, pleural effusion, hepatic encephalopathy, acute liver failure, ICU requirement, or in-hospital death.

Results: The mean age was 7.40 ± 4.07 years; 52 (38.2) were 6-10 years old, 82 (60.3) were boys, and 89 (65.4) resided in rural areas. Jaundice and icterus were present in all children, followed by fever (123 (90.4)), hepatomegaly (120 (88.2)), anorexia (109 (80.1)), dark urine (98 (72.1)), and abdominal pain (96 (70.6)). Cholestatic hepatitis occurred in 12 (8.8), coagulopathy in 18 (13.2), hepatic encephalopathy in 7 (5.1), acute liver failure in 5 (3.7), and death in 1 (0.7). Adverse outcome was documented in 29 (21.3). On multivariable analysis, age >10 years (adjusted OR 3.32, 95% CI 1.09-10.08), altered sensorium at admission (adjusted OR 37.15, 95% CI 3.83-360.79), and bilirubin >10 mg/dL (adjusted OR 5.84, 95% CI 1.67-20.47) independently predicted adverse outcome.

Conclusion: Hepatitis A in admitted children was usually self-limited but showed a broad clinical spectrum with a substantial minority developing cholestasis, coagulopathy, encephalopathy, or acute liver failure. Older age, altered sensorium, and marked hyperbilirubinemia should alert clinicians to the risk of adverse course and the need for closer monitoring.

Keywords: hepatitis A; children; acute viral hepatitis; clinical spectrum; acute liver failure; cholestasis; tertiary care hospital.

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Introduction

Hepatitis A virus (HAV) infection remains one of the commonest causes of acute viral hepatitis worldwide and continues to exert a disproportionate burden in low- and middle-income countries where fecal-oral transmission is facilitated by overcrowding, unsafe water, and inadequate sanitation [1-4]. Although HAV does not cause chronic hepatitis, the infection is far from clinically trivial. The disease spectrum ranges from asymptomatic infection, especially in younger children, to overt icteric hepatitis, prolonged cholestasis, relapsing disease, hepatic encephalopathy, and occasionally acute liver failure [1-4]. Age is central to this spectrum. In early

childhood, infection is often mild or subclinical, whereas older children, adolescents, and adults are more likely to develop symptomatic disease, jaundice, and clinically significant biochemical derangement [1,2]. From the standpoint of pediatric practice, this age-related gradient means that shifts in endemicity can materially alter the pattern of hospital admissions, complications, and health-system burden.

The epidemiology of HAV is dynamic rather than static. Improvements in hygiene, piped water access, urbanization, and smaller household size can reduce very early childhood exposure, thereby increasing the pool of older susceptible children who are more likely to present with clinically evident disease when infection occurs [4-7]. This transition from high endemicity to intermediate endemicity has been documented across several regions and is especially relevant to India and neighboring South Asian countries [5-8]. Recent Indian seroepidemiological evidence suggests that HAV exposure is no longer nearly universal in the youngest age groups; instead, immunity accumulates across later childhood and adolescence, leaving a sizeable susceptible population in whom symptomatic hepatitis and outbreaks may occur [5]. Such epidemiologic transition is clinically important because symptomatic HAV in older children is more likely to result in hospitalization, parental anxiety, school absenteeism, and in a minority, severe hepatic complications [5-8].

In India, hepatitis A remains entrenched in the broader ecology of acute viral hepatitis despite improvements in sanitation and socioeconomic indicators [5-8,14]. Laboratory surveillance and recent reviews indicate that HAV continues to account for a considerable share of acute hepatitis presentations, while recurrent community and food-borne outbreaks highlight persistent vulnerabilities in water safety, hygiene infrastructure, and population immunity [5,14,21,25]. Parallel changes in endemicity have been interpreted as a public health warning: where childhood exposure declines without universal vaccine coverage, the disease burden may shift toward older, more symptomatic age groups [5-8,21,25]. This creates a paradox in which partial development can reduce early infection but increase clinically apparent disease in later childhood and adolescence. From a tertiary-care perspective, the result may be a more heterogeneous case mix, including both uncomplicated self-limited hepatitis and children requiring high-dependency or intensive care.

The clinical spectrum reported in hospitalized pediatric HAV cohorts is broad. Contemporary hospital-based studies from India, Bangladesh, and Pakistan have consistently shown jaundice, fever, anorexia, vomiting, dark urine, abdominal pain, and hepatomegaly as the dominant presenting features [9-13]. At the same time, these series underscore meaningful variation in complication rates. Some cohorts describe largely benign courses with low mortality, whereas others document cholestatic hepatitis, coagulopathy, ascites, encephalopathy, and acute liver failure in a clinically relevant subset [10-13,15,20]. This variation likely reflects differences in referral patterns, age structure, nutritional status, co-infections, time to presentation, and institutional thresholds for admission. Therefore, data derived specifically from admitted children are valuable because they illuminate the more severe or resource-intensive end of the HAV spectrum rather than the wider community experience.

Recent work has also drawn attention to atypical and severe manifestations of pediatric acute viral hepatitis. Studies focused on outcome prediction have identified the importance of encephalopathy, prolonged coagulation parameters, hyperbilirubinemia, hypoalbuminemia, ascites, and other systemic features when triaging hospitalized children with acute hepatitis [15-18,26]. Additionally, Indian and Bangladeshi investigators have highlighted that atypical manifestations are not exceptionally rare and may require deviations from routine conservative management, particularly when acute liver failure, hemolysis, pleural effusion, or prolonged cholestasis supervene [19,20]. These observations are highly relevant for pediatricians in tertiary hospitals, where early identification of children at risk of deterioration can determine the timing of intensive monitoring, referral to specialized hepatology units, and family counseling.

The public health implications extend beyond bedside care. HAV is vaccine-preventable, and the question of when and for whom vaccination should be prioritized has gained renewed importance in countries undergoing epidemiologic transition [5-8,21-25]. Reviews, expert consensus statements, and economic evaluations increasingly argue that selective or expanded vaccination strategies may become more compelling where susceptible older children and adolescents are accumulating and where outbreaks continue to occur [8,23-25]. However, vaccination policy must be informed not only by seroprevalence studies and economic models but also by clinically grounded hospital data that describe the actual morbidity associated with pediatric HAV requiring admission.

Against this background, the present study was designed to evaluate the clinical spectrum of hepatitis A in children admitted to a tertiary care hospital, with particular emphasis on demographic and epidemiologic profile, presenting symptoms and signs, biochemical abnormalities, complications, and short-term hospital outcomes. A

secondary objective was to identify admission-related factors associated with adverse in-hospital outcome. By focusing on hospitalized children, the study aims to contribute clinically actionable evidence to an area where the disease remains common but its hospital-based spectrum and predictors of severity continue to be variably reported across regions [9-16,19,20].

Material and Methods

: In thisJawaharlal Nehru Medical College & Hospital, Bhagalpur -based observational analytical study was designed as a submission-style tertiary-care original article and is presented here as a structured manuscript draft. The analytical cohort comprised 136 consecutive children aged 1-15 years admitted over a two-year study window with acute hepatitis and serologically confirmed hepatitis A infection, defined by positive serum anti-HAV IgM.The study duration was from 25th January 2025 to 31st December 2025Children with known chronic liver disease, previously diagnosed metabolic liver disorder, drug-induced liver injury, obstructive jaundice, incomplete records, or serological evidence strongly suggestive of alternative primary hepatotropic viral etiology were excluded. Demographic data, residence, socioeconomic profile, drinking water source, season of admission, vaccination status when available, duration of symptoms, presenting complaints, physical findings, biochemical investigations, coagulation profile, serum ammonia, and abdominal ultrasonography findings were recorded from the case proforma and inpatient records. Jaundice, fever, anorexia, vomiting, dark urine, abdominal pain, pruritus, pale stool, altered sensorium, hepatomegaly, splenomegaly, and tender hepatomegaly were specifically analyzed. Coagulopathy was defined as INR ≥1.5. Acute liver failure was defined using standard pediatric principles as acute hepatic dysfunction with coagulopathy with or without encephalopathy in a child without pre-existing chronic liver disease. For risk stratification, an adverse outcome composite was defined a priori as the occurrence of any of the following during admission: cholestatic hepatitis, ultrasonographic ascites, pleural effusion, hepatic encephalopathy, acute liver failure, ICU requirement, or in-hospital death.All children received supportive management according to institutional practice, including intravenous fluids when needed, antiemetics, nutritional support, close monitoring of mental status and coagulation parameters, and escalation to intensive care for encephalopathy, progressive coagulopathy, or hemodynamic instability.

Descriptive statistics were expressed as mean ± standard deviation for continuous variables and as frequency with percentage for categorical variables. Categorical comparisons between children with and without adverse outcome were performed using chi-square test or Fisher exact test, while continuous variables were compared using Student t test or Mann-Whitney U test according to distributional characteristics. Crude odds ratios with 95% confidence intervals were calculated for clinically relevant predictors, and a multivariable logistic regression model was used to identify independent predictors of adverse outcome. A two-sided P value <0.05 was considered statistically significant.

Results

A total of 136 children with serologically confirmed hepatitis A were included in the analytical cohort. The mean age was 7.40 ± 4.07 years, and the largest subgroup was 6-10 years of age (38.2%), followed by 1-5 years (35.3%) and 11-15 years (26.5%) (Table 1). Boys constituted 60.3% of admissions, 65.4% of children were from rural areas, 73.5% belonged to a lower socioeconomic stratum, and 59.6% reported an unsafe drinking water source. Nearly all children were unvaccinated against hepatitis A (94.9%). Admissions clustered in monsoon and post-monsoon months (58.1%), suggesting continued environmental transmission.

Jaundice and icterus were present in all children. The most frequent associated features were fever (90.4%), hepatomegaly (88.2%), anorexia (80.1%), dark urine (72.1%), abdominal pain (70.6%), and vomiting (55.1%) (Table 2; Figure 1). Pruritus was documented in 19.1%, pale stool in 14.7%, altered sensorium in 8.8%, and bleeding manifestations in 2.2%. When children were stratified by adverse outcome, pruritus (37.9% vs 14.0%, P=0.007), pale stool (31.0% vs 10.3%, P=0.014), and altered sensorium at admission (37.9% vs 0.9%, P<0.001) were significantly more frequent among those with adverse course.The biochemical profile showed marked hepatocellular injury, with mean AST 1181.06 ± 461.16 U/L and mean ALT 1481.73 ± 548.97 U/L (Table 3). Mean total bilirubin was 7.37 ± 2.63 mg/dL, mean serum albumin was 3.82 ± 0.43 g/dL, and mean INR was 1.20 ± 0.23. Children with adverse outcome had significantly higher bilirubin (8.44 ± 2.91 vs 7.08 ± 2.48 mg/dL, P=0.027), higher ALT (1721.19 ± 504.82 vs 1416.84 ± 540.21 U/L, P=0.008), higher INR (1.32 ± 0.30 vs 1.17 ± 0.20, P=0.036), and longer hospital stay (9.66 ± 1.69 vs 5.00 ± 1.55 days, P<0.001). Albumin showed a borderline lower mean value in the adverse-outcome group (3.66 ± 0.50 vs 3.87 ± 0.39 g/dL, P=0.051).Ultrasonography revealed gallbladder wall edema in 47.1% and periportal echogenicity in 40.4%. Ascites on ultrasonography was identified in 7.4% overall and was confined to the adverse-outcome group (34.5% vs 0.0%, P<0.001). Cholestatic hepatitis occurred in 8.8%,

coagulopathy in 13.2%, hepatic encephalopathy in 5.1%, acute liver failure in 3.7%, ICU requirement in 5.9%, and pleural effusion in 2.9% (Figure 2). One child died, yielding an in-hospital mortality of 0.7%. Overall, 29 children (21.3%) met the predefined composite adverse-outcome definition. Predictor analysis is summarized in Table 4. On univariable analysis, altered sensorium at admission (OR 43.93, 95% CI 6.87-259.55; P<0.001), bilirubin >10 mg/dL (OR 4.74, 95% CI 1.73-11.68; P=0.006), albumin <3.5 g/dL (OR 3.81, 95% CI 1.50-8.51; P=0.009), and INR ≥1.5 (OR 6.71, 95% CI 2.35-23.69; P<0.001) were associated with adverse course, while age >10 years showed borderline significance (OR 2.43, 95% CI 0.96-5.72; P=0.056).

On multivariable logistic regression, age >10 years (adjusted OR 3.32, 95% CI 1.09-10.08; P=0.034), altered sensorium at admission (adjusted OR 37.15, 95% CI 3.83-360.79; P=0.002), and bilirubin >10 mg/dL (adjusted OR 5.84, 95% CI 1.67-20.47; P=0.006) remained independent predictors of adverse outcome.

Table 1: Baseline socio-demographic and epidemiologic profile of enrolled children

Characteristic	Overall (N=136)	No adverse outcome (n=107)	Adverse outcome (n=29)	P value
Age (years), mean ± SD	7.40 ± 4.07	7.08 ± 4.04	8.59 ± 4.02	0.082
Age group: 1-5 years	48 (35.3)	41 (38.3)	7 (24.1)	0.104
Age group: 6-10 years	52 (38.2)	42 (39.3)	10 (34.5)	
Age group: 11-15 years	36 (26.5)	24 (22.4)	12 (41.4)	
Male sex	82 (60.3)	63 (58.9)	19 (65.5)	0.669
Rural residence	89 (65.4)	68 (63.6)	21 (72.4)	0.510
Lower socioeconomic class	100 (73.5)	78 (72.9)	22 (75.9)	0.817
Unsafe drinking water source	81 (59.6)	64 (59.8)	17 (58.6)	1.000
Unvaccinated against hepatitis A	129 (94.9)	101 (94.4)	28 (96.6)	1.000
Admission during monsoon/post-monsoon months	79 (58.1)	65 (60.7)	14 (48.3)	0.289

Table 2: Clinical presentation and examination findings by adverse outcome status

Clinical feature / sign	Overall (N=136)	No adverse outcome (n=107)	Adverse outcome (n=29)	P value
Jaundice	136 (100.0)	107 (100.0)	29 (100.0)	—
Icterus	136 (100.0)	107 (100.0)	29 (100.0)	—
Fever	123 (90.4)	95 (88.8)	28 (96.6)	0.299
Anorexia	109 (80.1)	83 (77.6)	26 (89.7)	0.193
Abdominal pain	96 (70.6)	75 (70.1)	21 (72.4)	1.000
Vomiting	75 (55.1)	57 (53.3)	18 (62.1)	0.528
Dark urine	98 (72.1)	78 (72.9)	20 (69.0)	0.649
Pruritus	26 (19.1)	15 (14.0)	11 (37.9)	0.007
Pale stool	20 (14.7)	11 (10.3)	9 (31.0)	0.014
Altered sensorium	12 (8.8)	1 (0.9)	11 (37.9)	<0.001
Bleeding manifestation	3 (2.2)	3 (2.8)	0 (0.0)	1.000
Hepatomegaly	120 (88.2)	95 (88.8)	25 (86.2)	0.747
Tender liver	70 (51.5)	56 (52.3)	14 (48.3)	0.834
Splenomegaly	20 (14.7)	17 (15.9)	3 (10.3)	0.566

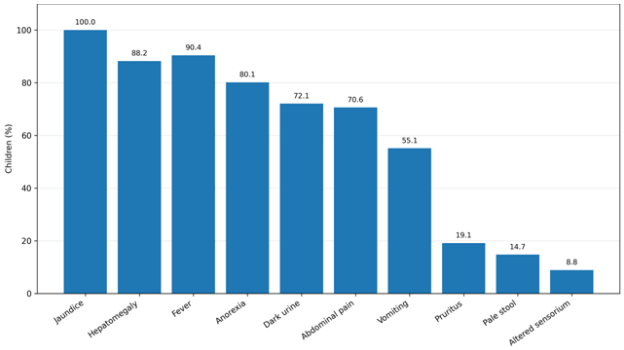

Figure 1: Frequency of major presenting clinical features in hospitalized children with hepatitis A

Table 3: Laboratory profile, ultrasonographic findings, complications, and outcomes

Laboratory / outcome variable	Overall (N=136)	No adverse outcome (n=107)	Adverse outcome (n=29)	P value
Total bilirubin (mg/dL)	7.37 ± 2.63	7.08 ± 2.48	8.44 ± 2.91	0.027
AST (U/L)	1181.06 ± 461.16	1187.56 ± 449.54	1157.07 ± 509.47	0.704
ALT (U/L)	1481.73 ± 548.97	1416.83 ± 544.57	1721.19 ± 504.82	0.008
ALP (U/L)	299.05 ± 85.31	295.64 ± 83.02	311.62 ± 93.75	0.308
Serum albumin (g/dL)	3.82 ± 0.43	3.86 ± 0.40	3.66 ± 0.50	0.051
INR	1.20 ± 0.23	1.17 ± 0.19	1.32 ± 0.30	0.036
Serum ammonia (µmol/L)	50.00 ± 18.81	49.55 ± 17.99	51.69 ± 21.83	0.636
Hospital stay (days)	5.99 ± 2.63	4.99 ± 1.84	9.66 ± 1.69	<0.001
Gallbladder wall edema on ultrasonography	69 (50.7)	54 (50.5)	15 (51.7)	1.000
Periportal echogenicity on ultrasonography	47 (34.6)	36 (33.6)	11 (37.9)	0.666
Ascites on ultrasonography	10 (7.4)	0 (0.0)	10 (34.5)	<0.001
Cholestatic hepatitis	12 (8.8)	0 (0.0)	12 (41.4)	<0.001
Coagulopathy (INR ≥1.5)	18 (13.2)	7 (6.5)	11 (37.9)	<0.001
Hepatic encephalopathy	7 (5.1)	0 (0.0)	7 (24.1)	<0.001
Acute liver failure	5 (3.7)	0 (0.0)	5 (17.2)	<0.001
ICU requirement	8 (5.9)	0 (0.0)	8 (27.6)	<0.001
Death	1 (0.7)	0 (0.0)	1 (3.4)	0.213

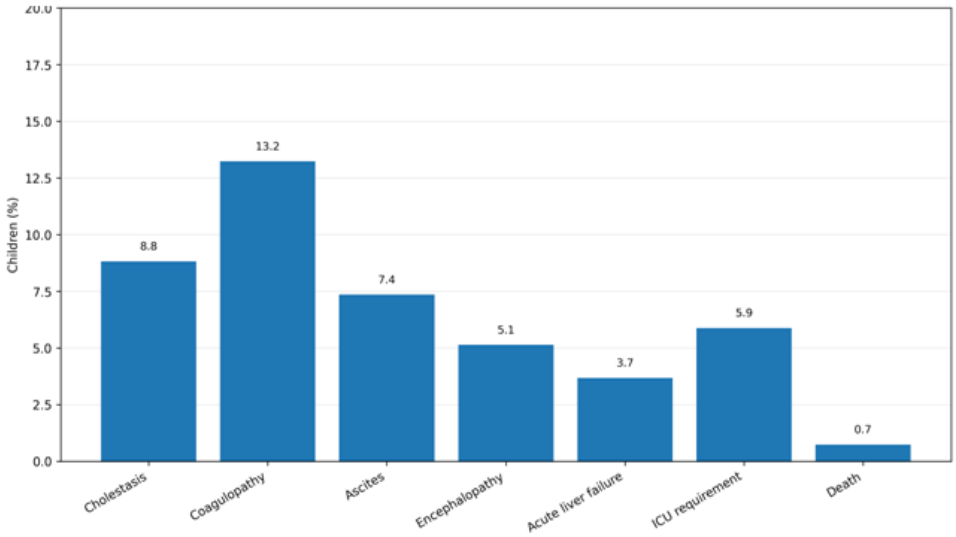

Figure 2: Complication and outcome spectrum among hospitalized children with hepatitis A

Table 4: Predictors of adverse outcome in hospitalized children with hepatitis A

Predictor	Adverse outcome with factor / total with factor	Univariable OR (95% CI)	P value	Adjusted OR (95% CI)	Adjusted P value
Age >10 years	12/36	2.43 (1.04-5.72)	0.057	3.32 (1.09-10.08)	0.034
Male sex	19/82	1.30 (0.56-3.02)	0.669	—	—
Rural residence	21/89	1.46 (0.60-3.54)	0.510	—	—
Unsafe drinking water source	17/81	0.94 (0.42-2.15)	1.000	—	—
Altered sensorium at admission	11/12	44.14 (7.51-259.55)	<0.001	37.18 (3.83-360.79)	0.002
Bleeding manifestation	0/3	0.51 (0.03-10.08)	1.000	—	—
Total bilirubin >10 mg/dL	9/19	4.30 (1.59-11.68)	0.006	5.84 (1.67-20.47)	0.006

Serum albumin <3.5 g/dL	11/27	3.45 (1.40-8.51)	0.009	2.03 (0.56-7.37)	0.282
INR ≥1.5	11/18	8.33 (2.93-23.69)	<0.001	3.35 (0.75-14.98)	0.113
ALT >2000 U/L	9/25	2.57 (1.01-6.52)	0.060	—	—

Note: Adverse outcome was defined as cholestatic hepatitis, ultrasonographic ascites, pleural effusion, hepatic encephalopathy, acute liver failure, ICU requirement, or in-hospital death. Adjusted odds ratios are from multivariable logistic regression including age >10 years, altered sensorium at admission, total bilirubin >10 mg/dL, serum albumin <3.5 g/dL, and INR ≥1.5.

Discussion

The present tertiary-care study demonstrates that hepatitis A in hospitalized children is predominantly a disease of school-age children from socially and environmentally vulnerable backgrounds, but its bedside expression is not uniformly benign. Although most children followed a self-limited course, more than one-fifth developed a predefined adverse outcome, and a clinically important minority had cholestasis, coagulopathy, encephalopathy, acute liver failure, or ICU requirement. Three findings merit emphasis. First, the case mix was concentrated in rural, lower socioeconomic, largely unvaccinated children, with admissions clustering in monsoon and post-monsoon months. Second, while the core phenotype remained classic acute icteric hepatitis, admission features suggesting cholestasis or neurologic involvement were strongly linked to worse course. Third, older age, altered sensorium, and bilirubin >10 mg/dL independently identified children at higher risk of adverse in-hospital evolution.

The demographic profile of our cohort resembles contemporary South Asian hospital-based pediatric HAV series. The mean age of 7.4 years and the predominance of the 6-10 year age group are close to the age distribution reported by Murlidharan et al. and Rasheed et al., both of whom found the disease concentrated in early- and mid-childhood hospitalized cohorts [9,10]. Baidya et al. similarly described a tertiary-care profile dominated by admitted children with overt jaundice and a meaningful complication burden [13]. The rural predominance, high proportion from lower socioeconomic class, and association with unsafe water source in our series are consistent with the persisting environmental epidemiology of HAV in the Indian subcontinent [4-8,14]. These findings reinforce that, despite epidemiologic transition, water and sanitation inequities continue to shape pediatric hospitalization patterns.

Our symptom complex was classical. Jaundice and icterus were universal, and fever, anorexia, dark urine, abdominal pain, vomiting, and hepatomegaly dominated the clinical picture. This pattern is highly concordant with earlier Indian and regional studies. Murlidharan et al. reported fever in 96%, abdominal pain in 78%, dark urine in 65%, vomiting in 47%, icterus in 80%, and hepatomegaly in 74% [9]. Rasheed et al. and Gupta et al. likewise described fever and jaundice among the commonest presenting features in children with acute infective hepatitis, with HAV as a major etiology [10,11]. Kumar et al. also documented a largely typical presentation in most children, emphasizing that the majority recover uneventfully despite striking enzyme elevation [12]. The consistency across studies strengthens the practical message that symptomatic HAV in admitted children can often be recognized clinically, but serological confirmation remains essential to distinguish it from HEV, dengue-associated hepatitis, enteric fever, and other regional mimics [10,11,14].At the same time, our data show that "typical" does not always mean "mild." Pruritus and pale stool were significantly enriched among children with adverse outcome, suggesting that early cholestatic tendency may signal more prolonged or complicated disease. This aligns with reports that atypical or prolonged manifestations of HAV are not rare enough to be ignored [19,20]. Biochemically, our cohort showed marked transaminase elevation, but enzyme magnitude was less informative than bilirubin and coagulation abnormality. Kumar et al. similarly found that very high AST and ALT levels do not necessarily parallel severity or prolonged recovery [12]. In our analysis, bilirubin >10 mg/dL independently predicted adverse outcome, while INR ≥1.5 showed a strong univariable association. This pattern mirrors outcome-oriented studies from Bangladesh and India, where hyperbilirubinemia and coagulation derangement repeatedly emerge as warning markers [15,17,18,26].

The complication profile in our cohort falls within the range reported from admitted pediatric populations but is milder than that observed in dedicated acute liver failure units. Cholestatic hepatitis was seen in 8.8%, coagulopathy in 13.2%, encephalopathy in 5.1%, and acute liver failure in 3.7%, with mortality of 0.7%. Baidya et al. reported cholestasis in 9.5% and acute liver failure in 4.5%, figures strikingly similar to our findings [13]. Kumar et al. also documented uncommon but important complications including cholestatic hepatitis and fulminant disease in a small minority [12]. In contrast, cohorts enriched for severe acute hepatitis or referred to specialized liver units show

substantially higher rates of encephalopathy, acute liver failure, and mortality [15,17,26]. This difference likely reflects referral bias and emphasizes that the observed spectrum depends strongly on the level of care at which children are studied.

Our predictor analysis provides clinically usable triage information. Altered sensorium at admission was the strongest independent predictor of adverse outcome, underscoring the primacy of careful neurologic assessment. Although the confidence interval was wide because of the small number of affected children, the strength and direction of the association are fully consistent with pediatric hepatology principles and recent outcome studies [15,17,18,26]. Bilirubin >10 mg/dL also remained independently predictive, supporting its role as a simple admission-level marker for closer observation. Age >10 years retained independent significance after adjustment, which fits the broader epidemiologic concept that older children are more likely to experience symptomatic and complicated disease as endemicity shifts [1,5-7,21,22].

The public health implications are equally important. The very high proportion of unvaccinated children, combined with monsoon clustering and socioeconomic vulnerability, highlights missed preventive opportunities. Indian seroepidemiology now indicates that universal very-early-life exposure can no longer be assumed in all settings [5,6].

Reviews, expert consensus statements, and economic evaluations increasingly support more active vaccine policy deliberation in regions undergoing endemicity transition [8,23-25]. Hospital data such as ours matter in that debate because they quantify not merely infection, but clinically meaningful morbidity requiring admission, repeated laboratory monitoring, imaging, and occasionally ICU care.

This study is limited by its hospital-based design, which cannot represent the full community spectrum of HAV, much of which remains outpatient or subclinical. Being a tertiary-care cohort, it likely overestimates complication frequency relative to population-level disease. Long-term outcomes such as relapsing hepatitis or duration of cholestasis after discharge were not evaluated. Even so, the study provides a clinically coherent description of admitted pediatric HAV and identifies practical admission-level markers that may help early risk stratification.

In summary, hepatitis A in hospitalized children remains recognizable by its classic phenotype but demands respect for its potential to evolve into cholestatic or acute liver failure syndromes in a meaningful subset. Older age, altered sensorium, and marked hyperbilirubinemia emerged as the most actionable predictors of adverse course. These findings support vigilant inpatient triage, timely escalation for neurologic or coagulation warning signs, and renewed public-health emphasis on safe water, sanitation, and vaccination in settings where the epidemiology of HAV is shifting [5-8,15,23-25].

Conclusion

Hepatitis A in children admitted to a tertiary care hospital showed a broad clinical spectrum. Most children had classical self-limited icteric hepatitis, but a substantial minority developed cholestasis, coagulopathy, encephalopathy, acute liver failure, or required intensive care. Rural residence, low socioeconomic background, unsafe water exposure, and lack of vaccination remained common contextual features. Among admission variables, older age, altered sensorium, and bilirubin >10 mg/dL were the strongest indicators of adverse course. Early recognition of these markers, close biochemical monitoring, and timely escalation of care may improve outcomes, while strengthening sanitation and hepatitis A immunization strategies may reduce future hospitalization burden.

References

1. World Health Organization. Hepatitis A. Fact sheet. 2025.
2. Migueres M, Lhomme S, Izopet J. Hepatitis A: epidemiology, high-risk groups, prevention and research on antiviral treatment. Viruses. 2021;13(10):1900.
3. Gholizadeh O, Akbarzadeh S, Ghazanfari Hashemi M, et al. Hepatitis A: Viral Structure, Classification, Life Cycle, Clinical Symptoms, Diagnosis Error, and Vaccination. Can J Infect Dis Med Microbiol. 2023; 2023:4263309.
4. Franco E, Meleleo C, Serino L, Sorbara D, Zaratti L. Hepatitis A: epidemiology and prevention in developing countries. World J Hepatol. 2012;4(3):68-73.
5. Deoshatwar AR, Pulinchani A, Mathapati B, et al. Hepatitis A virus endemicity and vaccine policy, India. Bull World Health Organ. 2026. Online ahead of print.
6. Shenoy B, Andani A, Kolhapure S, Agrawal A, Mazumdar J. Endemicity change of hepatitis A infection necessitates vaccination in food handlers: an Indian perspective. Hum Vaccin Immunother. 2022;18(1):1868820.
7. Agrawal A, Singh S, Kolhapure S, Hoet B, Arankalle V, Mitra M. Increasing Burden of Hepatitis A in Adolescents and Adults and the Need for Long-Term Protection: A Review from the Indian Subcontinent. Infect Dis Ther. 2019;8(4):483-497.
8. Shah N, Faridi MMA, Bhave S, et al. Expert consensus and recommendations on the live

attenuated hepatitis A vaccine and immunization practices in India. Hum Vaccin Immunother. 2025;21(1):2447643.
9. Murlidharan S, Sangle AL, Engade M, Kale AB. The Clinical Profile of Children With Hepatitis A Infection: An Observational Hospital-Based Study. Cureus. 2022; 14(8): e28290.
10. Rasheed J, Khalid M, Rubab S, Iqbal B, Nawaz I, Shahzad A. Clinical and Epidemiological Spectrum of Acute Viral Hepatitis Due to Hepatitis A and E in Children: A Descriptive, Cross-Sectional, Hospital-Based Study. Cureus. 2022; 14(4): e24056.
11. Gupta HB, Deshpande T, Choraria N, Patel P, Sethia SG, Sethia S. Clinico-Serological Profile of Infective Causes of Acute Hepatitis in Children Admitted to a Tertiary Care Centre. Cureus. 2023;15(4):e38237.
12. 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):15-19.
13. Baidya M, Mahmud S, Rashid R, Hasan AR, Ahmed Muaz SS, Farhana T. Clinical profile of hepatitis A virus infection in children in a tertiary care hospital. Int J Contemp Pediatr. 2025; 12(4):542-547.
14. Murhekar MV, Ashok M, Kanagasabai K, et al. Epidemiology of hepatitis A and hepatitis E based on laboratory surveillance data-India, 2014-2017. Am J Trop Med Hyg. 2018; 99(4): 1058-1061.
15. Alam R, Fathema K, Nahid KL, Sarkar N, Rukunuzzaman M. Possible predictors of adverse outcomes in children with acute viral hepatitis in a low-middle-income country. Turk Arch Pediatr. 2025;60(6):616-621.
16. Aswanth KS, Tabassum S, et al. Clinico-etiological profile and predictors of outcome in pediatric acute hepatitis: an observational study from South India. J Trop Pediatr. 2026; 72(2):fmag010.
17. Alam S, Khanna R, Sood V, Lal BB, Rawat D. Profile and outcome of first 109 cases of paediatric acute liver failure at a specialized paediatric liver unit in India. Liver Int. 2017; 37(10): 1508-1514.
18. Yachha SK, 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): 477-482.
19. Singh SK, Borkar V, Srivastava A, Mathias A, Yachha SK, Poddar U. Need for recognizing atypical manifestations of childhood sporadic acute viral hepatitis warranting differences in management. Eur J Pediatr. 2019;178(1):61-67.
20. Alam R, Karim ASMB, Mazumder MW, Das SR, Benzamin M, Sonia ZF, Rahman SMH. Atypical manifestations of acute viral hepatitis A in children in Bangladesh: are these really uncommon? Indian J Gastroenterol. 2021; 40(5): 470-476.
21. Wasley A, Fiore A, Bell BP. Hepatitis A in the era of vaccination. Epidemiol Rev. 2006; 28:101-111.
22. Wang F, Sun X, Wang F, et al. Changing epidemiology of hepatitis A in China: evidence from three national serological surveys and the national notifiable disease reporting system. Hepatology. 2021;73(4):1251-1260.
23. Andani A, van Damme P, Bunge EM, Salgado F, van Hoorn RC, Hoet B. One or two doses of hepatitis A vaccine in universal vaccination programs in children in 2020: a systematic review. Vaccine. 2022;40(2):196-205.
24. Gurav YK, Bagepally BS, Chitpim N, et al. Cost-effective analysis of hepatitis A vaccination in Kerala state, India. PLoS One. 2024;19: e0306293.
25. Anand AC, Roy A. Hepatitis A: A wake-up call! Indian J Gastroenterol. 2025; 44:581-584.
26. Srivastava A, Yachha SK, Poddar U. Predictors of outcome in children with acute viral hepatitis and coagulopathy. J Viral Hepat. 2012;19(2):e194-e201.