Original Article

Clinical Profile of Hepatitis A in Children: A Hospital-Based Study from GMC Handwara, District Kupwara, Kashmir

Showkat Mohmad Bhat[1], Irshad Ahmad Bhat[2], Gazala Gul[3], Naseer Yousuf Mir[2], Khalid Rahim Malik[4]

[1]Senior Resident, Department of Pediatrics, Government Medical College Handwara, District Kupwara, Jammu and Kashmir, India.

[2]Assistant Professor, Department of Pediatrics, Government Medical College Handwara, District Kupwara, Jammu and Kashmir, India.

[3]Assistant Professor, Department of Pathology, Government Medical College Handwara, District Kupwara, Jammu and Kashmir, India.

[4]Consultant, Department of Pediatrics, Government Medical College Handwara, District Kupwara, Jammu and Kashmir, India

Abstract

Background: Hepatitis A remains an important cause of acute viral hepatitis in children, and clinical severity may increase with age. District-specific data from Kashmir are limited. **Objective:** To describe the demographic, clinical, biochemical, seasonal, severity, and short-term outcome profile of laboratory-confirmed hepatitis A in children presenting to a tertiary-care hospital in northern India. **Material and Methods:** This hospital-based observational study included children aged 1 month to 16 years presenting with clinical and biochemical features of acute hepatitis from August 2025 to July 2026. Acute hepatitis A was confirmed by anti-hepatitis A virus immunoglobulin M (IgM) detected by enzyme-linked immunosorbent assay (ELISA). Screening for Hepatitis B, C, and E was performed in all participants. Demographic, clinical, biochemical, severity, and outcome data were recorded and analyzed using IBM SPSS Statistics version 20. **Results:** Among 210 children, 121 (57.6%) were male and 89 (42.4%) were female; mean age was 7.4 ± 3.6 years. Children aged 5–10 years constituted 102 (48.6%) cases. Fever (92.8%), jaundice (89.5%), anorexia (84.3%), vomiting (71.4%), and hepatomegaly (76.2%) were frequent. Mean total bilirubin, alanine aminotransferase, and aspartate aminotransferase were 6.8 ± 3.2 mg/dL, 412 ± 156 IU/L, and 386 ± 142 IU/L, respectively. Acute liver failure occurred in 14 (6.7%) children. Older age groups had higher mean bilirubin, alanine aminotransferase, and international normalized ratio values (P=0.031, 0.042 and 0.028 respectively). Monsoon and post-monsoon periods accounted for 78 (37.1%) and 62 (29.5%) cases, respectively. All children survived without documented short-term sequelae. **Conclusion:** Hepatitis A predominantly affected school-aged children in this cohort. Acute liver failure occurred in a minority, while short-term survival was excellent. The seasonal concentration of cases supports continued emphasis on safe water, sanitation, hygiene, surveillance, and locally appropriate prevention strategies.

Keywords: Hepatitis A; Child; Liver Failure, Acute; Seasonality; India.

Received: 05 July 2026 **Revised: 20 July 2026** **Accepted: 10 August 2026** **Published: 09 September 2026**

Introduction

Hepatitis A is an acute infection caused by hepatitis A virus (HAV), a non-enveloped RNA virus transmitted predominantly through the fecal-oral route. Transmission is strongly linked to unsafe water, inadequate sanitation, food contamination, and close contact with infected individuals. Although most infections resolve completely without chronic liver disease, clinical severity increases with age and a small proportion of patients develop fulminant hepatitis or acute liver failure.[1,2]

Worldwide, the epidemiology of HAV is changing as socioeconomic conditions, sanitation, and vaccination coverage improve. In highly endemic settings, infection commonly occurs during early childhood and is often asymptomatic. In settings undergoing an epidemiological transition, a larger proportion of children may remain susceptible to infection until later childhood or adolescence, when symptomatic disease is more common and severe outcomes become more likely.[1,3] The World Health Organization recognizes safe water, sanitation, hygiene, food safety, and vaccination as major tools for prevention and notes that vaccination strategies should be tailored to local epidemiology and cost-effectiveness considerations.[1,3]

India has historically had high levels of HAV exposure during childhood, although regional variation and evidence of changing age at infection have been reported. Hospital-based pediatric studies from different parts of India demonstrate substantial variation in age distribution, clinical manifestations, biochemical abnormalities, and complications.[4-7] A recent study from Jammu reported that fever and biochemical evidence of hepatic injury were common among children with hepatitis A, while an earlier Cureus study from Maharashtra documented a broad spectrum of clinical and laboratory findings.[5,6]

Data from the Kashmir region are comparatively limited. In a

Address for correspondence: Dr. Irshad Ahmad Bhat, Assistant Professor, Department of Pediatrics, Government Medical College Handwara, District Kupwara, Jammu and Kashmir, India.
E-mail: bhat297@gmail.com

DOI:
10.21276/amit.2026.v13.i3.1020

How to cite this article: Bhat SM, Bhat IA, Gul G, Mir NY, Malik KR. Clinical Profile of Hepatitis A in Children: A Hospital-Based Study from GMC Handwara, District Kupwara, Kashmir. Acta Med Int. 2026;13(3):64-69.

study from neighboring Shopian district, all 83 children presenting with acute icteric hepatitis were anti-HAV IgM positive, suggesting a substantial contribution of HAV to pediatric acute hepatitis in that population.[4] However, district-specific clinical and outcome data from Kupwara are scarce. Government Medical College Handwara provides secondary and tertiary pediatric services to a predominantly rural population of District Kupwara and therefore offers an important setting for describing the local disease profile.

The present study was undertaken to characterize the demographic and clinical profile of laboratory-confirmed hepatitis A in children presenting to Government Medical College Handwara, describe biochemical abnormalities and seasonal variation, determine the frequency of acute liver failure, and document short-term outcomes. These findings may help clinicians recognize severe disease promptly and may provide locally relevant information for preventive health planning.

Materials and Methods

Study design and setting: This was a hospital-based observational study conducted in the Department of Pediatrics, Government Medical College Handwara, District Kupwara, Jammu and Kashmir, India, over 12 months from August 2025 through July 2026. The study population consisted of children presenting with clinical features suggestive of acute hepatitis and subsequently confirmed to have acute HAV infection by serology.

Participants and case ascertainment: Children aged 1 month to 16 years with clinical and biochemical features compatible with acute hepatitis were eligible. Laboratory confirmation required detection of anti-HAV immunoglobulin M (IgM) by enzyme-linked immunosorbent assay (ELISA). All enrolled children underwent testing for HBsAg, anti-HCV antibodies, and anti-HEV IgM to identify alternative or concurrent hepatotropic viral infections. Children with chronic liver disease, known hepatotoxic drug ingestion, or evidence of hepatitis B, C, or E infection were excluded.

Clinical and laboratory assessment: Demographic information, presenting symptoms, duration of illness, and physical examination findings were recorded using a predesigned case record form. Clinical variables included fever, jaundice, anorexia, vomiting, abdominal pain, dark urine, pale stools, pruritus, hepatomegaly, splenomegaly, ascites, and hepatic encephalopathy. Laboratory assessment included total, direct, and indirect bilirubin; alanine aminotransferase (ALT); aspartate aminotransferase (AST); alkaline phosphatase (ALP); prothrombin time (PT); international normalized ratio (INR); and complete blood count.

Definition of acute liver failure: Acute liver failure (ALF) was defined using criteria adapted from the Pediatric Acute Liver Failure Study Group: evidence of acute liver injury in a child without known chronic liver disease together with an INR ≥1.5 in the presence of hepatic encephalopathy or an INR ≥2.0 in the absence of encephalopathy, after assessment and correction of vitamin K deficiency where appropriate.[9]

Management and outcome assessment: Treatment was supportive because uncomplicated HAV infection has no specific antiviral therapy. Management included hydration, nutritional support, antiemetic therapy when required, monitoring of glucose and coagulation status, and vitamin K for clinically relevant coagulopathy. Fresh frozen plasma was administered when clinically indicated, including marked prolongation of coagulation parameters. Children meeting criteria for ALF were managed with intensive monitoring. The recorded outcomes included survival, clinical recovery, documented sequelae, and duration of hospitalization.

Statistical analysis: Data were entered and analyzed using IBM SPSS Statistics version 20 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation and categorical variables as frequencies and percentages. Comparisons of continuous biochemical variables across the three age groups were performed using one-way analysis of variance, with categorical comparisons performed using the chi-square test or Fisher's exact test when appropriate. A two-sided p-value <0.05 was considered statistically significant. Because individual-level data were not used to generate the figures in this manuscript, the graphical displays are descriptive and do not introduce additional inferential analyses beyond those reported in the tables.

Results

During the 12-month study period, 210 children fulfilled the eligibility criteria and were included in the final analysis. All participants had laboratory confirmation of acute HAV infection and underwent screening for hepatitis B, C, and E as described in the Methods.

Demographic characteristics: The cohort comprised 121 (57.6%) males and 89 (42.4%) females, giving a male-to-female ratio of 1.36:1. The mean age was 7.4 ± 3.6 years, with an age range of 1–16 years. Children aged 5–10 years represented the largest group (102, 48.6%), followed by those aged 11–16 years (58, 27.6%) and 1–4 years (50, 23.8%).

Table 1: Age-group and sex distribution of the study participants.

Age group	Male, n	Female, n	Total, n (%)
1–4 years	28	22	50 (23.8%)
5–10 years	61	41	102 (48.6%)
11–16 years	32	26	58 (27.6%)
Total	121	89	210 (100%)

Clinical presentation: Fever was the most frequent presenting symptom, reported in 195 (92.8%) children, followed by jaundice in 188 (89.5%), anorexia in 177 (84.3%), vomiting in 150 (71.4%), and abdominal pain in 138 (65.7%). Dark urine was reported in 167 (79.5%) children and pale stools in 92 (43.8%). Hepatomegaly was

present in 160 (76.2%) children, while splenomegaly and ascites were documented in 58 (27.6%) and 18 (8.6%), respectively. Hepatic encephalopathy was recorded in 12 (5.7%) children. The mean duration of symptoms before presentation was 6.4 ± 2.8 days.

Table 2: Clinical features of children with laboratory-confirmed hepatitis A.

Clinical feature	n	%
Fever	195	92.8
Jaundice	188	89.5
Anorexia	177	84.3
Vomiting	150	71.4
Abdominal pain	138	65.7
Dark urine	167	79.5
Pale stools	92	43.8
Pruritus	45	21.4
Hepatomegaly	160	76.2
Splenomegaly	58	27.6
Ascites	18	8.6
Hepatic encephalopathy	12	5.7

Biochemical profile: The mean total bilirubin concentration was 6.8 ± 3.2 mg/dL, with a mean direct bilirubin of 4.2 ± 2.1 mg/dL. Mean ALT and AST concentrations were 412 ± 156 IU/L and 386 ± 142 IU/L, respectively. The mean ALP was 245 ± 78 IU/L. Mean PT was 14.2 ± 4.1 seconds and mean INR was 1.2 ± 0.5, with an observed INR range of 0.9–4.2.

Table 3: Biochemical profile of children with laboratory-confirmed hepatitis A.

Parameter	Mean ± SD	Range
Total bilirubin (mg/dL)	6.8 ± 3.2	2.1–18.4
Direct bilirubin (mg/dL)	4.2 ± 2.1	1.2–11.6
AST (IU/L)	386 ± 142	120–980
ALT (IU/L)	412 ± 156	145–1120
ALP (IU/L)	245 ± 78	110–520
PT (seconds)	14.2 ± 4.1	11.2–35.6
INR	1.2 ± 0.5	0.9–4.2

Age-wise biochemical comparison: Biochemical severity increased across the age groups. Mean total bilirubin rose from 5.9 ± 2.8 mg/dL in children aged 1–4 years to 7.4 ± 3.1 mg/dL in those aged 11–16 years. Mean ALT increased from 378 ± 132 IU/L to 438 ± 152 IU/L, while mean INR increased from 1.1 ± 0.4 to 1.4 ± 0.6 across the same age groups. The reported between-group differences were statistically significant for bilirubin (p=0.031), ALT (p=0.042), and INR (p=0.028).

Table 4: Age-wise comparison of selected biochemical parameters.

Parameter	1–4 years	5–10 years	11–16 years	p-value
Total bilirubin (mg/dL)	5.9 ± 2.8	7.1 ± 3.4	7.4 ± 3.1	0.031
ALT (IU/L)	378 ± 132	420 ± 168	438 ± 152	0.042
INR	1.1 ± 0.4	1.3 ± 0.5	1.4 ± 0.6	0.028

Severity, acute liver failure, and outcomes: Most children (196, 93.3%) had acute viral hepatitis without ALF, while 14 (6.7%) met the study definition of ALF. Among the 14 children with ALF, nine (64.3%) had hepatic encephalopathy and five (35.7%) had INR ≥2.0 without encephalopathy. All ALF cases received vitamin K and were managed with intensive monitoring; eight (57.1%) received fresh frozen plasma. Mean hospitalization duration was 12.4 ± 4.2 days among ALF cases compared with 5.8 ± 2.1 days among children without ALF. All 210 children survived, and no documented mortality or residual sequelae were reported.

Table 5: Severity and outcomes.

Severity/outcome	n	%
Acute viral hepatitis without ALF	196	93.3
Acute liver failure	14	6.7
ALF with encephalopathy	9	64.3
ALF without encephalopathy	5	35.7
Complete recovery	210	100
Recovery with sequelae	0	0
Death	0	0

Seasonal distribution: Cases showed a clear seasonal concentration. Seventy-eight (37.1%) children presented during the monsoon period (July–September), while 62 (29.5%) presented during the post-monsoon period (October–December).

Together, these periods accounted for 140 (66.7%) cases.

Winter and summer contributed 38 (18.1%) and 32 (15.2%) cases, respectively.

Table 6: Seasonal distribution of pediatric hepatitis A cases.

Season	Cases, n	Percentage
Monsoon (July–September)	78	37.1
Post-monsoon (October–December)	62	29.5
Winter (January–March)	38	18.1
Summer (April–June)	32	15.2

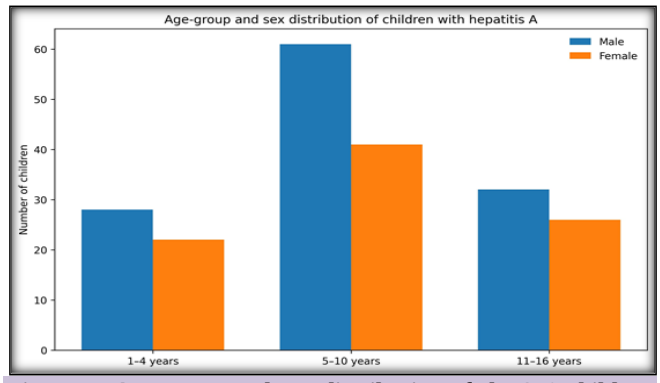

Figure 1: Age-group and sex distribution of the 210 children with laboratory-confirmed hepatitis A.

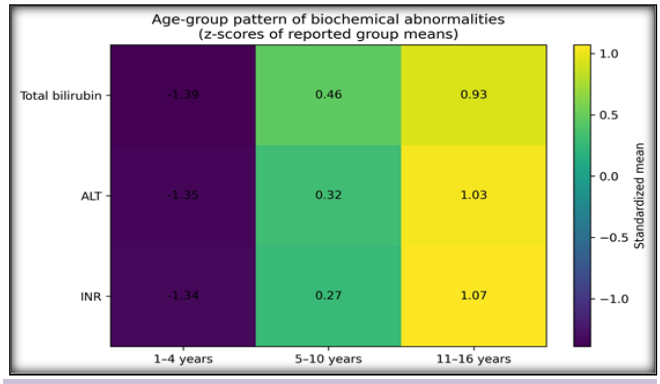

Figure 2: Standardized age-group pattern of selected biochemical parameters. Values are z-scores calculated from the reported age-group means for each analyte; the figure is descriptive and should not be interpreted as a patient-level correlation analysis.

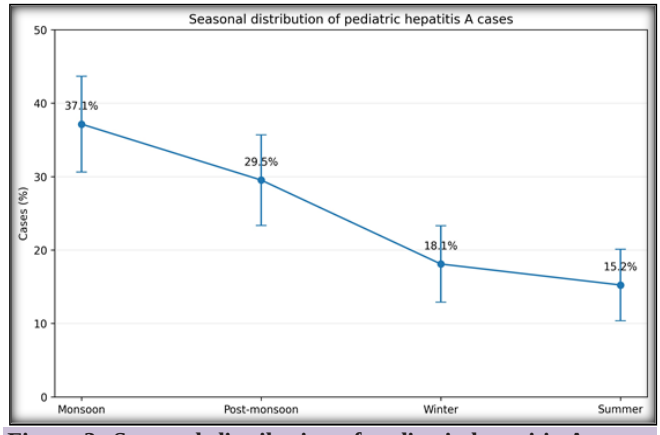

Figure 3: Seasonal distribution of pediatric hepatitis A cases. Points show the observed proportion of cases; vertical bars represent approximate 95% binomial confidence intervals calculated from the aggregate seasonal counts.

Discussion

This study describes the clinical and biochemical characteristics of 210 children with laboratory-confirmed hepatitis A managed at a tertiary-care government medical college in District Kupwara. Three findings are particularly relevant: the predominance of school-aged children, the substantial but limited occurrence of acute liver failure, and the concentration of presentations during the monsoon and post-monsoon periods. The study also demonstrates that, within this hospital cohort, survival was excellent despite the occurrence of ALF.

The age distribution is consistent with the clinical epidemiology of HAV in populations where childhood exposure remains common but symptomatic disease is increasingly observed beyond early childhood. The 5–10-year age group represented almost half of the cohort, whereas more than one-quarter of cases occurred among children aged 11–16 years. The latter finding is clinically important because older age at infection is associated with a greater likelihood of symptomatic and severe disease.[1,3] A recent Indian study involving a large laboratory dataset also described an increasing contribution of symptomatic HAV infection among adolescents and adults, supporting continued surveillance of age-specific patterns.[7]

Male children constituted 57.6% of the cohort. A male predominance has been reported in several hospital-based Indian series, although the magnitude and direction of sex differences vary between populations.[5,6] The present study cannot establish a biological explanation for this difference. Differences in exposure, household roles, healthcare-seeking behavior, or the composition of the hospital catchment population may contribute. Accordingly, the observed sex distribution should be interpreted descriptively rather than as evidence of sex-specific susceptibility.

The clinical presentation in our cohort was typical of symptomatic hepatitis A. Fever, jaundice, anorexia, vomiting, and hepatomegaly were frequent. These findings broadly resemble those reported in other Indian pediatric cohorts. In the 2022 Cureus study from Maharashtra, fever and hepatomegaly were common, while the Jammu study published in 2025 also identified fever as the leading clinical manifestation.[5,6] Differences in the frequency of individual symptoms between studies may reflect age distribution, referral patterns, diagnostic thresholds, and the proportion of children managed as outpatients versus inpatients.

The biochemical profile demonstrated substantial hepatocellular injury, with ALT and AST markedly elevated in the cohort. Bilirubin was also elevated, with predominantly direct hyperbilirubinemia. Importantly, older age groups had higher mean bilirubin, ALT, and INR values. This age-related pattern is biologically plausible because clinically apparent HAV becomes more frequent and severe with increasing age.[1,3]

However, the present data are aggregate and cross-sectional at the level of reported summary statistics; therefore, the study should not be interpreted as establishing an independent causal effect of age on biochemical severity.

Acute liver failure occurred in 14 children (6.7%). Published pediatric series have reported variable rates of severe disease and ALF, reflecting differences in case definition, referral setting, and study population.[5,8] In the Eastern Indian prospective study by Mohanty et al., three of 80 children died, emphasizing that although HAV is generally self-limited, fulminant disease can be life-threatening.[8] The Pediatric Acute Liver Failure Study Group established the importance of standardized coagulation-based criteria for identifying ALF in children, including children without encephalopathy who have persistent severe coagulopathy despite vitamin K.[9]

The absence of mortality in our cohort is notable but should be interpreted cautiously. A zero mortality rate in 210 hospitalized children does not imply that HAV-associated ALF is uniformly benign. Rather, it may reflect early recognition, supportive management, timely correction of coagulopathy, intensive monitoring, and the characteristics of the patients who reached our center. Referral bias may also influence outcomes: patients with rapidly progressive disease may be transferred to specialized liver-transplant centers, while our study captures children managed within the local institution. Consequently, the favorable outcome observed here should not be generalized to all children with HAV-associated ALF.

The seasonal pattern is another important observation. Nearly two-thirds of cases occurred during the monsoon and post-monsoon periods. HAV transmission is strongly influenced by environmental sanitation, water contamination, and fecal-oral spread.[1,2] Seasonal clustering has also been reported in pediatric hospital series.[5] The present study cannot establish that rainfall or water contamination caused the observed increase because environmental exposure data, water-quality testing, and community incidence rates were not collected. Nevertheless, the temporal pattern supports strengthening water-safety and hygiene measures before and during the high-incidence months.

The public-health implications are relevant to a district with substantial rural and semi-rural populations. Prevention of HAV transmission depends on safe drinking water, appropriate sewage disposal, hand hygiene, and food safety. Vaccination can provide an additional preventive strategy. The WHO 2022 position paper recommends that countries consider the introduction of hepatitis A vaccination into childhood schedules when there is evidence of increasing severe disease in older children or adults, a transition from high to intermediate endemicity, and favorable cost-effectiveness.[3] Our hospital-based findings alone cannot establish that routine vaccination should be introduced in Kupwara, but they provide a rationale for community-based sero-epidemiological and health-economic studies that could inform local policy.

This study has several strengths. It included a relatively large pediatric cohort for a single district, used laboratory confirmation with anti-HAV IgM, systematically assessed other major hepatotropic viral infections, and documented clinically important outcomes including ALF and mortality. It also provides district-level data from an area for which published pediatric HAV information is limited.

The study also has limitations. First, it was hospital-based and therefore cannot estimate community incidence or population prevalence. Mild and asymptomatic infections are likely to have been missed. Second, the single-center design limits generalizability to other districts and healthcare settings. Third, socioeconomic variables, vaccination history, household exposures, source of drinking water, sanitation practices, and environmental water-quality measurements were not incorporated, preventing direct assessment of transmission risk factors. Fourth, the study provides aggregate age-group biochemical data rather than individual-level longitudinal measurements; consequently, advanced patient-level predictive modelling or correlation analyses cannot be reliably performed from the summary data presented here. Finally, the follow-up was limited and long-term assessment of relapsing or cholestatic disease was not performed, and the reported absence of sequelae should therefore be interpreted as a short-term outcome.

Conclusion

In this hospital-based cohort from District Kupwara, laboratory-confirmed hepatitis A predominantly affected school-aged children and commonly presented with fever, jaundice, anorexia, vomiting, and hepatomegaly. Acute liver failure occurred in 6.7% of children, but no deaths were recorded during the study period. The concentration of cases during the monsoon and post-monsoon months highlights the importance of sustained attention to safe water, sanitation, hygiene, and seasonal surveillance. Further community-based studies incorporating seroprevalence, vaccination status, household exposures, and environmental risk factors are needed to guide district-specific prevention strategies.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. World Health Organization: Hepatitis A. (2026). Accessed: August 10, 2026: https://www.who.int/news-room /fact-sheets/detail/hepatitis-a
2. 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. 10.4254/wjh.v4.i3.68
3. World Health Organization: Hepatitis A vaccines: WHO position paper, October 2022. Wkly Epidemiol Rec. 2022, 97:493-512. https://www.who.int/publications/i/item/who-wer9740-493-512
4. Lone RA, Khurshid I, Mushtaq A: Incidence of hepatitis A in children with acute icteric hepatitis of district Shopian of Kashmir. Int J Contemp Pediatr. 2022, 9:680-683. 10.18203/2349-3291.ijcp20221613
5. 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:e28290. 10.7759/cureus.28290
6. Sharma HV, Khajuria A, Gupta RK: Clinical and biochemical characteristics of children with hepatitis A infection. J Coll Med Sci Nepal. 2025, 21:29-32. 10.3126/jcmsn.v21i1.70811
7. Grover M, et al.: Rising trend of symptomatic infections due to Hepatitis A virus infection in adolescent and adult age group: an observational study from a tertiary care liver institute in India. Indian J Med Microbiol. 2024, 50:100653. 10.1016/j.ijmmb.2024.100653
8. Mohanty N, Mishra S, Panigrahi S: Fulminant hepatitis A in children, its incidence, presentation, complications and outcome: a study from eastern India. J Nepal Paediatr Soc. 2017, 37:226-231. 10.3126/jnps.v37i3.18100
9. Squires RH Jr, Shneider BL, Bucuvalas J, et al.: Acute liver failure in children: the first 348 patients in the pediatric acute liver failure study group. J Pediatr. 2006, 148:652-658. 10.1016/j.jpeds.2005.12.051
10. Ciocca M: Clinical course and consequences of hepatitis A infection. Vaccine. 2000, 18 Suppl 1:S71-S74. 10.1016/S0264-410X(99)00470-3
11. Wasley A, Fiore A, Bell BP: Hepatitis A in the era of vaccination. Epidemiol Rev. 2006, 28:101-111. 10.1093/epirev/mxj012
12. Kumar KJ, Kumar HC, Manjunath VG, Anitha C, Mamatha S: Hepatitis A in children—clinical course, complications and laboratory profile. Indian J Pediatr. 2014, 81:15-19. 10.1007/s12098-013-1114-8.