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Muthanna Medical Journal
2022; **9**(2):132-145

http://dx.doi.org/10.52113/1/1/2022-132-145

Acute viral hepatitis a in children an epidemiological and clinical course in Babylon

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Abstract

The hepatitis A virus, a picornavirus, is a common cause of hepatitis worldwide. Spread of infection is generally by feco-oral route. Hepatitis A is endemic in developing countries, and most residents are exposed in childhood. The aim of study is to evaluate epidemiology, clinical course, and laboratory profile of hepatitis A in children. Method: In this cross-sectional investigation, 250 children identified with acute viral hepatitis A by IgM antiHAV were hospitalized to our infectious unit from March 2018 to December 2018. The child's location of residence, source of drinking water, manner of fecal sewage, nursery attendance, eating outside the house, fever, gastrointestinal symptoms, and history of contact with a hepatitis patient were recorded. Total serum bilirubin, blood urea nitrogen, liver functions test (AST, ALT), bleeding profile, complete blood count, and abdominal ultrasound were performed. Results: (7.3 2.6) year-olds averaged. The study included 128 women (51.20%) and 122 men (48.80%). 238 (95.2%) patients reported yellow sclera, fever in 181 (72.4%), vomiting in 180 (72%), and stomach pain in 171 (67.41%). 168 individuals had hepatomegaly, 34 had splenomegaly, and 6 had ascites. 72 (28.8%) patients had anemia, 15 (6%), leucopenia, 41 (16.4%), and 9 (3.6%) had thrombocytopenia. Rural areas exhibited greater ALT, TSB, PT, PTT, and blood urea (P-values = 0.006, 0.020, 0.027, 0.010, and 0.003). 17 (6.8%) people experienced hepatic encephalopathy (8.3%), ascites (6.4%), and pleural effusion (3.2%) 233 (96%) had good results, while 3% needed tertiary care. One patient died from the illness (0.4%). Conclusion: Although hepatitis A is a self-limiting disease, it’s have low rate of complications. hepatitis A is one of important health problems but, still affected large numbers of children.

Keywords: Acute, Viral Hepatitis, Children, Epidemiological, Clinical course, Babylon

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Received 22 August 2022, Accepted 13 October 2022, Available online 3 November 2022.

Introduction

Hepatitis A is a vaccine-preventable virus. Hepatitis A is spread by the fecal-oral route by contaminated food or water and/or contact with diseased people [1]. Hepatitis A is a self-limiting, acute liver illness, although its severity increases with age [2]. 50% of children under 6 are asymptomatic, but 75% of children over 6 and adults have signs including jaundice and black urine [3]. Hepatitis A virus is a 27-nm, nonenveloped, positive-polarity RNA virus of the family picornaviridae [4]. It's a frequent source of infectious jaundice in epidemics [5]. Natural host is humans [6]. The virus is stable from low pH to moderate temperatures. Two viral types exist. Naked, non-enveloped HAV virions excreted in faeces by infected people are tiny, 27nm icosahedral protein capsids [7]. All human hepatoviruses have the same antigenic structure and serotype. A second kind of infectious virus [8] is quasi-enveloped and non-lytic. These virions are observed in blood and supernatant fluids of infected cell cultures and consist of RNA-containing capsids encapsulated in membrane vesicles with no virally-encoded proteins on their surface [9]. They are a new discovery that plays a key role in HAV pathogenesis [10]. Hepatitis A is prevalent worldwide, with prevalence rates of 40%, 20.1%, 29%, 30.2%, 22.3%, 50%, 12.2%, and 10.1% in Iraq, the UAE, Turkey, Saudi Arabia, Iran, Egypt, the US, and the UK, respectively. Globally, there are 1.4 million symptomatic hepatitis A cases [11] and tens of millions of infections [12]. Babylon Health Directorate reported 404, 132, 316, 1068 hepatitis A infection in 2015, 2016, 2017, and 2018. In Low- and Middle-Income Countries, poor hygiene and sanitation constitute the highest risk for HAV infection [13]. All ages may have hepatitis A. People over 50 had a 1.8% case fatality ratio [14]. Vaccines prevent Hepatitis A. Sanitation, housing, personal hygiene, pre- and post-exposure passive immune globulin prophylaxis, or pre- or post-exposure active HAV vaccination [15] prevent HAV infection. Sanitation and personal cleanliness decrease hepatitis A transmission [16]. Handwashing and nail trimming prevent sickness. Wash your hands before meals, after diapering and using the bathroom. Handling faces requires gloves and handwashing. Intramuscular (I.M.) IG is effective for 12-20 weeks at 0.02- 0.06ml/kg. Post-exposure prophylaxis is 80-90% effective within 14 days following injection. IG may prevent hepatitis A symptoms, but not infection [17]. IG is used in immunocompromised, chronic liver disease, or vaccine-intolerant persons for post-exposure hepatitis A prevention [18]. The aim of study is to evaluate epidemiology, clinical course, and laboratory profile of hepatitis A in children.

Method

This study was carried out in Babylon maternity and children teaching hospital for 250 children were admitted as hepatitis A infection, the survey was conducted over a period from 1 March 2018 to 28 December 2018. Children below 14 years old with +ve HAV IgM and clinical features.

Exclusion criteria

- ✓ Age more than 14 years old
- ✓ Hepatitis B, and C
- ✓ Hepatitis due to metabolic disorders
- ✓ Drugs induced hepatitis
- ✓ Hepatitis due to other infection like CMV and EBV
- ✓ Parents refused to do laboratory investigations outside hospital

This is a cross-sectional study which was designed to determine the clinical course, laboratory profile, complications, and frequency of hepatitis A in children less than 14 years' age.

A focused history was taken from the child or/and his/her care provider including the place of residence, source of drinking water, method of fecal sewage, nursery attendance, taking food outside home, fever, GIT symptoms and history of contact with hepatitis patient. physical examination was done. with investigations were send as HAV IgM, TSB, blood urea nitrogen, liver enzymes (AST, ALT), bleeding profile and CBC. investigations as TSB, blood urea nitrogen, LFT (AST, ALT) were done by using spectrophotometers device (CECIL company). bleeding profile and CBC were done by RUBY device (Abbott Diagnostic 2012). Hepatitis virology test were done by minividas device (BIOMERIEUX company). The abdominal US was performed by Voluson D62168 device by 3 different sonarists. SPSS® Software (version 23.0 for Linux®) was used to perform statistical analysis for this study. Qualitative data are presented as numbers and percentages, while continuous numerical data are presented as mean ± standard deviation. Comparisons of study groups were performed using chi-square test for categorical data, and Student's t-test for continuous data. Correlations were assessed using Pearson's product-moment correlation coefficient. P value of < 0.05 was considered statistically significant.

Results

This study included a total of (250) cases diagnosed with hepatitis A. Age of the patients ranged from (1) year up to (14) years, with a mean age of (7.3 ± 2.6) years and a median of (4.9) years. Figure (2) illustrates the age group distribution of the study population. More than half of the cases are between (5-10) years old.

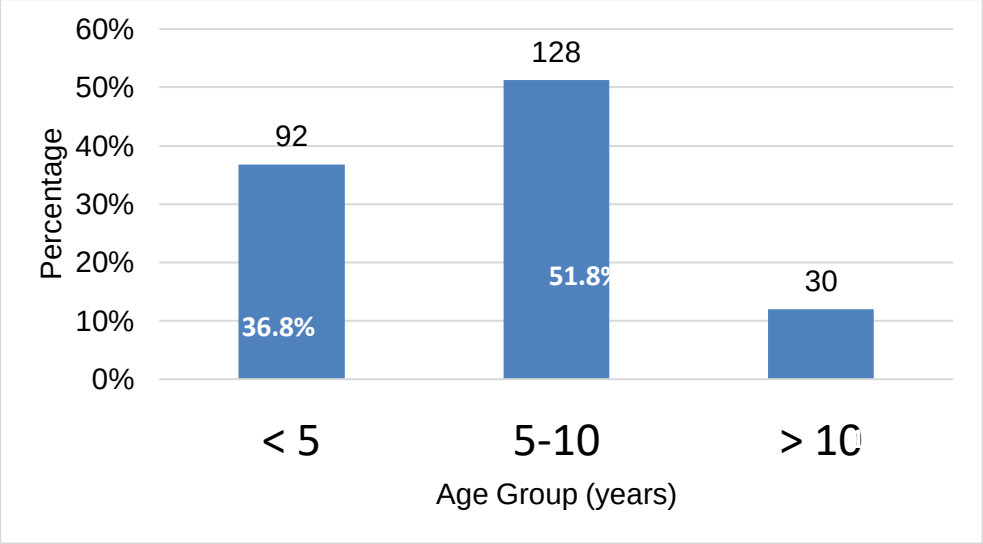

Figure 2.

Age group distribution of the study population

Demographic characteristics of the cases are detailed in Table (1). Females comprised 128 (51.20%) of the study population while males comprised the remaining 122 (48.80%). Rural residents' proportion was s higher than urban residents' proportion (62.4% vs. 37.6%).

Table 1.

Demographic characteristics of the cases

Demographic characteristics		Frequency	Percent
Gender	Male	122	48.80%
	Female	128	51.20%
Total		250	100%
Residence	Urban	94	37.6%
	Rural	156	62.4%
Total		250	100%

The sanitary conditions in the houses of the cases were detailed in Table (2). Drinking water was obtained from filtered R.O. water (outside home) in (56.40%) of the cases, while (46.60%) used filtered R.O. water (homemade). More than half of the cases (59.6%) had domestically implemented sewage system, while (40.4%) had proper installation of sewage system. Almost three quarters (72.80%) of patients were reported to consume food recently from sources outside the home-cooked meals, but only (14.40%) of patients attended daycare nurseries. On history inquire about contact with infected persons there were (63.2%) of patient have history contact with infected person.

Table 2.
Sanitary conditions of the houses of cases

	Characteristics	Frequency	Percent
Water Source	Filtered R.O. water (homemade)	109	46.60%
	Filtered R.O. water (outside home)	141	56.40%
Total		250	100%
Sewage System	Proper Installation	101	40.4%
	Domestically implemented	149	59.6%
Total		250	100%
Food Source	Home made	68	27.20%
	From Outside	182	72.80%
Total		250	100%
History of contact		92	36.8%

Clinical manifestations of the patients are described in detail in Table (3). The commonest symptoms was jaundice which was present in (95.2%) of cases, followed by fever in (72.4%) of them, vomiting (72%) in them and abdominal pain in (67.41%) of the cases. On clinical examination, a total of (168) patients were found to have hepatomegaly, while only (34) had splenomegaly (Table 3).

Table 3.

Clinical characteristics of cases

Clinical Characteristics	Frequency (%)	
	Present	**Not present**
Jaundice	238 (95.2%)	12 (4.8%)
Fever	181 (72.40%)	69 (27.60%)
Vomiting	180 (72.00%)	70 (28.00%)
Abdominal Pain	171 (68.40%)	79 (31.60%)
Hepatomegaly	168 (67.20%)	82 (32.80%)
Anorexia	159 (63.60%)	91 (36.40%)
Splenomegaly	34 (13.60%)	216 (86.40%)
Diarrhea	18 (7.20%)	232 (92.80%)
Disturbed level of consciousness (Hepatic encephalopathy)	8 (3.20%)	242 (96.8%)

Various laboratory investigations were performed for the cases enrolled in the study table (4), including total serum bilirubin (TSB), AST, ALT, Prothrombin time (PT), Partial Thromboplastin Time (PTT), INR and blood urea nitrogen level.

Table 4.

Laboratory investigations express by mean

Test	Overall Value (Mean ± SD)
TSB	7.30 ± 4.14
AST	608.41 ± 476.05
ALT	628.91 ± 537.08
PT	17.29 ± 5.24
PTT	33.34 ± 10.19
INR	1.41 ± 0.33
Urea	4.86 ± 1.55
Platelets	176.52 ± 52.87
WBC	4.900 ±2.364
Hb	10.01±0.97

Hematological finding of study details in table (5). anemia was seen in (28.8%), leucopenia in (6%), leukocytosis in (16.4%) and thrombocytopenia in (3.6 %). And their mean was Hb (10.01±0.97), WBC (4.900 ±2.364) and platelets (176.52 ± 52.87).

Table 5.

Hematological Finding of study cases

Hematological complications	Numbers	Percent%
Anemia	72	28.8%
Leucopenia	15	6%
Leukocytosis	41	16.4%
Thrombocytopenia	9	3.6%
Normal	113	45.2%
Total	250	100%

Ultrasound findings was show ascites in 51 patients (6 have moderate ascites and 45 have mild ascites), Gallbladder wall swelling 70 and Pleural Effusion in 3 of them while the remaining have no finding. Ultrasound findings among study cases are summarized in Table (6).

Table 6.

Ultrasound findings of study cases

Ultrasound Findings	Frequency	Percent
Ascites	51	20.4%
Pleural Effusion	3	1.20%
Gallbladder wall swelling	70	28.00%
Normal	11	4.4%
Total	250	100%

Various laboratory investigations were performed for the cases enrolled in the study, including total serum bilirubin (TSB), AST, ALT, Prothrombin time (PT), Partial Thromboplastin Time (PTT), INR and blood urea nitrogen level. The values of those tests were compared between patients of urban residence and patients with rural residence. It was noticeable that the ALT, TSB, PT, PTT, and blood urea level were significantly higher in rural areas compared with urban areas (P-value = 0.006, 0.020, 0.027, 0.010, and 0.003, respectively). Table (7) summarizes the findings.

Table 7.

Comparable Laboratory finding of the cases by residence

Test	Residence (Mean ± SD)		P-value
	Urban	Rural	
TSB	6.68 ± 3.74	7.90 ± 4.42	**0.020***
AST	663.77 ± 495.43	555.64 ± 452.46	0.073
ALT	538.53 ± 530.87	723.73 ± 529.24	**0.006***
PT	16.54 ± 2.57	18.00 ± 6.82	**0.027***
PTT	31.65 ± 4.89	34.95 ± 13.25	**0.010***
INR	1.39 ± 0.31	1.43 ± 0.35	0.415
Urea	4.56 ± 1.41	5.14 ± 1.63	**0.003***

* Significant P-value (< 0.05)

About (6.8%) of the patients had complications, which were mainly hepatic encephalopathy (3.2%) and pleural effusion (1.2%). More than (96%) of patients had good outcome, while about (3%) of them needed referral to a tertiary center. One patient died as a result of the disease, giving a case fatality rate of (0.4%), Table (8). It is worth noting that the single case with mortality had water source from filtered water and had a domestically implemented sewage and used to consume food from sources outside of the house.

Table 8.

Complications and outcome of the cases

	Variable	Frequency	Percent
Complications	Hepatic encephalopathy	8	3.2%
	Pleural effusion	3	1.2%
	No complications	239	95.6%
Total		250	100%
Outcome	Good outcome	241	96.40%
	Referral	8	3.20%
	Death	1	0.40%
Total		250	100%

Discussion

The HAV positivity in different age groups in this study was: 5 years (36.8%), 6-10 years (51.8%), and >10 years (12%). This study suggests more than half of cases age between 5 and 10 years old, which agrees with studies by Kamath SR et al [19]. Other studies suggest majority of cases were aged above 10 years [20].

This may be because sickness symptoms worsen with age and children this age have greater contact. In the current research, females predominated, similar to Parekh Z et al [21], but previous investigations revealed boys' preponderance (22). Small sample size may be at blame. Rural participants had a greater HAV prevalence (62.4%) than urban ones (37.6%). Altnkaynak, Sevin, et al [23] found similar results. Poor hygiene and illness ignorance make this feasible. In (56.40%) of the homes, R.O. water was used for drinking, while (46.60%) utilised filtered water. This implies that water pollution is not the major mechanism for hepatitis A transmission, but water storage may play a role. Those using water filters are more concerned with disease prevention and greater cleanliness, which helps prevent transmission.

Over half of the instances (59.6%) had a domestically implemented sewage system, whereas 40.4% had appropriate installation. This fits with a study by Almeida, L. M., et al [24]. According to Yu, Ping, et al. [25], 72.80% of patients recently ate outside the house.

asymptomatic food handlers, inadequate hand washing, and contaminated food may be to blame. This research focuses on fever (72.4%), vomiting (72%), hepatomegaly (67.2%), diarrhoea (7.2), and splenomegaly (13.6%). Jaundice (94%) and fever (82%) were the most prevalent presenting ailments. Behera AK et al [26] found jaundice as the most prevalent symptom in their research. Non-icterus hepatitis was detected in 4.8%, which matches Behera AK et al's (6%) report. Anemia, leucopenia, leukocytosis, and thrombocytopenia were reported in 28.8% of patients, similar to prior studies. autoimmune viral hepatitis or illness or dietary issues may cause it. Ascites (20.4%), pleural effusion (1.2%), hepatitis characteristics (46%) and gallbladder wall thickness (28%). (21.74%) of hepatitis A children had ascites, according to Kamath et al. Yacha et al. [27] found that 67% of ascitic acute viral hepatitis was related to HAV infection and concluded that it affects children.

TSB, ALT, and PT were greater in rural patients. This is likely owing to rural populations delaying medical help and lacking illness knowledge. In 6.8% of our sample, hepatic encephalopathy was the predominant problem; Kamath et al. (19) reported 5.8% of hepatitis A children had difficulties. 3.2% of our sample had hepatic encephalopathy, similar to Mohanty et al. [28]. (3.7%) In a study from Korea, hepatic encephalopathy due to hepatitis A was seen in 6.2% of 568 patients. This high percent of encephalopathy in our study in compares with global percent (0.5%) possible due to our hospital is pooling hospital of province, many cases of hepatitis treated as out-patient at private clinic, most cases of hepatitis go undiagnosed and small sample of our study. 2.4% had ascites and 1.2% had pleural effusion, according to Mohanty et al. [28] pleural effusion is an uncommon consequence of acute hepatitis. Tesovic et al. [29] claim pleural effusion is a benign early consequence of acute hepatitis A that resolves spontaneously. In our investigation, one patient died, giving a mortality rate of (0.4%).

Conclusion

Although hepatitis A is a self-limiting disease, it has many complications. hepatitis A is one of important health problems and affected large numbers of children. Physicians all over this region should be aware of these features of hepatitis A because in small children it’s not so evident always. All cases should be followed up till complete recovery and only a strong index of suspicion will enable us to recognize the complications.

References

1. de Jong G. Guidelines for the Control of Hepatitis A in South Africa. South Africa: National Institute of Communicable Disease; 2007.
2. Franco E, MeleleoC, SerinoL, SorbaraD, ZarattiL. Hepatitis A: epidemiology and prevention in developing countries. WorldJHepatol.2012;4:68–73.
3. GossnerCM, SeveriE, DanieissonN, HutinY, CoulombierD. Changing hepatitis A epidemiology in the European Union: new challenges and opportunities. EuroSurveill.2015;20.pii:21101.
4. Joon A, Rao P, Shenoy S M, Baliga S. Prevalence of hepatitis A virus (HAV) and hepatitis E virus (HEV) in the patients presenting with acute viral hepatitis. Indian J Med Microbiol. 2015;33:102-5.
5. Groover AA, Huang NA. Treatment of disseminated TB with drug induced hepatitis/case study. American Journal of BioMedicine 2021;10(1):1-5.
6. Cuthbert JA. Hepatitis A: old and new. *Clin Microbiol Rev.* 2001;14(1):38-58.
7. Wang X, Ren J, Gao Q, et al. Hepatitis A virus and the origins of picornaviruses. *Nature.* 2015;517(7532):85-88.
8. Feng Z, Hensley L, McKnight KL, et al. A pathogenic picornavirus acquires an envelope by hijacking cellular membranes. *Nature.* 2013;496(7445):367-371.
9. Jacob DH, Zeng IU, Tsung Y, et al. Tpl2 exaggerated ischemic hepatic injury through induction of apoptosis: Downstream of caspase-9. American Journal of BioMedicine 2015;3(1):39-56.
10. Feng Z, Lemon SM. Hepatitis A virus. In: Ehrenfeld E, Domingo E, Roos RP, eds. *TheU Picornaviruses.* Washington, D.C.: ASM Press; 2010:383-396.
11. World Health Organization. Hepatitis A. 2015.
12. Wasley A, Flore A, Bell BP. Hepatitis A in the era of vaccination. Epidemiol Rev. 2006; 28:101–111.
13. Heathcote J, Elewaut A, Fedail S, Gangl A, Hamid S, Shah M, et al. Managment of acute viral hepatitis. 2003.
14. Wick JY. Hepatitis in the elderly: still a scourge. Consult Pharm. 2012; 27:472–481.
15. Manor Y, Lewis M, Ram D, et al. Evidence for Hepatitis A virus endemic circulation in Israel despite universal toddlers' vaccination since 1999 and low clinical incidence in all age groups. *J Infect Dis.* 2016.
16. 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.

17. Liu JP, Nikolova D, Fei Y. Immunoglobulins for preventing hepatitis A. *Cochrane Database Syst Rev.* 2009(2):CD004181.
18. Nelson NP, Link-Gelles R, Hofmeister MG, et al. Update: Recommendations of the Advisory Committee on Immunization Practices for use of hepatitis A vaccine for postexposure prophylaxis and for preexposure prophylaxis for international travel. *MMWR Morb Mortal Wkly Rep* 2018;67(43):1216–1220.
19. Kamath SR, Sathiyasekaran M, Raja TE, Sudha L. Profile of viral hepatitis A in Chennai. Indian Pediatr. 2009;46(7):642-3.
20. Nandi B, Hadimani P, Arunachalam R, Ganjoo R. Spectrum of acute viral hepatitis in Southern India. Med J Armed Forces India. 2009;65(1):7-9.
21. 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.
22. Poddar U, Thapa BR, Prasad A, Singh K. Changing spectrum of sporadic acute viral hepatitis in Indian children. J Trop Pediatr. 2002;48(4):210-3.
23. Altinkaynak S, Selimoglu MA, Ertekin V, Kilicaslan B. Epidemiological factors affecting hepatitis A seroprevalence in childhood in a developing country. Eurasian J Med 2008; 40: 25-28.
24. Almeida LM, Werneck GL, Cairncross S, Coeli CM, Costa MC, Coletty PE. The epidemiology of hepatitis A in Rio de Janeiro: environmental and domestic risk factors. Epidemiol Infect 2001; 127:327-33.
25. Yu, P., Huang, L., Li, H., Liu, M., Zong, J., Li, C., & Chen, F. (2015). Epidemiological investigation of an outbreak of hepatitis A in rural China. *International Journal of Infectious Diseases*, *33*, 191-195.
26. Behera MR, Patnaik L. Clinico-biochemical profile and etiology of acute viral hepatitis in hospitalized children: a study from Eastern India. Indian J Child Health. 2016;3(4):317-20.
27. Yachha SK, Goel A, Khanna V, Poddar U, Srivatsava A, Singh U. Ascitic form of sporadic acute viral hepatitis in children: A distinct entity for recognition. J Pediatr Gastroenterol Nutr. 2010;50:184–7.
28. MOHANTY, Niranjan; SHUBHANKAR, Mishra; SAMBEDANA, Panigrahi. Fulminant Hepatitis A in Children, its Incidence, Presentation, Complications and Outcome: A Study from Eastern India. *Journal of Nepal Paediatric Society*, 2017, 37.3: 226-231.
29. Tesovic G, Vukelic D, Vukovic B, Benic B, Dragomir B. Pleural effusion associated with acute Hepatitis A infection.Pediatr Infect Dis J 2000;19:585-6.