Section: Pediatrics

Original Research Article

STUDY ON CLINCO – EPIDEMIOLOGY AND LABORATORY PROFILE OF VIRAL HEPATITIS A IN CHILDREN ADMITTED TO TERTIARY CARE CENTRE MEDICAL HOSPITAL IN SOUTH INDIA

S.Mekalai[1], Ponkeerthi, S.Aravind[3], C. Suganya[4]

[1]Professor, Department of Pediatrics, Institute of Child Health, Egmore, Tamilnadu, India.
[2]Postgraduate, Department of Pediatrics, Institute of Child Health, Egmore, Tamilnadu, India.
[3]Assistant Professor, Department of Pediatrics, Institute of Child Health, Egmore, Tamilnadu, India.
[4]Assistant Professor, Department of Pediatrics, Institute of Child Health, Egmore, Tamilnadu, India.

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Corresponding Author:
Dr. C. Suganya
Email: ajayselvasugi@gmail.com

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Abstract

Background: Hepatitis A is a non-enveloped, positive-stranded RNA virus that causes global economic loss and morbidity. It is a vital cause of sporadic acute hepatitis among children, with 90% of adults and almost most preschool children having acquired immunity. The study aims to evaluate the hepatic and extrahepatic complications in children with hepatitis A infection. **Material & Methods:** This observational study was conducted in the Institute of Child Health and Hospital for Children, Madras Medical College, for 12 months. Totally 125 children who were admitted as inpatients with features of acute hepatitis satisfying the inclusion criteria were selected. Liver function test, Prothrombin time, activated partial thromboplastin time, blood sugar, blood urea, creatinine, and other viral hepatitis markers were done. **Results:** Most of the study participants were males, 67 (53.6%). The majority of the study participants belonged to the age group of 6 to 10 years 76 (60.8%). All the study participants had fever and vomiting. Ultrasound showed hepatomegaly among all the study participants, followed by the majority having Gall bladder wall edema 62 (49.6%). HAV seropositivity is highest among lower middle and upper lower-class socioeconomic status. It is also high among those using tap water as a water source and open fields as a mode of excreta disposal. None of the study participants had complications at 2nd and 4th week of follow-up. **Conclusions:** It is recommended to keep a good watch with a series of laboratory investigations till the complete recovery for early identification and treatment of complications.

INTRODUCTION

Globally, hepatitis A virus infection accounts for nearly 14 lakh cases annually.[1] Hepatitis is the inflammation in hepatocytes, which can occur due to viral infections such as hepatitis A, B, C, D and E. Meanwhile, for all liver diseases with jaundice development, proper diagnosis can be made only after testing for the virus-specific antibodies in the patient's serum.[2] Hepatitis A belongs to the picornavirus family. It is a non-enveloped, positive-stranded RNA virus, leading to global economic loss and morbidity.[3]

The spread of Hepatitis A is by contaminated excreta. The course of this enteric infection has four phases clinically. The incubation period, pre-icteric, icteric and convalescent phase.[4] Literature has shown that the epidemiology of hepatitis has significantly shifted in the past few years.[5] The past few decades have shown an increased prevalence of hepatitis A among children and clinically manifests as non-specific gastrointestinal symptoms and persistent jaundice for more than 12 weeks.[6] Studies have shown an increased prevalence of anti-HAV antibodies among children from lower socioeconomic status.

Officially, the first epidemic of viral hepatitis occurred in Delhi in 1955.[7] Since then, many outbreaks have been reported in various parts of India. It remains a vital cause of sporadic acute hepatitis among children.[8] A study done in 2000 in India showed that 90% of adults and almost most preschool children had acquired immunity towards hepatitis A infection.[9] Life-threatening complication due to hepatitis A is low. The mortality rate is approximately 0.2% among the icteric patients. Rarely, extensive necrosis in the liver can happen in the initial 6 to 8 weeks of the

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disease. Severe pain in the abdomen, jaundice, vomiting, and the progress into hepatic encephalopathy with seizures and coma are the clinical manifestation of fulminant hepatitis, resulting in mortality among 70 to 90% of the hepatitis patients.[10] Hepatitis A virus is the primary causative agent for the development of Fulminant hepatic failure among children. The mortality among patients with hepatitis B and C increases when they are superinfected with the hepatitis A virus.[10]

HAV hepatitis is diagnosed by estimating the anti-HAV IgM antibodies present in serum. When the clinical symptoms are apparent, anti-HAV remains positive for nearly 4 to 6 months after the acute infection. Neutralising anti-HAV IgG antibodies can be detected within eight weeks from the onset of symptoms, and it converses long-term protection.[12] Hence, the study aims to evaluate the hepatic and extrahepatic complications in children with hepatitis A infection.

MATERIALS AND METHODS

This observational study was conducted in the Institute of Child Health and Hospital for Children, Madras Medical College, for 12 months. Totally 125 children who were admitted as inpatients with features of acute hepatitis satisfying the inclusion criteria were selected.

Inclusion criteria: Age 1 to 12 years, IgM Anti-HAV- Reactive, duration of jaundice less than three months, and icteric and anicteric hepatitis were included.

Exclusion criteria: Jaundice persisting for over three months, Hemolytic Jaundice, surgical causes of jaundice, IgM HAV- Nonreactive, and chronic liver disease were excluded.

The study was undertaken after taking written informed consent from the guardian/ parents. Data was collected by oral questionnaire regarding relevant history from parents or patient's family members. Required investigations were sent to the laboratories and recorded in a pre-structured case study proforma.

Clinical history includes fever, abdomen pain, nausea, vomiting, bleeding manifestation, urine discolouration, dark urine, abdomen distension, altered mental status, Clinical signs like hepatomegaly, splenomegaly, free fluid abdomen were noted down. Extrahepatic manifestations like evanescent rash, arthralgia, hematological manifestation like pancytopenia, AIHA , neurologia cal complications, Cholecystitis, pancreatitis, Vasculitis were also monitored and noted down. Diagnosis was confirmed by specific Viral serology (Ig/m anti-HAV positive, Method Enzyme-Linked Immunosorbent Assay). Liver function tests, Prothrombin time, activated partial thromboplastin time, blood sugar, blood urea, creatinine, and other viral hepatitis markers were done. Routine investigations such as CBC with Hb%, TC, DC and platelet count were also done. Ultrasound abdomen was advised, with particular emphasis on the hepatobiliary system.

All the children were monitored for complications during the hospital stay. They were discharged when clinically stable and afebrile for >24 hours with a modest increase in appetite & general well-being. Follow-up was done at 2 and 4 weeks of discharge. All complications during this follow-up were recorded.

The collected data were checked for completeness before entering the Microsoft Excel spreadsheet. The validation of the data was checked at regular intervals. Data analysis was performed to treat the approach using Statistical Package for Social Sciences (SPSS IBM) 21. The quantitative data were expressed in frequency and percentage.

RESULTS

Most of the study participants were males, 67 (53.6%). The majority of the study participants belonged to the age group of 6 to 10 years 76 (60.8%). All the study participants had fever and vomiting. Ultrasound showed hepatomegaly among all the study participants, followed by the majority having Gall bladder wall edema 62 (49.6%) [Table 1].

Table 1: Patient characteristics in the study

		Frequency	Percentage
Gender	Male	67	53.6
	Female	58	46.4
Age (years)	1- 5	10	8
	6 -10	76	60.8
	11-12	39	31.2
Clinical features	Fever	125	100
	Vomiting	125	100
	Abdominal pain	93	74.4
	Yellow coloured urine	63	50.4
	Loss of appetite	56	44.8
USG findings	Hepatomegaly	125	100
	Gall bladder wall edema	62	49.6
	Splenomegaly	61	48.8
	Free fluid	52	41.6

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Seroprevalence was more in the lower middle class and upper lower class socioeconomic status, 20% and 66%. Most of the education status of parents completed primary school (58%), middle school (40.5%), and illiterate (40%).
HAV seropositivity is high among people who are using tap water as a water source (50%) and also increased among the people who are using open fields as a mode of excreta disposal (65%) [Table 2].

Table 2: The overall impact of all factors on HAV seroprevalence

Socioeconomic status	Upper	5
	Upper middle	20
	Lower middle	66
	Upper lower	55
	Lower	4
Education status of parents	Professional;	1
	Graduate	1.5
	Intermediate	2
	High school	20
	Middle	40.5
	Primary school	58
	Illiterate	40
Source of drinking water	Tap water	50
	River	10
	well water	10
	Hand pump	30
Excreta disposal	Open field	65
	Public toilet	20
	Private toilet outside house	10
	Private toilet within the house	5

he means total bilirubin was 11.28 ± 5.56, ranging from 17.8 to 20. The mean Direct bilirubin was 0.48 ± 0.21, ranging from 0.7 and 0.8. The mean SGOT was 252.63 ± 90.81, ranging from 292 and 396, and the mean SGPT was 331.98 ± 100.9, ranging from 151 to 498.
The mean ALP in the present study was 96.34 ± 26.73, and the mean prothrombin time was 12.89 ± 0.5. The mean APTT was 25.04 ± 0.82, and the mean INR was 1.05 ± 0.02 [Table 3].

Table 3: Mean parameters of the study

	Mean	**SD**
Heart rate	104.79	3.13
Respiratory rate	34.78	3.08
CRT	1.49	0.30
Total bilirubin	11.28	5.56
Direct bilirubin	0.48	0.211
SGOT	252.63	90.81
SGPT	331.98	100.96
ALP	96.34	26.73
Prothrombin time	12.89	0.50
APTT	25.04	0.82
INR	1.05	0.02

None of the study participants had complications at 2nd and 4th week of follow-up. No extrahepatic manifestations were seen in any of the study group during hospital stay or during follow up.

DISCUSSION

Acute hepatitis in children can happen due to many causes, including non-infectious and infectious. In India, acute hepatitis remains a major contributor to mortality and morbidity among children and adults. The cause of acute hepatitis among the paediatric population varies with a wide range of demographic determinants. Due to the varied differential diagnosis, Hepatitis A cannot be detected merely based on clinical grounds and cannot be recognised from other kinds of hepatitis without laboratory investigation. Suspicion of hepatitis A is raised when there is a history of consumption of raw vegetables or fruit, uncooked/ undercooked foods and drinking water that is not sanitised or in contact with HAV-infected patients.
The clinical manifestation of hepatitis A creates it hard to distinguish from other types of acute viral hepatitis as the symptoms have similarities with many febrile and gastrointestinal diseases. The differential diagnosis of hepatitis A includes other viral infections, toxins, drugs, parasitic/ bacterial infections, and autoimmune hepatitis. Diagnosis is usually made by measuring serum immunoglobulin M (IgM) anti-HAV antibodies. Anti-HAV antibody detection helps it to distinguish from types of hepatitis. The specificity and sensitivity of Anti HAV IgM is above 95 percent. The present study has shown that most participants belonged to the age

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group of 6 to 10 years. Similarly, a study done by Dhak S et al.[13] showed that the mean age of the study participants was 6.65 ± 2.46 years, and also this corresponds with the study done by Kumar KJ et al.[14]

The present study has shown that most study participants were males, which corresponds with the study done by Blechova Z et al.[15], who showed that most participants with hepatitis among children were males. The present study has demonstrated that seroprevalence was more seen in the lower middle-class to upper-middle-class socioeconomic status, and this corresponds with a study done by Mitra et al.[16]

The present study has shown that all participants had fever and vomiting, followed by abdominal pain and yellow-coloured urine. Similarly, in a study by Dhak et al.[13], all participants with hepatitis A had fever. The manifestation spectrum in hepatitis A infection range from asymptomatic to fulminant hepatitis. The symptoms of hepatitis A are highly dependent upon the age. Usually, it's asymptomatic in 70% of children under six years. However, 70% of adults present with jaundice and increased aminotransferases. After an incubation period of 2 to 7 weeks, typical manifestation develops in hepatitis A in the form of fever, nausea/ vomiting, malaise, abdominal pain, jaundice, and dark-coloured urine.

The role of other variables, such as parents' educational status, drinking water source, and sanitary conditions with HAV seropositivity, was also analysed. The seroprevalence of HAV was high among the lower middle-class socioeconomic class. HAV seropositivity is high among people using tap water as a water source and those using open fields as a mode of excreta disposal. The present study has shown that ultrasound showed hepatomegaly among all the study participants, followed by the majority having Gall bladder wall edema. Similarly, in a study by Dhak et al.[13], 100% of the study participants had hepatomegaly. The clinical findings can be supplemented by ultrasound to give a definite diagnosis of hepatitis A.

The present study showed that the mean total bilirubin was 11.28 ± 5.56, ranging from 17.8 to 20. The mean Direct bilirubin was 0.48 ± 0.21, ranging from 0.7 and 0.8. Intrahepatic viral inflammation disrupts conjugated bilirubin transport, leading to jaundice. In a study done by Blechova Z et al.[15], it was shown that the mean total bilirubin and conjugated bilirubin among the symptomatic icteric patients were 69.94 and 43.63, respectively. Similarly, a study done by Dhak et al.[13] showed that the mean bilirubin level was 6.86.

The present study stated that the mean SGOT was 252.63 ± 90.81, ranging from 292 and 396, and the mean SGPT was 331.98 ± 100.9, ranging from 151 to 498. In a study done by Dhak et al.13, the mean AST was 463.19. A study by Kumar KJ et al.[14] showed a more than 5-fold increase in Aspartate transaminase and Alanine transaminase among 79.5% and 70.5%, respectively. Literature has shown a substantial increase in the SGOT (serum glutamic oxalacetic) and SGPT (serum glutamic pyruvic transaminase) in acute viral hepatitis. Such an increase is normally 2000 to 4000 percent.

The distinctive features of the transaminases in hepatitis are an increase in both SGOT and SGPT activities, a relative increase in SGPT to SGOT, and a decrease in the SGOT/SGPT ratio to <1. The mean ALP in the present study was 96.34 ± 26.73, and the mean prothrombin time was 12.89 ± 0.5. It also stated that the mean APTT was 25.04 ± 0.82. The mean INR was 1.05 ± 0.02. Liver disorders are usually related to haemostatic function impairment because of the reduced or abnormal synthesis of the clotting factors. A study by Kumar KJ et al.[14] showed that an INR of more than 1.5 was seen among 15.4% of the study participants. 12.8% had abnormal APTT.

Limitations

A multicentric study with a larger sample size can yield a generalised result for giving proper recommendations.

CONCLUSION

Hepatitis remains a global public health problem, notably in developing countries like India. Acute viral hepatitis is estimated to account for nearly 1.6 percent of hospital admissions. Although Hepatitis A is a self-limiting infection, it is not uncommon for extrahepatic manifestation and development complications like fulminant hepatitis, leading to mortality. It is recommended to keep a good watch with a series of laboratory investigations till the complete recovery for early identification and treatment of complications.

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