

Prevalence of hepatitis A in southern part of Delhi, India

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Abstract

Background: Hepatitis A virus (HAV) infection is one of the important causes of hepatitis in developing countries. It is transmitted through orofecal route. They pose major health problems.

Objective: To determine the prevalence of HAV in patients presenting with acute viral hepatitis.

Materials and Methods: Serum samples of the patients presenting with fever, nausea, vomiting, and jaundice were collected and analyzed by using commercially available mini VIDAS (Biomerieux, France) to detect immunoglobulin M (IgM) against HAV. A one-year retrospective study was conducted in the Department of Microbiology, Hamdard Institute of Medical Sciences and Research, New Delhi, India.

Result: Of the 89 serum samples subjected to mini VIDAS for detection of HAV IgM in patients, 30 (33.7%) were found to be positive. The prevalence was found to be more in male patients than in female patients, that is, 21 (70%) and 9 (30%), respectively, and was predominantly seen in the 5–15 years of the school-going age group. Peak cases were found in the monsoon season.

Conclusion: Regular monitoring of clinical, serological, and molecular characteristics would help in understanding the epidemiology of HAV and in planning the intervention studies. Prevalence was lower in the age group of 0–5 years, which shows epidemiological shift and improvement in hygiene and in socioeconomic conditions.

KEY WORDS: HAV, serology, anti-HAV IgM

Introduction

Hepatitis A (formerly known to be infectious hepatitis) is an acute infectious disease of the liver caused by Hepatitis A virus (HAV).^[1] It is a nonenveloped 27 nm ribonucleic acid (RNA) virus, which is resistant to heat, acid, and ether. It belongs to family Picornaviridae and genus *Hepatovirus*.^[2] It has one serotype and six genotypes. Genotypes I–III are involved in human infections.^[3] HAV is transmitted through feco-oral route either by direct contact or by consumption of contaminated water/food.

Approximately 1.5 million cases are reported every year throughout the world. Acute hepatitis A (AHA) is supposed to be endemic in developing countries because of low socioeconomic status, increased density of houses, and untreated water consumption. Frequent infection leads to development of acquired immunity in nearly 90% of children below 10 years of age.^[4]

With the development of effective vaccine against HAV in the 1990s and improvement in socioeconomic conditions, epidemiology of hepatitis A has changed. The age group more affected has shifted from first decade to second and third decade severity of disease, which was nearly asymptomatic or mild in children become symptomatic in adults.^[5–8]

Antibodies to HAV (anti-HAV immunoglobulin M [IgM]) can be detected in serum during acute illness whereas during convalescence immunoglobulin G (IgG) becomes predominant.

We carried out this study to find out the trend of infection or epidemiological changes in our area and present our findings of serological studies on the samples referred to our laboratory for HAV diagnosis.

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Materials and Methods

This study was conducted in Microbiology Department, Hamdard Institute of Medical Sciences and Research, South Delhi, India, from January 2014 to December 2014 for a period of 1 year. It is a retrospective study. We included patients of both sex from all age groups. Patients or their parents who came themselves to submit the sample were informed verbally about the study and they were inquired about education of their mother, type of drinking water, and toilets being used.

On the basis of history of fever, nausea, vomiting, and jaundice, blood samples of the OPD and IPD patients were collected as per the laboratory protocols. Serum was separated and tests were run on mini VIDAS (Biomérieux, France) for the detection of anti-HAV IgM. mini VIDAS is based on enzyme-linked fluorescent assay technology and claims sensitivity and specificity of 98.8% and 99.44%, respectively. All the kits were supplied by Biomérieux and used according to supplier's instruction manual. Positive and negative controls were provided along with the kit. Test value of ≥ 0.5 was considered positive and lesser than < 0.4 was considered negative. Test values ≥ 0.4 and < 0.5 were equivocal, which should be repeated. Interpretation of the test was made taking into consideration the patient history and the result of any other test performed.

Result

Of the 89 serum samples, 59 (66.2%) were found negative in the assay and 30 (33.7%) were found to be positive. Test value ≥ 0.5 for anti-HAV IgM. Among which 21 (70%) male patients were found positive as compared with 9 female patients (30%). Of the 30 cases, 18 (60%) mothers were found educated, 13 (43.3%) use RO water, and none used boiled water [Tables 1–4].

Discussion

In India, we do not have much data about HAV, but some recent reports have suggested a change in the scenario in the epidemiology of HAV.^[9,10] So we collected data of AHA from our hospital and compared them with other studies; we found significant rise in the seroprevalance in the children of the age group 5–15 years as compared with small children, which shows improvement in the care of children at home and their socioeconomic status. This age group is usually the school-going age group, which is exposed to contaminated environment, and who are not mature enough to take care of themselves. HAV multiplies in liver cell hampering its functions and leading to stimulation of immune response resulting in liver inflammation and antibody synthesis of both IgM and IgG type.^[11] Usually AHA remains asymptomatic in children but symptomatic patients present with fever, malaise, weakness, nausea, and vomiting, followed by jaundice. As the symptoms of HAV are very similar to other hepatitis, it can be

diagnosed with serological testing of antibodies specifically to HAV. IgM can be detected with the appearance of symptoms till 6 months and IgG throughout life. So the presence of either one or both cannot signify acute infection. Therefore, symptoms and liver function test (liver enzymes) should be done along with antibodies to detect acute infection.^[9,11,12] In our study, patients having jaundice and deranged liver function test were only sent for confirmation of HAV infection.

We found 30 (33.7%) patients positive for hepatitis A IgM but in our studies they were all acutely infected patients. In other studies, seroprevalence of 34.02% in children and 28.70% in adults has been documented for IgM.^[13] Refer Table 1 for age and seroprevalence ratio.

Men were found more positive than women [Table 2], this may be due to the greater exposure of men in their professional and social lifestyle and this correlates with other studies.^[14–16] So many studies have noticed epidemiological shift in HAV infection [Table 3]. This may be attributed to various regions. Previously children used to be affected more. In India, areas with different types of people, exposed or susceptible coexist in various areas. Approximately 15 years ago, anti-HAV level in cord blood of neonate was almost 100%, which in turn reflected the prevalence of antibodies in mothers. In new studies, this has come down to 50%–60% suggesting lower infectivities of mothers.^[17–19] Symptoms also vary with age. Approximately 50% of children below 6 years have been reported to be asymptomatic. A total of 75% developed jaundice after 6 years.^[12,20] Only 0.2% of HAV infection can cause acute liver failure, and mortality rises with chronic liver diseases and with age.^[21] So these patients should be immunized for HAV. In our study, we did not find any patient with chronic liver disease or immunization history. Immunization against HAV is costlier than conducting seroprevalence test. So in countries where asymptomatic or mild infection leads to acquired immunity, a prior seroprevalence test should be performed. HAV is endemic in North India and found throughout the year. Still some studies have reported seasonal variation as it peaks in summer and monsoon months of the year.^[13] Similarly, we have reported more cases in the month of August, that is, monsoon season [Table 4].

Because of scarcity of resources and facilities, we could not perform molecular studies although it has been documented that there are three genotypes of HAV, which infect humans, that is, 1A, 1B, and IIIA. In Indian population, genotype IIIA is found to be a predominant one.^[19,22–27] Having the knowledge of genotype is significant as different genotype and serotype causes disease of different severity in the population. Some can cause sporadic cases whereas some can cause outbreaks.^[3,7,8,19,22,23,28–30] This may help in taking control measures in the society to prevent outbreaks in that particular area. Although we have involved many factors such as socioeconomic conditions, cleanliness, education, access to clean drinking water except the molecular tool to understand the epidemiology better.

Table 1: Prevalence of HAV antibodies in other studies

Overall incidence of HAV reported in studies till now				
State/city	Adults	Children	Year	Reference no.
New Delhi	14%	67%	1984	32
Chandigarh	–	64.5%	2002	33
Chandigarh	17.5%	–	2007	34
New Delhi	–	8.1%	2010	31
Lucknow	26.6%	27.2%	2012	13

HAV, hepatitis A virus.

Table 2: Monthly sexwise distribution

Month	Sample		Females		Males	
	Total	Positive	Total	Positive	Total	Positive
Jan	12	2	7	0	5	2
Feb	5	0	2	0	3	1
Mar	3	2	1	0	2	0
Apr	5	3	1	1	4	1
May	10	2	5	1	5	2
Jun	6	2	4	1	2	1
Jul	3	6	2	1	1	1
Aug	6	2	4	4	2	2
Sep	13	3	5	1	8	1
Oct	18	3	6	0	12	3
Nov	4	4	1	0	3	3
Dec	4	0	0	0	4	4
	89		38	09	51	21

Table 3: Age- and sexwise prevalence of HAV IgM antibody in India

Age (years)	Total cases	Positive titer for HAV IgM antibody			
		Samples in females	Positive in females	Samples in males	Positive in males
0–5	12	9	(6)	3	(3)
6–15	24	16	(11)	8	(6)
16–40	46	22	(4)	24	(0)
>40	07	4	(0)	3	(0)

HAV, hepatitis A virus; IgM, immunoglobulin M.

Table 4: Monthwise/seasonal distribution of HAV cases

Month	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec
Total sample	12	5	3	5	10	6	3	6	13	18	4	4
Positive	2	1	0	2	3	2	2	6	2	3	3	4

HAV, hepatitis A virus.

Conclusion

This study investigated that infections have been found in school-going children of <5 years, which shows improvement in the education of mothers' hygiene and socioeconomic status. Hepatitis A, because of epidemiological shift, has now been

diagnosed more in teenagers and adults with more severe symptoms that are similar to other viral hepatitis, so the diagnosis must be confirmed by serological testing for the detection of IgM. In an infection, genotype, serotype, and seroprevalence of the nearby area should be tracked immediately to prevent outbreaks. More studies on seroepidemiology should be carried

out to evaluate the appropriate age of HAV vaccination, and also proper guidelines can be formulated for immunization in developing countries.

References

- Ryan KJ, Ray CG. *Sherri's Medical Microbiology*, 4th edn. McGrawHill, 2004. pp. 541–4.
- Francki RIB, Fauquet CM, Knudson DL, Brown F. The classification and nomenclature of viruses. Fifth report of the International Committee on Taxonomy of viruses. *Arch Virol* 1991;2:S320–6.
- Lu L, Ching KZ, de Paula VS, Nakano T, Siegl G, Weitz M, et al. Characterization of the complete genomic sequence of genotype II hepatitis A virus (CF53/Berne isolate). *J Gen Virol* 2004;85 (Pt 10):2943–52.
- Verma YS, Rajput N, Rajput SS. Seroprevalence of hepatitis A virus infection in different age groups of children. *Ann Trop Med Public Health* 2014;7(5):223–6.
- Braunwald E, Fauci AS, Kasper DL, Hauser SL, Longo DL, Jameson JL. *Harrison's Principles of Internal Medicine*, 5th edn. 2001. pp. 1694–710.
- Mathur P, Arora NK. Epidemiological transition of hepatitis A in India: issues for vaccination in developing countries. *Indian J Med Res* 2008;128(6):699–704.
- Arankalle VA, Sarada Devi KL, Lole KS, Shenoy KT, Verma V, Haneephabi M. Molecular characterization of hepatitis A virus from a large outbreak from Kerala, India. *Indian J Med Res* 2006;123(6):760–9.
- Nalbantoglu B, Donma MM, Ozdilek B, Karasu E, Nalbantoglu A. Shifting epidemiology of hepatitis a infection and vaccination status of children aged 6 months-12 years: time for mass vaccination. *Iran J Pediatr* 2013; 23(3):276–80.
- Krugman S. The Gordon Wilson Lecture. The ABC's of viral hepatitis. *Trans Am Clin Climatol Assoc* 1992;103:145–56.
- Lakshmi TM, Vaithilingam A, Franklin A, Reddy EP. The prevalence of serological markers of viruses causing acute hepatitis in South Indian population. *Int J Biol Med Res* 2011;2(4):925–8.
- Koff RS. Hepatitis A. *Lancet* 1998;351(9116):1643–9.
- Wasley A, Fiore A, Bell BP. Hepatitis A in the era of vaccination. *Epidemiol Rev* 2006;28:101–11.
- Jain P, Prakash S, Gupta S, Singh KP, Shrivastava S, Singh DD, et al. Prevalence of hepatitis A virus, hepatitis B virus, hepatitis C virus, hepatitis D virus and hepatitis E virus as causes of acute viral hepatitis in North India: a hospital based study. *Indian J Med Microbiol* 2013;31(3):261–5.
- Joon A, Rao P, Shenoy SM, 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:S102–5.
- Al-Naaimi AS, Turkey AM, Khaleel HA, Jalil RW, Mekhlef OA, Kareem SA, et al. Predicting acute viral hepatitis serum markers (A and E) in patients with suspected acute viral hepatitis attending primary health care centers in Baghdad: a one year cross-sectional study. *Glob J Health Sci* 2012;4(5):172–83.
- Kamal SM, Mahmoud S, Hafez T, El-Fouly R. Viral hepatitis A to E in South Mediterranean countries. *Mediterr J Hematol Infect Dis* 2010;2(2):e2010001.
- Mathur P, Arora NK. Epidemiological transition of hepatitis A in India: issues for vaccination in developing countries. *Indian J Med Res* 2008;128(6):699–704.
- Chadha MS, Lole KS, Bora MH, Arankalle VA. Outbreaks of hepatitis A among children in western India. *Trans R Soc Trop Med Hyg* 2009;103(9):911–6.
- Barzaga BN. Hepatitis A shifting epidemiology in South-East Asia and China. *Vaccine* 2000;18(Suppl 1):S61–4.
- Shapiro CN, Margolis HS. Worldwide epidemiology of hepatitis A virus infection. *J Hepatol* 1993;18(Suppl 2):S11–4.
- Keeffe EB. Hepatitis A and B superimposed on chronic liver disease: vaccine-preventable diseases. *Trans Am Clin Climatol Assoc* 2006;117:227–37; discussion 237–8.
- Chobe LP, Arankalle VA. Investigation of a hepatitis outbreak from Shimla Himachal Pradesh. *Indian J Med Res* 2009;130(2):179–84.
- Chitambar S, Joshi M, Lole K, Walimbe A, Vaidya S. Cocirculation of and coinfections with hepatitis A virus subgenotypes IIIA and IB in patients from Pune, western India. *Hepatol Res* 2007;37(2):85–93.
- Hussain Z, Das BC, Husain SA, Murthy NS, Kar P. Increasing trend of acute hepatitis A in north India: need for identification of high-risk population for vaccination. *J Gastroenterol Hepatol* 2006;21(4):689–93.
- Arankalle VA, Chadha MS, Chitambar SD, Walimbe AM, Chobe LP, Gandhe SS. Changing epidemiology of hepatitis A and hepatitis E in urban and rural India (1982-98). *J Viral Hepat* 2001; 8(4):293–303.
- Singh J, Charu P, Panda R, Bora D, Jain DC, Datta KK. Acute sporadic viral hepatitis in urban population of a tribal district in Madhya Pradesh. *Indian Pediatr* 1998;35(2):105–9.
- Kulkarni MA, Walimbe AM, Cherian S, Arankalle VA. Full length genomes of genotype IIIA hepatitis A virus strains (1995-2008) from India and estimates of the evolutionary rates and ages. *Infect Genet Evol* 2009;9(6):1287–94.
- Yoon YK, Yeon JE, Kim JH, Sim HS, Kim JY, Park DW, et al. Comparative analysis of disease severity between genotypes IA and IIIA of hepatitis A virus. *J Med Virol* 2011;83(8):1308–14.
- Hollinger FB, Emerson SU. Hepatitis A virus. In: *Fields virology*, 5th edn, Knipe D, Howley P (Eds.). Philadelphia, USA: Lippincott Williams Wilkins, 2007. pp. 911–47.
- Robertson BH, Jansen RW, Khanna B, Totsuka A, Nainan OV, Siegl G, et al. Genetic relatedness of hepatitis A virus strains recovered from different geographical regions. *J Gen Virol* 1992;73(Pt 6):1365–77.
- Irshad M, Singh S, Ansari MA, Joshi YK. Viral hepatitis in India: a report from Delhi. *Glob J Health Sci* 2010;2(2):96–103.
- Tandon BN, Gandhi BM, Joshi YK. Etiological spectrum of viral hepatitis and prevalence of markers of hepatitis A and B virus infection in north India. *Bull World Health Organ* 1984;62(1):67–73.
- 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.
- Kumar S, Ratho RK, Chawla YK, Chakraborti A. The incidence of sporadic viral hepatitis in North India: a preliminary study. *Hepatobiliary Pancreat Dis Int* 2007;6(6):596–9.

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