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Status of Hepatitis A infection: A three year study in a tertiary care hospital of Uttarakhand, India.

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Abstract: Background: *Acute viral hepatitis (AVH) is a major public health problem and an important cause of morbidity and mortality. Amongst various Hepatitis viral infections, Hepatitis A virus is known to be the commonest cause of AVH.*
Objective: *The objective of the present study was to determine the prevalence of hepatitis A virus (HAV) in the population attending our tertiary care hospital and clinically diagnosed as hepatitis.* ***Method:*** *This prospective cross-sectional study was carried out over a period of three years (2015 to 2017). Serum was separated and tests were run on miniVIDAS and VIDAS (bioMerieux, France) for the detection of anti-HAV IgM. Patients of both genders from all age groups were included in this study.*
Results: *A total of 1048 serum samples were tested over a period of three years. Throughout the three year study period it was observed that the maximum cases fell into the age groups of 20-30 years rather than in 0-10 years and 11-20 years.*
Conclusion: *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.*
Key words*: Hepatitis A, Anti-HAV IgM, viral hepatitis.*

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I. Introduction

Hepatitis A (HA) does not recognize country, state, and international borders [1], but it is a usually mild and self-limited disease, and rarely causes chronic illness [2]. Hepatitis A virus (HAV) is a member of the Hepatovirus genus of the family Picornaviridae, and is a nonenveloped single-stranded RNA virus. HAV is transmitted via the fecal-oral route either by direct contact with an infectious person or by ingestion of contaminated food or water. Persons with hepatitis A can shed the virus in their stool beginning several weeks before the onset of symptoms. The viral concentration in stool is highest in the prodromal phase. For those who develop symptoms, the viral concentration is usually very low by the time jaundice appears and undetectable before symptoms resolve [3].

Predictors of past or recent infection with hepatitis A virus include low household socioeconomic status (low income, wealth, and/or educational level), a larger household size and crowding, residence in a rural area, membership in certain ethnic groups, limited access to improved water sources, and limited access sanitation facilities [4].

Hepatitis A infection has four clinical phases, although these do not occur in all patients. The first stage is an incubation period of 15 to 50 days (mean 28 to 30 days). This stage is asymptomatic, but the infected person may be actively shedding the virus in the stool. The second stage is a pre-icteric period of several days to weeks that may precede the onset of jaundice. This prodromal period is characterized by nonspecific symptoms followed by gastrointestinal symptoms such as anorexia (loss of appetite), nausea, vomiting, abdominal pain, fatigue, malaise, and fever. Other symptoms at this stage may include myalgia (muscle pain), arthralgia (joint pain), cough, pharyngitis, constipation, diarrhea, pruritus (itchiness), and urticaria (hives). Dark urine caused by elevated bilirubin levels usually occurs prior the onset of jaundice. In the third stage, the characteristic yellowing of the skin and eyes of jaundice appear and most symptoms subside, although clinical signs such as hepatomegaly and hepatic tenderness are found in about half of patients. There is no treatment for HAV infection. Jaundice usually resolves within a few weeks. The final stage is a convalescent period during which the patient recovers [5].

The vast majority of hepatitis A patients make a full recovery, and the case fatality rate is low. The estimated mortality rate is 0.1% for children less than 15 years old, 0.3% for adults ages 15 to 39, and 2.1% for adults ages 40 and old [6]. Current or recent infection can be determined by the presence of anti-HAV IgM antibodies in serum, which are detectable soon after infection and can remain detectable for about 6 months (or longer, in some cases). Past infection can be determined by the presence of anti-HAV IgG antibodies in serum, which appear shortly after the onset of symptoms and confer long-term (usually lifelong) immunity. In endemic areas, most children are born with circulating antibodies, but these generally disappear by about 6 months of age [7] [8] [9] [10].

Acute viral hepatitis (AVH) is a major public health problem in India and other developing nations having inadequate sanitary conditions. Few studies describing the pattern of hepatitis viruses are available from North India [11] [12] [13] [14].

Involvement with hepatitis A in childhood is directly proportionate to the hygiene level of the community and observance of the rules of health by the people [15].

We carried out this study to assess the trend of infection and the epidemiological changes in our area based on our findings of serological studies on the samples referred to our laboratory for HAV diagnosis over a period of three years.

II. Materials And Methods

This prospective study was conducted in Department of Microbiology and Immunology, Sri Guru Ram Rai Institute of Medical and Health Sciences (SGRRIM&HS) and Shri Mahant Indiresh Hospital (SMIH), Dehradun, over a period of 3 years (Jan 2015 to Dec 2017).

Patients of both genders from all age groups were included in this study. Subjects were selected based on the clinical picture and suspicion by the clinicians of various departments. 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 and VIDAS (bioMerieux, France) for the detection of anti-HAV IgM. The test is based on enzyme linked fluorescent assay technology and claims sensitivity and specificity of 98.8% and 99.44%, respectively. The tests were conducted as per manufacturer's instructions. 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 considered equivocal, and were repeated.

The demographic data was collected and the results were analysed.

III. Results

A total of 1048 serum samples were tested over a period of three years, i.e.,195 in 2015, 469 in 2016 and 384 in 2017. Of these 18.47% (36/195), 13.22% (62/469) and 23.70% (91/384) were found to be positive for Anti HAV IgM antibodies in the years 2015, 2016 and 2017 respectively. Seasonal predilection of Hepatitis A infection when observed was found to range from month of May to September (Fig. 1 & 2). However, an extended period upto November was observed in 2017(Fig. 3). When the gender wise distribution was analysed it was found that more of males 20% (21/105) were positive for Anti HAV IgM antibodies in 2015, while with 17.75% (30/169) and 27.75% (48/173) females were found to dominate in Anti HAV IgM positivity in the years 2016 & 2017. Throughout the three years study period, it was observed that the maximum cases fell into the age groups of 20-30 years and less number of cases were seen in 0-10 and 10-20 years age groups (Fig. 4).

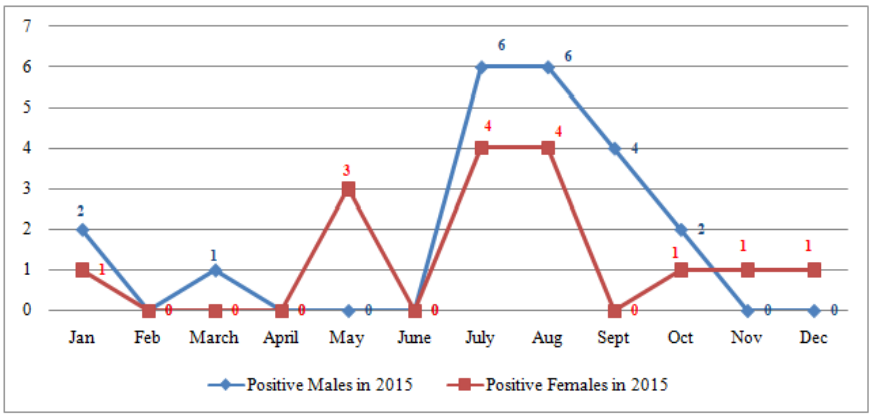

Fig. 1: Seasonal variation of Hepatitis A infection in 2015

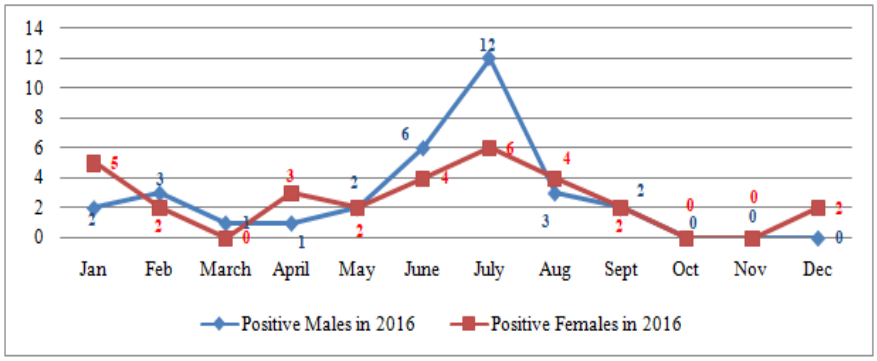

Fig. 2: Seasonal variation of Hepatitis A infection in 2016

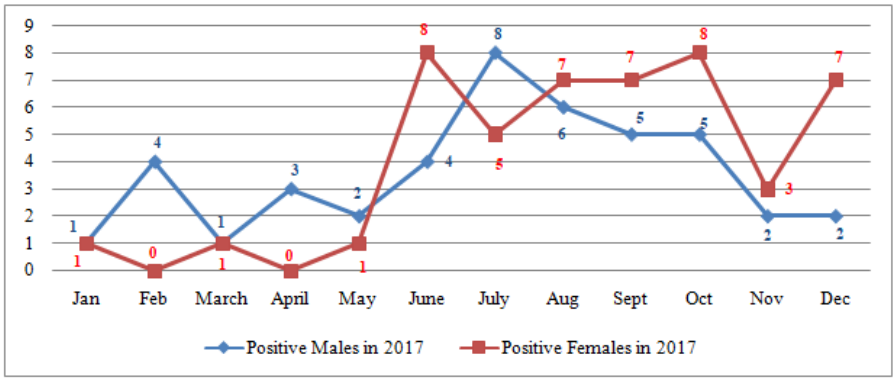

Fig. 3: Seasonal variation of Hepatitis A infection in 2017

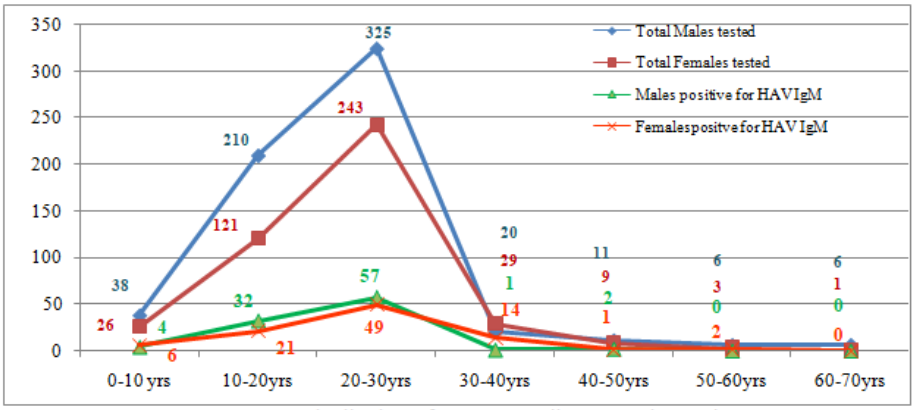

Fig. 4: Distribution of cases according to age interval

IV. Discussion

Being an area highly affected with monsoon showers all round the year, the chances of getting infected with HAV are much higher in this part of Uttarakhand as compared to other parts of the country. HAV is endemic in North India and found throughout the year [16] [17]. Still some studies have reported seasonal variation as it peaks in summer and monsoon months of the year. Although the data available on HAV in our country is not enough but a change in scenario has been observed over the last few years. In our study we found a significant rise in cases of HAV in age group of 20-30 years which shows improvement in the care of small and school going children. The lowest HAV antibody seroprevalence rates in India have consistently been reported from Kerala; Mathews *et al* and Mall *et al* have reported the lowest seroprevalence rates of 4.5 and 10.3 per cent respectively in children below 5 yr [18] [19]. This is in contrast to studies from across the country which have shown the seroprevalence to be between 60-80 per cent in children below 5 yr [20] [21].

Being a hospital based study; the socioeconomic status of the study population was not undertaken. Out of the total population tested for presence of HAV 616/1048 (58.7%) were males and 432/1048 (41.3%) were females. Men were found to be infected more than women, this may be due to the greater exposure of men in their professional and social lifestyle and this correlates with other studies [22].

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. Having the knowledge of genotype is significant as different genotype and serotype causes disease of different severity in the population.

HAV is usually a self limiting disease and if not diagnosed at appropriate time it may go unnoticed and is most commonly confused with rotavirus infection also affecting in early childhood. In high endemicity regions, majority of HAV infection is acquired in early childhood. Hepatitis A virus (HAV) infection in early childhood is mostly asymptomatic or mildly symptomatic. In the absence of specific anti-viral drugs, it requires only supportive management. The relative frequency of symptomatic hepatitis and asymptomatic infection has been reasonably well characterized and appears to be strikingly age dependent [23]. The major limitation of our study was that only those patients who were having jaundice and deranged liver function test were sent for confirmation of HAV by the gastroenterologist and paediatricians so it does not represent the true picture of the population suffering from HAV infection.

V. Conclusion

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.

Seroprevalence data relating to healthy pediatric population are necessary in order to assess the need for routine or mass vaccination. Moreover, in India, the indication for HAV vaccination is not clear due to the contradictory seroepidemiology data on children [24]. 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.

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References

[1]. Rosenthal P. Hepatitis A: A preventable threat. J Pediatr Gastroenterol Nutr. 2002; 35: 595-596.
[2]. Lynda JD, Lynn EG, Maria VK, Denis PR. Calming the panic over hepatitis A. Nursing. 2004; 34: 44–47.
[3]. Cuthbert JA. Hepatitis A: old and new. Clin Microbiol Rev 2001; 14: 36-58.; Koff RS. Hepatitis A. Lancet 1998; 351: 1643-1649.
[4]. Jacobsen KH, Koopman JS. Changing hepatitis A seroprevalence: a global review and analysis. Epidemiol Infect 2004; 133: 1005-1022.
[5]. Cuthbert JA. Hepatitis A: old and new. Clin Microbiol Rev 2001; 14: 36-58.; Hollinger FB, Ticehurst JR. Hepatitis A virus. In: Fields Virology. 3rd ed. Philadelphia: Lippincott- Raven; 1996. p. 735-782.
[6]. Hollinger FB, Ticehurst JR. Hepatitis A virus. In: Fields Virology. 3rd ed. Philadelphia: Lippincott- Raven; 1996. p. 735-782.
[7]. Abdool Karim SS, Soutsoudis A. Sero-epidemiology of hepatitis A in black South African children. S Afr Med J 1993; 83: 748-749.
[8]. Chadha MS, Chitambar SD, Shaikh NJ, Arankalle VA. Exposure of Indian children to hepatitis A virus & vaccination age. Indian J Med Res 1999; 109: 11-15.
[9]. Jacobsen KH, Koopman JS. Changing hepatitis A seroprevalence: a global review and analysis. Epidemiol Infect 2004; 133: 1005-1022.
[10]. Jacobsen KH, The Global Prevalence of Hepatitis A Virus Infection and Susceptibility: A Systematic Review. World Health Organization Department of Immunization, Vaccines and Biologicals. 2009
[11]. Irshad M, Singh S, Ansari MA, Joshi YK. Viral hepatitis in India: A Report from Delhi. Glob J Health Sci 2010; 2:96‑ 103.
[12]. 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; 62:67‑ 73.
[13]. 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; 6:596‑ 9.
[14]. 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; 31:261-5.
[15]. Sherlock S, Dooley J. Disease of the Liver and Biliary System. 11th ed. Oxford; Blackwell 2002: 274-81.
[16]. 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.
[17]. Tewari R, Makeeja V, Dudeja M. Prevalence of hepatitis A in southern part of Delhi, India. Int J Med Sci Public Health 2016; 5:2067-2070.
[18]. Mathew P, Bobba R, Zacharias P. Hepatitis A seroprevalence in Kerala. *Indian J Gastroenterol* 1998; *17*: S71-2.
[19]. Mall ML, Rai RR, Philip M, Naik G, Parekh P, Bhawnani SC, *et al*. Seroepidemiology of hepatitis A infection in India: changing patterns. *Indian J Gastroenterol* 2001; *20*: 132-5.
[20]. Tandon BN, Gandhi BM, Joshi YK. Aetiological spectrum of viral hepatitis and prevalence of markers of hepatitis A and B virus infection in North India. *Bull World Health Organ* 1984; *62* : 67-73.
[21]. Mittal SK, Rastogi A, Kumar N, Talukdar B, Kar P. Seroprevalence of hepatitis A in children - Implications for hepatitis A vaccine. *Trop Gastroenterol* 1998; *19* : 120-1.

[22]. 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.
[23]. Hadler SC, Webster HM, Erben JJ, Swanson JE, Maynard JE. Hepatitis A in day care centres. A community- wide assessment. *N Engl J Med* 1980; *302*: 1222-7.
[24]. Batra Y, Bhatkal B, Ojha B, Kaur K, Saraya A, Panda SK, *et al*. Vaccination against hepatitis A virus may not be required for schoolchildren in Northern India: Results of a seroepidemiological survey. Bull World Health Organ 2002; 80:728-31.

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