Seroprevalence of Hepatitis E from a Tertiary Care Hospital in Central Delhi, India

Shalini Malhotra, Sanjiv Ahuja, Nirmaljit Kaur Bhatia, Shweta Sharma, Ankit Chauhan and Charoo Hans

¹Department of Microbiology, R.M.L Hospital & P.G.I.M.E.R. India.

(Received: 21 October 2011; accepted: 11 December 2011)

Hepatitis E virus (HEV) is a spherical, non-enveloped, single stranded RNA virus. It has five genotypes. *Enterically* transmitted hepatitis E virus is a major cause of outbreaks & sporadic cases of viral hepatitis in developing countries like India. Several reports are available in India about well characterised HEV cases, which are mainly about large scale epidemics in communities. However information about the clinical and epidemiological characteristics of HEV cases which may be epidemic or sporadic in origin are scanty in India. This study was planned to determine IgM antibodies and thereby evaluate the *Seroprevalence* of Hepatitis E in patients with acute hepatitis in Central Delhi & adjoining areas. Prevalence of hepatitis E was studied over a period of two year in 3945 patients by the demonstration of IgM antibodies. In 3945 patients 661 (16.75%) were positive for hepatitis E. Hepatitis E was mainly seen in between April-June. Primary prevention is the cornerstone of HEV control which mainly depends upon improvement of sanitary conditions.

Key Words: Seroprevalence, Hepatitis E virus, Viral Hepatitis, Enterically transmitted.

Acute viral hepatitis E, a self limiting disease presenting as acute, icteric hepatitis, is caused by Hepatitis E virus (HEV). It is a small, non-enveloped RNA virus, icosahedral in shape and 27-34nm in diameter¹. It has five genotypes: genotype 1 (Asia-Africa), genotype 2 (United States), genotype 3 (Mexico), genotype 4 (Beijing, China) and genotype 5 (Europe). HEV is excreted in faeces and is transmitted by the fecal-oral route usually through contaminated water². It has a long incubation period (28-40days) and affects older children and adults³.

HEV has been responsible for major outbreaks of acute infections in developing countries of Asia, Africa and Latin America over the last 50 years. The first documented epidemic of HEV was reported in New Delhi, India in 1955-1956, and 29,300 people were affected⁴. Outbreaks have been reported from several countries of South and South-East Asia, Eastern Europe and North and East Africa. A major outbreak was reported from South Xinjiang in Uighar region of China, in 1986-1988 which lasted for 20 months. In this outbreak 1,19,000 cases were reported⁵. In 1991 HEV outbreak occurred in India (Kanpur) and it affected over 79,000 cases⁶. However information about clinical and epidemiological characteristics of HEV cases which may be from epidemic source or sporadic in origin are scanty in India. So the present study aims to determine the occurrence of specific IgM antibodies to HEV in clinical cases of hepatitis and to correlate the same with existing liver conditions.

Mob.: +91-9810778233

E-mail: drshalinimalhotra@yahoo.com

 $^{^{\}ast}$ To whom all correspondence should be addressed.

MATERIAL AND METHODS

A total of 3945 patients (out-patient department and admitted) with suspected diagnosis of infectious hepatitis presenting to Microbiology Department of Dr R.M.L. Hospital & P.G.I.M.E.R., New Delhi were included. The study was carried out between July 2009 to June 2011. 3-5ml of blood was collected from all these patients. IgM antibodies to HEV was determined using a commercially available IgM capture ELISA (Asialion Biotechnology, China) using manufacturer's instructions. Test was considered positive if the sample optical density (OD) was equal to or higher than cut off value (mean of negative control+0.10). The serological status for other hepatitis markers namely HBsAg (HBV), IgG HCV and IgM HAV were also reviewed. A record of liver function tests (LFT) profile of all these patients were also maintained along with demographic and environmental variants.

RESULTS AND DISCUSSION

Out of a total of 3945 samples, 661 serum samples were positive for HEV IgM. Amongst the sero-positive patients, percentage of males affected were much more as compared to females. Age-wise distribution of Hepatitis E positive patients is shown in Table 1. There was association of HEV and other liver conditions as shown in Table 1. Analysis of results in various months showed its presence mainly in summer season as shown in Fig 1.

HEV is the causative agent of hepatitis E and has been assigned the genus *Hepevirus*, family Hepeviridae. HEV is enterically transmitted and causes an acute and generally self-limiting infection of the liver but with a higher mortality in general when compared to infections with Hepatitis A virus (HAV), which is transmitted via the same route⁷. The classical epidemiological studies by Vishwanathan⁴ and recent serological study by Wong et al8, Khuroo9, Panda et al., 10 have convincingly demonstrated that HEV is an important cause of Non-A Non-B viral hepatitis. Occurrence of HEV specific IgM antibodies was noted in 16.75% of serum samples in our study. Similar findings (17.3% from Tamil Nadu³ and 18.8% from Karnataka¹¹) have been reported from other parts of India and also from Iraq (19.4%)12 and Pakistan (14.1%)¹³. In the current study male preponderance was noted (70.8%) and this correlates with findings of another study from India (67.3% from Karnataka). 11 The youngest person found to be sero-positive was 8 months old and oldest person was 78 years old male. However, older children and adults upto 50 years were found to be more susceptible (19.28%) as compared to younger children and adults over 50 years of age. This was similar to a study from Tamil Nadu in which older children (>12years) and adults were more susceptible (18.5%)³ but in another Indian study from Karnataka adults above 20 years of age were more susceptible (20.17%)¹¹. Amongst the 661 HEV positive cases 13 (1.97%) were found to be co-infected with hepatitis B virus (HBsAg positive). This corroborates with another study

Table 1. Age and sex-wise distribution of IgM-HEV Seropositive patients and its association with other viral markers

Age group in years	Male	Female	Total no positive (%)	No. (%) of co-infection with other viral markers		
				HBsAg	HCV	HAV
1-10	27/219	8/145	35/364 (9.6)	0	0	6
11-20	75/256	27/181	102/437 (23.3)	3	0	2
21-30	123/515	126/584	249/1099 (22.7)	4	0	0
31-40	185/1057	16/159	201/1216 (16.5)	6	1	1
41-50	41/251	8/119	49/370 (13.2)	0	0	0
51-60	12/163	5/85	17/248 (6.8)	0	0	0
>60	5/114	3/97	8/211 (3.8)	0	0	0
Total	468/2575	193/1370	661/3945 (16.75)	13(1.97)	1(0.15)	9(1.36)

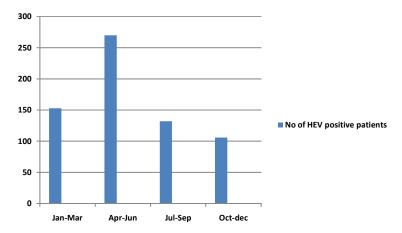


Fig. 1. Seasonal distribution of HEV positive cases

from Karnataka¹¹. These findings can be attributed to the fact that Delhi & Karnataka are meso-endemic zones for HBV. Only 1.36% HEV infected patients were positive for IgM HAV antibodies. This may be due to common mode of transmission i.e contaminated food and water. A case (0.9%) was also associated with hepatitis C and this correlates with co-infection rate reported in other studies ^{11,14}.

CONCLUSION

In summary, we found that HEV was commonly seen in older children and adults. Since hepatitis E is rampant in India, for both epidemiological and sporadic hepatitis cases, the prevention strategies should aim at providing clean drinking water, proper sewage disposal and good health. Travelers to endemic regions must take precautions against the consumption of contaminated water. Although infection via food appears to be much less common for hepatitis E than for hepatitis A, travellers must maintain vigilance about the risks of contaminated water, ice and food.

REFERENCES

- Mahtab MA, Rahman S, Khan M, Khan M, Karim F. HEV Infection as an aetiological factor for acute hepatitis: Experience from a Tertiary Hospital in Bangladesh. *J Health Popul Nutr*. 2009; 27(1): 14-19
- 2. Alavian SM, Fallahian F, Lankarani KB. Epidemiology of Hepatitis E in Iran and

- Pakistan. Hep Mon 2009; 9(1): 60-65
- Radhakrishnan S, Raghuraman S, Abraham P, Kurian G, Chandy G, Sridharan G. Prevalence of Enterically Transmitted hepatitis viruses in patients attending a tertiary – Care Hospital in South India. *Indian J Pathol Microbio* 2000; 43(4): 433-436
- 4. Viswanathan R. Infectious hepatitis in Delhi (1955-1956): a critical study with Epidemiology. *Ind J Med Res, Suppl* 1957; **45**: 1-29.
- Zuang H, Cao X Y, Liu CB. Enterically transmitted non-A, non-B hepatitis in China. In Shikata T, Purcell RH, Uchida T, editors. Viral hepatitis C,D and E: proceedings of the international Meeting on Non-A, Non-B Hepatitis. Amsterdam. Elsevier Science 1991: 275-285
- Ray R, Aggarwal R, Salunke PN, Mehrotra NN, Talwar GP, Naik SR. Hepatitis E virus genome in stools of hepatitits patients during large epidemic in north India. *Lancet* 1991; 338: 783-784
- Anderson DA. Hepatitis E Virus. In: Mandell GL, Bennett JE, Dolin R editors. Principles and practice of infectious disease. 7th ed. *Philadelphia*, PA. Churchill Livingstone 2010; 2: 2411-2421
- 8. Wong Dc, Purcell RH, Srinivasan MA, et al. Epidemic and endemic hepatitis in India: Evidence for Non-A Non-B etiology. *Lancet* 1980; **2**: 876.
- 9. Khuroo MS, Rustogi VK, Dawson GJ, et al. Spectrum of hepatitis E virus infection in India. *J Med Virol* 1994; **43**: 281-286.
- Panda SK, Dutta R, Kaur J, et al. Enterically transmitted non-A, non-B hepatitis: recovery of virus like particles from an epidemic in South

- Delhi and transmission studies in rhesus monkey. *Hepatology* 1989; **10**: 466.
- 11. Mishra B, Srinivasa H, Muralidharan S, Charles S, Macaden RS. A Hospital based study of hepatitis E by serology. *Ind J Med Microbiol* 2003; **21**(2): 115-117.
- 12. Turky AM, Akram W, Al-Naaimi AS, Omer AR, Al-Rawi JR. Analysis of acute viral hepatitis in Iraq. *Global Journal of Health Science* 2011;
- **3**(1): 70-75.
- 13. Saeedi MI, Mahmood K, Amanullah, Zianuddin M, Ilyas N, Zarif M. Frequency and clinical course of hepatitis E in tertiary care hospitals. *J Coll Physicians Surg Pak* 2004; **14**(9): 527-529.
- 14. Tandon BN. Viral hepatitis in tropics and its management, *JAMA India The physicians*' Update 2001; **4**: 102-106.