

Exposer Rate of Hepatitis E (IgG) in a Selected Population of Children and Adults in Karachi

Huma Qureshi, Anjum Shahid (PMRC Research Centre, Jinnah Postgraduate Medical Centre, Karachi.)
S.A. Mujeeb (Blood Bank, Jinnah Postgraduate Medical Centre, Karachi.)

Abstract

Aims: To see exposure rate of hepatitis E (IgG) in 100 apparently healthy children and adults.

Subjects and Methods: Sera from 100 healthy children aged 1 day to 10 years and too healthy adults aged 1.8-45 years were analysed for exposure to hepatitis B (IgG) using ELISA.

Results: Two samples from children were excluded from the study due to improper storage leaving 98 samples for analysis. Of 98 sera from children 19.4% were positive for IgG indicating previous exposure to hepatitis B. The exposure rate increased with age and was 10% in children below 1 year of age, 14% at 2 years, 19% at 3 years and 28% at 1.0 years. In adults overall exposure was 16%. There was no predominance of either sex in both the groups and all individuals belonged to middle to lower socioeconomic strata.

Conclusion: An exposure of 19% in children and 16% in adults indicates high faecal contamination of drinking water and re-addressing of the issue of use of boiled water on individual level, supply of potable water on the government level and a need to produce a vaccine on international level (JPMA 50:352, 2000).

Introduction

Hepatitis E virus is one of the major causes of water borne epidemics of non A non B (NANB) hepatitis. The predominant mode of transmission is through the faeco-oral route. Large epidemics have been reported from India¹⁻³, Nepal⁴, Burma⁵, Algeria⁶ and Soviet Union⁷. Two major and multiple mini epidemics have been reported from Pakistan⁸⁻¹⁴. The clinical disease occurs either as epidemic or sporadic. The diagnosis is made using ELISA kits which has both IgM and IgG antibodies either separately or combined as total test. Both IgM and IgG antibodies appear 10-15 days after HEV infection, the IgM in majority becomes undetectable after 40-50 days but IgG persists for 10-15 years¹⁵. Seroprevalence studies of HEV infection are based on the presence of IgG antibodies in the healthy population.

The seropositivity is low in developed countries where it varies from 2% in USA, 3% in UK and Europe to less than 1% in Japan and Australia^{16,17}. In developing countries like Burma⁵, Nepal¹⁸, India¹⁻³, China^{19,20}, Sudan²¹, the figures are as high as 10-45%. Intermediate prevalence rate of 2-10% is reported from Indonesia, Korea, Taiwan and some Latin American and African countries^{22,23}, advancement seropositivity.

Seropositivity increases with the of age. A Chinese study reported a of 7.2% in preschool children, 18.9% in adults to 33% in older age amongst adolescents 24.5% group²⁴. In Sudan 70% exposure rate was reported in children and 20% in adults²¹. In Pakistan 17% children and 29% adults are exposed to this virus²⁵. In Western India 60% of children showed exposure while only 5% adults were IgG positive²⁶. Both sexes are equally affected but the disease is more in rural areas. The present study was done to determine the seropositivity of HEV in healthy population using IgG antibodies.

Subjects, Methods and Results

Sera from well baby clinics and school going children, whose ages ranged from 1-10 years were collected. Adults included voluntary blood donors whose ages ranged from 1 8-45 years. Preliminary

work included a filling of Performa containing baseline information on age, sex, place of residence and socioeconomic status. Individuals having a past history of jaundice were excluded. Sera were labelled and stored at -70°C till analyzed. All blood samples were initially tested for the presence of IgG antibodies to HEV (Abbott HEV EIA, Abbot Laboratories, Chicago, USA). All initially reactive samples were tested in repeat and no supplemental test was done to verify the results.

During 1999 a total of 98 sera from children and 100 from adults were collected. Of the 98 sera of children 19.4% were IgG positive. The exposure rate increased with the age and was 10% in children under 1 year age to 14% at 2 years, 19% at 3 years and 28% at 10 years. Overall exposure in children was 19.4% and in adults the exposure rate was 16%. There was no sex predilection and all individuals belonged to the urban setting of low to middle socioeconomic group.

Comments

The exposure rate to hepatitis E virus (HEV) in healthy population varied from 19% in children to 16% in adults. This high exposure rate is an under-estimation of the actual figures, because this study included only those subjects who had no previous exposure to jaundice. Had the patients with history of jaundice in the past been included, the figures might have been much higher. The high exposure rate also indicates the extent of faeco-oral contamination and thus shows the quality of the potable water. High faeco-oral contamination is also confirmed by an almost 97% exposure to hepatitis A virus in our country^{27,28}. Although mini epidemics of hepatitis E have been reported in our country but the virus runs a sporadic course all the year round.

High maternal mortality has been reported in the third trimester in infected females, during epidemics from Kashmir²⁹, Ahmedabad³⁰, Azamgarh³¹, New Delhi³², Kirzighistan³³, Burma³⁴, Indonesia³⁵ and China³⁶. The risk to mother varies from acute liver failure, fulminant hepatitis to encephalopathy with or without coagulopathy within 4 weeks of onset of jaundice without previous evidence of chronic liver disease. Foetal outcome also varies from abortion, still birth, intrauterine growth retardation, teratogenic effect to normal progression of pregnancy. In a study done in Karachi on 13 fulminant hepatic failure cases admitted during endemic season, foetal loss was 50% with 16.6% maternal loss³⁷. As maternal loss is over 40% during epidemics therefore it has been suggested by Khurro that in all high endemic areas pregnant women should be given immune globulin during epidemics³⁸.

Although HEV infection has low mortality but its morbidity is very high causing lot of stress on individual plus government funds. It is therefore suggested that a joint venture needs to be initiated at the local level, government level and internationally. Boiling of water needs to be stressed at local level, supply of potable water is to be made a priority on the government level and development of vaccine to be stressed internationally.

References

1. Wang DC, Purcell RH, Sreenivasan MA, et al. Epidemic and endemic hepatitis in India: evidence for a non A non B hepatitis virus etiology. *Lancet*, 1980; 2:876-9.
2. Khurro MA. Hepatitis E. Enterically transmitted non-A non-B hepatitis *Indian J. Gastroenterol.* 1991; 9: 96-100.
3. Naik SR, Aggarwal R, Salunke PN, et al. A large waterborne viral hepatitis E epidemic in Kanpur, India. *Drill. WHO.*, 1992; 70: 597-604.
4. Kane MA, Bradley DW, Shrestha SM, et al. Epidemic non A non B hepatitis in Nepal. Recovery of a possible etiological agent and transmission studies in marmosets. *JAMA.*, 1984; 252: 3140-42.
5. Shwe S, Soc MM. Epidemiological criteria as indication of non A non B hepatitis in a community (letter). *Lancet*, 1985; 2: 828.
6. Belabbes El-i, Bouguermouth A, Ahanted HA, et al. Epidemic non A non B viral hepatitis in Algeria; strong evidence for its spreading by water, *J. Med. Virol.*, 1985; 16: 257-8.

7. Balayan MS, And aparidze AG, Sttvinskaya SS, et al. Evidence of a virus in non Anon B hepatitis transmitted via the featoral route. *intervirology*. 1983;20 23.
8. Smego RA, Klmalig AA. Epidemic non A 'non B hepatitis in urban Karachi, Pakistan. *Ant. J. Trop. Med. Flyg.*, 1988; 38: 628-32.
9. Iqbal M, Ahmed A, Qamar A, ci at. An outbreak of enterically transmitted non A non B hepatitis in Pakistan. *Atn. 3. Trop. Mcd. ilyg.*, 1988; 40:438-43.
10. Malik IA, Qureshi MS, Luqinan M, et al, Epidemic non A non B hepatitis in Pakistan, *Trop. Doct.*, 1988: 18: 99 101.
11. Quraishi MS, Manzoor A, Rashid H, et al, tiepatitis non A non B: report of a water-borne out break. *J Pak Med Assoe.* 1988: 38: 203-5.
12. Ahmad M, Quraishi MS. Mushtaq 5, et at. Acute sporadic hepatitis non A non B clinical features and biochemical profile J. 'ak. Mcd, Assoe. 1989: 39: 307-9.
13. Khan R. A study of non A nout B hepatitis with specific reference to hepatitis E. *Pak. 3. Health*, 1993; 301: 37-42.
14. Rab MA, Bile MK, Mubarak MM. et at. Water-brone hepatitis E virus epidemic in Islamabad, Pakistan: A common source of outbreak traced to the malfunction of a modern water treatment plant. *Am, 3, Trop. Med, Etyg.*, 1997; 57: 151-57.
15. Khuroo MS, Kamili 5, Oar MY, et al. Hepatitis F; and long term antibody statuts. *Lancet*, 1993: 34 1:1355.
16. Dowson GJ, Chau KI I Cabal CM, et al. Solid phase enzyme linked immunsorbent assay for hepatitis E virus IgG and 1gM antibodies utilizing recombinant antigens amid peptides. *J. Viral. Methods.*, 1992; 38: 175-86.
17. Dowson G, Paul S, Geutierrez T, et al. viral hepatitis and liver diseases. 1994, pp.371-4.
18. Longer CE, Shreshtha MP, Mac Arthy PD. Epidemiology of hepatitis H virus (11Ev): a cohort study in Kathmandu, Nepal. lit: *Viral hepatitis and liver disease.*(eds) Nishoka K, Suzuki H,Mishiro 5, Oda Tokyo. Springer-Verlag: 1994.
19. Zhttatig II. Epidemiology at Hepatitis E in China In: *Hepatitis E virus epidemiology to candidate vaccine* (eds) Tandon BN, Aeharva SK. London a tropical gastroenterology publication 1995, p 23.
20. Lee SD, Wang Ti, Ltt RH et al. Seroprevalance of antibody to hepatitis E virus amongst Chinese subjects in Taiwan. *Hepatology*, 1993; 19. 866-70.
21. Kenneth C, Hyams MA, Purdy MK et at. Acute sporadic hepatitis E in Sudanese children: analysis based on new Western blot assay. *J. Infect Dis.*, 1992; 165: 1001-5.
22. Tandon B.N, Aeharya SK. *Hepatitis E virus - Epidemiology to candidate vaccine* London. A tropical gastroenterology publication. 1995.
23. Tucker 'TJ. Kirsch RE, Louw M, et at. Epidemiology of hepatitis H in South Africa. In *Hepatitis E virtis epidemiology to candidate vaccine* (eds) 'faitdon ltN, Aeharya SK. London, A tropical gastroenterology pumblication 1995 p 33.
24. Loe A, Kwan WK, Moeckil RR, et al. Sero epidemiology survey of hepatitis E in Hong Kong by recombiant based enzyme immunoassays *Lancet*, 1992: 340: 1205-8.
25. Bryan JP, Tsarev SA, Iqbal M, et al, Epidemic hepatitis F in Pakistan: Pattern of serologic response and evidence that antibody to hepatitis E protects against disease. *3. Infect. Dis.*, 1994; 17: 517-21.
26. Khuroo MS, Rustgi VK, Dowson GJ et at. Spectrum of hepatitis E virus infection in India. *3. Med. Viral.*, 1994: 43. 281-6.
27. Malik IA, Legters Li, Luqman M, et al. The serological markers of hepatitis A and B in healthy population in Northern Pakistan. *3 Pak. Med. Assoc*, 1988; 38; 69-72.
28. Lodi TZ. Zuberi Si. Cost effective approach for serological diagnosis of hepatitis. *3. Pak. Med, Assoc*, 988: 38: 199.
29. Khuroo MS. Tali MR. Skidmore 5, et at. Incidence and severity of viral hepatitis in pregnancy. *Am. 3. Med.*, 1981; 71); 252-55.

30. Sreemtiyasan MA, Banerjee K, Pandya PC, et al. Epidemiology investigations of an outbreak of infectious hepatitis in Ahmadabad city during 1975-76. *Indian J. Med. Res.*, 1978; 67: 197-206.
31. Tandon BN, Joshi YK, Jan SK, et al. An epidemic of non A non B hepatitis in north India. *Indian J. Med. Res.*, 1982; 75: 739-44.
32. Vishwanathan R. Infectious hepatitis in Delhi (1955-56): A critical study; epidemiology. *Indian J. Med. Res.*, 1957; 45: 1-30.
33. Scrguev NW, Paksoris EA, Ananov VA. General characteristics of Botkin's disease occurring in Kirgiz Republic of USSR in 1955-56. *Soviet Healthcare Kirgizii* 1957; 5: 16-23.
34. Myint H, Soc MM, Khin T, et al. A clinical and epidemiological study of an epidemic of non A non B hepatitis in Rangoon. *Am. J. Trop. Med. Hyg.*, 1985; 31: 103-10.
35. Krawczynski K. Hepatitis E. *Hepatology*. 1993; 17: 932-41.
36. Zhuang H, Cao XV, Lu CB, et al. Enterically transmitted non A non B hepatitis in China. In Shikata T, Purcell RH, Uchida T (eds). *Viral hepatitis CDE*. Amsterdam, Elsevier Science Publishers, 1991, pp. 277-85.
37. Hamid SS, Jafri SM, Khan H, et al. Fulminant hepatic failure in pregnant women: acute fatty liver or acute viral hepatitis? *J. Hepatol.*, 1996; 25: 20-7.
38. Khurro MS. Hepatitis E; The enterically transmitted non A non B hepatitis. *Indian J. Gastroenterol.*, 1991; 10: 96-100.