

Editorial

World Hepatitis Day- new challenges

Viral hepatitis is the most common cause of acute and chronic liver disease in the world with over half the world's population exposed to the different hepatotropic viruses. The estimated 400-500 million people with chronic viral hepatitis has been recently described as a game changer in Hepatology¹. Hepatitis B and C contribute to a very high grade of disease. The spectrum of viral hepatitis differs with respect to the aetiological agents in different geographical regions of the world. In India, HEV infection is responsible for most of the epidemics of viral hepatitis. HEV infection is responsible for 30-70 per cent of the cases of sporadic hepatitis and the major cause of acute liver failure (ALF)²⁻⁶. Many a times even epidemics of sporadic outbreaks of hepatitis A, have also been reported from our country⁷⁻¹¹. Since hepatitis E is rampant in India, the need of the hour is to adopt preventive strategies that could aim at providing clean drinking water, proper sewage disposal and health education. HEV ORF 2 proteins as candidate vaccine¹² could turn out to be an additional armentonium in near future.

In India, hepatitis B accounts for 15-30 per cent of the cases of the acute hepatitis and 70 per cent cases of chronic hepatitis while HCV is an infrequent cause of acute icteric hepatitis though it is responsible for most of the cases of post transfusion hepatitis^{13,14}. In our country, hepatitis D is responsible for less than 10 per cent of the patients of acute and chronic HBV infection¹⁵. In India, the HBV carrier rate is approximately 4 per cent¹⁶. The majority of severe sequelae occur in patients who are chronically infected with HBV; a significant proportion develop liver cirrhosis or hepatocellular

carcinoma. Moreover, these chronically infected patients serve as reservoir for continuing HBV transmission. HBV is transmitted by precutaneous or mucosal contact with infectious blood or other body fluids (serum, semen, and saliva). In case of infants and children, the infection is acquired through prenatal transmission and horizontal transmission from infected household contacts. Adolescents and adults are mostly infected through sexual activity, sharing needles in case of injected drugs use (IDU) or accidental needle stick injuries in health care settings. Since the epidemiological data reflect that HBV causes a considerable disease burden in India the basic need is to target higher risk population for HBV vaccination and to insist on inclusion of HBV vaccine in the universal immunization schedule so as to reduce the HBV carrier frequency. Quality control of donor screening in India is another area which needs attention. Awareness campaign by the India National Association of the Study of Liver with respect to the risk of community acquired infection and steps to prevent household and nosocomial spread of HBV infection need to be launched.

In India, HCV infection is acquired mostly through transfusion of blood or blood products¹⁷⁻¹⁹. In our country, HCV infection as a cause of acute viral hepatitis has been reported to vary between 0-21 per cent²⁰⁻²³ and responsible for 14-26 per cent cases of chronic liver disease^{17,24,25}. A considerable number of cases have asymptomatic HCV presentation. The magnitude of HCV infection amongst the patients of chronic liver disease is likely to increase in future since blood banks in India have only recently introduced the policy of anti-HCV screening. Hence

all those individuals who have been exposed before this are likely to develop the disease in next 15-30 years. No vaccine has yet been developed against hepatitis C because of the large and frequent genetic variation. Screening and testing of blood and organ donors, strong education programme and infection safety practices in health care settings are currently the most effective preventive measures for hepatitis C. It is believed that failure to address acute transmission of HCV infection will undermine long term attempts to reduce HCV associated disease burden. Further, spending more resources in this direction would allow identification of iatrogenic and nosocomial infections which still occur and are largely unrecognized. A co-ordinated multilevel approach is a priority.

Further improvement of chronic HBV and HCV screening will add to the demand for vaccination and care services, thus increasing the need to prepare added public health and medical systems to administer and care for people with viral hepatitis. There is a need to collect data at the national, State, and direct level so as to evaluate and supervise the prevention policies. The existing viral hepatitis scrutiny systems are not enough in their competence to monitor chronic infections and to measure the burden of morbidity and mortality due to viral hepatitis. The epidemiology of viral hepatitis is shifting and presents new prevention challenges. A better public health response will be required involving governmental, academic, and community-based organizations. A combined effort and sensible stride towards the direction of prevention of viral hepatitis infection and disease control can help accomplished the goal.

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References

1. <http://www.aasld.org/news/060211/Pages/default.aspx>.
2. Nanda SK, Yalcinkaya K, Panigrahi AK, Acharya SK, Jameel S, Panda SK. Etiological role of hepatitis E virus in sporadic fulminant hepatitis. *J Med Virol* 1994; 42 : 133-7.
3. Madan K, Gopalkrishna V, Kar P, Sharma JK, Das UP, Das BC. Detection of hepatitis C and E virus genomes in sera of patients with acute viral hepatitis and fulminant hepatitis by their simultaneous amplification in PCR. *J Gastroenterol Hepatol* 1998; 13 : 125-30.
4. Acharya SK, Panda SK, Saxena A, Gupta SD. Acute hepatic failure in India: A perspective from the East. *J Gastroenterol Hepatol* 2000; 15 : 473-9.
5. 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.
6. Kar P, Budhiraja S, Narang A, Chakravarthy A. Etiology of sporadic acute and fulminant non-A, non-B viral hepatitis in north India. *Indian J Gastroenterol* 1997; 16 : 43-5.
7. Acharya SK, Batra Y, Bhatkal B, Ojha B, Kaur K, Hazari S, et al. Seroepidemiology of hepatitis A virus infection among school children in Delhi and north Indian patients with chronic liver disease: Implications for HAV vaccination. *J Gastroenterol Hepatol* 2003; 18 : 822-7.
8. Chadha MS, Walimbe AM, Chobe LP, Arankalle VA. Comparison of etiology of sporadic acute and fulminant viral hepatitis in hospitalized patients in Pune, India during 1978-81 and 1994-97. *Indian J Gastroenterol* 2003; 22 : 11-5.
9. Mall ML, Rai RR, Philip M, Naik G, Parekh P, Bhawnani SC, et al. Seroepidemiology of hepatitis A infection in India. Changing pattern. *Indian J Gastroenterol* 2001; 20 : 132-5.
10. Das K, Jain A, Gupta S, Kapoor S, Gupta RK, Chakravorty A, et al. The changing epidemiological pattern of hepatitis A in an urban population of India: Emergence of a trend similar to the European countries. *Eur J Epidemiol* 2000; 16 : 507-10.
11. Dhawan PS, Shah SS, Alvares JF, Kher A, Shankaran, Kandoth PW, et al. Seroprevalence of hepatitis A virus in Mumbai, and immunogenicity and safety of hepatitis A vaccine. *Indian J Gastroenterol* 1998; 17 : 16-8.
12. Li TC, Suzaki Y, Ami Y, Dhole TN, Miyamura T, Takeda N. Protection of cynomolgus monkeys against HEV infection by oral administration of recombinant hepatitis E virus-like particles. *Vaccine* 2004; 22 : 370-7.
13. Dasarathy S, Misra SC, Acharya SK, Irshad M, Joshi YK, Venugopal P, et al. Prospective controlled study of posttransfusion hepatitis after cardiac surgery in a large referral hospital in India. *Liver* 1992; 12 : 116-20.
14. Saxena R, Thakur V, Sood B, Guptan RC, Guruja S, Sarin SK. Transfusion associated hepatitis in a tertiary referral hospital in India. A prospective study. *Vox Sang* 1999; 77 : 6-10.
15. Irshad M, Acharya SK. Hepatitis D virus (HDV) infection in severe forms of liver diseases in north India. *Eur J Gastroenterol Hepatol* 1996; 8 : 995-8.
16. Tandon BN, Acharya SK, Tandon A. Epidemiology of hepatitis B virus infection in India. *Gut* 1996; 38 (Suppl 2) : S56-9.
17. Panigrahi AK, Panda SK, Dixit RK, Rao KV, Acharya SK, Dasarathy S, et al. Magnitude of hepatitis C virus infection in India; Prevalence in healthy blood donors, acute and chronic liver disease. *J Med Virol* 1997; 51 : 167-74.
18. Hazari S, Panda SK, Gupta SD, Batra Y, Singh R, Acharya SK. Treatment of hepatitis C virus infection in patients of northern India. *J Gastroenterol Hepatol* 2004; 19 : 1058-65.
19. Amarapurkar D, Dhorda M, Kirpalani A, Amarapurkar A, Kankonkar S. Prevalence of hepatitis C genotypes in Indian patients and their clinical significance. *J Assoc Physicians India* 2001; 49 : 983-5.

20. Mehta SK, Singh V, Bhasin DK, Kumar YR, Kochhar R. Hepatitis C virus in patients with acute and chronic liver disease. *Indian J Gastroenterol* 1992; 11 : 146.
21. Irshad M, Acharya SK. Status of hepatitis viral markers in patients with acute and chronic liver diseases in northern India. *Intervirology* 1994; 37 : 369-72.
22. Jaiswal SP, Chitnis DS, Naik G, Artwani KK, Pandit CS, Salgia P, *et al.* Prevalence of anti-HCV antibodies in central India. *Indian J Med Res* 1996; 104 : 177-81.
23. Arankalle VA, Tungatkar SP, Banerjee K. Anti-HCV positivity among blood donor population from Pune, India (1981-1994). *Vox Sang* 1995; 69 : 75.
24. Ramesh R, Munshi A, Panda SK. Prevalence of hepatitis C virus antibodies in chronic liver disease and hepatocellular carcinoma patients in India. *J Gastroenterol Hepatol* 1992; 7 : 393-5.
25. Nandi J, Bhawalkar V, Mody H, Elavia A, Desai PK, Banerjee K. Detection of HIV-1, HBV and HCV antibodies in blood donors from Surat, western India. *Vox Sang* 1994; 67 : 406-7.