EDITORIAL

# Review of Hepatitis C in Malaysia

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The identification of the Hepatitis C Virus (HCV) by Choo *et al* in 1989[1] was rapidly followed in 1990 by the development and commercialisation of HCV-Antibody screening tests. This made it possible to conduct epidemiological and prevalence studies on this new member of the family of Hepatitis Non-A Non-B Viruses.

HCV-Antibody prevalence data in Malaysian population is presently available from a number of as yet unpublished studies conducted locally. One such study (M Sinniah & BG Ooi, unpublished) in the Institute for Medical Research (IMR) involving 1434 human sera from eleven clinical/risk groups collected between 1985 and 1991 indicated that HCV infection occurred in individuals who are already known to be at risk for Hepatitis B and that the HCV-antibody prevalence was lowest in healthy blood donors (3%) and highest in intravenous drug abusers (85.3%). The prevalence according to each risk group was Haemophiliacs (64.3%); Dialysis Patients with Chronic Renal Failure (CRF) (53.9%); Liver Cirrhosis (28%); Hepatocellular Carcinoma (HCC) (13.8%); Chronic Active (CAH) and Chronic Persistent Hepatitis (CPH) (6.5%); Male Homosexuals (10.8%); Female Prostitutes (9%); Personnel in haemodialysis Unit (0%) and IMR laboratory (0%).

Further evidence that haemodialysis patients in Malaysia are at increased risk for HCV infection comes from Zaki *et al*'s study (Personal communication) in General Hospital, Kuala Lumpur where a total of 287 CRF patients were enrolled for HCV-AB screening comprising three groups viz. those on Home Haemodialysis (HHD), Chronic Ambulatory Peritoneal Dialysis (CAPD) and those undergoing Centre Haemodialysis. The overall HCV-AB positivity was 43.2%, the highest prevalence being among Centre Haemodialysis patients (55.5%) followed by HHD patients (41.9%) and lowest among CAPD patients (18.9%).

That blood and blood products cannot be guaranteed to be absolutely safe from HCV infection is concluded from various studies which report HCV-AB prevalence rates ranging from 64.3% in haemophiliacs (M Sinniah & BG Ooi, unpublished) to 58% in multiple transfused patients (Zuraidah *et al*, Personal communication); also I. Ishak *et al*, (Personal communication) reported the presence of HCV-AB in 5.77% of multiple transfused thalassemic patients in their clinic as compared to none of non-transfused thalassemics.

The HCV exposure rate in Malaysian blood donors varied from 3% in 363 donors (M Sinniah & BG Ooi, unpublished) to 2% in 344 blood donors (VJL How *et al*, Personal communication), the difference may perhaps be due to racial differences in the sample population, which therefore requires further study to determine how Malaysian donors compare with those of developed countries, which report 1.4% in American Blood Transfusion Centres[2] and 1.1% in Japanese blood donors.[3]

The major transmission routes of HCV in Malaysia appear to be parenteral via sharing of intravenous needles, transfusion of blood and blood products and haemodialysis. The sexual route, which has been

implicated by Alter MS *et al*[4] as possibly constituting a transmission route for HCV must be further studied in selected populations in Malaysia. One study indicated evidence of past exposure to HCV in 10.8% of 37 male homosexuals and 9% of 100 commercial female sex workers, in whom no other obvious transmission routes could be identified such as intravenous drug abuse or blood transfusion (M Sinniah & BG Ooi, unpublished). The role of occupational exposure in HCV transmission requires further study although in one Kuala Lumpur based study of 123 laboratory and 53 haemodialysis unit personnel, none had HCV-antibodies, thereby indicating perhaps that strict adherence to hygienic clinical and laboratory practices may reduce HCV transmission.

No data is currently available to indicate the role of perinatal transmission of HCV in Malaysia.

HCV infection is serious because 40–50%[5] of all patients exposed to HCV develop chronic liver disease with its attendant high morbidity and mortality in contrast to Hepatitis B Virus infection where spontaneous recovery is the rule, with only 10% of patients developing chronic sequelae.

Some of the unanswered questions in Malaysia prior to 1990 were those relating to the proportion of chronic liver disease due to HCV, and the association if any of HCV with other known viral causes of liver disease such as HBV. In Malaysia, a major proportion (85%) of all forms of chronic liver disease including CAH, CPH, Cirrhosis and HCC were found to be associated with the Hepatitis B Virus. In three quarters of the cases, HBV was the sole viral agent detected; 10% were due to coinfecting HBV and HCV and only 1.5% of cases were due to HCV alone. Thus, although HCV plays an etiological role in the development of chronic liver disease in Malaysia, this role is perhaps less important than that of HBV in our population (M Sinniah & BG Ooi, unpublished).

The role HCV plays in the etiology of Acute Viral Hepatitis in Malaysia is yet to be established, although some unpublished data indicate that NANBV was responsible for 33.2% of 1157 consequetive acute viral hepatitis patients (Vijayamalar B, Sinniah M *et al*, unpublished) and HCV-AB was detected in 41.6% of 12 fulminant hepatitis cases (VJL How, Personal communciation).

A major issue to be addressed from the various prevalence data presented here, is whether to introduce routine HCV screening in Malaysian laboratories.

The advantages of HCV screening are obvious in terms of protection of organ transplant and blood transfusion recipients. However there are certain limitations that have to be considered. The test techniques currently available for routine HCV diagnosis are not for detection of the actual virus itself, but for the host's immune (antibody) response to one or more of the HCV viral proteins. The test undoubtedly has successfully identified both infective blood donors and recipients who developed the disease after transfusion. Its practical usefulness, however is somewhat limited because, firstly seroconversion may not occur for weeks or months after onset of acute hepatitis. Secondly, it is not clear if all HCV-AB positive individuals will be persistently infective or if the antibody confers immunity.

The currently available commercial HCV-AB tests are also rather expensive and in most instances require further confirmation by alternative, more expensive, specificity tests such as the Neutralisation Assay, Recombinant Immunoblot Assay (RIBA),[6] or Polymerase Chain Reaction (PCR),[7] which are currently available as research tools only in specialised reference laboratories.

Some of these issues should be resolved hopefully in future when new improved tests for both HCV-antigen and antibodies become available at affordable prices.

## References


1 Choo QL, Kuo G, Weiner AJ, Overby LR, Bradley DW, Houghton M. Isolation of a cDNA bome non-A non-B viral hepatitis genome. Science 1989; 244 : 359–62.

2. Stevens CE, Taylor PE, Pindyck J, *et al.* Epidemiology of Hepatitis C Virus – A preliminary study in volunteer blood donors. JAMA 1990; 263 : 49–53.

3. Nishimura Y, Yamaguchi K, Williams NP, Takatsuki K. Antibodies to Hepatitis C Virus in Japanese blood donors and in hospital personnel. Transfusion 1990; 30 : 667–8.

4. Alter MJ, Coleman PJ, Alexandar WJ, *et al.* Importance of heterosexual activity in the transmission of hepatitis B and non-A non-B hepatitis. JAMA 1989; 262 : 1201–5.

5. Cuthbert JA. Southwestern Internal Medicine Conference: Hepatitis C. Am J Med Sci 1990; 229(5) : 346–55.

6. Weiner AJ, Quan S, DiNello R, Van Boven JJP, Winkle I *et al.* Confirmation of Hepatitis C Virus infection by new four-antigen recombinant immunoblot assay. Lancet 1991; 337 : 317–9.

7. Cristiano K, Di Biseglie AM, Hoofnagle JH, Feinstone SM. Hepatitis C viral RNA in serum of patients with chronic non-A, non-B hepatitis: Detection by the polymerase chain reaction using multiple primer sets. Hepatology 1991; 14 : 51–5.