Local Epidemic of Non-A Non-B Hepatitis in Hyderabad

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Abstract
Fifty cases with acute, self-limiting hepatitis, which occurred in a local area in Hyderabad during the period March 1984 to May 1984, were studied clinically and serologically for hepatitis A and B markers. The results showed that this was a localised epidemic of non-A non-B hepatitis.

Key words: Non-A non-B hepatitis, epidemic.

Introduction
Non-A non-B (NANB) hepatitis is a well-known clinical entity, but very little is known about the nature of the etiological agent(s), although one study believes that it might be a retrovirus.1 NANB is now considered to be the most common cause of post-transfusion hepatitis in western countries.2 Feeco-orally transmitted NANB infection is believed to be an important cause of sporadic and epidemic hepatitis in India.3-4 The diagnosis is made by exclusion of hepatitis due to A and B viruses.

We report here a small epidemic of viral hepatitis due to NANB infection in a local area of Hyderabad. Such data on small epidemics are not available.

Material and Methods
Lallanpura is a small area in the city of Hyderabad. About 70 cases with acute hepatitis occurred here between March 1984 and May 1984. Fifty of these cases, including 30 males and 20 females (45 adults, 5 children) were examined clinically and blood samples were collected. Serum bilirubin, transaminases and alkaline phosphatase were estimated at weekly intervals. HBSAg, IgM anti-HBc and IgM anti-HAV were studied using commercially available ELISA kits (Abbott Laboratories, North Chicago, Illinois, USA).

Results
The fifty cases had mild prodrome, moderate icterus and a mild clinical course lasting 2-3 weeks. The cases were followed up for 6-8 months, during which none developed chronic liver disease and there was no related death.

The mean serum bilirubin was 5.93 ± 4.28 mg/dl (range 1.89-18.8), SGPT 277.6 ± 185.9 units % (115-782) and serum alkaline phosphatase 10.2 ± 2.6 KA units (6-33.9).

HBSAg was positive in four cases and IgM anti-HBc in none. IgM anti-HAV was positive in two cases, both of them children below the age of 10 years. Excluding these two cases the remaining 48 cases were classified as having NANNB hepatitis.

Discussion
Of the 50 cases, we excluded recent virus A and B infection in 48 samples. The 4 HBSAg positive patients were chronic carriers since recent infection was ruled out by a negative IgM anti-HBc. Thus this was a small epidemic of NANB hepatitis. The source of infection appears to be fecal contamination of the common water supply because of a faulty drainage system. Most of the cases in this epidemic were adults while HAV is known to predominantly affect children.

Khuroo3 first reported a NANB epidemic in Srinagar (India), caused by fecal contamination of water, raising the possibility of a hepatitis virus distinct from post-transfusion hepatitis virus. Wong et al4 made a similar suggestion in a retrospective study on samples collected from the Delhi and Ahmedabad epidemics and from Pune.

Most of the epidemics in our country are now believed to be due to NANB virus.5-6 A localised school epidemic in Kerala6 was however due to the A virus.

Since earlier reports of epidemics involve large numbers with significant mortality and morbidity, we wish to emphasize that even small localised epidemics like the present one may be due to NANB virus.

References