

Hepatitis A epidemic in Kerala state (India) in 1980

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An epidemic of hepatitis which occurred in 1980 in a Primary School in Kerala (south India) was investigated and proved to be due to virus A infection. The source of infection was well water in the school campus. A total of 362 of 778 children and 14 of 55 teachers had clinically apparent illness. The onset of the illness was acute and the prodromal phase was short. The clinical course of the disease was mild and there was no mortality. All the patients improved within four weeks and the epidemic subsided within six weeks. Blood samples collected during phases of recovery and convalescence as well as from controls were tested for anti-HAV IgM and total serum IgM; 85 per cent of the children and 71 per cent adult patients were positive for anti-HAV IgM and total IgM levels were elevated significantly in all of them. None of the controls gave positive results for anti-HAV IgM and all had normal serum IgM levels. Thus, these serologic findings were diagnostic for virus A infection. This is perhaps the first etiologically proved epidemic of hepatitis A in India.

Viral hepatitis is an endemic disease in India and outbreaks of this condition in an epidemic form are also common^{1,2}. Hepatitis A virus has been incriminated as an etiologic agent for such epidemics of hepatitis, mainly because the transmission was through contaminated drinking water¹. Recent reports, however, have established that water-borne epidemics of viral hepatitis could be due to non-A non-B virus infection²⁻⁴ rather than of virus A infection, as had been believed earlier. It is now possible to study the etiology of viral hepatitis with the help of serologic markers for A and B infection and consequently to diagnose infection due to non-A non-B groups, by a process of elimination. In the present communication we report a confirmed epidemic of hepatitis A virus in India.

Material & Methods

In 1980 a primary school in one of the small towns near Cochin in Kerala (south India) had to be closed due to an epidemic of viral hepatitis. The Rajgarhia Liver Unit of the Department of Gastroenterology, All India Institute of Medical Sciences (AIIMS), New Delhi was requested to investigate this epidemic. The Unit made a field visit from October 25 to November 5, 1980, to collect epidemiologic data and also serum samples to study the markers of virus A and B.

Study area : Muvattupuzha is a small town about 45 km from Cochin with a population of 33,000. It has an uninterrupted central water supply, but no central sewage disposal system. Most of the

houses have deep underground tanks for sewage disposal. Waste water flows through open drains. According to local health authorities, hepatitis is an endemic disease in the town.

Nirmala Junior School, in which the epidemic was reported is located in the centre of the town. The school runs only primary classes up to the fourth standard for children between 4 and 10 yr of age. A total of 778 children had been registered at the time of this study. Most of the children belonged to middle class families. The school building is situated in the compound of Nirmala Bhavan which is a residential building for the sisters of the mission which runs the School. Fifty five sisters and five servants stay in the residential campus. Nirmala Bhavan and the School have a central water connection from the town. The campus also has a drinking water well. Nirmala Bhavan has an underground tank (with uncemented walls) for sewage disposal. The distance between the sewage tank and the well is 12 m. Only water from the central water supply of the town was used by the residents and school children for drinking purposes till the end of June 1980. However, subsequently, due to shortage of water in the town, the main supply was found to be insufficient, necessitating the use of water from the well in the first week of July 1980. The well water was not subjected to chlorination or any other form of treatment since its construction.

The School had been closed at the time the Surveying Team arrived on October 25, 1980. A list of all the children who were reported to be sick was made available to the Team. During the first three days, the Team visited the houses of those children situated within 15 km

radius from the town to examine the children, find out the nature and duration of their illness, collect the reports of investigations, if any, and to detect any fresh case in the house or, in the neighbourhood. Sixty five houses were visited during three days and 92 children were examined. Fifty two blood samples were collected from the convalescent children (with their informed consent). Four well known general practitioners and four Government Medical Officers, including two practitioners of the traditional system of medicine, who had treated the maximum number of children, were interviewed.

When the school reopened on November 1, 1980, a note was sent to all the parents whose children were absent on the first day, asking if the child was absent because of hepatitis. It was discovered that out of 68 children who were absent on the first day, only three were absent due to hepatitis. These three children were examined and their blood and stool samples were collected.

Fourteen sisters of the Mission who had hepatitis were examined and their blood samples were collected. Samples were also collected from 11 sisters who did not suffer from hepatitis and who had no past history of jaundice. The Team could not obtain permission to collect blood from the healthy children.

Investigations: Serum bilirubin and serum transaminases (SGOT, SGPT) were determined by standard methods. All the 80 samples were tested for HBsAg by enzyme-linked immunosorbent assay (Auszyme, Abbott Labs, USA). Anti-HBs was tested in 39 pediatric samples and 11 adult blood samples by radio-immunoassay (Ausab, Abbott Labs,

USA). Anti-HAV IgG by RIA (Havab, Abbott Labs, USA) was done in only nine children and seven adults. The test is not diagnostic of recent infection, and in India, about 90 per cent of those above 5 yr give positive results⁵ for anti-HAV IgG. Anti-HAV IgM was tested in all the samples by ELISA method of Moller and Mathiesen⁶. Five stool samples from three sick children collected within three days of the onset of clinical symptoms were screened for hepatitis A virus by ELISA and immune-electron microscopy (IEM).

Results

In the present epidemic we could prove that the well water was contaminated and served as the sole source of the infection, because of the following evidence : (i) the interval between the use of the well water and the spurt in the occurrence of hepatitis coincided with the incubation period of hepatitis A; (ii) the well was closed by September 10 and a sharp decline of cases occurred by November; (iii) chlorination and microbiologic examinations of the well water had not been carried out before consumption; (iv) the sewage dumping tank was within a distance of 12 m from the well and had no cement walls; (v) microbiologic examination of well water done later showed very high *Escherichia coli* count and the water was declared unfit for drinking purposes; and (vi) in families of the school children no other member (child or adult) developed hepatitis.

Of the 362 children and 14 adults who suffered from hepatitis, 346 children and all the 14 adults fell ill in the month of October 1980. The age distribution of the 95 affected children was as follows : 14

aged 4 yr, 12 aged 5 yr, 16 aged 6 yr, 12 aged 7, 14 aged 8, 19 aged 9 and 8 aged 10 yr. Thirteen patients were registered during September and only three new cases in the month of November 1980. Two patients reported in June and one each in July and August 1980, but these could not be attributed to the epidemic, as hepatitis is endemic in the town. The school was closed from 1st October, 1980.

There were no deaths due to this epidemic. Though the patients were not seen during the acute phase of illness, a retrospective analysis of the information collected from the doctors, school records and parents showed that the course of the disease was mild, and only a few patients required hospitalization. More than 90 per cent of the children and adults improved after 2-3 wk of the icteric phase. Onset of the illness was acute and the prodromal phase was short, lasting for only 3-4 days. Dark colour urine and fever were the most frequent symptoms followed by nausea, vomiting and anorexia. Mild icterus was present in 57 per cent children and 64 per cent adults. Liver was palpable in all the 95 children and 14 adults at the time of examination by the team. There was no splenomegaly.

Clinical diagnosis was established by physicians who had attended on the patients during the acute stage, on the following information : The patients belonged to Nirmala school—the locale where the outbreak of hepatitis occurred; the illness had an acute onset and short pre-icteric phase; the disease was characterised by jaundice and high coloured urine positive for bile salts and pigments; as well as hepatomegaly. Recovery occurred usually 2-3 wk after the onset of the icteric phase.

Serum bilirubin in the phase of convalescence phase ranged between 1.4-1.9 mg/dl in three patients who had hepatitis in the second week of October and between 1.2-1.6 mg/dl in three new patients. Serum enzyme (SGOT/SGPT) levels were above 120 Karmen Units in the three new patients.

HBsAg was positive in only two children. Eight of the 39 children and one of 10 adult patients were anti-HBs positive. Anti-HAV IgG was found in all the nine children and seven adults tested. Anti-HAV IgM was demonstrated in 85 per cent children and 71.4 per cent of adult patients but in none of the 11 adult controls (Table). Serum IgM levels were elevated in the sera of all patients and were within the normal range in the 11 adult controls. Virus A was not detected in any of the five stool samples collected from the children.

Discussion

The outbreak reported here is perhaps the first confirmed epidemic of type A virus hepatitis in India. In a short period of 2 months, 47 per cent of children in a

school suffered from an illness which had the characteristic features of viral hepatitis. The etiological diagnosis of hepatitis A was considered on the basis of hepatitis A specific IgM antibody positivity and elevation of total IgM levels in the patients.

The criteria suggested⁷ as diagnostic of hepatitis A are : detection of the virus particles in the stool; demonstration of rising titres of anti-HAV; and demonstration of anti-HAV IgM. The diagnosis of type A virus infection can be established by any one of these criteria. Demonstration of HAV-specific IgM antibody has been used as a simple and dependable test. Viral particles may be absent even in definite cases of type A virus infection as excretion of virus in stool is very short-lived and occurs only in the early phase of infection⁸.

At the past epidemics of viral hepatitis reported in India were thought to be due to virus A on the basis of clinical, biochemical and epidemiologic studies and the absence of HBsAg. After the advent of diagnostic techniques for virus A, a study of the stored sera samples² as well as recent epidemics^{3,4} have established that water-borne epidemics of hepatitis

Table. Serologic tests in children and adult patients with hepatitis and adult controls

Test	Children		Adults		Adult controls*	
	No. tested	No. positive	No. tested	No. positive	No. tested	No. positive
HBsAg	55	2 (4)	14	0	11	0
Anti-HBs	39	8 (20.5)	10	1 (10)	11	1 (10)
Anti HAV IgM	55	47 (85.5)	14	10 (71.4)	11	0

*There were no controls among the children; Figures in parentheses indicate the percentage values

in India were due to non-A non-B (NANB) infection. Of 254 sera samples from patients of epidemic NANB hepatitis tested by us^{4,5}, only 19 (7.5%) were positive for anti-HAV IgM. The present report seems to be the only one in which type A virus etiology was established. Clinical and epidemiologic characteristics of this epidemic are quite different from NANB epidemics reported from India^{3,4}. NANB usually affects all age groups, although adults suffer in a high proportion. Onset of illness is usually insidious, the clinical course is quite stormy and mortality high. Clinical and epidemiological characteristics can help to suggest type A or NANB, virus etiology of water-borne epidemics of hepatitis. If future studies could confirm the epidemiologic and clinical characteristics of type A virus hepatitis epidemic reported here, it would be possible to predict with reasonable certainty the etiology of epidemic hepatitis and initiate appropriate control measures even in the absence of or while awaiting serological reports. Three recent reports⁹⁻¹¹ of type A hepatitis confirmed by serological studies have also highlighted short and acute pre-icteric phase, benign course of the illness and the absence of mortality in these episodes, two of which were due to food-borne infection^{9,10}. In the epidemic reported in a country club by Osterholm *et al*⁹, there was no special predilection for children. The other food-borne epidemic occurred in the navy recruits. The third epidemic was in a school for the mentally retarded in which none of the adults were reported to have suffered with hepatitis¹¹. In the present study on 55 adults and 778 children who were exposed to contaminated well water, despite the use of the same source of contaminated water by all the adults, only 25 per

cent developed icteric hepatitis, as against 47 per cent of the children. This suggests a predilection of the children to virus A hepatitis which is supported by our earlier study on sporadic viral hepatitis⁵. A high, incidence rate of type A hepatitis in Indian children; considered in the light of the fact that 95 per cent of the population had anti-HAV by the age of five years presents interesting possibilities which need further exploration.

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