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OUTBREAK OF ACUTE VIRAL HEPATITIS DUE TO HEPATITIS E VIRUS IN HYDERABAD

*P Sarguna, A Rao, KN Sudha Ramana

Abstract

Purpose: A waterborne outbreak of viral hepatitis occurred in the old city of Hyderabad from March through August 2005. An attempt was made to study the outbreak clinically, serologically, and etiologically. Methods: Five hundred and forty-six clinically and biochemically documented cases were screened for the hepatotropic viral markers, hepatitis A, B, C, and E by the ELISA method. Their demographic characteristics and outcomes were analyzed. Point source contamination of the water supply with sewerage was identified. Result: Our data confirms hepatitis E as the major cause of the outbreak (78.57%). Occasionally, mixed infection of HEV-HAV (5.31%) or HEV-HBV (0.91%) was detected in the present series of acute viral hepatitis. Conclusions: HEV was confirmed as the major etiological agent in this outbreak that was transmitted by contaminated drinking water. The study highlights the importance of screening for both enterically transmitted hepatotropic viral markers as well as the parenterally transmitted hepatotropic viral markers during outbreaks of acute viral hepatitis.

Key words: Acute viral hepatitis, epidemic, Hepatitis E virus, mixed infection

Hepatitis E virus (HEV) is the agent largely responsible for epidemic as well as sporadic hepatitis in the developing countries.1-4 The virus is transmitted by the feco-oral route, often through contaminated water5,6 and affects travelers from developed countries who have been to endemic areas.7 Primarily a self-limiting disease, it produces chronic sequelae. A mortality of 20-30% has been reported, particularly in pregnant woman who contract the disease in the third trimester.8,9 HEV has been implicated as an important etiological agent for sporadic fulminant hepatic failure (FHF) in developing countries.10

A waterborne outbreak of viral hepatitis affected a large population in the old city of Hyderabad from March 2005 through August 2005. The water distribution system in the old city of Hyderabad was examined for any recent changes in the supply system. Although acute viral hepatitis (AVH) could be differentiated into enterically transmitted virus or parenterally transmitted virus based on the mode of presentation, confirmation of etiology needs to be determined serologically. It has been well established that in endemic areas infection with HEV can be seen in association with infection by other hepatotropic viruses, such as hepatitis A virus (HAV), hepatitis B virus (HBV), and hepatitis C virus (HCV);11 this may lead to FHF worsening the already affected liver cells. In view of the above facts, this study was undertaken to determine the etiology of the outbreak and the incidence of mixed hepatotropic viral infections (HAV, HBV, HCV, and HEV) among the individuals who were affected with hepatitis E in Hyderabad.

Materials and Methods

Hyderabad city is divided into seven circles (Fig. 1). Water supply for the population of 40,36,347 is maintained by the Hyderabad metropolitan water supply and sewerage board (HMWS and SB). In March 2005, there was a breach in the sewerage pipelines located in circles 1 and 2, which contaminated the drinking water supply. The layout of the water and sewerage distribution lines in circles 1 and 2, obtained from HMWS and SB, revealed some unsafe pipelines which passed through open drains—a possible source of the infection (Fig. 2). The local inhabitants had noticed that the tap water was dirty. Tap water samples (117) were analyzed

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Figure 1: Incidence of acute hepatitis in seven circles of Hyderabad; March–August 2005

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for residual chlorine levels and fecal contamination as per the standard protocol.12

Following this event, 1455 patients with clinically suspected AVH from these localities reported to Sir Ronald Ross Institute of Tropical and Communicable Diseases (SRRITCD), Hyderabad, A.P., from March 2005 through August 2005. For the purposes of this study, a case of acute hepatitis was defined as acute illness with jaundice, dark urine, loss of appetite, and right hypochondrial tenderness. Exclusion criteria included history of chronic hepatitis, exposure to hepatotoxic drugs or chemicals, and chronic alcohol use. The study population included 546 consecutive patients who met the case definition of AVH and had biochemical evidence of liver function derangement. These individuals belonged to both genders, with the ages ranging from 10-70 years. The above patients reported to the hospital at different stages of illness following exposure to the contaminated water. The period of reporting varied from 1 day to 2 months after the onset of illness.

Urine was tested for the presence of bile pigments and bile salts. The liver function tests included estimation of serum bilirubin, serum alkaline phosphatase, and serum alanine aminotransferase (ALT).

Blood samples were collected from all the patients on the day of admission to the hospital. Serum was separated and preserved at −20°C until it was tested. Sera were screened within 4 days of collection for the hepatotropic viral markers. Anti-HAV IgM (ImmunoVision) was assessed using HAV-specific immunodominant recombinant antigens by capture enzyme immunoassay; anti-HEV IgM (ImmunoVision), using ORF2 as well as ORF3 recombinant antigens by capture enzyme immunoassay; HBsAg was screened for by using third-generation enzyme immunoassay (PATHOZYME); and anti-HCV antibody was detected by third-generation enzyme immunoassay (General Biologicals Corp.) using synthetic HCV peptides, core and NS4 antigens, and recombinant antigens NS3 and NS5 by sandwich assay. All tests were carried out using procedures as per the manufacturers’ instructions.

**Results**

The epidemic was short-lived; the majority of the cases occurred during the third week of March and between the last week of April and the first week of May. A high incidence was observed in circle 1 and circle 2 around the old city of Hyderabad.

Thirty-four of the 117 randomly collected samples of water from various taps in and around the old city during the outbreak revealed evidence of inadequate chlorination (<0.1 ppm) and presence of coliform organisms (more than 20/dL). All the 34 contaminated water samples were from the affected area of the old city of Hyderabad.

Among the 546 patients studied, males outnumbered females with a ratio of 2.3:1. The most affected age-group was that between 15 and 25 years, with an incidence of 73% in males. We found a relative sparing of children below 10 years of age. Of the 13 pregnant women with AVH, 3 (23%) women who were in the third trimester had a fatal outcome. No untoward effects were observed in the remaining 10 HEV infected pregnant women during their follow-up.

The clinical profile of the patients is depicted in Table 1. Of the 546 patients, 119 (21.79%) gave a history of usage of herbal medicines for treatment of jaundice after the onset of the present illness. All the 546 patients had abnormal liver function tests suggestive of acute hepatitis (Table 2); the tests were repeated at weekly intervals as it was found to be useful in predicting the prognosis. There was a moderate rise in serum alkaline phosphatase levels, and a relative cholestatic picture was observed in 50.36% (275/546) patients. The average duration of symptoms was 11 to 15 days. The nature of the symptoms was similar in men, women, and children.

Seroanalysis of 546 serum samples from the outbreak (Table 3) revealed the presence of at least one seromarker of hepatitis in 534 (98 %) cases.

Anti-HEV IgM positivity was significantly higher among individuals >15 years of age than in those >15 years (P < 0.001). In contrast, anti-HAV IgM positivity was statistically significant in subjects <15 years old, with a P value < 0.001 (Fig. 3).

**Discussion**

Contaminated water, as a source of infection, is intimately related to outbreaks of hepatitis due to HEV; this was the case in the old city of Hyderabad, similar to several other outbreaks in other places. Cases were clustered around those water supply lines that were found to be crossing the open drains. Overcrowding and poor sanitation and living conditions contributed to the rapid spread of the outbreak.

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**Figure 2:** Incidence of acute hepatitis by block in the circles 1 and 2 of the city of Hyderabad; March–August 2005

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Diagnostic tests with high sensitivity and specificity, such as peptide-based ELISA, have enabled an extensive seroanalysis of the epidemic of hepatitis. Analysis of viral makers revealed isolated viral infection in 91.57% cases; coinfection with multiple viruses was detected in 6.22% of AVH patients. The majority of single infections were due to HEV (78.57%), followed by HAV (9.70%). The presence of HBV in 3.29% probably reflects sporadic cases in the community. The age distribution of cases was similar to that described in previous epidemics of hepatitis due to HEV: adults in the age-group of 15–25 years being predominantly affected. Children below 10 years were spared, as has been also observed in earlier epidemics of NANB hepatitis.3,9 This could be because anicteric hepatitis or subclinical infection is common in children under 9 years of age in endemic hepatitis.13 An alternative explanation could be that HEV is maintained in the community as a sporadic infection; thus, HEV is acquired early in life, making infants and children immune to another attack.1,14-16 However, seroanalysis of the epidemic of hepatitis revealed anti-HEV IgM in a few children.17

Although HEV and HAV have a common route of transmission, HAV infection was infrequent, being the predominant form in individuals less than 15 years (81.12%). Hepatitis due to HAV is considered a childhood disease in developing countries. Association of HAV infection with fulminant disease and relapsing hepatitis is frequently observed in adults over the age of 40 years. The case fatality

Table 1: Clinical profile in acute viral hepatitis (n = 546)

<table>
<thead>
<tr>
<th>Clinical manifestation</th>
<th>HEV n = 429 No. (%)</th>
<th>HAV n = 53 No. (%)</th>
<th>HEV-HAV n = 29 No. (%)</th>
<th>HEV-HBV n = 5 No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jaundice</td>
<td>429 (100)</td>
<td>53 (100)</td>
<td>29 (100)</td>
<td>5 (100)</td>
</tr>
<tr>
<td>Pyrexia</td>
<td>329 (76.68)</td>
<td>43 (81.13)</td>
<td>17 (58.62)</td>
<td>4 (80)</td>
</tr>
<tr>
<td>Malaise</td>
<td>195 (45.45)</td>
<td>18 (34)</td>
<td>5 (17.24)</td>
<td>2 (40)</td>
</tr>
<tr>
<td>Loss of appetite</td>
<td>195 (45.45)</td>
<td>18 (34)</td>
<td>8 (27.58)</td>
<td>4 (80)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>212 (49)</td>
<td>28 (52.83)</td>
<td>14 (48.27)</td>
<td>2 (40)</td>
</tr>
<tr>
<td>High coloured urine</td>
<td>429 (100)</td>
<td>31 (58.49)</td>
<td>29 (100)</td>
<td>5 (100)</td>
</tr>
<tr>
<td>Pale coloured stools</td>
<td>60 (14)</td>
<td>6 (11.32)</td>
<td>1 (3.44)</td>
<td>-</td>
</tr>
<tr>
<td>Pain right hypochondrium</td>
<td>333 (77.62)</td>
<td>28 (52.83)</td>
<td>14 (48.27)</td>
<td>-</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>220 (51.28)</td>
<td>31 (58.49)</td>
<td>2 (6.89)</td>
<td>-</td>
</tr>
<tr>
<td>Itching</td>
<td>255 (59.44)</td>
<td>22 (41.35)</td>
<td>8 (27.58)</td>
<td>-</td>
</tr>
</tbody>
</table>

Table 2: Liver function tests in acute viral hepatitis (n = 546)

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Normal values</th>
<th>HEV n = 429 No. (%)</th>
<th>HAV n = 53 No. (%)</th>
<th>HEV-HAV n = 29 No. (%)</th>
<th>HEV-HBV n = 5 No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum bilirubin</td>
<td>0.2-1.2 mg %</td>
<td>429 (100)</td>
<td>53 (100)</td>
<td>29 (100)</td>
<td>5 (100)</td>
</tr>
<tr>
<td>Serum alkaline phosphatase</td>
<td>100-280 U/L</td>
<td>417 (97)</td>
<td>49 (92.45)</td>
<td>29 (100)</td>
<td>5 (100)</td>
</tr>
<tr>
<td>Serum alanine amino transferase (ALT)</td>
<td>40 U/L</td>
<td>429 (100)</td>
<td>49 (92.45)</td>
<td>29 (100)</td>
<td>5 (100)</td>
</tr>
</tbody>
</table>

Table 3: Serological profile in AVH (n = 546)

<table>
<thead>
<tr>
<th>Viral marker</th>
<th>No. of positive</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-HAV IgM</td>
<td>53</td>
<td>9.70</td>
</tr>
<tr>
<td>Anti-HEV IgM</td>
<td>429</td>
<td>78.57</td>
</tr>
<tr>
<td>HBsAg</td>
<td>18</td>
<td>3.29</td>
</tr>
<tr>
<td>HEV and HAV</td>
<td>29</td>
<td>5.31</td>
</tr>
<tr>
<td>HEV and HBV</td>
<td>5</td>
<td>0.91</td>
</tr>
<tr>
<td>Total</td>
<td>534</td>
<td>97.80</td>
</tr>
</tbody>
</table>

Figure 3: Age and gender distribution in AVH (n = 546)
rate is quoted as being less than 1.5% in icteric cases. Prompt hospitalization following the onset of illness and complete rest was important in inducing recovery within a period of 2-6 weeks in the majority of the cases. We did not observe any sequelae suggestive of chronicity. There was a fatal outcome in 23% of pregnant women (all in the third trimester) due to isolated HEV infection. Pregnant women are at higher risk of a fulminant course and the case fatality rate increases with the length of the pregnancy period, as was evident in the present study.

Occasionally dual infections with HAV and HBV in acute HEV patients have been observed in the present series of AVH, affecting adults predominantly the adult age 86.20% and 100%, respectively. All the HAV and HEV dual infections were probably coinfections as they have a common route of transmission. There was no essential difference in the clinical behavior or biochemical profile between the single and multiple infection groups of AVH patients. Recovery was uneventful, without any sequelae. Coinfections with HAV and HEV have been implicated as the single largest etiologic subgroup causing AHF and sporadic fulminant hepatitis in children in North India. Yet others have reported mixed infections of HEV and HAV in a pediatric population in association with FHF (22.5%). This contrasts with our clinical experience of an uneventful course in AVH due to HAV–HEV coinfection. The discrepancy could be because their studies comprised proven cases of acute liver failure and FHF, whereas our study included cases selected on the basis of clinical criteria and deranged liver function during the waterborne outbreak of viral hepatitis.

In our study, dual infection of HEV and HBV was observed in 0.91% of acute hepatitis in adults without any sequelae, while a group of researchers observed dual infection of HEV and HBV in 5.4% of patients of acute hepatitis. We did not observe any sequelae suggestive of chronicity. There was a fatal outcome in 23% of pregnant women (all in the third trimester) due to isolated HEV infection. Pregnant women are at higher risk of a fulminant course and the case fatality rate increases with the length of the pregnancy period.

To conclude, HEV and HAV coexist during epidemic hepatitis but with different peak age positivity. Gross contamination of drinking water supplies results in outbreaks. In endemic areas, infection with HEV may be seen in association with other hepatotropic viruses (HAV, HBV, and HCV) as observed in the present study. These mixed infections could worsen the prognosis in patients with preexisting impaired liver function and may lead to AHF or FHF. HEV positivity is considered a strong marker for multiple infection; hence, investigations during HAV/HEV outbreaks should also include screening for HBV and HCV to identify mixed infections and thereby enable better management and prevention of sequelae.

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References


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