Outbreak of Viral Hepatitis E in a Regimental Training Centre

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Abstract

Background : An outbreak of viral hepatitis occurred in a regimental centre with 265 cases occurring during a 3 months period. **Methods :** 190 serum samples were tested for IgM antibodies against viral hepatitis E by Enzyme Immuno Assay (EIA) and for antibodies against Hepatitis A and Hepatitis B viruses. Epidemiological investigation comprised review of surveillance data, filling up epidemiological case sheet, sanitary survey, inspection of water supplies and bacteriological examination of water for coliforms.

Result : 97.4% of the serum samples were positive for IgM antibodies against Hepatitis E virus. Two leaks were detected in water pipelines, which were passing through contaminated areas around improperly functioning septic tanks and soak pits. The attack rate among recruits being supplied water through leaking pipelines was 11.1% whereas it was 2.89% in those not directly exposed. This difference was statistically significant ($p < 0.001$). Bacteriological examination of water showed a high coliform count.

Conclusion : The outbreak of viral hepatitis E occurred due to sewage contamination of water pipelines.

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Key Words: Hepatitis E; Outbreak

Introduction

Most outbreaks of enterically transmitted hepatitis in young adults in India have been due to hepatitis E virus infection [1-8]. Fecal contamination of drinking water is the commonest cause of such outbreaks. The causative agent hepatitis 'E' virus (HEV), based on its genomic characterization, has been classified in the family *Caliciviridae*. The present study pertains to an outbreak of enterically transmitted HEV in a regimental training center in central India. This study comprises of 265 viral hepatitis cases, which were reported during 3 months i.e. from end March to end June 2002. The method of investigation of the outbreak and relevant findings are presented.

Material and Methods

Clinical case description: Acute illness compatible with the following clinical description – jaundice, dark urine, anorexia, malaise, extreme fatigue and right upper abdominal pain. There was increase of Alanine Aminotransferase (ALT) > 8 times and serum bilirubin > 2mg%.

Case classification: (i) Suspect : A case that is compatible with clinical description (ii) Confirmed case: A suspect case that is laboratory confirmed. The outbreak was described in terms of time, person and place.

Laboratory investigations: Of the total 265 clinically suspected cases of viral hepatitis, 190(71.7%) serum samples were transported in cold chain from the Regimental Training Centre (Central India) to the Department of Microbiology, AFMC, Pune. The samples were tested for the IgM antibodies against viral hepatitis E by Enzyme Immuno Assay (EIA) [kit used was manufactured by Giuliana Diagnosticci S.e.l. Via Galileo Galilei, 38-20096, Seggiano di Pioltello (Milan), Lot No AD018AC]. The serum samples were also tested for antibodies against Hepatitis A virus (HAV) and Hepatitis B virus (HBV) [kit manufactured by Biochem immunosystems (Italy)]. In addition, 151(57%) serum samples were also sent to National Institute of Communicable Diseases (NICD), New Delhi for screening for anti-HEV and anti-HAV IgM antibodies. Biochemical parameters such as serum bilirubin, alanine aminotransaminase (ALT), urine for urobilinogen and bilirubin were also assessed in all the cases at the local service hospital to establish diagnosis of viral hepatitis.

Epidemiological investigation: Detailed information was recorded on epidemiological case sheet such as date of joining the recruiting centre, date of onset of signs and symptoms, history of movement and other relevant data. Active case findings by surveys among recruits was carried out from 25 April 02. Persons who were ill and met clinical description and classification were included [9].

A sanitary survey was carried out to detect the sources of

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water contamination and to study methods of sewage disposal. The available blue print of the water supply pipelines was examined. Place distribution of the cases along the distribution of water supply was mapped out. Record of bacteriological examination of water for coliforms was reviewed.

Results

Clinical profile: The signs and symptoms included pain abdomen (38%), anorexia(45%), malaise(32%), fever(39%), nausea(62%), vomiting(74%), icterus(97%) and yellow discoloration of urine(95%). Few had arthralgia (2%) and myalgia (5%). Cases detected by active survey (about 1/3rd of the total cases) had milder symptoms. The disease followed an uneventful self-limiting course with average length of stay in hospital of about 2 weeks. Only one patient who had reported late with serum bilirubin level of over 18 mg/dL developed features of hepatic failure and was evacuated to a tertiary care service hospital. There were no fatalities.

Laboratory findings: Of the 190 serum samples tested at AFMC, Pune, 185(97.4%) were positive for IgM antibodies for Hepatitis E virus. None of the cases demonstrated IgM antibodies against Hepatitis A and B virus. Of the 151 samples sent to NICD, 142(94.04%) were positive for IgM against HEV and none had IgM against hepatitis A virus.

Epidemiological findings: There were a total of 265 cases of viral hepatitis among recruits and troops in the regimental centre within a span of two and a half months with an attack rate of 8.14%. Two leaks were detected in water pipelines, which were passing through sewage-contaminated areas around improperly functioning septic tanks and soak pits. The first leak was detected and repaired on 06 April 2002 and the second leak repaired on 15 April 2002. A cohort of 132 recruits who reported to the regimental centre after 15 April 2002, i.e. after repair of the second leaking pipeline was also followed for a period for two months each (maximum incubation period of Hepatitis E).

There was no unusual incidence of cases among the troops and families residing outside the regimental center. From records it was seen that only 12 cases of viral hepatitis occurred in the station in 2000 and 6 in 2001. Table 1 shows attack rates in recruits/troops who were exposed to leaking pipelines compared to those not exposed. The relative risk for those exposed against those non-exposed was 3.92(95% CI of RR 2.73, 5.63). The difference in attack rates between the two groups was statistically significant ($p < 0.0001$).

The regimental centre was supplied water through several tubewells and a network of pipelines. The water supply was intermittent with negative pressure leading to suction developing in the two decade old corroded pipes. The age and rank distribution is shown in Tables 2 and 3 respectively.

Time distribution: All cases were admitted to local service hospital between 24 March 2002 to 23 June 2002 (Table 4). There were no cases after third week of June 2002. A large number of cases were detected in end April and first and second weeks of May 2002 due to active surveillance. No case of viral hepatitis occurred among the cohort of 132

recruits who reported to the regimental center after 15 April 2002(after rectification of sewage contamination of leaking pipelines).

Bacteriological examination of water in first week of April 2002 (just after onset of the outbreak), showed a high coliform count at five different consumer ends in the affected cookhouses, confirming fecal contamination of drinking water. Prior to this period record of bacteriological examination of water was not available.

Discussion

In the present outbreak, 185 (97.4%) of the 190 serum samples showed IgM antibodies against HEV establishing it as cause of outbreak beyond reasonable doubt [9].

Hepatitis E was not recognized as a distinct clinical entity until 1980, when sensitive and specific tests for antibodies to Hepatitis A virus (anti-HAV) were first applied to study epidemic waterborne hepatitis in India [10,11]. The first direct evidence for the existence of additional waterborne hepatitis agent was reported in 1983 by Balayan et al who described successful fecal-oral transmission of hepatitis to a volunteer from patients

Table 1
Attack rates in recruits/troops consuming water from defective pipelines compared to those not exposed to leaking pipes

Source of water	Number affected	No. not affected	Total
Leaking pipes	233 (11.01%)	1884 (88.99%)	2117 (100%)
Normal pipes	32 (2.81%)	1107 (97.19%)	1139 (100%)
Total	265 (8.14%)	2991 (91.86%)	3256 (100%)

RR = 3.92, (95% CI = 2.73 < RR < 5.63), Chi Sq = 52.72, df = 1, P < 0.0001

Table 2
Age distribution of viral hepatitis

Age group (years)	Number of cases (%)
17-20	195 (73.58)
21-25	40 (15.09)
26-30	11 (4.15)
Above 30	19 (7.16)
Total	265 (100)

Table 3
Rankwise distribution of cases

Rank	Number of cases (%)
Recruit	184 (69.43)
Sep	53 (20.00)
L/Nk	7 (2.64)
Nk	9 (3.39)
Hav	11 (4.15)
JCO	1 (0.37)
Officers	Nil
Total	265 (100)

Table 4**Time distribution of hospital admission**

Week	Week ending	No. admitted	Cumulative	Remarks
First	24 Mar 02	1	1	Index case
Second	31 Mar 02	3	4	
Third	07 Apr 02	16	20	
Fourth	14 Apr 02	26	46	Leaking pipes repaired on 15 Apr 02
Fifth	21 Apr 02	9	55	
Sixth	28 Apr 02	12	67	Active case detection started from 25 Apr 02
Seventh	05 May 02	18	85	
Eighth	12 May 02	61	146	
Ninth	19 May 02	63	209	
Tenth	26 May 02	20	229	Admitted in convalescent phase
Eleventh	02 Jun 02	10	239	Admitted in convalescent phase
Twelfth	09 Jun 02	3	242	Admitted in convalescent phase
Thirteenth	16 Jun 02	02	244	Admitted in convalescent phase
Fourteenth	23 Jun 02	01	245	Admitted in convalescent phase
Fifteenth	30 Jun 02	Nil		
Others*		20	265	

*recruits posted out to various units (between Mar-May 2002), and information of their having contacted viral hepatitis obtained from their new units.

with Hepatitis A like disease in Tashkent, Uzbekistan [12]. Virus particles 27 to 30 nm in diameter were detected by immune electron microscopy (IEM) in faeces collected from the volunteer between 28 and 45 days after exposure. This form of Non-A, Non-B hepatitis came to be known as epidemic or enterically transmitted Non-A, Non-B hepatitis (ENANB)[13]. The complete sequence of the viral genome is now known and the agent was renamed Hepatitis E virus (HEV)[14]. It is a single-strand, positive sense RNA virus, approximately 32 nm in diameter [15,16]. HEV lacks an envelope making it stable in bile and thus, transmissible by ingestion of fecally contaminated water. HEV is resistant to pH extremes of the gastrointestinal tract, although water chlorination decreases infectivity [17].

Faecal contamination of water supplies is a frequent finding in evaluation of HEV outbreaks [2,4,5,6,7, and 17]. The association in present outbreak of higher attack rates of viral hepatitis in those exposed to leaking water pipelines passing adjacent to sewage overflowing septic tanks/soak pits strongly indicates that the outbreak was due to sewage contamination of water supplies, is confirmed by high coliform counts at consumer end and is further reinforced by the fact that no case occurred among the cohort after the pipelines were repaired.

The attack rates of viral hepatitis E in previous outbreaks have varied between 1.9% to 17% [2,4,5]. In the present outbreak the overall attack rate was 8.14% with the recruits exposed to sewage contaminated water showing a higher rate of 11.1% as compared to 2.89% in those not exposed water.

Outbreaks of HEV mostly affect young adults. This

has been explained by less frequent transmission of HEV and postponement of infection to later years, when it is clinically apparent[17]. Although HEV infection has been demonstrated in children [18] in many studies, the seroprevalence is low until the third decade of life [19,20,21,22]. On the other hand, others have demonstrated higher seroprevalence rates of HEV infection in children [23]. Both views i.e. lack of infection in childhood and infection in childhood not producing durable immunity may explain the childhood immunity being not durable in our country.

Person to person HEV transmission is less efficient than Hepatitis A virus, as indicated by low secondary attack rates [17]. In the present outbreak no case occurred among the cohort after the sewage-water contamination was eliminated.

Humans are the only proven reservoir [17]. However, a highly related virus has been found in swine [24,25]. Moreover, antibody that cross-reacts with HEV antigens has been detected in swine, rats and other animals [26].

Senior medical authorities should advocate replacement of vintage water distribution and sewage systems in a phased manner. The Station Health Committee in each station should periodically review the progress in improvement of water distribution and sewage disposal. Medical authorities should also maintain surveillance for acute diarrhoea, typhoid and jaundice and also monitor the water quality, as at present there is no vaccine available for Hepatitis E [8]. An increase in the incidence of diarrhoea and/or typhoid and poor water quality could indicate impending epidemic of viral hepatitis. Surveillance should include daily check for free chlorine at the consumer end. Any absence of

free chlorine should be taken as indication of fecal contamination until unless proved otherwise and investigated thoroughly. Coliform counts on water from different consumer ends should be carried out periodically.

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