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RESEARCH ARTICLE

CLINICAL PROFILE OF OUTBREAK OF VIRAL HEPATITIS AND ITS OUTCOME AT TERTIARY CARE CENTRE IN JAIPUR

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ABSTRACT

Introduction and Objectives: Hepatitis E virus causes acute viral hepatitis. Being waterborne infection, Monsoon season is associated with high risk of epidemics Monsoon season is associated with high risk of epidemics.Our study aimed to evaluate the clinical profile and predictors of severity in patients of viral hepatitis epidemic in Jaipur during May 2014 to Jan 2015. Cases evaluated in the study were both Index cases and referred cases from epidemic area, for worsening of jaundice and other complications.

Material and Method*:* This study was conducted on 64 patients presenting with acute hepatitis in Metro MAS hospital over period of 9 months from May 2014 to Jan 2015. A detailed history of each patient was recorded, including travel history, blood transfusion, food and water intake from outside sources. The diagnosis of acute viral hepatitis was based on accepted clinical, biochemical and serological criteria.

Results: In present study, viral hepatitis was predominantly seen in middle age. Males (81%) were predominantly affected. Out of female (19%) patients, 2 were pregnant. Maximum patients presented with jaundice (90%), followed by nausea and vomiting (60%), fever (30%) and abdominal pain (13%). Most common sign was icterus followed by tender hepatomegaly (28%). Virology study showed 40 cases positive for HEV, 3 cases for HBV and 5 cases were positive for HAV. In 3 cases, RFT was raised above 2mg/dl out of which 1 required hemodialyses. 8 cases were found with deranged prothrombin time and 6 received FFP in view of active bleeding. No Mortality was seen in our study.

Conclusion: Our study suggests that the outbreaks in Jaipur district were due to Hepatitis E virus. Jaundice was the most common presenting feature followed by vomiting; fever and pain.Early presentation and judicious treatment can prevent mortality. Majority of cases were cured with supportive treatment.

INTRODUCTION

Hepatitis E was first recognised during an epidemic of hepatitis in Kashmir valley in 1978[1]. It is an enterically transmitted disease that spreads through faecal contamination of drinking water. It occurs both in the form of epidemics as well as sporadic infection in developing countries[2-5]. According to the South East Asia Regional office of the World Health Organization (WHO), hepatitis E is widespread in developing countries, accounting for up to 90 per cent of all sporadic cases of acute viral hepatitis[6]. Hepatitis E virus affects young to middle aged adults and causes high mortality in pregnant women, 20-30 per cent as compared to 0.2-1 per cent in general population[7]. It has been implicated as an important aetiological agent for sporadic fulminant hepatic failure (FHF) in developing countries[8].

Aetiology of acute viral hepatitis (AVH) cannot be differentiated on the basis of mode of presentation; confirmation is done serologically. Hepatitis E virus is an important hepatotropic virus that causes acute viral hepatitis. Most of the studies on prevalence of hepatitis E virus are from epidemic setting and some from sporadic setting.

Hepatitis E virus (HEV) causes a self-limiting viral infection that is transmitted by the faeco-oral route, primarily through the consumption of contaminated food and water. It can occur as both epidemic (Arankalle *et al*., 1994) and sporadic cases (Arankalle *et al*., 1993) in developing countries, with sporadic HEV infection occurring with increased frequency in both developing and developed countries (Aggarwal, 2011). Interestingly, this virus results in 20-30 % mortality among pregnant women (Kumar *et al*., 2004) and has been implicated as an important aetiological agent for sporadic fulminant hepatic failure in developing countries (Nanda *et al*., 1994).

Most documented studies on the incidence of acute sporadic HEV infection to date have been from the northern, western and south central parts of India (Amarapurkar *et al*., 2008; Chadha *et al*., 2003; Chandra *et al*., 2012b; Das *et al*., 2000;

Jain *et al*., 2013; Kaur *et al*., 2003; Khuroo *et al*., 1983; Kumar *et al*., 2007; Madan *et al*., 1998; Radhakrishnan *et al*., 2000). HEV is restricted to tropical countries and affects older children and young adults and its epidemics are common[1-3]. In developing countries like India, HAV and HEV both are endemic. Exposure rates over a period of time are different in different parts of the country and in different socioeconomic groups[1]. Comparing the epidemiology of HEV in the countries with similar environmental conditions contributing to feco-oral transmission shows that HAV is universally acquired by the age of 5 in both India and Egypt, whereas HEV most commonly infects young adults. Seroprevalence of HEV is only 30 to 40% in the adult population in India, whereas it exceeds 60% by the age of 10 in Egypt [2]. It is unknown why age-specific seroprevalence rates of HAV and HEV are dissimilar in the same country and why antibody acquisition rates are different in different countries with similar environmental conditions. Previously in India, Age-specific prevalence of HAV and HEV in Pune was checked in 1982, 1992 and 1998[1,4]. Similar studies were carried out in Andaman-Nicobar islands in 1989 and 1999 [5]. We have reported age-specific HEV prevalence in Mumbai in the population < 40 years of age in 1993.[3]

Hepatitis E virus causes acute viral hepatitis. Being waterborne infection, Monsoon season is associated with high risk of epidemics Monsoon season is associated with high risk of epidemics. Our study aimed to evaluate the clinical profile and predictors of severity in patients of viral hepatitis epidemic in Jaipur during May 2014 to Jan 2015. Cases evaluated in the study were both Index cases and referred cases from epidemic area, for worsening of jaundice and other complications.

MATERIAL AND METHOD

This study was conducted on 64 patients presenting with acute hepatitis in Metro MAS hospital over period of 9 months from May 2014 to Jan 2015. Informed written consent was taken from each patient. A detailed history of each patient was recorded, including travel history, blood transfusion, and food and water intake from outside sources. The diagnosis of acute viral hepatitis was based on accepted clinical, biochemical and serological criteria. Serum bilirubin, alanine aminotransferase (ALT), prothrombin time international normalized ratio (PT INR) and serum creatinine were the key parameters in our study.

Patients with pre-existing liver disease were excluded. Complete liver function tests (LFT), renal function tests (RFT), prothrombin time and ultrasound abdomen of each patient was done on admission and investigations were repeated at regular intervals thereafter for two months. Upper normal limit of ALT, alkaline phosphatase, bilirubin, creatinine and PT INR was 40 IU/l, 170 IU/l, 2.0 mg/dl, 1.2 mg/dl and 1.3, respectively. All patients were given supportive treatment, symptomatic treatment for nausea, vomiting and specific treatment for hepatic encephalopathy, coagulopathy and renal failure when required. Patients were followed up at regular intervals for eight weeks for assessment of clinical and biochemical parameters. Serum samples from consecutive individuals with acute hepatitis of less than 42 days' duration were collected and stored at 280 uC for further analysis. An acute viral hepatitis (AVH) case was defined as a person having an acute illness with a discrete onset of any sign or symptom (e.g. fever, headache, malaise, anorexia, nausea, vomiting, diarrhoea, abdominal pain) and either jaundice or elevated serum alanine aminotransferase levels higher than 100 IU l^{21} on at least two occasions during a week without any history of pre-existing liver disease.

Immunoassays

Blood samples were collected from all patients under aseptic conditions and centrifuged at 1200 *g*. Serum was separated and stored at -80^0 C for further analysis. The samples were screened using commercially available Micro ELISA for markers of hepatitis A (IgM anti-HAV, Adaltis, Spain), hepatitis B (HBsAg, Biorad Monalisa HBsAg plus, IgM anti HBc in HBsAg positive cases Abbott laboratories North Chicago, IL) hepatitis C (Innotest HCV ab IV, Belgium) and hepatitis E (EIAgen HEV IgM, Adaltis, Spain). On the basis of serological tests, viral hepatitis was classified as acute hepatitis A (presence of IgM anti-HAV), acute hepatitis B (presence of HBsAg & IgM anti HBc), and hepatitis C may be acute or chronic (presence of anti-HCV) or acute hepatitis E (presence of IgM anti HEV), acute hepatitis E and B (presence of anti IgM HEV, HBsAg, IgM anti HBc) and HBV carrier (HBsAg positive). The ELISA kit used for IgM anti HEV were coated with recombinant proteins for open reading frame (ORF) 1 and 2 with 98 per cent sensitivity and specificity. ELISA was performed as per manufacturers' protocol.

RESULTS

In present study, viral hepatitis was predominantly seen in middle age. Males (81%) were predominantly affected. Out of female (19%) patients, 2 were pregnant. Maximum patients presented with jaundice (90%), followed by nausea and vomiting (60%), fever (30%) and abdominal pain (13%). Most common sign was icterus followed by tender hepatomegaly (28%). Virology study showed 40 cases positive for HEV, 3 cases for HBV and 5 cases were positive for HAV. In 3 cases, RFT was raised above 2mg/dl out of which 1 required hemodialyses. 8 cases were found with deranged prothrombin time and 6 received FFP in view of active bleeding. No Mortality was seen in our study

Table1 showing characteristic and clinical features of patients in the study group

parameter	Value (n=64)
age	30 +_ 9.2 yr
fever	30%
anorexia	46%
jaundice	90%
hepatomegaly	28%
vomiting	60%
Abdominal discomfort	13%

Table 2 showing Complications in the study group

Complications	Total (n=64)
Hepatic coagulopathy	8
Fulminant hepatic failure	15
Renal failure	3
Hepatic encephalopathy	11
Overall mortality	0

Table 3. Comparison of the clinical spectrum of patients seropositive for HEV

Data are expressed as mean ± SD.

Marker	Anti-HEV IgM positive (n=41)	Anti-HEV IgG positive (n=51)	Anti-HEV IgM and IgG positive (n=25)	*P* value*
ALT ($IU\ l^{-1}$)	877.1 ± 600.4	226.2 ± 386.5	564.7 ± 562.2	0.001
AST ($IU\ l^{-1}$)	1051.7 ± 660.7	449.7 ± 484.4	745.1 ± 670.4	0.001
TBil ($mg\ dl^{-1}$)	5.2 ± 5.0	5.7 ± 4.0	6.7 ± 6.2	0.018
ALP ($KA\ U\ l^{-1}$)	1253.3 ± 706.9	646 ± 535.7	844 ± 588.1	0.005

*P<0.05 was considered statistically significant. ALT, Alanine aminotransferase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; TBil, total bilirubin. *P* value compared by ANOVA test.

DISCUSSION

Hepatitis E virus is a major hepatotropic virus for acute viral hepatitis. Hepatitis E exists as sporadic hepatitis with periodic resurgence. This infection is responsible for 30-70 per cent cases of acute sporadic hepatitis[11] and is the major cause of acute liver failure (ALF) [12]. It has been widely reported that HEV primarily affects young adults between 15-40 yr of age in endemic region[13]. Similar findings were observed in the present study.

HEV positivity ranging from 12.6-78.6 per cent has been reported by others from different parts of India.

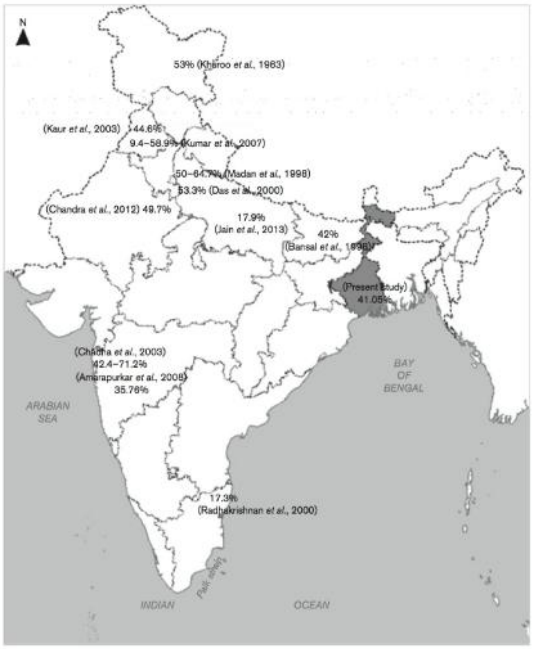

Fig. 1. Prevalence of acute sporadic HEV infection in different parts of India.

In most disease-endemic areas like India, HEV seroprevalence is as low as 5% in the population under the age of 10, but increases to 10-40% among adults > 25 years of age [1, 10]. The results of the present epidemiological study showed the presence of an overall high endemicity of HEV infection (35.68%) and a significantly lower seroprevalence in the age groups <10 years and <20 years as compared to any age group > 20 years. Compared to other series, the higher rate of HEV seroprevalence in the population under the age of 10 was seen in the present series (17.75%), in series from Andaman- Nicobar (32.9%) [5] And in one from North India (>60%) [11]. This was in sharp contrast to series from Pune, where age >15 years was strongly associated with HEV infection [1]. These differences might be due to either varying environmental conditions in different geographical areas or the differences in the diagnostic tests used.

Age distribution of acute HEV cases, both epidemic and sporadic, is almost similar and remains comparable to previous data [12]. HEV mainly affects young adults of 15-40 years of age and relatively spares children [10]. In India, HEV is responsible for 50-70% of all cases of sporadic acute viral hepatitis [13, 14], and also is responsible for large outbreaks with source of infection mainly being contaminated water supplie. [2, 10] The occurrence of large epidemics of HEV in disease-endemic areas, as it was the case in the present study, suggests the possibility of doubtful protection from the antibody, gradual decline in the protective level of the antibody or infection from divergent strains of the virus [4, 10]. However, previous studies have shown that antibodies do protect against the disease [15], HEV virus strains involved in multiple outbreaks in different parts of India are closely related [16], and anti HEV titres persist for a long time [15].

CONCLUSION

We concluded that acute viral hepatitis E is a self- limiting illness with the time to resolution directly correlating with initial severity at presentation and occurrence of complications.

Our study suggests that the outbreaks in Jaipur district were due to Hepatitis E virus. Jaundice was the most common presenting feature followed by vomiting; fever and pain.Early presentation and judicious treatment can prevent mortality. Majority of cases were cured with supportive treatment.

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