An Epidemiological study of acute viral Hepatitis caused by Hepatitis E virus in Surendranagar district, Gujarat

Vidhi Motka¹, Tejas Choksi², Atul Shrivastav³, Miral Dalsania⁴, A. S. Agnihotri⁵

ABSTRACT

Background:-In developing country like India, particularly Surendranagar of Gujarat state where there is inadequate safe drinking water and sewage disposal problem, Hepatitis E virus out-break is very common. This study was carried out to know the effect of unsafe drinking water, unhealthy sewage disposal & its impact on patient's health. Objective: To study clinical presentation and various laboratory parameters of acute viral hepatitis caused by Hepatitis E virus in Surendranagar district. Materials and Method: Retrospective study was carried out from Jan 2011 to March 2011. Results: In this study, total 1500 patient data were collected. Of them, majority of patients had higher Serum bilirubin and SGPT level. Serum bilirubin in patients was ranging from 1mg/dL to 45 mg/dL. SGPT was also higher ranging from 15 IU/L to >4000 IU/L. HEV Ig M by ELISA method was positive in 95% of all advised cases. They had symptoms of acute viral hepatitis like nausea, abdominal pain, jaundice, diarrhea and vomiting. Conclusion: For epidemic of acute viral hepatitis by Hepatitis E virus, unsafe drinking water, unhealthy sewage disposals are an important responsible factors and symptoms were similar to other viral hepatitis.

Key Words: Acute viral hepatitis, Hepatitis E virus, Serum bilirubin, SGPT

^{1,4}Resident Doctors, ²Professor ³Assistant Professor., ⁵Sr. Professor & Head

Department of Pathology, C. U. Shah Medical College, Dudhrej Road, Surendranagar.

Corresponding author mail: atulshri@ymail.com

Conflict of interest: None

INTRODUCTION

Viral hepatitis caused by Hepatitis Virus A and E (HAV & HEV) is the major health problem in India¹. Out of six different types of viral hepatitis known (A, B, C, D, E, and G), hepatitis E virus

(HEV) is the agent responsible for hepatitis outbreak as well as sporadic cases of hepatitis in developing countries ^{1,2,3}. Hepatitis E appears to be wide spread problem in developing countries where there is inadequate safe drinking

water and sewage disposal problem⁴. Almost all outbreaks of viral hepatitis in India are due to feaco-orally transmitted hepatitis E^5 .

The first documented epidemic of HEV was reported in New Delhi, India, in 1955-1956, and 29,300 people were affected 6 . HEV is a small, non enveloped RNA virus, icosahedral in shape, and 27-34 nm in diameter 7 . Classified as member of hepeviridae family 8 . In general population HEV carries low mortality rate of 0.5-4% 9 while in pregnant women mortality rate is up to 20% 10 .

RNA of HEV remains a more specific tool for the diagnosis of the infection, which remains positive in serum for a short duration, and this test commercially available. So, detection of anti HEV Ig M still remains the main diagnostic marker of acute HEV infection 11, it appears in the serum of infected patients the onset symptoms, precedes appearance of anti-HEV Ig G, and wanes within 2-5 months after infection ¹².

MATERIALS AND METHOD

This was a retrospective study, for the period from Jan 2011 to March 2011. During this period a large number of patients were affected with hepatitis in Surendranagar district. In this study data of 1500 patients were collected.

Patients were included from the findings like, deranged lab parameters (like abnormal liver function test (e.g. abnormal values of S. bilirubin, &/or SGPT) and these features were correlated with clinical presentation of patients. Acute viral hepatitis was diagnosed by a serum bilirubin level > 1 mg/dL, SGPT > 65 IU/L or by sero-positivity for any of the hepatotropic virus by using serological tests like Ig M antibodies to HAV, HEV, HCV and HBsAg (hepatitis B surface antigen). Here during initial phase of epidemic the patients were first tested for Ig M antibody to HAV and after that patients were tested for Ig M antibody to HEV, then HBsAg and HCV by ELISA method. Serum Bilirubin and SGPT were done on fully automated Bio chemistry analyzer – Dimension Rx L by Siemens.

RESULTS

In this study data of 1500 patients were collected for the period from Jan 2011 to March 2011. In which

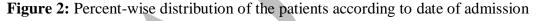
majority of patients were detected for hepatitis during 15 Jan to 15 March.

22.54

Males
Females

Figure 1: Hepatitis E: Gender-wise distribution

Figure 1 revealed that majority of patients were males.



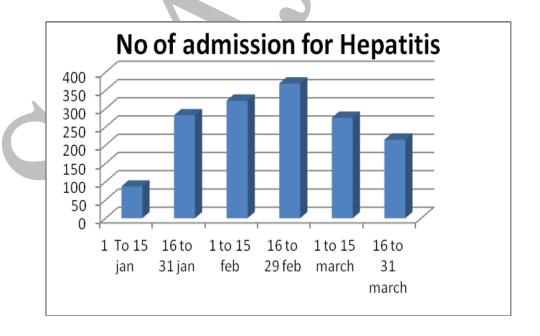


Figure 3: Age-wise distribution of patients.

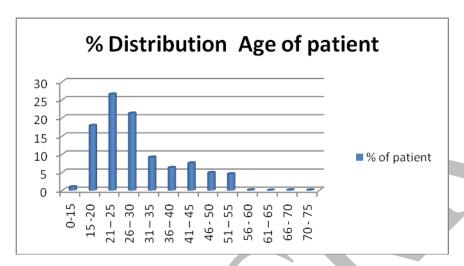


Figure 3, shown that majority of patients (65.86 %) were from 15 to 30 years of age. Here X-axis indicates age of patients and Y-axis indicates percentage of patients.

Table 1: Serum Bilirubin level in patients.

	S. Bilirubin mg/dl	No of patient	% of patient
	1.2 - 5.0	715	47.67
ا د	5.1 – 10.0	499	33.27
	10.1 – 15.0	188	12.53
	15.1 – 20.0	67	4.47
	20.1 – 25.0	24	1.60
	25.1 – 30.0	3	0.2
	30.1 – 35.0	2	0.13
	35.1 – 40.0	0	0
	40.1 -45	2	0.13

Table 1 shown that maximum number of patients have S. bilirubin in range of 1.25 mg/dL to 10 mg/dL.

Two patients had above 40 mg/dl. In almost all patients bilirubin value returned to normal within 1 to 3 weeks.

Table 2: Range of SGPT and serum bilirubin in number of patients.

SGPT range	Bilirubin range	No of patient
15 – 40	1.92 – 18.22	102
40 – 400	1.27 – 26.54	480
400 – 800	1.42 – 26.11	328
800 – 1200	2.03 – 46.11	203
1200 – 1600	1.52 - 21.70	145
1600 – 2000	2.06 – 22.54	95
2000 – 2400	2.72 – 45.77	54
2400 – 2800	1.72 – 27.87	40
2800 – 3200	1.27 – 21.52	25
3200 – 3600	2.07 – 27. 10	14
3600 – 4000	2.43 – 21.41	03
>4000	2.86 – 18.98	11

Table 2 shown SGPT level varying from 15 IU/L to 4000 IU/L, there was no correlation between SGPT level and rise in bilirubin.

All patients had presence of bile salts and bile pigments in urine and 9 % of patients had presence of protein (trace) in urine. HEV Ig M was advised in 150 patients only, as it could not advised in all patients due to un affordability of patients.

HEV Ig-M was found positive in 142 patients. It was 95 % of all advised cases. Among HEV Ig-M confirmed cases, mean total bilirubin level was 10.74 mg/dL, mean conjugated bilirubin was 8.3 mg/dL and mean unconjugated bilirubin was 2.3 mg/dL Mean SGPT was 1452.29 IU/L. The rise in conjugated

bilirubin was higher than unconjugated bilirubin and rise in SGPT was more than rise in bilirubin which was suggestive of hepatic cell injury.

In 5 of the patients Ig M antibody to HAV was positive. In 3 patients hepatitis B surface antigen was positive. From all the above findings it was clear that this epidemic of hepatitis was caused by HEV.

The clinical picture varies depending on the grades of severity from mild asymptomatic or anicteric to more severe icteric forms. Signs and symptoms of positive patients showed that 90% of patients had nausea and abdominal pain, 60 % had jaundice, 40 % had fever, 20 % diarrhea and vomiting. None of the patients had urticaria and pruritus. In most of the patients abdominal pain and nausea appeared before rise in bilirubin was detected. Almost all symptoms were subsided within 3 weeks, without any chronic sequel.

DISCUSSION

This study confirms that there was an outbreak of hepatitis in Surendranagar district between Jan 2011 and March 2011. ELISA testing for anti HEV Ig M in 142 patients confirmed that

Hepatitis E virus was an important attributor to the etiological factor in this epidemic. This observation was not different from other studies. HEV Ig-M could not done in all patients, as they came from poor socio economic conditions. It was positive in 95 % of all cases tested.

All these came from patients same locality and had same water supply and drainage facility. So concluded that this epidemic of viral hepatitis was caused by HEV. Contamination of water was an important causative factor in this epidemic. specific outbreak in present study was found to be maximum in 15-30 years of age, which was also epidemiological evidence in favor of hepatitis E.

This finding was also similar to the other studies ^{2, 3,15,16}. Hepatitis E infection causes asymptomatic to mild illness in children, this is in contrast to other feaco-orally transmitted agents like HAV and polio ¹⁷. Due to variable immunity, resistance and continuous mutations, clinical behavior keeps changing from time to time and region to region. There is no definitive treatment for hepatitis E. Patients are managed symptomatically

only and most of them recover in course of time. In case of hepatic failure patients are managed in intensive care unit.

CONCLUSION

We have concluded from this study, Hepatitis E is mainly spread due to improper hygienic habits, impure water supply & unhealthy waste disposal practice. During investigation it has prove that person who are living in the vicinity of contamination of water, poor sanitation and unhealthy hygienic conditions are more prone for HEV virus infestation. HEV causes self limiting illness, but in many cases it hospitalization requires and during epidemic it was major cause of work loss and drain of hospital resources.

For proper treatment as well as for prevention of HEV infection good personal hygiene, high quality standards for public water supply and proper disposal of sanitary waste are recommended.

REFERENCES

- 1.Tandon BN, Joshi YK, Jain SK, Gandhi BM. An epidemic of non-A, non-B hepatitis in North India. Indian J med Res 1982;75:739-44.
- 2. National Institute of Communicable Disease. Vol 4. Outbreak of viral hepatitis

- E: Public health system need to be alert. CD Alert, Delhi: National Institute of Communicable Disease; 2000.
- 3. Sarguna P, Rao A, Sudha Raman KN. Outbreak of acute viral hepatitis due to hepatitis E virus in Hyedrabad. Indian J med Microbiol 2007;25(4):378-82.
- 4. Park K. Park's text book of preventive and social medicine. 18th ed. Bhanot Publisher: Jabalpur; 2005.p.175.
- 5. Directorate of Heaith services, Maharashtra. Disease surveillance – Training Module , National Surveillance Programme for communicable Disease: 1999.p.45-55.
- 6. Vishwanathan R. Infectious hepatitis in Delhi (1955-56): a critical study. Epidemioliogy.Indian J Med *Res* 1957;45:1-29.
- 7. Margolis HS, Alter MJ, Halder SC. Viral hepatitis. In: Evans AS, Kaslow RA, editors . Viral infection of humans : epidemiology and control. 4thed.New York, NY: Plenum Medical Book Company, 1997:363-406
- 8. Dienstag J.L. Acute viral Hepatitis. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL Jameson JL, et al. editors. Harrison's Internal Medicine. 17th ed. New York: McGraw-Hill Companies; 2008.p 1932-49.

- 9. Krawczynski K. Hepatitis E. Hepatology 1993;17:932-41.
- 10. Wang L, Zhuang H. Hepatitis E: an overview and recent advances in vaccine research. World J Gastroenterol 2004;10(15): 2157-62.
- 11.Clayson ET, Myint KS, Sntibhan R, Vaughn DW, InhisBL,Chan L et al. Viremia, fecal shedding, and Ig M and Ig G responses in patients with hepatitis E. J Infect Dis 1995;172:927-33.
- 12. Bredley DW, Hepatitsis E virus: a brief review of the biology, molecular virology, and immunology of a novel virus. J Hepatol 1995;22(suppl 1):140-5.
- 13. Khuroo MS, Rustgi VK, Dawson GJ, Mushahwar IK, Yattoo GN, Kamili et al. Spectum of hepatitis E virus infection in India. *J Med Virol* 1994;43:281-6.
- 14. Hamid SS, Atiq M, Shehzad F, Yasmeen A, Nissa T, Salam A et al. Hepatitis E virus

- superinfection in patients with chronic liver disease. Hepatology 2002;36:474-8.
- 15. Dilawari JB, Singh K, Chawla YK,Ramesh GN, Chauhan A, Bhusnurmath SR, et al. Hepatitis E virus :Epidemiological, clinical and serological studies of a North Indian Epidemic. Indian J Gastroenterol 1994;13:44-8.
- 16. Bhagyalaxmi A, Gadhvi M, Bhavsar BS. Epidemiological Investigation of an outbreak of infectious Hepatitis in Dakor Town. Indian J Community Med 2007;32(4):277-9
- 17. Worm HC, Poel WHM, Brandstatter G. Hepatitis E: an over view. Microbes and infection 2002; 4:657-66.