Original Article

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Causative Agents in Patients Presenting with Acute Viral Hepatitis along with their Clinical Profile at a Tertiary Care Hospital, Western U.P.

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Abstract

Acute viral hepatitis (AVH) is a common health-related problem throughout the world, but morbidity and mortality are prevalently seen in developing countries like India. In India, AVH is most commonly caused by HAV (hepatitis A virus) and HEV (hepatitis E virus) and in few cases, HBV (Hepatitis B virus) is the etiological agent. HEV mainly affects the middle-aged population, is mild in symptoms however it is lethal with 30% mortality in pregnant females compared to the mortality of ≤1% in the general population. This study was focussed to identify the etiological agents, various clinical - laboratory factors, disease severity and associated complications in patients presenting with AVH. In our study, HEV was the most common etiological agent accounting for 43.6% of cases followed by HAV (29.1%), HBV (26.1%) and HCV (1.2%). The most common age group involved with AVH was the adult group i.e. from 21-30 yrs (35.1%) followed by 11-20 yrs (27.9%), below 10 yrs (15.1%) and 31-40 yrs (14.5%). The commonest symptom was yellow-colored urine (86.1%) followed by yellow discolouration of sclera and loss of appetite. Bleeding derangement was observed in all patients with acute liver failure (ALF) while Hepatic encephalopathy was observed in 3 patients with ALF. To conclude, integration of diagnostic measures, early diagnosis, treatment protocols, prevention and mass vaccination will help in the overall reduction of AVH cases and related complications.

Keywords: Acute viral hepatitis, acute liver failure, Anti -HEV

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Introduction

Acute viral hepatitis (AVH) is a common health-related problem throughout the world, but morbidity and mortality are prevalently seen in developing countries like India. Multiple factors like socioeconomic conditions, poor sanitation, contaminated food and water, lack of awareness are responsible for its spread in developing countries. Hepatitis A, B, C, D, E and G are the common viral agents causing AVH, of which Hepatitis D (Delta virus) is called an incomplete/ defective virus because it cannot replicate on its own and requires the help of Hepatitis B virus (HBV) for its replication hence its infection can occur only along with HBV infection. In India, AVH is most commonly caused by HAV (hepatitis A virus) and HEV (hepatitis E virus) and in few cases HBV (Hepatitis B virus) is the etiological agent.[1,2] Majority of epidemics are caused by Hepatitis E virus (HEV) especially in developing countries and sometimes causing complications like a fulminant hepatic failure.[3,4] Both HAV and HEV are transmitted by faeco-oral route while HBV, HCV (Hepatitis C virus), and HDV (Hepatitis D virus) are transmitted through parenteral route, sexually or through mother to child transmission. The incidence of HAV is more among children, however this pattern is changing in recent times due to increased vaccination of HAV among children and hence the trend of HAV has been shifted towards the adult and adolescent population in India.[1] HEV mainly affects the middle-aged population, is mild in symptoms however it is lethal with 30% mortality in pregnant females compared to the mortality of ≤1% in the general population. Hepatitis B and hepatitis C are more associated with chronicity later progressing to cirrhosis and hepatocellular carcinoma.[5,6] Regular testing for viral markers, mass vaccination against Hepatitis A &Hepatitis B, early diagnosis and appropriate treatment can improve health outcomes for at-risk patients. In view of the above discussion, this study was

conducted with the aim to identify the etiological agents, various clinical and laboratory parameters, severity of disease and associated complications in patients presenting with AVH.

Subjects and Methods

A prospective observational study was carried out for 3 years in the hospital of western U.P. North India from July 2017 to June 2020. Acute hepatitis case was defined as an acute illness with clinical symptoms (e.g. fever, headache, malaise, nausea, vomiting, loss of appetite, dark urine, and abdominal pain) with jaundice or serum (ALT) levels >200 IU/L or at least twice upper limit of normal without having a history of chronic liver disease.[7] Patients presenting with clinical manifestations of AVH were included in the study. Patients with underlying chronic liver disease, liver cirrhosis, hepatitis induced by drugs, alcoholic hepatitis, metabolic diseases leading to hepatitis and sepsis-related multiorgan failure were excluded from our study. All relevant clinical and medical history were recorded in the case record form. All the patients were tested for IgM HAV, IgM HEV, HBsAg and IgM HCV using ELISA. All the patients were tested for basic investigations like Complete blood count (CBC profile) and liver function test (LFT) and were recorded. All patients included in the study were informed regarding the study protocol and informed consent was signed by these patients. Statistical analysis was done using the latest SPSS software version 16.0 (IBMCorp., Armonk, NY, United States of America).

Results

During the study period of three years, 165 patients with a diagnosis of Acute Viral Hepatitis were detected and included. Out of them, HEV was the most common etiological agent accounting for 43.6% of cases followed by HAV (29.1%), HBV (26.1%) and HCV (1.2%) as shown in [Table 1]. Males (55.75%) were more commonly involved compared to females (44.25%). The most common age group involved with AVH was the adult group i.e. from 21-30 yrs (35.1%) followed by 11-20 yrs (27.9%), amongst children i.e. less than 10 yrs (15.1%) and 31-40 yrs (14.5%) as depicted in [Table 2]. The commonest symptom was yellow coloured urine (86.1%) followed by yellowish discolouration of sclera and loss of appetite. Other symptoms commonly seen in our study were malaise, fever, nausea, vomiting and itching as shown in table 3. Icterus was the most common sign seen followed by hepatomegaly, pallor, splenomegaly, ascites and altered sensorium as shown in [Table 3]. Ultrasound abdomen was done in 150 patients and the most common ultrasound finding was coarse liver echotexture with periportal cuffing and circumferential gall bladder thickening. Total serum bilirubin, SGOT and SGPT levels were raised in all cases of AVH however the maximum bilirubin level noted was 29.51mg/dl, while peak transaminases level i.e. SGOT and SGPT were 5,368 IU/L and 5,594 IU/L respectively. Maximum PT (prothrombin time) (sec), INR (international normalized ratio) and APTT (activated partial thromboplastin time) (sec) noted were 34.5, 3.66, 56.3 respectively. Clinically significant bleeding was observed in 6 patients who were already in acute liver failure (ALF) and were transfused with FFP. Hepatic encephalopathy was observed in 3 patients with ALF and they were shifted to ICU and were put on a ventilator. We had one mortality in our study.

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Table 1: Distribution of cases according to the etiological agents

Etiological agents	No. of patients	Percentage of patients
Anti HAV IgM positive	48	29.1%
Anti HEV IgM positive	72	43.6%
HbsAg positive	43	26.1%
Anti- HCV positive	2	**1.2%**
Total patients	165	

Table 2: Age-wise and sex-wise analysis of patients with AVH:

Age group	No. of isolates	Males	Females
≤10 yrs	25 (15.1%)	14	11
11- 20 yrs	46 (27.9%)	25	21
21-30 yrs	58 (35.1%)	32	26
31-40 yrs	24 (14.5%)	14	10
41-50 yrs	8 (4.8%)	5	3
≥50 yrs	4 (2.4%)	2	2
Total	165	92 (55.75%)	73 (44.25%)

Discussion

AVH is a major health-related issue affecting the young age group population especially in developing countries. In our study, 165 patients were diagnosed with AVH during the study period of three years. HEV was the most common etiological agent accounting for 43.6% of cases followed

Table 3: Clinical symptoms and signs seen in patients with acute viral hepatitis:

Symptoms & signs	HAV (n=48)	HEV (n=72)	HBV (n=43)	HCV (n=2)	Total (n=165)
Yellowish Dis-colouration of Sclera	41 (85.4%)	65 (90.3%)	31 (72.1%)	1 (50%)	138 (83.6%)
Yellowish Dis-colouration of Urine	42 (87.5%)	67 (93.1%)	32 (74.4%)	1 (50%)	142 (86.1%)
Nausea & Vomiting	36 (75%)	62 (86.1%)	29 (67.4%)	1 (50%)	128 (77.6%)
Loss of Appetite	42 (87.5%)	65 (90.3%)	31 (72.1%)	1 (50%)	139 (84.2%)
Abdominal Pain	28 (58.3%)	52 (72.2%)	22 (51.2%)	0 (0%)	102 (61.8%)
Fever	27 (56.2%)	48 (66.7%)	19 (44.2%)	0 (0%)	94 (56.9%)
Malaise	27 (56.2%)	50 (69.4%)	20 (46.5%)	0 (0%)	97 (58.8%)
Itching	18 (37.5%)	46 (63.9%)	9 (20.9%)	1 (50%)	74 (44.8%)
Altered Sensorium	4 (8.3%)	6 (8.3%)	7 (16.3%)	0 (0%)	17 (10.3%)
Icterus	41 (85.4%)	65 (90.3%)	31 (72.1%)	1 (50%)	138 (83.6%)
Pallor	17 (35.4%)	22 (30.5%)	9 (20.9%)	1 (50%)	49 (29.7%)
Hepatomegaly	18 (37.5%)	34 (47.2%)	9 (20.9%)	0 (0%)	61 (36.9%)
Splenomegaly	3 (6.2%)	6 (8.3%)	2 (4.6%)	0 (0%)	11 (6.7%)
Pedal Edema	2 (4.2%)	6 (8.3%)	3 (6.9%)	1 (50%)	12 (7.3%)
Ascitis	1 (2.1%)	7 (9.7%)	2 (4.6%)	1 (50%)	11 (6.7%)
Acute Liver Failure	0 (0%)	3 (4.2%)	3 (6.9%)	0 (0%)	6 (3.6%)

Table 4: Laboratory parameters derangement in patients with acute viral hepatitis:

Laboratory parameters	HAV (n=48)	HEV (n=72)	HBV (n=43)	HCV (n=2)	Total (n=165)
Total Bilirubin (mg/dl)					
1.3-3	1 (2.1%)	11 (15.3%)	2 (4.6%)	0	14 (8.5%)
3.1-10	37 (77.1%)	44 (61.1%)	29 (67.4%)	1 (50%)	111 (67.3%)
10.1-20	10 (20.8%)	13 (18.1%)	9(20.9%)	1 (50%)	33(20%)
≥20	0 (0%)	4 (5.5%)	3 (6.9%)	0 (0%)	7 (4.2%)
SGOT (IU/L)					
41-1000	9 (18.7%)	17 (23.6%)	8 (18.6%)	0 (0%)	34 (20.6%)
1001-2000	32 (66.7%)	46 (63.9%)	24(55.8%)	1 (50%)	103 (62.4%)
2001-3000	7 (14.6%)	7 (9.7%)	8 (18.6%)	0 (0%)	22 (13.3%)
>3000	0 (0%)	2 (2.8%)	3 (6.9%)	1 (50%)	6 (3.6%)
SGPT (IU/L)					
41-1000	8 (16.7%)	13 (18.1%)	6 (13.9%)	0 (0%)	27 (16.4%)
1001-2000	34 (70.8%)	48 (66.7%)	26 (60.5%)	1 (50%)	109 (66.1%)
2001-3000	6 (12.5%)	8 (11.1%)	8 (18.6%)	1 (50%)	23 (13.9%)
>3000	0 (0%)	3 (4.2%)	3 (6.9%)	0 (0%)	6 (3.6%)
PT (sec)*	11.2sec	20.2sec	18.4sec	13.6sec	
INR*	1.4	1.82	1.6	1.7	
APTT (sec)*	31.2	34.6	29.8	34.9	

mean value

by HAV (29.1%), HBV (26.1%) and HCV (1.2%). A study conducted by Desai et al[8] showed hepatitis E (70%) as the most common cause of AVH, followed by hepatitis B (15.8%), hepatitis A (12.8%), and hepatitis C (1.4%). Another study was done by Dabadghao et al.[9] and Shah et al,[10] found hepatitis E in 45% and 50% cases of AVH respectively. It is important to note that in our study HAV was seen in 29.1% of the patients with AVH. This is because of the fact that all age group was included and India being endemic for diseases like HAV, most of the children get exposed to HAV in childhood and become immune till adolescence due to subclinical exposure.[11,12] However this trend is changing and now the age group getting infected with HAV has been shifted to adolescents and adult population. The main reason for this shift is the easy availability of HAV vaccine and awareness among the population regarding the disease.

The most common age group involved with AVH in our study was the adult group i.e. from 21-30 yrs (35.1%) followed by 11-20 yrs (27.9%), amongst children i.e. less than 10 yrs (15.1%) and 31-40 yrs (14.5%). A study conducted by Shah et al,[10] and Desai et al,[8] showed the mean age of presentation being 30±12.4 years and 27.25±9.5 years respectively. Dabadghao et al,[9] and Birajdar et al.[13] also observed young adults being most affected with AVH. In the present study males were more commonly affected than females, similar to the studies conducted by Monica A et al and Handa S et al.[14,15]

In the present study, the commonest symptom was yellow coloured urine (86.1%) followed by yellow discolouration of sclera and loss of appetite. Other symptoms commonly seen in our study were malaise, fever, nausea, vomiting and itching. Icterus was the most common sign seen followed by hepatomegaly, pallor, splenomegaly, ascites and altered sensorium. A study done by Shah et al observed jaundice (86.10%) as the most common symptom followed by anorexia (76.50%), yellow-colored urine (73%), fever presenting with chills and rigors (66.1%), and pain in the abdomen (36.3%).[10] Zhang et al. also observed jaundice, fatigue, and anorexia as the common clinical presentation.[16]

In the present study, total serum bilirubin, SGOT and SGPT levels were raised in all cases of AVH however the maximum bilirubin level noted was 29.51mg/dl, while peak transaminases level i.e. SGOT and SGPT were 5,368 IU/L and 5,594 IU/L respectively. A study conducted by Desai et al observed that raised serum bilirubin and SGOT in all cases while SGPT was raised in 98.5% of patients.[8] In our study maximum PT (sec), INR and APTT (sec) noted were 34.5, 3.66, 56.3 respectively. Other studies conducted by Birajdar et al,[13] and Dabadghao et al. found PT >15 sec and altered PT/INR was present in 30% of cases of AVH Clinically significant bleeding (coagulation failure) in our study was observed in all patients with acute liver failure (ALF) and they were transfused with FFP.[9] Coagulopathy is considered as apoor prognostic marker in cases with AVH. Hepatic encephalopathy was also observed in 3 patients with ALF and one patient in our study died due to hepatic encephalopathy.

Conclusion

Viral hepatitis cannot be differentiated on the basis of clinical manifestations, Liver function test, and disease severity. In an endemic country like India, viral hepatitis is usually diagnosedon the basis of detection of IgM antibodies or antigen using ELISA, but now even rapid cards are available for diagnosis and the results are determined within 30 minutes thereby reducing the time taken in diagnosis. This also helps in initiating the treatment of such patients based on its diagnosis. Integration of diagnostic measures, early diagnosis, treatment protocols, prevention and mass vaccination will help in the overall reduction of AVH cases and related complications.

References

1. Rawat S, Gill PS, Gupta T, Malhotra P, Parmar A. Prevalence of hepatitis A virus and hepatitis E virus in the patients presenting with acute viral hepatitis in Rohtak. Haryana;.
2. Thiruvasagam P, George KJ, Arumugam S, Prasad AR, , , et al. IPSec: Performance Analysis in IPv4 and IPv6. J ICT Standardization. 2019;7(1):61–80. Available from: https://dx.doi.org/10.13052/jicts2245-800x.714.
3. Murhekar MV, Ashok M, Kanagasabai K, Ravi JV, Subarinathan M, R. Epidemiology of hepatitis A and hepatitis E based on laboratory surveillance Data-India. Am J Trop Med Hyg. 2014;99:1058–61. Available from: https://dx.doi.org/10.4269/ajtmh.18-0232.
4. Satsangi S, Chawla YK. Viral hepatitis: Indian scenario. Med J Armed Forces India. 2016;72(3):204–210. Available from: https://dx.doi.org/10.1016/j.mjafi.2016.06.011.
5. Hossain MS, Alam MR, Hasan MI, Sharif JU, Kabir MA, Islam MA. Prevalence of serological markers of viruses in patients of acute hepatitis. Mymensingh Med J. 2019;25:278–285.
6. Ringehan M, McKeating JA, Protzer U. Viral hepatitis and liver cancer. Philos Trans R Soc Lond B Biol Sci. 2017;372(1732):20160274. Available from: https://dx.doi.org/10.1098/rstb.2016.0274.
7. ; 2012. Available from: http://www.cdc.gov/nndss/document/2012_Case%20Definitions.pdf.
8. Desai H, Ansari AZ, Makwana D, Jadeja D, Gusani J. Clinical-biochemical profile and etiology of acute viral hepatitis in hospitalized young adults at tertiary care center. J Family Med Prim Care. 2020;9(1):247. Available from: https://dx.doi.org/10.4103/jfmpc.jfmpc_727_19.
9. Dabadghao V, Barure R, Sharma S, Mangudkar S. A study of the clinical and biochemical profile of acute viral hepatitis. Int J Biomed Adv Res. 2015;6:68993.
10. Shah NA, Kadla SA, Shafi PM, Dar IH. Clinico-serological profile of acute sporadic viral hepatitis in Kashimiri adults: Hospital based prospective study. JMSCR. 2014;2:3119–3145.

11. Acharya SK, Madan K, Dattagupta S, Panda SK. Viral hepatitis in India. Natl Med J India. 2006;19:203–220.
12. Nayak S, Panda M. VLSI Cell Partitioning Using Data Mining Approaches. International J Comput Sci Eng. 2018;6:1019–1027. Available from: https://dx.doi.org/10.26438/ijcse/v6i8.10191027.
13. Birajdar SV, Chavan SS, Mundhe SA, Bhosale MG. Clinical and biochemical profile of patients with viral hepatitis at tertiary care centre. Int J Adv Med. 2017;4(2):412. Available from: https://dx.doi.org/10.18203/2349-3933.ijam20171002.
14. Agrawal M, Ruchi K, Ashish B, Pallab S. A Study of Seroprevalence and Co-infection of Hepatitis A and Hepatitis E Viruses in Sporadic Cases in an Endemic Area. Int J Med Sci Public Health. 2016;02(03):1–5. Available from: https://dx.doi.org/10.46347/jmsh.2016.v02i03.001.
15. Handa S, Wasim S, Kalra BP, Rawat A, Chandar V, Gupta A. Clinico-epidemiological profile of hepatitis A virus and hepatitis E virus co-infection in pediatric age group: a hospital based retrospective study. Int J Contemp Pediatr. 2019;6(2):588. Available from: https://dx.doi.org/10.18203/2349-3291.ijcp20190692.
16. Zhang S, Wang J, Yuan Q, Ge S, Zhang J, Xia N. Clinical characteristics and risk factors of sporadic hepatitis E in Central China. Virol J. 2011;8:1–5. Available from: https://dx.doi.org/10.1186/1743-422X-8-152.

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