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Prevalence of Hepatitis A and E Infection in a Suburban Tertiary Care Hospital

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Abstract

Background: Acute viral hepatitis causes high morbidity in children and young adults. Hepatitis A and E can lead to fulminant hepatitis in 0.1% -2%. Objective: To find prevalence of HAV and HEV among suspected cases of acute viral hepatitis and to co-relate the laboratory findings with clinical presentations, over a one year period **Material & Methods:** A retrospective analysis of 396 suspected acute viral hepatitis cases, whose samples were received in the laboratory during over a period of 1 year was performed. All sera samples were tested for IgM anti HAV & IgM anti HEV using commercially available solid phase ELISA, in Microbiology laboratory, at our hospital. **Results:** Majority infected were male (69%) and young adults (98 %). Clinical presentations included fever (92%), jaundice/icterus (74%), nausea/vomiting (60%), hepatomegaly (45%), abdominal pain (40%), darkcolored urine(15%), itching/rash (8.3%). ELISA revealed overall positivity :21.2% ; HAV: 38% ,HEV:62% and dual infection:5%. Increased serum bilirubin, AST and ALT occured in 68%. **Conclusions:** Awareness regarding sanitation and hygiene is imperative to curb the spread of acute viral hepatitis, especially in developing countries. Laboratory diagnosis is an essential supplementary tool in confirmation of suspected clinical cases and reduce transmission of this infection.

Keywords:- Acute Viral Hepatitis.

INTRODUCTION

Hepatitis viruses A and E share many similarities and occupy similar ecological niches. They present considerable public health challenges with increased morbidity especially in children, young adults and pregnant women. The World Health Organization estimates that there are 1.4 million cases of hepatitis A globally annually, resulting in approximately 7,000 deaths.[1] The estimated HEV infections each year is 20 million, 3.3 million being symptomatic cases and approximately 44,000 deaths.[2] These figures represent parts of the world where HEV is endemic are a gross underestimate of the actual global disease burden.[3] Both HAV and HEV present clinically as acute viral hepatitis and sequelae includes fulminant hepatitis, 0.1% in HAV and 1-2% in HEV. Both these infections are preventable by active and passive immunoprophylaxis. Detection of

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cases would help us reduce the source of infection and prevent its spread.[4] This will enable all to achieve the "global hepatitis elimation target by 2030".[3]

Aim & objective:

To find out the prevalence of HAV and HEV among suspected cases of acute viral hepatitis and to co-relate the positive findings with clinical presentations.

MATERIAL AND METHODS

After obtaining Institutional Review board approval, a retrospective analysis of 396 suspected acute viral hepatitis cases, whose samples were received in the laboratory during June 2018 to May 2019 over a period of 1 year, was initiated in the Microbiology Department of a tertiary level suburban teaching hospital in Mumbai, India. Inclusion criteria: 1.Gender:All suspected cases of HAV or HEV acute hepatitis presenting to ward/ OPD. 2.

Age: > 1 year.

Exclusion criteria:

1. Patient having chronic liver disease or metabolic disease, biliary obstruction.
2. Children < one year.
3. Records with incomplete data.

Study procedure

All the data was collected in a period of one to two months from the laboratory records and from Medical Record Department (MRD) if required. Laboratory records were analysed by the following parameters: demographic details age, sex, clinical presentation, positivity for IgM anti HAV/ IgM anti HEV/both and outcome of the patients those who were positive. The tests were performed at the department laboratory using ELISA by Recombilisa (CTK Biotech Inc, California). Study procedure: Total 396 whole blood samples from patients were processed for detection of IgM anti HAV & IgM anti HEV ELISA by Recombilisa (CTK Biotech Inc, California).

HAV IgM ELISA: The Recombilisa HAV IgM ELISA is a solidphase enzyme-linked immunosorbent assay based on the principle of the IgM capture technique for the detection of anti-HAV IgM in human serum or plasma. The HAV IgM ELISA is composed of two key components: 1. Solid microwells pre-coated with monoclonal anti-human IgM antibody, 2. Liquid conjugates composed of HAV antigens conjugated with horseradish peroxidase (HRP-HAV conjugates). During the assay, the test specimen is first incubated in the coated microwell. The anti-HAV IgM , if present in the specimen, binds to the antibody coated on the microwell surface, and any unbound specimen is then removed by a wash step. During a second incubation with the HRP-HAV conjugate, the anti-HAV IgM antibody adsorbed on the surface of microwell binds to antibody in the HRP conjugate, forming a conjugate complex. Unbound conjugates are then removed by washing. After addition of the TMB substrate, the presence of the conjugate complex is shown by development of a blue colour resulting from a reaction between the enzyme and substrate. This reaction is then quenched by addition of the stop solution, and the absorbance value for each microwell is determined using spectrophotometer at 450/620-690 nm.

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HEV IgM ELISA: The Recombilisa HEV IgM ELISA is a solidphase enzyme-linked immunosorbent assay based on the principle of the IgM capture technique for the detection of anti-HEV IgM in human serum or plasma. The HEV IgM ELISA is composed of two key components: 1. Solid microwells pre-coated with polyclonal anti-human IgM antibody, 2. Liquid conjugates composed of HEV antigens conjugated with horseradish peroxidase (HRP-HEV conjugates).

During the assay, the test specimen is first incubated in the coated microwell. The anti-HAV IgM , if present in the specimen, binds to the antibody coated on the microwell surface, and any unbound specimen is then removed by a wash step.

During a second incubation with the HRP-HEV conjugate, the anti-HEV IgM antibody adsorbed on the surface of microwell binds to antibody in the HRP-HEV conjugate, forming a conjugate complex.

Unbound conjugates are then removed by washing. After addition of the TMB substrate, the presence of the conjugate complex is shown by development of a blue colour resulting from a reaction between the enzyme and substrate. This reaction is then quenched by addition of the stop solution, and the absorbance value for each microwell is determined using spectrophotometer at 450/620-690 nm.

Statistical analysis & Outcome measures: Sample size calculation has been mentioned above.The data entry was made in Microsoft Excel sheet.Statistical analysis was performed using SPSS software.The chi square test was used for assessing association between categorical variables.The p value of 0.05 or less was considered significant.

RESULTS

In our study the overall positivity of HEV and HAV by ELISA testing was 84/396 (21.2%). Distribution of HAV, HEV and dual infection is shown in [Figure 1]. As noted HEV positivity was higher compared to HAV. Agewise, majority were young adults >12 years: 387/396 (98%). Gender wise, total number of females cases were higher :204/396 (55%). However both HAV and HEV infection was higher in males [Figure 2]. The commonest clinical feature was fever followed by jaundice, icterus, nausea and vomiting [Table 1]. Laboratory investigations revealed increased serum bilirubin, aspartate and alanine aminotransferase (AST and ALT) levels in 57/84 (68%) patients [Table 2].

Table 1: Clinical Features in HAV/HEV infected patients n=84

Clinical features	**Number(%)**
Fever	77/84(92%)
Jaundice / icterus	62/84(74%)
Nausea /vomiting	50/84 (60%)
Hepatomegaly	38/84 (45%)
Abdominal pain	34/84 (40%)
Darkcolored urine	13/84 (15%)
Itching /rash	7/84 (8.3%)

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Table 2: Laboratory findings in infected patients (n=57)

Parameter	**Number(%)**
ALT>1000IU	21/57(37%)
AST >1000IU	16/57(28%)
Serum bilirubin 5-10	16/57(28%)
Serum bilirubin >5	6/57(11%)

Table 3: Comparison of HAV and HAV infection with other studies

Authors	**City**	**Year**	**HAV %**	**HEV %**
Sarangi et al	Cuttack	2022	54.3	43.5
K Sravanthi	Kurnool	2021	71	29
Desai et al	Ahmedabad	2020	12.8	70
Khan et al	Bangladesh	2020	19	10
Kalita et al	Uttarakhand	2020	14.7	28.4
Sammadar et al	Central Mumbai	2019	6.96	9.63
Agarwal S et al	New Delhi	2017	15.5	27.2
Behera Patnaik	Bhuvaneshwar	2016	75	6.2
Agarwal M et al	Gurgaon	2016	9.4	23.3
Sandhya et al	South Mumbai	2016	10.6	20
Behera et al	Sambhalpur	2016	17	47
Joon et al	Mangalore	2015	19.3	10.5
Jain et al	Lucknow	2013	26.9	17.9
Amen et al	Yemen	2010	86.6	10.7
Present study			38	62

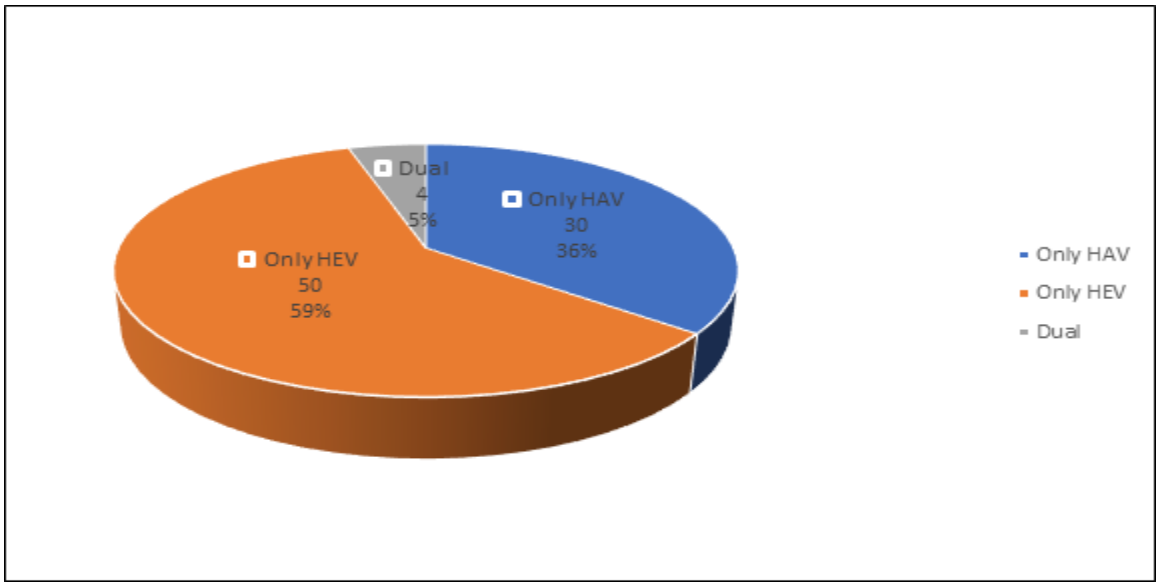

Figure 1: Distribution of HAV, HEV and dual infection in positive cases n=84

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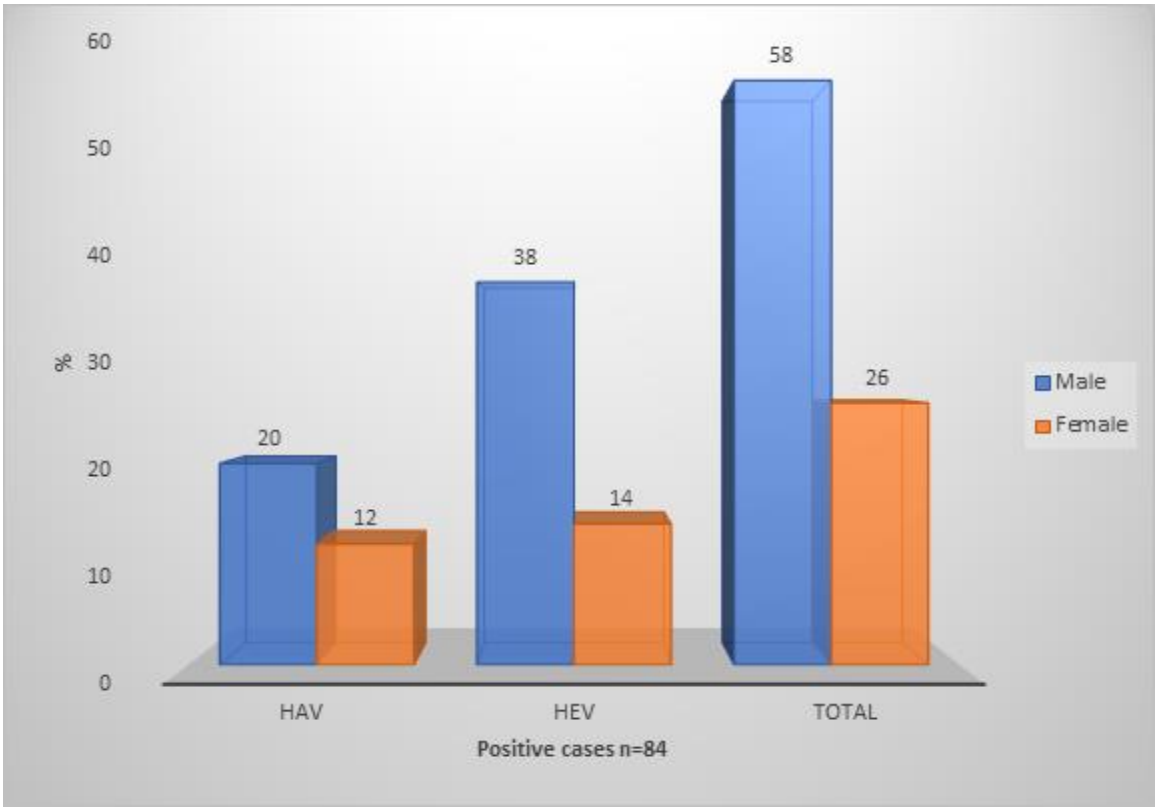

Figure 2: Genderwise distribution of in positive cases n=84

DISCUSSION

Overall positivity of HEV and HAV by ELISA testing, in our study was 84/396 (21.2%). This was closer to Bangladesh study 29%.[5] Comparatively high positivity was reported from Gurgaon, New Delhi and Cuttack 38.1%, 42.7% and 85.5% respectively.[6,7,8]

HAV positivity in our study was 32/84 (38%). High HAV positivity was reported by Sarangi et al 54.3%, K Sravanthi et al 71%, Behera and Patnaik 75 % and Bawazir et al 86.6%.[8,9,10,11] Comparatively low HAV figures were reported by Jain et al 26.96%, Joon et al 19.31%, Khan et al 19%, Behera et al 17%, Agarwal M et al 15.5%, Kalita et al 14.7%, Desai et al 12.8%,Sandhya et al 10.6 %,Agarwal S et al 9.4 %, SR Moebbi et al 9.3 % and Samaddar et al 6.96% [Table 3].[5,6,7,12,13,14,15,16,17,18,19]

HEV positivity in our study was 52/84 (62 %). Comparatively high HEV positivity was noted by Mohebi et al 90% and Desai et al (70%).[16,18] In contrast low HEV positivity was reported from Jaipur (Chandra et al) 49.7%, Sambhalpur (Behera et al) 47%,Cuttack (Sarangi et al) 43.5%, Bhuvaneshwar (Behera & Patnaik) 6.2%, Kurnool (K Sravanthi et al) 29% ,Uttarakhand (Kalita et al) 28.4%, New Delhi (Agarwal S et al) 27.2%, Gurgaon (Agarwal M et al) 23.3%, South Mumbai (Sandhya et al) 20 %, Lucknow (Jain et al) 17.9%, Mangalore (Joon et al) 10.54%, Yemen (Bawazir et al)10.7%, Bangladesh (Khan et al) 10%, Central Mumbai

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(Samaddar et al) 9.63% and Spain (Buti et al)4.6% [Table 5].[5,6,7, 8,9,10,11,12, 13,14,15,17,19,21]

We noted higher HEV cases as compared to HAV: 62% and 38% respectively. Similarly higher HEV > HAV was reported by Desai et al, Sandhya et al, Gurgaon study and New Delhi study.[6,7,16,17] Lower figures HEV < HAV was observed by: Chandra et al, Sarangi et al and Behera et al [Table 5].[8,14,20]

In our study dual HAV and HEV infection was seen in 4/84 (5%). Similar findings were reported from south Mumbai (5.9%), Uttarakhand (5.95%), Gurgaon (5.2%), and New Delhi (5.1%).[6,7,15,17] Higher coinfection of above was noted by Joon et al (11.5%), whereas comparatively low figures were reported from Bhuvaneshwar (3.1%), Cuttack (2.2%),Central Mumbai (2.07%)and Jaipur (1.2%).[8,10,13,19,20]

Gender wise, in our study both HAV and HEV infection was higher in males (69%) compared to females (31%) [Table 2]. Male preponderance was also noted by by Behera et al (71%), Joon et al (68%), Chandra et al (66%), Agarwal S et al (60%), K Sravanthi et al (57.14%), Sarangi et al (55%), Desai et al (53%).[7,8,9,13,14,16,20] As opposed to this high HEV in females was observed by Gurav et al M/F :8/17(68%).[21,22] However in few other studies one gender was predominant in HAV and reverse was true for HEV. For example, Sandhya et al found HAV infection to be more in males and HEV infection more common in females (HAV M>F 50/42; HEV M<F 57/117).[17] Samaddar et al also revealed similar picture (HAV M>F 7.3/6.7; HEV M<F 8.8/10.7.[19] In contrast higher HAV in females and higher HEV in males was shown by Kalita et al (HAV M<F 14.2/15.36 %; HEV M>F 34.2/21.6%) and Agarwal S et al (HAV M<F 14.6/16.6; HEV M>F 29.8/23.4).[7,15] Khan et al, also noted higher HAV in females 24.9% and higher HEV in males 11.2%.[5]

In our study young adults >12 years constituted 387/396 (98%). This was similar to 96% by Agarwal M et al and 91.8% by Sandhya et al.[6,17] Likewise young adults was the major age group in studies from Uttarakhand and Mangalore.[13,15] Infection in lower age <18 years was noted by Sarangi et al 62% and Agarwal S et al 60 %.[7,8] Khan et al found HAV to be higher in children and HEV to be higher in age group 15-60 years.[5] Jain et al have indicated an epidemiologic shift of HAV from children to adults.[12]

Clinically fever was the commonest presentation seen in 77/84 (92%) cases in our study. Similar finding was reported by K Sravanthi et al study fever 100%, Gurav et al in 88%, Khan et al study 75.5%.[5,9,22] Comparatively low cases of fever were reported and from Bhuvaneshwar 50%, Ahmedabad 37 % and Sambhalpur 34%.[6,14,16]

Jaundice and icterus was seen in 62/84 (74%) cases in our study. Comparatively high incidence was reported by Gurav et al (100%), Khan et al 100%, Behera and Patnaik (95.8%), Desai et al (90 %), K Sravanthi et al study 88% and Behara et al 82%.[5,9,10,11,16,22]

Nausea/vomiting occured in 50/84 (60%) of our cases. This was closer to 50 % cases of nausea and vomiting reported by Behera and Patnaik.[10] Khan et al noted vomiting in 57 % patients.[5] Nausea was reported by K Sravanthi et al in 88% and by Khan et al in 80 % of cases, which was higher compared to our

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findings.[5,9] Lower figures of the above features were reported from Sambhalpur 13% and Ahmedabad 40 %.[14,16]

Hepatomegaly was manifested by 38/84 (45%) patients; higher number of this finding was reported by K Sravanthi et al (80%) and Behera et al (68.8%) and lower by Desai et al (31.4%).[9,14,16]

Abdominal pain occurred in 34/84 (40%) in our study. Comparatively higher number of patients showing above presentation, was noted by Behera and Patnaik (95.8%), Khan et al (78.4%), Desai et al (65.7%) and K Sravanthi et al (57%) and fewer cases by Behera et al (21%).[5,9,10,14,16]

Darkcolored urine occurred in 13/84 (15%) of our cases. Comparatively high incidence was reported from Ahmedabad 84%, Karad 80% and Kurnool 74%.[9,16,22]

Itching/rash occurred in 7/84 (8.3%) of our patients which was lower than 27.1% observed by Behera et al.[14]

Thus we can see that varying clinical features were observed by different authors. Therefore an amalgamation of clinical profile and laboratory testing is required for accurate diagnosis.

Laboratory findings revealed increased serum bilirubin, aspartate and alanine aminotransferase (AST and ALT) levels in 57/84 (68%) in our study; AST >1000IU in 16/57 (28%) ALT 21/57(37%); bilirubin 5-10 mg/dL in16/57 (28%) and >5 mg/dL in 6/57 (11%). In Kurnool study elevation of all three parameters was seen in 100 % patients; AST >1000Ul 17%, ALT>1000Ul 11% and bilirubin 5-10 in 34%.[9] Behera et al reported 12.5% increased AST and 20.8% increased ALT.[14] Elevation of above parameters in 100% (all) patients was reported from Jaipur and Ahmedabad.[16,20] These findings of overall and individual levels of AST, ALT and bilirubin were comparable to our values.

Outcomewise 76/84 (90%) of our patients recovered and one died. Death rates closer to our finding was 3% from Jaipur and 2% Bhuvaneshwar.[10,20] Desai et al reported higher death rate (7.2%).[16] Sequelae of complication eg acute liver failure was reported by studies from Jaipur, Bhuvaneshwar and Ahmedabad.[10,16,20] Desai et al in addition also observed 25.7% cases of coagulopathy.[16] Although Joon et al reported 6 cases of fulminant hepatitis there was no mortality in 4 cases, however 2 patients with associated Wilson's disease died.[13]

We noted seasonal variation, reflecting increase number of samples during monsoon and beginning of winter. Similar observation was noted by Joon et al, Kalita et al and Agarwal s et al.[7,13,15] Samaddar et al noted more cases occurring in summer and monsoon whereas Gurav et al and Behera et al observed monsoon peak.[14,19,22]

CONCLUSIONS

The spread of HAV and HEV can be prevented by good hygiene practices, proper sanitation, and immunoprophylaxis. Thorough hand washing and careful food handling practices are essential. The U.S. Centers for Disease Control and Prevention recommends vaccination for healthy people aged 12 months to 40 years and immune globulin for children

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under 12 months, immunocompromised individuals and preferably for those aged over 40.[23]

Thus it is evident that various modalities are available for prevention. Immediate diagnosis and laboratory confirmation are of paramount importance to curtail transmission. Further studies are required to estimate the exact burden, to monitor evolving trends worldwide and to ascertain whether the strategies implemented are effective. This will lead us to the ultimate goal of global elimination.

Newer vistas of molecular epidemiology has evolved and play an important role in improving the understanding of this infection by identifying sources and the dynamics of virus evolution. Molecular biomarkers help to determine the genetic composition, track modes and chains of transmission and characterize the evolution of organisms. New approaches from disciplines such as bioinformatics and molecular evolution have added to the understanding of both the epidemiology of agents within host populations and need to be explored in future.[24]

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