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STATUS OF HEPATITIS A VIRUS (HAV) AND HEPATITIS E VIRUS (HEV) INFECTION IN THE PATIENTS PRESENTING WITH ACUTE VIRAL HEPATITIS ATTENDING A TERTIARY CARE HOSPITAL IN JHALAWAR, RAJASTHAN

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Abstract

Viral hepatitis refers to a primary infection and inflammation of the liver by any of the heterogenous group of hepatitis virus types A,B,C,D and E.[1] The condition can be self-limiting or can progress to fibrosis (scarring), cirrhosis or liver cancer. Hepatitis viruses are the most common cause of hepatitis in the world but other infections, toxic substances (e.g. alcohol, certain drugs), and autoimmune diseases can also cause hepatitis. There are 5 main hepatitis viruses, referred to as types A, B, C, D and E. These 5 types are of greatest concern because of the burden of illness and death they cause and the potential for outbreaks and epidemic spread. In particular, types B and C lead to chronic disease in hundreds of millions of people and, together, are the most common cause of liver cirrhosis and cancer. Around 400 million people all over the world suffer from chronic hepatitis and the Asia-Pacific region constitutes the epicentre of this epidemic.[2]

Introduction

Hepatitis A and E are typically caused by ingestion of contaminated food or water. Hepatitis B, C and D usually occur as a result of parenteral contact with infected body fluids. Common modes of transmission for these viruses include receipt of contaminated blood or blood products, invasive medical procedures using contaminated equipment and for hepatitis B transmission from mother to baby at birth, from family member to child, and also by sexual contact. Acute infection may occur with limited or no symptoms, or may include symptoms such as jaundice (yellowing of the skin and eyes), dark urine, extreme fatigue, nausea, vomiting and abdominal pain.

In India, the estimated burden of viral hepatitis is very high, necessitating focus on prevention and control measures of hepatitis to mitigate the morbidity and mortality due to hepatitis. HAV and HEV are important causes of acute viral hepatitis and acute liver failure. As these viruses continue to perplex clinicians and virologists alike, this review attempts to discuss the nuances in our understanding of this virus, its pathogenesis and diagnosis with reference to the Indian scenario.

Due to paucity of data, the exact burden of disease for the country is not established. Data thus obtained from this study will be essential for planning of future vaccination strategies and better sanitation program in this part of the country.

Material and Methods

Study site — The study was conducted at Viral Research & Diagnostic Laboratory in the Department of Microbiology of S.R.G Hospital & Jhalawar Medical College, Jhalawar, Rajasthan.

Study duration — The study was carried out for a period of 1 year i.e. November 2018 to November 2019 after approval from the institutional ethical committee.

Study subjects — After obtaining informed consent, complete clinical and epidemiological history of the all the patients satisfying the inclusion and exclusion criteria were recorded. The inclusion and exclusion criteria were as follows —

Inclusion criteria — All patients suspected of Acute Viral Hepatitis attending the outpatient & inpatient Departments of Medicine, Paediatrics and Obstetrics & Gynaecology were included in this study.

Exclusion criteria —

1. Patients with Chronic viral hepatitis with underlying with Hepatitis B and Hepatitis C.
2. Non-infective cases of jaundice (physiological, hereditary & acquired haemolytic anaemias, blood transfusion reactionary anaemia, obstructive jaundice, alcoholics, drug & toxin reactions, malignancies, as a complication of some primary disease.
3. Non - hepatotropic viral etiological cases of jaundice.
4. Non-viral etiological cases of jaundice.
5. Neonatal cases of jaundice.

Clinico-epidemiological history — A complete Epidemiological (Name, age, gender, address, source of drinking water, h/o travel, presence of similar cases in household or neighbourhood and practice of open defecation etc.), Clinical (history of hospital admission, duration of illness, clinical signs & symptoms like fever, nausea & vomiting, jaundice, dark urine, hepatomegaly & abdominal pain) & Laboratory diagnostic history (Liver function tests like SGOT, SGPT, S.bilirubin, ALP) were recorded in a requisition form.

Sample collection — 5-8ml of venous blood was collected with sterile and aseptic precautions in plain red-capped vials. It was allowed to clot for around 20 minutes and transported to Viral Research & Diagnostic Laboratory (VRDL) in the Department of Microbiology, where it was centrifuged at for 10mins, serum was separated and stored at -20^{o}C till analysis.

Observation & Results

Study population characteristics —

This study was conducted, after obtaining clearance from institutional ethics committee, on a total of 267 patients comprising of 154 (57.67%) Males & 113 (42.32%) Females within age groups ranging from 3months to 80 years of age. Out of 113 females, 27 (15.04%) cases were pregnant. Out of 267 cases, a total of 102 (38.02%) patients were hospitalised.

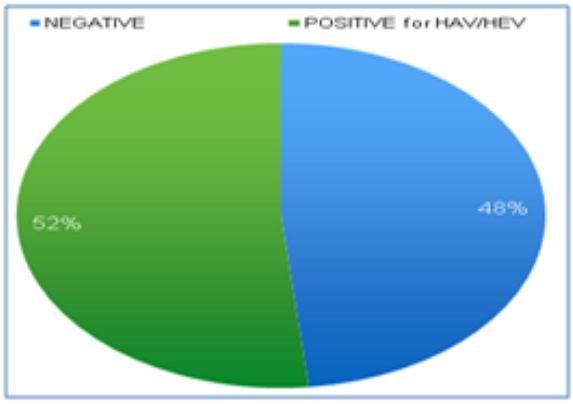

Figure 1: Total Seroprevalence of AVH due to HAV/HEV -

In this study, **52% (138/267) patients** were found to be suffering from Acute Viral Hepatitis either due to HAV or HEV, which implies that more than 50% burden of AVH comprises of HAV & HEV infection. (Fig 1)

Table 1: Seroprevalence of HAV & HEV -

HAV	**21.34%** (57/267)
HEV	**26.59%** (71/267)
HAV + HEV coinfection	**3.74%** (10/267)
TOTAL AFFECTED POPULATION	**51.68%** (138/267)

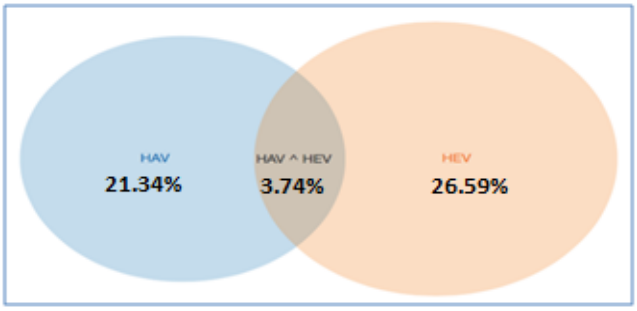

Figure 2: Seroprevalence of HAV & HEV -

HAV IgM antibodies were detected in 67 subjects, with an overall seroprevalence rate of **25.09%.** HEV antibodies were detected in 81 subjects, with an overall seroprevalence rate of **30.33%.** The overall incidence of **HEV** was found to be higher in the study population as compared to **HAV.** HAV & HEV co infection was found to be **3.74%**. [Table 1] (Fig 2)

Table 2: Gender wise distribution of HAV & HEV infected cases —

SEX	HAV	HEV
MALES	**17.53%** (27/154)	**33.11%** (51/154)
FEMALES	**35.39%** (40/113)	**26.54%** (30/113)
TOTAL	67	81

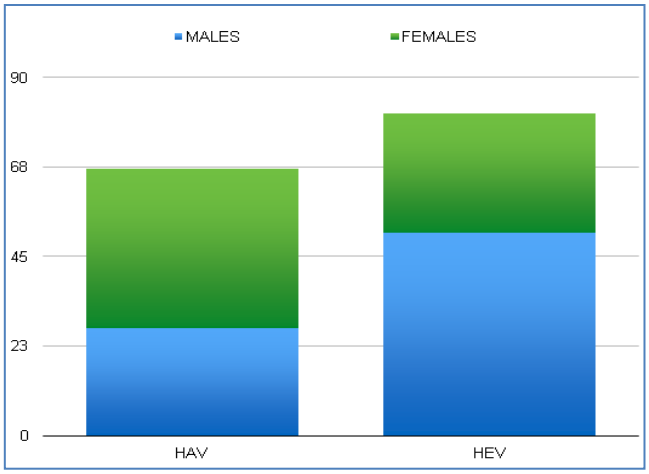

Figure 3: Gender wise distribution of HAV & HEV infected population

The seropositivity rate of HAV IgM was significantly higher in females **(35.39%)** as compared to males **(17.53%).** Whereas, the seropositivity rate of HEV IgM was found to be higher in males **(33.11%)** as compared to males **(26.54%).** [Table 2] (Figure 3)

Table 3: Gender comparison between HAV infected cases -

	HAV +ve	HAV -ve	TOTAL
MALES	27	127	154
FEMALES	40	73	113
TOTAL	67	200	267

Table 4: Gender comparison between HEV infected cases -

	HEV +ve	HEV -ve	TOTAL
MALES	51	103	154
FEMALES	30	83	113
TOTAL	81	186	267

Chi squared equals 10.137 with 1 degrees of freedom.

The gender comparison showing **females to be more prone to HAV infection** than males was found to be statistically significant. (**P value equals 0.0015)** [Table 3]Gender comparison in HEV infection was however not found to be statistically significant. [Table 4]

Table 5: Age wise distribution of cases in HAV & HEV infected population -

Age (years)	HAV +ve	HEV +ve	HAV+HEV
<10	**80.76%** (42/52)	**7.69%** (4/52)	**5.76%** (3/52)
11-20	**36.17%** (17/47)	**27.65%** (13/47)	**6.38%** (3/47)
21-30	**7.35%** (5/68)	**42.64%** (29/68)	**4.41%** (3/68)
31-40	0/39	**46.15%** (18/39)	0/39
41-50	**4.16%** (1/24)	**33.33%** (8/24)	**4.16%** (1/24)
51-60	**5.55%** (1/18)	**27.77%** (5/18)	0/18
>60	**5.26%** (1/19)	**21.05%** (4/19)	0/19
Total	67	81	10

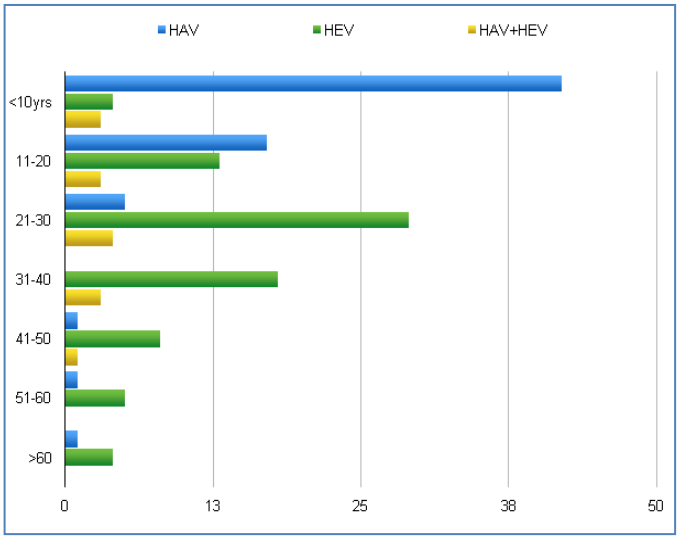

Figure 4: Age wise distribution of cases in HAV & HEV infected population -

Table 6: Age wise comparison of HAV infected population -

POPULATION	HAV +ve	HAV -ve	TOTAL
Paediatric	79.10% (53)	16	69
Adult	20.89% (14)	184	198
TOTAL	67	200	267

Chi squared equals 128.718 with 1 degrees of freedom.

The overall prevalence of **HAV infection is predominantly higher in paediatric population** as compared to adults and is considered to be extremely statistically significant. (P value is less than 0.0001) [Table 5]

Table 7: Age wise comparison of HAV infected population -

POPULATION	HEV +ve	HEV -ve	TOTAL
Paediatric	11.11% (9)	64	73
Adult	88.88% (72)	122	194
TOTAL	81	186	267

Chi squared equals 12.761 with 1 degrees of freedom.

The two-tailed **P value equals 0.0004**

The overall prevalence of **HEV infection is predominantly higher in adults** as compared to paediatric population and is considered to be extremely statistically significant. [Table 6]

Table 8: History of admission (OPD/IPD) in HAV & HEV infected population —

ADMISSION	HAV	HEV
OPD	**12.72%** (21/165)	**24.24%** (40/165)
IPD	**45.09%** (46/102)	**40.19%** (41/102)
TOTAL	67/267	81/267

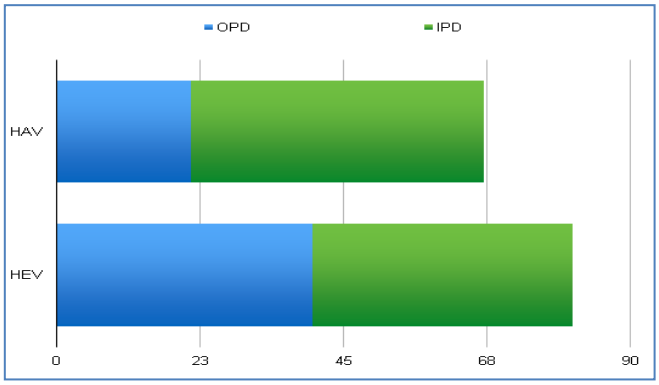

Figure 5: History of admission (OPD/IPD) in HAV & HEV infected population —

Chi squared equals 30.629 with 1 degrees of freedom. The rate of hospital admission as in-patient (IPD) was found to be higher in **HAV (45.09%)** infected individuals as compared to out patient (OPD) visit and this comparison was found to statistically significant. (P value is less than 0.0001) [Table 7] (Figure 5)

Chi squared equals 6.855 with 1 degrees of freedom. The rate of hospital admission as in-patient (IPD) seemed to be higher in **HEV (40.19%)** infected individuals as compared to outpatient (OPD) visit and the comparison was found to be statistically significant. (P value is less than 0.0088) [Table 7] (Figure 5)

Table 9: Area wise distribution (Urban/Rural) of HAV & HEV infected population —

AREA	HAV	HEV
URBAN	**25.64%** (50/195)	**31.79%** (62/195)
RURAL	**23.61%** (17/72)	**26.38%** (19/72)
TOTAL	67	81

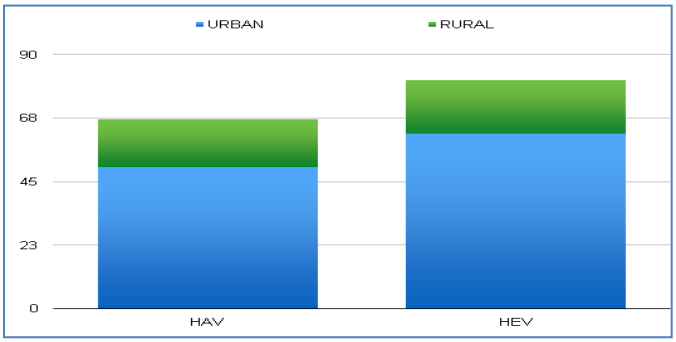

Figure 6: Area wise distribution (Urban/Rural) of HAV & HEV infected population —

In this study, the seropositivity rate of HAV and HEV in Urban (25.64% & 31.79%) population was slightly higher than rural areas (23.61% & 26.38%) areas respectively. However this difference was not found to be statistically significant. (Chi squared equals 0.006 with 1 degrees of freedom. The two-tailed P value equals 0.9378)

In this study, the rate of hospital admission as in-patient (IPD) was found to be higher in **HAV (45.09%)** infected individuals as compared to out patient (OPD) visit. The rate of hospital admission as in-patient (IPD) was found to be higher in **HEV (40.19%)** infected individuals as compared to out patient (OPD) visit. However this comparison was not found to be statistically significant. (Chi squared equals 0.006 with 1 degrees of freedom. The two-tailed P value equals 0.9378) [Table 7] (Figure 5)

Table 10: Source of drinking water in HAV & HEV infected population—

Source	HAV	HEV
Local Tubewell	**17.69%** (20/113)	**34.51%** (39/113)
Open well	**57.14%** (4/7)	**71.42%** (5/7)
Filtered tap water	0/18	**27.77%** (5/18)
Tap water	**33.33%** (43/129)	**24.80%** (32/129)
TOTAL	67	81

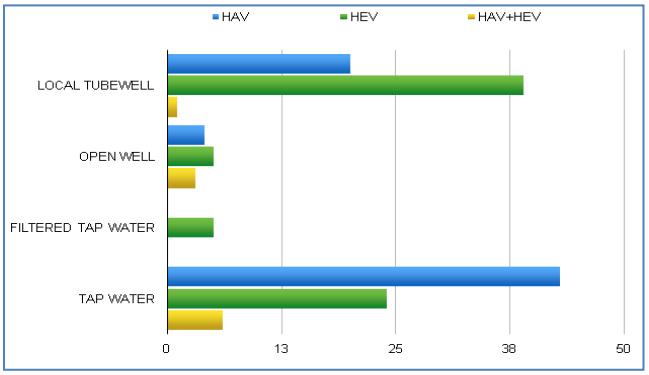

Figure 7: Source of drinking water in HAV & HEV infected population—

The overall incidence of **HAV & HEV** infection was found to be considerably higher in people utilising **Open Well** as the source of drinking water, with HEV **(71.42%)** infection predominating HAV **(57.14%)**, as compared to Tap water, Local tube well and Filtered tap water.

Table 11: History of Open Defecation in HAV & HEV infected population —

OPEN DEFECATION	HAV +ve	HEV +ve
YES	59.70% (40/67)	64.19% (52/81)
NO	43.28% (27/67)	35.80% (29/81)
TOTAL	67	81

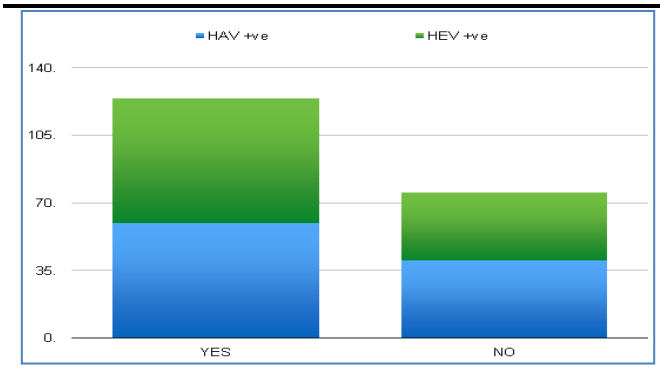

Figure 8: History of Open Defecation in population infected with HAV & HEV

Amongst the study population of this study, those patients who openly defecated were more prone to acquire **HAV (59.70%) & HEV (64.19%)** infection as opposed to those utilising proper sanitation. [Table 11] (Figure 8)

Table 12: Month wise distribution of HAV & HEV infected population —

MONTH	HAV	HEV	HAV+HEV	TOTAL
November	**9.52%** (2/21)	**19.04%** (4/21)	**14.28%** (3/21)	**42.85%** (9/21)
December	0/27	**29.62%** (8/27)	**7.40%** (2/27)	**37.03%** (10/27)
January	0/19	**57.89%** (11/19)	0/19	**57.89%** (11/19)
February	0/9	**55.55%** (5/9)	**11.11%** (1/9)	**66.66%** (6/9)
March	**12.50%** (1/8)	**37.50%** (3/8)	**12.50%** (1/8)	**62.50%** (5/8)
April	**7.69%** (1/13)	**38.46%** (5/13)	0/13	**46.15%** (6/13)
May	**19.14%** (9/47)	**23.40%** (11/47)	**2.12%** (1/47)	**44.68%** (21/47)
June	**33.33%** (11/33)	**18.18%** (6/33)	**3.03%** (1/33)	**54.54%** (18/33)
July	**23.80%** (5/21)	**28.57%** (6/21)	0/21	**52.38%** (11/21)
August	**25%** (6/24)	**12.5%** (3/24)	0/24	**37.5%** (9/24)
September	**58.33%** (14/24)	**20.83%** (5/24)	0/24	**79.16%** (19/24)
October	**38.09%** (8/21)	**19.04%** (4/21)	**4.76%** (1/21)	**61.90%** (13/21)
TOTAL	67	81	10	158

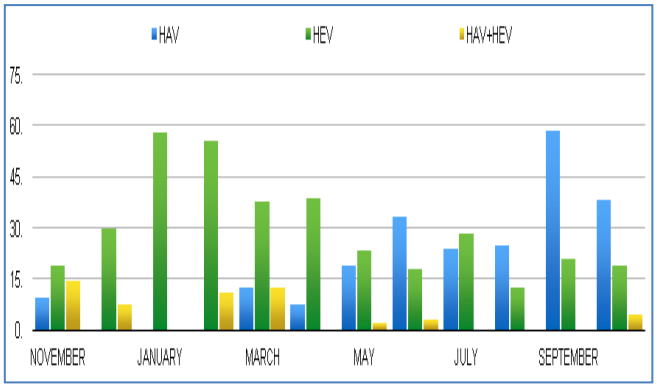

Figure 9: Month wise distribution of HAV & HEV infected population —

According to this study, HAV and HEV were seen to be prevalent all around the year with predominance seen towards the end of monsoons and beginning of winters and more so even the co-infection showed a similar seasonal trend. A peak in HEV was also noted in the beginning of rainy season. [Table 12] (Figure 9)

Table 13: Clinical manifestations of HAV & HEV infected population —

	HAV	HEV
Duration of illness		
<7d	**71.64%** (48/67)	**90.12%** (73/81)
>7d	**28.35%** (19/67)	**9.87%** (8/81)
Fever	**62.68%** (42/67)	**62.96%** (51/81)
Nausea/vomitting	**85.07%** (57/67)	**85.19%** (69/81)
Loss of appetite	**82.08%** (55/67)	**75.30%** (61/81)
Jaundice	**94.02%** (63/67)	**92.59%** (75/81)
Dark Urine	**79.10%** (53/67)	**62.96%** (51/81)
Abdominal Pain	**25.37%** (17/67)	**18.51%** (15/81)
Hepatomegaly	**16.41%** (11/67)	**11.11%** (9/81)

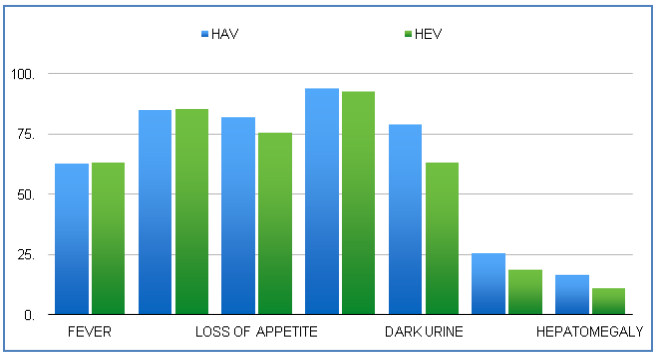

Figure 10: Clinical manifestations of HAV & HEV positive patients —

Table 14: Liver Function tests of HAV & HEV infected population -

LIVER FUNCTION TESTS	HAV +ve	HEV +ve
AST/ALT		
>1	**79.10%** (53/67)	**67.90%** (59/81)
<1	**20.89%** (14/67)	**27.16%** (22/81)
Elevated Bilirubin	**100%** (67/67)	**100%** (81/81)
Mean Bilirubin	45.79	41.06
Elevated ALP	**70.14%** (47/67)	**71.60%** (58/81)

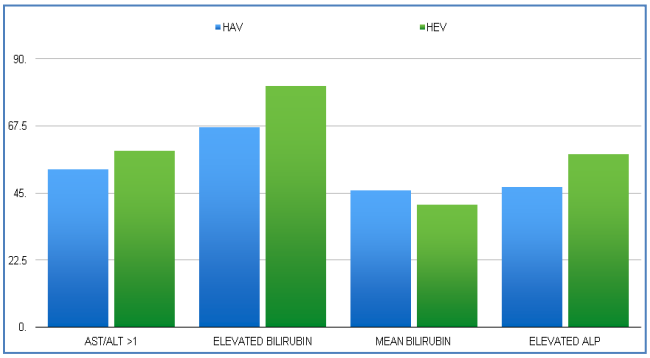

Figure 11: Liver Function tests of HAV & HEV positive patients -

Clinical characteristics of people affected with HAV & HEV were as depicted in Table. Also, individuals infected with either HAV or HEV had deranged liver function tests with elevated transaminases, bilirubin & ALP levels. [Table 13] (Figure 10)

The mean bilirubin of HAV infected patients were 45.79 IU/L , whereas those infected with HEV had their mean bilirubin around 41.06 IU/L. [Table 13] (Figure 11).

Discussion

In the present study, **52% (138/267) patients** were found to be suffering from Acute Viral Hepatitis either due to HAV or HEV. In 2016, a study done by Agrawal *et al.* on 475 samples, 181 samples were positive for HAV and/or HEV infection with an overall prevalence of 38.1%, which was comparatively lower than our study.[3] In 2015, another study by Joon *et al,* only 29.9% of the suspected cases had a positive viral marker which is comparatively lower than our study.[4] However in 2014, a population based cross-sectional study conducted by Vitral *et al.*, 359 out of 397 cases were found to be infected with HAV/HEV with an overall seropositivity rate of 90.42% which is higher when compared to our study.[5] Another study by Al-Naaimi *et al. in 2012.* suggested an overall seropositivity rate of 49%.[6]

In this study, HAV IgM antibodies were detected in **67** subjects, with an overall seroprevalence rate of **25.09%** and HEV antibodies were detected in **81** subjects, with an overall seroprevalence rate of **30.33%.** When compared with a study done in 2018 by Netra *et al.,* out of a total of 1751 serum samples, 908 serum samples were tested for IgM HAV and 843 serum samples were tested for IgM HEV. Of these 908 suspected HAV cases, IgM antibodies were detected in 73 (8.03%) serum samples. Among 843 suspected HEV cases, 185 (21.94%) serum samples were positive for IgM antibodies, is comparatively lower than our study finding.[7] In a viral hepatitis surveillance - India (2011– 2013) by Kumar *et al.* in 2015, 599605 cases tested for HAV, 7.4% were positive, and among 187040 samples tested for HEV, 10.4% were positive.[8]

The rate of co-infection in present study was found to be 3.74% which is in accordance with a recent study done in 2019 by Samaddar *et al.,*[9] with a co-infection rate of 2.07% and is much lower than the study conducted in 2018 by Netra *et al.,*[7] depicting HAV & HEV co-infection rate of 11.5%.

In the present study, the seropositivity rate of HAV IgM was significantly higher in females **(35.39%)** as compared to males **(17.53%).** Whereas, the seropositivity rate of HEV IgM was found to be higher in males **(33.11%)** as compared to males (26.54%). In a recent study done in 2019 by Samaddar *et al.,*[9] seropositivity rate of HAV & HEV among males was 7.3% & 8.8% respectively and in females 6.7% & 10.2% respectively, which was found to be lower than our study. However, in 2018, a study by Netra *et al.,*[7] the prevalence of both HAV [Males - 52 (71.2%) Females- 21

(28.8%)] and HEV [Males - 142 (76.7%), Females-43(23.3%)] was higher in males than in females, this finding is consistent with other studies *(*Al-Naaimi *et al., 2012;* Kamal *et al., 2010)*[10].

In this study, the overall prevalence of HAV infection was predominantly higher in paediatric population 79.10% (53/67) as compared to adults 20.89% (14/67) whereas the overall prevalence of HEV infection was predominantly higher in adults 88.88% (72/81) as compared to paediatric population 11.11% (9/81). In a study done 2018 by Netra *et al.,* HAV infection was more common in young children 79.11% (58/73) while the prevalence of HEV infection was more in adults 82.70% (153/185) in comparison to children.[7] In a recent study by 2017 by Agrawal *et al.,* HAV infection is a disease of infants and young children, but the same was not found in our study as children below < 15 years, older children and adult were found with the same seroprevalence; on the other side, we can say seroprevalence is decreasing in its respective age group. On the other side, HEV was found maximum positive (96) in population of 16-40 age group with in comparison to 18 children in the age group of < 15 years.[11]

In the present study, considering the source of drinking water of the cases included in our study, the overall incidence of **HAV & HEV** infection was found to be considerably higher in people utilising **Open Well** as the source of drinking water, with HEV **(71.42%)** infection predominating HAV **(57.14%)**, as compared to Tap water, Local tube well and Filtered tap water. In 2014, a population based cross-sectional study conducted by Vitral *et al*, the source of water utilised by their study subjects were either open well or river stream, amongst which only 308/379 subjects utilised filtered water as their source of drinking water out of which 83.40% were positive for HAV IgM and out of 302 subjects utilising filtered water as their source of drinking water, 13.20% were positive for HEV IgM.[5]

Amongst the study population of this study, those patients who openly defecated were more prone to acquire **HAV (59.70%) & HEV (64.19%)** infection as opposed to those utilising proper sanitation. When compared with a case study done in 2010 in Bhavnagar by Raval *et al.,* it was seen that out of 20 case-patients, 19 were found positive for hepatitis E virus (HEV) IgM antibodies. The water samples taken from households contained more than 10 coliforms in 100 ml sample. The relative risk of developing hepatitis E among people using pipeline water against those using ground water was 3.23 (95% CI of RR 1.59, 6.57), concluding that fecal contamination of water was the most likely source of this bimodal outbreak of hepatitis E in that area.[12]

According to this study, HAV and HEV were seen to be prevalent all around the year with predominance seen towards the end of monsoons and beginning of winters and more so even the co-infection showed a similar seasonal trend. A peak in HEV was also noted in the beginning of rainy season. This seasonal variation in transmission of acute viral hepatitis might be probably due to, to mixing of contaminated soil into wells, rivers and other common sources of drinking water during periods of heavy rains or floods. In a very recent study conducted by *Samaddar et al. in* 2019, HAV and HEV infections were prevalent all-round the year with maximum number of cases seen from May to September, that is, during summer and rainy seasons.[10]

In the present study, the clinical characteristics of people affected with HAV & HEV were evaluated under the following parameters, such as, duration of illness, fever, nausea & vomiting, loss of appetite, jaundice, dark urine, abdominal pain & hepatomegaly. In 2014, a study done by Sarker *et al.,* sowed that most of the icteric children presented with fever, anorexia and nausea /vomiting.[13]

Studies conducted by Kamaal *et al.,* Vitral *et al.,* Radhakrishnan *et al*. & Davalkhaam *et al.* also showed similar observations.[10,5,14,15]

In this study, individuals infected with either HAV & HEV had deranged liver function tests with elevated transaminases (79.10% & 67.90%), bilirubin & ALP (70.14% & 71.60%) levels. The mean bilirubin of HAV infected patients were 45.79 IU/L , whereas those infected with HEV had their mean bilirubin around 41.06 IU/L. In a recent study done by Samaddar *et al. i*n 2019, the total serum bilirubin (normal range: 0.2–1.2 mg/dL), AST (normal range: 10–40 U/L), ALT (normal range: 7–56 U/L) and ALP (normal range: 20–140 IU/L) levels were raised in 68.1%, 61.7%, 59.6% and 74.5% of the HAV-positive patients and in 78.5%, 72.3%, 61.5% and 70.8% of the HEV-positive patients, respectively. In HAV-HEV co-infection, it was observed that ALP level was raised in all patients while total bilirubin, AST and ALT levels were raised in 85.7%, 64.3% and 85.7% cases, respectively. Three patients with co-infection had very high levels of liver enzymes (AST and ALT >2000 IU/L). This suggests that dual infection with HAV and HEV can lead to severe disease manifestations such as acute liver failure and hepatic encephalopathy.[9]

Summary and Conclusion

It was found that more than half of the burden of AVH was caused by HAV & HEV combined. Also the overall seroprevalence of HEV was found to be comparatively more as than HAV. However, significant difference was observed in age wise distribution of HAV & HEV infection. Age distribution was remarkable. HAV infection is predominantly higher in paediatric population as compared to adults whereas HEV infection is predominantly higher in adults as compared to paediatric population. People openly defecating & utilising the drinking water of open wells were more prone to HAV &

HEV infection as compared to other sources of drinking water.

Even though the therapeutic options are limited in HAV and HEV infections, instituting proper surveillance and preventive measures is important not only for the care of patients but also for contacts who may be candidates for immunisation. Vaccines give active and long lasting protection against hepatitis A. As far as HEV is concerned, at least two distinct recombinant HEV vaccines have gone to clinical trials, but exact public health role of this vaccine remains unclear at this time and none has been sanctioned for use. As all HAV & HEV infections are spread by the faeco - oral route, good personal hygiene, high quality standards for public water supplies and proper disposal of sanitary waste will definitely reduce prevalence of these infections. Data thus obtained from this study will be essential for planning of future vaccination strategies and better sanitation program in this part of the country. The establishment of a national surveillance system for acute viral hepatitis can serve a vital role in early detection and control of infection and to monitor the impact of national sanitation and health care programs. Also, the knowledge of the strength and limitations of ELISA will allow the rational use and interpretation of results and it will be of immense help in arriving at a probable diagnosis of Acute Viral Hepatitis.

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