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Original Research

Assessment of hepatitis A and E viruses in patients of acute viral hepatitis

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ABSTRACT:

Background: HAV is one of the major feco-orally transmitted agents and responsible for 1.4 million new cases per year. The present study was conducted to hepatitis A and E viruses in patients of acute viral hepatitis. **Materials & Methods:** 56 patients of acute viral hepatitis of both genders were enrolled. 5 ml blood samples were collected aseptically. Serum was assessed for anti HAV immunoglobulin M (IgM) and anti-HEV IgM antibodies. All analyses were performed using commercial kits based on the enzyme-linked immunosorbent assay. **Results:** Out of 56 patients, males were 32 and females were 24.Hepatitis A was seen in 20, Hepatitis E in 25 and Hepatitis A+ E in 11 patients. The clinical features found in Hepatitis A and Hepatitis E was pallor seen in 42 and 40, encephalopathy in 38 and 27, hepatomegaly in 26 and 13 and yellowish discoloration in 52 and 54 respectively. The difference was significant (P< 0.05). **Conclusion:** Common clinical features found to be pallor, encephalopathy, hepatomegaly and yellowish discoloration.

Key words: Hepatitis A, encephalopathy, hepatomegaly

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INTRODUCTION

Acute viral hepatitis is a systemic infection affecting theliver predominantly.HAV is one of the major feco-orally transmitted agents and responsible for 1.4 million new cases per year. HAV is an RNA virus belonging to the genus Hepatovirus and family Picornaviridae. Anti-HAV antibodies in human sera are detectable in acute illness as serum liver enzyme level reaches a high level and fecal HAV shedding is still going on.[1] IgM class antibody elevates initially persisting for a few months (6-12 months usually). In the later stage (convalescence) IgG class of anti-HAV emerges predominantly. HAV is a self-limiting infection without any chronic sequelae. From the early 1990s, a safe and effective vaccine became available against HAV infection.[2]

Hepatitis E virus (HEV) is another agent transmitted feco-orally and predominantly found in Asia, parts of Africa, and in core Central America. It is an RNA virus falling under the genus Hepevirus of the family Hepeviridae.[3] The anti–HEV IgM antibody appears soon after an acute infection and falls to a very low level within 6 months of infection.There is a significant body of evidence that suggests that although both HAV and HEV are non-enveloped viruses, they can also enjoy at least some of the benefits of enveloped viruses.[4] HAV and HEV virions, which are shed in the stool, are naked protein capsids, ideally suited to their role of reaching new hosts across both time and distance in a potentially hostile environment.[5]The present study was conducted to hepatitis A and E viruses in patients of acute viralhepatitis.

MATERIALS & METHODS

The present study comprised of 56 patients of acute viral hepatitis of both genders. The consent was obtained from all enrolled patients.

Data such as name, age, gender etc. was recorded. 5 ml blood samples were collected aseptically. The venipuncture site was cleaned with soap and water, rinsed with sterile water and 1-2% tincture iodine or povidone iodine was applied and allowed to dry for 1-2 min (povidone-iodine) or 30 seconds. The blood was collected in sterile serum tubes. The tubes were then transported to the laboratory and kept on the rack and allowed to clot. Serum was assessed for anti HAV immunoglobulin M (IgM) and anti-HEV IgM antibodies. All analyses were performed using commercial kits based on the enzyme-linked

immunosorbent assay. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 56		
Gender	**Males**	**Females**
Number	32	24

Table I shows thatout of 56 patients, males were 32 and females were 24.

Table II Hepatitis A and E

Viral infection	Number	P value
Hepatitis A	20	0.91
Hepatitis E	25	
Hepatitis A+ E	11	

Table II, graph I shows that Hepatitis A was seen in 20, Hepatitis E in 25 and Hepatitis A+ E in 11 patients. The difference was significant (P< 0.05).

Graph I Hepatitis A and E

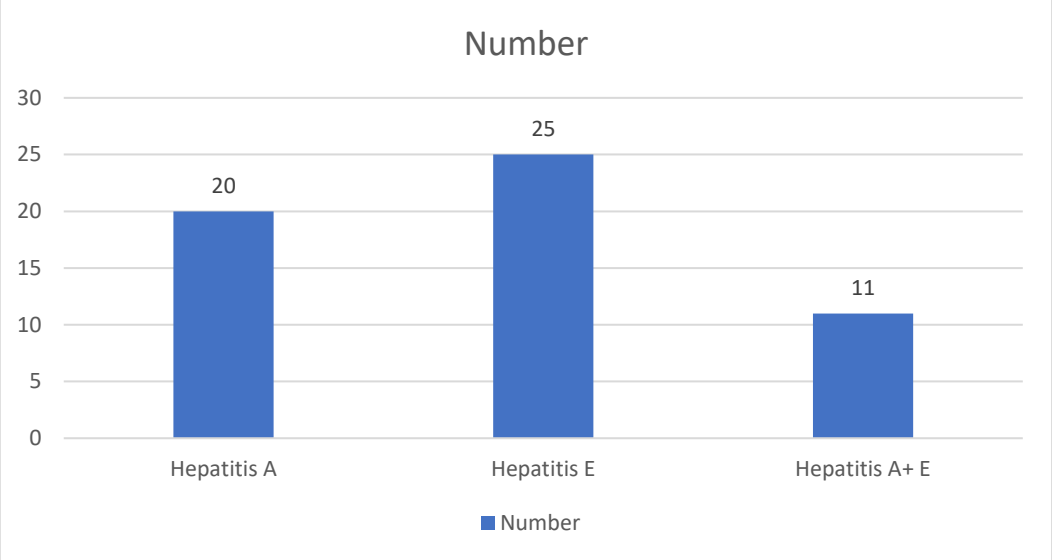

Table III Assessment of clinical profile

Clinical profile	Hepatitis A	Hepatitis E	P value
Pallor	42	40	0.94
Encephalopathy	38	27	0.05
Hepatomegaly	26	13	0.02
Yellowish discoloration	52	54	0.95

Table III shows that clinical features found in Hepatitis A and Hepatitis E was pallor seen in 42 and 40, encephalopathy in 38 and 27, hepatomegaly in 26 and 13 and yellowish discoloration in 52 and 54 respectively. The difference was significant (P< 0.05).

DISCUSSION

The primary route of transmission for HAV is fecal-oral, primarily through direct person-to-person contact, but also via contaminated food or water.[6] Men who have sex with men are at increased risk of infection, as are any persons engaging in oral-anal sexual contact regardless of gender or sexual orientation. Parenteral transmission via contaminated blood products has been described and injecting drug users are at high risk, with increased prevalence positively correlated with low incomes.[7] Infected individuals shed virus in their stool for around 2 weeks before becoming symptomatic and typically for a few days after but may continue to do so for several weeks. Even with good standards of hygiene and sanitation facilities, the rate of infection in close contacts of cases is high, suggesting very efficient interpersonal transmission.[8]The present study was conducted to hepatitis A and E viruses in patients of acute viral hepatitis.

We found that out of 56 patients, males were 32 and females were 24.Takahashi et al[9] reported that in

HEV, risk and severity of AVH enhance with age. A relevant point to note here is that HEV infection is mostly manifested without icterus and hence missed in children.

We found that Hepatitis A was seen in 20, Hepatitis Ein 25 and Hepatitis A+ E in 11 patients.We found that clinical features found in Hepatitis A and Hepatitis E was pallor seen in 42 and 40, encephalopathy in 38 and 27, hepatomegaly in 26 and 13 and yellowish discoloration in 52 and 54 respectively. The main methods by which the spread of HAV can be prevented are good hygiene practices, proper sanitation, case investigation and contact tracing during outbreaks, and active and passive immunoprophylaxis.[10] Thorough hand washing and careful food handling practices are of great importance. Postexposure prophylaxis can be considered for close contacts of infected individuals, subject to local guidelines. This can consist of either vaccination or intramuscular immune globulin.[11] The U.S. Centers for Disease Control and Prevention recommends vaccination for healthy people aged 12 months to 40 years; immune globulin for those aged over 40, although vaccination is acceptable; and immune globulin for children under 12 months, immunocompromised individuals, patients with chronic liver disease, and anyone with contraindications for vaccination.[12]

CONCLUSION

Authors found that common clinical features found to be pallor, encephalopathy, hepatomegaly and yellowish discoloration.

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