Original Research Article

Study of clinical profile of patients with Hepatitis E infection

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Abstract

Hepatitis E is one of the most important cause of acute clinical hepatitis in adults throughout Asia, the MiddleEast and Africa. Present study was aimed at finding out the clinical features of hepatitis Einfection. The study concluded that when compared with liver function tests, study had no statistical correlation with Hepatitis E infection.

Keywords: Hepatitis E,Clinical, hepatitis.

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Introduction

Hepatitis E is one of the commonest causes of acute clinical hepatitis in adults throughout Asia, the MiddleEast and Africa[1]. Hepatitis E virus (HEV) is a small round RNA-containing virus that is the only member of the genus Hepavirus in the family Hepaviridae[1].HEV accounts for more than 50% of acute viral hepatitis in young adults in developing countries[3]. Initial symptoms of acute hepatitis E are typically non-specific and include flu-like myalgia, arthralgia, weakness and vomiting.However,more severe forms of acute liver disease can occur in pregnant women or patients with underlying chronic liver diseases, sometimes progressing to fulminant hepatic failure[3]. The most frequent clinical and biological signs of HEV infection are jaundice, vomiting, hepatalgia, hepatomegaly, asthenia, distended abdomen, fever and high levels of transaminases (ALT and AST)[3].

Objective

To study the clinical and laboratory profile of patient with hepatitis Einfection.

Methods

The study was conducted on 50 patients diagnosed with HEV infection at Mahatma Gandhi Mission Hospital, Kamothe and Mahatma Gandhi Mission hospital, Kalamboli, Navi Mumbai. Patients were enrolled after matching inclusion and exclusion criteria.

Inclusion Criteria

1. Age grp-12-65yrs.
2. No genderspecification.
3. Diagnosed Hepatitis E infection

Exclusion Criteria

1. Patients of hepatitis A, B, C, D were excluded.
2. Alcoholic population.
3. Hepatotoxic drugs (for eg:-statins, isoniazid etc) were excluded from the study.
4. Study excluded Infections which affect liver (for eg:-plasmodium falciparum and plasmodium vivax, amoebic liver abscess)
5. Other liver diseases like nonalcoholic steatohepatitis, inborn errors of metabolism and genetic liver diseases.

Investigations

- CBC,
- LFT,
- RFT,
- Serum Creatinine,
- PT-INR,
- Anti HEV-IgM,
- RT-PCR for HEV Genotype if affording.

Statistical Analysis

Data collected was analyzed by using specialized software keeping the objective of the study in mind.

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Table 1:Mean total and direct bilirubin level at baseline, day 7 and day 15 amongst study population

	Mean	Std. Deviation	P value
Total Bilirubin	5.3	3.3	
Day7	5.9	4.0	0.085
Day15	5.0	5.1	0.678
Direct Bilirubin	3.6	2.5	
Day7	3.6	2.4	0.876
Day15	8.5	3.7	0.383

As seen in the above table, there was no statistically significant difference in mean total and direct bilirubin level at baseline, day 7 and day 15. The total bilirubin was at presentation, day 7 and day 15 was 5.3 ± 3.3, 5.9 ± 4, 5 ± 5.1 respectively while direct bilirubin was at presentation, day 7 and day 15 was 3.6 ± 2.5, 3.6 ± 2.4, 8.5 ± 3.7 respectively.

Fig 1: Mean total bilirubin levels at day1, day 7 and day 15 amongst study population

Fig 2: direct bilirubin levels at day 1, day 7 and day 15 amongst study population

Table 2:Mean SGOT, SGPT and Alkaline Phosphatase level at baseline, day 7 and day 15 amongst study population

	Mean	Std. Deviation	P value
SGOT	820.4	2343.8	
Day7	563.0	580.9	0.411
Day15	390.1	390.8	0.204
SGPT	594.8	721.5	
Day7	572.0	704.5	0.620
Day15	370.9	431.1	0.010
Alkaline Phosphatase	191.1	121.8	
Day7	202.9	124.8	0.197
Day15	197.5	164.5	0.677

As seen in the above table, there was no statistically significant difference in mean SGOT and Alkaline Phosphatase level at baseline, day 7 and day 15 while there was significant fall in SGPT at day 15 as compared to baseline and day 7. The mean SGOT was at presentation, day 7 and day 15 was 820.4 ± 2343.8, 563 ± 580.9, 390.1 ± 390.8 respectively while SGPT was atpresentation, day 7 and day 15 was 594.8 ± 721.5, 572 ± 704.5, 370.9 ± 431.1 respectively and whileAlkaline Phosphatase was at presentation, day 7 and day 15 was 191.1 ± 121.8, 202.9 ± 124.8, 197.5 ± 164.5 respectively.

Fig 3:Mean SGOT levels at day 1, day 7 and day 15 amongst study population

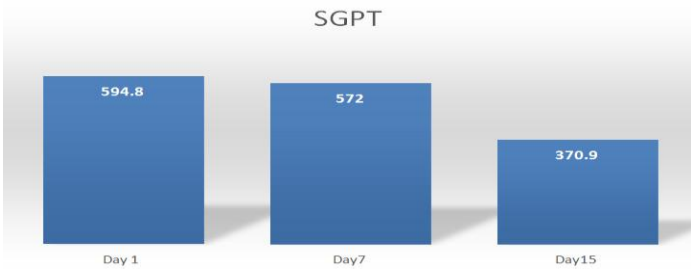

Fig 4:Mean SGPT levels at day 1,day 7 and day 15 amongst study population

Fig 5: Mean Alkaline phosphatase levels at day 1, day 7 and day 15 amongst study population

Table 3: Mean Total Protein and Albumin level at baseline, day 7 and day 15 amongst study population

	Mean	Std. Deviation	P value
Total Protein	6.4	0.7	
Day7	6.3	0.9	0.239
Day15	6.2	0.8	0.039
Albumin	3.5	0.8	
Day7	3.4	0.8	0.597
Day15	3.4	0.8	0.365

As seen in the above table, there was no statistically significant difference in mean albumin level atbaseline,day7and day15while there was significant fall in total protein at day15as compared to baseline and day 7.

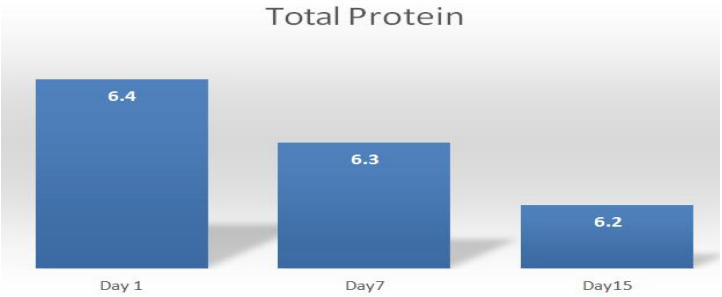

Fig 6:Mean Total protein levels at day1, day 7 and day 15 amongst study population

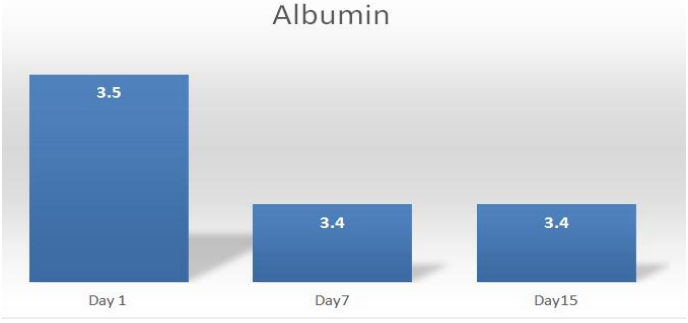

Fig 7:Mean albumin levels at day 1,day 7 and day 15 amongst study population

Table 4 :Mean PT and INR at baseline, day 7 and day 15 amongst study population

	Mean	Std. Deviation	P value
PT	14.6	2.1	
Day7	15.0	1.5	0.179
Day15	14.5	1.8	0.721
INR	1.1	0.4	
Day7	1.0	0.2	0.81
Day15	1.0	0.2	0.79

As seen in the above table, there was no statistically significant difference in mean PT and INR at baseline, day 7 and day 15.

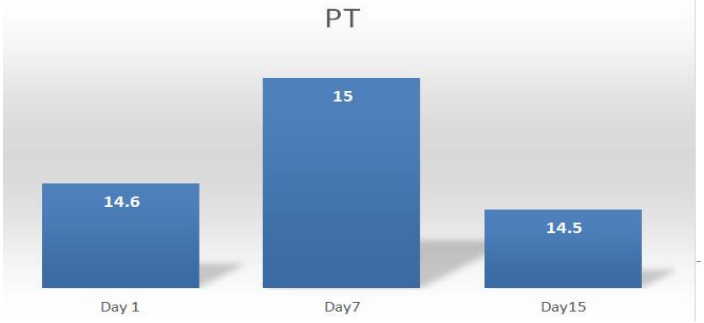

Fig 8:Mean PT levels at day 1,day 7 and day 15 amongst study populations

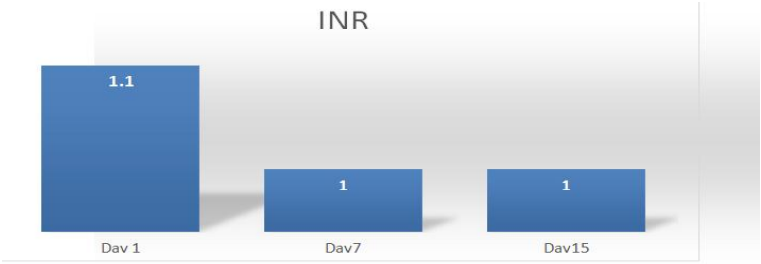

Fig 9:Mean INR levels at day 1, day 7 and day 15 amongst study population

Table 5: Correlation of Serum HEV IgM and liver function test amongst study population

		Total Bilirubin	Direct Bilirubin	SGOT	SGPT	Alk Po4
Serum HEV IgM	Pearson Correlation	.055	-.096	-.004	-.069	-.047
	Sig. (2-tailed)	.705	.506	.977	.635	.744
	N	50	50	50	50	50

As seen in the above table, there was no statistically significant correlation was observed between Serum HEV IgM and Total Bilirubin, direct Bilirubin, SGOT, SGPT and Alkaline Phosphatase.

Table 6: Correlation of Serum HEV IgM and Total protein, albumin, PT and INR amongst study population

		Total Protein	Albumin	PT	INR
Serum HEV IgM	Pearson Correlation	-.120	.194	-.239	-.148
	Sig. (2-tailed)	.408	.178	.095	.305
	N	50	50	50	50

As seen in the above table, there was no statistically significant correlation was observed between Serum HEV IgM and Total protein, direct albumin, PT and INR.

Results and Discussion

In the present study, there was no statistically significant difference in mean total and direct bilirubin level at baseline,day 7 and day15.Thetotal bilirubin was at presentation,day7and day 15 was 5.3 ± 3.3, 5.9 ± 4, 5 ± 5.1 respectively while direct bilirubin was at presentation, day 7 and day 15 was 3.6 ± 2.5, 3.6 ± 2.4, 8.5 ± 3.7 respectively.In the present study, there was no statistically significant difference in mean SGOT and Alkaline Phosphatase level at baseline, day 7 and day 15 while there was significant fall in SGPT at day 15 as compared to baseline and day7.The mean SGOT was at presentation,day7 and day 15 was 820.4 ± 2343.8, 563 ± 580.9, 390.1 ± 390.8 respectively while SGPT was at presentation, day 7 and day 15 was 594.8 ± 721.5, 572 ± 704.5, 370.9 ± 431.1 respectively and while Alkaline Phosphatase was at presentation, day 7 and day 15 was 191.1 ± 121.8, 202.9 ± 124.8, 197.5 ± 164.5 respectively.This findings is in agreement with the study conducted by TejasN Modi etal., observed that on presentation serum bilirubin level was elevated in100% patients with ALT raised in 86%. They found that 34%

of patients presented with bilirubin in range of 2.1-10 mg/dl and 43% with ALT in range of 500-1,000. Values started decreasing by 2nd week and laboratory profile of all survivors (93) became normal by 8th week[1]. In the present study, there was no statistically significant difference in mean albumin level at baseline, day 7 and day 15 while there was significant fall in total protein at day 15 as compared to baseline and day 7. This findings correlate well with the study conducted by YK Gurav et al., 2007 observed the mean serum bilirubin was 4 mg/dl, mean S.G.O.T. was 270 IU, mean S.G.P.T. was 294 IU, and mean serum alkaline phosphatase was 321 IU[3].Similarly in the study conducted by KBashir et al.,reported that the values of alkaline phosphatase (ALP), serum glutamate pyruvate transaminase (SGPT), serum glutamate oxaloacetate transaminase (SGOT) and bilirubin were increased in positive patients as compared with the control group whereas there was no effect on albumin and total protein[1].The HEVaffects the hepatocytes resulting in the abnormal function of the liver.The development of jaundice is a characteristic feature of viral infection. A comparison of liver function tests in HEV seropositive and seronegative patients revealed that liver enzymes were raised in hepatitis E patients indicating the malfunctioning of the liver. SGPT and SGOT are important liver enzymes that help to process proteins.Theymight be raised if the liverisinflame dorinjured.ALPenzymes might be raised when there is a blockage in the liver or bile duct. It has been reported in a case study that the function of the liver shows abnormal values for SGPT, SGOT and ALP due to HEV[1].In the present study, there was no statistically significant difference in mean PT and INR at baseline, day 7 and day 15.In the present study,there was no statistically significant correlation observed between Serum HEV IgM and Total Bilirubin, direct Bilirubin, SGOT, SGPT and Alkaline Phosphatase. Similarly in the study conducted by Nidhi Chandra et al., reported that anti-HEV IgM positivity was significantly associated with higher levels of serum alanine aminotransferase, aspartate aminotr-ansferase, alkaline phosphatase and total bilirubin[1].In the present study,there was no statistically significant correlation observedbetweenSerum HEV IgM and Total protein, direct albumin, PT and INR.Globally, 57 000 deaths and 3.4 million cases of acute hepatitis E are attributable to infection with HEV genotypes 1 and 2. Over 60% of all hepatitis E infections and 65% of all hepatitis E deaths occur in East and South Asia, where seroprevalence rates of 25% are common in some age groups. An epidemiologic feature that distinguishes HEV from other enteric agents is the rarity of secondary person-to-person spread from infected persons to their close contacts[2].

Conclusion

The study concluded that when compared with liver function tests, study had no statistical correlation with Hepatitis E infection.

Conflict of Interest: Nil
Source of support:Nil

References

1. Tam AW,Smith MM,Guerra ME,HuangCC,Bradley DW,Fry KE,Reyes GR.Hepatitis E virus (HEV): molecular cloning and sequencing of the full-length viral genome. Virology.1991;185:120–131.
2. Kamar N,Bendall R,Legrand-Abravanel F,XiaNS, IjazS ,Izopet J,Dalton HR.Hepatitis E. Lancet.2012;379:2477–2488.
3. Wang L, Zhuang H. Hepatitis E: an overview and recent advances in vaccine research. World J Gastroenterol. 2004;10:2157–2162.
4. Bhatia V, Singhal A, Panda SK, Acharya SK. A 20-year single center experience with acute liver failure during pregnancy: is the prognosis really worse? Hepatology. 2008;48:1577-85.
5. Panda SK, Thakral D, Rehman S: Hepatitis E virus. Rev Med Virol. 2007, 17:151-180.
6. TejasNModi,AStudy ofClinical Profile and Outcome in Acute Viral Hepatitis E, Indian Journal of Clinical Practice, 2013; 23(10):635-37.
7. Bashir K, Hussain N, Hasnain S, Elahi S. Seroprevalence of hepatitis E virus immunoglobulin G and M antibodies in adults: A hospital-based study. Indian J Med Microbiol2009;27:139-41.
8. Okuda K, Hai Y. Acute hepatitis and acute hepatic failure. In: Hepatobiliary diseases: Pathophysiology and Imaging, Ist ed. Okuda K, Mitchell DG, Itai Y, AriyamaJ. Eds., (Blackwell Science, U.K)2001:88-89.
9. Borgohain S, Agarwal AK, Dudeja RK, Sumeet S, Abhijeet R. Acutepancreatitis associated with acute hepatitis E virus infection (case report). J Indian Acad Clin Med 2000; 3:282-4.
10. Nidhi Subhash Chandra, Contribution of hepatitis E virus in acute sporadic hepatitis in north western India, Indian J Med Res. 2012; 136(3):477–482.
11. Longo DL, Fauci AS, Kasper DL, et al., editors. Harrison's Principles of Internal Medicine. 18th ed. Vol 2. New York: McGraw Hill Inc; 2012. Acute viral hepatitis. By Jules L. Dienstag; p.2548.