Original Research Paper

Volume - 13 | Issue - 07 | July - 2023 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Biochemistry

ASSOCIATION OF HEPATITIS C VIRUS WITH SERUM BILIRUBIN & LIVER ENZYMES

Ishita Jain* Senior Resident Department of Biochemistry IGMC Shimla HP *Corresponding Author

Nita Garg Professor & Head Department of Biochemistry ASMC Firozabad UP

ABSTRACT **Aim:** To estimate an association between Hepatitis C virus with serum bilirubin and liver enzymes. **Study Design:** The study comprised of 133 Hepatitis C patients attending the out patient Department of Medicine of Shri Mahant Indiresh Hospital Dehradun,(UK) for a period of 12 months from Jan 2017 to Dec 2017. Out of the 133 Hepatitis C seropositive patients 74 were males and 59 were females. The blood samples from all 133 Hepatitis C seropositive patients were subjected to HCV RNA viral load quantification and estimation of Total Bilirubin and liver enzymes namely AST, ALT & GGT were performed. Out of 133 samples analyzed for HCV RNA viral load, target was detected in 72 (54.16 %) cases and in 61 (45.86 %) samples, target was not detected. So 72 cases (37 Males + 35 Females) and 61 controls (37 Males+ 24 Females) were considered for this study. **Methodolgy:** 5 ml of blood was collected from all the Anti-HCV Ab +ve patients and were subjected to estimation of liver panel of enzymes and Serum Bilirubin. **Results:** Our study showed that the levels of AST,ALT,GGT and Total bilirubin were significantly higher in the cases both male and female than in the respective controls. ALP levels showed no significant difference in the study group.

KEYWORDS : HCV, ALT, AST, GGT, ALP.

INTRODUCTION:

Hepatitis C, a serious viral disease of the liver and caused by Hepatitis C virus infection is a major health issue of global concern [1-2]. Earlier known as Non A, Non B hepatitis, it was identified as putative viral hepatitis occurring after transfusion of blood products or intravenous drug use. There was evidence that Non A, Non B hepatitis could lead to presistent infection in a high proportion of infected individuals and could progress to chronic liver disease, cirrhosis and hepatocellular carcinoma. HCV was discovered to be the major cause of Non A, Non B Hepatitis in 1989 and is known to be a leading cause of chronic liver disease in developing countries. The annual mortality due to HCV related liver diseases is about 3.5-5 million [3-4].

HCV is a positive-sense, single-stranded enveloped RNA virus approx 9600 nucleotide in length, HCV also displays remarkable genetic diversity & propensity for selection of immune evasion or drug resistant mutations.

AST and ALT are aminotransferase which require pyridoxal phosphate as coenzyme, effect of pyridoxal phosphate deficiency is greater on ALT activity than on AST [6-7]. Both aminotransferases are highly concentrated in liver.

In cases of acute viral hepatitis, aminotransferase levels usually peak before jaundice appears and have more gradual decrease thereafter, and there is a greater increase in serum bilirubin levels[8]. It is important to emphasize that the degree of aminotransferase alteration is a poor guide to the severity of the disease in patients with established chronic viral hepatitis unless an AST/ALT ratio greater than 1 is found [9].

Liver and bone diseases are the most common causes of pathological elevation of ALP levels, althrough ALP may originate from other tissues such as placenta, kidneys or intestines or from leucocytes [10]. Cholestasis enhances synthesis & release of ALP and accumulating bile salts increase its release from cell surface [11].

GGT is an enzyme that is present in hepatocytes and biliary epithelial cells. GGT is a microsomal enzyme and its activity can be induced by several drugs such as anticoagulants & oral contraceptives .[12]

If test results for HCV antibodies are negative and there is no evidence of Acute Hepatitis A or B infection, testing for HCV RNA may be a useful strategy since recent studies show that these patients are rarely at a risk of acute liver failure but they may benefit from early antiviral treatment.[13]

MATERIALAND METHODOLOGY :

The study comprised of 133 Hepatitis C patients attending the out patient Department of Medicine of Shri Mahant Indiresh Hospital Dehradun,(UK) for a period of 12 months from Jan 2017 to Dec 2017. Out of the 133 Hepatitis C seropositive patients 74 were males and 59 were females. The blood samples from all 133 Hepatitis C seropositive patients were subjected to HCV RNA viral load quantification and estimation of liver enzymes namely ALT (14), AST (15), ALP (16) GGT (17) and Total bilirubin (18) were performed.

Out of 133 samples analyzed for HCV RNA viral load, target was detected in 72 (54.16 %) cases and in 61 (45.86 %) samples, target was not detected. So 72 cases (37 Males + 35 Females) and 61 controls (37 Males+ 24 Females) were considered for this study.

RESULTS:

The data so obtained was subjected to statistical analysis using SPSS Software. The results were expressed as Mean ± SD ± SE. The t-values and P- values were calculated using the Unpaired t Test. ANOVA or Test of Variance was used to analyse the Liver enzyme and total Bilirubin levels amongst patients infected with different HCV genotypes. The result are tabulated and depicted graphically.

Table 01 : Comparison of Liver Enzymes and Total Bilirubin levels between Cases and Controls:

Parameter	Cases (72)	Controls (61)	CI (95%)	df	t-value	p-value	Significance
AST(U/L)	88.53±53.62±6.16	42.39±33.82±4.44	-61.84 to -30.43	131	5.81	<0.001	HS
ALT(U/L)	85.47±46.50±5.34	45.82±41.75±5.49	-54.93 to -24.36	131	5.133	<0.0001	HS
ALP(U/L)	138.99±64.94±7.46	120.39±61.93±8.14	-40.48 to 3.28	131	1.681	0.09	NS
GGT(U/L)	72.65±65.51±7.53	46.16±40.60±5.34	25.74 to 69.73	131	2.74	0.007	S
TB(mg/dl)	2.20±0.82±0.09	1.20±1.16±0.15	-1.34 to -0.66	131	5.80	<0.0001	HS

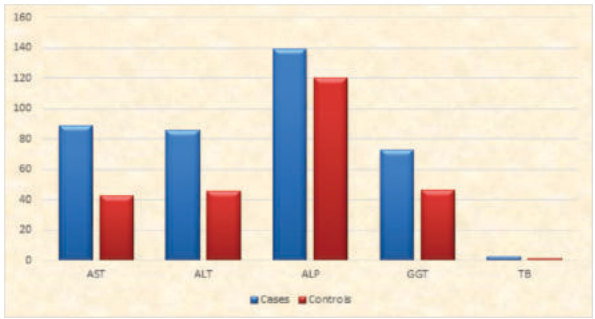


Table 02: Comparison of Liver Enzymes and Total Bilirubin levels between Male cases and Male controls:

Parameter	Male cases (37)	Male controls (37)	CI(95 %)	df	t-value	P -value	Significance
AST(U/L)	94.51±61.84±10.14	49.70±40.77±6.61	-0.34 to 0.43	72	3.68	0.0004	S
ALT(U/L)	85.19±30.36±4.98	54.24±51.35±8.32	-50.49 to -11.40	72	3.15	0.0023	S
ALP(U/L)	148.49±76.75±12.58	129.81±70.04±11.35	-52.73 to 15.37	72	1.09	0.27	NS
GGT(U/L)	62.05±49.26±8.08	60.46±54.74±8.87	-25.72 to 22.54	72	0.13	0.89	NS
TB(mg/dl)	2.17±0.81±0.13	1.34±0.83±0.14	-1.21 to -0.45	72	4.35	<0.0001	HS

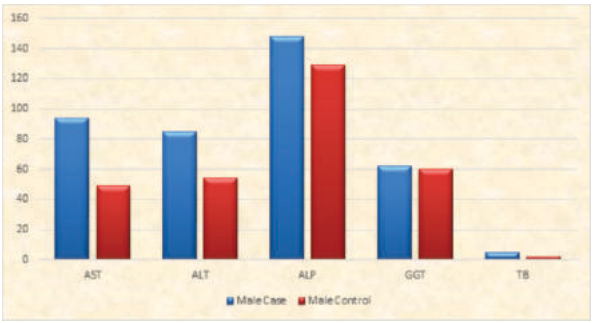
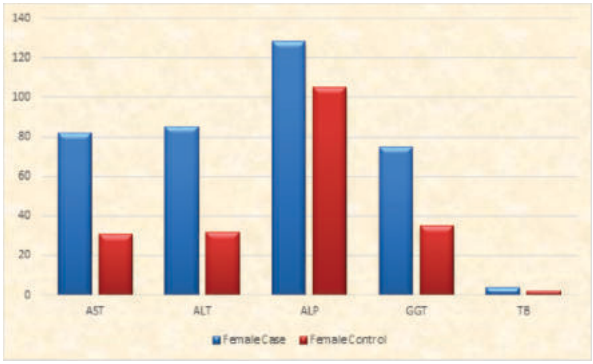


Table 03 : Comparison of Liver Enzymes and Total Bilirubin levels between Female Cases and Female Controls:

Parameter	Female cases (35)	Female controls (24)	CI (95%)	df	t-value	p-value	Significance
AST(U/L)	82.20±43.30±7.40	31.13±12.77±2.66	-69.33 to -32.81	57	5.63	0.0001	HS
ALT(U/L)	85.77±59.48±10.17	32.83±11.40±2.38	-77.62 to -28.26	57	4.29	0.0001	HS
ALP(U/L)	128.94±48.66±8.32	105.83±44.29±9.23	-48.02 to 1.80	57	1.87	0.07	NS
GGT(U/L)	75.86±63.04±10.78	35.71±44.08±9.18	-69.96 to -10.34	57	2.69	0.009	NS
TB(mg/dl)	2.22±0.83±0.14	1.00±1.54±0.32	-1.84 to -0.59	57	3.93	0.0002	HS



DISCUSSION:

Hepatitis C virus is a blood borne pathogen that is endemic in most parts of the world, with an estimated prevalence of nearly 3%[19]. Approximately 80% of patients with Hepatitis C virus develop chronic infection , and progression to cirrhosis occurs in nearly 20% of these subjects. Patients infected with Hepatitis C virus have different clinical outcomes, ranging from acute resolving Hepatitis to chronic liver disease including liver cirrhosis or hepatocellular carcinoma[20]. HCV infections have become the leading cause of chronic liver disease worldwide[21]. Those with cirrhosis will develop complications such as liver failure, liver cancer or oesophageal varices[22].

Our study showed that the levels of AST, ALT and Total Bilirubin were significantly higher in the cases than in the control group ($p<0.0001$) . There was no statistical difference in the levels of liver enzymes and Total Bilirubin in between the male and female cases, implying that HCV has no gender variation in its presentation ($p > 0.05$). The levels of AST, ALT and Total Biliruin were significantly higher in male cases as compared with male controls and in female cases as compare with female controls ($p<0.0001$). The levels of GGT showed slightly significant higher values in cases as compared to controls ($p< 0.05$) whereas ALP levels showed no significant difference in the study group ($p>0.05$). Bozdayia et al had got similar results in their study and they had suggested that Hepatitis C patients with higher ALT levels have more active response to chronic viral infections[23].

Ahmed et al observed elevate ALT and AST levels in PCR positive patients compared to normal range. He also showed that Serum ALP levels are not considered valuable markers during HCV diagnosis but he showed that change in ALP levels greater than 120 U/l can be indicative of advanced disease progression[24]. Wahib et al showed that 35% of HCV PCR positive patients had elevated results of Total bilirubin levels[25].

CONCLUSION:

Since treatment is expensive focus should be on development of a vaccine to prevent Chronic HCV infection is essential. Till then Maximum thrust is on prevention followed by early detection and treatment of the affected group. It is important to organize public awareness and health education campaigns targeting health care providers, private practitioners and public. Early diagnosis and treatment of HCV infection minimizes the risk of both long term complications and transmission of infection in the community.

REFERENCES:

1. Mohamed, A. A., Elbedewy, T. A., El-Serafy, M., et al. Hepatitis C virus: A global view. World Journal Journal of Hepatology, 2015; 7(26): 2676-80.
2. Stanaway, J. D., Flaxman, A. D., Naghavi, M., et al.. The global burden of viral hepatitis from 1990 to 2013: findings from the Global Burden of Disease S Study 2013. The The Lancet. doi.org/10.1016/S0140-6736(16)30579-7.
3. Graham. C. S., & Swan, T. A path to eradication of hepatitis C in low- and middle-income countries. Elsevier. 2015; 119: 89-96.
4. Lozano, R., Naghavi, M., Foreman, K., et al.Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012: 380(9859): 2095-128.
5. De Francesco R, Miliaccio G. Challenges and successes in developing new therapies for hepatitis C. Nature 2005;436:953-960.
6. Dufour DR, Lott JA, Nolte FS, Gretch DR, Koff RS, Seeff LB. Diagnosis and monitoring of hepatic injury. I. Performance characteristics of laboratorytests. Clin Chem 2000;46(12):2027-49.
7. Vanderlinde RE. Review of pyridoxal phosphate and the transaminases in liver disease. Ann Clin Lab Sci 1986;16(2):79-93.
8. Clemont RJ, Chalmers TC. The transminase tests in the liver disease. Medicine 1967;46(2): 197-207.
9. Williams AL, Hoofnagle JH. Ratio of serum aspartate to alanine aminotransferase in chronic hepatitis. Relationship to cirrhosis. Gastroenterology 1988;95 (3):734-9.
10. Giannini E, Botta F, Testa E, Romagnoli P, Polegato S, Malfatti, et. al. The 1-year and 3 month prognostic utility of the AST/ALT ratio and model for end-stage liver disease score in patients with viral liver cirrhosis. Am J Gastroenterol 2002; 97(11): 2855-60.
11. Moss DW. Physiochemical and pathophysiological factors in the release of membrane-bound alkaline phosphate from cells. Clin Chim Acta 1997;257(1): 133-40.
12. Rosalki SB, Tarlow D, Rau D. Plasma gamma-glutamy transpeptidase elevation in patients receving enzyme-inducing drugs. Lancet 1971;2:376-7.
13. Wedemyer H, Jackel E, Wiegand J, Corberg M, Manns MP. Whom? When? How? Another piece of evidence for early treatment of acute hepatitis C. Hepatology 2004;39(5):1201-3.
14. Tietz NW (cd). Fundamentals of Clinical Chemistry. ed. 3. Philadelphia: WB Saunders; 369-371; 1987.
15. Tietz NW (ed). Fundamentals of Clinical Chemistry. ed. 3. Philadelphia: WB Saunders; 369371; 1987.
16. Tietz NW (ed). Fundamentals of Clinical Chemistry. ed. 3. Philadelphia: WB Saunders; 385-386; 1987.
17. Tietz NW (ed). Fundamentals of Clinical Chemistry. ed. 3. Philadelphia: WB Saunders; 391-392; 1987
18. Tietz NW (ed). Fundamentals of Clinical Chemistry. ed. 3. Philadelphia: WB Saunders; 730-736; 1987.
19. Wasley A, Alter MJ. Epidemiology of hepatitis C: geographic differences and temporal trends. Semin Liver Dis.2000; 20: 1-16.
20. Lee YS, Yoon SK, Chung ES, Bae SH, Choi JY, et al. The relationship of histologic activity to serum ALT, HCV genotype and HCV RNA titers in chronic hepatitis C. J Korean Med Sci. 2001; 16: 585-591.
21. Kiyosawa K, Sodeyama T, Tanaka E, Gibo Y, Yoshizawa K, et al. Interrelationship of blood transfusion, non-A, non-B hepatitis and hepatocellular carcinoma: analysis by detection of antibody to hepatitis C virus. Hepatology. 1990; 12: 671-675.
22. Ryan KJ, Ray CG, eds. Sherris Medical Microbiology (4th ed.). McGraw Hill.2004 pp. 551-2. ISBN 0-8385-85299.
23. Zeuzem S, Franke A, Lee JH, Herrmann G, Ruster B, et. al. Phylogenetic analysis of hepatitis C virus isolates and their correlation to viremia, liver function tests, and histology. Hepatology. 1996; 24: 1003-1009.
24. Ahmad W, Ijaz B, Javed FT, Kausar H, Sausar H, Sarwar MT, et. al. HCV genotype-

specific correlation with serum markers: higher predictability for genotype 4a. Virol J. 2011; 2011;8: 293.
25. Wahib AA, Seif El Nasr MS, Mangound AM, El Shazly AM, Morsy AT. The liver function profile in PCR-RNA Egypatin HCV-patients and normal controls. J Egypt Soc Parasitol. 2005;35: 451-466.