



## A STUDY ON LIVER ENZYMES IN HEPATITIS PATIENTS AND ROLE OF INTERFERON THERAPY AS A PROGRESSIVE TREATMENT IN PAKISTAN

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### Abstract

**Introduction:** Hepatitis C is a worldwide health issue. Approximately, 71 million HCV cases are present in the world. Viral hepatitis claimed more annual deaths than HIV or malaria. It is the 7th leading cause of deaths worldwide. **Objective of the study:** The main objective of the study is to analyze the liver enzymes in hepatitis patients and role of interferon therapy as a progressive treatment in Pakistan. **Methodology of the study:** This cross sectional study was conducted at Shaikh Zayed Hospital, Lahore during September 2018 to January 2019. The data was collected from 200 patients of both genders who were suffering from hepatitis. The data was collected through a detailed questionnaire through which we collect the demographic data of patients. Blood investigation including Hemoglobin (Hb), total leucocytes count (TLC), differential leucocytes count (DLC), platelet count, X-ray chest, ultrasound abdomen and LFT were done in all patients. **Results:** The data were collected from 200 patients. The baseline characteristics of patients were recorded for the start of the treatment including age, blood complete picture, viral load and liver function test etc. The demographic values of patient group and control group shows a significant difference. The data suggest clearly that CD4 count decreases in abnormal liver function. There were non-significant relationship present in diseased group treated with different therapies like interferon and glutathione as  $p < 0.05$ . **Conclusion:** It is concluded that hepatitis directly increase the liver enzymes even after receiving medication and other therapies.

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**INTRODUCTION:**

Hepatitis C is a worldwide health issue. Approximately, 71 million HCV cases are present in the world. Viral hepatitis claimed more annual deaths than HIV or malaria. It is the 7th leading cause of deaths worldwide. Approximately 10 million hepatitis C cases are present in Pakistan. Hepatitis C prevalence is 4.9% in general population while in Injecting drug users and thalassemia patients the prevalence is 72% and 55% respectively [1]. The major routes of hepatitis transmission are contaminated blood and blood products used for transfusions, unsterilized dental and surgical instruments, reuse of needles and injections and shaving from barbers. Approximately, 1% of HCV positive patients are getting treatment each year in Pakistan and most of them are out-of-pocket payments. HCV has seven major genotypes and 86 confirmed subtypes [2]. The Pakistani dominant genotype is 3Chronic liver disease (CLD) is a major cause of morbidity and mortality and is quickly becoming an increasing burden on the health care system. Both CLD and cirrhosis are the fifth leading cause of death in the 45–61 age group and 12th leading cause overall. Liver is a pivotal organ of the body and play very important role in the metabolism [3]. Hepatitis is a major public health problem and is endemic throughout the world especially in tropical and developing countries. Hepatitis means inflammation of the liver. The liver is indispensable to our survival [4]. It has synthetic, storage and detoxification functions. An abnormal LFT may signify a serious disease that can be identified only through further testing. These conditions include liver diseases, such as primary biliary cirrhosis (PBC), diseases of other organs such as Paget's disease of bone, and multi-organ diseases such as haemochromatosis. However, the majority of people with an abnormal LFT in primary care settings will not have any such previously undetected disease [5].

**Table 01:** LFTs of hepatitis patients

S.O.V	Sum of Squares	df	Mean Squares	f	Sig.
ALP	15292.855	4	3823.214	18.288	.000
AST	4181.198	20	209.060	23.794	
ALT	19474.054	24		35.391	.000

**Objective of the study**

The main objective of the study is to analyze the liver enzymes in hepatitis patients and role of interferon therapy as a progressive treatment in Pakistan.

**METHODOLOGY OF THE STUDY:**

This cross sectional study was conducted at Shaikh Zayed Hospital, Lahore during September 2018 to January 2019. The data was collected from 200 patients of both genders who were suffering from hepatitis. The data was collected through a detailed questionnaire through which we collect the demographic data of patients. Blood investigation including Hemoglobin (Hb), total leucocytes count (TLC), differential leucocytes count (DLC), platelet count, X-ray chest, ultrasound abdomen and LFT were done in all patients.

**Statistical analysis**

The data were sampled and entered into the SPSS worksheet for analysis. A two-tailed P-value was calculated for all tests and  $P \leq 0.05$  was considered as statistically significant.

**RESULTS:**

The data were collected from 200 patients. The baseline characteristics of patients were recorded for the start of the treatment including age, blood complete picture, viral load and liver function test etc. The demographic values of patient group and control group shows a significant difference. The data suggest clearly that CD4 count decreases in abnormal liver function. There were non-significant relationship present in diseased group treated with different therapies like interferon and glutathione as  $p < 0.05$ .

**Table 02:** Association between serum markers and fibrosis stage

Marker	Adjusted model <sup>a</sup>			Marker	Adjusted model <sup>a</sup>		
	Mean	SD	P-value		Mean	SD	P-value
AST (normal: 17–59)				Total bilirubin (normal: 0.2–1.2)			
Stage 0	58.14	4.29	Ref.	Stage 0	1.71	1.03	Ref.
Stage 1	50.49	4.74	0.2283	Stage 1	0.81	0.90	0.0872
Stage 2	63.39	4.27	0.2222	Stage 2	1.33	0.89	0.4867
Stage 3	70.02	4.27	0.0423	Stage 3	1.98	0.95	0.5085
Stage 4	73.25	4.28	0.0014	Stage 4	2.73	0.92	0.0188
ALT (normal: 0–55)				Albumin (normal: 3.5–5.0)			
Stage 0	55.65	10.46	Ref.	Stage 0	3.48	0.21	Ref.
Stage 1	63.98	8.82	0.2226	Stage 1	3.91	0.21	0.0007
Stage 2	70.58	8.26	0.0092	Stage 2	3.81	0.18	0.0117
Stage 3	75.63	9.37	0.0025	Stage 3	3.88	0.17	0.0031
Stage 4	58.24	9.43	0.7787	Stage 4	3.34	0.18	0.0607

**DISCUSSION:**

INR and albumin are often regarded as important markers of synthetic function, and our study revealed a normal range of INR for all pre-cirrhotic stages. The INR was statistically significant for stage 4 fibrosis when compared to those individuals with no discernible liver disease; however, it remained near the upper limit of normal [6]. Albumin is a major protein synthesized by the liver. We did not see a progressive decrease with advanced disease, and the albumin level in stage 4 remained close to the normal value and was not statistically significant. As such, makers for hepatic synthetic function (albumin and INR) do not appear to be a good measure of hepatic fibrosis [7].

CLD and cirrhosis have gained increasing attention due to the newly approved therapies for hepatitis C and the increasing obesity epidemic. Because of the morbidity and mortality associated with CLD and cirrhosis, there is significant utilization of health care resources. Identification of affected individuals is critical to help mitigate the progression of disease and, more importantly, identify those with end stage liver disease who are at significant risk for decompensating [8]. Identification of those individuals with treatable disease, such as HCV, HBV, and autoimmune hepatitis, may slow or even reverse fibrosis and early cirrhosis. Damage to the structural integrity of liver is reflected by an increase in the level of serum transaminase because these are cytoplasmic in location and are released into circulation after cellular damage [9]. Over 4 million acute hepatitis B cases are diagnosed every year which leads to one fourth of cases becoming chronic carriers. The chronic stage accounts for 1 million deaths per year due to chronic active hepatitis, cirrhosis and hepatocellular

carcinoma [10]. This study shows distribution of acute viral hepatitis cases with age group and gender in children. It also shows that HBV is responsible for 18.75% of acute hepatitis, so an important cause of morbidity in this part of the country.

It is generally accepted that the toxicity of carbon tetrachloride depends on the cleavage of the carbon-chlorine bond to generate a trichloromethyl free radical, and this free radical reacts rapidly with oxygen to form a trichloro methyl peroxy radical, which may contribute to the hepatotoxicity and subsequent increase in hepatic enzymes [11].

**CONCLUSION:**

It is concluded that hepatitis directly increase the liver enzymes even after receiving medication and other therapies. With new and effective treatments becoming available for the treatment of liver disease, identification of such individuals with proper risk factor determination by history and physical examination.

**REFERENCES:**

1. Pradhan SC and C Girish (2006). Hepato protective herbal drug, silymarin from experimental pharmacology to clinical medicine Indian J Med Res 124, pp 491-504.
2. Patel, V.K. and Bhatt H.V., 1985.Toxicity antiseptic effect of chicory root extract in Pyorrhea. The antiseptic 904-906.
3. Crapnell K., Zanjani E. D., Chaudhuri A., Ascensao J. L., Jeor S. S., Maciejewski J. P. In vitro infection of megakaryocytes and their precursors by human cytomegalovirus. *Blood*. 2000;95(2):487–493.

4. Gavrilovskaya I. N., Shepley M., Shaw R., Ginsberg M. H., Mackow E. R.  $\beta_3$  integrins mediate the cellular entry of hantaviruses that cause respiratory failure. *Proceedings of the National Academy of Sciences of the United States of America*. 1998;95(12):7074–7079. doi: 10.1073/pnas.95.12.7074.
5. Martell M., Gomez J., Esteban J. I., et al. High-throughput real-time reverse transcription-PCR quantitation of hepatitis C virus RNA. *Journal of Clinical Microbiology*. 1999;37(2):327–332.
6. Chen C.-H., Yang P.-M., Huang G.-T., Lee H.-S., Sung J.-L., Sheu J.-C. Estimation of seroprevalence of hepatitis B virus and hepatitis C virus in Taiwan from a large-scale survey of free hepatitis screening participants. *Journal of the Formosan Medical Association*. 2007;106(2):148–155.
7. Seeff L. B. Natural history of chronic hepatitis C. *Hepatology*. 2002;36(5, supplement 1):S35–S46.
8. Li X., Jeffers L. J., Garon C., et al. Persistence of hepatitis C virus in a human megakaryoblastic leukaemia cell line. *Journal of Viral Hepatitis*. 1999;6(2):107–114.
9. Ward J. W. The hidden epidemic of hepatitis C virus infection in the United States: occult transmission and burden of disease. *Topics in Antiviral Medicine*. 2013;21(1):15–19.
10. Bataller R, North KE, Brenner DA. Genetic polymorphisms and the progression of liver fibrosis: a critical appraisal. *Hepatology*. 2003;37(3):493–503.
11. Iredale JP. Models of liver fibrosis: exploring the dynamic nature of inflammation and repair in a solid organ. *J Clin Invest*. 2007;117(3):539–548.