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Research Article

A STUDY ON LIVER FUNCTION TEST (LFT'S) IN HEPATITIS PATIENTS AND ROLE OF INTERFERON THERAPY AS A PROGRESSIVE TREATMENT IN PAKISTANDr Muhammad Waqar Rehan¹, Dr Ayesha Shaukat², Dr Muhammad Yasir³¹House Officer at Shalamar Hospital, Lahore, ²Nishtar Institute of Dentistry, Multan,³Lahore Medical and Dental college, Lahore.

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Abstract:

Introduction: Chronic 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.

Objective of the study: The main objective of the study is to analyze the liver function test (LFT's) 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 Shalamar Hospital, Lahore during January 2018 to November 2018. 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 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 $p < 0.05$. The normal range of AST is 5–34 U/L. The AST values in the fibrosis stages 3 and 4 were significantly elevated when compared to stage 0.

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.

Corresponding author:**Dr. Muhammad Waqar Rehan,**

House Officer at Shalamar Hospital, Lahore.

QR code



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INTRODUCTION:

Chronic 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 [1]. If there is any problem in the liver then the herbs or different plants play an important role for the treatment of liver disorders¹. There are a number of plants which shows hepatoprotective property [2]. Hepatitis B and C viruses can lead to hepatocellular carcinoma and cirrhosis-related end-stage liver disease, which are potentially life-threatening liver diseases [3]. Every year, about 600,000 people die because of the acute or chronic consequences of hepatitis B, and more than 350,000 people die from hepatitis C-related liver diseases worldwide [4].

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 [5]. 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 [6].

Objective of the study

The main objective of the study is to analyze the liver function test (LFT's) 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 Shalamar Hospital, Lahore during January 2018 to November 2018. 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. A thorough clinical examination was carried out and stigmata of chronic liver disease, hepatosplenomegaly, ascites, etc. if present were noted.

Biochemical analysis

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. The LFT included serum bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), serum alkaline phosphatase (SAP) and serum albumin. Abnormal values were defined as serum Bilirubin ≥ 1.5 mg/dl, ALT/AST ≥ 50 IU/ml.

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 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 as $p < 0.05$. The normal range of AST is 5–34 U/L. The AST values in the fibrosis stages 3 and 4 were significantly elevated when compared to stage 0 (70 vs 58, $P < 0.05$; 73 vs 58, $P < 0.01$, respectively). When stages 1 and 2 were compared to stage 0, the difference was not significant.

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

Table 02: Association between serum markers and fibrosis stage

Marker	Adjusted model ^a			Marker	Adjusted model ^a		
	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:

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 decompensation [7]. Identification of those individuals with treatable disease, such as HCV, HBV, and autoimmune hepatitis, may slow or even reverse fibrosis and early cirrhosis [8]. 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]. 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 [5].

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.

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, physical examination, and targeted laboratory testing will better insure earlier treatment and potentially decrease the number of patients with both known and unknown advanced and decompensated liver disease.

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 Pyorrhoea. The antiseptic 904-906.
3. 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. doi: 10.1016/S0929-6646(09)60231-X.
4. 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.
5. Seeff L. B. Natural history of chronic hepatitis C. *Hepatology*. 2002;36(5, supplement 1):S35–S46.

6. 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. doi: 10.1046/j.1365-2893.1999.00140.x
7. *Blood Screening by Blood Center*. Taiwan Blood Services Foundation; 2009. <http://www.sc.blood.org.tw/Internet/main/docDetail.aspx?uid=6677&pid=6389&docid=24905>.
8. 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.
9. Gavrilovskaya I. N., Shepley M., Shaw R., Ginsberg M. H., Mackow E. R. β_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.
10. 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.