ORIGINAL RESEARCH PAPER

Volume-7 | Issue-11 | November-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

STUDY OF GAMMA GLUTAMY & TRANSFERASE: A SENSITIVE INDICATOR OF HEPATOBILIARY DISEASE

Biochemistry

Manpreet Saini Tutor, Dept. of Biochemistry, Govt. Doon Medical College, Patel Nagar, Dehradun

Gurpreet Kaur Associate Prof, Psychiatry Dept. SGRR College of Nursing, Patel Nagar Dehradun

Nita Garg* Professor and Head, Deptt of Biochemistry, SGRRIM&HS, Patel Nagar, Dehradun
*Corresponding Author

ABSTRACT

Aim: The aim of our study was to estimate Liver enzymes and to establish GGT as a sensitive indicator of hepatobiliary diseases.
Study design: A hospital based cross sectional study was conducted on patients attending the Outpatient Department of Medicine of SMIH for a period of 06 months from June 2017 to December 2017. 112 clinically confirmed liver disease cases and 80 age and sex matched controls in the group 20-80 years were included in the study. Exclusion criteria was age less than 20 years & more than 80 years.
Methodology: 5ml of venous blood was collected from both cases & controls and analyzed for Liver Function-Total Serum Bilirubin, SGOT, SGPT, ALP, GGT and Total Protein on a fully automated analyzer 5600 of OCD.
Results: We found a significant increase in the values of Total Serum Bilirubin, Serum Transaminases and Serum ALP, Serum GGT when compared with normal controls in both males & females.

KEYWORDS

GGT, Hepatobiliary diseases.

INTRODUCTION:

Chronic liver disease is defined as series of liver disorder with varying aetiologies and severities, with which hepatic inflammation and necrosis continue for atleast 06 months0[1]. A variety of biochemical parameters like serum bilirubin, transaminases, ALP and GGT etc are elevated to assess the liver cell damage[2].

GGT is a microsomal enzyme present in hepatocytes and biliary epithelial cells, renal tubules, pancreas and intestines. It is also present in cell membrane performing transport of peptides into the cell and across the cell membrane and involved in glutathione metabolism serum GGT activity is mainly attributed to hepatobiliary system even though it is found in more concentration in renal tissue[3]. The normal level of GGT is 9-85µ/l[4].

A comprehensive review by whitfield in 2001 described GGT as a marker of liver dysfunction, bile duct conditions and alcohol consumption[5]. The GGT activity is considered as a sensitive index of the hepatobiliary dysfunction than alkaline phosphatase, due to its presence in the microsomes & the plasma membranes of hepatocytes[6,7].

METHODOLOGY:

A hospital based cross sectional study was conducted on patients attending the Outpatient Department of Medicine of SMIH for a period of 06 months from June 2017 to December 2017. 112 clinically confirmed liver disease cases and 80 age and sex matched controls in the group 20-80 years were included in the study. Exclusion criteria was age less than 20 years & more than 80 years. 5ml of venous blood was collected from both cases & controls and analyzed for Liver Function-Total Serum Bilirubin[8], SGOT[9], SGPT[9], ALP[10], GGT[11] on a fully automated analyzer 5600 of Ortho Clinical Diagnostics.

RESULTS:

We found a significant increase in the values of Total Serum Bilirubin, Serum Transaminases, Serum ALP and Serum GGT when compared with normal controls in both males & females. The results are tabulated in Table (i) and (ii) and shown graphically in Fig (i) and (ii).

Table(I) showing comparison between Male cases and controls

Parameter	Male Cases (60)	Male Control (40)	t-value	p-value	Significance
Total serum bilirubin	4.62±2.39±0.29	0.67±0.18±0.03	-10.4	<0.0001	HS
SGPT	89.89±35.41±4.32	28.41±4.31±0.23	-10.7	<0.0001	HS
SGOT	133.76±42.23±5.15	28.79±6.35±1.22	-15.5	<0.0001	HS
ALP	166.33±126.78±15.46	83.66±16.50±3.17	-4.0	<0.0001	HS
GGT	226.29±184.76±22.53	22.69±2.14±0.41	-6.9	=0.0001	HS

Table(ii) showing comparison between female cases and controls

Parameter	Female Cases (52)	Female Control (40)	t-value	p-value	Significance
Total serum bilirubin	4.09±2.10±0.30	0.79±0.20±0.04	-9.89	<0.0001	HS
SGPT	107.20±48.17±6.99	29.16±5.00±0.96	-10.19	<0.0001	HS
SGOT	119.96±47.22±6.85	29.04±4.46±0.86	-12.12	<0.0001	HS
ALP	211.91±120.32±17.46	83.60±18.40±3.54	-6.6	<0.0001	HS
GGT	185.47±140.70±20.42	22.76±2.05±0.39	-7.3	<0.0001	HS

Fig(I) showing comparison between male cases and controls

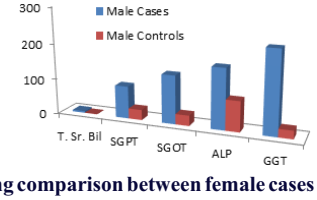
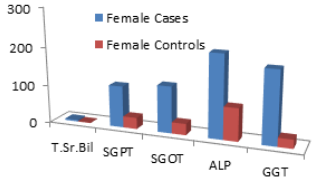


Fig (ii) showing comparison between female cases and controls



DISCUSSION:

In patients with hepatobiliary diseases, GGT serum levels can be markedly altered (>10 times the upper reference value) where as ALP levels may be normal or only slightly altered (GGT/ALP Ratio >2.5).

The strong correlation between Serum GGT & ALP in liver diseases confirms that changes in the activity of GGT reflects principally alterations in biliary function rather than damage to the parenchymal cells(12). The GGT levels rise & return to normal levels later in liver diseases than the transaminases[13]. So the estimation of GGT is of some

value in monitoring the progress of acute to chronic hepatitis, when the values persist in high levels. Serum GGT has been used in the detection of hepatobiliary diseases due to its sensitive index of chronic liver damage[1].

The reasons for elevated GGT values in those with hepatobiliary disease include increased denovo synthesis, increased release from cell membrane owing to the detergent effect of bile salts backflow into the blood stream, increased permeability and destruction of biliary epithelia[14,15].

CONCLUSION:

We conclude that serum GGT is a valuable diagnostic aid when used in conjunction with ALP assays for the investigation of latent chronic liver diseases. We also came to the conclusion that GGT is a more sensitive diagnostic marker, but a single laboratory liver test is of little value in screening for liver disease. The pattern of enzyme abnormality interpreted in the context of patients symptoms can aid in directing the subsequent diagnosis.

REFERENCES:

1. Jules L Dienstag. Harrison's Principles of Internal Medicine. 18th Vol. 2567. United States of America: McGraw Hill companies; 2012. Chronic Hepatitis. In Longo, Fauci, Kasper, Hauser, et al., editors;pp. 3588-90.
2. Laker MF. Liver Function Tests. Br Med J. 1990;301:250-51. [PMC free article] [PubMed]
3. Mauro P, Renze B, Wouter W. In: Tietz test book of clinical chemistry and molecular diagnostics. 4th edition. Carl AB, Edward R, David EB, editors. Elsevier; 2006. Enzymes; pp. 604-616.
4. Diana Nicoll C. In: Current medical diagnosis and treatment. 46th edition. Stephen JM, Maxine AP, editors. Mc Graw hill; 2007. Appendix: Therapeutic drug monitoring and laboratory reference ranges; pp. 1767-1775.
5. Whitfield J. B. Gamma glutamyl transferase. Critical Reviews in Clinical Laboratory Sciences. 2001;38(4):263-355. Doi: 10.1080/20014091084227.[PubMed] [Cross Ref].
6. Penn R, Worthington DJ. Is serum y-glutamyl transferase a misleading test. Br Med J. 1983;286:531-35. [PMC free article] [Pubmed].
7. Rosalki SB. Gamma-glutamyltranspeptidase. Advclin Chem. 1975;17:53-107. [Pubmed].
8. Tietz NW(ed). Fundamentals of Clinical Chemistry. Ed. 3. Philadelphia:WB-Saunder; 1987:730-736.
9. Tietz NW(ed). Fundamentals of Clinical Chemistry. Ed. 3. Philadelphia:WB-Saunder; 1987:361-371.
10. Tietz NW(ed). Fundamentals of Clinical Chemistry. Ed. 3. Philadelphia:WB-Saunder; 1987: 385-386.
11. Tietz NW(ed). Fundamentals of Clinical Chemistry. Ed. 3. Philadelphia:WB-Saunder;1987: 391-392.
12. J.B whitfield, RE Pounder, G Neale, DW Moss. Serum Gamma-Glutamyl transpeptidase activity in liver disease. Gut.1972 Sep;13(9):702-708.PMID:440-4786.
13. Percy-Robb IW, Finlayson NDC. Disease of the gastrointestinal tract and liver. 3rd, London: Churchill Livingstone; 1977. Clinical Chemistry of liver disease. In: Shearman JC. Finlayson NDC Camilleri M, eds; pp. 735-61.
14. Sotil EU, Jensen DM. Serum enzymes associated with Cholestasis. Clin liver Dis. 2004;8:41-54. [PubMed].
15. Loria P, Ballestri S, Lonardo A, Gli Enzimi Epatobiliari nella diagnostica clinica delle epatopatie. Intern Emerg Med. 2008;3:1-6.