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Comparative Study of ALT, AST, GGT & Uric Acid Levels in Liver Diseases

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Abstract:*Hepatic injury is associated with distortion of the metabolic function. Hepatic disease/Cirrhosis of liver can be evaluated by biochemical analysis of serum tests, includes levels of serum Alanine and Aspartate amino transferases, Alkaline Phosphatase, and also by Uric Acid estimation. The present study was continued to assay liver associated enzymes on patients with cirrhosis of liver, Amoebic liver abscess and hepatitis and to find out the comparative levels of enzymes and uric acid among the groups. In this study total 80 male subjects (25 healthy controls and 55 patients as case groups) aged between 20 to 60. yrs. Was enrolled. One case group consists 25 male patients(cirrhosis of liver) and second case group consists of 15 male patients (Amoebic liver abscess disease) and third case group consists of 15 male patients (Hepatitis) suffering from corresponding diseases ad controls group subjects are 25 in number. ALT, AST, ALP,GGT and uric acid levels are estimated in the above groups by standard methods. Several folds of variations in the analysed enzymes were found between healthy controls and case groups. There is a significant elevation in the Uric acid levels comparatively with controls group. Comparitive elevation of serum associated enzymes were observed to indicate the degree of hepatic damage in cirrhosis, amoebic abscess of liver diseases and hepatitis. Data analysed using SPSS software.*

Key Words: *Cirrhosis of liver, Amoebic abscess of liver disease, hepatitis, Uric acid , and hepatic damage.*

I. Introduction:

The liver is the largest organ of the body, weighing 1 to 1.5 kg and representing 1.5 to 2.5% of the lean body mass.Liver is a complex organ with interdependent metabolic, excretory and defense functions .The use of several screening tests improves the detection of hepato-biliary abnormalities, helps differentiate the basis for clinically suspected disease and determine the severity of liver disease [1]. Blood tests used for initial assessment of liver disease include measuring levels of serum Alanine and Aspartate aminotransferases (ALT and AST), alkaline phosphatase, and others. The pattern of abnormalities generally points to hepatocellular versus cholestatic liver disease and helps to decide whether the disease is acute or chronic and whether cirrhosis and hepatic failure are present [2]. Serum enzyme levels fluctuate widely from normal to moderately abnormal, with values rarely into the high hundreds [3, 4, 5]. Marked elevation of aminotransferases in the appropriate clinical context indicates acute cell necrosis caused by viral infection, drugs, toxins, alcohol, or Ischemia [6]. Viral hepatitis is a global health problem that affects hundreds of millions of children and adults; viral hepatitis remains a leading cause of virus-associated morbidity and mortality, affecting millions of individuals worldwide [7]. The number of enzymes present, or pathologically increased in the plasma during viral hepatitis, and their comparative behavior in other type of liver diseases like Alcoholic liver diseases, cirrhosis and others [8].

In humans and higher primates, uric acid (UA) is the final oxidation product of purine metabolism and is excreted in urine. Hyperuricemia has long been recognized as a cause of gouty arthritis and kidney stones. More recently, hyperuricemia has also been implicated in the development of hypertension, kidney disease, metabolic syndrome, and cardiovascular disease(reviewed by Feig et al. [9] and Edwards[10]) . Although hyperuricemia has traditionally been considered a result of these conditions or an epiphenomenon, mechanisms have been proposed by which hyperuricemia could actually cause them. Such mechanisms include the induction by hyperuricemia of endothelial dysfunction, insulin resistance, oxidative stress, and systemic inflammation [9,10]. Oxidative stress, insulin resistance, and systemic inflammation are now known to be important risk factors for the development or progression of the most important liver diseases. For example, these conditions are considered central in the pathogenesis of nonalcoholic fatty liver disease (NAFLD) and non-alcoholic steatohepatitis(NASH) [11]. In addition, they contribute to the progression of hepatitis C virus (HCV)–related and alcoholic liver diseases[12]. Therefore, we hypothesized that hyperuricemia, which strongly reflects and

may even cause oxidative stress, insulin resistance, and systemic inflammation, is a risk factor for the development of cirrhosis or the presence of hepatic necroinflammation.

Alcoholic liver disease (ALD) represents a spectrum of clinical illness and morphological changes that range from fatty liver to hepatic inflammation and necrosis (alcoholic hepatitis) to progressive fibrosis (alcoholic cirrhosis)[13]. A reliable history is helpful; in reality this can be difficult. A biochemical clue is the ratio of AST to ALT (2:1 atleast), reflecting the low level of activity of ALT in people with alcoholic liver disease. [14].Present study was conducted to find the comparative levels of liver associated enzymes and uric acid levels among Cirrhosis of liver, Amoebic liver abscess and Viral Hepatitis.

II. Materials And Methods

Present study was comprised of total 80 male subjects (25 Healthy controls and 55 case group) aged between 30 - 60 years, each case group consisted of 25 male patients of similar age suffering with Viral Hepatitis, Alcoholic Liver disease (more than 10 yrs) and liver cirrhosis. Histopathology Liver Biopsy report confirmed the Liver cirrhosis case group. All case groups were the patients admitted to KONASEEMA INSTITUTE OF MEDICAL SCIENCES & RESEARCH FOUNDATION,AMALAPURAM,PIN-533201,EG DISTRICT, AP, INDIA. Ethical approval was obtained from concerned authority and informed consent was taken from each participant. Alanine aminotransferase (ALT), Aspartate aminotransferase (AST) , Alkaline phosphatase(ALP) and Gamma Glutamyl Transferase (GGT) were assayed by kinetic methods,SPAN kits[15] .Uric Acid was determined by Uricase end point method,MERCK kit[16]

III. Statistical Analysis

Statistical analysis was done using SPSS software and results expressed as mean ±SD. Comparison of variables between two groups performed with student t-test for continuous variables. The p values < 0.05 were considered statically significant.

IV. Inclusion Criteria

Inclusion criteria for Cirrhosis of liver is jaundice, ascites, pedal edema, spider naevi, enlarged veins over the abdomen & palpable spleen. Inclusion criterion for Hepatitis was acute tenderness in the right hypochondrium, pyrexia, jaundice and repeated vomiting. For amoebic liver abscess the inclusion criterion were intermittent fever with rigors, pain in liver area, commences as dull ache later becoming sharpened and stabbing, hepatic tenderness, cysts and vegetative forms seen in stool examination, jaundice, mild response to metronidazole & ultrasound report.

V. Exclusion Criteria

Carcinoma of liver , cardio vascular associated liver diseases and other infectious diseases are excluded.

VI. Results :

Statistical Analysis: Statistical analysis was done using by SPSS software.Results expressed as mean ± SD

TABLE I: SERUM PARAMETERS IN CIRRHOSIS OF LIVER AND THE MATCHED CONTROLS

SERUM PARAMETERS	MEAN±SD (CIRRHOSIS OF LIVER, n=25)	MEAN±SD (CONTROL, n=25)	't'	'p'
SGOT (K units)	67±31.4	35.8±5.9	4.78	<0.001
SGPT (K units)	65±27.5	35.8±5.9	5.16	<0.001
ALP (KA units)	7.1±2.4	6.2±1.4	1.62	<0.10 (non significant)
GGT	116.33±29.3	26.7±4.026	4.5	<0.01
UA	10.6±2.0	5.0±2.0	1.6	<0.01

TABLE II: SERUM PARAMETERS IN AMOEBIC LIVER ABSCESS AND THE MATCHED CONTROLS

SERUM PARAMETERS	MEAN±SD (AMOEBIC LIVER ABSCESS, n=15)	MEAN±SD (CONTROL, n=25)	't'	'p'
SGOT (K units)	60±24	35.8±5.9	3.995	<0.001
SGPT (K units)	61±24	35.8±5.9	3.995	<0.001
ALP (KA units)	7.2±3.1	6.2±1.4	1.179	<0.20 (non significant)
GGT	114±22.0	26.7±4.026	4.5	<0.01
UA	9.5±2.1	5.0±2.0	1.8	<0.01

TABLE III: SERUM PARAMETERS IN HEPATITIS AND THE MATCHED CONTROLS

SERUM PARAMETERS	MEAN±SD (HEPATITIS, n=15)	MEAN±SD (CONTROL, n=25)	't'	'p'
SGOT (K units)	62±22.4	35.8±5.9	4.426	<0.001
SGPT (K units)	78±28.7	35.8±5.9	5.624	<0.001
ALP (KA units)	7.2±2.5	6.2±1.4	1.421	<0.01
GGT	120±20	26.7±4.026	4.8	<0.01
UA	9.9±2.2	5.0±2.0	1.6	<0.01

TABLE IV: TABLE SHOWING THE MEAN ± SD OF AST, ALT, ALP,GGT & URIC ACID IN CONTROLS & LIVER DISEASES

S.No	PARAMETERS	CONTROLS	CIRRHOSIS OF LIVER	AMOEBIC LIVER ABSCESS	HEPATITIS
1	AST (K UNITS)	36.4±8.1	67.4±31.4	60±31	62±22
2	ALT (K UNITS)	35.8±5.9	65±27.5	61±24	78±28.7
3	ALP (K UNITS)	6.2±1.4	7.1±2.4	7.2±3.1	7.2±2.5
4	GGT	26.7±4.026	116±30.0	117±21.0	120±20.0
5	UA	5.0±2.0	10±2.1	9.5±2.1	9.9±2.2

VII. Discussion

The liver associated enzymes, Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), and gamma glutamyl transferase (GGT) are measures of liver homeostasis [17]. Serum amino transferases such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) indicate the concentration of hepatic intracellular enzymes that have leaked into the circulation. These are the markers for hepatocellular injury [18].The aminotransferases (transaminases) are sensitive indicators of liver cell injury and are most helpful in recognizing acute hepatocellular diseases such as hepatitis. The pattern of the aminotransferase elevation can be helpful diagnostically. In most acute hepatocellular disorders, the ALT is higher than or equal to the AST. An AST: ALT ratio>2:1 is suggestive while a ratio >3:1 is highly suggestive of alcoholic liver disease. The AST in alcoholic liver disease is rarely >300 U/L and the ALT is often normal. A low level of ALT in the serum is due to an alcohol linduced deficiency of pyridoxal phosphate. [19]. In this study, the AST: ALT ratio is 1 for normal, 0.65(<1) for viral hepatitis, consistent with F .DE RITIS et al[20], >2 for ALD group, which similar to reported by several other studies conducted earlier [21], and 1.24 in cirrhosis , > 1 but < 2 also documented by Nyblom et al [22] and others . This helps to differentiate ALD from other liver diseases.

In this study AST, ALT ALP, GGT levels were significantly raised in viral hepatitis, and cirrhosis patients as compared to control. In viral hepatitis AST, ALT and ALP Levels were significantly high as compared to alcoholic liver disease and cirrhosis. In viral hepatitis ALT is greater than AST. The peak levels of Transminases have been reported to vary from 400- 4000 IU/L or more [23]. In alcoholic liver disease AST activity has been reported to be greater than ALT and usually does not exceed 300 IU/L. AST/ALT ratio is greater than 2 because of existing mitochondrial damage [23, 24].

This study also confirms that in cirrhosis AST and ALT levels are normal or slightly elevated. If the etiological factors are present or with active alcohol abuse increases AST and ALT levels [23]. In viral hepatitis and in alcoholic liver disease ALP usually increases.In cirrhosis ALP is either normal or slightly elevated [20], increased in serum ALP is associated with liver disease is caused by intra or extra hepatic cholestatis and some destruction of hepatic cell membrane .Elevation of ALP is observed in patients who have some form of extra hepatic and intra hepatic bile duct obstruction. Any mechanism that impaired excretion of ALP in bile will result in regurgitation of enzyme into circulation via the hepatic sinusoid. The increased ALP present in the patients with disease closely resembles the ALP that can be extracted from liver. The increased cholestatis stimulates the synthesis of ALP by the bile ductules cell providing more ALP which ultimately enters the blood, the amphillic nature of bile salts facilitates the release of ALP from its membranes bound site and entry into blood [25] .

In Viral hepatitis GGT levels were significantly increased as compared to cirrhosis and high as compared to Amoebic Abscess of liver disease and cirrhosis , moreover GGT levels are also high in case cirrhosis than Amoebic liver disease. GGT present in the cell membranes of hepatobiliary system, it is an extremely sensitive enzyme to identify cholestatis disease both intra and extra hepatic. In viral hepatitis in absence of cholestasis, it increases upto 5 times and in the presence of cholestatsis it increases upto 10 times of upper limits [23]. In the alcoholic liver disease it is 8-20 times the upper limits and persistence elevation of GGT may be an indicator if Cirrhosis [24] .In our study we observed the increasing pattern of GGT value in different folds among patients of Viral Hepatitis, Amoebic abscess of liver diseases and Liver Cirrhosis respectively.

We have reported novel associations between serum UA levels and the incidence of cirrhosis-related hospitalization or the presence of elevated serum ALT or GGT. These associations are largely independent of other known liver disease risk factors. The elevated serum UA level might be a risk factor for the incidence of chronic liver disease. Future studies should investigate whether this association is causal or has clinical utility in the prediction of the presence or incidence of liver disease. If this is confirmed, further consideration should be given to measures that reduce the serum UA levels as a means of preventing cirrhosis in persons with elevated levels.

VIII. Conclusion

There are significant elevations of values of of ALT,AST,ALP,GGT and Uric acid level in the cirrhosis of liver, amoebic liver abscess and viral hepatitis.Hence the elevation of serum uric acid levels along with the classical liver enzymes might be a risk factor for incidence of chronic liver diseases

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