ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

A COMPARATIVE STUDY IN ASSESSING THE USEFULNESS OF SERUM CHOLINESTERASE, HIGH SENSITIVITY C-REACTIVE PROTEIN WITH LIVER FUNCTION TESTS IN ALCOHOLIC LIVER DISEASE"

Dr.A. Jyotsna, Dr.T.Aruna Kumari, Dr.G.Anil Kumar

1,&3. Assistant Professor, Department of Biochemistry, Kakatiya Medical College/MGM Hospital, Warangal, Telangana, India.
2. Associate Professor, Department of Biochemistry, Kakatiya Medical College/MGM Hospital, Warangal, Telangana, India.
Corresponding author: Dr.A. Jyotsna

ABSTRACT

Background and Objectives: Chronic alcoholism causes liver damage. Alcoholic liver disease includes fatty liver, alcoholic hepatitis, and cirrhosis. >90% of binge and chronic drinkers have fatty liver, with a lower number developing alcoholic hepatitis, a precursor to cirrhosis. Conventional liver function tests have been used for years to diagnose liver disorders,however they lack 100% sensitivity and specificity. Often, non-liver disorders elevate liver function test parameters. Many research have been done, but more are needed to confirm its utility in liver disease diagnosis. Assays of serum cholinesterase and hs-CRP will be used to diagnose alcohol liver damage**. Materials and methods:** 50 men with alcoholic liver disease were compared to 50 normal men and 50 men with non-alcoholic liver disease. Interview, questionnaire, clinical indicators of liver disease, lab tests (bilirubin, total protein, serum albumin, A:G, AST,ALT,ALP and GGT), and ultrasonography led to the diagnosis.**Results:**Both alcoholic and non-alcoholic cirrhosis patients had inadequate liver function tests compared to controls. Serum cholinesterase levels were significantly lower in alcoholic cirrhosis patients (p 0.001), while hs-CRP levels were significantly higher in non- alcoholic cirrhosis patients (1.590.28) compared to alcoholic cirrhosis patients (1.34 0.42) (p 0.001). 38 patients had 0-48 U/L (76%) and 12 had >48 (24%). 100 percent of 50 controls were
0-48 U/L. 46 Non-ALCs had >48 U/L (92%) and 4 had 0-48 U/L (8%).**Conclusion:** In conclusion, a significant decrease in serum cholinesterase levels in patients with alcoholic cirrhosis suggests that the activity of this enzyme may be a specific indicator of liver dysfunction and may be used for the diagnosis of alcoholic cirrhosis patients. On the other hand, high-sensitivity C-reactive protein levels can be used as a strong predictor of non- alcoholic cirrhosis.

Key words: *alcoholic cirrhosis, non-alcoholic cirrhosis, serum cholinesterase, hs-crp*

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

INTRODUCTION

Alcoholic liver disease is a term that encompasses the hepatic manifestations of alcohol over consumption which includes fatty liver, alcoholic hepatitis and chronichepatitis with hepatic fibrosis.[1]

Alcoholism causes over 200 diseases and injuries.Alcohol causes 3 million fatalities annually worldwide. 5.3% of deaths.Overall, 5.1% of the worldwide disease and injury burden is related to alcohol (DALYs).Alcohol abuse causes social and economic damages for people and society.Alcoholism causes early death and disability. Alcohol causes 13.5% of deaths in 20–39-year-olds.Alcohol abuse causes mental and behavioural issues, noncommunicable diseases, and injuries. India diagnoses 10 lakh new liver cirrhosis patients annually. - WHO classifies liver disease as India's 10th leading cause of mortality. - Every fifth Indian has liver disease.[2] Alcohol causes cirrhosis globally, including India, and is one of the ten leading causes of death. Due to rising socioeconomic class, India's alcohol consumption has risen in the last decade. Chronic and heavy drinking promotes liver damage. Alcoholism and hepatitis B or C are frequent causes of liver cirrhosis. Gender, ethnicity, and geography affect liver cirrhosis prevalence. [2-5].

The amount of alcohol consumed over a prolonged period of time is the single most important risk factor that contributes to the development of alcoholic liverdisease. If a man consumes more than 60 to 80 grammes of alcohol per day for ten years, he has an increased risk of developing alcoholic liver disease. On the other hand, a woman has an increased risk of acquiring similar degrees of liver injury if she consumes 20 to 40 grammes of alcohol per day. Consuming 160 grammes of alcohol on a daily basis is related with a 25-fold greater chance of developing alcoholic cirrhosis. There is a gender gap in understanding of the effects of oestrogen and how alcohol is metabolised, which leads in gender differences. It has been hypothesised that social factors, immunologic factors,

and hereditary factors are all involved in the development of the pathogenic process. Prolonged infection with hepatitis C is an essential co-morbidity that has a role in the progression of alcoholic liver disease to cirrhosis in individuals who have a history of excessive and chronic drinking. [3]

The severity of alcoholic liver disease can range from asymptomatic liver enlargement to severe liver failure and/or portal hypertension with a high mortality rate. This spectrum is known as the alcoholic liver disease spectrum.

In the beginning stages of an inflammatory response, cell byproducts such as proteinases and reactive oxygen radicals have the potential to start the necrosis of hepatocellular tissue, which then leads to the release of a large number of cytokines. After hepatic damage, there is an increase in extracellular matrix, activation of stellate cells, an increase in rough endoplasmic reticulum, and expression of smooth muscle specific alpha chain. All of these changes take placein the liver[6] Activated stellate cells are susceptible to influence from a wide variety of cytokines.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

While some of them have an impact that is similar to that of proliferative on stellate cells, others stimulate fibrogenesis [6-8].

OBJECTIVES OF THE STUDY

1. To study the levels of Serum cholinesterase and hs-CRP in alcoholic cirrhotics and healthy controls.
2. To study the levels of Serum cholinesterase and hs-CRP in alcoholic cirrhoticis and non-alcoholic cirrhotics.
3. To study the efficacy of Serum cholinesterase and hs-CRP levels so as to differentiate alcoholic cirrhosis from non-alcoholic cirrhotics.

METERIALS AND METHODS

Place and Duration: The study is done Kakatiya Medical College/MGM Hospital, Warangal, Telangana, India. . The study period is from July 2019 to June 2021.

A) Selection of study subjects:

The case control study involved 150 subjects.

Based on inclusion and exclusion criteria a total number of 100 subjects (50 cases and 50 controls) were selected for the present study.

Inclusion Criteria

50 males who are diagnosed with alcoholic liver disease with history of consumption of alcohol spirits of >60g in males on daily basis for duration of more than 8 to 10 years, clinical signs of liver disease, supporting biochemical tests and ultrasonographic features.

Controls: 50 males who are non-alcoholic, normal, healthy individuals.

Exclusion Criteria

- Patients with clinical evidence of hypertension, diabetes mellitus, pancreatitis and renal failure.
- Patients having documented evidence of chronic hepatitis B or C.
- Patients having documented evidence of organophosphorus poisoning.
- Patients having history of intake of hepatotoxic drugs.
- Patients with history of congestive heart failure and myocardial infarction.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

- Patients with history of any inflammatory diseases.
- Other causes of cirrhosis.

Based on the inclusion and exclusion criteria, age matched cases and controls were included in the present study after obtaining informed consent. A proforma was used to record relevant information and patient's data. Alcohol drinking history was assessed by interview and questionnaire. Data from questionnaire was used to establish consumed duration type and pattern of alcohol intake.

Additional Study Group: 50 non-alcoholic cirrhosis cases confirmed by ultrasonography were included in this study as additional study group.

B) Collection of blood samples:

Following selection of subjects and after obtaining informed consent about the proposed study, clinical history was taken from subjects and examination findings were noted down. About 5ml of venous blood sample wascollected from median cubital vein by venipuncture with aseptic precautions. Serum was separated by centrifugation and separated serum was used for analysis of parameters.

C) Parameters estimated in the study subjects:

In the present study following parameters were estimated:

1. Serum Cholinesterase (CHE)
2. High sensitive C-reactive protein (hs-CRP)
3. Total Bilirubin
4. Total Protein
5. Serum Albumin
6. A:G ratio
7. Aspartate aminotransferase (AST)
8. Alanine aminotransferase (ALT)
9. Alkaline Phosphatase (ALP)

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

10. Gamma glutamyl transferase (GGT)

Estimation of total bilirubin by photometric colour test [6,7]

Statistical analysis:

The Statistical software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0 and R environment ver.2.11.1 were used for analysis of the data and Microsoft Word and Microsoft Excel have been used to generate tables, graphs etc.

RESULTS

Study design: A comparative case-control study involved 80 subjects of whom 40 were cases admitted to MGM Hospital attached to Kakatiya Medical College with diagnosis of alcoholic cirrhosis and 40 were age and sex matched healthy non- alcoholic controls and other 40 were Non-alcoholic cirrhosis as additional study group. The results were tabulated in master chart and statistically analysed.

AGE DISTRIBUTION AMONG STUDY SUBJECTS:

Among 50 cases in this study, 10 were in 31-40 years age group with 33.33%, 11 were in 41-50 years age group with 36.67% and 9 were in 51-60 years age group with50%.
Among 50 controls in this study, 7 were in 31-40 years age group with 23.33%, 15 were in 41-50 years age group with 50% and 8 were in 51-60 years age group with 26.67%.
Among 50 Non-ALC group in this study, 9 were in 31-40 years age group with 30%,12 were in 41-50 years age group with 40% and 9 were in 51-60 years age group with 30%.

Table No.1: Age distribution of patients studied

Age in years	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
31-40	15	30	12	24	15	30
41-50	21	42	25	50.00	22	44
51-60	14	28	13	26	13	26
Total	50	100.00	50	100.00	50	100.00
Mean ± SD	45.56±8.21		45.9±7.61		45.26±9.2	

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

DISTRIBUTION OF SERUM BILIRUBIN AMONG STUDY SUBJECTS:

The Total Bilirubin was studied accordingly, among 50 cases, ca

ses were >1.2mg/dl with 90 % and 5 cases were <1.2 mg/dl with 10%. Among 50 controls, 48 were <1.2 mg/dl group with 96 %. Among 50 Non-ALC group, 47 were >1.2 mg/dl with 94 % and 3 was <1.2 mg/dl group with 6 %.

The Direct bilirubin among cases were >0.5 in 88 %, <0.25 in 8 % and 0.25-0.5in 4 % whereas in controls it was <0.25 in 90 %. Among Non-ALC group, 15 were in >0.5 with 30 %, 22 were in <0.25 with 44 % and 13 were in 0.25-0.5 with 26 %.

Distribution of total bilirubin and direct bilirubin were shown in Table No.2a &2band Graph No.2a & 2b respectively.

Table No. 2a: Distribution of total bilirubin (mg/dl) among patients studied

Total Bilirubin (mg/dl)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
<1.2	5	10	48	96	3	6
>1.2	45	90	2	4	47	94
Total	50	100.00	30	100.00	50	100.00

Table No. 2b: Distribution of direct bilirubin (mg/dl) among patients studied

Direct Bilirubin (mg/dl)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
<0.25	4	6.67	45	90	15	30
0.25-0.5	2	3.33	2	4	22	44
>0.5	44	90.00	3	6	13	26
Total	50	100.00	50	100.00	50	100.00

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

DISTRIBUTION OF TOTAL PROTEIN AMONG STUDY SUBJECTS:

Among 50 cases, 24 cases were in 6.0-7.8 g/dl group with 48%, 16 were in <6.0 g/dl group with 32% and 10 were in > 7.8 g/dl group with 20 %. Among 50 controls, 32 were in 6.0-7.8 g/dl group with 64 % and 16 were in > 7.8 g/dl group with 32 %. Among 50 Non-ALC group, 29 were in <6.0 g/dl group with 58 % and 21 were in 6.0-7.8 g/dl group with 42%.

Table No. 3: Distribution of total protein (g/dl) among patients studied

Total Protein (g/dl)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
<6.0	16	32	2	4	29	58
6.0-7.8	24	48	32	64	21	42
>7.8	10	20	16	32	0	0.0
Total	50	100.00	30	100.00	50	100.00

DISTRIBUTION OF ALBUMIN AMONG STUDY SUBJECTS:

Among 50 cases, 46 cases were in <3.5 g/dl group with 92% and 3 was in 3.5- 5.2 g/dl group with 6%. Among 50 controls, all 50 were in 3.5-5.2 g/dl group with 100%. Among 50 Non-ALC group, 38 were in <3.5 g/dl group with 76 % and 12 werein 3.5-5.2 g/dl group with 24 %.

Table No. 4: Distribution of Serum albumin (g/dl) among patients studied

Serum Albumin (g/dl)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
<3.5	46	92	0	0.0	38	76
3.5-5.2	3	6	50	100.0	12	24
>5.2	1	2	0	0.0	0	0
Total	50	100.00	50	100.00	50	100.00

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

DISTRIBUTION OF A:G RATIO AMONG STUDY SUBJECTS:

Among 50 cases, 46 cases were in <1 group with 92% and 4 was in 1-1.5 group with 8%. Among 50 controls, 43 were in 1-1.5 group with 86 % and 7 were in >1.5 group with 14%. Among Non-ALC group, 30 were in 1-1.5 group with 60% and 20 were in <1 group with 40%.

Table No. 5: Distribution of A:G Ratio among patients studied

A:G Ratio	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
<1	46	92	0	0.0	30	60
1-1.5	4	8	43	86	20	40
>1.5	0	0.0	7	14	0	0.0
Total	50	100.00	50	100.00	50	100.00

DISTRIBUTION OF AST AMONG STUDY SUBJECTS:

Among 50 cases,44 cases were in >42 U/L group with 88 % and 6 were in 0-42 U/L group with 12 %. Among 50 controls, all50 were in 0-42 U/L groupwith100%.Among 50 Non-ALC group, all 50 were in >42 U/L group with 100%.

Table No. 6: Distribution of AST (U/L) among patients studied

AST (U/L)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
0-42	6	12	50	100.00	0	0.0
>42	44	88	0	0.0	50	100.00
Total	50	100.00	50	100.00	50	100.00

DISTRIBUTION OF ALT AMONG STUDY SUBJECTS

Among 50 cases, 38 were in 0-48 U/L group with 76 % and 12 were in >48 group with 24 %. Among 50 controls, all 50 were in 0-48 U/L with 100%. Among 50 Non-ALC group, 46were in >48 U/L group with 92 % and 4 were in 0-48 U/L group with 8 %.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

Table No. 7: Distribution of ALT (U/L) among patients studied

ALT (U/L)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
0-48	38	76	50	100.00	4	8
>48	12	24	0	0.0	46	92
Total	50	100.00	50	100.00	50	100.00

DISTRIBUTION OF ALP AMONG STUDY SUBJECTS

Among 50 cases, 40 cases were in 100-200 U/L group with 80%, 8 were in >200 U/L group with 16 % and 4 were in <100 U/L group with 8 %. Among 50 controls, 30 were in <100 U/L group with 60% and 20 were in 100-200 U/L group
with 40 %. Among 50 Non-ALC group, 42 were in 100-200 U/L group with 84 %

and 8 were in <100 U/L group with 16.0%.

Table No. 8: Distribution of ALP (U/L) among patients studied

ALP (U/L)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
<100	4	8	30	60	8	16
100-200	40	80	20	40	42	84
>200	8	16	0	0	0	0
Total	50	100.00	50	100.00	50	100.00

DISTRIBUTION OF GGT AMONG STUDY SUBJECTS

Among 50 cases, 26 cases were in 50-150 U/L group with 52 %, 14 were in <50 U/Lgroup with 28 % and 10 were in >150 U/L group with 20 %. Among 50 controls, all50 were in <50 U/L with 100%. Among 50 Non-ALC group, all 50 were in <50 U/Lwith100%.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

Table No. 9: Distribution of GGT (U/L) among patients studied

GGT (U/L)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
<50	14	28	50	100.00	50	100.00
50-150	26	52	0	0.0	0	0.0
>150	10	20	0	0.0	0	0.0
Total	50	100.00	50	100.00	50	100.00

DISTRIBUTION OF SERUM CHOLINESTERASE AMONG STUDY SUBJECTS

Among 50 cases, 44cases were in <4000 IU/L group with 88 % and 6 were in4000-8000 IU/L group with 12 %.Among 50 controls, 34 were in 4000-8000 IU/L group with 68 % and 16 were in>8000 IU/L group with 32 %.Among Non-ALC group, 26 were in <4000 IU/L group with 52 % and 24 werein 4000-8000 IU/L group with 48 %.Distribution of Serum Cholinesterase is shown in Table No. 10 and Graph No. 10

Table No. 10: Distribution of Serum Cholinesterase (IU/L) among patients studied

Serum Cholinesterase (IU/L)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
<4000	44	90.00	0	0.0	26	32
4000-8000	6	10.00	34	70.00	24	28
>8000	0	0.0	16	50.00	0	0.0
Total	50	100.00	50	100.00	50	100.00

DISTRIBUTION OF hs-CRP AMONG STUDY SUBJECTS

Among 50 cases, all50 cases were in 0.5-2 mg/dL group with 100%. Among 50 controls, all 50 were in <0.5 mg/dL group with 100%. Among 50 Non-ALC group, 38were in 0.5 -2 mg/dL group with 72 % and 12were in >2 mg/dL group with 24 %.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

Table No. 11: Distribution of hs-CRP among patients studied

hs-CRP (mg/dL)	Cases		Controls		Non-ALC	
	No.	%	No.	%	No.	%
<0.5	0	0.0	50	100	0	0.0
0.5-2	50	100.00	0	0.0	38	76
>2	0	0.0	0	0.0	12	24
Total	50	100.00	50	100.00	50	100.00

Table No. 12: Comparison of Study variables in Cases and Controls

Parameters	Cases	Controls	p value
Total bilirubin	8.45±7.90	0.55±0.31	<0.001***
Direct bilirubin	3.78±3.88	0.12±0.14	<0.001***
Total Protein	6.54±1.22	7.48±0.52	<0.001***
Albumin	2.76±0.62	4.22±0.34	<0.001***
A:G	0.50 ±0.34	1.54 ±0.28	<0.001***
AST	128.23±97.43	29.5 ±7.78	<0.001***
ALT	41.52±35.12	23.22 ±8.92	0.0112*
ALP	165.16±74.65	87.86±19.02	<0.001***
GGT	91.65 ±47.45	22.44±9.06	<0.001***
hs-CRP	1.34 ±0.42	0.17±0.15	<0.001***
Serum Cholinesterase	2212.32±1185.34	7755.1±566.22	<0.001***

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

Table No. 13: Comparison of Study variables in Cases and Non-ALC group

Parameters	Cases	Non-ALC	p value
Total bilirubin	8.45±7.90	2.17±0.58	<0.001***
Direct bilirubin	3.78±3.88	0.55±0.42	<0.001***
Total Protein	6.54±1.22	5.65±0.86	0.0051**
Albumin	2.76±0.62	2.18±0.43	<0.001***
A:G	0.50 ±0.34	1.02±0.17	<0.001***
AST	128.23±97.43	142.4±52.11	0.4536
ALT	41.52±35.12	86.2±31.09	<0.001***
ALP	165.16±74.65	121.7±27.17	0.0016**
GGT	91.65 ±47.45	30.43±7.13	<0.001***
hs-CRP	1.34 ±0.42	1.59±0.28	<0.001***
Serum Cholinesterase	2212.32±1185.34	4032.73±991.03	<0.001***

DISCUSSION

In the present study, which is a case control study, serum cholinesterase, serum hs-CRP, and other liver function tests in 30 alcoholic cirrhosis patients were compared with those in 50 healthy age- and sex-matched non-alcoholic controls along with 50 non-alcoholic cirrhosis patients. This comparison was made to determine whether or not there was a significant difference between the groups. The data was collated, and a statistical analysis was performedon it.In this particular research project, the ages of the participants ranged anywhere from 31 to 60 years old, which was also the situation with the controls and the Non-ALC group.The average age of patients diagnosed with alcoholic cirrhosis was 45.56 years, with a standard deviation of 8.21 years, while the average age of healthy controls was 45.9 years, with a standard deviation of 7.61 years. The p value for this comparison was 0.3601, which is in linewith the findings of a study conducted by Diana C., who found that alcoholic cirrhosis is mostcommonly diagnosed in people aged 50 to 50 years. According to Helga Paula et al., the age range between 45 and 64 years old had the highest rate of age-specific death. The mean age ofthose in the Non-ALC group was 45.26 9.2 standard deviations [10,11].

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

There was a substantial elevation in Serum Bilirubin levels in alcoholic cirrhosis cases when compared to controls with a significant p value of less than 0.001. This elevation was seen in both Total and Direct forms.

The values of Mean SD for Direct Bilirubin among cases were 3.783.88, >0.5 in 88 percent,

0.25 in 8 percent, and 0.25-0.5 in 4 percent, whereas the values for Mean SD for Direct Bilirubin among controls were 0.120.14 and were 0.25 in 90 percent of the cases. In the Non-

ALC group, 15 people landed in the >0.5 category with a percentage of 30%, 22 people landed in the 0.25 category with a percentage of 44%, and 13 people landed in the 0.25-0.5 category with a percentage of 26%.

There was a statistically significant difference between the total and direct bilirubin levels in those with alcoholic cirrhosis and those in the non-ALC group, with a p value of less than 0.001 indicating a significant difference.According to the findings of this investigation, cases of alcoholic cirrhosis had lower levels of total protein in their serum than controls did. The mean and standard deviation of the total protein content in the cases and controls, respectively, were
6.54 and 1.22, and 7.48 and 0.52. With a p value of less than 0.001, the drop in serum total protein levels seen in instances is statistically extremely highly significant. This conclusion is consistent with the findings of the study by Gopinath et al., which demonstrated a considerable reduction in the levels of total protein in alcoholic liver disease. In comparison to cases with alcoholic cirrhosis, the total serum protein level was found to be significantly lower in the non-ALC group. In the cases group, the mean and standard deviation of total protein was 6.54 1.2,whereas in the Non-ALC group, it was 5.65 0.86. With a p value of 0.005, the decrease in serum total protein seen in the Non-ALC group is highly significant from a statistical point of view. This indicates that the liver's ability to synthesise substances is being weakened. The liver is the principal organ responsible for the production of plasma proteins. When the function of the liver is damaged, there is a disruption in the process of protein synthesis. Cirrhosis is one of the causes, and one of its effects is an enhanced catabolic state. Other causes include decreased availability of amino acids.

In this particular investigation, the levels of serum albumin were considerably lower in cases compared to controls, as indicated by a significant p value of less than 0.001. Albumin levels in the serum were measured to have a mean of 2.76 0.62 and a standard deviation of 4.22 0.34 for cases and controls, respectively. On the other hand, the levels of albumin in the serum dropped significantly with a p value of less than 0.001 in both the alcoholic patients and the Non-ALC group. Albumin levels in the serum were measured to have a mean and standard deviation of 2.76 and 0.62 for the cases and 2.18 and 0.43 for the non-ALC group, respectively. The main reasons of hypoalbuminemia in cirrhosis include decreased hepatic production of albumin as well as loss of albumin in ascitic fluid. cirrhosis can be diagnosed by measuring albumin levels in the blood.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

In this particular investigation, the ratio of albumin to globulin was substantially lower in patients compared to controls, with a mean of 0.47 and 1.28 in cases and controls, respectively, and a standard deviation of 0.17 and 0.19, respectively, with a significant p value of less than

[11]. On the other hand, when alcoholic patients were compared to the Non-ALC group, the A:G ratio was significantly lower in the alcoholic cases than in the Non-ALC group with ap value of less than 0.001. In this particular study, the A/G ratio works in the opposite direction.

The estimated levels of AST in cases of alcoholic cirrhosis were substantially higher than those in controls, with a p value of less than 0.001 indicating a highly significant difference between the two groups. Comparing patients and controls, the mean and standard deviation of AST came in at 128.2397 and 29.57.78, respectively. However, as compared to the controls, there was no significant increase in ALT levels in cases, and the p value for this comparison was 0.0112. In the cases, the mean and standard deviation of ALT were 41.33 and 36.13, respectively, while in the controls, the mean and standard deviation were 25.63 and 8.62, respectively. It is possible that the increased appearance of mitochondrial AST, the decrease in ALT production, and injury to other tissues that produce AST but not ALT are all factors that contribute to the elevation of AST in alcoholic cirrhosis. The mean and standard deviation of AST values were respectively 128.2397.43 for the cases and 142.452.11 for the Non-ALC group, with p=0.4526. With a very significant p value of 0.001, the levels of ALT were found to be marginally higher in cases of non-alcoholic cirrhosis as compared to cases of alcoholic cirrhosis. The mean and standard deviation of ALT levels were respectively 41.52 and 35.12 for cases and 86.2 and 31.09 for non-ALC individuals.

In this particular study, the mean level of ALP in patients with the disease was 166.16, with a standard deviation of 73.53, whereas the level of ALP in healthy controls was 87.86, with a standard deviation of 19.29. The increase in ALP levels in patients, as opposed to those in the control group, was extremely significant, with a p value of less than 0.001. The mean and standard deviation of ALP values were 165.16 and 74.65 for cases, and 121.7 and 27.17 for the Non-ALC group, respectively. There was a marginal increase in ALP levels in cases when compared to the Non-alcoholic cirrhosis group, and the difference was statistically significant($p = 0.0016$).

The standard deviation for GGT levels in cases was 48.61, while the mean was 90.7. The mean GGT level in the controls was 22.56, with a standard deviation of 9.06. The Non-ALC group had a mean GGT level of 30.45 with a standard deviation of 7.12. The serum GGT levels of

patients with alcoholic cirrhosis were considerably higher than those of the controls and the Non-ALC group, with a significant p value of less than 0.001. Because of this increase in serum GGT levels, microsomal induction can be attributed to alcohol consumption.

In this investigation, the levels of serum cholinesterase were shown to be lower in alcoholics compared to controls, with a p value of less than 0.001 indicating a very significant difference. According to the findings of the research conducted by Jeyamani Ramachandran, the mean andstandard deviation of serum cholinesterase are as follows: The Non-ALC group had a mean blood cholinesterase level of 4004.73, with a standard deviation of 971.03 [12]. The levels of serum cholinesterase dropped in both the alcoholic cirrhosis and the non-alcoholic cirrhosis groups. However, the levels dropped a lot more in the alcoholic cirrhosis cases compared to the non-alcoholic

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

cirrhosis cases, and the difference was statistically significant with a p valueof less than 0.001.With a p value of less than 0.001, this study found that the levels of hs-CRPin patients with the disease were significantly higher than those in the control group. The meanand standard deviation of the cases was 1.34 0.42, while the mean and standard deviation of the controls was 0.17 0.15. The levels of hs-CRP were found to be elevated in both the alcoholiccases and the Non-alcoholic group. However, the levels were found to be significantly higher in the Non-ALC group when compared to the alcoholic cases, with a p value of less than 0.001. The Non-ALC group had a mean value of 1.59 with a standard deviation of 0.28. It has been demonstrated in a number of studies that patients with non-alcoholic liver disease had elevatedlevels of hs-CRP, which indicates that this protein may perform as a biomarker.

CONCLUSION

In conclusion, the significant reduction in serum cholinesterase that is seen in patients with alcoholic cirrhosis suggests that the activity of this enzyme may be a specific indicator of liverdysfunction and may be helpful in diagnosing patients with alcoholic cirrhosis. Furthermore, the hs-CRP can be used as a strong predictor of non-alcoholic cirrhosis. To determine whetheror not they are useful as reliable and cost-effective biomarkers in alcoholic and non-alcoholic liver disease, similar studies on large study groups of patients suffering from alcoholic and non-alcoholic cirrhosis are required. The outcomes of the patients' liver diseases will serve as the basis for the studies.

Acknowledgment

The author is thankful to Department of Biochemistry for providing all the facilitiesto carry out this work.

Conflict of Interest

None

Funding Support Nil

REFERENCES

1. Tomita, Y., Misaka, T., Yoshihisa, A., Ichijo, Y., Ishibashi, S., Matsuda, M., ... & Takeishi, Y. (2022). Decreases in hepatokine Fetuin-A levels are associated with hepatic hypoperfusion and predict cardiac outcomes in patients with heart failure. *Clinical Research in Cardiology*, 1-9.
2. https://www.who.int/news-room/fact-sheets/detail/alcohol [assessed on 30 june 2022]
3. Maurya N.K "A Review: Patients Generated Subjective Global Assessment (PG- SGA)" in Journal International Research Journal of Pharmacy 2018, 9(11): 5-8.
4. NK Maurya. Nephrotoxic Effect of Herbal Medicine and Supplements: A Review. *Research & Reviews: A Journal of Toxicology*. 2019; 9(2) :28-35.
5. Rodriguez-Roisin R, Krowka MJ, Herve P, Fallon MB, Pulmonary-hepatic vascular disorders (PHD). Eur Respir 2004; 24(5):861-880.
6. Friedman SL. The cellular basis of hepatic fibrosis-Mechanism and treatment strategies. N Engl J Med 1993;328:1828.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 05, 2022

7. .Pinzami M. Hepatic stellate (Ito) cell: Expanding roles for a liver specific pericyte. J Hepatol 1995;22:700-706.
8. .Alan H Gowenlock, Janet R McMurray, Donald M McLauchlan. Varley's Practical Clinical Biochemistry. 6th Ed. New Delhi, Bangalore, Pune, Cochin, Chennai. CBS Publishers & Distributors Pvt. Ltd. 1988; 326, 350, 362, 499, 518, 532, 715, 796.
9. .https://www.beckmancoulter.com IFU of each test method in Beckman coulter AU480.
10. Helga Paula, Sumeet K. Asrani, Nicholas C. Boetticher, Rachel, Pedersen, Vijay H. Shah et al. Alcoholic Liver Disease-Related Mortality in the United States: 1980- 2003; Am J Gastroenterol. 2010 August; 105(8) 1782-87.
11. Knut Stokkeland, Gunnar Hilm, Fredrik Spak, Johan Franck, Rolf Hultcrantz; Different drinking patterens for women and men with alcohol dependence with and without alcoholic cirrhosis. Alcohol & Alcoholism, 2008;43(1):39-45.
12. .Jeyamani Ramachandran, KG Sajith, Sophiya Priya, Amit K Dutta, KA Balasubramanian. Serum cholinesterase is an excellent biomarker of liver cirrhosis. Tropical Gstroenterology 2014; 35(1):15-20.