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## Alcoholic liver disease – Clinical profile and correlation of severity with the help of electrocardiography

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### Abstract

**Background:** alcohol affects liver, and affects heart also so we try to correlate these cardiac changes with severity in patients of alcoholic liver disease patients. **Material and methods:** The present study entitled has been conducted in Department of Medicine, Jayarogya group of hospitals, GRMC Gwalior M.P. from Nov. 2012 to Nov. 2013. The present study consisted of 102 chronic liver disease patients without any evidence of prior cardiac dysfunction and were evaluated by using electrocardiography. **Results:** Majority of the patients (80%) are 30-60 years old. Among Group I (ALD) patients (n=68), maximum 32(47%) belongs to child class C. Similarly, among Group II (NALD) patients (n=34), 16(47%) belongs to child class C. Abdominal distension is the most common presenting symptom in the study population present in 73.5% of patients followed by swelling over legs in 35.2% of patients & yellowish discoloration of eyes & abdominal pain in 18.6% of patients. Icterus is the most common clinical sign seen in 63 patients followed by ascites in 53 & edema in 28 patients. In the study population, 14 patients had no Coronary risk factors, 45 have one Coronary risk factors; 36 had two Coronary risk factors; 6 had three Coronary risk factors; 1 had four Coronary risk factors. Majority of them had dyslipidemia (61 patients) followed by smoking history (34 patients) & diabetes (17 patients). ECG abnormalities correlated significantly with history of alcoholism with abnormal ECG recorded in 67.7% of Group I (ALD) patients as compared to 53% of Group II (NALD) patients. Among the Group I (ALD) patients significantly, 50% had prolonged QTc as compared to 29.4% of Group II (NALD) patients. ECG abnormalities correlated significantly with severity of liver disease in the Group I (ALD) with 4 of Child A patients had abnormal ECG as compared to 17 of Child B & 27 of Child C patients. In the Group I (ALD) only 1 of Child A patients had prolonged QTc as compared to 6 of child B & 27 of Child C patients which was significant. In Group I (ALD) 62 patients had abnormal ECG out of them 28 had two or more Coronary risk factors thus showing the strong association between them. **Conclusion:** ECG correlates with ALD patients severity.

**Keywords:** liver disease, alcoholic liver disease, Electrocardiography.

### 1. Introduction

Alcohol consumption is responsible for 3.8% of global mortality and 4.6% of disability-adjusted life-years (DALYs) lost due to pre-mature death<sup>1</sup>. Chronic and excessive alcohol ingestion is one of the major causes of liver disease. The pathology of alcoholic liver disease consists of three major lesions, with the injury rarely existing in a pure form: (1) fatty liver, (2) alcoholic hepatitis, and (3) cirrhosis. Ethanol use may cause transient hypotension after acute use. But chronic use of alcohol may cause persistent rise of blood pressure in a dose dependent manner. There is also 6 times risk of coronary artery disease with alcohol use. Also have increased risk of cardiomyopathy through direct action of alcohol on myocardium, that may manifest itself as arrhythmias, heart failure, cardiac chambers enlargements etc.<sup>2</sup> The current study has been done to find out various cardiac manifestations in ALD patients and to correlate their existence in ALD patients with or without additional coronary risk factors & to statistically find out a significant difference in these manifestations in various stages of ALD patients, & comparing them to non-Alcoholic liver disease patients demonstrating alcohol a major causative & contributing factor so to treat them ahead time and in explaining prognosis.

### 2. Material and Methods

The present study was conducted in department of medicine of G.R. Medical College,

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Gwalior, M.P. The present study is a case control study, conducted over a period between November 2012 to November 2013 on the total 102 patients of alcoholic & non-alcoholic liver disease patients with informed and written consent. In the present study two groups has been created and studied. Group I comprises of patients of Alcoholic liver disease which are taken as cases & Group II comprises of patients of Non-Alcoholic liver disease patients which are taken as controls in the present study. The selection criteria for inclusion in study were-

1. **Alcohol intake history:** of more than 40 g/day (men) for >10 yrs.
2. **One & more of the following:**
  - A. **Lab findings:** Deranged Liver function test i) AST (SGOT)  $\geq 84$  ( $>3$  times of ULN); ii) ALT (SGPT)  $\geq 56$ ; iii) SAP  $\geq 166$ ; iv) Serum bilirubin  $>1.5$  mg/dl;
  - B. **Ultrasonographic evidence:** of liver disease (altered liver echotexture)
  - C. **Histopathology:** findings suggestive of liver disease.
  - D. Patients satisfying both of the **inclusion criteria** are taken as cases & those satisfying only second inclusion criteria are taken as controls in the present study. Patients were **excluded** from study if-Patients with pre-existing coronary artery disease; documented evidence of pre-existing cardiomyopathy secondary to a non-alcoholic

non-cirrhotic cause; Diagnosed Patients of chronic airway disease / primary pulmonary hypertension. A detailed history was elicited from all patients with emphasis on symptomatology and history of presenting & past illness; personal & family history; drug & addiction history was taken. Detailed clinical evaluation including history including questioning about risk factors for chronic liver disease, history of hepatitis, alcohol consumption, diabetes mellitus, use of illicit drugs (by injection or inhalation), transfusions, family history of liver disease, travel, and the presence of autoimmune diseases (including inflammatory bowel disease, rheumatoid arthritis and thyroid disease). The review of systems including questioning related to fatigue, easy bruisability, lower extremity edema, fever, weight loss, pruritus, increasing abdominal girth, and confusion or sleep disturbance (possibly indicating encephalopathy). Clinical signs including spider naevi, gynaecomastia, anemia, low grade fever, white opaque nails, clubbing of nails, foetor hepaticus, jaundice, ascites, encephalopathy, Prominent veins over abdomen, caput medusa, hepatomegaly, splenomegaly. Patients were classified on disease severity on basis of CHILD PUGH TURCOTTE SCORE which is mentioned as below.

**Child Pugh Turcotte Score** <sup>[3]</sup>

Measure	1 point	2 points	3 points
Total bilirubin, $\mu\text{mol/l}$ (mg/dl)	$<34$ ( $<2$ )	34-50 (2-3)	$>50$ ( $>3$ )
Serum albumin, g/dl	$>3.5$	2.8-3.5	$<2.8$
PT INR	$<1.7$	1.71-2.30	$>2.30$
Ascites	None	Mild	Moderate to Severe
Hepatic encephalopathy	None	Grade I-II (or suppressed with medication)	Grade III-IV (or refractory)

The patients were also enquired & examined for the presence of following coronary risk factors: 1. POSITIVE FAMILY HISTORY OF CAD. 2. SMOKING. 3. HYPERTENSION (SBP $>120$  / DBP $>80$ ). 4. DIABETES / IFG / IGT (FBS $>126$  / PPBS  $>140$ ) (ADA). 5. DYSLIPIDEMIA (TC $>200$ , TG $>150$ , HDL $<40$ ) (AHA)

The patients are also classified on the basis of number of CAD risk factors mentioned above.

CAD RF 0 –having no CAD risk factor. CAD RF 1- having one CAD risk factor. CAD RF 2- having two CAD risk factor. CAD RF 3- having three CAD risk factor. CAD RF 4- having four CAD risk factor. CAD RF 5- having five CAD risk factor.

All patients were subjected to the following investigation at the time of inclusion into the study.

Routine hemogram. (Hb, TLC, DLC, Platelet), Liver function test (Serum Bilirubin, SGOT, SGPT, SAP, S. protein, PT), Fasting and post prandial blood sugar, Ultrasonography of abdomen, Lipid profile (total cholesterol, triglycerides, LDL, HDL, VLDL), Blood urea and serum creatinine, and electrocardiography.

### 2.1 Statistical Methods and statistical analysis

Analysis of variance (ANOVA) has been used to find the significance of study parameters between three or more groups of patients analyzing the quantitative measurements of LV dimensions, Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups (Inter group analysis) and chi square test has been used to analyse the data having ordinal variables.

Significant figures were analysed, the Statistical software namely SPSS 22.0, for the analysis of the data and Microsoft word and Excel were used to generate graphs, tables. A p value of  $<0.05$  was considered as significant.

### 3. Results

In this present study population maximum number of patients 44 (43.1%) were 41-50 years old, 10 (9.8%) patients were less than 30 years old; 25 (24.5%) patients were 31-40 years old; 13 (12.7%) patients were 51-60 years old & 10 (9.8%) patients were older than 60 years (table-1). Out of 102 patients Group I comprising of 68 ALD patients & Group II comprising of 34 NALD patients served as cases & controls respectively in the present study. Among ALD patients, maximum 32 (47%) patients belongs to Child pugh class C. 8 (11.7%) belongs to Child pugh class A; 28 (41.3%)

belongs to Child pugh class B; Similarly, among NALD patients, maximum 16(47%) patients belongs to Child pugh class C. 4(11.7%) belongs to Child pugh class A; 14 (41.3%) belongs to Child pugh class B(table 2). In the study population 75(73.5%) patients had complaint of abdominal distension suggesting it as the most common presenting symptom in the study followed by swelling over legs in 36(35.2%) patients & yellowish discoloration of eyes & abdominal pain in 19(18.6%) patients. hematemesis/malena and fever(15.6%), weakness(14.7%) and altered sensorium(12.7%) were presents in decreasing trends(table 3).In the study population 63 (61.7%) patients had icterus on examination showing it as the most common clinical sign followed by ascites seen in 53(51.9%) patients, edema in 28(27.4%) patients, palpable liver in 20(19.6%) patients, palpable spleen in 17(16.6%) patients, clubbing in 10(9.8%) patients & asterixis in 12(11.7%) patients(table 4).In the study population none of the patients had given a positive family history of CAD.Among ALD patients (group I )23(33.8%) were smokers; 9(13.2%) were diabetics; 10(14.7%) were hypertensives & 38(55.8%) had deranged lipid profile. Among NALD (group II patients) 11(28.9%) were smokers; 8(21%) were diabetics; 4(10.5%) were hypertensives & 23(60%) had deranged lipid profile(table 5).In Group I (ALD), 10 patients had no CAD RISK FACTORS, 30 have one CAD RISK FACTORS; 23 had two CAD RISK FACTORS; 4 had three CAD RISK FACTORS; 1 had four CAD RISK FACTORS.In Group II (NALD), 4 patients had no CAD RISK FACTORS, 15 have one CAD RISK FACTORS; 13 had two CAD RISK FACTORS; 2 had three CAD RISK FACTORS.(table 6).A total of 93(91.1%) patients out of 102 had anemia out of them 49 (48%) had severe anemia while 8(7.8%) had leucopenia, 21(20.5%) had leukocytosis& 41 (40.19%) had thrombocytopenia(table 7).A total of 62(60.78%) patients out of 102 had abnormal ECG. Significantly, among the ALD patients 46(67.7%) had

abnormal ECG while among NALD patients only 16(53%) had abnormal ECG (table-8). Significantly, among the ALD patients 34(50%) had prolonged QTc as compared to 10 (29.4%) NALD patients which is the most common abnormality in ECG followed by chamber enlargement i.e. enlarged LA & LV observed in 13(19.11%) ALD patients. (table 9) In the Group I significantly only 2 of Child A patients had abnormal ECG as compared to 17 of child B & 27 of Child C patients. In the GROUP II significantly only 1 of Child A patient had prolonged QTc as compared to 7 of child B & 8 of Child C patients.- (table 10)Significantly, in GROUP I ,46 patients had abnormal ECG out of them 3 had no CAD risk factor, 15 had one CAD risk factor, 23 had two CAD risk factor, 4 had three CAD risk factor; & 1 had four CAD risk factor(table 12-A)In GROUP II 16 patients had abnormal ECG out of them 1 had no CAD risk factor, 9 had one CAD risk factor, 6 had two CAD risk factor.(table 12-B).Maximum ecg abnormalities were in patients with 1 risk factor (30 )followed by 2 risk factors(23) then 0 risk factor(10)and then 3 risk factor (4), min patients were associated with 4 (1)risk factors. QTc interval (16) in patients with 2 risk factor (table 13-A). Maximum patients in this group were of 1 risk factor (15), followed by 2 risk factor(13) , then 0 risk factor( 4), and then 3 risk factor(2) . Minimum patients were in group with 4 risk factor (0) . QTc prolongation was presents in 5 patients each with 1 and 2 risk factors (table 13-B)

**Table 1:** Age Distribution of Study Population

Age (Years)	TOTAL (%)
<31	10 (9.8%)
31-40	25 (24.5%)
41-50	44 (43.1%)
51-60	13 (12.7%)
>60	10 (9.8%)

**Table 2:** Disease Severity Distribution in Study Population

GROUP I (ALD) N=68			GROUP II (NALD) N=34			TOTAL (%)
CHILD PUGH A	CHILD PUGH B	CHILD PUGH C	CHILD PUGH A	CHILD PUGH B	CHILD PUGH C	
8 (11.7%)	28 (41.3%)	32 (47%)	4 (11.7%)	14 (41.3%)	16 (47%)	102

**Table 3:** Symptom Presentation in Study Population

SYMPTOMS	GROUP I (ALD) N=68			GROUP II (NALD) N=34			TOTAL (%)
	CHILD PUGH A (N=8)	CHILD PUGH B (N=28)	CHILD PUGH C (N=32)	CHILD PUGH A (N=4)	CHILD PUGH B (N=14)	CHILD PUGH C (N=16)	
ABDOMINAL DISTENSION	5	23	25	2	10	10	75(73.5%)
SWELLING OVER LEGS	2	14	12	0	3	5	36(35.2%)
YELLOWISH DISCOLORATION OF EYES & URINE	-	2	10	0	1	6	19(18.6%)
ABDOMINAL PAIN	4	5	2	0	4	4	19(18.6%)
HEMATEMESIS / MALENA	1	2	7	1	5	0	16(15.6%)
FEVER	1	5	3	1	2	4	16(15.6%)
WEAKNESS	3	4	2	1	5	0	15(14.7%)
ALTERED SENSORIUM	-	2	7	0	1	3	13(12.7%)

**Table 4:** Clinical Sign Presentation in Study Population

SIGNS	GROUP I (ALD) N=68			GROUP II (NALD) N=34			TOTAL (%)
	CHILD PUGH A (N=8)	CHILD PUGH B (N=28)	CHILD PUGH C (N=32)	CHILD PUGH A (N=4)	CHILD PUGH B (N=14)	CHILD PUGH C (N=16)	
ICTERUS	2	15	26	1	7	12	63(61.7%)
ASCITES	4	16	14	1	7	11	53(51.9%)
EDEMA	1	7	3	1	7	9	28(27.4%)
HEPATOMEGALY	-	2	8	1	7	2	20(19.6%)
SPLENOMEGALY	4	2	2	0	3	6	17(16.6%)
CLUBBING	0	2	7	1	0	0	10(9.8%)
ASTERIXIS	0	2	8	0	0	2	12(11.7%)

**Table 5:** Distribution of Cad Risk Factors in Study Population

CAD Risk factor	GROUP I (ALD) N=68			TOTAL (%)	GROUP II (NALD) N=34			TOTAL (%)
	CHILD PUGH A (N=8)	CHILD PUGH B (N=28)	CHILD PUGH C (N=32)		CHILD PUGH A (N=4)	CHILD PUGH B (N=14)	CHILD PUGH C (N=16)	
1. POSITIVE FAMILY HISTORY	0	0	0	0 (0%)	0	0	0	0 (0%)
2. SMOKER	3	10	10	23 (33.8%)	2	5	4	11 (28.9%)
3. DIABETIC	2	4	3	9 (13.2%)	1	4	3	8 (21%)
4. HYERTENSION	1	5	4	10 (14.7%)	0	2	2	4 (10.5%)
5. DYSLIPIDEMIA	3	18	17	38 (55.88%)	1	11	11	23 (60.5%)

**Table 6:** Distribution of Number of Cad Risk Factors in Study Population

CAD Risk factor	GROUP I (ALD) N=68				GROUP II (NALD) N=34			
	CHILD PUGH A (N=8)	CHILD PUGH B (N=28)	CHILD PUGH C (N=32)	Total	CHILD PUGH A (N=4)	CHILD PUGH B (N=14)	CHILD PUGH C (N=16)	Total
RISK FACTOR 0	2	2	6	10	1	1	2	4
RISK FACTORS 1	4	12	14	30	2	5	8	15
RISK FACTORS 2	2	12	9	23	1	7	5	13
RISK FACTORS 3	0	2	2	4	0	1	1	2
RISK FACTORS 4	0	0	1	1	0	0	0	0

**Table 7:** Hemogram parameters in study population

Parameters	TOTAL (%) N=102
1. Normal Hb(>13g/dl)	9 (8.8%)
2. Anemia (Hb <13g/dl)	93 (91.1%)
3. Severe anemia (Hb <8g/dl)	49 (48%)
4. Normal Leucocytic count (TLC 4000-11000)	73 (71.5%)
5. Leucopenia (TLC<4000)	8 (7.8%)
6. Leucocytosis (TLC>11000)	21 (20.5%)
7. Normal platelet count (Platelet>1.5 lakhs)	61(59.8%)
8. Thrombocytopenia (Platelet<1.5 lakhs)	41 (40.19%)

**Table 8:** Distribution of ECG findings in study population

	GROUP I (ALD) N=68	GROUP II (NALD) N=34	Total N=102
1.NORMAL ECG	22(32.3%)	18(47%)	40(39.22%)
2.ABNORMAL ECG *P = 0.047	46(67.7%)	16(53%)	62(60.78%)
Total	68(66.67%)	34(33.33%)	102(100%)

**Table 9:** Pattern of ECG abnormalities in study population

ECG FINDINGS	GROUP I (ALD) N=68	GROUP II (NALD) N=34
1.SINUS TACHYCARDIA	3(4.4%)	2(5.8%)
2.SINUS BRADYCARDIA	5(7.3%)	0
3. CHAMBER ENLARGEMENT (Dilated LA & LV)	13(19.11%)	0
4. CONDUCTION ABNORMALITIES (like BBB & fascicular blocks)	8(11.6%)	0
5. ST-T CHANGES	7(10.3%)	3(8.8%)
6.QTC PROLONGATION *P = 0.047	34(50%)	10(29.4%)
7.ARRYTHMIA's (like SA, SVT, VPC)	5(7.3%)	3(8.8%)
8.LOW VOLTAGE COMPLEXES	0(0%)	1(2.9%)

**Table 10:** Distribution of ECG Findings in Study Population as Per Severity Class

ECG FINDINGS	GROUP I (ALD) N=68				GROUP II (NALD) N=34			
	CHILD PUGH A (N=8)	CHILD PUGH B (N=28)	CHILD PUGH C (N=32)	TOTAL%	CHILD PUGH A (N=4)	CHILD PUGH B (N=14)	CHILD PUGH C (N=16)	TOTAL %
1.NORMAL ECG	6	11	5	22(32.3%)	3	7	8	18(47%)
2.ABNORMAL ECG *P = 0.015	2	17	27	46(67.7%)	1	7	8	16(53%)

**Table 11:** Pattern of ECG Abnormalities in Study Population as Per Severity Class

ECG FINDINGS	GROUP I (ALD) N=68				GROUP II (NALD) N=34			
	CHILD PUGH A (N=8)	CHILD PUGH B (N=28)	CHILD PUGH C (N=32)	TOTAL%	CHILD PUGH A (N=4)	CHILD PUGH B (N=14)	CHILD PUGH C (N=16)	TOTAL %
1.SINUS TACHYCARDIA	0	0	3	3 (4.4%)	0	2	0	2 (5.8%)
2.SINUS BRADYCARDIA	0	2	3	5 (7.3%)	0	0	0	0
3. CHAMBER ENLARGEMENT	0	4	9	13 (19.11%)	0	0	0	0
4. CONDUCTION ABNORMALITIES	0	5	3	8 (11.6%)	0	0	0	0
5. ST-T CHANGES	0	5	2	7 (10.3%)	1	1	1	3 (8.8%)
6. QTC ROLONGATION *P = 5.7e <sup>-7</sup> (highly significant)	1	6	27	34 (50%)	0	5	5	10 (29.4%)
7. ARRYTHMIA's	1	1	3	5 (7.3%)	0	0	3	3 (8.8%)
8.LOW VOLTAGE COMPLEXES	0	0	0	0 (0%)	0	0	1	1 (2.9%)

**Table 12A:** Distribution of ECG Findings in Group I as Per Number of Cad Risk Factors

ECG FINDINGS	GROUP I (ALD) N=68					Total	%
	CAD RISK FACTOR 0 N=10	CAD RISK FACTOR 1 N=30	CAD RISK FACTOR 2 N=23	CAD RISK FACTOR 3 N=4	CAD RISK FACTOR 4 N=1		
1.NORMAL ECG	7 (30%)	15 (50%)	0 (0%)	0 (0%)	0 (0%)	22	32.4%
2.ABNORMAL ECG *P = 0.0008	3 (30%)	15 (50%)	23 (100%)	4 (100%)	1 (100%)	46	67.6%

**Table 12B:** Distribution of ECG Findings in Group Ii as Per Number of Cad Risk Factors

ECG FINDINGS	GROUP II (NALD) N=34					Total	%
	CAD RISK FACTOR 0 N=4	CAD RISK FACTOR 1 N=15	CAD RISK FACTOR 2 N=13	CAD RISK FACTOR 3 N=2	CAD RISK FACTOR 4 N=0		
1.NORMAL ECG	3 (67%)	6 (40%)	7 (53.8%)	2 (100%)	0 (0%)	18	53%
2.ABNORMAL ECG	1 (33%)	9 (60%)	6 (47.2%)	0 (0%)	0 (100%)	16	47%

**Table 13A:** Pattern of ECG Abnormalities in Group I as Per Number of Cad Risk Factors

ECG FINDINGS	GROUP I ALD (N=68)					Total (%)
	CAD RISK FACTOR 0 N=10	CAD RISK FACTOR 1 N=30	CAD RISK FACTOR 2 N=23	CAD RISK FACTOR 3 N=4	CAD RISK FACTOR 4 N=1	
1.SINUS TACHYCARDIA	1 (10%)	1 (3.3%)	0 (0%)	0 (0%)	1 (100%)	3(4.4%)
2.SINUS BRADYCARDIA	1 (10%)	2 (6.6%)	1 (4.3%)	1 (25%)	0 (0%)	5(7.3%)
3.CHAMBER ENLARGEMENT	2 (20%)	7 (23.3%)	4 (17.2%)	0 (0%)	0 (0%)	13(19.11%)
4. CONDUCTION ABNORMALITIES	0 (0%)	3 (9.9%)	5 (21.5%)	0 (0%)	0 (0%)	8(11.6%)
5. ST-T CHANGES	1 (10%)	1 (3.3%)	3 (12.9%)	2 (50%)	0 (0%)	7(10.3%)
6. QTC PROLONGATION	3 (30%)	12 (40%)	16 (68.8%)	2 (50%)	1 (100%)	34(50%)
7.ARRYTHMIA	1 (10%)	3 (9.9%)	1 (4.3%)	0 (0%)	0 (0%)	5(7.3%)
8.LOW VOLTAGE COMPLEXES	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0(0%)

**Table 13B:** Pattern of ECG Abnormalities in Group Ii as Per Number of Cad Risk Factors

ECG FINDINGS	GROUP II NALD (N=34)					Total (%)
	CAD RISK FACTOR 0 N=4	CAD RISK FACTOR 1 N=15	CAD RISK FACTOR 2 N=13	CAD RISK FACTOR 3 N=2	CAD RISK FACTOR 4 N=0	
1.SINUS TACHYCARDIA	0 (0%)	1 (6.6%)	1 (7.6%)	0 (0%)	0 (0%)	2(5.8%)
2.SINUS BRADYCARDIA	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0
3.CHAMBER ENLARGEMENT	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0
4. CONDUCTION ABNORMALITIES	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0
5. ST-T CHANGES	1 (25%)	1 (6.6%)	1 (7.6%)	0 (0%)	0 (0%)	3(8.8%)
6. QTC PROLONGATION	0 (0%)	5 (33%)	5 (38%)	0 (0%)	0 (0%)	10(29.4%)
7.ARRYTHMIA	0 (0%)	2 (13.2%)	1 (7.6%)	0 (0%)	0 (0%)	3(8.8%)
8.LOW VOLTAGE COMPLEXES	0 (0%)	0 (0%)	1 (7.6%)	0 (0%)	0 (0%)	1(2.9%)

**4. Discussion**

The present study entitled as “to study 2D-echocardiography findings in alcoholic liver disease patients” has been conducted in Department of Medicine, Jayarogya group of hospitals, GRMC Gwalior M.P. from Nov. 2012 to Nov. 2013. There is dual effect on heart in ALD patient owing to

alcohol & liver disease per se. Patients with alcoholic cirrhosis and end-stage liver disease may have alcohol-related heart disease (alcoholic cardiomyopathy), heart disease associated with cirrhosis per se (cirrhotic cardiomyopathy), or coincidental heart disease (e.g. CAD). There are limited data regarding the prevalence of these

types of heart disease in patients with ESLD, particularly in patients with alcoholic cirrhosis. The current study has been done to find out various cardiac manifestations in ALD patients and to correlate their existence in ALD patients with or without additional coronary risk factors & to statistically find out a significant difference in these manifestations in various stages of ALD patients & comparing them to non-Alcoholic liver disease patients demonstrating alcohol as a major causative or contributive factor so to treat them ahead time and in explaining prognosis.

#### 4.1 Age & Disease Severity

A total of 102 patients of chronic liver disease are studied. 80% of the patients are of 30-60 year age group clearly reflecting the disease is effecting the most productive and economically earning age group. Group I comprising of 68 ALD patients & Group II comprising of 34 NALD patients served as cases & controls respectively in the present study. 8 patients were of Child class A, 28 were of Child class B & 32 were of Child class C severity of liver disease in GROUP I (ALD). 4 patients were of Child class A, 14 were of Child class B & 16 were of Child class C severity of liver disease in GROUP II (NALD). It shows clearly that patient comes to medical care services very late in the course of disease which reflects low awareness & lack of knowledge in population about the disease.

#### 4.2 Symptomatology

In the study population 73.5% patients had complaint of abdominal distension suggesting it as the most common presenting symptom in the study followed by swelling over legs present in 35.2% of patients & yellowish discoloration of eyes & abdominal pain in 18.6% of patients. Specifically altered sensorium is seen in 13 patients is clearly more common in CHILD C class (10 patients) as compared to CHILD B (3 patients) patients suggesting hepatic encephalopathy is more common in CHILD C class patients. In the study population 63 patients had icterus on examination showing it as the most common clinical sign followed by ascites seen in 53 & edema in 28 patients. Important point in the study is that Asterixis is clearly more commonly seen in CHILD C class as compared to CHILD B patients suggesting hepatic encephalopathy is more common in CHILD C class patients. Similarly Hepatomegaly is more common in CHILD A & B class suggesting shrinking of liver secondary to cirrhosis is more common in CHILD class C i.e. during later part of disease.

#### 4.3 Coronary Risk Factors

Among ALD patients 23(33.8%) were smokers; 9(13.2%) were diabetics; 10(14.7%) were hypertensives & 38(55.8%) had deranged lipid profile. Among NALD patients 11(28.9%) were smokers; 8(21%) were diabetics; 4(10.5%) were hypertensives & 23(60%) had deranged lipid profile. On classifying the study population on the basis of number of CAD risk factors: 14(13.7%) patients had no CAD risk factors, 45(44.1%) have one CAD risk factors; 36(35.2%) had two CAD risk factors; 6(5.8%) had three CAD risk factors; 1(0.9%) had four CAD risk factors.

#### 4.4 ECG Abnormalities

A total of 62 patients out of 102 had abnormal ECG out of them 46 were ALD & 16 were NALD patients. This significant difference concurs with the prior studies.<sup>[4]</sup> Among the ALD patients 50% had prolonged QTc as compared to 29.4% of NALD patients which shows Alcoholics have much higher incidence of prolonged QTc. This significant difference also concurs with the prior studies.<sup>[7-9]</sup> Campbell *et al.* (1953) showed presence of QT and QTC prolongation in alcoholics which served as a marker of mortality.<sup>[5]</sup> Kochegurov VN. *et al.* (1983) studied ECG changes in alcoholic patients, the most common ECG changes recorded at rest were shortened PQ interval, lengthened QT, rhythm and conductivity disorders, flattened P waves, whereas typical exercise-associated changes were elevated systolic parameters. As alcoholism progressed, there was an obvious tendency to increased incidence of conductivity disorders, flattened P waves and elevated systolic values during exercise.<sup>[6]</sup>

#### 4.5 ECG Abnormalities & Liver Disease

In present study in GROUP I (ALD) 4 of Child A patients had abnormal ECG as compared to 17 of child B & 27 of Child C patients which is significant difference clearly showing higher incidence of ECG abnormalities among patients having more severe liver disease. Also, in GROUP I (ALD) only 1 of Child A patients had prolonged QTc as compared to 6 of child B & 27 Child C patients which is significant clearly showing with increasing severity of disease the incidence of prolonged QTc is also increasing. These findings are consistent with prior studies.<sup>[10-12]</sup> Anand *et al* done preoperative evaluation of OLT patients in which Electrocardiography recording showed T-wave inversion in 7 patients, ventricular premature beats in 3 patients, evidence of old myocardial infarction in 1, and normal rhythms in rest of the patients. In present study 10 patients had ST-T changes & 8 had arrhythmias (like SA, SVT, VPC, WPW syndrome) on ECG but it was not significant.<sup>[13]</sup> The present study supports the conclusion of the results of a trial conducted by Puthumana *et al.*

<sup>[14]</sup> and Bernardi *et al.*<sup>[15]</sup> that explains that the degree of liver dysfunction is significantly correlated with the prevalence of prolonged QTc-interval. Zamirian *et al* in 2012 in his study concluded that prolonged QTc interval was a common finding in cirrhotic patients with majority of values subsequently normalizing after OLT.<sup>[16]</sup> QTc-interval > 440 msec indicates that the cardiac muscle cells that work in response to electric signals cannot cause normal pulsation anymore, but prolonged QTc-interval is not always correlated with the appearance of clinical symptoms. Literature explains that prolonged QTc-interval > 600 msec often triggers Torsades de pointes that can further proceed to be ventricular fibrillation, and even end in sudden death. It is expected that caution should be exercised to do preventive measures such as avoiding drugs that can cause prolonged QTc-interval and/or trigger Torsade de points as emphasized by Morissette P *et al.*<sup>[17]</sup> and Yee Guan Yap *et al*<sup>[18]</sup>. In reality, the tendency of correlation of the severity degree of liver dysfunction with prolonged QTc-interval in the present study revealed significance. The more severe liver

dysfunction is, the higher the tendency to trigger cardiac emergency due to such an prolonged QTc-interval. It may be a predictor of ventricular arrhythmia and a negative prognostic factor in patients with cirrhosis. The QTc interval prolongation in patients with cirrhosis may be an important marker for cirrhotic cardiomyopathy. This entity involves chronic cardiac dysfunction, including systolic and diastolic changes, and electrophysiological abnormalities. The cause of prolonged QTc in patients with advanced parenchymal liver disease remains controversial. Prolonged QTc interval in cirrhotic patients may be related to autonomic neuropathy which may reduce baroreflex activity and heart rate variability. In addition, chronotropic incompetence is a feature of autonomic neuropathy in these patients. Autonomic dysfunction may play an important role in cirrhotic cardiomyopathy.

#### 4.6 ECG Abnormalities & Coronary Risk Factors

In GROUP I, significantly 46 patients had abnormal ECG out of them 3 had no CAD risk factor, 15 had one CAD risk factor, 23 had two CAD risk factor, 4 had three CAD risk factor; & 1 had four CAD risk factor showing clearly that abnormality in ECG increases with increasing number of CAD RF among patients of Liver disease. The abnormalities in ECG in present study includes sinus tachycardia, sinus bradycardia, chamber enlargement (dilated LA, LV), ST-T changes, QTc prolongation, Arrhythmia's and Low voltage complexes. Shlomo stern (2009) concluded in his study & concluded that even early in the course of diabetes mellitus, ECG alterations such as sinus tachycardia, long QTc, QT dispersion, changes in heart rate variability, ST-T changes, and left ventricular hypertrophy may be observed<sup>[19]</sup>. Devi MR *et al* (2013) compared ECG changes in smokers with non-smokers. The heart rate was increased, The RR interval, the QT interval and the ST segment were shortened in the smokers as compared to those in the non-smokers, which was highly significant statistically<sup>[20]</sup>. Stavros Dimopoulos *et al* told that the electrocardiographic findings in patients with arterial hypertension can include LVH with strain pattern, ST-T changes, prolonged QTc & arrhythmias.<sup>[21]</sup>

#### 5. Conclusion

The present study is a modest attempt in assessment of cardiac function in chronic liver disease patients. The present study demonstrates that patients with chronic liver disease have cardiac dysfunction as evidenced by electrocardiography. The cardiac dysfunction is significantly higher in patients of alcoholic liver disease as compared to non-alcoholic liver disease patients thus demonstrating alcohol as a major causative & contributing factor in cardiac pathology in patients of chronic liver disease. These effects may be asymptomatic earlier in course but later may become symptomatic. Hence physician should be vigilant to identify these complications and treat them as soon as possible. Looking at these effects & keeping in mind their vital importance with possible contribution in the final outcome in the health of the patient these should be detected as soon as possible so as measures can be taken timely to prevent them & manage them properly. There is a strong correlation of cardiac status with severity of liver dysfunction with

increasing severity of liver dysfunction the cardiac dysfunction also increases steeply. The cardiac dysfunction is well known to occur in patients having coronary risk factors but the effects are intensified and added in presence of chronic liver disease and that too of alcohol induced owing to the effect of alcohol & liver disease per se. Thus, as electrocardiography acts as an important diagnostic tool in assessing the status of liver function in chronic liver disease patients, Electrocardiography should be employed routinely in these patients as it plays a significant role in detecting early cardiac changes in patients of chronic liver disease especially in those where it is of alcoholic in origin & in presence of additional coronary risk factors. It can also be used as a good predictor in explaining prognosis & to detect the cardiac dysfunction at earlier stage so that it can be treated ahead time.

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