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Echocardiographic changes in alcoholic Liver disease

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Abstract

Background- There are limited data regarding the prevalence of these types of heart disease in patients with ESLD, particularly in patients with alcoholic cirrhosis, but anecdotal observations suggest that CAD is a more common overt clinical problem than either alcoholic or cirrhotic cardiomyopathy. There is a need for prospective study of cardiac function in alcoholic and non-alcoholic patients.

Aims and objectives-- To study the 2D- Echocardiographic findings in alcoholic liver disease patients.

Material and methods- The present study was conducted in J A group of hospital, Gwalior, M.P. The present study is a case control study, conducted over a period between November 2012 to November 2013 on the patients of alcoholic & non-alcoholic liver disease patients. 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. Total of 102 patients were studied.

Results- ECHO abnormalities is higher significantly in Group I (ALD) patients (66.7%) as compared to Group II (NALD) patients (44.2%) hence showing the detrimental effect of alcohol over heart. Among Group I (ALD) patients chamber enlargement (dilated LV & LA); diastolic dysfunction; regional wall motion abnormalities; Valvular abnormalities (like mild MR, TR) is significantly higher than Group II (NALD) in the present study. Echocardiography strongly correlated with severity of liver disease as in study population (n=102) only 33% of Child A (n=12) patients had abnormal ECHO as compared to 59.6% of Child B (n=42) & 68.8% of Child C (n=48) patients which is statistically significant. In Group I (ALD), 2 of Child A patients had chamber enlargement i.e. enlarged LA & LV as compared to 6 of child B & 20 of Child C patients. Similarly, diastolic dysfunction is seen in 0 of Child A patients as compared to 6 of Child B (n=42) & 14 of Child C patients which are statistically significant difference thus reflecting the effect of severity of liver disease over heart. In the study population 61 patients had abnormal ECHO out of them 46 were of Group I (ALD). Out of these 46 significantly, 2 had no Coronary risk factors, 19 had one Coronary risk factor, 20 had two Coronary risk factors, 4 had three Coronary risk factors; & 1 had four Coronary risk factors. 18 patients had regional wall motion abnormalities in their Echocardiography, out of them 17 were of Group I (ALD) & significantly, 13 of these 17 patients had two or more Coronary risk factor's thus reflecting the strong association between them.

Conclusion- Echocardiography 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.

Keywords: alcohol, alcoholic liver disease, coronary artery disease, 2D- echocardiography

1. Introduction

Patients with alcohol induced liver cirrhosis and end-stage liver disease may have alcohol-related heart disease (alcoholic cardiomyopathy), heart disease associated with liver cirrhosis per se (cirrhotic cardiomyopathy), or coincidental heart disease (e.g., CAD).¹ There are limited data regarding the prevalence of these types of heart disease in patients with ESLD, particularly in patients with alcoholic cirrhosis, but anecdotal observations suggest that CAD is a more common overt clinical problem than either alcoholic or cirrhotic cardiomyopathy. There is a need for prospective study of cardiac function in alcoholic and non-alcoholic patients. 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.

Aims and objectives- To study the 2D- Echocardiographic findings in alcoholic liver disease patients.

Material & Methods

The present study was conducted in J A group of hospital, Gwalior, M.P. The present study is a case control study, conducted over a period between November 2012 to November 2013 on the patients of alcoholic & non-alcoholic liver disease patients. 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. **Total of 102 patients were studied.**

Inclusion criteria: **A- Alcohol intake history** of more than 40 g/day (men) for >10 yrs. **B-One & more of the following:**

Lab findings: Deranged Liver function test-AST (SGOT) >=84 (>3 times of ULN); ALT (SGPT) >=56; SAP >=166; Serum bilirubin >1.5 mg/dl; **Ultrasonographic evidence:** of liver disease (altered liver echotexture)

C- Histopathology: findings suggestive of liver disease.

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.

Exclusion criteria: 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.

Method and Data Collection-All the liver disease patients attending JAH Groups of Hospital, Gwalior, M.P. were screened for eligibility. Informed consent was taken from the eligible patients and enrolled in the present study. The patients were interviewed and underwent thorough physical examination.

History and Examination-A detailed history was elicited from all patients with emphasis on symptomatology and history of presenting & past illness; personal & family history; drug & addiction history is taken.

Patients were classified on disease severity on basis of CHILD PUGH TURCOTTE SCORE which is mentioned as below.

Child Pugh Truscott Score

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 are also enquired & examined for the presence of following coronary risk factors: POSITIVE FAMILY HISTORY OF CAD, SMOKING, HYPERTENSION (SBP>120 / DBP >80), DIABETES / IFG / IGT (FBS >126 / PPBS >140) (ADA), 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.

Investigations-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, ECG, Ultrasonography of abdomen, Lipid profile(total cholesterol, triglycerides, LDL, HDL, VLDL),Blood urea and serum creatinine and 2-Dimensional Echocardiography.

Technique of 2-dimensional Echocardiography

Two-dimensional, pulsed Doppler, M-mode and color flow Doppler echocardiographic studies were performed by an experienced cardiologist using a commercially available cardiac ultrasound machine on vivid 5GE Echocardiography machine employing TEICHOLZ method. Echocardiographic images were obtained from the parasternal and apical windows with the patient reclining on the left side, according to the recommendations of American Echocardiography Committee. Mitral inflow velocity pattern was recorded by placing the pulsed wave Doppler sample volume between the mitral valvular endings. Left ventricle outflow pattern was recorded from the apical five space window by placing the pulsed wave Doppler sample volume just under the aortic valve. With M-mode measurements, interventricular septum (IVS) and left ventricle posterior wall (LVPW) thicknesses separately at diastole and systole and left ventricle end-diastolic (LVED), end systolic (LVES) diameters, & left atrial dimension (LAD), Ejection fraction (EF), & fractional shortening were determined.

Statistical Methods-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 (+ Suggestive significance (P value: 0.05<P<0.10),

* Moderately significant (P value: 0.01<P _ 0.05),** Strongly significant (P value: P_0.01).

Statistical software-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.

Observations

Table 1: Distribution of 2-D Echo Findings In Study Population

	GROUP I (ALD) N=68	GROUP II (NALD) N=34
1.NORMAL ECHO	22(32.3%)	19 (55.8%)
2.ABNORMAL ECHO *P = 0.022	46(67.7%)	15(44.2%)

A total of 61 patients had abnormal ECHO out of 102.Significantly, among ALD patients 46(66.7%) had abnormal ECHO as compared to 15(44.2%) NALD patients.

Table 2: Pattern of Echo Abnormalities in Study Population

ECHO ABNORMALITIES	GROUP I (ALD) N=68	GROUP II (NALD) N=34
1. CHAMBER ENLARGEMENT (Dilated LV & LA) *P = 0.0008	28(41.1%)	3 (8.8%)
2. DIASTOLIC DYSFUNCTION *P = 0.006	20(29.4%)	2 (5.8%)
3. SYSTOLIC DYSFUNCTION	4(6%)	1 (2.9%)
4. RWMA *P = 0.003	17(25%)	1 (2.9%)
5. VALVULAR ABNORMALITIES (MR, TR etc.) *P = 0.019	20(29.4%)	3(5.8%)
6. PERICARDIAL EFFUSION	5(7.3%)	7(20.5%)
7. PULMONARY HYPERTENSION	4(5.8%)	1(2.9%)

Significantly, among ALD patient’s chamber enlargement (dilated LV & LA) is present in 28(41.1%) as compared to 3 (8.8%) NALD patients; diastolic dysfunction is present in 20(29.4%) ALD patients as compared to 2(5.8%) NALD

patients; RWMA in 17(25%) ALD patients as compared to 1 (2.9%) NALD patients; Valvular abnormalities (like mild MR, TR) in 20(29.4%) ALD patients as compared to 3(5.8%) NALD patients.

Table 3: Distribution of Lv Dimensions on 2-D Echo in Study Population

LV Dimension (Normal values)	Group I (ALD) N=68 MEAN	Group II (NALD) N=34 MEAN
Ejection Fraction (>55)	61.5%	60.9%
Fractional Shortening (35-45)	32.6%	34%
LVSTD (0.6-1.1)	0.94	0.88
LVIDD (3.6-5.6) *P = 0.021	4.95	4.8
LVIDS (2.4-4.0) *P = 0.01	3.7	3.23
LVPWD (0.6-1.1)	1.04	1.01
LAD (2.7-3.8) *P = 0.01	3.8	3.46

In the study population there was a significant difference noted in LVIDD, LVIDS & LAD. These dimensions were significantly larger in ALD patients (Mean value: LVIDD-5.2,

LVIDS-3.64, LAD-4.0) as compared to NALD patients (Mean value: LVIDD-4.9, LVIDS-3.25, LAD-3.4).

Table 4: Distribution of Echo Findings in Study Population as Per Severity Class

Echo 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 ECHO	5	8	9	22(32.3%)	4	9	6	19 (55.8%)
2. Abnormal ECHO *P = 0.021	3	20	23	46(67.7%)	0	5	10	15(44.2%)

In the study population only 3(33%) Child A patients had abnormal ECHO as compared to 25(59.6%) child B & 33

(68.8%) Child C patients which is statistically significant difference.

Table 5: Pattern of Echo Abnormalities in Study Population as Per Severity Class

Echo 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. Chamber Enlargement*P = 0.009	2	6	20	28 (41.1%)	0	1	2	3 (8.8%)
2. Diastolic Dysfunction *P = 0.025	0	6	14	20 (29.4%)	0	0	2	2 (5.8%)
3. Systolic Dysfunction	1	3	0	4 (6%)	0	0	1	1 (2.9%)
4. RWMA	0	7	10	17	0	0	1	1 (2.9%)

				(25%)				
5. Valvular Abnormalities (MR, TR etc.)	0	8	12	20 (29.4%)	0	1	2	3(5.8%)
6.Pericardial Effusion	0	2	3	5 (7.3%)	0	4	3	7(20.5%)
7.Pulmonary Hypertension	0	1	3	4 (5.8%)	0	0	1	1(2.9%)

In the GROUP I (ALD) only 2 of Child A patients had chamber enlargement i.e. enlarged LA & LV as compared to 6 of child B & 20 of Child C patients which is statistically significant difference. Similarly, in Group I (ALD) diastolic

dysfunction is seen in 0 of Child A patients as compared to 6 of child B & 14 of Child C patients which is also statistically significant difference.

Table 6: Distribution of Lv Dimensions On 2d-Echo in Study Population as Per Severity Class

Lv Dimension (Normal Values)	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)	Mean	Child Pugh A (N=4)	Child Pugh B (N=14)	Child Pugh C (N=16)	Mean
Ejection Fraction (>55)	63.8%	58.2%	62.6%	61.5%	63.2%	57.2%	62.3%	60.9%
Fractional Shortening (35-45)	34.7%	32.5%	30.5%	32.6%	34.7%	33.5%	33.8%	34%
Lvstd (0.6-1.1)	0.98	0.89	0.91	0.94	0.87	0.87	0.9	0.88
Lvidd (3.6-5.6) *P = 0.000	4.87	4.7	5.27	4.95	4.55	4.94	5.04	4.8
Lvids (2.4-4.0) *P = 0.041	3.22	3.42	3.8	3.7	3.0	3.28	3.44	3.23
Lvpwd (0.6-1.1)	1.02	1.07	1.03	1.04	1.12	0.89	0.91	1.01
Lad (2.7-3.8) *P = 0.005	3.45	4.1	4.3	3.8	3.3	3.4	3.7	3.46

In the GROUP I (ALD) there was a significant difference noted inLVIDd, LVIDs & LAD. These dimensions were significantly larger in Child C (Mean value: LVIDd-5.27, LVIDs-3.8, LAD-4.3) patients as compared to Child B (Mean

value: LVIDd-4.7, LVIDs-3.42, LAD-4.1) & Child A (Mean value: LVIDd-4.87, LVIDs-3.22, LAD-3.45). There were no significant difference in the EF, FS, LVISTd & LVPWd.

Table 7: Distribution of Echo Findings in Group I as Per Number of Cad Risk Factors

Echo 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 Echo	8 (80%)	11 (36.6%)	3 (13%)	0 (0%)	0 (0%)	22	32.3%
2.Abnormal Echo *P = 0.014	2 (20%)	19 (63.4%)	20 (87%)	4 (100%)	1 (100%)	46	67.7%

Significantly, in the Group I (ALD) 46 patients had abnormal ECHO out of them 2 had no CAD RISK FACTORS, 19 had one CAD RISK FACTORS, 20 had two CAD RISK

FACTORS, 4 had three CAD RISK FACTORS; & 1 had four CAD RISK FACTORS.

Table 8: Distribution of Echo Findings in Group II as Per Number of Cad Risk Factors

ECHO 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 ECHO	4 (100%)	9 (60%)	6 (46.1%)	0 (0%)	0 (0%)	19	55.8%
2.ABNORMAL ECHO	0 (0%)	6 (40%)	7 (53.9%)	2 (100%)	0 (0%)	15	44.2%

Significantly, in the study population 15 patients had abnormal ECHO out of them 6 had one CAD RISK

FACTORS, 7 had two CAD RISK FACTORS & 2 had three CAD RISK FACTORS.

Table 9: Pattern of Echo Abnormalities in Group I as Per Number of Cad Risk Factors

ECHO 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. CHAMBER ENLARGEMENT (DILATED LV, LA)	2 (20%)	14 (46.2%)	11 (47.8%)	1 (25%)	0 (0%)	28(41.1%)
2. DIASTOLIC DYSFUNCTION	2 (20%)	7 (23.1%)	6 (26%)	4 (100%)	1 (100%)	20(29.4%)
3. SYSTOLIC DYSFUNCTION	0 (0%)	3 (9.9%)	1 (4.3%)	0 (0%)	0 (0%)	4(6%)
4. RWMA *P = 0.006	0 (0%)	4 (13.2%)	9 (39%)	4 (100%)	0 (0%)	17(25%)
5. VALVULAR ABNORMALITIES (MR, TR etc.)	2 (20%)	8 (26.4%)	9 (39%)	1 (16.6%)	0 (0%)	20(29.4%)
6. PERICARDIAL EFFUSION	0 (0%)	1 (3.3%)	4 (17.2%)	0 (0%)	0 (0%)	5(7.3%)
7. PULMONARY HYPERTENSION	0 (0%)	1 (3.3%)	3 (12.9%)	0 (0%)	0 (0%)	4(5.8%)

Significantly, 17 patients had RWMA in their ECHO out of them 4 had one CAD RISK FACTOR, 9 had two CAD RISK FACTORS & 4 had three CAD RISK FACTORS.

Table 10-Pattern of Echo Abnormalities in Group II as Per Number of Cad Risk Factors

ECHO 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. CHAMBER ENLARGEMENT (DILATED LV, LA)	0 (0%)	0 (0%)	3 (23%)	0 (0%)	0 (0%)	3 (8.8%)
2. DIASTOLIC DYSFUNCTION	0 (0%)	1 (6.6%)	0 (0%)	1 (50%)	0 (0%)	2 (5.8%)
3. SYSTOLIC DYSFUNCTION	0 (0%)	1 (6.6%)	0 (0%)	0 (0%)	0 (0%)	1 (2.9%)
4. RWMA	0 (0%)	1 (6.6%)	0 (%)	0 (0%)	0 (0%)	1 (2.9%)
5. VALVULAR ABNORMALITIES (MR, TR etc.)	0 (0%)	1 (6.6%)	2 (15.3%)	0 (0%)	0 (0%)	3(5.8%)
6. PERICARDIAL EFFUSION	1 (25%)	2 (13.2%)	3 (23%)	1 (50%)	0 (0%)	7(20.5%)
7. PULMONARY HYPERTENSION	0 (0%)	0 (0%)	0 (0%)	1 (50%)	0 (0%)	1(2.9%)

Discussion

The present study consisted of 102 chronic liver disease patients without any evidence of prior cardiac dysfunction and were evaluated by two dimensional echocardiography. Group I comprising of 68 ALD patients & Group II comprising of 34 NALD patients served as cases & controls respectively in the present study. The findings are then correlated among ALD & NALD patients; on basis of severity of liver disease & presence of coronary risk factors. Majority of the patients (80%) were 30-60 years old. Among Group I (ALD) patients (n=68), 8(11.7%) belongs to Child pugh class A; 28 (41.3%) belongs to child class B; 32(47%) belongs to child class C. Similarly, among Group II (NALD) patients (n=34), 4(11.7%) belongs to child pugh class A; 14 (41.3%) belongs to child class B; 16(47%) belongs to child class C. ECHO ABNORMALITIES & ALCOHOLIC LIVER DISEASE-A total of 61 patients had abnormal ECHO out of 102. Significantly, among ALD patients 46(66.7%) had abnormal ECHO as compared to 15(44.2%) NALD patients showing clearly the ALD patients had higher abnormal ECHO's which is significant and thus demonstrating the

effect of Alcohol over heart. Among these 61 patients having abnormal ECHO's 15 had normal ECG clearly showing that electrocardiography being insensitive in picking abnormalities of cardiac dysfunction and a treating physician cannot solely rely on ECG while assessing cardiac status especially in these patients. Among ALD patient's significantly, chamber enlargement (dilated LV & LA) is present in 28(41.1%) as compared to 3 (8.8%) NALD patients; diastolic dysfunction is present in 20(29.4%) ALD patients as compared to 2(5.8%) NALD patients; RWMA in 17(25%) ALD patients as compared to 1 (2.9%) NALD patients; Valvular abnormalities (like mild MR, TR) in 20(29.4%) ALD patients as compared to 3(5.8%) NALD patients. There is significant difference in Chamber enlargement, diastolic dysfunction, RWMA & valvular abnormalities showing these ECHO abnormalities more common among ALD as compared to NALD patients thus reflecting again that detrimental effect of alcohol heart in form of Dilation of chambers, diastolic dysfunction, & increased risk of CAD if taken in substantial amount. The findings of the present study concurs with the prior studies.²⁻⁴ Out of 31 patients who had ECHO proven chamber enlargement only 10 had signs of chamber enlargement on

ECG again showing the insensitivity of electrocardiography in picking up the this abnormality. Again, out of these 31 patients who had ECHO proven chamber enlargement 18 patients doesn't have severe anemia hence chamber enlargement in these patients are attributed to the liver disease. Henning *et al.* studied cardiac performance in asymptomatic alcoholics and concluded that patients with alcoholic liver cirrhosis, although free of cardiac symptoms, may have a latent or preclinical cardiomyopathy that is manifest during physical stress.⁵ & also Henning and colleagues demonstrated the existence of latent cardiomyopathy in non-cirrhotic chronic alcoholics as most of ALD patients who showed ECHO abnormalities doesn't have any symptoms of cardiac dysfunction.⁶ These effects may be asymptomatic earlier in course but later may become symptomatic. Symptoms of overt heart failure is rare because of the peripheral vasodilatation characteristic of cirrhosis, in effect "autotreating" the ventricle by systemic vasodilatation reducing afterload, and compensatory diminution of inhibitory influences such as the cardiac muscarinic system. Although patients had complaints of dyspnoea, reduced exercise capacity, peripheral fluid retention (dependent edema) and ascites, these symptoms are common to both heart failure and advanced cirrhosis. It is therefore difficult to determine by symptoms if patients are indeed suffering from symptomatic heart failure. The only differentiating feature is that dyspnoea in cirrhosis is usually associated with hydrothorax from ascitic fluid tracking into the pleural cavity or tense ascites pushing up against the respiratory diaphragms, and not with pulmonary vascular congestion or frank pulmonary edema. The presence of such pulmonary congestion strongly points to a diagnosis of heart failure. Also, in the study population there was a significant difference noted in LVIDd, LVIDs & LAD. These dimensions were significantly larger in ALD patients (Mean value: LVIDd-5.2, LVIDs-3.64, LAD-4.0) as compared to NALD patients (Mean value: LVIDd-4.9, LVIDs-3.25, LAD-3.4). It appeared that Left ventricular dilation with preserved EF and impaired LV relaxation characterized LV function in chronic asymptomatic alcoholic patients. This concurs with the prior studies.^{7,8,9} So, there is a definite relationship of cardiomyopathy & liver disease in patients of chronic alcoholism as proved by Ramon & colleagues¹⁰. The finding of present study regarding the association between left atrial enlargement and alcohol consumption is in line with the results of two previous studies.^{11,12} 12 patients had pericardial effusion. 23 patients had Valvular abnormalities like (Mild MR & TR). The exact cause cannot be attributed to cardiac dysfunction as these patients had diastolic dysfunction along with ascitis and portal hypertension. Doppler echocardiography detected raised Pulmonary arterial pressures (PAP) in 5 patients (4 ALD & 1 non-ALD). This was similar to Yasemin Soyoral, Ali Süner *et al.*¹³ Mechanism of increased PAP is not fully understood, previous studies suggested that increased levels of vasoactive substances in pulmonary circulation and the probable toxic effects of these substances on endothelial cells. Torregrosa *et al.*¹⁴ showed & Basil. N. Saeed *et al.*¹⁵ showed the importance of Doppler echocardiography in detecting the presence of pulmonary hypertension in patients with chronic liver disease.

ECHO ABNORMALITIES & CHRONIC LIVER DISEASE- In the study population only 3(33%) Child A patients had abnormal ECHO as compared to 25(59.6%) Child B & 33 (68.8%) Child C patients which is statistically significant difference clearly showing higher incidence of ECHO abnormalities among patients having more severe liver

disease. In the GROUP I (ALD) only 2 of Child A patients had chamber enlargement i.e. enlarged LA & LV as compared to 6 of child B & 20 of Child C patients which is statistically significant difference clearly showing with increasing severity of disease the chamber size is also increasing. Similarly, diastolic dysfunction is seen in 0 of Child A patients as compared to 6 of child B & 14 of Child C patients in GROUP I (ALD) which is also statistically significant difference clearly showing with increasing severity of disease the LV diastolic dysfunction is also increasing. The present study clearly shows that cardiac changes parallel the severity of hepatic dysfunction in cirrhotics. This is concordant with Gaskari *et al.* who suggested that cardiac dysfunction parallels the severity of liver dysfunction. In the GROUP I (ALD) there was a significant difference noted in LVIDd, LVIDs & LAD as compared to that of GROUP II (NALD) with these dimensions being larger in GROUP I (ALD) patients. In GROUP I (ALD) these dimensions were significantly larger in Child C (Mean value: LVIDd-5.27, LVIDs-3.8, LAD-4.3) patients as compared to Child B (Mean value: LVIDd-4.7, LVIDs-3.42, LAD-4.1) & Child A (Mean value: LVIDd-4.87, LVIDs-3.22, LAD-3.45) patients suggesting clearly the effect of Alcohol & disease severity on the dilatation of the LV & LA. This concurs with the prior studies of Eldeeb *et al.*¹⁶ G. Finucci & colleagues¹⁷, Pozzi *et al.*¹⁸, Moller *et al.*¹⁹ There were no significant difference in the EF, FS, LVISTd & LVPWd. In study population left ventricular & left atrial enlargement in 30%, Left ventricular dysfunction were present in 26%, significant valvular regurgitation in 22.5%, and regional wall motion abnormalities in 18%, pericardial effusion in 11% & Pulmonary hypertension in 5%. Similar findings were observed by Donovan *et al.*²⁰. In 2005 Torregrosa *et al.* showed Ascitic patients exhibited more diastolic dysfunction at rest and during stress compared to non-ascitic patients. Liver transplantation caused regression of ventricular wall thickness improvement of diastolic function, and normalization of systolic response and exercise capacity during stress (significant increases in heart rate, ventricular ejection fraction, stroke volume and cardiac index) thus reflecting that these changes are reversible with liver transplantation²¹. This present study shows a strong relation between cardiomyopathy and severity of cirrhosis of liver more so in the later stages of cirrhosis of liver as told by earlier study of Samiullah *et al.*²². Therefore, highlighting the importance of recognizing the hemodynamic challenges encountered by chronic liver disease patients and how these response can be exacerbated by underlying cardiac pathology is critical in developing recommendations for the risk assessment and management of these patients as emphasized by Raval & *et al.*²³

ECHO ABNORMALITIES & CAD RISK FACTORS

In the Group I (ALD), significantly 46 patients had abnormal ECHO out of them 2 had no CAD RISK FACTORS, 19 had one CAD RISK FACTORS, 20 had two CAD RISK FACTORS, 4 had three CAD RISK FACTORS; & 1 had four CAD RISK FACTORS, showing clearly that abnormality in ECHO increases with increasing number of CAD risk factors. 18 patients had RWMA in their ECHO out of them 5 had one CAD risk factor, 9 had two CAD risk factors & 4 had three CAD risk factors which is significant, suggestive of clearly the RWMA the ECHO marker of CAD is more common in those having more number of CAD RF. This concurs with the prior studies.^{24, 25} In the present study, segmental wall motion abnormalities were associated with the presence of more CAD risk factors. These results are

consistent with previous observations from the SHS of an association of abnormal global LV function with diabetes mellitus.²⁶ The presence of regional wall motion abnormality on 2-D echocardiography in the presence of other cardiovascular risk factors necessitates further investigation and management to minimize later complications of coronary artery disease.

Conclusion-

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 ultrasonography acts as an important diagnostic tool in assessing the status of liver function in chronic liver disease patients, Echocardiography 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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References

- Liu H, Gaskari SA, Lee SS. Cardiac and vascular changes in cirrhosis: Pathogenic mechanisms. *World J Gastroenterol* 2006; 12:837-42.
- Timothh J. Regan, Gilmert E. Levinson, Henry A. Oldewurt, Martin J. Franx, Allen B. Weisse, And Christos B. Moscios- Ventricular Function in Noncardiacs with Alcoholic Fatty Liver: Role of Ethanol in the Production of Cardiomyopathy-The Journal of Clinical Investigation Volume 48 1969
- Lawrence Gould, Mahmood Shariff, Mohammad Zahir, And Mary Di Lieto -Cardiac Hemodynamics in Alcoholic Patients with Chronic Liver Disease and a Presystolic Gallop -The Journal of Clinical Investigation Volume 48 1969
- S. Sultan Ahmed, Maceo Howard, William ten Hove, Carroll M. Leevy, Timothy J. Regan-Cardiac function in alcoholics with cirrhosis: Absence of overt cardiomyopathy—myth or fact? *Journal of the American College of Cardiology* Volume 3, Issue 3, March 1984, Pages 696–702
- Henning Kelbaek, Jan Eriksen, Ingelise Brynjolf, Alan Raboel, Jens Otto Lund, Ole Munck, Olaf Bonnevie, John Godtfredsen -Cardiac performance in patients with asymptomatic alcoholic cirrhosis of the liver -The American Journal of Cardiology Volume 54, Issue 7, 1 October 1984, Pages 852–856
- Henning Kelbnk, Bjarne M Nielsen, Jan Eriksen, Alan Rab0l, Niels J Christensenjens Lund, Olaf Bonnevie, Ole Munck, John Godtfredsen- Left ventricular performance in alcoholic patients without chronic liver disease - *Br Heart J* 1987;58:352-7
- M. Dancy, G. Leech, J.M. Bland, M.K. Gaitonde, J.D. Maxwell_ preclinical left ventricular abnormalities in alcoholics are independent of nutritional status, cirrhosis, and cigarette smoking-The *lancet* Volume 325, Issue 8438, 18 May 1985, Pages 1122–1125
- L. L. CREGLER, T. M. WORNER, H. MARKM, - Echocardiographic Abnormalities in Chronic Asymptomatic Alcoholics-Clin. *Cardiol.* 12, 122-128 (1989)
- Aleksandar M Lazarević, Satoshi Nakatani, Aleksandar N Nešković, Jelena Marinković, Yoshio Yasumura, Djordjo Stojičić, Kunio Miyatake, Milovan Bojić, Aleksandar D Popović- Early changes in left ventricular function in chronic asymptomatic alcoholics: relation to the duration of heavy drinking -*Journal of the American College of Cardiology*_Volume 35, Issue 6, May 2000, Pages 1599–1606
- Ramon Estruch, Joaquim Fernández-Solá, Emilio Sacanella, Carles Paré, Emanuel Rubín, Alvaro Urbano-Márquez- Relationship between cardiomyopathy and liver disease in chronic alcoholism-Hepatology Volume 22, Issue 2, pages 532–538, August 1995
244. Djoussé L, Hunt SC, Eckfeldt JH, Arnett DK, Province MA, Ellison RC: Alcohol consumption and plasma atrial natriuretic peptide (from the HyperGEN study). *Am J Cardiol* 2006, 98:628–632.
- Breithardt G, Zeitter J, Moritz K, Bluschke V, Lehmann H, Borggrete M: The effect of alcohol on the heart in chronic alcoholics. *Z Kardiol* 1988, 77:642–648.
- Cheitlin, MD, Armstrong, WF, Aurigemma, GP, *et al.* - guideline for the clinical application of echocardiography ACC/AHA/ASE 2003
- Torregrosa, Mireia; Genesca, Joan; Gonzalez, Antonio; Evangelista, Arturo; Mora, Angels; Margarit, Carlos; Esteban, Rafael; Guardia, Jaime- Role of Doppler Echocardiography in the Assessment of Portopulmonary Hypertension in Liver Transplantation Candidates1-BRIEF COMMUNICATIONS: Clinical Transplantation 27 February 2001 - Volume 71 - Issue 4 - pp 572-574
- Basil. N. Saeed, Sameer Hakeam, Lutf Ahmed, Jalal Ahmed- The Value of Using Echocardiography in Patients of Advanced Liver Disease with Cardio Pulmonary Complications THE IRAQI POSTGRADUATE MEDICAL JOURNAL VOL.8, NO.4, 2009
- 249.Manal Eldeeb, Ragai M. F. R. Fouda, Mona M.R. Hammady and Laila Rashed-Echocardiographic Evaluation of Cardiac Structural and Functional Changes in Hepatitis C Positive Non-Alcoholic Liver Cirrhosis Patients and Their Plasma NT-ProBNP Levels-Life Science Journal, 2012;9(1)
- G. Finucci, A. Desideri, D. Sacerdoti, M. Bolognesi, C. Merkel, P. Angeli and A. Gatta-Left Ventricular Diastolic Function in Liver Cirrhosis -1996, Vol. 31, No. 3 , Pages 279-284
- M Pozzi, S Carugo, G Boari, V Pecci, S de Ceglia, S Maggiolini, G B Bolla, L Roffi, M Failla, G rassi, C Giannattasio, G Mancina -Evidence of functional and structural cardiac abnormalities in cirrhotic patients with and without ascites- *Hepatology* Volume 26, Issue 5, pages 1131–1137, November 1997
- S Møller, J H Henriksen_ Review Cirrhotic cardiomyopathy: a pathophysiological review of circulatory dysfunction in liver disease- *Heart* 2002;87:9-15
- Donovan, Carolyn L.; Marcovitz, Pamela A.; Punch, Jeffrey D.; Bach, David S.; Brown, Kimberly A.; Lucey,

- Mi- Two-Dimensional and Dobutamine Stress Echocardiography in the Preoperative Assessment of Patients With End-Stage Liver Disease Prior to Orthotopic Liver Transplantation- Clinical Transplantation 27 April 1996 - Volume 61 - Issue 8 - pp 1180-1188
21. Mireia Torregrosa, Santi Agudé, Laura Dos, Rosa Segura, Antonio González, Artur Evangelista, Joan Castell, Carlos Margarit, Rafael Esteban, Jaume Guardia, Joan Genescà-Cardiac alterations in cirrhosis: reversibility after liver transplantation-Journal of Hepatology Volume 42, Issue 1, January 2005, Pages 68–74
 22. Samiullah Shaikh, Mukhtiar Abro, Iftikhar Qazi, Akbar Yousfani- Frequency of cirrhotic cardiomyopathy in patients with cirrhosis of liver: A tertiary care hospital experience Pak J Med Sci 2011 Vol.27 No.4
 23. Zankhana Raval, Matthew E. Harinstein, Anton I. Skaro, Ata Erdogan, Andre M. DeWolf, , Sanjiv J. Shah, Oren K. Fix, Nina Kay, RN, Michael I. Abecassis, Mihai Gheorghide, and James D. Flaherty -Cardiovascular Risk Assessment of the Liver Transplant Candidate - J Am Coll Cardiol, 2011; 58:223-231
 24. Silvana Cicala, Thomas K. Welty, James M. Galloway, Barbara V. Howard and Richard B. Devereux Giovanni de Simone, Mary J. Roman, Lyle G. Best, Elisa T. Lee, Wenyu Wang-Prevalence and Prognostic Significance of Wall-Motion Abnormalities in Adults Without Clinically Recognized Cardiovascular Disease: The Strong Heart Study Circulation. 2007;116:143-150;
 25. Gardin JM, Siscovick D, Anton-Culver H, Lynch JC, Smith VE, Klopfenstein HS, Bommer WJ, Fried L, O'Leary D, Manolio TA. -Sex, age, and disease affect echocardiographic left ventricular mass and systolic function in the free-living elderly: the Cardiovascular Health Study. Circulation. 1995; 91:1739 –1748.