RESEARCH ARTICLE

Do you have to evaluate the heart of the cirrhotic patients?

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Abstract

Objective: To identify patients with occult cardiac dysfunction and its relationship with the severity of liver impairment.
Method: This is a Judgment (Purposive) Sampling, cross-sectional study that was conducted at Kafrelsheikh University Hospital, Egypt, from November 2019 to December 2020, and comprised adult patients of either gender with liver cirrhosis. After detailed history, a clinical examination, pathological assessment and cardiac evaluation based on electrocardiogram and echocardiography, the patients were divided into three groups. Patients who had dyspnoea or cyanosis were in group A, those who did not have dyspnoea or cyanosis but had electrocardiogram and echocardiography abnormalities were in group B, and patients who did not have dyspnoea, cyanosis or electrocardiogram and echocardiography abnormalities were in group C. The severity of the liver disease was evaluated using Child-Pugh and Model of End Liver Disease scores. Data was analysed using SPSS 20.
Results: Of the 300 patients, 153(51%) were males and 147(49%) were females. The overall mean age was 55.1±5.1 years (range: 20-60 years). There were 58(19.33%) patients in group A, 108(36%) in group B and 134(44.66%) in group C. Group A patients showed higher Child-Pugh and Model of End Liver Disease scores than the other groups (p<0.05). Child-Pugh score >6 and Model of End Liver Disease score >37 yielded the best accuracy for detecting cardiac abnormalities in group B (p<0.05).
Conclusion: There were significant cardiac changes in cirrhotic patients.
Keywords: Liver cirrhosis, Electrocardiography, Cirrhotic cardiomyopathy, Hyperdynamic circulation.
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Introduction

Cirrhosis of the liver is a longstanding disorder marked by organ fibrosis, lessened liver functionality and structural pathologies that lead to portal hypertension (PHT).[1] Cirrhotic patients exhibit hyperdynamic circulation, which includes higher cardiac output and stroke volume, higher organ blood flow, lower peripheral vascular resistance, and reduced systemic arterial pressure. Additionally, the proportions of circulating vasoactive intestinal peptide, glucagon, tumour necrosis factor (TNF), prostacyclin, nitric oxide, endothelin-1, and endothelin-3, are not down-regulated by the liver and are raised.[2] Owing to these haemodynamic characteristics, patients with compensated or decompensated liver cirrhosis potentially suffer from various repercussions involving deficient cardiac health which usually is not discerned.[3] Besides, insufficient heart function could contribute to more hepatic damage.[4] The tie between cardiac and hepatic malfunction can result in high fatalities, as evidenced by data which suggests that impaired heart function caused 25.8% of cirrhotic patients' deaths.[5] Cardiovascular disorders, in the majority of patients with chronic liver disease, emerge as subclinical conditions for a long period in cirrhosis. As with the progression of cirrhosis, there is a continuous worsening of cardiac function due to a failure in heart function and decreased cardiac output, resulting in the absence of the hyperdynamic circulation.[6] Unfortunately, decompensated cirrhosis manifestations may reflect those of heart failure, making a differential diagnosis difficult.[7] Heart failure manifestations may arise during stressful procedures, such as transjugular intrahepatic portosystemic collaterals (TIPS) and liver transplantations (LTs), both of which have poor outcomes.[8] Furthermore, recent cohort studies have revealed that cirrhotic individuals selected for LT had a significant frequency of silent coronary artery disease (CAD).[9-11] Additionally, the key factor in determining the post-LT prognoses is the existence of CAD, and cardiovascular problems are the main reason for non-graft-related death post-LT.[12] Consequently, while these patients are waiting for an LT, a therapy that focusses on their circulatory insufficiency may improve their short-term prognosis.[13-14] There is currently no conclusive evidence for or against regular

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cardiac checking for silent cirrhotic patients. Early screening may well be useful in identifying patients with occult cardiovascular dysfunction before clinical changes occur, as well as refining notions of preclinical disease, both of which have a major impact on liver disease and survival.

The current study was planned to investigate the prevalence of cardiac abnormalities in cirrhotic patients to identify patients with occult cardiac dysfunction and the relationship of the condition with the severity of liver impairment.

Patients and Methods

This is a Judgement (Purposive) Sampling, cross-sectional study that was conducted at Kafrelsheikh University Hospital, Egypt, from November 2019 to December 2020. After approval from the institutional ethics review committee, the sample size was calculated using EpiInfo calculator 3.01 with 95% confidence level, 80% power and expected outcome of cirrhotic cardiomyopathy 49%.[15-16] The sample size was inflated to cover up for dropouts.

The sample was raised from among those admitted or treated as outpatients in Hepatology, Gastroenterology and Infectious Disease departments. Those included were liver cirrhosis patients of either gender aged 20-65 years. The diagnoses were based on clinical findings, laboratory results, and imaging studies. Patients with known cardiac disease, hypertension (HTN), diabetes mellitus (DM), thyroid disease, obesity, severe anaemia with haemoglobin (Hb) <8gm/dl, bleeding, hepatocellular carcinoma (HCC), pulmonary disease, other malignancies, pregnancy, history of recent intake of drugs that could affect heart or liver function were excluded.

After taking informed consent from all the participants, they were subjected to detailed medical history, clinical assessment, and regular laboratory tests, including complete blood count (CBC), liver function test (LFT), serum creatinine, international normalisation ratio (INR), and hepatitis B surface antigen (HBsAg) and anti-hepatitis C virus antibodies (HBsAg-HCV Abs). All subjects underwent ultrasound (U/S) of the abdomen to assess liver condition, spleen diameter, portal vein diameter and portosystemic collaterals; arterial blood gases (ABG) to assess oxygen (O2) saturation. The severity of the liver disease was evaluated using Child-Pugh and Model of End Liver Disease (MELD) scores.[17-18]

Cardiac changes were determined using electrocardiogram (ECG) to assess P wave, QT interval, QRS, or any other changes, and two-dimensional (2D) echocardiography (ECHO) (Philips EPIC7) to assess ejection fraction (EF), pulmonary artery (PA) diameter, PA pressure, right atrium (RA) or any other change.

Based upon the symptoms, signs, ECG and ECHO, the patients were divided into three groups. Patients who had dyspnoea or cyanosis were in group A, those who did not have dyspnoea or cyanosis but had ECG and ECHO abnormalities were in group B, and patients who did not have dyspnoea, cyanosis or ECG and ECHO abnormalities were in group C.

Data was analysed using SPSS 20. Data normality was confirmed using Kolmogorov-Smirnov test, and Chi-square test was used to compare groups for categorical variables (Monte Carlo [MC]). For quantitative variables, student t-test and Mann Whitney test were used, as appropriate. The groups were compared using analysis of variance (ANOVA) or Kruskal-Wallis test, and pairwise comparisons of quantitative variables were compared using the Tukey or Dunn's multiple comparison test as the Post-Hoc test. $P<0.05$ was considered statistically significant.

Results

Of the 300 patients, 153(51%) were males and 147(49%) were females. The overall mean age was 55.1±5.1 years (range: 20-60 years). Most patients 278(92.7%) had HCV-related cirrhosis, while 15(5%) had HBV-related disease. Those who presented with dyspnoea were 68(22.7%) and those who had cyanosis were 39(13%) (Table 1). Regarding cardiac findings, QT prolongation was the most common abnormal finding 120(40%) (Table 2).

There were 58(19.33%) patients in group A, 108(36%) in group B and 134(44.66%) in group C, and laboratory findings showed significant inter-group differences ($p\leq0.05$) (Table 3).

Also, there were significant inter-group differences in terms of ECG and ECHO findings (Table 4).

Group A patients showed higher Child-Pugh and MELD scores than the other groups ($p<0.05$), while MELD scores were not significantly different between groups B and C ($p>0.05$).

There was a significant relation of Child-Pugh and MELD scores with ECG and ECHO abnormal findings (Table 5).

Child-Pugh score >6 and MELD score >37 yielded the best accuracy for detecting cardiac abnormalities in group B (Figure).

Table-2: Demographic and laboratory parameters of the subjects (n=300).

	n (%)
Age	
Mean ± SD.	55.1 ± 5.1
Median (Min. – Max.)	56 (36 – 63)
Gender	
Male	153(51%)
Female	147(49%)
Aetiology	
HCV	260(86.7%)
Bilharizial+ HCV	18(6%)
HBV	15(5%)
Autoimmune	4(1.3%)
Haemochromatosis	3(1%)
Dyspnoea	
Absent	143(47.7%)
1	89(29.7%)
2	43(14.3%)
3	22(7.3%)
4	3(1%)
Cyanosis	39(13%)
Manifest	56(18.7%)
Occult	
non-occult non-manifested	108(36%)
136(45.3%)	
Hb	
Mean ± SD.	11 ± 1.9
Median (Min. – Max.)	11 (8 – 17.7)
WBCs	
Mean ± SD.	6.4 ± 3.5
Median (Min. – Max.)	5.6 (1.1 – 24)
PLT	
Mean ± SD.	119.9 ± 57.5
Median (Min. – Max.)	110 (20 – 397)
ALT	
Mean ± SD.	35.8 ± 19.8
Median (Min. – Max.)	30(13 – 124)
AST	
Mean ± SD.	46.3 ± 22.7
Median (Min. – Max.)	42 (15 – 117)
BIL	
Mean ± SD.	2.8 ± 3.3
Median (Min. – Max.)	1.8 (0.3 – 23)
ALB	
Mean ± SD.	3.5 ± 0.9
Median (Min. – Max.)	3.4 (1 – 5)
Creat	
Mean ± SD.	1 ± 0.5
Median (Min. – Max.)	0.8 (0.4 – 6.2)
INR	
Mean ± SD.	1.5 ± 0.4
Median (Min. – Max.)	1.5 (1 – 3.4)

SD: Standard deviation, HCV: Hepatitis C virus, HBV: Hepatitis B virus, Hb: Haemoglobin, WBC: White blood cell counts, PLT: Platelets, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, Creat: Creatinine, ALB: Albumin, BIL: Bilirubin, INR: International normalisation ratio.

Table-2: Ultrasound (U/S), electrocardiogram (ECG) and echocardiography (ECHO) parameters (n=300).

		n (%)
	Total number of Patients	300
	liver cirrhosis	
	liver cirrhosis +Periportal fibrosis	
	Spleen (n= 269)	
	Mean ± SD.	16 ± 2.5
	Median (Min. – Max.)	16 (11 – 22)
U/S	**PV diameter (n= 290)**	
	Mean ± SD.	13.3 ± 1.9
	Median (Min. – Max.)	14 (10 – 18)
	Collaterals	90(30%)
	QT interval	
	Normal	180(60%)
	Prolonged	120(40%)
	P wave	
	Absent	264(88%)
	Pulmonale	34(11.3%)
	Mitrale	2(0.7%)
ECG	**QRS**	
	Normal	298(99.3%)
	Prolonged	2(0.7%)
	Others	
	Normal	290(96.7%)
	1st HB	2(0.7%)
	AF	8(2.7%)
	EF	
	Mean ± SD.	63.9 ± 35.9
	Median (Min. – Max.)	64 (40 – 666)
ECHO	**RA**	68(22.7%)
	PP	65(21.7%)
	PA	65(21.7%)
	Other	26(8.7%)
O2 Saturation		
Mean ± SD.	92.9 ± 3.1	
Median (Min. – Max.)	93.0 (75.0 – 99.0)	
Child score		
Mean ± SD.	8.1 ± 3.3	
Median (Min. – Max.)	6.5 (5.0 – 14.0)	
MELD Score		
Mean ± SD.	43.7 ± 18.3	
Median (Min. – Max.)	37.8 (25.8 – 132.5)	

SD: Standard deviation, PV: portal vein, HB: Heart Block, AF: Atrial fibrillation, EF: Ejection fraction, RA: Right atrium, PP: Pulmonary pressure, PA: Pulmonary artery, O2: Oxygen, MELD: Model of End Liver Disease.

Table-3: Intergroup comparison of demographic and laboratory parameters.

	Normal ranges of labs	Manifest (n = 58)	Occult (n = 108)	Non-occult Non-Manifest (n = 134)	Test of sig.	*p-value*
Age (years)						
Mean ± SD.		54.4 ± 4.6	55.2 ± 4.7	55.3 ± 5.6	F= 0.763	0.467
Median (Min. – Max.)		55 (41 – 63)	55 (40 – 62)	57 (36 – 63)		
Gender						
Male		38 (65.5%)	67 (62%)	48 (35.8%)	χ2= 22.511*	<0.001*
Female		20 (34.5%)	41 (38%)	86 (64.2%)		
Sig. bet. Grps				p1=0.657,p2<0.001*,p3<0.001*		
Hb (g/l)	**(12-14)**					
Mean ± SD.		9.6 ± 1.4	10.6 ± 1.7	11.9 ± 1.7	F= 42.225*	<0.001*
Median (Min. – Max.)		9.3 (8 – 15)	10 (8 – 16)	12 (8 – 17.7)		
Sig. bet. Grps				p1=0.002*,p2<0.001*,p3<0.001*		
WBCs (× 103/L)	**(4-11)**					
Mean ± SD.		4.7 ± 2.6	6.7 ± 4.6	6.9 ± 2.4	H= 39.044*	<0.001*
Median (Min. – Max.)		4 (1.1 – 15.7)	5 (2.3 – 24)	6.9 (2.4 – 15)		
Sig. bet. Grps				p1=0.001*,p2<0.001*,p3=0.001*		
PLT(× 103/L)	**(150-450)**					
Mean ± SD.		91.4 ± 49.7	104.2 ± 50.1	144.9 ± 56.3	H= 59.799*	<0.001*
Median (Min. – Max.)		83.5 (20 – 274)	94 (39 – 397)	145 (29 – 312)		
Sig. bet. Grps				p1=0.085,p2<0.001*,p3<0.001*		
ALT(U/L)	**(Up to 4o)**					
Mean ± SD.		29.2 ± 22	31.3 ± 16.4	42.3 ± 19.4	H= 39.636	<0.001*
Median (Min. – Max.)		21 (14 – 124)	25 (14 – 87)	39 (13 – 96)		
Sig. bet. Grps				p1=0.119,p2<0.001*,p3<0.001*		
AST (U/L)	**(Up to 4o)**					
Mean ± SD.		38.6 ± 16.4	50.9 ± 27.5	45.8 ± 19.7	H= 6.888*	0.032*
Median (Min. – Max.)		36 (17 – 84)	50 (15 – 117)	42 (16 – 102)		
Sig. bet. Grps				p1=0.010*,p2=0.033*,p3=0.524		
Serum BIL (mg/L)	**(0.3- 1)**					
Mean ± SD.		4.6 ± 3.8	4 ± 4	1 ± 0.8	H=138.566*	<0.001*
Median (Min. – Max.)		3.3 (1.6 – 18)	3 (0.5 – 23)	0.8 (0.3 – 4)		
Sig. bet. Grps				p1=0.064,p2<0.001*,p3<0.001*		
Serum ALB (g/dl)	**(3.5-5)**					
Mean ± SD.		2.8 ± 0.4	3.1 ± 0.6	4.1 ± 0.7	F=117.080*	<0.001*
Median (Min. – Max.)		2.8 (2.2 – 3.7)	3 (2.2 – 4.4)	4.2 (1 – 5)		
Sig. bet. Grps				p1=0.020*,p2<0.001*,p3<0.001*		
Creat (mg/dL)	**(M:0.7-1.3) (F:0.6-1.1)**					
Mean ± SD.		1.2 ± 0.9	1.1 ± 0.4	0.8 ± 0.1	H=72.070*	<0.001*
Median (Min. – Max.)		0.9 (0.6 – 6.2)	0.9 (0.6 – 2.2)	0.8 (0.4 – 1.2)		
Sig. bet. Grps				p1=0.438,p2<0.001*,p3<0.001*		
INR	**(0.8 to 1.1)**					
Mean ± SD.		1.8 ± 0.4	1.7 ± 0.4	1.3 ± 0.2	F=113.887*	<0.001*
Median (Min. – Max.)		1.7 (1.3 – 3.4)	1.6 (1 – 3.4)	1.3 (1 – 2.7)		
Sig. bet. Grps				p1=0.019*,p2<0.001*,p3<0.001*		

SD: Standard deviation, HCV: Hepatitis C virus, HBV: Hepatitis B virus, Hb: Haemoglobin, WBC: White blood cell counts, PLT: Platelets, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, Creat: Creatinine, ALB: Albumin, BIL: Bilirubin, INR: International normalisation ratio. Sig. bet. Grps : Significance between Groups, $\chi 2$: Chi square test

F: F for analysis of variance (ANOVA) test, Pairwise comparison bet. each 2 groups were done using Post Hoc Test (Tukey)

H: H for Kruskal Wallis test, Pairwise comparison bet. each 2 groups were done using Post Hoc Test (Dunn's for multiple comparisons test)

p: p value for comparing the studied groups

p1: p value for comparing the manifest and occult

p2: p value for comparing the manifest and normalv

p3: p value for comparing the occult and normal

*: Statistically significant at $p \leq 0.05$

Table-4: Intergroup comparison regarding different parameters.

	Manifest (n = 58)	Occult (n = 108)	Normal (n = 134)	Test of sig.	*p-value*
QT interval					
Normal	20 (34.5%)	26 (24.1%)	134 (100%)	χ^2 =163.149*	<0.001*
Prolonged	38 (65.5%)	82 (75.9%)	0 (0%)		
Sig. bet. Grps		p1=0.153,p2<0.001*,p3<0.001*			
P wave					
Absent	47 (81%)	85 (78.7%)	132 (98.5%)	χ2= 41.547*	^{MC}p <0.001*
Pulmonale	11 (19%)	23 (21.3%)	0 (0%)		
Mitrale	0 (0%)	0 (0%)	2 (1.5%)		
Sig. bet. Grps		p1=0.723,MCp2<0.001*,MCp3<0.001*			
QRS					
Normal	56 (96.6%)	108 (100%)	134 (100%)	χ2=5.024*	^{MC}p=0.035*
Prolonged	2 (3.4%)	0 (0%)	0 (0%)		
Sig. bet. Grps		FEp1=0.121, FEp2=0.090,p3=–			
Others					
Normal	55 (94.8%)	101 (93.5%)	134 (100%)	χ2= 10.424*	^{MC}p=0.006*
1st HB	0 (0%)	2 (1.9%)	0 (0%)		
AF	3 (5.2%)	5 (4.6%)	0 (0%)		
Sig. bet. Grps		MCp1=0.869,FEp2=0.008*,MCp3=0.002*			
EF					
Mean ± SD.	57.4 ± 9.8	58.7 ± 9	70.9 ± 52	H= 66.372*	<0.001*
Median (Min. – Max.)	59.5(40 – 90	60(40 – 75)	66(54 – 666)		
Sig. bet. Grps		p1=0.324,p2<0.001*,p3<0.001*			
RA					
Normal	39 (67.2%)	62 (57.4%)	131 (97.8%)	χ2=59.733*	<0.001*
Dilated	19 (32.8%)	46 (42.6%)	3 (2.2%)		
Sig. bet. Grps		p1=0.216,p2<0.001*,p3<0.001*			
PP					
Normal	41 (70.7%)	60 (55.6%)	134 (100%)	χ2=72.075*	<0.001*
Increased	17 (29.3%)	48 (44.4%)	0 (0%)		
Sig. bet. Grps		p1=0.057,p2<0.001*,p3<0.001*			
PA					
Normal	41 (70.7%)	60 (55.6%)	134 (100%)	χ2=72.075*	<0.001*
Increased	17 (29.3%)	48 (44.4%)	0 (0%)		
Sig. bet. Grps		p1=0.057,p2<0.001*,p3<0.001*			
Other					
Normal	44 (75.9%)	96 (88.9%)	134 (1%)	χ2=31.069*	<0.001*
Abnormal	14 (24.1%)	12 (11.1%)	0 (0%)		
Sig. bet. Grps		p1=0.028*,FEp2<0.001*,p3<0.001*			
O2 Saturation					
Mean ± SD.	89.6 ± 3.4	92.7 ± 2.8	94.5 ± 1.7	F=108.779*	<0.001*
Median (Min. – Max.)	90 (75 – 94)	93 (84 – 99)	95 (90 – 97)		
Sig. bet. Grps		p1<0.001*,p2<0.001*,p3<0.001*			
Child score					
A	3 (5.2%)	27 (25%)	120 (89.6%)	F=160.560*	<0.001*
B	6 (10.3%)	15 (13.9%)	4 (3%)		
C	49 (84.5%)	66 (61.1%)	10 (7.5%)		
Sig. bet. Grps		p1=0.003*,p2<0.001*,p3<0.001*			
Mean ± SD.	10.8 ± 1.9	9.6 ± 3.1	5.7 ± 1.9	F=40.182	<0.001*
Median (Min. – Max.)	11 (6 – 14)	10 (5 – 14)	5 (5 – 14)		
Sig. bet. Grps		p1=0.019*,p2<0.001*,p3<0.001*			
MELD score					
Mean ± SD.	55.5 ± 19.8	51.1 ± 20.3	32.7 ± 5	F=150.501*	<0.001*
Median (Min. – Max.)	49.9(33.7 – 129.8)	46.1(29.1 – 132.5)	31.5(25.8 – 51)		
Sig. bet. Grps		p1=0.062,p2<0.001*,p3<0.001*			

SD: Standard deviation, HB: Heart block, AF: Atrial fibrillation, EF: Ejection fraction, RA: Right atrium, PP: Pulmonary pressure, PA: Pulmonary artery, MELD: Model of End Liver Disease, Sig. bet. Grps: Significance between Groups, χ2: Chi square test, MC: Monte Carlo H: H for Kruskal Wallis test, pairwise comparison bet. each 2 groups were done using Post Hoc Test (Dunn's for multiple comparisons test)
p: *p-value* for comparing between the studied groups p1: *p-value* for comparing between manifest and occult
p2: *p-value* for comparing between manifest and normal p3: *p-value* for comparing between occult and normal
*: Statistically significant at p ≤ 0.05

Table-5: Relation between Child-Pugh score, MELD score and cardiac assessment parameters (n = 300).

QT interval	n	Mean ± SD	Mean ± SD.Age
Normal	180	6.5 ± 2.6	35.8 ± 9.9
Prolonged	120	10.4 ± 2.7	55.6 ± 21.2
U(p)			
P wave			
Absent	264	7.9 ± 3.2	42.4 ± 16.3
Pulmonale	34	9.2 ± 3.3	54.7 ± 27.4
Mitral	2	5 ± 0	33.6 ± 0
H(p)			
QRS			
Normal	298	8.1 ± 3.3	43.4 ± 17.9
Prolonged	2	11 ± 0	88.7 ± 0
U(p)			
Others			
Normal	290	8 ± 3.3	43.2 ± 17.9
1st HB	2	9 ± 1.4	42.7 ± 5.2
AF	8	10.8 ± 2.4	63.4 ± 22.1
H(p)			
RA			
Normal	232	7.7 ± 3.2	41.4 ± 15.9
Dilated	68	9.3 ± 3.2	51.7 ± 23
U(p)			
PP			
Normal	235	7.8 ± 3.2	41.8 ± 16
Increased	65	9 ± 3.3	50.5 ± 23.6
U(p)			
PA			
Normal	235	7.8 ± 3.2	41.8 ± 16
Increased	65	9 ± 3.3	50.5 ± 23.6
U(p)			
Other			
Normal	274	7.9 ± 3.2	41.8 ± 15.4
Abnormal	26	10.3 ± 3	63.5 ± 31
U(p)			

U: Mann Whitney test H: H for **Kruskal Wallis test**
p: p value for comparing between different category
***: Statistically significant at p ≤ 0.05**
SD: Standard deviation, HB: **Heart Block,** AF: **Atrial fibrillation, ECHO:** Echocardiography, **EF:** Ejection fraction, **RA:** Right atrium, **PP:** Pulmonary pressure, **PA: Pulmonary artery, MELD:** Model of End Liver Disease.

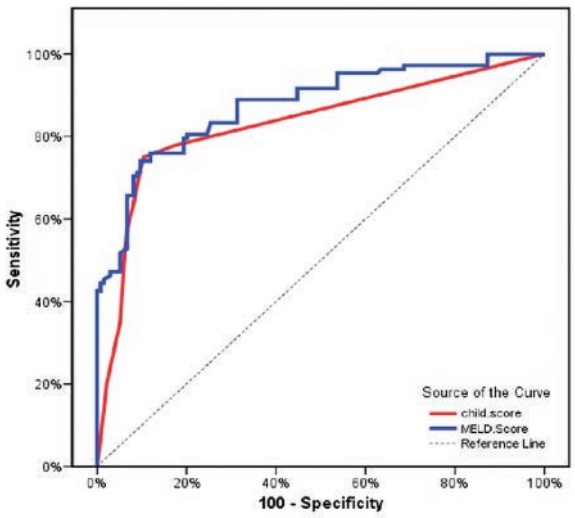

Figure 2: ROC curve for Child score and MELD score to discriminate Occult patients (n=108) from normal (n=134).

Discussion

The study found that the most common cause of liver cirrhosis, either compensated or decompensated, was HCV (92.7%), which was in agreement with Mokdad AA et al.[19] Occult cardiac changes occur in about 36% of cirrhotic patients, magnifying the need for cardiac evaluation of every cirrhotic patient because cardiomyopathy was found in 33.8% of cirrhotic patients in a study.[20]

Prolonged QT was the most common finding in the current study, which has been reported earlier as well, suggesting that this prolongation may be due to alterations of ion channel activity in cardiomyocyte plasma membranes.[21] Different ECHO changes were noticed in decompensated cirrhosis who did not have manifestations. This was also reported earlier.[22] Porto-pulmonary hypertension in patients was asymptomatic in the current study. This was in line with an earlier study.[23] The current study found a positive correlation between the severity of liver cirrhosis and cardiac changes. The result agrees with earlier findings, but also is in contrast with some other findings.[24-25] The disagreement could be due to the difference in sample sizes, and also because there is no universally accepted definition of cirrhotic cardiomyopathy. Also, there are many

Table-6: Validity (AUC, sensitivity, specificity) for Child score and MELD score to discriminate Occult patients (n=108) from normal (n=134)

	AUC	P	95% C.I	Cut off	Sensitivity	Specificity	PPV	NPV
Child score	0.830	<0.001*	0.774-0.886	>6	75.0	89.55	85.3	81.6
MELD score	0.876	<0.001*	0.831-0.921	<37	75.93	88.06	83.7	81.9

AUC: Area Under a Curve P value: Probability value
CI:Confidence Intervals PPV: Positive Predictive Value
NPV: Negative Predictive Value
*: significally significant at p≤0.05
#Cut off was choose according to Youden index

different instruments to assess the condition, and studies adopt different inclusion criteria.

Conclusion

There were significant cardiac changes in cirrhotic patients. Occult changes may occur early and need close follow-up and management. Cardiac evaluation may be needed for every cirrhotic patient.

Disclaimer: This manuscript is a part of the third co-author's M.Sc. thesis in Hepatology, Gastroenterology and Infectious diseases. The abstract has not been previously presented or published in a conference.

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