International Journal of Advances in Medicine
Ranjith KGK et al. Int J Adv Med. 2021 Jul;8(7):940-946
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20212405

Original Research Article

Study of cardiac involvement in liver cirrhosis patients

Ranjith Kumar G. K.[1]*, Manjunath F. V.[1], Parameshwar[2], Nagabhushana S.[1]

[1]Department of General Medicine, [2]Department of Cardiology, Shimoga Institute of Medical Sciences (SIMS), Shimoga, Karnataka, India

Received: 28 May 2021
Revised: 20 June 2021
Accepted: 21 June 2021

***Correspondence:**
Dr. Ranjith Kumar G. K.,
E-mail: ranjithkumargk@gmail.com

ABSTRACT

Background: Cirrhosis liver is characterized by diffuse destruction and regeneration of hepatic parenchymal cells. Various studies have been carried out over the years to evaluate the cardiac and hemodynamic changes in cirrhosis of the liver.
Methods: Study is conducted on 50 consecutive patients with cirrhosis liver admitted in various medical wards of the Mc Gann Hospital attached to Shimoga institute of medical sciences, Shimoga from January, 2020 to September 2020. Detailed history was taken and a through physical examination was done. Blood investigations are done. Echocardiography, Chest radiography done. All patients were subjected to USG abdomen to confirm the diagnosis of cirrhosis.
Results: There were 35 males and 15 females. Average age is 54±6.6 years. Alcoholism (64%) and idiopathic (16%) are commonest associations. Commonest symptom is anaemia (88%) followed by pedal oedema (80%) followed by fatigue (66%), ascites (60%) and jaundice (60%). Pleural effusion seen in 38%, diastolic dysfunction 32%, pericardial effusion 16%, wall motion abnormality in 11%. Hypertension, arrhythmias and LVF is seen in 8%. Cardiomyopathy in 6%. ECG changes are Hemiblock in 4 (8%), QTc prolongation in 34%. LVH in 12%, Low voltage complex are seen in 24%. cardiomegaly seen in 20%.
Conclusions: Study shows that, there was significant incidence of subclinical cardiac abnormalities observed in chronic liver disease patients which increase with duration of illness. Early detection of subclinical cardiac changes is important to reduce morbidity and mortality in chronic liver disease patients.

Keywords: Cirrhosis of liver, Echocardiography, Pleural effusion, Arrhythmias, Anemia

INTRODUCTION

Cirrhosis liver is characterized by diffuse destruction and regeneration of hepatic parenchymal cells leading to deposition of connective tissue with resulting disorganization of the lobular and vascular architecture. Eventhough liver is able to regenerate, once regenerative capacity is exceeded, clinically overt or decompensated liver disease (cirrhosis of liver) enshues. Development of portal hypertension due to obstuction to blood flow through the liver parenchyma results in increased portal venous pressure, which in turn leads to diversion of blood flow to portosystemic collaterals, which is a low resistance channel.

The current study is to evaluate the cardiovascular system in a group of patients with hepatic cirrhosis based on clinical examination.

Cirrhosis is a irreversible chronic injury to the liver characterized by abundant fibrosis along with regenerative nodules leading to disorganized lobular and vascular

architecture of liver, this leads to portal hypertension and liver cell failure. Hyperdynamic circulation more indirectly indicates the presence of portal hypertension and liver cell failure. Many studies had been carried out previously to know the cardiac and hemodynamic changes in cirrhosis of the liver.[1] Portal hypertension develops when there is obstruction to blood flow through the liver this leads to increase in portal venous pressure leading to diversion of blood flow to low pressure portosystemic collaterals thereby bypassing the hepatic circulation. As there is diversion of blood flow from portal circulation to systemic circulation, there will be increased blood flow in systemic circulation which leads to hyperdynamic systemic circulation.

Chronic liver diseases (cirrhosis) produce high cardiac output states.[2] The exact mechanism was not clear but had been attributed to increased blood volume, intrahepatic AV shunts, mesenteric AV shunts and defective inactivation of circulating vasodilators. 2D echocardiography is a useful noninvasive method of studying the various morphological and functional parameter of Heart.[3] In patients with cirrhosis liver, earlier studies have shown that RV end diastolic volume and RV end systolic volume were significantly reduced, whereas LV end diastolic volume and LV end systolic volume and LA volume were normal or slightly increased.[4]

The right ejection fraction (EF) was significantly increased and the left EF was slightly decreased. Stroke volume was significantly higher. There is also evidence of cardiac muscle contractile function impaired and ventricular muscle hyporesponsiveness to pharmacological or physiological stress.[5] Left ventricular Diastolic dysfunction (DD) was found to be thirty five percent in prior studies and more common in alcoholic than in non-alcoholics. These changes are reversed following hepatic transplantation. Pericardial effusion has been demonstrated in many patients with cirrhosis and sometimes it is massive.[6]

Objectives of the study

To clinically evaluate patients with hepatic cirrhosis with respect to cardiac examination and to document the electrical and morphological alterations in the heart in patients with cirrhosis by means of noninvasive investigations like electrocardiography, X-ray and 2-dimensional echocardiography.

METHODS

Source of data

The study will be conducted for period of one year from January 2020 to September 2020. All documented cirrhotic patients admitted in Mcgann teaching district hospital attached to shimoga institute of medical sciences are taken into the study.

Inclusion criteria

Patients with age more than 18 years will be taken for study. Only patients with clinical, biochemical and sonographic evidence of cirrhosis liver are selected.

Exclusion criteria

Patients with previously detected heart disease, Patients with intercurrent illness, Patient with cardiac cirrhosis.

Method of collection of data

A detailed history was elicited from the patient with special reference to cardiovascular symptoms. A thorough physical examination was performed in the patients and a special note was made regarding heart rate and rhythm, blood pressure, jugular venous pulse, and pressure and precordial examination. All patients were subjected to routine investigations viz, Blood urea, sugar, complete hemogram, serum cholesterol and liver function tests. electocardiography, HIV serology, hepatitis B surface antigen (HBsAg), anti-hepatitis C antibody (Anti HCV), urine for routine examination done. All patients were subjected to ultrasound scan abdomen to confirm the diagnosis of cirrhosis. Patients with ascites underwent abdominal paracentesis and fluid was analyzed for protein content and cells. All patients were then subjected to electrocardiography, chest X-ray,and M-mode 2-Dimensional echocardiography. Ethical clearance is taken from institutional ethical board before starting the study

Statistical analysis

Significance was evaluated Chi-square test and $p<0.05$ was considered as significant. The statistical software namely Statistical package for social sciences (SPSS) 11.0 were used for the analysis of the data.

RESULTS

Demographic data

50 consecutive patients with cirrhosis liver were studied from January 1st, 2020 to September 30th2020. There were 35 males and 15 females.

The age distribution ranged from 15 years to 80 years with maximum number of patients in the age group 51 to 60 years (38%). Average age is 54±6.6 years.

The mean age among men was 54.22±7.34 years and in women was 50.45± 6.56 years. Majority of the patients were in the age group of 40-60 years (Figure 2).

Alcoholism (64%) and idiopathic (16%) are commonest associations (Table 1).

Commonest symptom is anaemia (88%) followed by pedal oedema (80%) followed by fatigue (66%) and ascites, jaundice (60%). (Table 2)

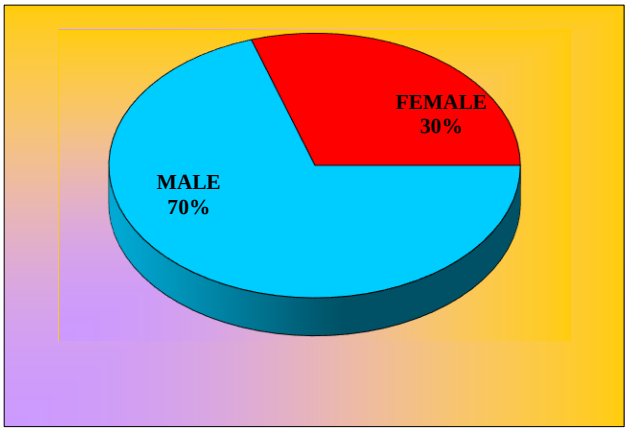

Figure 1: Sex distribution.

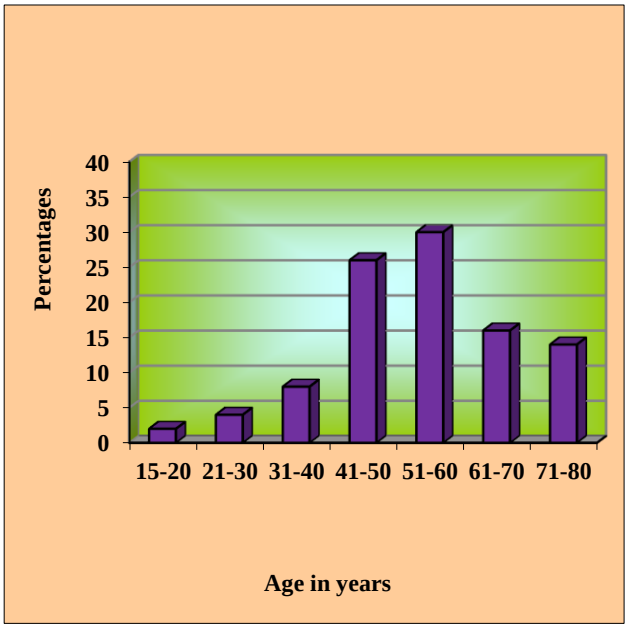

Figure 2: Age distribution.

Table 1: Patient profile.

Ranges	Number	Percentage
HBsAg	6	12
Diabetes mellitus	3	6
Alcoholic	32	64
Idiopathic	9	18

ECHO abnormalities, Cardiomyopathy Global hypokinesia and chamber enlargement present. 3 (Out of 3, 2 patients is alcoholic). Diastolic dysfunction in 32%. Pleural effusion in 38%, pericardial effusion 16%. IHD changes in 11%. LV systolic dysfunction in (LVEF<50%) 8%. (Table 3)

The average pulse rate was 90 and it ranged from 50/m to 130/m. The systolic blood pressure from ranged 85 mmHg to 170 mmHg. The average being 110 mmHg. The diastolic blood pressure ranged from 40 mmhg to 100 mmHg. The average being 60 mmHg.

Table 2: Symptomatology.

Symptoms	No. of patients (n=50)	Percentage
Dyspnea	24	48
Anemia	44	88
Decreased appetite	27	54
Fatigue	33	66
Malena	5	10
Pedal edema	40	80
Ascites	30	60
Pruritus	6	12
Cyanosis	5	10
Jaundice	30	60
Hemetemesis	7	14
Chest pain	6	12

Table 3: Incidence of cardiac complications in cirrhosis of liver.

Complications	No. of cases	Percentage
Hypertension	4	8
Arrhythmias	4	8
Left ventricular failure	4	8
Cardiogenic shock	6	6
Congestive cardiac failure	4	8
Myocardial wall motion abnormality	11	11
Pericardial effusion	8	16
Left ventricular hypertrophy	3	3
Pleural effusion	19	38
Diastolic dysfunction	16	32
Cardiomyopathy	3	6

Table 4: ECG changes is in cirrhosis of liver patients.

ECG changes		
LVH	6	12
Prolonged QTc	17	34
T wave inversion	6	12
Low QRS voltage	12	24
Hemiblock	4	8

The chest roentgenogram showed pleural effusion 38% in 4 patients it is unilateral, bilateral in rest. Cardiomegaly was evident in chest X – ray in 10 patients (20%).

The ECG showed an average heart rate of 90/min. The low QRS voltage in chest leads and limb leads were found in 12 patients. T wave inversion was found in chest leads (V1 to V3 / V6) in 2 (4%) patients, in II, III avf in 4 patients (8%). Hemiblock seen in 4 (8%) cases. QTc prolongation is seen in 34%. LVH is seen in 12% of people. (Table 4)

DISCUSSION

Cardiac dysfunction in cirrhosis of liver arises from multiple causes. Study is conducted on 50 consecutive patients admitted in various medical wards of the Mc Gann Hospital attached to Shimoga Institute of Medical Sciences, Shimoga. From January 2020 to September 2020. It is discussed here and the results compared with various other studies.

Table 5: Causes for cirrhosis of liver.

Ranges	Our study %	Sukhwani N *et al*[8] (%)	Punekar et al[7]
HBsAg	12	43	20
HCV	6	6	3
Alcoholic	64	51	57
Idiopathic	18		18

Sex

There were 35 males (70%) and 15 females (30%) in the present study. The male to female ratio was 1.3:1. This finding is consistent with study done by Punekar et al et al with male (72%) and female (28%).[7]

Age

The age distribution ranged from 15 years to 80 years with maximum number of patients in the age group 51 to 60 years (38%). Average age is 54±6.6 years.

Table 6: Symptomatology.

Symptoms	Our study percentage	Punekar et al[7] (%)	Sukhwani et al[8] (%)	Selvamani et al[10] (%)
Dyspnea	48			
Anemia	88		42.8	
Decreased appetite	54		88.5	
Fatigue	66			
Malena	10	5	54	
Pedal edema	80	34	68.5	
Ascites	60	54	94.2	78.1
Pruritus	12			
Cyanosis	10			
Jaundice	60	51	48.5	28
Hemetemesis	14	6	54	25
Chest pain	12			
Hepatic encephalopathy	4	4		

Table 7: Cardiac manifestation in cirrhosis of liver.

Complications	Our study percentage	Punekar et al[7] (%)	Selvamani et al[10](%)	Elango et al[11](%)
Hypertension	8		6	
Arrhythmias	8		9	
Left ventricular failure	8	6	3.13	6
Cardiogenic shock	6		3	
Congestive cardiac failure	8		3	6
Myocardial wall motion abnormality	11			10
Pericardial effusion	16	22	62.5	8
Left ventricular hypertrophy	3		6.25	
Pleural effusion	38			10
Diastolic dysfunction	32	32		
Cardiomyopathy	6			6

The mean age among men was 54.22±7.34 years and in women was 50.45± 6.56 years. Majority of the patients were in the age group of 40-60 years.

This is consistent with Sukhwani et al8. Where 50-59yrs age group (40%). In study by Sharma et al the mean age of the patients with cirrhosis was 44.70±8.65 years.[9]

In study by Punekar et al the mean age is between 40-49 years 40%.[7]

Our study showed higher incidence of alcoholism as cause of cirrhosis (64%) followed by idiopathic (18%) causes and HBsAg (12%) is third commonest cause. which is consistent with study done by Sukhwani et al and Punekar et al.[7,8] HBsAg and Idiopathic causes are 12 and 18% respectively in our study, which is consistent with study done by Punekar et al.[7] Higher incidence of alcoholic liver disease can be attributed increased malnutrition in Indian population and higher susceptibility of liver to alcohol induced damage in Indian population.

Majority of the patient presented with the symptoms of anemia (88%) and pedal edema (80%) followed by ascites (60%), icterus (60%). Dyspnea seen in 48% may be due to abdominal distension, anemia and cardiac disease combined. Decreased appetite seen in 54%. Values correlated with studies done by Punekar, Sukhwani, Selvamani et al.[7,8,10]

Higher incidence of anemia and hypoprotienemia can be due to preexisting anemia and malnutrition in Indian population, which is supported by other Indian studies.[7,8,10]

In our study 38% had pleural effusion, 32% of cases had diastolic dysfunction. Also, 11% of cases had grade 2 diastolic dysfunction. Also, diastolic dysfunction was significantly higher among Child class B and C compared to A (p=0.04). Albumin level decreased proportionately with diastolic dysfunction. Ascites in our study showed significant correlation with diastolic dysfunction (more the ascites more is the diastolic dysfunction), Hepatic encephalopathy also showed highly significant relation with diastolic dysfunction (p=0.004). This is in correlation with study done by Punekar et al.[7]

It has been said that cardiac arrhythmias in cirrhosis liver are always due to a definable precipitating event such as hypo or hyperkalemia, acidosis, hypoxia or cardiac decomponsation due to IV fluids and blood and blood product transfusion, in older patients the possibility of IHD can be considered. In one study done by Selvamani et al demonstrated that Sinus node dysfunction was seen in 3 patients (3.13%).[10] Ventricular premature complexes was seen in 3 patients (3.13%) and superaventricular premature complex was seen in 3 patients (3.13%). T wave inversion was noted in the precordial leads and limb leads in 12 patients.

In our study left ventricular hypertrophy is seen in 3% of patients. In most studies of patients with cirrhosis, the heart muscle mass had been found to be within the normal range.[12,13] However, some have reported an increased left ventricular muscle mass.[14,15] A recent experimental study of portal hypertensive rats, left ventricular eccentric hypertrophy was found to be correlating well with the degree of hyperdynamic circulation.[16]

Incidence of Systolic dysfunction was found in 8%, this was concordence with study report of echocardiography in chronic liver disease, a systematic review.[17]

Thus the present study confirms that there is an increase in heart rate in cirrhosis of liver as compared to the average heart rate of healthy subjects of same age and sex, this reflects a hyper dynamic circulatory state. The average systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) in the present study were 110 mm/Hg, 60 mm/Hg and 83 mmHg respectively. In the other studies they were as follows: In study done by Elango et al.[11] The SBP ranged from 90 mmHg to 160 mmHg. The average being 110 mmHg. The DBP ranged from 50 mmHg to 100 mmHg. The average being 70 mmHg. The MAP ranged from 70 mmHg to 110 mmHg the average being 86 mmHg. This is consistent with our study. Selvamani et al study MAP is 84.3 mmHg, which is comparable with our study.[10] Significant correlation was demonstrated between the heart rate and MAP both of which indicate a hyper dynamic circulation, which in turn showed correlation with serum albumin and serum bilirubin levels (indicator of liver disease). Our study clearly demonstrated that hyperdynamic circulation progressively increase with the severity of the liver dysfuntion. The study done by Ring et al, showed that the severity of cirrhosis is closely related to the degree of hyperkinetic circulatory state and portal hypertension.[19] In study done by Elango et al there is significant positive correlation is seen between decreased MAP (36 %) and increased HR (54 %), similarly decreased serum albumin (32 %) and increased serum bilirubin (44%).[11]

The current study shows that low voltage QRS complexes were present in 12 patients. Out of which 8 had pericardial effusion probably reflecting the presence of occult pericardial effusion.

In the present study, prolongation of QTc interval on ECG was observed in the majority (34%) of cirrhosis patients. In study done by Bhardwaj et a it was 80%. Similar studies done to evaluate QTc prolongation in cirrhosis patients have described a variable prevalence of abnormal QTc ranging from 30–84%.[18,20,21] This high variability is probably due to the differences in the clinical, demographic profile, presence of comorbidities and drug interactions in patients studied in different settings. On further analysis of data of chronic liver disease patients based on disease severity, findings showed that the mean QTc interval was highest in Child-Pugh Class C that is in severe disease. There is a definite correlation between QTc

prolongation and disease severity. QTc prolongation is many time associated with ventricular arrhythmias and sudden cardiac death since it is known to cause the same in non cirrhotic patients also. Therefore, QTc prolongation is associated with increased mortality cirrhotic patients as evidence by many studies.[22] Therefore, in regular monitoring of these patients for any ECG abnormalities is recommended for early detection of cardiac dysfunction and sudden cardiac death. In our study there was definite association observed between the severity of liver disease and cardiac abnormalities, which is in concordance with many studies, which shows cardiac abnormalities being parallel to severity of hepatic dysfunction in cirrhotic patients.[23]

However it is very difficult to understand the amount of cardiac dysfunction that can be attributed to cirrhosis of liver, due to the frequent presence of other conditions or comorbidities which affect the cardia, such as smoking, hypertension, diabetes, CAD etc. but these comorbidities sometimes could predate the cirrhosis of liver. Many More studies are needed to understand the underlying pathological mechanisms involved in cardiac dysfunction in patients with cirrhosis of liver so that diagnostic evaluation, risk categorization and treatment of patients can be improved.

One important limitation of our study is the small sample size. Others are difficulty in follow-up of the patients, poor compliance to drugs in patients and lack of advance tests such stress testing, thallium scan etc.

CONCLUSION

Study shows that, there is a significant higher incidence of subclinical cardiac abnormalities seen in chronic liver disease patients which increase with duration of the disease. Incidence of subclinical cardiac abnormalities also increases with severity of chronic liver disease. Early detection of subclinical cardiac changes is important to reduce morbidity, mortality and management of chronic liver disease patients.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Sahn DJ, De Maria A, Kisslo J, Weyman A. recommendations regarding quantitation in M-Mode echocardiography results of a survey of echocardiographic measurements. Circulation. 1978;58:1072-83.
2. Herry VL, DE Maria A, Gramiak R, King DL, Kisslo J, Popp RL, Sahn DJ, et al. Report of the American Society of Echocardiography, nomenclature and standards in two-dimensional echocardiography. Circulation. 1980;62:212-7.
3. Ma Z, Lee SS. Cirrhotic cardiomyopathy getting to the heart of the matter. Hepatology. 1996;24:451-9.
4. Shah A, Variyam E. Pericardial effusion and left ventricular dysfunction associated with ascites secondary to hepatic cirrhosis. Arch Int Med. 1988;148:585-688.
5. Moller S, Weinberg N, Henriksen JH. Non-invasive 25 –Hour ambulatory arterial blood pressure monitoring. Hepatology. 1995;22:88-95.
6. Moller S, Bendtsen F, Henriksen JH. Splanchnic and systemic hemodynamic derangement in decompensated cirrhosis. Can J Gastroenterol. 2001;15(2):94-106.
7. P. Punekar, Thakur DK. Echocardiographic changes in chronic liver disease. International Journal of Contemporary Medical Research. 2018;5(3):C1-4.
8. Sukhwani N, Nayak OP, Kumbhkar T. Cardiac changes in patients with chronic liver disease: A prospective descriptive study. Int J Med Res Rev. 2016;4(4):569-74.
9. Sharma N, Chaudhary A. A Comparative Study to Assess the Cardiovascular Complications in Patients of Liver Cirrhosis. Int J Sci Stud. 2021;8(11):78-81.
10. Selvamani S, Vignesh C. Cardiac Changes in Hepatic Cirrhosis: IOSR Journal of Dental and Medical Sciences (IOSR-JDMS). 2016;15(7):01-4.
11. P. Elango, G. Indumathi. Cardiac changes in hepatic cirrhosis in Government Dharmapuri Medical College Hospital, Dharmapuri. IAIM. 2017; 4(9):19-24.
12. Ahmed SS, Howard M, ten Hove W, Leevy CM, Regan TJ. Cardiac function in alcoholics with cirrhosis: absence of overt cardiomyopathy--myth or fact? J Am Coll Cardiol. 1984;3(3):696-702.
13. Møller S, Søndergaard L, Møgelvang J, Henriksen O, Henriksen JH. Decreased right heart blood volume determined by magnetic resonance imaging: evidence of central underfilling in cirrhosis. Hepatology. 1995;22(2):472-8.
14. Friedman HS, Fernando H. Ascites as a marker for the hyperdynamic heart of Laennec's cirrhosis. Alcohol Clin Exp Res. 1992;16(5):968-70.
15. Wong F, Liu P, Lilly L, Bomzon A, Blendis L. Role of cardiac structural and functional abnormalities in the pathogenesis of hyperdynamic circulation and renal sodium retention in cirrhosis. Clin Sci (Lond). 1999;97(3):259-67.
16. Perello A, Inserte J, Godoy A. Cardiac structure and function in experimental intra-hepatic portal hypertension. J Hepatol. 2000;32:65.
17. Mota, Gomes V, Filho M, Brivaldo. Echocardiography in chronic liver disease: systematic review. Arquivos Brasileiros de Cardiologia. 2013;100:376-785.
18. Bhardwaj A, Joshi S, Sharma R, Bhardwaj S, Agrawal R, Gupta N. QTc prolongation in patients of cirrhosis and its relation with disease severity: An observational study from a rural teaching hospital. J Family Med Prim Care. 2020;9:3020-4.

19. Ring – Larsen H, Henriksen JH, Wilken C, Clausen J, Pals H, Christensen NJ. Diuretic treatment in decamp cirrhosis and congestive heart failure; effect of posture. Br Med J. 1986;292:1361-53.
20. Tsiompanidis E, Siakavellas SI, Tentolouris A, Eleftheriadou I, Chorepsima S, Manolakis A, et al. Liver cirrhosis-effect on QT interval and cardiac autonomic nervous system activity. World J Gastrointest Pathophysiol. 2018;9:28-36.
21. Bal JS, Thuluvath PJ. Prolongation of QTc interval: Relationship with etiology and severity of liver disease, mortality and liver transplantation. Liver Int. 2003;23:243-8.
22. Kim SM, George B, Alcivar-Franco D, Campbell CL, Charnigo R, Delisle B, et al. QT prolongation is associated with increased mortality in end stage liver disease. World J Cardiol. 2017;9:347-54.
23. Jaue DN, Ma Z, Lee SS. Cardiac muscarinic receptor function in rats with cirrhotic cardiomyopathy. Hepatol. 1997;25:1361-5.

Cite this article as: Ranjith KGK, Manjunath FV, Parameshwar, Nagabhushana S. Study of cardiac involvement in liver cirrhosis patients. Int J Adv Med 2021;8:940-6.