International Journal of Advances in Medicine
Raghavendra et al. Int J Adv Med. 2016 Nov;3(4):865-868
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: http://dx.doi.org/10.18203/2349-3933.ijam20163497

Original Research Article

A study of cardiovascular abnormalities among cirrhosis of liver cases

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Received: 20 September 2016
Accepted: 29 September 2016

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ABSTRACT

Background: The correlations between alcohol consumption and cirrhosis of liver have been dealt with great detail. As with the many studies that have looked upon the mortality of the patients with cirrhosis, the speed of progression of the disease and the various situations in which the condition of the patient deteriorates, many have been inconclusive about the reason for death in patients of cirrhosis of liver as the disease progresses.
Methods: This study was conducted in the department of medicine, Vijayanagara Institute of Medical Sciences, a tertiary care hospital in Bellary, Karnataka Study subjects: were a group of 50 patients with alcoholic cirrhosis, a group of 50 patients with non-alcoholic cirrhosis and 50 normal subjects without cirrhosis.
Results: The ECG findings among alcoholics, low voltage complex 10%, long QT3% LAE 8% LVH16%. ST T changes 10%. Among non-alcoholic patients low voltage complex 4% Long QT 2% LAE 7%, LVH 16% ST T changes 16%.
Conclusions: The cardiovascular abnormalities did not show much difference between the alcoholic and non-alcoholic patients.

Keywords: Alcohol, Cardiovascular, Cirrhosis of liver

INTRODUCTION

Heart and liver are organs that are closely related both in health and disease chronic liver disease may affect cardiac function in absence of other heart disease, these are called cirrhotic cardiomyopathy Cirrhosis is a common hepatological disorder seen in clinical practice. Cirrhosis is a pathologically defined entity that is associated with a spectrum of characteristic clinical manifestations.[1]

WHO (World Health Organization) described cirrhosis in 1978 as "a diffuse process characterized by fibrosis and conversion of normal liver architectures into structurally abnormal nodules This peculiar transformation of the liver was identified by the first anatomic pathologist, Gianbattista Morgagni in his 500 autopsies published in 1761 but the name of "cirrhosis" ('cirr' is orange color in greek) was given by Laennec in 1826 because of the yellowish-tan color of the cirrhotic liver.[2]

The correlations between alcohol consumption and cirrhosis of liver have been dealt with great detail. As with the many studies that have looked upon the mortality of the patients with cirrhosis, the speed of progression of the disease and the various situations in which the condition of the patient deteriorates, many have been inconclusive about the reason for death in patients of cirrhosis of liver as the disease progresses.[3]

Cardiac abnormalities have been studied by many researchers, of which Kowalski et al were the first to report that patients with cirrhosis had abnormal cardiovascular function and a prolonged QT interval.[4] Advanced liver cirrhosis is associated with an increase in blood volume, a reduction in systemic vascular resistance, and an increase in cardiac output. How this

hyperkinetic circulation affects cardiac function and structure has been incompletely described, however. That is, while evidence has been produced that left ventricular systolic function is usually normal at rest in cirrhotic patients, scanty information is available on whether this applies to diastolic function and cardiac structure as well.[5] This is of pathophysiological relevance because in other diseases diastolic function has proved to be an early marker of cardiac structural abnormality that in advanced cirrhosis may be favored by the influence that stimulation of the renin angiotensin - aldosterone and the sympathetic nervous systems exerts on tissue growth.

Hence, this study will evaluate the clinical aspects of cirrhosis, its effect on cardiac functions and structure by means of echocardiogram.

METHODS

Design of this study was a comparative (descriptive) cross sectional study. This study was conducted in the department of medicine, Vijayanagara Institute of Medical Sciences, a tertiary care hospital in Bellary, Karnataka. Subjects in this study were a group of 50 patients with alcoholic cirrhosis, a group of 50 patients with non-alcoholic cirrhosis and 50 normal subjects without cirrhosis

Inclusion criteria

All patients with cirrhosis of liver presenting to medical OPD and 50 patients without cirrhosis was taken as controls

Exclusion criteria

- Not willing to participate
- Patients with other known aetiology of cardiac disease
- Patients with severe anemia
- Patients with GI bleed
- Patients with hepatorenal syndrome
- Patients with HIV infections

The sample size was based on the study period, i.e the number of patients who fits for the inclusion criteria attending the medicine during the study period.

RESULTS

Table 1: Distribution of study subjects.

Study subjects	Frequency	Percentage
With alcoholic cirrhosis (a)	50	33.3%
With non-alcoholic cirrhosis (b)	50	33.3%
Normal subjects (c)	50	33.3%
Total	**150**	**100%**

Totally 150 study subjects were considered for the study and among them, 50 patients with alcoholic cirrhosis, 50 patients with cirrhosis of non alcoholic etiology and 50 persons without cirrhosis as controls. Non-alcoholic includes hepatitis B 25 g, Hepatitis C 20. One cryptogenic, steotohepatitis.[4]

Table 2: Distribution of study subjects based on age.

Age group	Groups		
	A	**B**	**C**
18 - 40 years	03 (06%)	06 (12%)	08 (16%)
41 - 60 years	33 (66%)	25 (50%)	32 (64%)
61 - 80 years	14 (28%)	19 (38%)	10 (20%)
Total	**50 (100%)**	**50 (100%)**	**50 (100%)**

P value<0.05.

Among patients with alcoholic cirrhosis, majority of patients belong to the age group of 41 - 60 years (66%) followed by 61 - 80 years (28%) and 18 - 40 years (6%). Among patients with non- alcoholic cirrhosis, majority of patients belong to the age group of 41 - 60 years (50%) followed by 61 - 80 years (38%) and 18 - 40 years (6%) among controls, majority of them belong to the age group of 41 - 60 years (64%) followed by 61 - 80 years (20%) and 18 - 40 years (16%).

Table 3: Distribution of study subjects based on ECG changes.

ECG	Groups		
	A	**B**	**C**
Yes	23 (46%)	21 (42%)	04 (08%)
No	27 (54%)	29 (58%)	46 (92%)
Total	**50 (100%)**	**50 (100%)**	**50 (100%)**

P value<0.05.

The ECG findings among alcoholics, low voltage complex 10%, long QT3% LAE 8% LVH16%. ST T changes 10% among non-alcoholic patients low voltage complex 4% long QT 2% LAE 7%, LVH 16% ST T changes 16%.

In ECHO, abnormal LAD was observed among 100% patients. Among patients with non-alcoholic 96% patients had abnormal LAD.

Table 5: Distribution of study subjects based on ECHO findings of LVIDd.

LVIDD (MM)	Groups		
	A	**B**	**C**
35-56 (normal)	00	00	50 (100%)
>56 (abnormal)	50 (100%)	50 (100%)	00
Total	**50 (100%)**	**50 (100%)**	**50 (100%)**

P value>0.05.

ECHO revealed that all the patients with alcoholic and non-alcoholic cirrhosis had abnormal LVIDd.

Table 6: Distribution of study subjects based on ECHO findings of LV mass.

LV MASS (GM/M2)	Groups		
	A	B	C
Normal (M<170, F<160)	17 (34%)	12 (24%)	44 (88%)
Abnormal (M>17(F>160)	33 (66%)	38 (76%)	06 (12%)
Total	**50 (100%)**	**50 (100%)**	**50 (100%)**

P value>0.05.

LV mass abnormality was found among 76% of patients with non-alcoholic cirrhosis compared to 66% of patients with alcoholic cirrhosis.

Table 7: Distribution of study subjects based on ECHO findings of ejection fraction.

Ejection fraction	Groups		
	A	B	C
Normal (M:55-75, F:55- 80)	50 (100%)	48 (96%)	50 (100%)
Abnormal (M>75, F>80)	00	02 (04%)	00
Total	**50 (100%)**	**50 (100%)**	**50 (100%)**

P value>0.05.

Ejection fraction abnormality was found among only 4% of patients with non-alcoholic cirrhosis.

Table 8: Distribution of study subjects based on E/A ratio.

E/A Ratio	Groups		
	A	B	C
Normal (M<1.3, F<1.2)	48 (96%)	46 (92%)	50 (100%)
Abnormal (M>1.3, F>1.2)	02 (04%)	04 (08%)	00
Total	**50 (100%)**	**50 (100%)**	**50 (100%)**

P value>0.05.

The abnormal E/A ratio was observed among 8% of patients with non-alcoholic cirrhosis compared to 4% of alcoholic cirrhosis patients.

DISCUSSION

In the cirrhotic population among the study group, 44% (44 cases) had ECG changes, of which, 23 were alcoholics and 21 were non alcoholics. Among the controls 4 patients had nonspecific T wave inversion ECG abnormalities. More cases had LVH 32% and ST T changes 26% and, some of them had long QT intervals 4%. These cases showed a significant correlation with cardiac deformities in accordance with the Bernadi et al study.[6] The comparison of ECG variations was statistically significant in comparison with the control subjects. There was no significant difference between the ECG findings between alcoholics and non-alcoholics. Long QTc is associated with sudden death due to arrhythmias.[7]

The LAD was above normal limits (>40 mm) in 98 patients of the study group. Much significant changes were not found between the alcoholics and non-alcoholics. There was significant difference in the values of LAD compared to the controls among which only 2 of them had an elevated value more than 40. These findings were in line with the studies conducted by Wong et al and Pozzi et al.[8]

The echo findings showed that in all patients in our study group, the value of LVIDd was more than 56, which was the value taken as the cut off. This shows that almost all cirrhotic patients will have some sort of cardiac enlargement during the course of the disease. As the follow up of the patients were not done, it was not able to assess the rate of progression and its rapidity. The findings correlated with the studies conducted by Wong et al and Pozzi et al.[8]

The machine calculated LV mass was abnormal in 71 of the cirrhotic patients as compared to 6 of the controls. Among this 33 of them were alcoholics and 38 were non alcoholics. This increased value among non-alcoholics could be attributed to the etiologies which could cause concurrent cardiomyopathy and cirrhosis. The difference in LV mass was statistically significant as compared to the controls. The increase in LV mass was mostly among those in the age group 41-60 years, which could be explained by the increased number of subjects in that age group. The findings correlated with studies by pozzi et al.[8] The major cardiac structural abnormality of the myocardium in such patients was myocardial hypertrophy. One possible explanation for this would be myocardial adaptation to a chronically elevated blood volume.

Alternatively, ventricular hypertrophy or remodelling could be related to the trophic effects of activated neurohormonal systems such as noradrenaline, or angiotensin II with or without the synergistic effects of endothelin-1.[9]

Abnormal E/A ratio were found in 6% of the cirrhotic cases as compared to none of them among the controls. Studies have proven that there is E/A normalization in patients that receive liver transplant.[10] The difference in this study was statistically significant.

LVEF was normal in 148 of the patients of the study group, with mean value of 64.53±0.97%, similar to results in same above mentioned studies. This paradoxical normal EF value in the face of diastolic dysfunction could probably be because of normal pre and after load of the cirrhotic heart as explained by Muller et al.[10]

CONCLUSION

ECG variations, mainly in the form of low voltage complexes, left ventricular hypertrophy and QT prolongation were present in many cirrhotic patients irrespective of the aetiology of cirrhosis.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

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Cite this article as: Raghavendra, Kotresh N, Naik S. A study of cardiovascular abnormalities among cirrhosis of liver cases. Int J Adv Med 2016;3:865-8.