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General Medicine

"IMPORTANCE OF ECG AND ECHO IN PATIENTS OF CHRONIC LIVER DISEASE WITH SPECIAL REFERENCE TO ALCOHOLIC LIVER DISEASE"

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ABSTRACT **Background-** Alcohol consumption has many detrimental effects on various organs of the body, especially liver, heart and brain. Cardiac dysfunction in cirrhotic patients is common especially in patients with alcoholic CLD. Cardiac involvement in these patients may be masked by the clinical features of CLD. Moreover, cardiac dysfunction in chronic alcoholics remain asymptomatic till very advanced stage. **Objectives-** Present study was carried out in order to find cardiac involvement in patients of CLD and to study the difference in ECG and ECHO findings among alcoholic and non-alcoholic CLD, if any. **Materials and Methods**- Present case control study was conducted in Medicine Department of a tertiary care hospital over a duration of one year on 100 randomly selected cases of CLD, which were divided into cases (n=50, patients of CLD of alcoholic etiology) and control (n=50, the etiology of which was non-alcoholic). Both the groups were compared to different parameters and cardiac function was evaluated. **Result-** In our study, we found that the patients having QT prolongation (p=0.023), chamber enlargement (p=0.025) and conduction defects (p=0.046) were significantly higher among the cases as compared to control group. Abnormal ECHO findings including chamber enlargement (p=0.004), diastolic dysfunction (p=0.023), valvular defects (p=0.003) and RWMA (p=0.041) were significantly higher among cases as compared to control group. **Conclusion-** We recommend the screening of patients of CLD via Echocardiography, for early detection of cardiac involvement allowing timely intervention for better outcomes. Due weightage should be given for cardiac screening in chronic alcoholics with CLD without waiting for signs and symptoms pertaining to heart.

KEYWORDS : Cirrhosis, Alcohol, Cardiac dysfunction, CLD, Cardiomyopathy, ECHO

INTRODUCTION

Alcohol consumption is a common practice in rural as well as urban population with broad range of consumption. Alcoholic beverages are widely consumed throughout the world. The broad range of alcohol consumption patterns from daily heavy drinking to occasional hazardous drinking creates significant public health and safety problems in nearly all countries. Alcohol related toxicity is the third most common cause of morbidity and the fifth most common cause of disease burden worldwide.[1] Alcohol abuse is the leading cause of mortality in people aged 15–49 years, and the total expenditure amounts to billions of dollars. In developing countries, alcohol is the most common aetiology of cirrhosis.[2] Overall, 3.5% of the global burden of disease is attributable to alcohol, which accounts for as much death and disability as tobacco and hypertension.[3]

The relationship between alcohol consumption and cardiovascular diseases is complex- "the alcoholic's paradox" i.e. light intake of alcohol is actually cardioprotective whereas heavy intake of alcohol (as present in most cases of alcoholic liver disease) leads to increased cardiovascular morbidity and mortality. Light to moderate drinking may have a beneficial impact on morbidity and mortality for ischemic heart disease and ischemic stroke. However, the beneficial cardioprotective effect of drinking disappears with heavy drinking occasions. Rehm(2010) have shown, based on meta-analyses that, on average, light to moderate drinkers experienced no protective effect if they reported at least one heavy drinking occasion per month. Moreover, alcohol consumption has detrimental effects on hypertension, cardiac dysrhythmias and haemorrhagic stroke, regardless of the drinking pattern.[3]

We all are well aware about the detrimental effects of alcohol on various organs of the body, especially liver, but other organs are also not spared by the alcohol. Its effects on heart and brain are also well known, identified and described, regardless of the drinking pattern. Apart from many causative factors for CLD (chronic liver disease), alcohol still remains to be the most stressed and discussed factor as it is one of the most common cause and most importantly it is a modifiable factor. The diagnosis of CLD in most cases is delayed till the decompensating symptoms become evident.

Cardiac dysfunction in cirrhotic patients is common especially in patients with alcoholic CLD as seen in electrocardiographic and echocardiographic findings. Various studies have shown that 50% of asymptomatic chronic alcoholics have LVH (Left ventricular hypertrophy) and diastolic dysfunction whereas 30% of asymptomatic chronic alcoholics have LV systolic dysfunction. Cardiac involvement in these patients may be masked by the clinical features of CLD. Moreover, cardiac dysfunction in chronic alcoholics remain asymptomatic till very late and advanced stage. These signs and symptoms become evident when heart failure sets in.

As a clinician, our prime focus should be on how to detect various organ dysfunction in chronic alcoholics, be it liver or heart, as early as possible before the decompensation sets in.

AIMS AND OBJECTIVES

- To study the electrocardiographic findings in patients of alcoholic chronic liver disease
- To study the echocardiographic findings in patients of alcoholic chronic liver disease
- To compare the electrocardiographic and echocardiographic findings in alcoholic and non-alcoholic chronic liver disease
- To correlate the electrocardiographic and echocardiographic findings with modified Child Pugh Turcotte Score

MATERIALS AND METHODS

Study Type:
Case control study

Study Place:
Department of General Medicine, G.R. Medical College, Gwalior (M.P.)

Sample Size:
100, We included 100 patients of CLD which were divided into Cases (n=50, chronic liver disease of alcoholic etiology) and Control (n=50, the etiology of which will be non-alcoholic). Patients having pre-existing cardiac disease, cerebrovascular accidents, diabetes mellitus, hypertension, chronic kidney disease and chronic obstructive pulmonary disease were excluded from the study.

Both the groups were compared to different parameters and following observations were made.

OBSERVATIONS AND RESULTS

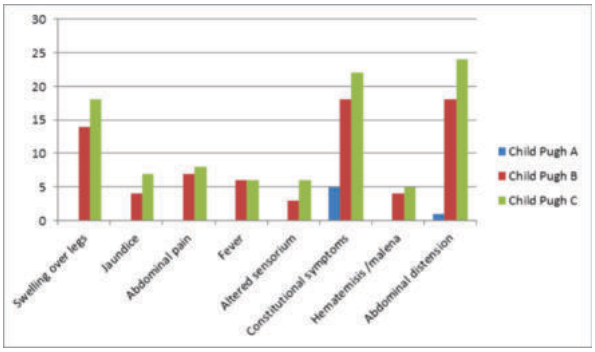
In our study, majority of the patients belonged to age group of 51-60 (n=18) and 41-50 years (n=19) among case and control respectively.

There was equal distribution of ages among both the groups (p=0.068). Majority of the subjects in study population had Child Pugh Score of C (n=45) followed by B (n=41) and A (n=14). Among cases, majority of the patients had modified Child Pugh Score of C (n=24) followed by B (n=20) and A (n=6) whereas among the control group, Modified Child Pugh Score B (n=21) and C (n=21) were equally distributed and Modified Child Pugh Score A had 8 patients.

Symptomatology is similar in both the groups. Majority of the patients had constitutional symptoms followed by abdominal distension, swelling over legs, abdominal pain and fever. When we correlated the symptoms with Modified Child Pugh Score among cases, we found that majority of the patients with swelling of legs, jaundice, abdominal pain, fever, altered sensorium, constitutional symptoms, hematemesis /malena and abdominal distension had Child Pugh Class C.

Table 1: Symptom distribution among cases and controls

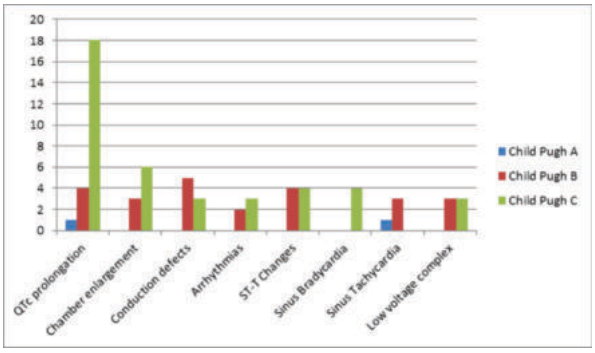
Symptoms	Case	Control	Total	P value
Swelling over legs	32	32	64	1.00
Jaundice	11	13	24	0.640
Abdominal Pain	15	13	28	0.656
Fever	12	12	24	1.00
Altered sensorium	9	9	18	1.00
Constitutional Symptoms	45	42	87	0.372
Hemetemesis/Malena	9	8	17	0.790
Abdominal distension	43	37	80	0.134



Graph 1: Symptom wise distribution among cases according to Child Pugh score

Table 2: Comparing ECG findings among cases and controls

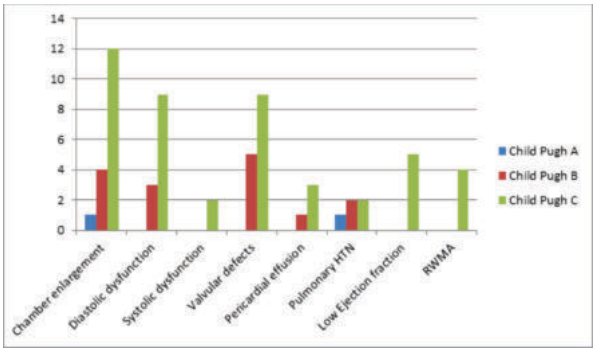
ECG findings	Case	Control	Total	P value
QT prolongation	23	10	33	0.023
Chamber enlargement	9	2	11	0.025
Conduction defects	8	2	20	0.046
Arrhythmias	5	4	9	0.727
ST-T Changes	8	6	14	0.564
Sinus Bradycardia	4	1	5	0.169
Sinus Tachycardia	4	5	9	0.727
Low voltage complex	6	6	12	1.00



Graph 2: Distribution of ECG findings among cases according to Child Pugh Score

Table 3: Comparing individual ECHO findings among cases and controls

ECHO findings	Case	Control	Total	P value
Chamber enlargement	17	5	22	0.004
Diastolic dysfunction	12	3	15	0.023
Systolic dysfunction	2	2	4	1.00
Valvular defects	14	3	17	0.003
Pericardial effusion	4	4	8	1.00
Pulmonary HTN	5	5	10	1.00
Low Ejection fraction	5	5	10	1.00
RWMA (Regional wall motion abnormality)	4	0	4	0.041



Graph 3: Distribution of 2D Echo findings among cases according to Child Pugh Score

DISCUSSION

Cirrhosis is associated with a host of cardiovascular abnormalities including hyperdynamic circulation, portal hypertension, hepatopulmonary syndrome and changes in several different vascular territories such as the renal and cerebral vasculature.[4] Peripheral vasodilatation by reducing cardiac "after load" may prevent any clinical evidence of cardiac dysfunction; however, many studies have demonstrated that under physiological or pharmacological stress the ventricular systolic function may give an inadequate response.[5] Cirrhosis is associated with an increased cardiac output and heart rate, as well as decreased systemic peripheral vascular resistance and blood pressure. Splanchnic arterial vasodilatation and impaired autonomic activity play a role.[6] Cirrhotic cardiomyopathy is a pathological condition defined as "a chronic cardiac dysfunction in patients with cirrhosis characterized by blunted contractile responsiveness to stress and/or altered diastolic relaxation with electrophysiological abnormalities in the absence of known cardiac disease.[7]

In cirrhosis, cardiac output increases while systemic vascular resistance and arterial pressure decreases.[8] Thus, many a times cirrhotic patients are dismissed as having normal cardiac function. However, when these patients are subjected to physiological or pharmacological stress they are prone to develop clinical signs of suboptimal perfusion including renal failure and acidosis. Most patients with liver disease have subtle defects in myocardial function that are not apparent on examination and these defects become clinically overt only when these patients are exposed to stress.[9]

In our study, we found that the patients having QT prolongation (p=0.023), chamber enlargement (p=0.025) and conduction defects (p=0.046) were significantly higher among the cases as compared to control group. Patients having arrhythmias, ST-T changes , sinus bradycardia, sinus tachycardia and low voltage complex were equally distributed between both groups (p>0.05). Among cases, majority of the patients with QT prolongation (p=0.024) and chamber enlargement (p=0.025) had Child pugh score of C (18 and 6 patients respectively) while distribution of patients with conduction defects, arrhythmias, ST-T changes and low voltage complex among different Child Pugh Class was statistically insignificant (p>0.05).

Abnormal ECHO findings including chamber enlargement (p=0.004), diastolic dysfunction (p=0.023), valvular defects (p=0.003) and RWMA (p=0.041) were significantly higher among cases as compared to control group. Among cases, abnormal ECHO findings including chamber enlargement (p=0.004), diastolic dysfunction (p=0.012), valvular defects (p=0.003) and RWMA (p=0.041) were more common in Child pugh score C while distribution of systolic dysfunction, pericardial effusion and pulmonary hypertension among different Child Pugh Class was statistically insignificant (p>0.05).

In agreement to present study, Bernardi M et al stated QT prolongation as the major ECG abnormality in cirrhotic patients which parallels

with the severity of liver disease.[4] The author showed a prevalence of 42.9% in alcoholic and 47.1% in non alcoholic cirrhosis. By contrast, Bal and Thuluvath[10] reported that the prolonged QTc was a more common finding in patients with alcoholic cirrhosis (60%) than in non-alcoholic ones (35%), and that alcoholic cirrhosis was an independent predictor of QT interval prolongation.

In study by Pozzi et al the clinical and laboratory data of 60 patients with liver cirrhosis were classified according to their Child- Pugh score into 3 groups. Twenty eight patients had ECG changes in the form of prolongation of the QT interval which is the most widely recognized electrophysiological abnormality in cirrhosis. An elevated prevalence of QT interval prolongation was first shown in patients with alcoholic liver disease.[7] Several investigations have now shown that QT interval prolongation increases with the severity of liver disease, but can also occur in patients with well compensated cirrhosis. In fact, its prevalence was 25% in class A of Child–Pugh classification, 51% in class B, and 60% in class C. Prolongation of the QT interval is well known to increase the risk for ventricular tachyarrhythmias.[4]

Sun et al demonstrated diastolic dysfunction in 48.8% of cirrhotics. [11] The author also associated the cardiac changes with Child score. Ruiz-Del-Arbol et al also found diastolic dysfunction in patients with cirrhosis.[12] Hansen et al, studied the cardiac function in patients with liver cirrhosis. Thirty patients with liver cirrhosis, referred to as group I (G I), were selected. They were subdivided according to Child-Pugh classification. Thirty healthy subjects, referred to as group II (G II), were selected as a control group. This study showed significant difference between two groups with regards to LV (left ventricular) diastolic parameters (E/A wave), (E/Em) and left atrial diameter.[13]

Pavarino et al, who investigated a fifty-two patients with cirrhosis, after excluding those with any medical condition that could affect the cardiac condition, cardiac structural and functional assessment was performed non-invasively using transthoracic echocardiography (TTE) found that there was significant increase in ejection fraction (EF) in cirrhotic group which was more evident in the early stage of liver disease compared to controls. There was also a significant increase in left ventricular wall thickness and diastolic dysfunction (decreased E/A ratio and prolonged deceleration time of E wave (EDT)) which were more marked in advanced stages of cirrhosis. Left atrial diameter (LAD) and aortic root diameter (ARD) showed a highly significant increase in patients with cirrhosis.[14]

Wakiri et al., studied the effects of liver cirrhosis due to viral hepatitis on cardiac ventricular functions, thirty patients (mean age 43.6±12,20 male) with liver cirrhosis underwent echocardiographic studies and were compared with 30 healthy controls (mean age 37.3±2, 22 male) showed that there were no significant differences in left ventricle systolic and diastolic functions between the groups.[15] Naschitz et al 2000 showed the significant difference between GI and GII regarding RV diameter, RV stroke volume and RV EF%. Trevisani et al. 2001 showed the impaired right ventricle diastolic functions in cirrhotic patients. Kowalsky and Abelmann described an impaired contractility function in patients with alcoholic cirrhosis.[16] Nevertheless, the cause of chronic liver disease per se could be the cause of cardiomyopathy (e.g. alcohol), and accordingly, heavy drinkers may suffer from heart failure before a significant liver damage has occurred. In this regard, in the study by An et al[17] subgroup analysis showed that alcoholic cirrhosis was an independent risk factor, unlike hepatitis virus related disease. Therefore, the real impact of cardiovascular risk in cirrhosis is still undefined; at this time, no specific guidelines have been developed in this clinical setting to evaluate cirrhotic patients for cardiovascular risk factors or presence of cardiac dysfunction.

CONCLUSION

- The echocardiographic study should be a part of the screening of patients with CLD, because patients with systolic and/or diastolic dysfunction have higher morbidity and mortality. A better understanding of the echocardiographic findings, mainly cardiac strain imaging, in patients with CLD will help in improving their management.
- By the time patient is diagnosed of alcoholic hepatitis or CLD, it is advised to screen the patient for cardiac involvement as well. Due weightage should be given for cardiac screening in chronic alcoholics with CLD without waiting for signs and symptoms pertaining to heart.
- During this time, patient may remain asymptomatic for further one or two decades. Most of the time, signs and symptoms of cardiac involvement are attributed to liver decompensation. RV dysfunction again mimics decompensated CLD. Hence patient should be subjected to ECG/ECHO.
- This allows timely intervention to prevent further damage and progression of the disease, thus providing morbidity and mortality benefits.

Think Of Cardiac Involvement!!

- When there is exertional dyspnoea
- When pedal edema is out of proportion of S. Protein/ albumin levels
- In chronic alcoholics- As USG abdomen is done for liver, USG heart (ECHO) should also be done.
- Not to miss cardiac disease in the setting of CLD.
- See the fatty liver or its sequlae in alcoholics, Imagine the coronaries.
- Clinicians must be very vigilant while dealing with a case of chronic liver disease especially alcohol related.
- Efforts should be made for earliest possible detection of liver disease while it is in compensated stage and same rule should be applied for cardiac dysfunction before it becomes symptomatic.

Limitations Of The Study

Present study has few limitations. First cross sectional nature of the present study was the main limitation which restricts the use of present study findings to large population. Second is the small sample size; a large randomized clinical trial is required to strengthen the present study findings.

Conflict Of Interest – Nil

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