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PREVALENCE OF DILATED CARDIOMYOPATHY IN PATIENTS WITH ALCOHOLIC LIVER DISEASE

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ABSTRACT *Alcoholic cardiomyopathy is a clinico pathologic state induced by chronic alcoholism and a major cause of dilated cardiomyopathy, characterised by cardiac enlargement and low cardiac output state. It is associated with a 3 year mortality rate of 40-80% and it may present with mild symptoms to even sudden death from ventricular fibrillation. It is associated with the duration and the amount of alcohol drunk. On the other hand development of liver dysfunction in alcoholics is also associated with duration of alcohol intake. In this study we will search the prevalence of alcoholic cardiomyopathy in patients of alcoholic liver disease.*

KEYWORDS : Alcoholic cardiomyopathy, Alcoholic liver disease, cirrhosis, Dilated Cardiomyopathy

INTRODUCTION:

Alcoholic liver disease (ALD) is one of the most serious complications of alcoholism, but other organs may also be affected by long-term alcohol dependence or abuseChronic alcohol use has been implicated as the etiology of left ventricular (LV) dysfunction in as many as one third of cases of Dilated Cardiomyopathy. The mechanism by which alcohol causes cardiac damage remains unclear. Over many years, several theories have arisen based on clinical and scientific data obtained in human and animal studies, including oxidative stress, apoptosis, mitochondrial dysfunction, derangements of fatty acid metabolism/transport, and accelerated protein catabolism.

AIMS AND OBJECTIVE:

To find the prevalence of Dilated cardiomyopathy in patients of Alcoholic liver disease.

MATERIALS AND METHODS:

The study was undertaken in patients attending OPD and Indoor Department of Medicine, JLN Medical College, Bhagalpur. The study was undertaken in patients attending OPD and Indoor Department of Medicine, JLN Medical College, Bhagalpur. The study was undertaken in patients attending OPD and Indoor Department of Medicine, JLN Medical College, Bhagalpur. After informed consent 100 patients were included in the study.100 diagnosed cases of alcoholic liver disease(based on History of alcohol consumption, USG findings and laboratory investigations suggestive of alcoholic liver disease) were enrolled in the study.

INCLUSION CRITERIA:

1. H/O alcohol intake of 40-80 gm for atleast 10 years
2. Raised SGOT and SGPT both 2 to 7 folds
3. SGOT/SGPT> 1
4. USG findings of alcoholic liver disease- fatty liver or cirrhosis
5. No other co morbid conditions

EXCLUSION CRITERIA:

Presence of other conditions of dilated cardiomyopathy like Hypertension, diabetes mellitus, Ischemic heart disease,peripartum cardiomyopathy were excluded from the study.

Also presence of other factors which can lead to liver disease like Infective Hepatits(B,C), Wilsons disease were excluded from the study.

RESULTS:

Table 1. Distribution of the cases according to stages of liver disease on USG

	NUMBER	PERCENT
FATTY LIVER	72	72
CIRRHOSIS	28	28

Table 2.Distribution of Dilated cardiomyopathy in the study according to Echocardiographic findings

DILATED CARDIOMYOPATHY	NUMBER	PERCENTAGE
PRESENT	23	23
ABSENT	77	77
TOTAL	100	100

It was also seen that 18 patients out of the 23 patients who were having cirrhosis had dilated cardiomyopathy implying severe degree of liver disorder is associated with heart dysfunction.

DISCUSSION:

Alcoholic cardiomyopathy is a type of acquired dilated cardiomyopathy associated with long-term heavy alcohol consumption (commonly defined as >80 g per day over a period of at least five years). Like other types of dilated cardiomyopathy, alcoholic cardiomyopathy is characterized by a dilated left ventricle (LV), increased LV mass, and LV systolic dysfunction (generally detected as reduced LV ejection fraction).The original theories regarding the mechanism focused on nutritional deficiencies (eg, thiamine deficiency), secondary exposures (eg, tobacco, cobalt, arsenic), and other comorbidities (eg, hypertension). However, although these mechanisms may play a role in selected patients, most evidence in the literature indicates that the effects of alcohol on the myocardium are independent of these factors and that the effect is a direct toxic result of ethanol or its metabolites. Some studies have suggested that a genetic vulnerability exists to the myocardial effects of alcohol consumption. Individuals with certain mitochondrial deoxyribonucleic acid (DNA) mutations and angiotensin-converting enzyme (ACE) genotypes (DD genotype) may be particularly susceptible to the damaging effects of alcohol. Exactly how these genetic variables create this higher risk is not known. Also role of different mitochondrial dysfunctions as well as dysfunction of mitochondrial dysfunction has been elucidated.

Alcoholic cardiomyopathy is most common in men between the ages of 35 and 50, but the condition can affect women as well. People with alcoholic cardiomyopathy often have a history of heavy, long-term drinking, usually between five and 15 years. Heavy drinking is alcohol consumption that exceeds the recommended daily limits. For men, heavy drinking is defined as more than four

drinks per day or more than 14 drinks per week. For women, it's defined as more than three drinks per day or more than seven drinks per week. Alcoholic cardiomyopathy doesn't always cause symptoms. When symptoms do occur, they're often those of heart failure. They commonly include fatigue, shortness of breath, and swelling of the legs and feet.

People with alcoholic cardiomyopathy might experience: shortness of breath, swelling of the legs, feet, and ankles, fatigue, weakness, dizziness or fainting, loss of appetite, a rapid and irregular pulse,a cough that produces a frothy, pink mucus,a change in urine output. It's important to note that alcoholic cardiomyopathy may not cause any symptoms until the disease is more advanced. At that point, the symptoms are often the result of heart failure.

Physical examination findings in alcoholic cardiomyopathy are not unique compared with findings in dilated cardiomyopathy from other causes. Elevated systemic blood pressure may reflect excessive intake of alcohol, but not alcoholic cardiomyopathy per se.Frequently, a relative decrease occurs in systolic blood pressure because of reduced cardiac output and increased diastolic blood pressure due to peripheral vasoconstriction, resulting in a decrease in the pulse pressure.

Cardiac percussion and palpation reveal evidence of an enlarged heart with a laterally displaced and diffuse point of maximal impulse. Auscultation can help to reveal the apical murmur of mitral regurgitation and the lower parasternal murmur of tricuspid regurgitation secondary to papillary muscle displacement and dysfunction. Third and fourth heart sounds can be heard, and they signify systolic and diastolic dysfunction. Pulmonary rales signify pulmonary congestion secondary to elevated left atrial and LV end-diastolic pressures. Jugular venous distention, peripheral edema, and hepatomegaly are evidence of elevated right heart pressures and right ventricular dysfunction.

Other findings may include cool extremities with decreased pulses and generalized cachexia, muscle atrophy, and weakness due to chronic heart failure and/or the direct effect of chronic alcohol consumption. Electrocardiographic findings are frequently abnormal, and these findings may be the only indication of heart disease in asymptomatic patients. Palpitations, dizziness, and syncope are common complaints and are frequently caused by arrhythmias (eg, atrial fibrillation, flutter) and premature contractions. Other supraventricular tachyarrhythmias and sudden death have also been associated with alcohol use and AC, with the latter being most likely secondary to the development of ventricular fibrillation. Conduction disturbances, such as degrees of atrioventricular block, left or right bundle-branch block and hemiblocks, are also observed. The mainstay of therapy for alcoholic cardiomyopathy is to treat the underlying cause, ie, to have the patient exercise complete and perpetual abstinence from all alcohol consumption. Medical therapy for alcoholic cardiomyopathy is identical to conventional therapy for other forms of heart failure. This includes treatment with an ACE inhibitor and with digoxin (for patients with symptomatic LV dysfunction), as well as the symptomatic use of diuretics. Newer therapies, such as beta blockers in stable patients without decompensated heart failure, are also used. Thiamine (200 mg once daily), multivitamins, vitamin B-12, folate, and mineral supplementation are beneficial for patients with AC because of the significant prevalence of concomitant nutritional or electrolyte deficiencies in these patients. Animal studies have suggested a benefit from vitamins B-1 and B-12, speculated to be due to protective effects against apoptosis and protein damage.

CONCLUSION:

In our study we have found that 23 patients out of the 100 patients enrolled in the study had dilated cardiomyopathy that means almost ¼ of the liver disease patients had evidence of dilated cardiomyopathy. The results of our study are at a variance with the commonly held view of an inverse relationship between alcoholic liver disease and cardiomyopathy. The pathogenesis of alcoholic liver disease and dilated cardiomyopathy has not been completely elucidated but the findings of our study suggest that there may be a common pathogenic mechanism at cellular mechanism. Timely intervention of alcoholic cardiomyopathy is the keystone to success in treating them, so all patients admitted with alcoholic liver disease must have a proper cardiological examination to rule out dilated cardiomyopathy.

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