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A CROSS STUDY OF CARDIAC FUNCTION BASED ON CLINICAL FEATURES

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ABSTRACT

Cirrhosis is a diffuse process of fibrosis and conversion of normal liver parenchyma into abnormal regenerative nodules. The clinical features associated with chronic liver disease reflect the severity of its pathogenesis. Activation of the SNS, increases levels of norepinephrine, or increased cytokines from the portal venous bacteraemia and endotoxemia in cirrhotic patients might stimulate carbon monoxide production. Carbon monoxide reduces the contractility of cardiac myocytes through cyclic GMP and decreases the influx of calcium. Heme oxygenase messenger ribonucleic acid transcription, protein expression, and total hemeoxygenase activity were increased in cirrhotic hearts compared to sham-operated control rat hearts. The sodium overload causes an increase in end-systolic volume in cirrhotic subjects in a standard, resting hemodynamic set-up. In the presence of ascites in cirrhotic patients, contractile dysfunction is prominent despite an increase in both arterial vasodilation (after-load) and venous return (pre-load). Due to the lack of proper functioning of the pump, the cardiac index is decreased, and systemic vascular resistance is elevated in cirrhosis with compensation, which is not related to the patient's hypertensive status. The capacity of the heart to increase pulse rate or LV EF with heavy work or drug intake is dampened in cirrhotic patients. Thus results in a decrease in liver parenchymal mass, decrease in liver function and alters the blood flow. Fall in the heart rate and cardiac output due to a decline in cardiac response may be because of impaired sensitivity to activate SNS and cardiovascular reflex regulation, which add to the chronotropic in adaptiveness.

Keywords: SNS, LV EF, left ventricular dilatation, Hypertrophy, Hyperdynamic.

INTRODUCTION

In this section presents the introduction of this research work. In many studies, it is found that structural and functional changes have been noted in the left heart, but not on the right [1,2]. Left atrial dilatation and left ventricular dilatation or hypertrophy has been observed.[3,4] This is reflected in a hyperdynamic state wherein there is an increased pulse rate and decreased cardiac output in the initial stages where the patient is asymptomatic [5,6].

In the second compensatory phase, the patient becomes clinically apparent with palpitations, tachycardia [7]. At this point, there is a worsening hyperdynamic state. If untreated, the patient progresses to the third phase of cardiac failure where the patient might present with signs and symptoms of cardiac failure [8]. This includes pulmonary oedema. A variety of cardiac changes occur in CCM, that can be looked upon as high-output cardiac changes; the series of these events is not fully [9]. In this paper presents section 2 of this paper explains the detail on the related works. In section 3 presents the materials and methods adopted and section 4 presents the details of the experiments and discussions. Finally, section 5 concludes the paper by sharing our inferences and plans.

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RELATED WORKS

In this section presents focuses on the related works of this research work. Apoptosis is essential for myocardial remodelling in cardiac failure [10]. Mitogen-activated protein kinases (MAPKs) that respond to various stimuli are signally proteins [11]. Of all the kinases of the MAPK family, the one that is influential in growth differentiation, proliferation, and apoptosis is p38-MAPK [12]. It has been proven that there are many cirrhosis - inducing agents which target and activate p38-MAPK present in myofibroblasts [13]. There is a specific isoform (the p38-MAPK isoform, p38) demonstrated by the gene transfer technique that has shown to cause ischemia and then cardiomyocyte apoptosis ultimately leading to cell death [14]. There is also, SB203580, which selectively inhibits the p38 /p38 isoforms and thereby helps in protecting the cardiac myocytes from damage that is caused by ischaemia [15]. This helps to prove the role that p38 has in apoptosis. This is caused due to the inhibition of the -isoform and not the beta isoform. It is also found that there is a potent pro-fibrogenic and pro-apoptotic cytokine which is transforming growth factor. This factor is also increased in cirrhosis, and its mechanism is through non-smad pathways and smad proteins which includes both the JUN NH2-terminal kinases and p38 MAPK.

MATERIALS AND METHODS

In this section presents the materials and methods of this research work. Institutional ethical committee clearance was obtained. Fifty patients diagnosed with chronic liver disease, who were admitted in the medical wards were taken into the study. Informed and written consent from the patients was obtained to participate in the study.

The patients were subjected to detailed history (age, sex, alcohol intake and duration of alcohol intake, duration of liver disease and co-existing conditions like Diabetes Mellitus and Hepatitis B, if any, were noted) and clinical examination (pallor, icterus, oedema, splenomegaly, ascites and encephalopathy). The baseline investigations like liver function tests, coagulation parameters, renal function tests and electrolytes were taken. The second line investigations specific for cardiovascular system namely ECG (12 leads), Echo (2 dimensional) and cardiac markers are done using the methods mentioned in Table like troponin I done by CLIA method, in CK MB the IFCC method was used and NT pro BNP method of Fluorescence Immune Assay were taken.

RESULTS AND DISCUSSIONS

In this section focuses the results and discussions of this research work. The mean age was 46.48 ± 11.51(years) in the study population. The proportion of male participants were 42 (84.0%), and female were 8 (16.0%) in the study population. The proportion of participants who reported alcohol intakes were 34 (68.0%), and 16 (32.0%) people did not report with alcohol intake in the study population. The mean alcohol duration was 15.20 ± 9.171(years) in the study population. The average duration of liver disease was 10.19 ± 8.29 (years) in the study group. The proportion of participants who had reported with Type 2 Diabetes Mellitus were 13 (26.0%). Portal Hypertension is a common entity which results in a range of disorders like gastroesophageal varices, caput medusa, splenomegaly, hypersplenism. The primary pathophysiology involved in this can be explained by increased resistance and blood flow in the Porto-venous system. Ascites occurs due to Ascites is a common complication due to increased pressure in the hepatic vein. Complications of ascites are spontaneous bacterial peritonitis. The hepatorenal syndrome occurs due to destruction in the renal blood flow—hepatic encephalopathy when there is increased protein intake which is beyond the liver's capacity to metabolize.

Hepatopulmonary syndrome; Porto-pulmonary hypertension are complications involving the respiratory system. The other comorbidities include malnutrition, coagulopathy due to clotting factors deficiency, fibrinolysis, thrombocytopenia; bone disease- osteopenia, osteoporosis, osteomalacia; hematologic abnormalities – anaemia, hemolysis, thrombocytopenia, and in some cases

cirrhotic cardiomyopathy. Majority of study population 56% were under the age group of 41 to 60 years of age, followed by 32% participants under 21 to 40 years of age and 12 % patients under the age of above 60 years. None of the study subjects was in the age group of 18 to 20 years of age. In the present study, 23 patients (46.0%) were with pallor, 36 patients (72%) were with icterus, and 12 patients (24%) had oedema. And 24 patients (48%) were found to have splenomegaly, and eight patients (16%) had encephalopathy. Twenty-seven patients (54%) had ascites out of which19 patients (38%) had mild ascites and eight patients (16%) had moderate to severe ascites. It shows that majority of patients had indirect hyperbilirubinemia with mean total bilirubin 3.34 +/- 3.37, with moderately elevated transaminases and GGT. Coagulation parameters were prolonged with mean PT being 15.90 +/-2.55, INR was 1.303 +/- 0.197, and aPTT was 41.91+/- 3.107.

CONCLUSION

Finally, this work concludes that the enzyme representation occurs in the caveolae which are found on the sarcoplasmic reticulum by the neuronal and the endothelial forms of NOS. This NO that is produced as a result of this NOS helps in perfusion, and Inflammatory mediators help to stimulate the iNOS (the inducible form of NOS). While NO synthesized by neuronal NOS and endothelial NOS has cardioprotective effects through the improvement of perfusion and prevention of apoptosis, the NO produced from iNOS is cardiotoxic because of the inhibition of muscle wall contractility and stimulating apoptosis.

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