

Original Research Article

Dyslipidemia – Could It Be A Reliable Prognostic Factor In Cirrhosis Of Liver? An Approach to Find Out By A Cross Sectional Analytical Study in a Tertiary Care Hospital

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Abstract: Cirrhosis is the end stage consequence of fibrosis of hepatic parenchyma, resulting in nodule formation that may lead to altered hepatic function and blood flow. There are a wide variety of tests to evaluate the severity of liver dysfunction. One simple, non-expensive and reliable test to foretell severity of hepatic injury is the serum lipid profile. The aim was to determine the lipid profile in cirrhotic patients and to assess if there is correlation between the lipid profile derangement and severity of liver dysfunction. 50 cases of cirrhosis were recruited from patients attending the liver clinic in the department of medical gastroenterology in the age group of 30 to 70 years. Patients with Diabetes, renal failure, acute pancreatitis and with history of hyperlipidemia are excluded. 50 age matched healthy controls were selected. Fasting blood sample was collected and lipid profile was done using enzymatic method. Child-Pugh score and The Model for End-Stage Liver Disease, or MELD were used for assessing the severity of liver disease. In our study, it was found that all the four variables (Total cholesterol, Triglycerides, LDL and HDL) were significantly decreased in patients compared to normal subjects. Among these parameters total cholesterol, LDL, HDL correlated inversely well with the severity of liver dysfunction. Since metabolism of serum lipids is predominantly accomplished by liver, it is probable that lipid profile can be used as a laboratory marker of severity of liver damage in cirrhosis, a non-expensive easy test to foretell severity of hepatic injury.

Keywords: lipid profile, cirrhosis, MELD score, child-pugh score, hepatic injury

INTRODUCTION:

Lipids are essential component of biological membranes, free molecules and metabolic regulators that control cellular function and homeostasis. Lipids are integrators of cellular function and intercellular communication. Lipid- lipid and lipid protein interactions regulate cellular physiology [1]. Liver is a dynamic organ characterised by several unique properties, including self-renewal that permits its daily exposure to ingested nutrients, gut-derived endo biotics, and xenobiotic metabolism without adverse consequences. This unique position also confers vulnerability to a wide variety of insults and injury [2].

Liver plays a vital role in lipid metabolism. It contributes both in exogenous and endogenous cycles of lipid metabolism and transport of lipids through plasma. Synthesis of many apolipoproteins takes place in liver.

The apolipoproteins are required for the assembly and structure of lipoproteins. Lipoproteins play an important role in the absorption of dietary cholesterol, long chain fatty acids and fat soluble vitamins. The transport of triglycerides, cholesterol and fat soluble vitamins from the liver to peripheral tissue and transport of cholesterol from peripheral tissue to liver is by lipoproteins. Liver is the principal site of formation and clearance of lipoproteins. Thus in severe liver disease, lipid metabolism is profoundly disturbed. Cirrhosis of the liver is the histologic end point of varied chronic insults resulting in necrosis of the cell followed by fibrosis and nodular regeneration with gross distortion in liver architecture [3]. There are a wide variety of tests to evaluate the severity of liver dysfunction. One simple, non-expensive and reliable test to foretell severity of hepatic injury is the serum lipid profile, more the tissue damage, the more decline in serum lipids.

AIMS AND OBJECTIVES:

To determine the lipid profile in cirrhotic patients and to assess if there is correlation between the lipid profile derangement and severity of liver dysfunction

MATERIALS AND METHODS :

The present Cross Sectional Hospital Based study was conducted in the Department of Biochemistry, in association with Department of Gastroenterology in Stanley Medical College. Cases were recruited from patients attending the liver clinic in the department of medical gastroenterology . 50 Cases in the age group of 30 to 70 years were chosen.

Inclusion Criteria:

The diagnosis of chronic liver disease was based upon clinical features, liver function tests, prothrombin time, ultrasonography, upper gastrointestinal endoscopy. A similar group of 50 healthy persons, age and sex matched were chosen as controls. Serum triglyceride level, Total cholesterol, High density lipoprotein (HDL-C) was then measured in all cases and controls by maintaining the standard protocol. We excluded patients suffering from concomitant diseases, which can alter the lipid profiles, like diabetes mellitus, cancer, acute pancreatitis, and renal failure, patients who were on glucose or lipid lowering drugs.

Collection of 5 ml of fasting blood sample and lipid profile is done using enzymatic method.

Total cholesterol (TC), high-density lipoprotein (HDL-C) cholesterol and triglyceride were assayed by enzymatic reaction and low-density lipoprotein (LDL-C) cholesterol was calculated using by the Friedwald formula. Multiple scores have been created to categorize the severity of disease. The Child-Turcotte-Pugh score is the most widely used. It incorporates three laboratory values (PT, bilirubin, albumin) and two clinical features (ascites and hepatic encephalopathy) [4].

The Model for End-Stage Liver Disease, or MELD, is also a scoring system for assessing the severity of chronic liver disease. MELD uses the patient's values for serum bilirubin, serum creatinine, and the international normalized ratio for prothrombin time (INR) to predict survival. It is calculated according to the following formula,

$$\text{MELD} = 3.78[\text{Ln serum bilirubin (mg/dL)}] + 11.2[\text{Ln INR}] + 9.57[\text{Ln serum creatinine (mg/dL)}] + 6.43$$

MELD model has been widely accepted as a measure of chronic liver disease. It provides an objective, readily available measure for selection of liver transplantation candidates with chronic liver disease [5].

COMPARISON OF LIPID PROFILE IN VARIOUS CATEGORIES OF CIRRHOTIC PATIENTS

Data were analysed by SPSS software 14 version. ANOVA and t-test were used and p value was set to be significant if less than 0.05 all the values are expressed as mean ± S.D.

Table-1: Lipid profile categorised according to child classification

CATEGORY	TOTAL CHOLESTEROL	HDL	LDL	TGL
CHILD A	140.72 ±30.88	41.85 ± 7.58	79.41 ± 32.31	104 ± 31.15
CHILD B	126 ± 35.60	39.41 ± 10.01	66.22 ± 30.58	97.35 ± 60.54
CHILD C	76.75 ± 19.94	27.11 ± 9.87	32.15 ± 22.84	87.41 ± 34.68

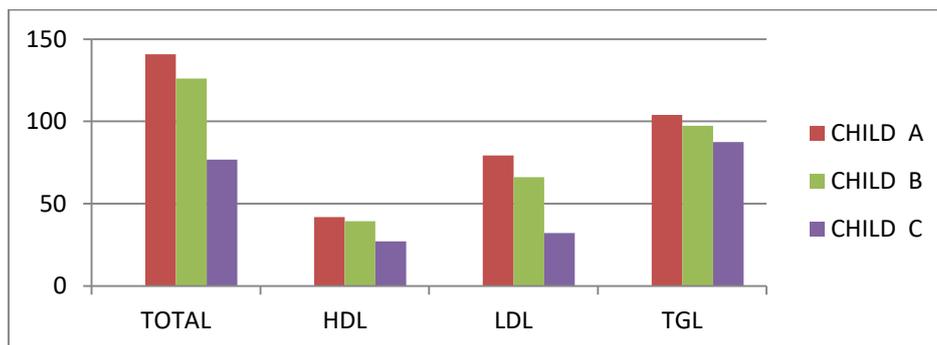


Fig-1: Bar diagram showing lipid profile according to CHILD criteria

Table-2: lipid profile categorised according to meld classification

CATEGORY	TOTAL CHOLESTEROL	HDL	LDL	TGL
MELD ≤ 10	146.08 ± 41.5	42±11.80	86.95±42.05	85.66± 26.03
MELD 11 - 19	125.82± 30.37	40.43±11.39	63.55± 26.97	109.21± 50.62
MELD ≥ 20	75.7±21	25.04±7.89	34.45 ±23.59	81 ±28.83

Table-3: Comparison of lipid profile between cases and controls

LIPID	PATIENTS (mg/dl)	CONTROLS(mg/dl)	p VALUES
LDL	63.59	143.38	p < 0.05
HDL	37.48	47.32	p < 0.05
TOTAL CHOL.	120.66	189.66	p < 0.05
TGL	97.92	152.78	p < 0.05

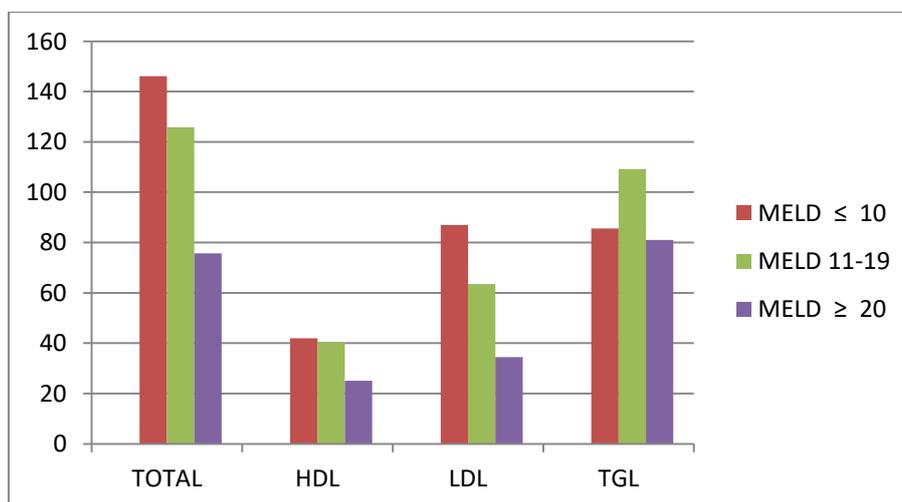


Fig-2: Bar diagram showing lipid profile according to MELD criteria

RESULTS:

In our study, it was found that all the four variables (Total cholesterol, Triglycerides, LDL and HDL) were significantly decreased in patients with

cirrhosis compared to normal subjects. The mean of various lipids like triglycerides, HDL, LDL and total cholesterol for patients versus controls are 97.92/152.78, 37.48/47.32, 63.59/143.38, 120.66/189.66

respectively according to Table 3. Among these parameters total cholesterol, LDL, HDL inversely correlated well with the severity of liver dysfunction. The lipid levels were lower in child pugh's class A and the lowest levels were found in child pugh class C. Similarly, the lowest values of lipids were found in the class of MELD > 20. (Tables 1 & 2)

DISCUSSION:

In chronic liver disease, collagen deposition in the space of Disse leads to defenestration of the sinusoidal endothelial cells (capillarisation of sinusoids) thereby altering exchange between plasma and hepatocytes and resulting in a decreased sinusoidal diameter that is further exacerbated by the contraction of stellate cells [6]. Liver is the main organ responsible for alcohol metabolism. In addition to forming cytotoxic metabolites such as acetaldehyde, ethanol metabolism can alter the cellular oxidation-reduction state thereby modulating liver injury [7]. Alcohol consumption cause fatty liver, alcoholic hepatitis and ultimately, alcoholic cirrhosis in some patients [8-10]. Several studies reveal that different lipid abnormalities are present in different liver diseases e.g. in chronic hepatitis, liver cirrhosis. Marked lipid abnormalities are found in patients suffering from Hepatitis C and HIV coinfection. Regardless of the cause of cirrhosis, the pathologic feature consists of development of fibrosis to the point that there is architectural distortion with the formation of regenerative nodules. These results in a decrease in hepatocellular mass thus function and alteration of blood flow [11].

In our study we found decreased levels of total cholesterol, triglycerides, LDL and HDL in chronic liver disease irrespective of etiology. LDL, HDL and total serum cholesterol levels decrease progressively with cirrhosis advancing from child Pugh A to C [12]. Cirrhotic patients need frequent visits and multiple hospitalizations for management of cirrhosis or its complications. However, choosing the proper treatment plan depends on the severity, type of liver damage and possibility of assessing its extent [13].

CONCLUSION

Since metabolism of serum lipids is predominantly accomplished by liver, it is probable that lipid profile can be used as a laboratory marker of severity of liver damage in cirrhosis, a non-expensive easy test to foretell severity of hepatic injury. It can be used as a reliable test in cirrhotic patients to estimate severity of hepatic damage.

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