e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2024; 16(3); 122-126

Original Research Article

An Observational Study to Assess the Lipid Profile of Decompensated Chronic Liver Disease: A Hospital-Based Assessment

Ujjwal Kumar[1], Rahul Kumar[2]

[1]DM Resident, Department of Gastroenterology, IGIMS, Patna, Bihar, India

[2]DM Resident, Department of Gastroenterology, IGIMS, Patna, Bihar, India

Received: 18-01-2024 / Revised: 22-02-2024 / Accepted: 21-03-2024

Corresponding author: Dr. Rahul Kumar

Conflict of interest: Nil

Abstract

Aim: The aim of the present study was to assess the lipid profile of Decompensated chronic liver disease.

Methods: This observational study was carried out amongst 50 Decompensated chronic liver disease patients that fulfils the inclusion and exclusion criteria and attended the gastroenterology OPD of IGIMS, Patna, Bihar, India.

Results: 43 (86%) were male and 7 (14%) were female. The age range was from 16 to 76. The average age of the patients in the study was 48.72±12.58 yrs. 34% of the patients were between 40 and 50 years of age. 91.67 % of the patients were alcoholic. Abdominal distension and ascites were most common presenting complaints. Pallor was present in 32 (64%) cases. Splenomegaly was present in 24 (48%) patients of Decompensated chronic liver disease. Renal dysfunction was present in 18 (36%) cases. Icterus was present in 12 (24%) cases. 33 (66%) of the patients had decreased platelet count. The comparisons between lipid profile of Decompensated chronic liver disease patients and healthy controls was significant ($p<0.05$).

Conclusion: Lipid abnormalities are commonly seen in patients with Decompensated chronic liver disease and screening for the same is essential for intervention with appropriate treatment to prevent adverse cardiovascular events. The levels of serum total cholesterol, TG, LDL and HDL in patients with Decompensated chronic liver disease are related to the advancement in Decompensated chronic liver disease. It helps in diagnosis of severity of liver disease and also acts as a good prognostic sign.

Keywords: Cholesterol, Decompensated chronic liver disease, High-density lipoproteins, Low density lipoproteins, Lipid profile, Triglyceride

Introduction

Chronic liver disease (CLD) is a term used to describe a wide spectrum of disorders, including idiopathic, infectious, genetic, drug-induced, toxin-induced, and autoimmune disorders. [1,2] The common consequence of chronic damage to the liver is cirrhosis. [3] This is characterized by the replacement of normal liver tissue by fibrotic tissue, occurring due to the accumulation of extracellular material, such as type I collagen activated by hepatic stellate cells and myofibroblasts. [2]

The result is progressive liver dysfunction and clinical complications including portal hypertension (HTN), hepatocellular carcinoma (HCC), liver failure which is characterized by decompensation events such as ascites, variceal bleeding, encephalopathy, spontaneous bacterial peritonitis, and hepato-renal syndrome, eventually leading to death. [3,4] CLD is responsible for approximately 2 million deaths per year worldwide. Most deaths occur due to the complications of cirrhosis and HCC, making them the 11th and 16th most common causes of death, respectively. [5]

In healthy people, a dynamic balance is maintained between uptake, synthesis, secretion, transformation, and storage of lipids. [6] In ongoing hepatocyte damage, such as progressive fibrosis of the liver, glycogen reserves are reduced, increasing the lipid catabolism [7] and then promoting the development of malnutrition. [8] Patients with chronic hepatitis C virus (HCV) infection often have lower levels of total cholesterol (TC) and low-density lipoprotein-cholesterol (LDLc). [9]

The mechanism regarding how HCV hurts lipid metabolism has not been sufficiently elucidated yet. It has been hypothesized that HCV interferes with the activity of very low-density lipoprotein (VLDL) transporter [10], decreasing the VLDL level and then leading to a reduction of cholesterol synthesis. [11] Additionally, patients with liver cirrhosis usually suffer from hypocholesterolemia and hypotriglyceridemia. [12]

The aim of the present study was to assess the lipid profile of Decompensated chronic liver disease.

Materials and Methods

This observational study was carried out amongst 50 Decompensated chronic liver disease that fulfils the inclusion and exclusion criteria and attended the gastroenterology OPD of IGIMS, Patna, Bihar, India for one year

Inclusion Criteria

All cirrhosis of liver patients irrespective of etiology was above 15 years of age. Decompensated chronic liver disease was diagnosed on the basis of combination of clinical features, hematological profile and radiological imaging. Clinical signs and symptoms were those of portal hypertension, i.e. ascites and/or gastrointestinal varices. Hematological profile included evidence of anemia and thrombocytopenia. Radiological features, with transabdominal ultrasound, had to demonstrate a small shrunken liver with or without splenomegaly and intra-abdominal varices. [13,14]

Exclusion Criteria

Patients suffering from concomitant diseases, which can alter the lipid profiles, like diabetes mellitus, malignancy, chronic renal disease, acute pancreatitis, recent parenteral nutrition, history of hyperlipidemia, patients who were on glucose or lipid lowering drugs, patient on immunosuppressive drug were excluded.

After due consideration into inclusion and exclusion criteria, detailed history and clinical examination was undertaken in all subjects. Patients were subjected to routine investigation and fasting lipid profile test. Routine test included complete blood count (CBC) (Sysmex XS-800i), renal function test, liver function test, HBsAg, HCV antibody, Ultrasonography of whole abdomen. A fasting serum lipid profile included serum cholesterol, triglyceride, HDL and LDL cholesterol on Fully Automated analyzer (Erba EM360).

In the present study, anemia was defined using the World Health Organization definition hemoglobin (Hb) concentration <12g/dl (females), <13g/dl (males).The severity of anemia was classified as mild anemia (Hb concentration between 11-12.9g/dl for males and 11- 11.9g/dl for females); moderate anemia (Hb concentration between 8-10.9g/dl), and severe anemia (Hb concentration <8g/dl).10 Thrombocytopenia was defined with a value <150×103/µl.

Statistical Analysis

All the data were entered on excel spreadsheet, and statistical analyses were made using SPSS version 21.0 software. Results were expressed in average + SD, frequencies and percentages. Continuous data were compared using Student's t-test. A p-value <0.05 was considered as statistically significant for all tests conducted.

Results

Table 1: Gender distribution

	Male	Female	Total
N	43	7	50
%	86	14	100
Mean age±SD	48.62±12.44	47.13±15.35	48.72±12.58

43 (86%) were male and 7 (14%) were female. The age range was from 16 to 76. The average age of the patients in the study was 48.72±12.58 yrs.

Table 2: Age distribution

Age group	Male	Female	Total	%
<20	1	1	2	4
21-30	4	0	4	8
31-40	6	0	6	12
41-50	14	3	17	34
51-60	7	2	9	18
61-70	9	1	10	20
71-80	2	0	2	4
Total	43	7	50	100

34% of the patients were between 40 and 50 years of age.

Table 3: Etiology of Decompensated chronic liver disease and symptoms and signs

Etiology	N	%
Alcoholic	45	90
Non-alcoholic	5	10
Hepatitis B	0	0
Hepatitis C	0	0
Total	50	100
Symptoms/ signs		
Pallor	32	64
Icterus	12	24
Edema	16	32
Asteriaxis	1	2
Ascites	41	82
Encephalopathy	0	0
Breathlessness	3	6
Hematemesis	3	6
Malena	4	8
Splenomegaly	24	48
Renal dysfunction/ decreased GFR	18	36
Abdominal distension	47	94

91.67 % of the patients were alcoholic. Abdominal distension and ascites were most common presenting complaints. Pallor was present in 32 (64%) cases. Splenomegaly was present in 24 (48%) patients of Decompensated chronic liver disease. Renal dysfunction was present in 18 (36%) cases. Icterus was present in 12 (24%) cases.

Table 4: Platelet levels in Decompensated chronic liver disease

Platelet count (lakh per ml)	N	%
<1.5 (Thrombocytopenia)	33	66
1.5-4.5	16	32
>4.5	1	2
Total	50	100

33 (66%) of the patients had decreased platelet count.

Table 5: Laboratory parameters of Decompensated chronic liver disease

Laboratory parameters	Decompensated chronic liver disease (Mean±SD)
Haemoglobin	7.95±2.21
RBC (M=4.7-6.1and F= 4.2-5.4)	3.15±0.94
MCV(80-100)	76.51±12.29
PCV(M=40.7-50.3 and F=36.1-44.3)	23.90±7.05
MCH (27-31)	25.34±3.75
MCHC (33.4-35.5)	33.13±6.24
RDW (11.5-14.5)	17.27±2.46
TPC (1.5-4.5)	1.43±0.83
Serum bilirubin total	4.07± 6.10
Serum bilirubin direct	1.97±2.79
SGPT	68.12±45.22
SGOT	84.68±60.29
ALP	132.79±85.69
Blood urea (10-40 mg/dL)	44.71±56.08
Serum creatinine(0.5-1.3 mg/dL)	1.58±1.59

The result showed different laboratory parameters of Decompensated chronic liver disease.

Table 6: Comparisons between lipid profile of Decompensated chronic liver disease

Indices	Decompensated chronic liver disease (MEAN±SD)	p-value
Serum cholesterol	123.94±37.15	<0.05
Serum triglyceride	111.71±51.25	<0.05
HDL	35.91±13.91	<0.05
LDL	71.50±26.30	<0.001

The comparisons between lipid profile of Decompensated chronic liver disease patients and healthy controls were significant ($p<0.05$).

Discussion

Lipids are vital constituent of biological membranes and it is a part of free molecules and metabolic regulators. This controls cellular function and homeostasis in the body. [15] Liver acts as a central character in lipid metabolism. It contributes both in endogenous and exogenous cycles of lipid metabolism and transport of lipids through plasma. The apolipoproteins are essential for assembling and structuring of lipoproteins. Lipoproteins take part in a vital 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 transport of cholesterol from peripheral tissue to liver is by lipoproteins. Apolipoproteins stimulate enzymes which are important in lipoprotein metabolism and to mediate the binding of lipoproteins to cell surface receptors.

Liver is the primary site of formation and clearance of lipoproteins. Hence, liver is involved in various steps of lipid metabolism and lipid transport. Thus, lipid metabolism is greatly disturbed in severe liver disease which is affected in an array of ways. Dyslipidemia found in chronic liver disease differs from secondary dyslipidemia associated with other etiologies, because circulating lipoproteins are not only present in abnormal amount; but they also often have abnormal composition, appearance and electrophoretic mobility. [16] 43 (86%) were male and 7 (14%) were female. The age range was from 16 to 76. The average age of the patients in the study was 48.72±12.58 yrs which was comparable with study by Suthar et al [17] (41 years), Sarin et al [18] (43±8.7 years). 34% of the patients were between 40 and 50 years of age. 91.67 % of the patients were alcoholic. Abdominal distension and ascites were most common presenting complaints. Pallor was present in 32 (64%) cases. Splenomegaly was present in 24 (48%) patients of Decompensated chronic liver disease. Renal dysfunction was present in 18 (36%) cases. Icterus was present in 12 (24%) cases. In previous studies also ascites was common finding Suthar et al [17] (60%), Pathak et al [19] (57.5%), Mendenhall (50.9%). [20]

Cardiovascular disease (CVD) risk stratification includes serum lipid profile. It is infrequently considered a useful screening tool for the assessment of liver diseases; however there is reason to think otherwise. [21] Many previous studies concluded an inverse association of lipid parameters such as total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) with severity of liver disease. However, some other studies did not find such correlation especially for the TG and HDL levels. [22,23] Further, Sen et al [24] showed that TC, HDL and TG were higher in grade 3 fatty liver. 33 (66%) of the patients had decreased platelet count. The comparisons between lipid profile of Decompensated chronic liver disease patients and healthy controls was significant ($p<0.05$). A study by Rosario Gonzale Z, et al, showed that anemia in CLD patients were 75%. [25] Macrocytic anemia was more common in males than females. Microcytic hypochromic anemia was predominant in Decompensated chronic liver disease patients. This may be due to the low socioeconomic and poor nutritional status of most of the cases. According to interesting article by Tody L Kujovich MD – "Haemostatic defects in end stage liver disease", critical care clinics [26] (2005), mild to moderate thrombocytopenia occurs in 49 to 64% of patients with decompensated chronic liver disease (DCLD).

Conclusion

Lipid abnormalities are commonly seen in patients with Decompensated chronic liver disease and screening for the same is essential for intervention with appropriate treatment to prevent adverse cardiovascular events. The levels of serum total cholesterol, TG, LDL and HDL in patients with Decompensated chronic liver disease are related to the advancement in Decompensated chronic liver disease. It helps in diagnosis of severity of liver disease and also acts as a good prognostic sign.

References

1. El-Kabbany ZA, Hamza RT, Ibrahim SA, Mahmoud NH. Dyslipidemia and hyperinsulinemia in children and adolescents with chronic liver disease: relation to disease severity. Int J Adolesc Med Health. 2014;26 (2):195-201.
2. Fujii H, Kawada N. Fibrogenesis in alcoholic liver disease. World J Gastroenterol. 2014 Jul 7;20(25):8048-54.
3. Chang PE, Wong GW, Li JW, Lui HF, Chow WC, Tan CK. Epidemiology and Clinical

Evolution of Liver Cirrhosis in Singapore. Ann Acad Med Singap. 2015 Jun;44(6):218-25.
4. Wigg AJ, McCormick R, Wundke R, Woodman RJ. Efficacy of a chronic disease management model for patients with chronic liver failure. Clin Gastroenterol Hepatol. 2013 Jul;11(7):850-8.e1-4.
5. Asrani SK, Devarbhavi H, Eaton J, Kamath PS. Burden of liver diseases in the world. J Hepatol. 2019 Jan;70(1):151-171.
6. Kroon PA, Powell EE. Liver, lipoproteins and disease: i. Biochemistry of lipoprotein metabol ism. J Gastroenterol Hepatol. 1992;7(2):214–224.
7. Jeon S, Carr R. Alcohol effects on hepatic lipid metabolism. J Lipid Res. 2020;61(4):470–479.
8. Palmer LB, Kuftinec G, Pearlman M, et al. Nutrition in Cirrhosis. Curr Gastroenterol Rep. 2019;21(8):38.
9. Bassendine MF, Sheridan DA, Felmlee DJ, et al. HCV and the hepatic lipid pathway as a potential treatment target. J Hepatol. 2011;55 (6):1428–1440.
10. André P, Perlemuter G, Budkowska A, et al. Hepatitis C virus particles and lipoprotein metabolism. Semin Liver Dis. 2005;25(1):93–104.
11. Aizawa Y, Seki N, Nagano T, et al. Chronic hepatitis C virus infection and lipoprotein metabolism. World J Gastroenterol. 2015;21 (36):10299–10313.
12. Halsted CH. Nutrition and alcoholic liver disease. Semin Liver Dis. 2004;24(3):289–304
13. Nagula S, Jain D, Groszmann RJ, Garcia-Tsao G. Histological-hemodynamic correlation in cirrhosis—a histological classification of the severity of cirrhosis. Journal of hepatology. 20 06 Jan 1;44(1):111-7.
14. European Association For The Study Of The Liver. EASL Clinical Practice Guidelines: management of cholestatic liver diseases. Journal of hepatology. 2009 Aug 1;51(2):237-67.
15. Chiang JY. Nuclear receptor regulation of lipid metabolism: potential therapeutics for dyslipidemia, diabetes, and chronic heart and liver diseases. Current opinion in investigational drugs (London, England: 2000). 2005 Oct;6(10):994-1001.
16. Mehboob F, Ranjha FA, Masud S. Changes in Serum Lipid Profile Among Patients Suffering From Chronic Liver Disease. Annals of King Edward Medical University. 2007;13(3):209.
17. Suthar H, Suthar K, Mewada B. Clinical profile of cases of alcoholic liver disease.
18. Sarin SK, Dhingra N, Bansal A, Malhotra S, Guptan R. Dietary and nutritional abnormalities in alcoholic liver disease: a comparison with chronic alcoholics without liver disease. American Journal of Gastroente -rology (Springer Nature). 1997 May 1;92 (5).
19. Pathak OK, Paudel R, Panta OB, Giri BR, Adhikari B. Retrospective study of clinical profile and prognostic indicators in patients of alcoholic liver disease admitted to a tertiary care teaching hospital in western Nepal. Saudi J Gastroenterol. 2009; 15(3):172-5.
20. Mendenhall CL. Alcoholic hepatitis. Clin Gastroenterol. 1981;10:417-41.
21. Jiang ZG, Mukamal K, Tapper E, Robson SC, Tsugawa Y. Low LDL-C and high HDL-C levels are associated with elevated serum transaminases amongst adults in the United States: a crosssectional study. PloS One. 2014; 9(1):e85366.
22. Mandal SK, Sil K, Chatterjee S, Ganguly J, Chatterjee K, Sarkar P, et al. A study on lipid profiles in chronic liver diseases. Nat J Med Res. 2013;3:70-2.
23. Ghadir MR, Riahin AA, Havaspour A, Nooranipour M, Habibinejad AA. The relationship between lipid profile and severity of liver damage in cirrhotic patients Hepat Mon. 2010;10:285-8.
24. Sen A, Kumar J, Misra RP, Uddin M, Shukla PC. Lipid profile of patients having non-alcoholic fatty liver disease as per ultrasound findings in north Indian population: A retrospective observational study. J Med Allied Sci. 2013;3:59.
25. Gonzalez-Casas R, Jones EA, MorenoOtero R. Spectrum of anemia associated with chronic liver disease. World J Gastroenterol. 2009;15 (37):4653-8.
26. Kujovich JL. Hemostatic defects in end stage liver disease. Crit Care Clin. 2005;21(3):563-87.