

Original Research Article

DYSLIPIDEMIA IN CHRONIC LIVER DISEASE PATIENTS: A HOSPITAL-BASED OBSERVATIONAL STUDY

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ABSTRACT

Background: Chronic liver disease (CLD) is associated with metabolic disturbances, including dyslipidemia, which may contribute to cardiovascular morbidity and disease progression. Alterations in lipid metabolism occur due to impaired hepatic function, and their extent often correlates with the severity of liver disease. This study aimed to assess the prevalence and patterns of dyslipidemia in patients with CLD and to evaluate its association with Child-Pugh score, comorbidities, and clinical outcomes.

Materials and Methods: This observational cross-sectional study was conducted in the Department of General Medicine, GMERS Medical College, Valsad, from March 2023 to January 2025. A total of 150 patients aged ≥18 years with confirmed CLD were included. Patients on lipid-lowering therapy, those with dietary restrictions, those receiving immunosuppressants, or those who had undergone liver transplantation were excluded. Demographic, clinical, and laboratory data were recorded using a structured proforma. Fasting lipid profile, liver function tests, and Child-Pugh classification were assessed. Statistical analysis was performed using Epi Info CDC version 7, with p < 0.05 considered significant.

Results: The mean age of patients was 45.9 ± 12.9 years, with most aged 30-50 years (58.0%); males predominated (75.3%). Diabetes (41.3%) and hypertension (40.7%) were common comorbidities. Ascites (48.7%) and organomegaly (48.0%) were the most frequent clinical features. Alcohol consumption was the predominant risk factor (80.0%). Laboratory findings revealed anemia (mean hemoglobin level 10.8 g/dL), thrombocytopenia (platelet count $98,693/\mu\text{L}$), hypoalbuminemia (albumin level 2.42 g/dL), elevated transaminases, hyperbilirubinemia, and prolonged INR (international normalized ratio 2.43). Dyslipidemia was present in 52.7% of patients, with LDL abnormalities being the most frequent (44.7%), followed by elevated triglycerides (16.7%) and cholesterol levels (12.0%), while HDL levels were preserved. More than half of patients were Child-Pugh class C (54.7%). Dyslipidemia was more common among males (82.3%, p=0.03), whereas hypertension was higher in the non-dyslipidemic group (50.7% vs. 31.6%, p=0.014). No significant association was found between dyslipidemia and Child-Pugh class (p = 0.16).

Conclusion: Dyslipidemia was prevalent in more than half of CLD patients, predominantly characterized by LDL abnormalities. Although no significant association was found with Child-Pugh class, male gender was significantly related to dyslipidemia, while hypertension appeared more common among non-dyslipidemic patients. Routine lipid evaluation in CLD patients is essential for comprehensive management and risk stratification.

Keywords: Chronic liver disease; Dyslipidemia; Child-Pugh score; Lipid profile; Comorbidities; Liver function tests.

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INTRODUCTION

Chronic liver disease (CLD) is a progressive condition characterized by persistent inflammation, fibrosis, and eventually cirrhosis. It is frequently caused by chronic viral hepatitis, alcoholic liver disease, non-alcoholic fatty liver disease (NAFLD), and autoimmune liver disorders. As the key organ for lipid metabolism, the liver is responsible for lipid synthesis, storage, and control.[1,2] Dyslipidemia, defined as "abnormal levels of lipids in the bloodstream, is commonly observed in patients with chronic liver disease due to compromised hepatic function. The interplay between liver dysfunction and lipid metabolism abnormalities underscores the clinical significance of studying dyslipidemia in this patient population.[3,4]

The severity and aetiology of liver dysfunction determine the spectrum of dyslipidemia in chronic liver disease. Typically, patients with CLD demonstrate changes in their lipid profiles, such as decreases in total cholesterol, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) levels. The liver's diminished ability to synthesize lipoproteins and metabolize cholesterol contributes to these lipid abnormalities. Dyslipidemia in patients with CLD has clinical implications, as it may contribute to the progression of the disease and the development of complications, including cardiovascular disease, portal hypertension, and hepatocellular carcinoma. [5,6]

The Child-Pugh score, a widely used categorization system for evaluating liver disease severity, categorizes patients into three groups (A, B, and C) based on clinical and laboratory criteria. This score provides information about liver functionality and helps predict outcomes, such as morbidity and mortality. Examining dyslipidemia patterns using the Child-Pugh classification enhances comprehension of how liver dysfunction affects lipid metabolism and aids in risk categorization and management of individuals with chronic liver disease (CLD).^[7,8]

Patients with CLD who have dyslipidemia may experience additional problems in addition to liver dysfunction. For example, changes in lipid profiles have been associated with a higher risk of events, cardiovascular coagulopathies, infections. Further aggravating liver damage, the imbalance in lipid homeostasis may also impact oxidative stress and inflammatory processes. Therefore, determining the frequency and pattern of dyslipidemia in patients with CLD is crucial to creating all-encompassing care plans meant to enhance therapeutic results.[9,10]

Comprehending the correlation between mortality and dyslipidemia in chronic liver disease is crucial from a clinical standpoint. The emergence of comorbidities such as variceal haemorrhage, ascites, encephalopathy, and multi-organ failure frequently affects mortality in CLD. Targeted therapies and better prognostication for this patient population may

result from the identification of dyslipidemia as a possible risk factor for mortality. Additionally, some of the adverse effects linked to chronic liver disease may be lessened by treating dyslipidemia with treatment measures.[11,12]

Dyslipidemia is a frequent metabolic abnormality in chronic liver disease (CLD), contributing to disease progression and adverse outcomes such as atherosclerosis, cardiovascular, and cerebrovascular events, while also aggravating glucose metabolism disturbances. The present study aimed to estimate the prevalence and patterns of dyslipidemia in patients with chronic liver disease and to evaluate its association with the Child-Pugh score, comorbidities, and clinical outcomes

MATERIALS AND METHODS

Study Setting, Type, and Design: The present study was conducted in the Department of General Medicine at GMERS Medical College and Civil Hospital, Valsad, Gujarat. The study was designed as an observational cross-sectional study and was conducted over a period from March 2023 to January 2025.

Study Participants: All patients diagnosed with chronic liver disease admitted to the inpatient department during the study period were considered eligible for participation. The inclusion criteria comprised adults aged 18 years and above with a confirmed diagnosis of chronic liver disease. Patients were excluded if they were pregnant, had undergone liver transplantation, were following lipid-lowering restrictions, were on lipid-lowering medications, or were receiving steroids or immunosuppressants.

Sample Size and Sampling Technique: The minimum required sample size was calculated using the hypothesis testing method based on the formula: $n = \frac{Z^2 p (1-p)}{L^2}$ where Z is the standard normal variate at 95%

$$n = \frac{Z^2 p (1-p)}{L^2}$$

confidence interval (1.96), p was the prevalence of dyslipidemia in chronic liver disease (80% as per Rashid M et al. [13]), 1-p was 20%, and L was the margin of error set at 7%. Considering a 10% nonresponse rate, the calculated sample size was 150. A purposive sampling technique was used to recruit all eligible participants.

Data Collection: A structured pro forma was used to record demographic and clinical details, including age, sex, medical history, and etiology of chronic liver disease. Laboratory investigations included fasting lipid profile parameters such as total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL). Venous blood samples of approximately 10 mL were collected from the cubital fossa following a minimum fasting period of 12 hours. Samples were centrifuged at 5000 rpm for 10 minutes within two hours of collection, and serum was stored at -40°C before

analysis, which was performed on the same day to ensure accuracy. The severity of liver disease was categorized using the Child-Pugh scoring system into grades A, B, or C. The outcome measures included the prevalence and patterns of dyslipidemia, as well as the complications associated with it.

Ethical Issues: Before enrolment, all participants were provided with a participant information sheet (PIS) in a language they could understand. Written informed consent was obtained from all participants. The study protocol was reviewed and approved by the Institutional Ethics Committee (IEC).

Data Analysis: Data entry and statistical analysis were performed using Epi Info CDC version 7 software. Continuous variables were expressed as mean and standard deviation, while categorical variables were summarized as proportions. A p-value of less than 0.05 was considered statistically significant.

RESULTS

[Table 1] presents the baseline characteristics of the study population. The majority of patients were aged 30-50 years (58.0%), with a mean age of 45.9 years. Males predominated (75.3%) compared to females (24.7%). Among comorbidities, diabetes mellitus (41.3%) and hypertension (40.7%) were the most prevalent, while ascites (48.7%), organomegaly (48.0%), and black stools (47.3%) were the most common clinical features. Alcohol consumption was the predominant risk factor (80.0%), followed by both alcohol and viral hepatitis (8.0%), with only a small proportion having viral hepatitis alone (0.7%). [Table 2] shows the laboratory investigations of the participants. Hemoglobin was reduced with a mean of 10.8 g/dL, and platelet counts were also low (mean 98,693/µL). Albumin levels were decreased (mean

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2.42 g/dL), indicating impaired liver function. Liver enzyme derangements were observed with SGOT (94.9 U/L) being higher than SGPT (57.3 U/L). Bilirubin levels were elevated (direct 3.15 mg/dL, indirect 1.64 mg/dL), and INR was prolonged (mean 2.43). In terms of lipid profile, total cholesterol (168.9 mg/dL), triglycerides (112.4 mg/dL), and LDL (102.6 mg/dL) were mildly elevated, while HDL was within normal range (51.5 mg/dL).

[Table 3] highlights the overall prevalence and patterns of dyslipidemia. Dyslipidemia was present in more than half of the patients (52.7%). LDL abnormality was the most frequent pattern (44.7%), followed by elevated triglycerides (16.7%) and abnormal cholesterol levels (12.0%). In contrast, HDL levels remained within normal limits in all patients (100%). Regarding disease severity, over half of the participants were in Child-Pugh class C (54.7%), with smaller proportions in class B (29.3%) and class A (16.0%).

[Table 4] presents the associations between dyslipidemia and Child-Pugh score, demographic variables, comorbidities, and risk factors. Although a higher proportion of patients with dyslipidemia were in Child-Pugh class C (62.0%), the association was not statistically significant (p=0.16). Males had significantly more dyslipidemia (82.3%) compared to females (17.7%), whereas hypertension was more frequent among those without dyslipidemia (50.7% vs 31.6%, p=0.014). Other comorbidities such as diabetes mellitus (35.4% vs 47.9%), CVD (21.5% vs 28.2%), stroke (13.9% vs 14.1%), and HRS (11.4% vs 15.5%) showed no significant differences. Similarly, etiological factors like alcoholism (75.9% vs 84.5%), viral hepatitis (1.3% vs 0.0%), and combined alcohol plus viral etiology (10.1% vs 5.6%) did not differ significantly between groups.

Variables	n (%)
Age (years)	
• <30	11 (7.3)
• 30–50	87 (58.0)
• >50	52 (34.7)
Gender	
• Male	113 (75.3)
• Female	37 (24.7)
Comorbidities	
• Diabetes mellitus	62 (41.3)
Hypertension	61 (40.7)
Cardiovascular disease	37 (24.7)
• Stroke	21 (14.0)
Hepatorenal syndrome	20 (13.3)
Clinical Features	
• Ascites	73 (48.7)
 Organomegaly 	72 (48.0)
Black stools	71 (47.3)
Pedal edema	70 (46.7)
Coagulopathy	42 (28.0)
Risk Factors	
• Alcoholic	120 (80.0)
• Viral	1 (0.7)
• Both	12 (8.0)
• None	17 (11.3)

Table 2: Laboratory Investigations of Study Participants (n=150)

Parameter	Mean ± SD	Range
Hemoglobin (g/dL)	10.80 ± 2.68	4.0–17.0
Platelet count (/μL)	$98,693.22 \pm 70,272.79$	10,100-484,000
Albumin (g/dL)	2.42 ± 1.96	0.7–23.0
SGOT (U/L)	94.89 ± 48.28	22–422
SGPT (U/L)	57.35 ± 32.84	18–290
ALP (U/L)	143.79 ± 101.37	55–1300
Direct bilirubin (mg/dL)	3.15 ± 2.83	0.3–20.1
Indirect bilirubin (mg/dL)	1.64 ± 1.85	0.1–18.1
INR	2.43 ± 0.92	0.8-5.0
LDL (mg/dL)	102.58 ± 23.25	57–198
Triglycerides (mg/dL)	112.41 ± 33.00	70–192
HDL (mg/dL)	51.53 ± 10.12	25–76
Total cholesterol (mg/dL)	168.86 ± 26.92	80–218

Table 3: Dyslipidemia in Chronic Liver Disease Patients (n=150)

Dyslipidemia	n (%)
Overall dyslipidemia	
• Present	79 (52.7)
• Absent	71 (47.3)
Pattern of Dyslipidemia	
Abnormal LDL	67 (44.7)
Abnormal TG	25 (16.7)
Abnormal HDL	0 (0.0)
Abnormal cholesterol	18 (12.0)
Child-Pugh Score	
• A	24 (16.0)
• B	44 (29.3)
• C	82 (54.7)

Table 4: Factors associated with Dyslipidemia (n=150)

Variables	Dyslipidemia	Dyslipidemia	
	Present (n=79)	Absent (n=71)	
Child-Pugh Score			
• A	11 (13.9)	13 (18.3)	0.16
• B	19 (24.0)	25 (35.2)	
• C	49 (62.0)	33 (46.5)	
Age group			
• <30	2 (2.5)	9 (12.7)	0.057
• 30–50	49 (62.1)	38 (53.5)	
• >50	28 (35.4)	24 (33.8)	
Gender			
• Male	65 (82.3)	48 (67.6)	0.03*
Female	14 (17.7)	23 (32.4)	
Comorbidities	, , ,		
Diabetes mellitus	28 (35.4)	34 (47.9)	0.08
Hypertension	25 (31.6)	36 (50.7)	0.014*
• CVD	17 (21.5)	20 (28.2)	0.22
• Stroke	11 (13.9)	10 (14.1)	0.58
• HRS	9 (11.4)	11 (15.5)	0.31
Risk Factors			
Alcoholic	60 (75.9)	60 (84.5)	0.19
• Viral	1 (1.3)	0 (0.0)	0.34
• Both	8 (10.1)	4 (5.6)	0.31
• None	10 (12.7)	7 (9.9)	0.58

^{*}Significant at p < 0.05

DISCUSSION

Chronic liver disease (CLD) continues to be a pressing global health problem, characterized by progressive hepatic dysfunction and multiple systemic complications. In this study, demographic, clinical, biochemical, and metabolic features of CLD patients were assessed, with special attention to dyslipidemia and its relationship with disease severity.

Most patients in this cohort fell within the middle-aged bracket of 30–50 years (58.0%), followed by those older than 50 years (34.7%), with a minority being younger than 30 years (7.3%). The mean age of 45.9 years indicates that CLD chiefly affects the working-age population. Similar age clustering was documented by Mandal SK et al.,^[14] who found 38.75% of males between 41 and 50 years old, and by Muhammed HP et al.,^[15] who observed 67.3% of cases between 30 and 60 years old. On the other hand, Bassani L et al,^[16] reported a higher concentration above 50 years (85.6%), whereas Irfan S et al,^[17]

described nearly equal distribution between younger patients (52.6%) and older patients (47.4%). Thus, the current study reinforces the evidence that CLD is primarily a disease of middle-aged and older adults, though regional variations exist. A clear male predominance was also evident, with 75.3% of the sample being male. This aligns closely with findings from Verma AK,^[18] (71.2%), Pandey T,^[19] (75.0%), and Muhammed HP (72.5%), all of whom reported higher male vulnerability, most likely explained by greater alcohol consumption among men in these populations.

The present study demonstrated a high burden of comorbidities, with diabetes mellitus in 41.3% and hypertension in 40.7% of cases. These rates are remarkably similar to those reported by Verma AK,[18] (38.5% and 36.2%) and Pandey T,[14] (44.8% and 39.5%). Cardiovascular disease affected roughly one-quarter of patients (24.7%), while stroke (14.0%) and hepatorenal syndrome (13.3%) were less frequent. Comparable frequencies have been noted by Irfan S,[17] and Mandal SK,[14] suggesting that metabolic and vascular complications are common accompaniments of CLD. Symptomatically, ascites (48.7%), organomegaly (48.0%), black stools (47.3%), and pedal edema (46.7%) dominated the clinical presentation, while coagulopathy was seen in 28.0%. These features mirror the observations of Verma AK,[18] and Pandey T,[19] who also described ascites and pedal edema in nearly half their patients. The etiological profile was heavily skewed toward alcohol consumption (80.0%), with only a small fraction due to viral hepatitis (0.7%) or mixed etiology (8.0%). This pattern is almost identical to Pandey T,[19] (81.5% alcohol-related) and Verma AK,[18] (76.0%), underscoring alcohol as the major driver of CLD in Indian cohorts.

With over half the patients categorized as Child-Pugh C (54.7%), the study depicts advanced disease at presentation, while 29.3% and 16.0% were in classes B and A, respectively. These proportions are nearly parallel to those of Verma AK, [18] (50.3% C, 31.5% B) and Pandey T,^[19] (55.2% C, 30.2% B). The biochemical profile also reflected severe hepatic compromise. Mean hemoglobin was reduced to 10.8 g/dL, and platelet counts were markedly low at 98,693/µL. Albumin levels were depressed (2.42) g/dL), while liver enzymes were elevated, with SGOT at 94.9 U/L and SGPT at 57.3 U/L. Bilirubin levels were raised (direct 3.15 mg/dL, indirect 1.64 mg/dL), and INR was prolonged at 2.43. These derangements closely match previous research, which documented similar trends in liver function markers, highlighting the biochemical signatures of progressive cirrhosis. [18,19]

Dyslipidemia was detected in just over half of the study population (52.7%). This prevalence was comparable to Verma AK,^[18] (54.3%), Pandey T,^[19] (50.8%), and Bassani L,^[16] (48.7%), though lower than the 83.6% reported by Irfan S. LDL abnormalities were most common (44.7%), followed by triglyceride (16.7%) and cholesterol

derangements (12.0%), whereas HDL levels remained normal in all patients. A similar pattern was described by Pandey T, [19] who noted LDL changes in 46.1% and triglyceride abnormalities in 15.7%, while Verma AK, [18] also highlighted LDL as the most consistently altered parameter. In contrast, Irfan et al,^[17] observed significant HDL changes (40.2%), suggesting possible population-specific variations. Although patients in Child-Pugh C had a higher share of dyslipidemia (62.0%), the association was not statistically significant, differing from Irfan S, who linked worsening lipid profiles to advancing disease.[17] Interestingly, gender differences were noted, with dyslipidemia more frequent among males (82.3%), while hypertension was paradoxically more common in the non-dyslipidemic group (50.7% vs 31.6%), indicating a complex interplay of metabolic and vascular factors.

CONCLUSION

The present study demonstrated that dyslipidemia is a frequent metabolic abnormality in patients with chronic liver disease, affecting more than half of the cohort and predominantly characterized by LDL abnormalities, while HDL remained preserved. Although no significant correlation was found between dyslipidemia and Child-Pugh classification, its higher prevalence in advanced disease and significant association with male gender underscores the need for regular lipid monitoring in CLD patients. Given the coexistence of comorbidities such as diabetes and hypertension, comprehensive metabolic evaluation should be integrated into routine care. It is recommended that clinicians incorporate early detection and management of dyslipidemia into treatment strategies for CLD to minimize cardiovascular risks and improve long-term outcomes. Larger multicentric studies are warranted to further establish the prognostic significance of dyslipidemia in this population.

REFERENCES

- Tanwar S, Rhodes F, Srivastava A, Trembling PM, Rosenberg WM. Inflammation and fibrosis in chronic liver diseases including non-alcoholic fatty liver disease and hepatitis C. World J Gastroenterol. 2020 Jan 14;26(2):109-133.
- Sharma A, Nagalli S. Chronic Liver Disease. [Updated 2023 Jul 3]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK554597/
- Kopin L, Lowenstein CJ. Dyslipidemia. Annals of internal medicine. 2017 Dec 5;167(11):ITC81-96.
- Arvanitis M, Lowenstein CJ. Dyslipidemia. Annals of internal medicine. 2023 Jun;176(6):ITC81-96.
- Mikolasevic I, Žutelija M, Mavrinac V, Orlic L. Dyslipidemia in patients with chronic kidney disease: etiology and management. Int J Nephrol Renovasc Dis. 2017 Feb 7;10:35-45.
- Arvind A, Osganian SA, Cohen DE, et al. Lipid and Lipoprotein Metabolism in Liver Disease. [Updated 2019 Jul 21]. In: Feingold KR, Ahmed SF, Anawalt B, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK326742/

- Tsoris A, Marlar CA. Use Of The Child Pugh Score In Liver Disease. [Updated 2023 Mar 13]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-Available from: https://www.ncbi.nlm.nih.gov/books/NBK542308/
- Pugh RN, Murray-Lyon IM, Dawson JL, Pietroni MC, Williams R. Transection of the oesophagus for bleeding oesophageal varices. Br J Surg. 1973 Aug;60(8):646-9.
- Unooque U, Lohano AK, Dahri Q, Arain N, Farukhuddin F, Khadke C, Prince F, Farooque R, Shehata MA, Bin Zafar MD. The Pattern of Dyslipidemia in Chronic Liver Disease Patients. Cureus. 2021 Feb 10;13(2):e13259.
- Dong J, Yang S, Zhuang Q, Sun J, Wei P, Zhao X, Chen Y, Chen X, Li M, Wei L, Chen C, Fan Y, Shen C. The Associations of Lipid Profiles With Cardiovascular Diseases and Death in a 10-Year Prospective Cohort Study. Front Cardiovasc Med. 2021 Nov 25;8:745539.
- 11. Diaz-Soto MP, Garcia-Tsao G. Management of varices and variceal hemorrhage in liver cirrhosis: a recent update. Therap Adv Gastroenterol. 2022 Jun 20;15:17562848221101712.
- 12. Sarin SK, Choudhury A, Sharma MK, Maiwall R, Al Mahtab M, Rahman S, Saigal S, Saraf N, Soin AS, Devarbhavi H, Kim DJ, Dhiman RK, Duseja A, Taneja S, Eapen CE, Goel A, Ning Q, Chen T, Ma K, Duan Z, Yu C, Treeprasertsuk S, Hamid SS, Butt AS, Jafri W, Shukla A, Saraswat V, Tan SS, Sood A, Midha V, Goyal O, Ghazinyan H, Arora A, Hu J, Sahu M, Rao PN, Lee GH, Lim SG, Lesmana LA, Lesmana CR, Shah S, Prasad VGM, Payawal DA, Abbas Z, Dokmeci AK, Sollano JD, Carpio G, Shresta A, Lau GK, Fazal Karim M, Shiha G, Gani R, Kalista KF, Yuen MF, Alam S, Khanna R, Sood V, Lal BB, Pamecha V, Jindal A, Rajan V, Arora V, Yokosuka O, Niriella MA, Li H, Qi X, Tanaka A, Mochida S, Chaudhuri DR, Gane E, Win KM, Chen WT, Rela M, Kapoor D, Rastogi A, Kale P, Rastogi A, Sharma CB, Bajpai M, Singh V, Premkumar M, Maharashi S, Olithselvan A, Philips CA,

- Srivastava A, Yachha SK, Wani ZA, Thapa BR, Saraya A, Shalimar, Kumar A, Wadhawan M, Gupta S, Madan K, Sakhuja P, Vij V, Sharma BC, Garg H, Garg V, Kalal C, Anand L, Vyas T, Mathur RP, Kumar G, Jain P, Pasupuleti SSR, Chawla YK, Chowdhury A, Alam S, Song DS, Yang JM, Yoon EL; APASL ACLF Research Consortium (AARC) for APASL ACLF working Party. Acute-on-chronic liver failure: consensus recommendations of the Asian Pacific association for the study of the liver (APASL): an update. Hepatol Int. 2019 Jul;13(4):353-390.
- Rashid M, Zafar M, Farooque U, Mirza M, Ahmed A, Khalid S. Evaluation of lipid profile in patients with liver cirrhosis: a cross-sectional study. Cureus. 2020;12(10):e11103.
- Mandal SK, Sinha A, Choudhury TA, Saha A, Pal S, Basu A. Lipid profiles in patients with chronic liver disease. Niger Med J. 2013;54(1):5-9.
- Muhammed HP, Jayaraj K. Correlation of lipid profile in patients with severity of liver disease: a cross-sectional study in a tertiary care hospital. Int J Res Med Sci. 2017;5(1):326-9.
- Bassani L, Fernandes SA, Franco JM, Alves VA, Brandão DF, Pereira FE. Lipid profile in patients with liver cirrhosis and its correlation with Child-Pugh and MELD scores. Arq Gastroenterol. 2015;52(2):121-5.
- Irfan S, Bashir S, Karim S, Shah S, Abbasi A. Frequency of dyslipidemia in patients with liver cirrhosis. J Ayub Med Coll Abbottabad. 2014;26(2):174-7.
- 18. Verma AK, Gautam SK, Giri R, Kushwaha JS, Gupta V, Singh R. Study of serum lipid profile in patients with chronic liver disease and its correlation with Child-Pugh class: a hospital-based observational study in North Indian patients. Int J Adv Med. 2023;10(5):388-91.
- Pandey T, Gautam R, Pokhrel S. The study of serum lipid profile in patients with cirrhosis of liver. Asian J Med Sci. 2023;14(6):108-12.