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Original Article

Dyslipidemia and impaired liver function biomarkers in patients with hepatitis B liver cirrhosis

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المخلص

أهداف البحث: أجريت الدراسة لتحديد التغيرات في التمثيل الغذائي للدهون وحالة إنزيم الكبد لمرضى تليف الكبد الإيجابي (الفيروس الكبدي ب).

طرق البحث: تم تضمين ما مجموعه 300 مريض تليف كبدي مصاب بفيروس التهاب الكبد ب و200 عنصر تحكم سليم في دراسة الحالة والشواهد هذه. تم تجنيد المرضى من مختلف مستشفيات الرعاية الثالثية في لاهور من مارس إلى أكتوبر 2021. تم جمع عينات الدم وتحليلها من أجل المستضد السطحي لالتهاب الكبد البائي ومستضد غلاف التهاب الكبد ب والعلامات الحيوية لوظائف الكبد ودهون المصل. تم تأكيد تليف الكبد عن طريق التصوير بالموجات فوق الصوتية وخزعة الكبد.

النتائج: كان من الواضح أن المؤشرات الحيوية لوظائف الكبد في الدم كانت أعلى بشكل ملحوظ، في حين أن مستويات الدهون في الدم كانت أقل بكثير في مرضى تليف الكبد المصابين بفيروس التهاب الكبد ب مقارنة بالضوابط. كشفت الدراسة عن عدم وجود علاقة معنوية بين الجنس والعمر مع عسر شحميات الدم في مرضى تليف الكبد. ارتبط تصنيف درجات تشمع الكبد وتدريجها سلبا بمستويات الكوليسترول الكلي. علاوة على ذلك، لوحظ الجنس والمستويات العالية من إنزيمات الكبد كعوامل خطر كبيرة مرتبطة بخلل شحميات الدم في مرضى تليف الكبد الإيجابي ب. كانت القيم الفاصلة المثلى لأنزيمات الكبد ودهون المصل من أجل تشخيص تليف الكبد أعلى من نطاقاتها الطبيعية.

الاستنتاجات: يمكن الاستنتاج أنه يمكن استخدام تركيزات الدهون في الدم كمؤشر سريري لتقييم تلف الكبد في المرضى المصابين بفيروس التهاب الكبد ب.

الكلمات المفتاحية: عسر شحميات الدم؛ المؤشرات الحيوية لوظائف الكبد؛ فيروس التهاب الكبد ب؛ دهون المصل؛ المستضد السطحي لالتهاب الكبد البائي

Abstract

Objective: This study was conducted to determine changes in lipid metabolism and liver enzyme status among HBV-positive patients with liver cirrhosis.

Methods: A total of 300 HBV-positive patients with liver cirrhosis and 200 healthy controls were included in this case–control study. The patients were recruited from several tertiary care hospitals in Lahore from March to October 2021. Their blood samples were collected and analyzed for HBsAg, HBeAg, liver function biomarkers, and serum lipids. Liver cirrhosis was confirmed by ultrasonography and liver biopsy. The data were analyzed with chi-square test, Student's t-test, logistic regression, and ROC curve analysis.

Results: Serum liver function biomarkers were significantly higher, and serum lipid levels were substantially lower, in HBV-infected patients with liver cirrhosis than in controls. No significant associations of sex and age with dyslipidemia were observed in patients with cirrhosis. Grading and staging scores for liver cirrhosis were negatively associated with total cholesterol levels. Moreover, sex and high levels of liver enzymes were significant risk factors associated with dyslipidemia in HBV-

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positive patients with liver cirrhosis. The optimum cut-off values of liver enzymes and serum lipids for the prognosis of liver cirrhosis exceeded normal ranges.

Conclusion: Serum lipid concentrations may serve as a clinical index to assess liver damage in HBV-positive patients.

Keywords: Dyslipidemia; HbsAg; Hepatitis B virus; Liver function biomarkers; Serum lipids

Introduction

Globally, HBV infection is one of the leading infectious diseases and is associated with high morbidity and mortality.[1] The World Health Organization has estimated that 296 million people were living with chronic hepatitis B (CHB) infection in 2019, and 1.5 million cases occur annually. Cirrhosis and hepatocellular carcinoma are two of the most common complications of CHB.[2]

The etiologies of cirrhosis differ among regions of the world.[3] Most liver cirrhosis in North America is caused by CHB infection, whereas chronic hepatitis C infection is responsible for most liver damage in Asia and the Pacific. Hemochromatosis, Wilson's disease, primary biliary cirrhosis, and autoimmune hepatitis are all considered to cause liver cirrhosis. In rare cases, cryptogenic or idiopathic causes have been found.[4] Despite the multifactorial causes of liver cirrhosis, liver cirrhosis presents several pathological characteristics, including liver function loss and fibrotic tissue replacement.[5] Currently, no proven treatment is available, because the molecular mechanism underlying liver cirrhosis is not fully understood. Therefore, better understanding of the pathogenesis of liver cirrhosis is required to enable the development of more effective treatments.[6]

Chronic liver diseases are associated with changes in serum lipids, because the liver plays a critical role in lipid metabolism. CHB and liver cirrhosis have been reported to be inversely associated with total cholesterol (TC) and high-density lipoprotein (HDL).[7] Different liver diseases impair plasma lipids in various ways.[8,9] CHB and cirrhosis may result in lower liver biosynthetic capacity, thus decreasing plasma triglycerides (TG) and cholesterol.[10] Data from Pakistan regarding impaired serum lipids in patients with HBV-caused liver cirrhosis are lacking.

The current study was performed to delineate the alterations in blood serum lipid profiles and subsequently elucidate the associations of serum lipids with liver enzymes and liver histology in HBV-infected patients with liver cirrhosis in Pakistan.

Materials and Methods

Study participants

A total of 300 HBV-positive patients who visited multiple tertiary care hospitals in Lahore, comprising the Pakistan Kidney & Liver Institute, The University of Lahore Teaching Hospital, Ganga Ram Hospital, Mayo Hospitals, and Sheikh Zayed Hospital, were included in the study regardless of their age and sex. Patients receiving antiviral therapy or blood transfusion, and those who were co-infected with HCV, HAV, or HIV were excluded. A total of 200 age and sex-matched individuals with no history of HBV infection were included in the control group. Individuals with a history of alcohol use, malignancy, or seropositive HBsAg were excluded. Patient medical history of any viral infection, malignancy, cholestatic liver diseases including primary biliary cirrhosis, autoimmune hepatitis, biliary ductal obstruction, primary sclerosing cholangitis, or sarcoidosis, as well as basic demographic features (age and sex), was recorded on questionnaires completed by patients after written consent had been obtained from the patients or their guardians. Patients with a history of cholestatic liver diseases and/or use of hypolipidemic drugs were excluded.

Laboratory measurements

Blood samples (5 ml) from patients and healthy controls who had fasted for 10−14 h were collected under sterilized conditions. HBV positivity was confirmed serologically through detection of HBsAg, HBeAg (ELISA, Abbott Laboratories, USA), and HBV DNA. Liver cirrhosis was diagnosed on the basis of physical signs and symptoms, including weight loss, fatigue, weakness, ascites, asterixis, digital clubbing, and collateral vessels in the abdominal wall, and was confirmed by ultrasonography (Figure S1) and liver biopsy (Figure S2). The grades and stages of biopsy samples were evaluated as described previously.[11] The grading was performed on a scale of 0−18 (modified HAI scale), whereas liver fibrosis staging was determined on a scale of 0−6. The levels of serum biochemical analytes, including TC, TG, HDL, very low-density lipoproteins (VLDL), low-density lipoproteins (LDL), bilirubin, aspartate aminotransferase (AST), alkaline phosphatase (ALP), and alanine transaminase (ALT), were measured with a clinical analyzer (Hitachi 7600, Japan).

Statistical analysis

The mean and standard deviation (SD) were calculated for each parameter. SPSS version 20 (SPSS Inc. Chicago, IL) was used to detect associations. Continuous variables are presented as mean ± SD, whereas categorical variables are described as frequencies or percentages. Student's t-test was applied to compare continous variables, whereas the chi-square test was used to compare categorical variables between the HBV-positive and control groups. Multivariable logistic regression analysis was performed to detect the independent associations of age, sex, and liver function

biomarkers with dyslipidemia in HBV-positive patients with liver cirrhosis. To estimate risk factors, we calculated odds ratios with 95% confidence intervals (CI). The predictive values of the identified risk factors were assessed according to the area under the receiver operating characteristic (ROC) curve. Optimum cut-off values were calculated with Youden's index. A *P*-value less than 0.05 indicated a significant difference.

Results

The baseline characteristics of the study participants are indicated in Table 1. The HBV-positive group comprised 137 (46%) males and 163 (54%) females, whereas the control group comprised 107 (53.5%) females and 93 (46.5%) males. The study participants were divided into five age groups. The mean age was 54.6 ± 13.3 years and 54.3 ± 12.7 years in the HBV-positive and control groups, respectively. Liver cirrhosis and hepatitis B more prevalent in females and patients in the 41−60 year age group. Blood liver function biomarkers (bilirubin, ALT, AST, and ALP) were significantly higher in HBV-positive patients with cirrhosis than in controls. In contrast, serum TC, TG, HDL, LDL, and VLDL levels were markedly lower in HBV-positive patients with cirrhosis than in controls.

Sex and age differences in the predictive value of the lipid profile

Evaluation of variations in serum lipids in relation to the age and sex of the participants revealed no statistical association between sex (Table 2) and dyslipidemia in HBV-positive patients and healthy controls. The predictive values of TC, VLDL, TG, HDL, and LDL were similar in the male and female populations of both groups.

Moreover, a comparison of mean values of serum lipids except HDL indicated no statistical association with the ages of the HBV-positive patients and controls. The level of HDL in HBV-positive patients with liver cirrhosis was low in age group I, followed by age groups V, III, IV, and II (Table S1).

Correlation of LFTs with the lipid profile

The correlation between serum lipids and liver function biomarkers was evaluated through Pearson's correlation. Among all serum lipids, HDL levels showed a strong and significant negative association with bilirubin and liver function biomarkers. LDL levels showed a weak but highly significant positive association with bilirubin and a negative correlation with liver enzymes. Importantly, TC levels were weakly but significantly associated with bilirubin levels but not significantly associated with liver enzymes. VLDL and TG levels had weak, negative, and non-significant associations with bilirubin and liver enzymes except ALP (Table 3).

Liver histology

The mean grading and staging scores for liver biopsy in HBV-positive patients with liver cirrhosis were 10.1 ± 4.7 and 3.4 ± 1.4, respectively. Analysis of the correlations of grading and staging scores with serum lipids and liver function biomarkers was performed with Pearson's rank correlation, which demonstrated positive correlations of grading score with serum lipids (except TC) and all liver function biomarkers (Table 4), and negative correlations of staging score with TC and VLDL. Moreover, none of the correlations between the staging score and serum biochemical analytes were statistically significant, whereas the grading score exhibited a statistically significant negative correlation with only TC.

Table 1: Demographic and clinical features of the study population.

Parameters	Reference Range	Patients (n = 300)	Controls (n = 200)	*P*
Age (years)	−	54.6 ± 13.3	54.3 ± 12.7	0.615
Age group n (%)				
≤20	−	2 (0.7)	2 (1)	0.969
21−40	−	43 (14.3)	29 (14.5)	
41−60	−	162 (54)	112 (56)	
61−80	−	84 (28)	52 (26)	
81−100	−	9 (3)	5 (2.5)	
Sex n (%)				
Male	−	137 (46)	93 (46.5)	0.855
Female	−	163 (54)	107 (53.5)	
LFTs				
Bilirubin	0−1.2 mg/dl	0.7 ± 0.92	0.63 ± 0.34	0.224
AST	10−35 U/L	52.4 ± 123.43	25.4 ± 8.9	<0.001
ALT	9−41 U/L	50.9 ± 70.80	24.2 ± 9.2	<0.001
ALP	30−120 U/L	121.8 ± 87.27	74.8 ± 8.9	<0.001
Lipid profile				
TC	<200 mg/dl	153.7 ± 9.2	190.0 ± 5.6	<0.001
TG	<200 mg/dl	134.8 ± 8.6	145.8 ± 7.3	<0.001
HDL	40−50 mg/dl	22.2 ± 4.4	40.8 ± 6.0	<0.001
LDL	<115 mg/dl	79.6 ± 11.6	101.9 ± 9.6	<0.001
VLDL	~50 mg/dl	29.9 ± 5.8	47.4 ± 4.2	<0.001

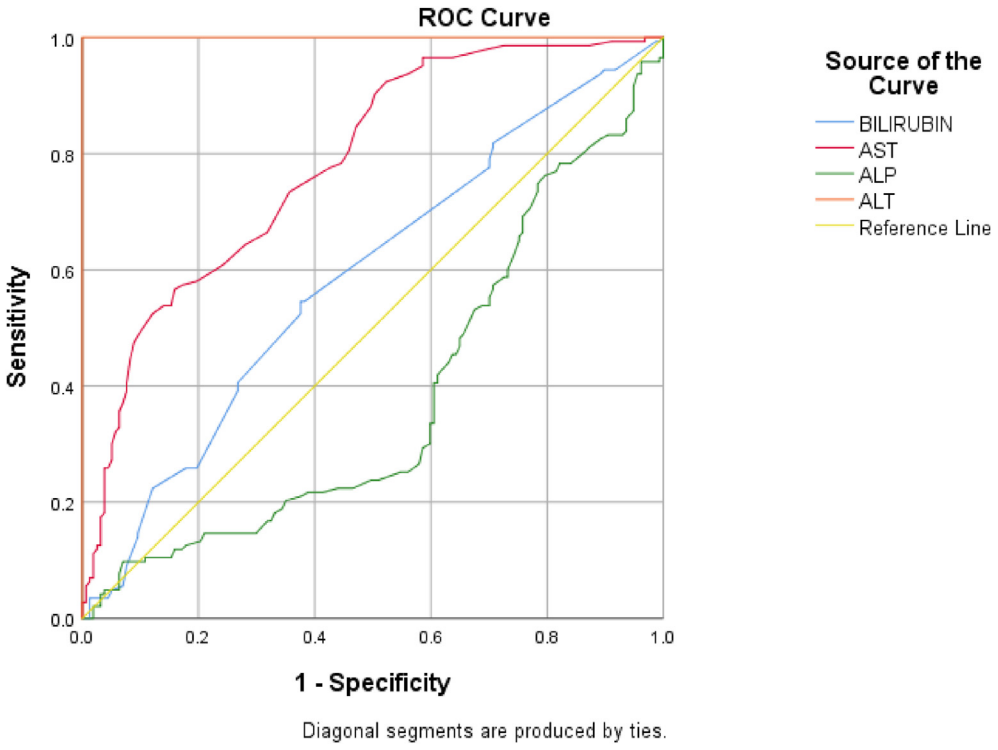

Figure 1: ROC curve analysis of serum liver function biomarkers for HBV-positive patients with liver cirrhosis.

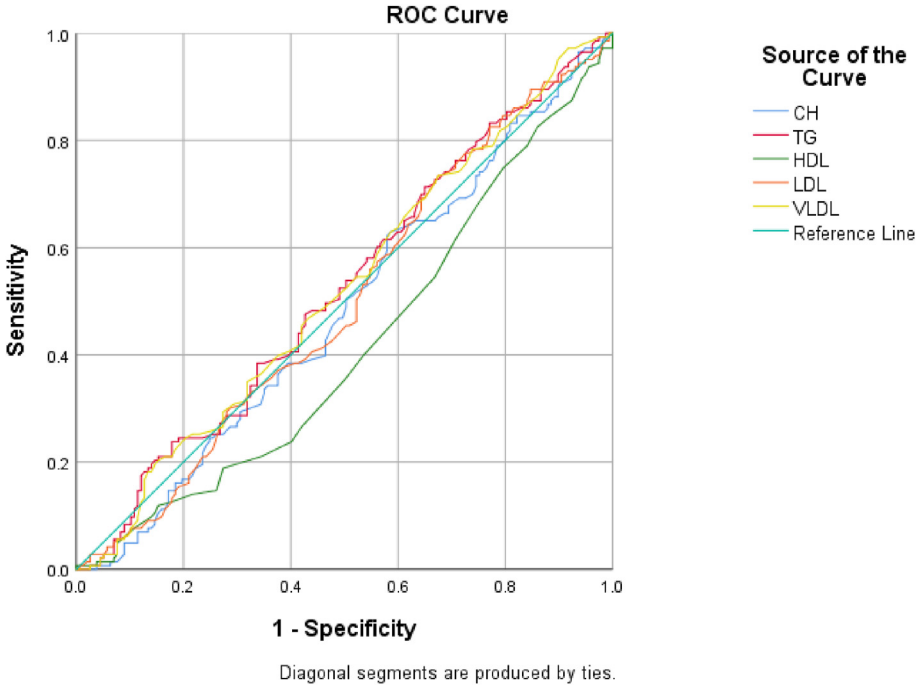

Figure 2: ROC curve analysis of serum lipid enzymes for HBV-positive patients with liver cirrhosis.

Table 2: Sex differences in lipid profiles in HBV-positive patients with cirrhosis and healthy controls.

	HBV-positive Group		Control Group		*P*
	Males	Females	Males	Females	
TC	152.5 ± 8.1	154.6 ± 9.8	190.1 ± 5.3	189.9 ± 5.9	0.630
TG	134.8 ± 8.6	134.8 ± 8.7	146.2 ± 8.2	145.2 ± 8.4	0.572
HDL	22.1 ± 4.3	22.3 ± 4.4	41.2 ± 5.8	40.5 ± 6.2	0.724
LDL	80.8 ± 11.7	78.8 ± 11.5	100.9 ± 9.4	102.3 ± 9.7	0.570
VLDL	29.5 ± 6.1	30.2 ± 5.6	46.8 ± 4.4	47.9 ± 4.1	0.450

Data are presented as mean ± SD.

Table 3: Pearson's correlation between liver enzymes and lipid profiles in HBV-positive patients with cirrhosis.

	Bilirubin		AST		ALT		ALP	
	r	*P*	r	*P*	r	*P*	r	*P*
TC	−0.07	<0.001	−1.42	0.8	−1.47	0.48	0.07	0.67
TG	−0.06	0.17	−0.06	0.96	−0.06	0.98	0.01	<0.001
HDL	−0.21	<0.001	−0.18	<0.001	−0.18	<0.001	−0.03	0.04
LDL	0.038	<0.001	−0.07	<0.001	−0.08	0.16	0.09	0.007
VLDL	−0.01	0.29	−0.06	0.06	−0.05	0.32	0.01	<0.001

Table 4: Correlations of serum lipids and liver function biomarkers with histological variables.

	Grading Score		Staging Score	
	r	*P*	r	*P*
TC	−0.12	0.04	−0.06	0.29
TG	0.02	0.97	0.02	0.76
HDL	0.10	0.08	0.01	0.84
LDL	0.05	0.94	0.04	0.49
VLDL	0.02	0.74	−0.07	0.20
Bilirubin	0.04	0.46	0.05	0.35
AST	0.12	0.04	0.07	0.20
ALP	0.02	0.97	0.06	0.27
ALT	0.08	0.16	0.10	0.07

Table 5: Risk factors associated with dyslipidemia in HBV-positive patients with liver cirrhosis.

Variable	OR	95% CI	*P*
Age	0.978	0.948–0.989	0.805
Sex	0.357	0.199–0.639	0.001*
Bilirubin	0.839	0.595–1.184	0.318
AST	0.878	0.845–0.912	<0.001*
ALP	0.927	0.913–0.941	<0.001*
ALT	0.919	0.897–0.942	<0.001*

OR = odds ratio, CI = confidence interval.

Risk factors associated with dyslipidemia in HBV-positive patients with liver cirrhosis

A multivariate logistic regression analysis was performed to assess the independent risk factors (age, sex, grading score, and staging score) associated with the impaired serum lipid profiles in HBV-positive patients with liver cirrhosis. The analysis revealed that sex and high levels of serum ALT, AST, and ALP were the significant risk factors associated with dyslipidemia in HBV-positive patients with liver cirrhosis (Table 5).

Receiver operative characteristics curve analysis of serum lipids and liver enzymes

ROC curve analysis demonstrated that the areas under the curve for liver enzymes (Figure 1) and serum lipids (Figure 2) predicted liver cirrhosis in HBV-infected patients and indicated the optimum cut-off values (Table 6).

Discussion

The current investigation explored the association of serum lipids with liver enzymes and liver histology in HBV-positive patients with liver cirrhosis—a patient group

Table 6: Area under the curve and optimum cut-off values for serum lipids and liver enzymes.

Variables	Area under the curve	Optimum cut-off values	Sensitivity	Youden Index
TC	0.48	206 mg/dl	0.399	−0.066
HDL	0.52	42.5 mg/dl	0.238	−0.164
LDL	0.42	153.5 mg/dl	0.098	−0.061
VLDL	0.49	29.5 mg/dl	0.734	0.059
TG	0.52	147.5 mg/dl	0.713	0.064
ALT	0.762	43.6 U/L	0.993	0.993
AST	0.766	42.5 U/L	0.566	0.407
ALP	0.800	90.5 U/L	0.615	−0.123

typically showing abnormally elevated liver enzymes.[12] This increase in liver enzyme concentration is directly associated with the liver disease grade. We found significantly higher levels of bilirubin, ALT, AST, and ALP in the HBV-positive group than in the control group. In contrast, the levels of serum lipids (TC, TG, HDL, LDL, and VLDL) were significantly lower in HBV-positive patients with cirrhosis than in controls. Hepatic impairment caused by HBV has been reported to disrupt lipid metabolism and to decrease serum lipid levels.[11,13,14] This decrease in lipid levels is associated with a compromised lipid-synthesis ability in the liver, owing to liver cirrhosis. No significant associations of sex or age with dyslipidemia were observed in patients with cirrhosis and healthy controls in the current investigation. Ghadir et al. have reported that dyslipidemia is associated with the progression of the disease rather than the age or sex of patients.[15]

In the current study, the association of serum lipids with liver function biomarkers suggested that HDL levels strongly and significantly correlated with liver enzyme levels and may serve as a strong predictor of liver function and cirrhosis. Trieb et al. have reported that HDL levels in patients with liver are significantly altered.[16] Ramcharran et al. have revealed the prognostic value of HDL in viral hepatitis liver cirrhosis.[17] An association of HDL with liver cirrhosis has also been reported by Manka et al.[18] Su et al. have reported poorer liver dysfunction outcomes in patients with liver cirrhosis with lower HDL levels.[19]

We found that the grading score was positively correlated with serum lipids (except TC) and all liver function biomarkers, and the staging score was negatively correlated with TC and VLDL. Both grading and staging scores were negatively associated with TC. Several studies have explored the relationship between serum lipids and liver fibrosis. A study from Spain has revealed significant associations of HDL and TG with fibrosis in HBV-infected patients.[20] A study from an American University has reported that LDL is a good predictor of liver fibrosis.[21] In HCV infection, Siagris et al. have found that the grading score is positively correlated with TC, whereas no correlation exists between the staging score and serum lipids (TC, LDL, HDL, or TG).[22]

A multivariate logistic regression analysis revealed that sex and high levels of serum ALT, AST, and ALP were significant risk factors associated with dyslipidemia in HBV-positive patients with liver cirrhosis. These findings indicate not only that cirrhosis is the main reason for the increase in liver enzymes, but that if liver enzyme levels are elevated for other reasons, such as fatty liver disease, cirrhosis can result.[18] Here, the ROC curve indicated the optimum cut-off values for liver enzymes, which exceeded the minimum values for the respective enzymes.

In conclusion, HBV infection is associated with dyslipidemia and abnormal levels of liver enzymes. Dyslipidemia, specifically diminished levels of HDL and TC in the blood, is associated with liver dysfunction, and liver cirrhosis grade or stage.

Recommendation

Further studies are required to clarify the pathophysiology of impaired lipid metabolism due to HBV infection. Serum lipids and liver enzymes may serve as the best index for the prognosis of liver cirrhosis, because long-term abnormal levels of both of these parameters are the main cause of liver cirrhosis in HBV-infected patients. Liver cirrhosis can be avoided if the levels of lipids and liver enzymes can be modulated through chemotherapeutic agents.

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CRediT authorship contribution statement

Naila Shoaib: Resources, Validation. **Zaman Khan:** Conceptualization, Methodology, Resources. **Marukh Ibrahim:** Formal analysis, Data Curation. **Anjam Hafeez:** Writing—Review & Editing. **Arooj Fatima:** Writing—Review & Editing. **Hassan Imran:** Writing—Review & Editing, Conceptualization, Formal analysis. **Fiza Saleem:** Writing—Review & Editing, Conceptualization, Formal analysis. **Syed Muhammad Hassan Askari:** Formal analysis, Data Curation. **Sidra Gull:** Conceptualization, Methodology, Resources.

Conflict of interest

The authors have no conflict of interest to declare.

Ethical approval

The study was approved by the institutional review board of The University of Lahore on 4-5-2021 under approval number IRB-UG-21947.

Consent

All patients involved in the study were informed that their identity would not be disclosed, and their data would be used for research purposes only.

Authors contributions

SG and ZK conceived and designed the work and provided logistic support, NS acquired data and conducted the laboratory work, MI and SMHA analyzed and interpreted data, AH and AF was involved in writing the final draft, and HI and FZ critically reviewed the manuscript for intellectual content and statistical analysis. All authors have critically reviewed and approved the final draft, and are responsible for the content and similarity index of the manuscript.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jtumed.2023.01.002.

References

1. Yuen M-F, Chen D-S, Dusheiko GM, Janssen HL, Lau DT, Locarnini SA, et al. Hepatitis B virus infection. **Nat Rev Dis Primers 2018**; 4: 1–20.
2. Tu T, Budzinska MA, Shackel NA, Urban S. HBV DNA integration: molecular mechanisms and clinical implications. **Viruses 2017**; 9: 75–79.
3. Saeed A, Virani SS, Mulukutla S, Chow CK. Dyslipidemia and cardiovascular disease prevention in South Asians: a review and discussion of causes, challenges and management strategies. **Curr Diabetes Rev 2021**; 17: 74–86.
4. Shiani A, Narayanan S, Pena L, Friedman M. The role of diagnosis and treatment of underlying liver disease for the prognosis of primary liver cancer. **Cancer Control 2017**; 24: 1–5.
5. Osna NA, Donohue Jr TM, Kharbanda KK. Alcoholic liver disease: pathogenesis and current management. **Alcohol Res Curr Rev 2017**; 38: 147–161.
6. Nicoletti A, Ponziani FR, Biolato M, Valenza V, Marrone G, Sganga G, et al. Intestinal permeability in the pathogenesis of liver damage: from non-alcoholic fatty liver disease to liver transplantation. **World J Gastroenterol 2019**; 25: 4814–4834.
7. Kim D, Touros A, Kim WR. Nonalcoholic fatty liver disease and metabolic syndrome. **Clin Liver Dis 2018**; 22: 133–140.
8. Pierantonelli I, Svegliati-Baroni G. Nonalcoholic fatty liver disease: basic pathogenetic mechanisms in the progression from NAFLD to NASH. **Transplantation 2019**; 103: 1–13.
9. Zoller H, Tilg H. Nonalcoholic fatty liver disease and hepatocellular carcinoma. **Metabolism 2016**; 65: 1151–1160.
10. Arain SQ, Talpur FN, Channa NA, Ali MS, Afridi HI. Serum lipids as an indicator for the alteration of liver function in patients with hepatitis B. **Lipids Health Dis 2018**; 17: 1–10.
11. Ishak K. Histological grading and staging of chronic hepatitis. **J Hepatol 1995**; 22: 696–699.
12. Lin C-S, Chang C-S, Yang S-S, Yeh H-Z, Lin C-W. Retrospective evaluation of serum markers APRI and AST/ALT for assessing liver fibrosis and cirrhosis in chronic hepatitis B and C patients with hepatocellular carcinoma. **Intern Med 2008**; 47: 569–575.
13. Vere CC, Streba CT, Streba L, Rogoveanu I. Lipid serum profile in patients with viral liver cirrhosis. **Med Princ Pract 2012**; 21: 566–568.
14. Vere CC, Neagoe D, Streba CT, Prejbeanu I, Ianoşi G, Comănescu VI, Pirici D. Steatosis and serum lipid patterns in patients with chronic viral hepatitis: differences related to viral etiology. **Rom J Morphol Embryol 2010**; 51(3): 509–514.
15. 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: 285–288.
16. Trieb M, Horvath A, Birner-Gruenberger R, Spindelboeck W, Stadlbauer V, Taschler U, Curcic S, Stauber RE, Holzer M, Pasterk L, Heinemann A. Liver disease alters high-density lipoprotein composition, metabolism and function. **Biochim Biophys Acta 2016**; 1861(7): 630–638.
17. Ramcharran D, Wahed AS, Conjeevaram HS, et al. Serum lipids and their associations with viral levels and liver disease severity in a treatment-naïve chronic hepatitis C type 1-infected cohort. **J Viral Hepat 2011**; 18(4): 144–152.
18. Manka P, Olliges V, Bechmann LP, Schlattjan M, Jochum C, Treckmann JW, et al. Low levels of blood lipids are associated with etiology and lethal outcome in acute liver failure. **PLoS One 2014**; 9: 102351–102357.
19. Su TC, Lee YT, Cheng TJ, Chien HP, Wang JD. Chronic hepatitis B virus infection and dyslipidemia. **J Formos Med Assoc 2004**; 103: 286–291.
20. Mena Á, Pedreira JD, Castro Á, López S, Vázquez P, Poveda E. Metabolic syndrome association with fibrosis development in chronic hepatitis B virus inactive carriers. **J Gastroenterol Hepatol 2014**; 29: 173–178.
21. Jaafar RF, Hajj Ali AM, Zaghal AM, Kanso M, Habib SG, Halaoui AF, et al. Fibroscan and low-density lipoprotein as determinants of severe liver fibrosis in diabetic patients with nonalcoholic fatty liver disease. **Eur J Gastroenterol Hepatol 2019**; 31: 1540–1544.
22. Siagris D, Christofidou M, Theocharis GJ, Pagoni N, Papadimitriou C, Lekkou A, et al. Serum lipid pattern in chronic hepatitis C: histological and virological correlations. **J Viral Hep 2006**; 13: 56–61.

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