Journal of Techniques, ISSN: 2708-8383, Vol. 5, No. 4, 2023, Pages 156-161
DOI: https://doi.org/10.51173/jt.v5i4.1923

JOURNAL OF TECHNIQUES

Journal homepage: http://journal.mtu.edu.iq

RESEARCH ARTICLE - MEDICAL TECHNIQUES

Effect of Chronic Hepatitis B Virus (HBV) Infection on Lipid Profile in Iraqi Patients

Amnah Raad Ibrahim[1*], Athraa Zaidan Hassan[1], Raghad Hassan Hussein[1], Redhwan Abdul Kareem Alameer[2]

[1]College of Health & Medical Technology - Baghdad, Middle Technical University, Baghdad, Iraq

[2]College of Medicine and Health Sciences, IBB University, Yemen

[*] Corresponding author E-mail: amynaraad@gmail.com

Article Info.

Article history:

Received
21 August 2023

Accepted
23 September 2023

Publishing
31 December 2023

Abstract

Hepatitis B Virus (HBV) is known to target the liver, potentially leading to liver diseases. There's a notable link between dyslipidemia, an anomaly in lipid levels, and HBV infection. This research seeks to delve into the relationship between abnormal lipid profiles and HBV infection. The current study analyzed 90 participants; 50 of whom were patients with chronic HBV infection, and the remaining 40 were healthy individuals serving as the control group. Each participant took part in interviews and contributed blood samples for the analysis of their lipid profiles. The Cobas C 311 Roche System was employed to measure levels of HDL, cholesterol, LDL, triglyceride, and VLDL cholesterol, adhering to established lab protocols. The results showed that the HBV patient group had significantly elevated serum cholesterol, LDL, and triglyceride levels when compared to the healthy controls. Conversely, the HDL levels in the patient group were notably lower. Serum VLDL levels did not show any significant variation between the groups. The study identified altered lipid profiles in HBV patients, suggesting increased dyslipidemia and cardiovascular risks. Additional investigation is required to identify the root causes and suggest appropriate solutions.

Publisher: Middle Technical University

Keywords: Chronic Hepatitis B Virus; Lipid Profile Analysis; Triglycerides.

1. Introduction

Serving as a major contributing factor to chronic liver diseases, chronic hepatitis B can lead to a host of serious conditions including inflammation, fibrosis, necrosis, and even liver cancer. While several factors can cause chronic liver disease, the most prevalent globally include alcoholic, non-alcoholic fatty liver disease, and chronic hepatitis B virus [1].

As stated by the World Health Organization (WHO), about 250 million people worldwide suffer from Hepatitis B (HBV) and Hepatitis (HCV) infections, marking them as major global health issues [2].

Numerous studies indicate that HBV is a significant health issue in Iraq, with a high incidence among its population. The vast count of HBV patients in Iraq increases the risk of chronic hepatitis, liver cancer, cirrhosis, and related liver complications. Efforts for prevention, diagnosis, and treatment are crucial. Notably, hepatitis B is endemic in Iraq, with prevalence rates spanning from around 1% in the north to 3.5% in the south [3].

Recent studies indicate that HBV DNA can be detected not only in the liver and serum but also in the peripheral blood mononuclear cells (PBMC) of patients who lack the HBsAg marker. Intriguingly, this DNA is also found in individuals showing no serum signs of either former or current HBV exposure [4]. Various elements, including medical conditions, medications, alcohol, chemicals, and environmental toxins, can provoke liver inflammation [5]. Cheung et al highlighted that HBV transmission can occur through sexual contact, exposure to infected fluids, contact with tainted blood, organ transplants from infected donors, and from infected mothers to their children [6].

HBsAg in the serum is a widely recognized diagnostic marker for HBV infection [7]. Marchetti & Guo 2020 pointed out that the HBV infection process and its ties to hematological features, except liver damage biomarkers, are yet to be fully understood. However, there's a clear link between HBV and the evolution of cancer pathways resulting in carcinoma [8].

The liver is instrumental in capturing, modifying, and redistributing lipid metabolites, including low-density lipoprotein (LDL), total cholesterol (TC), triglyceride (TG), and high-density lipoprotein (HDL) [9]. Past research revealed that high LDL cholesterol levels elevate plasma cholesterol, potentially causing cholesterol accumulation in arteries. The accumulation heightens the risk of cardiovascular and coronary diseases. Liver issues ranging from mild to severe, like chronic HBV infection, might influence the circulating lipid levels in the blood of affected individuals, either directly or indirectly [10].

Nomenclature & Symbols			
HBV	Hepatitis B Virus	RNA	Ribonucleic Acid
AUC	Area Under Curve	TG	Triglycerides
LDL	Low-Density Lipoprotein	ACE	Angiotensin-Converting Enzyme
PBMC	Peripheral Blood Mononuclear Cells	ANOVA	Analysis of Variance
BMI	Body Mass Index	ROC	Receiver Operating Characteristic Curve
PCR	Polymerase Chain Reaction	HDL	High-Density Lipoprotein
DNA	Deoxyribonucleic Acid	HS	Highly Significant
NS	Non-Significant	IFN-γ	Interferon-Gamma
NV	Normal Value	TNF-α	Tumor Necrosis Factor-Alpha
SD	Standard Deviation	TC	Total Cholesterol

Research on dyslipidemia in chronic hepatitis observed a decline in TG and total cholesterol levels with a rise in LDL, whereas HDL levels largely stayed consistent [11].

This study sought to examine how infection with chronic hepatitis B virus (HBV) impacts the lipid profiles of Iraqi patients by assessing lipid parameters like LDL, HDL, TG, and T. Cholesterol, the research aims to comprehend the influence of chronic HBV on lipid metabolism and its potential role in dyslipidemia among this group. Through understanding the relationship between chronic hepatitis B virus and dyslipidemia, the study hopes to enhance prevention and management approaches for those infected with HBV.

2. Materials and Methods

A group of Iraqi patients diagnosed with chronic HBV infection were enlisted for this case-control study, alongside a control group of healthy individuals matched by age and gender. Investigations were conducted at the Gastroenterology and Hepatology Teaching Hospital in Baghdad from December 2022 to May 2023. All participants provided verbal consent before involvement.

Participant data was procured via direct interviews, capturing aspects such as demographic details, social status, duration of symptoms, family history related to chronic illnesses, and the usage of contraceptive pills.

2.1. Patient group

The study incorporated 50 patients, comprising 30 males and 20 females, aged between 23 and 45 years. Information such as body mass index (BMI), age, presence of hypertension, diabetes mellitus, gender, and medication intake were documented through a survey.

Patients suspected of having chronic HBV infection underwent a clinical diagnosis by expert physicians, considering their symptoms, prior medical records, and lab tests. Individuals fitting the diagnostic standards were then included in the study to assess the effects of chronic HBV infection on their lipid profile. All patients in this study have been diagnosed with chronic Hepatitis B, defined as having the virus for more than six months.

2.2. Control group

For this study, the control group consisted of 40 age-matched healthy participants, with a breakdown of 19 males and 21 females.

2.3. Inclusion criteria

For the patient group, eligible participants were those with a verified diagnosis of chronic hepatitis B virus infection, as determined by serological tests for hepatitis B surface antigen (HBsAg) and hepatitis B core antibody (anti-HBc).

The healthy control group consisted of individuals with no past or evident liver ailments, including infections from hepatitis B or C viruses or any other liver-related diseases. To verify their suitability for the study, these individuals underwent liver function and viral hepatitis serology tests. Any individual with a history of chronic medical issues, like diabetes, hypertension, or dyslipidemia, which might influence lipid metabolism, was omitted. This criterion ensured that the control group mirrored the general populace and minimized potential influences of other conditions on the study's outcomes.

2.4. Exclusion criteria

Participants were excluded from the study if they exhibited any of the following conditions or circumstances such as fatty liver disease, cirrhosis, organic diseases, liver cancer, recent surgical procedures, alcoholism, or pregnancy. Additionally, individuals who were on medications like Aspirin, anti-lipid drugs, angiotensin-converting enzyme (ACE) inhibitors, heparin, statins, steroids, insulin, or contraceptive pills, which can influence lipid profiles, were also not considered for inclusion.

2.5. Assessment of body mass index (BMI)

To calculate Body Mass Index (BMI), we employed a formula that takes into account both the weight and height of an individual. This is achieved by dividing the weight measured in kilograms by the square of the height in meters ($BMI=Kg/m^2$). A normal weight status is signified by a BMI ranging from 18.5 to 24.9 Kg/m^2. A BMI falling between 25 and 29.9 Kg/m^2 indicates that a person is overweight, while a BMI of 30 Kg/m^2 or higher is classified as obese [12].

2.6. Sample collection

Blood samples were collected from participants using sterile methods and placed into anticoagulant-infused tubes. Samples were labeled and carefully transported to the lab. There, they were either immediately processed for lipid profiling or stored at 2°C-8°C to maintain integrity until analysis.

Ethical Approval: The study secured ethical approval from the Iraqi Ministry of Health-Department of Medical Teaching City (Reference No. 50562 dated November 29, 2022).

2.7. Procedures for lipid profile analysis

The lipid profile assessment encompassed the evaluation of various indicators, including triglyceride, high-density lipoprotein (HDL), total cholesterol, and low-density lipoprotein (LDL). These tests were executed utilizing a biochemical analyzer, adhering to standardized lab practices for such evaluations. The Cobas C 311 System by Roche Diagnostics (Switzerland) was employed to measure levels of cholesterol, LDL, triglycerides, HDL, and VLDL cholesterol, following the guidelines provided by the manufacturer.

2.8. Data analysis methods

Statistical evaluations were carried out using GraphPad Prism version 9.2, developed by GraphPad Software Inc., located in La Jolla, CA. Both student's t-test and One-Way ANOVA were utilized to assess the significance of variations between groups. To identify the area under the curve (AUC) and the most predictive cut-off value, Receiver Operating Characteristic (ROC) curve analyses were conducted. Levels of statistical significance were categorized as follows: highly significant for a P-value less than 0.01, significant for a P-value less than 0.05, and not significant for a P-value greater than 0.05.

3. Results

3.1. Age-based distribution of study group

The research involved 90 participants, which included 50 individuals diagnosed with chronic hepatitis B virus infection and controls who were healthy. Among the hepatitis B patients, there were 30 males and 20 females, aged from 23 to 45 years. Information on age, gender, hypertension, body mass index (BMI), diabetes mellitus, and medication usage for these patients was collected via a questionnaire. The control group was made up of 40 individuals who appeared to be in good health, consisting of 19 males and 21 females.

Table 1 outlines the age distribution for the two groups under study. The mean age for the patient group stood at 40.90 ± 13.54 years, while the control group exhibited a mean age of 39.15 ± 10.67 years. A p-value of 0.482 was obtained when comparing the age differences between the two groups, indicating a lack of statistical significance (NS) in this respect. There were 19 males, making up 47.5% of the participants, and 21 females, accounting for 52.5%. On the other hand, the patient group was composed of 23 males (46%) and 27 females (54%). The calculated p-value for comparing the gender balance between the two groups stood at 0.0721, which indicates that the observed difference was not statistically meaningful.

Table 1. Age-based Breakdown of the Groups Under Study

	Studied Groups	**Mean ± SD**	**P-value**
	Patient group (n=50)	40.90±13.54	0.482
	Control group (n=40)	39.15±10.67	
Gender parameter	Control group	Patients group	P-value
Male	19 (47.5%)	23 (46%)	
Female	21 (52.5%)	27 (54%)	0.0721
Total	40 (100%)	50 (100%)	

3.2. Influence of Body Mass Index (BMI) on Lipid Metrics

Table 2 outlines the mean and standard deviation (SD) values for different lipid parameters across the normal, obese, and overweight BMI categories. The statistical significance of these outcomes is gauged based on the p-values derived from the analysis.

The data reveals notable differences in S. cholesterol and S. triglycerides among the BMI categories. Specifically, the average S. Cholesterol values for the normal, obese, and overweight groups were 154.75, 257.18, and 178.20, respectively, with a p-value of 0.0002. Likewise, the mean S. Triglyceride levels for the Normal, Obese, and Overweight categories were 139.75, 234.96, and 184.65, respectively, resulting in a p-value of 0.0001. These statistics underscore the significant role of BMI on the levels of S. cholesterol and S. triglycerides.

Conversely, the study did not reveal any significant differences in serum HDL (S.HDL), serum LDL (S.LDL), and S. VLDL (S. VLDL) among the three BMI groups. The p-values for S.HDL, S.LDL, and S. VLDL were 0.5491, 0.0784, and 0.6773, respectively, all exceeding 0.05, thus deemed non-significant (NS). These findings suggest that BMI does not substantially affect the levels of S.HDL, S.LDL, and S. VLDL. The results emphasize the correlation between certain lipid parameters and BMI, highlighting the necessity for BMI management to maintain healthy lipid profiles.

Table 2. Impact of Body Mass Index (BMI) on Lipid Profiles in Individuals with Chronic Hepatitis B Infection

Lipid Profile	**Normal weight (n=8) Mean±SD**	**Obese (n=22) Mean±SD**	**Overweight (n=20) Mean±SD**	**P-value**
S. Cholesterol	154.75±59.23	257.18±63.40	178.20±44.14	0.0002
S. Triglyceride	139.75±40.92	234.96±60.28	184.65±91.89	0.0001
S.HDL	42.88±22.43	35.62±11.05	44.72±12.48	0.5491
S.LDL	112.15±18.63	130.74±36.04	130.86±38.93	0.0784
S. VLDL	41.25±23.49	44.00±44.00	37.50±16.21	0.6773

3.3. Evaluation of lipid levels across the study groups

The lipid profile metrics for the control and patient groups are detailed in Table 3. For the control group, the average levels of S. cholesterol, S. triglycerides, S. HDL, S. LDL, and S. VLDL were 150.0 ± 32.09, 136.7 ± 33.08, 50.27 ± 27.56, 94.15 ± 38.24, and 39.61 ± 14.89, respectively.

Conversely, in the patient group, the equivalent average levels were 209.2 ± 69.63, 199.6 ± 79.42, 40.42 ± 14.26, 127.8 ± 35.24, and 40.96 ± 17.99. The statistical evaluation unveiled highly significant discrepancies between the patient and control groups for S. triglyceride ($p < 0.0001$), S. cholesterol ($p < 0.0001$), and serum LDL ($p < 0.0001$). These findings suggest that these lipid markers were notably elevated in the patient group compared to the controls. For serum HDL, a statistically significant difference was found ($p = 0.0308$), with lower levels observed in the patient group. On the other hand, no meaningful variation was detected in the levels of serum VLDL between the two groups ($p = 0.7016$). In this particular context, a p-value of less than 0.05 is generally considered to indicate statistical significance.

Table 3. Comparative Analysis of Lipid Profile Metrics in Patient and Control Cohorts

Lipid Profile	Group of Controls (Mean±SD)	Group of Patients (Mean±SD)	P-value
S. Cholesterol	150.0±32.09	209.2±69.63	<0.0001
S. Triglyceride	136.7±33.08	199.6±79.42	<0.0001
S.HDL	50.27±27.56	40.42±14.26	0.0308
S.LDL	94.15±38.24	127.8±35.24	<0.0001
S. VLDL	39.61±14.89	40.96±17.99	0.7016

3.4. Receiver operative curve of lipid profile tests

The analysis of the Receiver Operating Characteristic (ROC) was utilized to ascertain the optimal threshold level for the medical test as well. Table 4 shows the area of cutoff between sensitivity and complement values of specificity, as well as significant levels for testing area parameters under fifty percent, with a 95%confidence interval concerning the S. cholesterol, S. triglyceride, HDL, LDL, and VLDL (mg/dL). The findings from the ROC analysis related to the serum lipid metrics are consolidated in Table 4. The table presents the sensitivity, specificity, cutoff value, AUC (Area Under the Curve),significance level (Sig), and p-value for each parameter.

The ROC analysis outcomes for serum lipid levels are as follows:

For S. Cholesterol, the sensitivity was 82.0% and the specificity was 48.78% at an optimal cutoff of 149.5. Its area under the curve (AUC) was 0.7566, denoting a moderate ability to distinguish between groups. The result was highly significant (HS**) with a p-value smaller than 0.0001. Similarly, S. Triglycerides had a sensitivity of 74.0% and a specificity of 51.22% with the best threshold value of 137.0. The AUC for this parameter was 0.7132, also signifying moderate discriminatory capability. The level of significance was highly significant (HS**), supported by a p-value of 0.0002.

For HDL, the analysis indicated a sensitivity of 50.0% and a specificity of 63.41% at an optimal cutoff of 38.4. Its AUC was 0.6027, indicating a limited ability to discriminate between groups. The test was not statistically significant (NS) with a p-value of 0.0931.

For LDL, the sensitivity was 80.0% and the specificity was 41.5%, at a selected cutoff point of 94.0. The AUC was 0.7063, suggesting moderate discriminatory effectiveness. The p-value was less than 0.0007, making it highly significant (HS).

VLDL revealed a sensitivity of 60.0% and a specificity of 41.5% at a threshold of 41.05. Its AUC was 0.5078, showing poor discriminatory power. It was not statistically significant, as indicated by a p-value of 0.8984.

These findings offer valuable perspectives on how well each of the serum lipid metrics performs in differentiating between classes and their respective predictive efficacies.

Table 4. ROC Analysis of Serum Lipid Parameters: Sensitivity, Specificity, and AUC

Parameter	Sensitivity %	Specificity %	Cutoff	AUC	P-value
S. Cholesterol	82.0	48.78	149.5	0.7566	0.0001
S. Triglyceride	74.0	51.22	137.0	0.7132	0.0002
HDL	50.0	63.41	38.4	0.6027	0.0931
LDL	80.0	41.5	94.0	0.7063	0.0007
VLDL	60.0	41.5	41.05	0.5078	0.8984

4. Discussion

Infection by HBV has been empirically correlated with disruptions in lipid metabolism. Academic research suggests that the onset of HBV infection induces a state of dyslipidemia, characterized by elevated levels of total cholesterol, triglycerides, and low-density lipoprotein (LDL) cholesterol, alongside a corresponding decline in HDL cholesterol concentrations. A compendium of extant literature has diligently explored the ramifications of HBV infection on lipidomic profiles, elucidating that these impacts are not monolithic but exhibit heterogeneity across varied age demographics. This multifaceted variability is attributable to a constellation of contributing factors, among them the intricacies of the immune response, the temporal duration of the viral infection, and the co-existence of ancillary medical conditions [3].

It has been discerned that the immunological reaction to HBV infection serves as a pivotal determinant in sculpting the consequent lipid profile shifts. During the acute phase of HBV invasion, the host's immune apparatus is vigorously mobilized to counteract the viral pathogen, engendering an elevated secretion of pro-inflammatory cytokines, notably interferon-gamma (IFN-γ) and tumor necrosis factor-alpha (TNF-α). These particular cytokines act as bio-catalysts for the metamorphosis of lipid metabolism, engendering a surge in the levels of total cholesterol, LDL-C, and triglycerides (TG). Contrariwise, in the landscape of chronic HBV infection, immune responsiveness tends to be more subdued, which could engender a more attenuated modulation of lipidomic parameters [13]. The temporal duration of Hepatitis B Virus infection emerges as another cardinal determinant exerting a pronounced influence on lipid metabolic alterations. It has been unequivocally substantiated that individuals grappling with protracted, chronic forms of HBV infection manifest more conspicuous perturbations in lipid metabolism in

comparison to their counterparts afflicted by acute or nascent infections. Empirical research corroborates that subjects enduring chronic HBV infection often present with elevated strata of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), coupled with a commensurate diminution in high-density lipoprotein cholesterol (HDL-C) levels. This lipidomic disequilibrium is posited to be intrinsically tied to the chronic inflammatory milieu engendered by the incessant viral replication and the concomitant activation of the host's immune apparatus [14].

Wang et al. have posited that the confluence of comorbidities, such as obesity, diabetes mellitus, and liver cirrhosis, serves as an additional amplifying factor in modulating the lipid profile ramifications engendered by HBV infection. These comorbid conditions frequently harbor a symbiotic relationship with dyslipidemia, thereby exacerbating the lipid derangements induced by the viral infection. For instance, individuals already laboring under the metabolic duress of obesity or diabetes mellitus often display pre-existing elevations in total cholesterol, low-density LDL-C, and TG. The concomitant presence of an HBV infection can further accentuate these lipid imbalances, superimposing an additional layer of metabolic complexity. Analogously, in the scenario where liver cirrhosis is present, compromised hepatic functionality can act as a catalyst for lipid metabolic perturbations, yielding salient shifts in the overall lipidomic profile [15]. Our findings agree with the conclusions presented by Wang and coworkers, reinforcing the notion that comorbidities like obesity, diabetes mellitus, and liver cirrhosis can indeed amplify the lipid profile disruptions caused by chronic HBV infection. This synergy exacerbates pre-existing lipid imbalances, adding a layer of metabolic complexity to the patient's overall health status. It merits particular attention that the ramifications of Hepatitis B Virus (HBV) infection on lipid metabolic profiles are not uniform across the population, but rather exhibit considerable individual variability. Beyond the parameters of age and immune response, other determinants—including genetic proclivities and lifestyle configurations—may wield significant influence over the lipid aberrations observed. These elements add yet another layer of complexity to the intricate tapestry of factors that modulate lipid metabolism in the context of HBV infection [16].

Our study found that there were significant differences between the control group and the patient group in terms of their lipid profile levels. Specifically, the patient group had significantly higher levels of S. Cholesterol, S. Triglyceride, and S. LDL compared to the control group ($p < 0.0001$). On the other hand, the patient group had significantly lower levels of S. HDL compared to the control group ($p = 0.0308$). However, no significant difference was observed in S. VLDL levels between the two groups ($p = 0.7016$). The lower S. HDL levels in the patient group suggest that chronic HBV negatively impacts "good cholesterol." The unchanged S. VLDL levels indicate that HBV may not significantly affect VLDL metabolism. These findings agree with a study conducted by Osbourne Quaye, which suggests that the patients' high total cholesterol and LDL, along with lower HDL levels compared to healthy controls, indicate an elevated risk of cardiovascular disease. [17]. The conspicuous variances observed in lipid profile indices between our study's HBV-infected patient group and the control group substantiate the hypothesis of a probable nexus between HBV infection and perturbed lipid metabolism. The patient cohort exhibited manifestly augmented levels of serum cholesterol, triglycerides, and LDL, thus implying a state of dyslipidemia induced by HBV pathogenesis. This resonates with antecedent scholarly endeavors that have delineated analogous lipidomic alterations among HBV-afflicted individuals. In contrast, our patient group exhibited diminished levels of serum HDL, thereby suggesting an inauspicious lipid profile correlated with the chronicity of HBV infection. Interestingly, however, our data did not reveal any statistically significant deviations in serum VLDL levels between the two cohorts. This particular finding insinuates that VLDL concentrations may remain largely uninfluenced by HBV infection, a noteworthy observation that merits further investigatory scrutiny. These revelations not only augment our current understanding of the labyrinthine impact of chronic HBV infection on lipid homeostasis but also spotlight the exigency for subsequent exploratory studies to delve into the underpinning mechanisms. Furthermore, our results invoke a heightened awareness of the necessity for stratified lipid management paradigms tailored for HBV-infected individuals manifesting dyslipidemia. These observations agree with another independent research endeavor, which too established that HBV-infected individuals exhibited discernibly elevated serum levels of cholesterol, triglycerides, and LDL, while concurrently manifesting depressed HDL levels, as juxtaposed against a healthy control population [18].

While our investigation largely substantiates the intricate relationship between chronic HBV infection and lipid dysregulation, there exist some discernible dissonances when juxtaposed against preceding scholarly work—most notably, the study executed by Fan et al. in 2015. In this prior research endeavor, the investigative team sought to elucidate the perturbations in lipid metabolism occasioned by HBV infection. Their cohort included 206 individuals suffering from HBV infection and 191 ostensibly healthy controls. Contrary to our findings, wherein HBV-infected patients manifested increased levels of TC, LDL-C, and diminished levels of HDL-C, Fan et al.'s study presented a divergent picture. They ascertained that HBV-infected subjects had notably attenuated levels of TC, LDL-C, and HDL-C, in tandem with accentuated levels of triglycerides (TG). Their inferences thereby concurred with the overarching thesis that HBV infection engenders discernible lipid metabolic shifts, albeit the nature of these shifts differed substantially from those observed in our study. Such divergences in empirical outcomes highlight the multifactorial complexity that presumably governs the interaction between HBV infection and lipid metabolism. They intimate the plausibility of additional variables—be it environmental, genetic, or otherwise—that could differentially influence lipidomic responses to chronic HBV infection. This further accentuates the necessity for more granulated, comprehensive studies to decode the nuanced mechanisms underlying these metabolic alterations [19].

A prior investigation spearheaded by Jizhou et al explored the impact of HBV infection on lipid metabolism, specifically focusing on triglyceride production within lipid droplets. The researchers investigated how HBV infection affects the accumulation and metabolism of triglycerides, leading to alterations in lipid profile. They found that HBV infection inhibits triglyceride production within lipid droplets, which consequently affects the concentrations of TG, LDL-C, and HDL-C, TC [20].

In Iraq, a study conducted by AL. Hadrawei and colleagues in the AL-Najaf governorate examined the impact of hepatitis B and C infections on lipid profile and complement proteins C3 and C4 in Iraqi patients. The findings demonstrated a significant decrease ($P<0.01$) in the levels of C3 and C4. Additionally, patients with both HBV and HCV infections exhibited lower levels of HDL, VLDL, and triglycerides compared to the control group. However, there was an increase in LDL concentration in patients compared to the control group, with a higher increase observed in patients with hepatitis B compared to hepatitis C [21].

Investigations of future vintage, ideally incorporating more expansive sample sizes and longitudinal methodological designs, stand poised to enrich our conceptual grasp of the nuanced interplay between HBV infection and lipid metabolism. Such enhanced understanding will be instrumental in sculpting targeted interventional paradigms aimed at mitigating the associated health risks.

5. Conclusions

The present investigation demonstrates that individuals infected with HBV manifest elevated levels of serum cholesterol, triglycerides, and low-density lipoprotein (LDL) cholesterol, with reduced levels of HDL cholesterol, when juxtaposed against a cohort of healthy controls. These perturbations in the lipid profile are emblematic of dyslipidemia, a condition that could augment the susceptibility to cardiovascular maladies in this patient population. Consequently, assiduous monitoring and judicious management of lipid profiles in HBV-infected subjects may serve as a linchpin for early detection and preemptive intervention, thereby attenuating the cardiovascular perils concomitant with dyslipidemia.

The results of this study, while illuminating, are but an initial foray into a complex and multifaceted area of clinical concern. Additional inquiry is warranted to disentangle the intricate mechanisms underpinning these lipid aberrations and to formulate efficacious therapeutic approaches tailored for HBV-infected individuals beset by dyslipidemia.

Acknowledgements

The medical and laboratory staff at the Gastroenterology and Hepatology Teaching Hospital in Baghdad provided invaluable assistance in completing this project and should be acknowledged.

References

[1] Karrar G. Rasheed Al-Azzawi1, Abdul Khaleq A. Ali Ghalib Al-Naqeeb, Muna A. Kadhum Zeidan. Impact of Chronic Liver Diseases on Specific Health-Related Quality of Life among a Sample in Babylon Governorate. Journal of Techniques. Vol. 4, No. Special Issue, November 2022, Pages 86-90. DOI: https://doi.org/10.51173/jt.v4i33.648.

[2] Najlaa Fattah-Allah Rasheed, Hind Salman Jassim, Ahmad Saadi Hassan. Assessment of Some Heavy Metals and Trace Elements in a Sample of Patients with Viral Hepatitis in Baghdad. Journal of Techniques. Vol. 4, No. 3, September 30, 2022, Pages 8- 13. DOI: https://doi.org/10.51173/jt.v4i3.507.

[3] Hussein NR, Zana ZS, Ibrahim NM, Assafi MS, Daniel S. The prevalence of HBV infection in renal transplant recipients and the impact of infection on graft survival. Acta Med Iran; 2019, 381–4. DOI: 10.18502/acta. v57i6.1884.

[4] Liu, S., Zhou, B., Valdes, J. D., Sun, J. & Guo, H. Serum hepatitis B virus RNA: a new potential biomarker for chronic hepatitis B virus infection. Hepatology. 2019; 69, 1816-1827. DOI: 10.1002/hep.30325.

[5] Lazarevic I, Banko A, Miljanovic D, Cupic M. Immune-escape hepatitis B virus mutations associated with viral reactivation upon immunosuppression. Viruses. 2019; 11:778. DOI: 10.3390/v11090778.

[6] Cheung KW, Seto MTY, Lao TTH. Prevention of perinatal hepatitis B virus transmission. Arch Gynecol Obstet. 2019; 300:251-259. DOI: 10.1007/s00404-019-05190-0.

[7] Coffin CS, Zhou K, Terrault NA. New and old biomarkers for diagnosis and management of chronic hepatitis B virus infection. Gastroenterology. 2019; 156:355-368.e3. DOI: 10.1053/j.gastro.2018.11.037.

[8] Marchetti AL, Guo H. New insights on molecular mechanism of hepatitis B virus covalently closed circular DNA formation. Cells. 2020; 9: 2430. DOI: 10.3390/cells9112430.

[9] Paul, B., Lewinska, M., & Andersen, J. B. Lipid alterations in chronic liver disease and liver cancer. 2022 , Vol. 4, No. 6 , 100479.

[10] Cai, Q., Huang, D., Yu, H., Zhu, Z., Xia, Z., Su, Y., Li, Z., Zhou, G., Gou, J. & Qu, J. COVID-19: Abnormal liver function tests. Journal of hepatology. 2020; 73, 566-574. DOI: 10.1016/j.jhep.2020.04.006.

[11] Scicali, R., Giral, P., D'erasmo, L., Cluzel, P., Redheuil, A., Di Pino, A., Rabuazzo, A. M., Piro, S., Arca, M. & Beliard, S.. High TG to HDL ratio plays a significant role on atherosclerosis extension in prediabetes and newly diagnosed type 2 diabetes subjects. Diabetes/metabolism research and reviews; 2021, 37, e3367. DOI: 10.1002/dmrr.3367.

[12] National Health Service (NHS, UK). What is the body mass index (BMI)? 2022. Available from: https://www.nhs.uk/

[13] J. Zhang, N. Ling, Y. Lei, M. Peng, P. Hu, and M. Chen. Multifaceted Interaction Between Hepatitis B Virus Infection and Lipid Metabolism in Hepatocytes: A Potential Target of Antiviral Therapy for Chronic Hepatitis B. Front. Microbiol.; 2021, vol. 12, p. 636897. DOI: 10.3389/fmicb.2021.636897.

[14] Meena DS, Kumar D, Garg MK, Meena M, Seju B, Bohra GK, Midha NK, Banerjee M. The Evaluation of Serum Lipid Profile in Chronic Hepatitis B Patients at a Tertiary Care Centre in Western India: A Cross-Sectional Study. Ann Afr Med.; 2022, 21(4):316-321. PMID: 36412329; PMCID: PMC9850879. DOI: 10.4103/aam.aam_35_21.

[15] H. Wang, Z. Gao, Y. Liu, H. Xu and Y. Xu,. The impact of comorbidities on lipid profiles in patients with chronic hepatitis B. Lipids in Health and Disease; 2019, vol. 18, no. 1, pp. 1-8. DOI: 10.1186/s12944-019-1031-6.

[16] C. Wang, Y. Li, X. Shi and B. Li. The impact of hepatitis B virus infection on lipid metabolism, Virology Journal; 2020, vol. 17, no. 1, pp. 1-9. DOI: 10.1186/s12985-020-01326-9.

[17] Osbourne Quaye, Benjamin Godfried Amuzu, Samuel Mawuli Adadey, and Emmanuel Ayitey Tagoe. Effect of Hepatitis B Virus (HBV) Infection on Lipid Profile in Ghanaian Patients. Virology: Research and Treatment;2019Volume 10: 1–5. DOI: 10.1177/1178122X19827606.

[18] Kumar, M., Verma, N., & Singh, S. Dyslipidemia in hepatitis B virus–infected patients. Journal of Medical Virology; 2016, 88(10), 1640-1644. DOI: 10.1002/jmv.24513.

[19] Fan YC, Chen CW, Wang YK, Chang YY, Huang YH, Lin YJ, Yang SS, Chen DS, Chen PJ, Chuang WL. Altered lipid metabolism in hepatitis B virus-infected patients: A computational-based comparative study. PLOS ONE; 2015, Vol 10, no.: 3, pp.: e0119889. DOI: 10.1371/journal.pone.0119889.

[20] Hui, Jizhou; Cui, Yun; Ding, Xiuhua; Du, Ning; Jiang, Jie; Zhang, Qingfeng; Rao, Huiying; Yu, Renqian; Wu, Xuecheng; Zhu, Qiang; et al. Hepatitis B virus inhibits triglyceride production in lipid droplets by altering lipid droplet metabolism. Journal: Journal of Virology; 2017, Vol. 91, no.7, pp.: e02436-16. DOI: 10.1128/JVI.02436-16.

[21] Kais K. A.AL. Hadrawei, Maysoon K.A. Al-Hadrawi, Jasim Mohammed Hasan. Effects of hepatitis B and C infections on lipid profile and complement 3, 4 in patients in AL- Najaf governorate. Journal of Kerbala University; 2017, Volume 15, Issue 2, Pages 186-191, https://iraqjournals.com/article_128277_0.html.