RESEARCH PAPER

Study of Metabolic Associated Fatty Liver Disease (MAFLD) and Pancreatic Steatosis in Obese Versus Lean Chronic Hepatitis B Patients

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Received: 6th Dec, 2025; Revised: 18th Feb 2026; Accepted: 19th Feb, 2026; Available Online: 28th Feb, 2026

ABSTRACT
Background: Recently, the coexistence of chronic hepatitis B (CHB) and metabolic associated fatty liver disease (MAFLD) has garnered increasing attention. Nevertheless, the frequency of MAFLD among individuals with HBV infection and its relationship with pancreatic steatosis remain subjects of ongoing debate. Aims of study: This study aims to investigate the prevalence of MAFLD, hepatic fibrosis, and pancreatic steatosis in obese versus lean patients with CHB, including both treatment-naive individuals and those receiving first-line nucleos(t)ide analogue therapy. Patients and Methods: This cross-sectional study encompassed 217 CHB participants were placed into two groups in accordance to their BMI. Hepatic steatosis (HS) and stiffness were determined via transient elastography, while the presence of pancreatic steatosis (PS) was evaluated via ultrasonography. Results: Our findings revealed that the frequency of significant HS among participants with CHB was 37.3%, which is less than that reported in the general Egyptian population. PS was observed in 64.1% of overweight/obese patients and 25.8% of lean patients. HS demonstrated a positive correlation with age, BMI, T2DM, pancreatic steatosis, and dyslipidemia, and an inverse association with HBV DNA levels ($p < 0.05$), but was not significantly associated with hepatic fibrosis ($p > 0.05$). Multivariate logistic regression analysis determined diabetes mellitus, pancreatic steatosis, increased BMI or waist circumference, elevated cholesterol and triglyceride levels, and reduced HDL levels as independent determinants with hepatic steatosis. Conclusion: Our data suggest that the frequency of HS is decreased in participants with CHB than in the general Egyptian population. DM, fatty pancreas, increase in BMI or waist circumference, elevated level of cholesterol and triglycerides and low level of HDL were independent indicators for hepatic steatosis. However, the coexistence of HS with HBV did not show association with developing fibrosis.

Keywords: Chronic hepatitis B, CHB, Metabolic dysfunction associated fatty liver disease, MAFLD, pancreatic steatosis, PS, Body mass index, BMI, prevalence.
How to cite this article: Salama S, Soliman H, Soliman S, Mokhtar HM, El Demerdash T, Study of Metabolic Associated Fatty Liver Disease (MAFLD) and Pancreatic Steatosis in Obese Versus Lean Chronic Hepatitis B Patients. Int J Drug Deliv Technol. 2026;16(5s): 357-364. DOI: 10.25258/ijddt.16.5s.47
Source of support: None
Conflict of interest: None

INTRODUCTION

Hepatitis B virus (HBV) infection considered a primary contributing cause of chronic liver disease. The pathological spectrum of HBV infection ranges from chronic hepatitis and fibrosis to cirrhosis and hepatocellular carcinoma (HCC)(1).

HBV infection may induce metabolic disturbances by disrupting hepatic signaling pathways associated with the metabolism of glucose, lipids, nucleic acids, bile acids, and vitamins (2).

Metabolic dysfunction–associated fatty liver disease (MAFLD) is diagnosed by the occurrence of hepatic steatosis (HS) in conjunction with overweight/obesity, type 2 diabetes mellitus (T2DM), or disrupted metabolic dysregulation characterized by a minimum of two risk factors, encompassing elevated waist circumference, prediabetes, high blood pressure, elevated triglycerides, and low serum high-density lipoprotein (HDL) cholesterol levels. (3).

According to this updated definition, MAFLD and chronic hepatitis B (CHB) can be diagnosed concurrently and due to the rapidly increasing prevalence of MAFLD, the coexistence of MAFLD and HBV infection has become more frequent (4).

Obesity represents an additional major public health concern. In particular, abdominal obesity is frequently accompanied with marked metabolic disorders and an elevated probability of cardiovascular diseases (5).

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Previous investigations have demonstrated associations between abdominal obesity and MAFLD, insulin resistance, type 2 diabetes mellitus (T2DM), and metabolic syndrome (6, 7).

Previous investigations have documented that CHB individuals with concomitant metabolic syndrome possess an elevated probability of liver fibrosis progression, irrespective of viral load or hepatitis activity (8). Obesity also contributes to increased hepatitis B–related mortality and HCC.(9)

Moreover, fatty pancreas (FP) can cause inflammation, fibrosis, B-cell malfunction, and possibly pancreatic cancer (10). Obesity has been known for decades as a key probable determinant for visceral fat deposition (11). However, our knowledge of fat distribution in the pancreas and its relation to hepatitis B is still in its early stages.

It is time to study MAFLD in CHB patients and its association with hepatic steatosis, fibrosis and pancreatic steatosis.

PATIENTS AND METHODS

This cross-sectional study was executed in tropical medicine and infectious diseases department, Tanta University during the period from April 2022 to January 2024. Two hundred seventeen patients with CHB infection were encompassed in the study, diagnosed by positive hepatitis B surface antigen (HBsAg) over an interval of more than 6 months. Individuals with HCV antibody positive, HCC, HIV, Pregnancy or any chronic liver disease other than CHB were excluded.

Participants were placed into two groups based on the body mass index (BMI) to study the frequency of MAFLD and PS in obese versus lean CHB individuals. In group I there were 128 participants of BMI ≥25 kg/m2 and in group II there were 89 participants of BMI <25 kg/m2.

Anthropometric measurements

BMI was quantified via equation dividing the weight in kilograms by height in meters squared. Waist circumference (WC) was identified at the midpoint from the upper portion of the iliac crests and the lower lateral edges of the ribs using a non-stretchable tape. Abdominal obesity, identified as a WC of ≥94 cm in males or ≥80 cm in females based on the guidelines of the International Diabetes Federation group (12).

laboratory investigations

Every participant underwent clinical assessment as well as laboratory testing as follow:

Complete blood count, liver functions tests, HBs Ag, quantitative PCR for HBV DNA and serum Lipid profile.

Imaging

Ultrasound on abdomen was done via Toshiba Nemio XG apparatus with a convex probe 3.5 MHz using (B-mode) to diagnose fatty pancreas. On longitudinal imaging near the abdominal midline, pancreatic echogenicity was assessed relative to liver echogenicity at the same depth. When the liver exhibited increased echogenicity, the pancreas was compared with the renal cortex. FP was diagnosed if the pancreatic echogenicity exceeded that of the liver or renal cortex. (13).

Transient elastography was conducted via the FibroScan® 502 (Echosens, France) by proficient operators in accordance to the manufacturer's instructions. M or XL probes were selected based on BMI to evaluate controlled attenuation parameter (CAP) and liver stiffness measurement (LSM) values.

Statistical Analysis

Data were entered and analyzed via IBM SPSS Statistics version 20.0 (Armonk, NY: IBM Corp). Categorical variables were summarized as frequencies and percentages. The Shapiro-Wilk test was employed to assess the normality of data distribution. Continuous variables were outlined using range (minimum and maximum), mean ± standard deviation, median, and interquartile range (IQR). Comparisons of categorical variables among groups were done via the Chi-square test, with Fisher's exact test or Monte Carlo correction applied when over 20% of the expected cell counts were less than five. For normally distributed continuous variables, the Student's t-test was applied to relate two groups, whereas the Mann–Whitney U test was applied for variables with non-normal distribution. Correlations between non-normally distributed continuous variables were assessed via Spearman's rank correlation coefficient. Multivariate logistic regression analyses were conducted to assess independent determinants of liver steatosis.

RESULTS

A total of 217 participants with CHB were recruited in this study. Of these, 128 participants had a BMI ≥25 kg/m² (group I) and 89 participants had a BMI <25 kg/m² (group II). The average age of group I was 45.9 ± 11.6 years, compared with 41.6 ± 11.8 years in group II, with group I being significantly older. Group I comprised 79 males (61.7%) and 49 females (38.3%), whereas group II included 63 males (70.8%) and 26 females (29.2%). In group I, 72 participants were treatment-naïve and 56 administered with nucleos(t)ide analogues, while in group II, 49 participants were treatment-naïve and 40 were on nucleos(t)ide analogue therapy. Diabetes mellitus was significantly more prevalent in group I than in group II ($p < 0.004$). Mean WC was also significantly elevated in group I relative with group II ($p < 0.001$). No significant differences were recorded among the groups concerning sex distribution, smoking status, or hypertension (Table1).

The mean values of the patients' cholesterol, triglycerides, low-density lipoprotein (LDL) and HDL cholesterol levels were significantly elevated in the overweight/obese group (group I) ($P < 0.001$ and $P = 0.003$, respectively). Conversely, mean platelet count, AST, ALT, and HBV DNA levels did not differ significantly among the two groups ($p > 0.05$) (Table 1).

Table (1). Demographic and clinical characteristics of patients in the studied groups

		Group I (n=128)	Group II (n=89)	t/ X^2	P-value
Age	**Range**	19 - 74	19 - 72	2.69[a]	0.008*
	Mean ±SD	45.9 ± 11.6	41.6 ± 11.78		
		n	**%**	**n**	**%**		
Sex	**Male**	79	61.7	63	70.8	1.529	0.216
	Female	49	38.3	26	29.2		
Smoking	**No**	103	80.5	63	70.8	2.225	0.136
	Yes	25	19.5	26	29.2		
Type II Diabetes mellitus	**No**	106	82.8	6	6.7	4.210	0.04*
	Yes	22	17.2	83	93.3		
Hypertension	**No**	105	82.0	77	86.5	0.484	0.486
	Yes	23	18.0	12	13.5		
HBV treatment	**Naïve**	72	56.3	49	55.1	0.560	0.756
	Entecavir	28	21.9	23	25.8		
	TDF	28	21.9	17	19.1		
WC (Cm)	**Range**	81 – 128		70- 112		5573.5[b]	<0.001*
	Mean ± SD	102.6 ± 10.12		87.25 ± 9.66			
Platelet ($x10^3/mm^3$)	**Range**	32 – 431		72 – 383		9627.5	0.873
	Mean ±SD	201.07 ± 61.87		201.41 ± 55.35			
ALT IU/L (up to 40)	**Range**	10 – 210		10 – 119		8931	0.091
	Mean ±SD	32.88 ± 28.62		25.74 ± 16.93			
AST IU/L (up to 40)	**Range**	10 – 161		10– 68		9071	0.166
	Mean ±SD	30.61 ± 20.96		26.56 ± 12.54			
HBV DNA (IU/ml)	**Range**	-ve - 2×10^9		-ve – 6.7×10^8		9892	0.593
	Mean ±SD	$3.1\times10^7 \pm 2.3\times10^8$		$8.1\times10^6 \pm 7.1\times10^7$			
Cholesterol (mg/dl)	**Range**	45 – 360		34 – 320		4.318	<0.001*
	Mean ±SD	163.62 ± 74.01		123.77 ± 54.93			
Triglycerides (mg/dl)	**Range**	42 – 310		43 – 311		4.016	<0.001*
	Mean ±SD	149.53 ± 71		113.77 ± 53.8			
LDL (mg/dl)	**Range**	10 – 172		18 – 148		3.523	<0.001*
	Mean ±SD	61.36 ± 33.66		46.01 ± 28.3			
HDL (mg/dl)	**Range**	15 – 93		17 – 89		2.927	0.003*
	Mean ±SD	55.1 ± 67.11		55.6 ± 15.18			

a,t test; b=Mann whitney test; n=number of patients; p=significant; SD= standard deviation; WC, Waist circumference.*

Our data revealed that 37.3% of the 217 CHB participants who had a transient elastography inspection had significant HS (S2-S3). After dividing patients into two groups according to BMI, 50 % of the overweight/obese patients had significant HS while in the lean group steatosis were significantly lower, only 19.1 % showed hepatic steatosis. (Table 2).

Group I had significantly higher number of participants with FP finding by ultrasound examination. The percentage of FP inthis study was 48.4%. In group I, 64.1% had fatty pancreas. While the frequency of FP in lean group was 25.8% which is significantly reduced (table 2).

Table (2): radiological finding in study population.

		Group I (n=128)		Group II (n=89)		Total		Chi-square	
		N	%	N	%	n	%	X^2	P-value
CAP	S0-S1	64	50	72	80.9	136	62.7	20.125	<0.001*
	S2-S3 (significant steatosis)	64	50	17	19.1	81	37.3		
Fatty pancreas	Yes	82	64.1	23	25.8	105	48.4	22.49	<0.001*
	No	46	35.9	66	74.2	112	51.6		

N: number of patients; p,significant; X2,Chi square test.*

There was marked linear correlation among CAP values and age, existence of diabetes mellitus, BMI, waist circumference, presence of pancreatic steatosis, cholesterol and triglycerides. Also, there was a significant negative correlation among CAP and HDL level and HBV DNA, the later was done on treatment naïve patients to overcome the effect of HBV treatment. While, no association was found between CAP values and sex, smoking, hypertension, ALT, presence of gallstones or LSM values (table 3).

Table (3): Correlation between CAP values and different parameters

	R	P-value
Age	0.143	0.036*
Sex	0.001	0.983
Smoking	-0.01	0.942
DM	0.179	0.008*
Hypertension	0.110	0.108
BMI	0.383	<0.001*
Waist circumference	0.454	<0.001*
Gall stones	0.109	0.110
Fatty pancreas	0.362	<0.001*
LSM	0.101	0.140
ALT	0.127	0.062
Cholesterol	0.322	<0.001*
Triglycerides	0.337	<0.001*
LDL	0.246	<0.001*
HDL	-0.23	0.001*
HBV DNA (in naïve patients)	-0.21	0.02*

p, significant; r,Spearman coefficient*

Multivariate regression analysis identified diabetes mellitus, pancreatic steatosis, increased BMI or waist circumference, and elevated cholesterol levels and triglycerides and low level of HDL were associated with HS (table 4).

Table (4): Multivariate logistic regression analysis for associated factors with HS in CHB patients

Associated factors	*P* value	OR	95% CI for OR	
			Lower	Upper
BMI	<0.001*	1.054	0.958	1.159
Waist circumference	<0.001*	1.031	0.988	1.076
WHtR	<0.001*	1.02	0.34	4.4
DM	0.02*	1.232	0.464	3.274
Fatty pancrease	<0.001*	2.062	1.057	4.024
Cholesterol	<0.001*	1.006	0.997	1.015
Triglycerides	<0.001*	1.000	0.991	1.008
HDL	0.016*	0.992	0.971	1.012

CI, confidence interval; DM, diabetes mellitus; OR, odds ratio; p,significant; WHtR, Waist/Height ratio*

Table (5) shows no significant differences between treatment naïve and patients on treatment regarding CAP, Waist circumference, cholesterol and triglycerides levels, (P > 0.05) and a significant difference regarding LSM (P=0.002).

Table (5): Comparison between naïve patients and patients on treatment regarding different parameters.

		Groups		Mann Whitney test	
		Naive (n=121)	On treatment (n=96)	T	P-value
CAP	**Range**	100 – 400	100 - 396	10288	0.702
	Mean ±SD	247.9 ± 57.9	247.7 ± 60.2		
LSM	**Range**	3.1 - 34.3	2.7 – 27	11900	0.002*
	Mean ±SD	6.2 ± 3.9	7.2 ± 3.5		
WC(cm)	**Range**	70 – 125	70 – 128	1.650@	0.1
	Mean ±SD	95.09 ± 12.16	97.9 ± 12.8		
Cholesterol (mg/dl)	**Range**	45 – 337	43 – 360	10589.5	0.786
	Mean ±SD	146.65 ± 70.09	148.1 ± 69.2		
Triglycerides (mg/dl)	**Range**	42 – 304	43 – 311	10375	0.847
	Mean ±SD	134.27± 63.78	135.6 ± 70.6		

@,t test

DISCUSSION

HBV infection is a key contributor to cirrhosis, HCC, and is accompanied with substantial morbidity and mortality (14).

MAFLD is among the most prevalent chronic liver diseases globally. Reported frequency of MAFLD in individuals with CHB varies widely across studies, ranging from 14% to 70%(15).

The complex interaction between metabolic disturbances and immune disorders in MAFLD further exacerbates the HBV-MAFLD relationship, possibly hastening the advancement of liver disease (16).

Pancreatic steatosis has been implicated in insulin resistance, T2DM. Moreover, pancreatic cancer have been linked to pancreatic fat, information regarding pancreatic fat content and its association with clinical characteristics remains limited in the literature (17). Obesity has been recognized for decades as a key probable determinant for visceral fat deposition (18). However, our knowledge of fat distribution in the pancreas and its relation to hepatitis B is still limited**.**

We aimed to evaluate the association between MAFLD and CHB infection and its link with hepatic steatosis, fibrosis and fatty pancreas.

By using transient elastography, we found that the prevalence of significant HS (S2&S3) was 37.3% in our study (table 2). Our data revealed that the identified steatosis prevalence in our participants with CHB was significantly reduced relative to the prevalence of liver steatosis found in the general Egyptian population which was (47.5%) (22). Additionally, a retrospective analysis of 2,097 patients from major tertiary liver care centers in Egypt demonstrated that 44.9% of participants had liver steatosis (23). These results were in agree with a population-based cohort study in Korean population recruited 83,339 patients who were without MAFLD at baseline and divided them into HBsAg-positive group and HBsAg-negative group, screened for HS through ultrasonography at yearly or two-year intervals, and underwent a ten-year follow-up. Following adjustment for potential biasing determinants as age, sex, follow-up duration, alcohol consumption, and BMI, the rate of hepatic steatosis was significantly lower in the HBsAg-positive group relative to the HBsAg-negative group. (24).

Similarly, another cohort investigation done by Yu et al on 2255 Chinese CHB patients and found that CHB were linked with decreased risk of steatosis (25). These studies indicate that the frequency of HS is lower in individuals with CHB, potentially as a result of the influence of HBV on hepatic lipid metabolism. HS has been shown to correlate positively with triglyceride and LDL levels, and negatively with HBV infection, pointing to that HBV may modulate or attenuate lipid metabolism in the liver (15). A cohort study of 62,287 non-cirrhotic, non-diabetic adults within a mean follow-up of 4.46 years demonstrated that serum HBsAg positivity was inversely accompanied with the progression of hypercholesterolemia, elevated LDL, and hypertriglyceridemia (24).

Conversely, Sayar et al. reported that HS is predominantly present in persons with CHB than in the general population (26). They explained their results by suggesting that HBV X protein causes inhibition of apolipoprotein B secretion and upregulates the fatty acid binding proteins (FABP1) and both mechanisms contribute to hepatic fat accumulation and the subsequent development of hepatic steatosis. (26). This discrepancy could be attributed to using different imaging modalities, study design, ethnicity, genetics, and environmental status of the studied groups. In fact, the association between HBV and lipid metabolism is complex and need additional clinical and fundamental investigations. In our study the rate of HS in overweight/obese group was 50% and in lean group was 19.1% (table 2). These data are in parallel with Zhu et al., who documented that overweight and obese individuals with CHB have an raised probability of developing MAFLD (27). Also, these results were in parallel with results from general population that showed a significantly linear association between BMI and MAFLD (28).

On studying the correlation between CAP results with other parameters, there was a significant linear link among CAP and age. This was in agreement with Sayar et al who found that CHB individuals with steatosis were notably older (26). CAP also correlated with lipid profile (positively with cholesterol, triglycerides and LDL and inversely with HDL. This agreed with van Kleef et al who reported that dyslipidemia is a probable determinates of HS in CHB individuals (29). Also, our results were in line with Tirosh study who reported that there is positive correlation among serum cholesterol level and occurrence of fatty liver in general population (30).

Our results revealed that CHB individuals with type II DM has higher CAP readings. This was similar to what was documented by Yu et al whose results showed a significant association among type II DM and liver steatosis (25).

Concerning the link between fatty liver disease and fibrosis in CHB individuals, it was expected to find increased fibrosis by having both HS and CHB, but our results showed that there was no significant correlation among CAP and LSM results in any group of the study. This agreed with A large meta-analysis that evaluated 48,472 CHB patients from 98 studies and the result was that liver stiffness grade had no significant relationship with the existence of HS in CHB participants and there was no significant difference in the presence of cirrhosis among CHB participants with or without HS (15). In contrast, Mak et al stated that hepatic fibrosis was linearly linked with the CAP value (8). On the other hand, a retrospective cohort study encompassed 6,786 CHB participants revealed a reduced occurrence of cirrhosis in those with fatty liver than those without, either before or after propensity score matching (PSM) (31). Collectively, the precise effect of HS on liver fibrosis among CHB participants is yet to be fully elucidated, and the variation in results could be partially attributed to differences in the intensity of fatty liver disease across study populations, resulting in variable levels of hepatic injury and following fibrosis.

Our data showed a significant inverse correlation among CAP values and HBV PCR in treatment naïve patients (table 3). This was in line with the data of Zheng et al who found that the existence of HS was indirectly linked with HBV DNA levels suggesting that HS may act to inhibit viral replication (32). Another study found that the CHB+HS patients showed not only a significant decrease in HBV DNA level but also decreased pgRNA and HBsAg levels suggesting that HS has a negative effect on hepatitis B virology (31).

Logistic regression analysis showed that increasing BMI, WC or Waist/Height ratio (WHtR) are probable determinates for HS in CHB participants (table 4). Similarly, Zheng et al found that BMI and WC can be predictors of HS in CHB patients (32). These findings were in parallel with studies on general populations as Sun et al study who reported that all anthropometric indicators are higher in HS (7).

Furthermore, diabetes mellitus showed a significant association with HS suggesting that it is one of the main independent predictors of HS. Also, high cholesterol and triglycerides and low HDL were found to be risk factors for HS. A similar study showed that diabetes and dyslipidemia was found to be a probable determinates of existence of MAFLD among patients with CHB as well as the general population (27).

Notably, pancreatic steatosis was significantly associated with liver steatosis, but we hope there will be more studies in the future in this concern in CHB patients.

Of note, our finding showed no significant differences between treatment naïve and patients on treatment as regard presence of steatosis based on transient elastography finding (table 5). These data disagreed with Chang et al who found that the occurrence frequency of newly developed HS in participants administrated entecavir as 50 per 1000 person per years which is nearly double that observed in the broader population (34). Different type of populations, type of study and method for diagnosis of HS may be responsible for the differences between their results and ours. Furthermore, relatively few studies have investigated whether antiviral therapy exacerbates hepatic steatosis in individuals with chronic hepatitis B (35).

CONCLUSION:

HS is less frequent in CHB individuals than the prevalence documented in the general Egyptian population. We verified that concomitant HS was exist in over one in three individuals with CHB and was linearly correlated with age, BMI, waist circumference, type II diabetes mellitus, pancreatic steatosis, dyslipidaemia and negatively correlated with HBV DNA levels. Notably, the existence of HS was not associated with an elevated rate of significant fibrosis

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