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Original Article

Hepatitis B virus infection in patients with metabolic syndrome: A complicated relationship. Results of a population based study

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ABSTRACT

Background: The presence of hepatitis B infection (HBI) and metabolic syndrome (MS) at the same time constitutes a high risk for liver cirrhosis and potentially hepatocellular carcinoma.
Aim: In this study we aim to explore the relationship between MS and HBI.
Methods: We used data from the cross-sectional HepaMeta study conducted in 2011 in Slovakia. Patients were tested for presence of MS, while lipid levels (total cholesterol, HDL, LDL, TG, apolipoprotein B100 and HBI (HBsAg and antiHBcIgG)) were also monitored. Viral load was measured in HBsAg positive patients.
Results: Altogether 855 patients were screened, MS was diagnosed in 25.1% of patients and 7.9% of patients presented with HBI. AntiHBcIgG antibodies were present in 34.6% patients. HBI patients had lower levels of total and LDL cholesterol along with a decreased apolipoprotein B100 (4.54 ± 0.84 vs. 5.0 ± 0.99 mmol/l, P = 0.001; 2.29 ± 0.58 vs. 2.6 ± 0.68 mmol/l, P = 0.001 and 0.71 ± 0.21 vs. 0.77 ± 0.23 mmol/l, P = 0.013 respectively). Patients diagnosed with MS had higher HBV DNA load than patients without MS − 1300.2 (95% CI 506.06–3440.41) vs. 7661.3 (95% CI 2008.17–29,228.06) IU/ml; P = 0.011. HBI patients with TC and apolipoprotein B100 in the reference range had lower HBV DNA load than patients with high or low values of TC or apolipoprotein B100.
Conclusion: Hepatitis B patients had lower levels of total and LDL cholesterol along with a decreased apolipoprotein B100. Viral load of chronic hepatitis B patients with MS was higher than that in patients without MS.

1. Introduction

Hepatitis B infection (HBI) leads to fibrogenesis, cirrhosis and eventually hepatocellular carcinoma (HCC). Patients with hepatitis B DNA (HBV DNA) load > 10^5 IU/ml have a 5 times higher risk of developing liver cirrhosis than patients with HBV DNA load ≤ 10^4/ml [1].

Patients with metabolic syndrome (MS) have a high risk not only for cardiovascular disease or diabetes mellitus but also for liver disease. According to current knowledge, non-alcoholic steatohepatitis is considered to be a hepatic manifestation of MS. In obese patients (BMI > 30 kg/m^2) the prevalence of nonalcoholic fatty liver disease (NAFLD) is estimated to be as high as 75%. Nonalcoholic fatty liver disease could on its own cause the progression of liver cirrhosis in about 0.5% of patients [2].

The presence of both diseases at the same time constitutes an even higher risk for liver cirrhosis and potentially HCC [3]. Unfortunately, there is not enough data about epidemiology of metabolic syndrome

Abbreviations: HDL, high density lipoproteins; LDL, low density lipoproteins; TG, triglycerides; MS, metabolic syndrome; HBI, hepatitis B infection; HBV, hepatitis B virus; NAFLD, nonalcoholic fatty liver disease; NASH, nonalcoholic steatohepatitis; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyltransferase; SD, standard deviation.

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in hepatitis B patients and the influence of metabolic syndrome on the course of hepatitis B. Therefore in this study we aim to explore the prevalence of metabolic syndrome, or partial clinical and laboratory signs, in hepatitis B patients and to compare it with the general population while also assessing the association between metabolic syndrome and viral load in HBV infected patients.

2. Methods

We used data from the cross-sectional HepaMeta study conducted in 2011 in Slovakia. This project aimed to map the prevalence of viral hepatitis B and MS in the population living in Eastern Slovakia including Roma settlements.

2.1. Sample and procedure

From the list of the general practitioners operating in the catchment area we contacted 26 general practitioners and 17 agreed to take part in our study (response rate 65%). From a list of patients based on information from the general practitioners 709 patients from majority population were conditionally randomly chosen to create an age and gender matched control sample. These patients were contacted by phone and mail by trained research assistants who gave the information about our study and invited them to participate. From the majority population 402 patients participated in our study (response rate 56.7%). Sample participants from the Roma population were recruited directly in the settlements by cooperation with the local Roma community workers. From these Roma settlements a total of 457 participated after providing information about our study. Patients younger than 45 years were included, because of the higher expected hepatitis B prevalence. The study was performed in accordance with the principles of the Declaration of Helsinki and approved by the Ethics Committee of the Medical Faculty at Safarik University in Kosice. Participation in the study was fully voluntary and anonymous. Inclusion criteria for the respondents were: no preventive medical check-up in the past two years, no acute illness, appropriate age and be able to take a time off from work during the week of data collection in the ambulance of their general practitioner. Exclusion criteria included antiviral or immunosuppressive therapy, anti-HCV or HIV antibody positivity, history or serological evidence of autoimmune liver disease, inheritable disorders such as hemochromatosis or Wilson's disease, renal insufficiency, a history of excess alcohol intake (average daily alcohol consumption > 20 g) or drug abuse. Detailed information about our study and its procedures was given to all the patients and informed consent was signed prior to medical check-ups. Trained medical personnel collected the blood and urine samples and performed anthropometric measures in ambulances of cooperating general practitioners.

2.2. Laboratory testing

A blood sample was obtained from each patient, while the tests included hepatitis B serology (HBsAg and antiHBcIgG antibodies) and HBV DNA. HBV DNA was only sampled in HBsAg positive participants. HBsAg and antiHBcIgG testing was performed by Enzygnost Siemens, Germany. HBV DNA was measured by HBV Cobas TaqMan AmpliPrep/Cobas v2.0 Roche Switzerland with detection limit of 20 IU/ml, upper limit 170,000,000 IU/ml. Clinical biochemistry tests (total serum cholesterol, HDL, LDL, TG, apolipoprotein B100, AST, ALT, GGT, uric acid, fasting plasma glucose) were performed by Advia 1650 and Advia 2400 autoanalyzers by Siemens, Germany. Furthermore anthropometric measures including weight, height, waist circumference and blood pressure (average of three measurements) were also documented.

Patients were considered to have active HBV infection if they were HBsAg positive. As this was a cross-sectional study we were unable to perform a follow-up HBsAg test after six months, therefore we cannot determine that all of our patients had indeed chronic hepatitis B. Patients with antiHBcIgG or antiHBs positivity were considered to have had encountered HBV in the past or were otherwise vaccinated.

2.3. Criteria of metabolic syndrome

Standard International Diabetes Federation criteria were employed. Patients were considered to have MS when central obesity was present (waist circumference >94 cm for males and >80 cm for females or BMI > 30 kg/m^2) and any two of the following factors were present

- Raised triglycerides ≥ 150 mg/dl (1.7 mmol/l) or specific treatment for this lipid abnormality
- Reduced HDL cholesterol <40 mg/dl (1.03 mmol/l) in males <50 mg/dl (1.29 mmol/l) in females or specific treatment for this lipid abnormality
- Raised blood pressure, systolic ≥ 130 or diastolic ≥ 85 mm Hg, or treatment of previously diagnosed hypertension
- Raised fasting plasma glucose ≥ 100 mg/dl (5.6 mmol/l), or previously diagnosed type 2 diabetes. If above 5.6 mmol/l or 100 mg/dl, oral glucose tolerance test is strongly recommended but is not necessary [4].

2.4. Statistical analysis

Results are presented as mean ± standard deviation for continuous variables and percentages for categorical variables unless stated otherwise. HBV DNA levels and AST, ALT and GGT levels were analyzed after log (10) transformation. Differences between two groups of participants were tested using T-test or the Mann–Whitney U test where appropriate for continuous variable while a Chi squared test was utilized for categorical variables. Association between metabolic parameters and metabolic syndrome with chronic hepatitis B infection was assessed by a series of logistic regression model adjusted for confounders. Association of individual components of MS with HBV DNA load was assessed by quadratic regression.

3. Results

Altogether 855 patients were screened, 452 Roma (mean age = 33.7; SD = 9.14; 35.2% men) and 403 non-Roma (Caucasian) population (mean age = 33.5; SD = 7.4; 45.9% men).

3.1. Factors associated with chronic hepatitis B

MS was diagnosed in 211 (25.1%) patients and HBV infection — HBsAg positivity in 66 (7.9%) of patients (18 values missing). AntiHBcIgG antibodies were present in 296 (34.6%) patients (19 values missing). Study parameters are summarized in Table 1.

Parameters in the study cohort are summarized in Table 1.

Patients with HBI (HBsAg positive) had statistically significant lower levels of total and LDL cholesterol and apolipoprotein B100. No difference was found in HDL, TG, glucose, BMI and waist circumference as well as prevalence of MS, obesity or overweight. Patients who encountered HBV (antiHBcIgG positive) had statistically significant lower levels of HDL while higher levels of TG, BMI and waist circumference, as well as higher prevalence of obesity or overweight, were also present.

Factors associated with chronic HBV infection were assessed by a series of univariate logistic regression models, results are summarized in Table 2. Total cholesterol (TC), LDL cholesterol and apolipoprotein B100 were significantly associated with hepatitis B infection even after adjustment for age, sex, BMI and waist circumference. No association with chronic hepatitis B was found for uric acid and HDL cholesterol. Furthermore, clinical parameters (BMI and waist circumference) were not found to be significantly associated with chronic hepatitis B after adjustment for age and sex.

Table 1
Baseline parameters of the study cohort.

	HBsAg positive	HBsAg negative	P	AntiHBcIgG positive	AntiHBcIgG negative	P
Age (years)	33.8 ± 6.9	34.1 ± 8.4	0.975	36.4 ± 7.8	32.7 ± 8.3	<0.001
TC (mmol/l)	4.54 ± 0.84	5.00 ± 0.99	0.001	5.00 ± 0.91	4.97 ± 1.03	0.434
LDL (mmol/l)	2.29 ± 0.58	2.60 ± 0.68	0.001	2.58 ± 0.65	2.58 ± 0.7	0.738
HDL[a] (mmol/l)	1.19 ± 0.35	1.19 ± 0.41	0.66	1.13 ± 0.30	1.22 ± 0.34	<0.001
TG[a] (mmol/l)	1.11 ± 0.59	1.31 ± 0.91	0.122	1.37 ± 1.01	1.26 ± 0.82	0.031
ApoB100[a] (g/l)	0.71 ± 0.21	0.77 ± 0.23	0.013	0.78 ± 0.22	0.76 ± 0.23	0.118
Uric acid (μmol/l)	229.9 ± 80.1	243.5 ± 83.8	0.182	239.1 ± 85.2	244.2 ± 82.8	0.460
Glucose[a] (mmol/l)	4.72 ± 0.73	4.86 ± 0.88	0.158	4.88 ± 0.94	4.83 ± 083	0.724
BMI[a] (kg/m^2)	25.0 ± 4.6	25.8 ± 5.4	0.600	26.7 ± 5.9	25.2 ± 5.0	<0.002
Waist (cm)	87.0 ± 12.6	87.9 ± 14.1	0.616	90.0 ± 15.2	86.7 ± 13.1	0.002
BMI >30 kg/m^2 (%)	14.1	21.0	0.121	25.7	17.7	0.007
BMI >25 kg/m^2 (%)	48.4	49.3	0.497	55.1	46.0	0.008
MS (%)	24.6	24.7	0.561	29.8	22.0	0.008

LDL — low density lipoproteins, HDL — high density lipoproteins, TC — total cholesterol, TG — triglycerides, ApoB100 — apolipoprotein B100, BMI — body mass index, MS — metabolic syndrome.
[a] Statistical significance measured by the Mann–Whitney U test.

3.2. Viral load and disease activity assessment in HBsAg positive patients

Out of 66 HBsAg positive patients 57 were also HBV DNA positive (90.5%). Viral load ranged from 40 to 5,340,000 IU/ml with mean viral load 218,036 IU/ml, median 2360 IU/ml, interquartile range 9316 IU/ml. Patients diagnosed with MS had significantly higher HBV DNA load (after log 10 transformation) than patients without MS — geometric mean 1300.2 (95% CI 506.06–3440.41) vs. 7661.3 (95% CI 2008.17–29,228.06); P = 0.011 (Fig. 1). This difference was independent of age of HBV DNA positive patients.

HBV DNA association with individual criteria of MS was assessed by quadratic regression model. This model was chosen instead of linear regression because of typical distribution of selected parameters (lipid profile, BMI, waist circumference) with pathological values at both extremes of the parameter range. Results are summarized in Table 3.

Cholesterol and apolipoprotein B100 had a significant quadratic relationship with HBV DNA (Figs. 2 and 3), other parameters of lipid profile were statistically and marginally insignificant. No relationship between HBV DNA and BMI, waist circumference or mean arterial pressure was found.

Hepatitis B activity is usually reflected by serum activity of transaminases. We found that patients with metabolic syndrome had significantly higher activity of ALT and GGT compared to patients without metabolic syndrome. No difference between serum levels of AST in HBsAg positive patients with MS compared to patients without MS was found (Table 4).

Table 2
Association between metabolic parameters and metabolic syndrome with chronic hepatitis B infection.

Factor	Adjusted OR	95% CI	P value
TC	0.592	0.435–0.806	0.001
LDL	0.442	0.281–0.695	<0.001
TG	0.652	0.401–1.058	0.083
HDL	0.987	0.410–2.374	0.976
ApoB100	0.206	0.051–0.839	0.027
Uric acid	0.996	0.993–1.000	0.058
Waist circumference	0.987	0.966–1.088	0.231
BMI	0.975	0.926–1.028	0.352
MS	0.637	0.289–1.403	0.263

Univariate logistic regression adjusted for age, sex, BMI and waist circumference. BMI, waist circumference were adjusted only for age and sex. LDL — low density lipoproteins, HDL — high density lipoproteins, TC — total cholesterol, TG — triglycerides, ApoB100 — apolipoprotein B100, BMI — body mass index, MS — metabolic syndrome.

4. Discussion

The first aim of this study was to compare the prevalence of MS between patients infected with hepatitis B virus and non-infected patients.

The overall prevalence of metabolic syndrome was 25.1%, even in this, relatively young, population. This result is very comparable to the general Slovak population (age independent prevalence of MS 38.1%) [18] and the European population (prevalence of metabolic syndrome in specific age groups: age 20–29 years — 6.2% (3.4–9.0%), 30–39 years 18.1% (14.2–22.1%) and 40–49 years 29.5(24.3–34.8)) [19]. The age plays of course a very important role in the development of metabolic syndrome. The prevalence of MS in our study was lower as it would have been in the population without age restriction. On the other hand, younger population, due to longer life expectancy, has a higher risk of developing hepatitis B and MS related complications. Furthermore, interventions aimed to prevent these complications have higher chance of success, therefore the selected population is still clinically relevant. The prevalence of hepatitis B was expectedly very high (7.9%) in this specific population and is not comparable to the general Slovak or European population. The estimated prevalence of HBsAg positivity in Slovakia

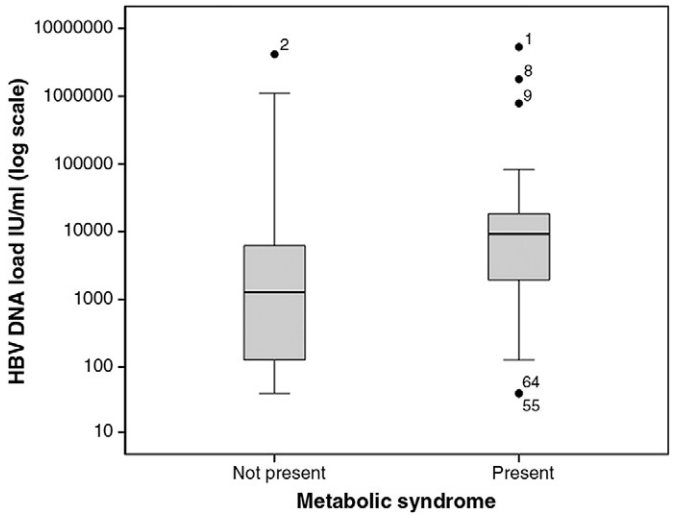

Fig. 1. HBV DNA levels in patients with and without metabolic syndrome. P for difference 0.011.

Table 3
Association between HBV DNA and components of metabolic syndrome assessed by quadratic regression analysis.

Independent variable	R^2	Formula	P value
Cholesterol	0.122	$0.432\ h^2 - 3.99\ h + 12.269$	0.028
HDL	0.086	$-1.671\ h^2 + 3.143\ h + 2.175$	0.085
LDL	0.087	$0.744\ h^2 - 3.499\ h + 7.229$	0.082
TG	0.011	$0.168\ h^2 - 0.333\ h + 3.489$	0.74
Apolipoprotein B100	0.186	$4.885\ h^2 - 6.482\ h + 5.276$	0.004
BMI	0.026	$-0.002\ h^2 + 0.14\ h + 1.081$	0.468
Waist circumference	0.059	$0\ h^2 + 0.046\ h + 0.311$	0.204
Mean arterial pressure	0.036	$0.001\ h^2 - 0.165\ h + 11.007$	0.375

LDL — low density lipoproteins, HDL — high density lipoproteins, TG — triglycerides, BMI — body mass index; h-value of independent variable.

is <2% [20]. On the other hand, Roma people make up an important ethnical minority in Slovakia and Europe. Our intent was to explore the relationship between hepatitis B and MS, therefore we chose to include the population with a high estimated prevalence of hepatitis B. It's important to note that the prevalence of MS was no different compared to the majority population.

As stated earlier, we cannot be sure that all of the participants had chronic hepatitis B, but there are other indirect signs which point to chronic hepatitis B as the reason for HBsAg positivity (in contrast to acute hepatitis B). There were only three patients with ALT over 3xULN, two of these three patients had very low viremia. Based on these arguments we believe that practically all of our patients had chronic HBV infection.

The prevalence of MS in patients with hepatitis B infection (HBsAg positive) was not significantly different compared to that of HBsAg negative patients. This was also true for BMI, waist circumference, glycemia HDL and TG levels, even after adjustment for age and sex (Table 2). Furthermore, we have not observed a significant difference in age per se between HBsAg positive and negative patients (Table 1), therefore we believe that the nonsignificant difference in the prevalence of MS between these two groups is unlikely to be caused by age confounder. Recently published data are in agreement with our finding. Most studies reported no association between hepatitis B and MS [21]. The largest study to date which included 593,594 patients with chronic hepatitis B even reported an inverse relationship between HBI and MS [11]. The prevalence of MS as well as hypertriglyceridemia in HBsAg positive patients was significantly lower compared to that in HBsAg negative controls in a Chinese cohort reported by Luo et al. The prevalence of hyperglycemia was insignificantly higher in HBsAg negative patients and both groups had similar, decreased levels of HDL [7].

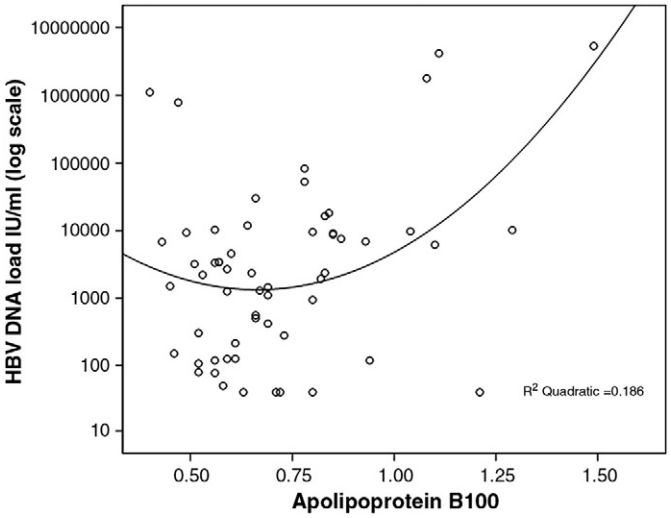

Fig. 2. Association between apolipoprotein B100 and HBV DNA load.

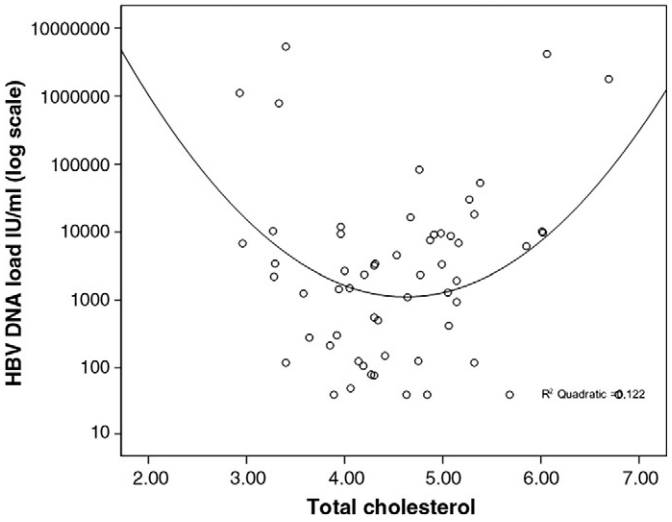

Fig. 3. Association between total cholesterol and HBV DNA load.

HBsAg positive patients had significantly lower levels of TC, LDL, and apolipoprotein B-100 than controls. This association was significant even after adjustment for age, sex, BMI and waist circumference. To our best knowledge ours is the first study reporting lower values of apolipoprotein B-100 in hepatitis B positive patients. In a recent Taiwanese study authors reported that HBsAg positive patients had significantly more often lower TG, LDL, and HDL levels and higher adiponectin levels. Total cholesterol level was also lower, albeit only in non-diabetic and non-obese HBsAg positive patients [6].

The reason behind lower levels of TC, LDL and apolipoprotein B100 in HBsAg positive patients remains unclear. Little published data allow a speculation that hepatitis B virus influences the metabolism of infected hepatocyte. Apolipoprotein H, plasma lipoprotein associated with chylomicrons and HDL-C, was reported to bind hepatitis B surface antigen (HBsAg) and presumed to react specifically with the HBsAg S protein and to play an important role in initiation of infection by hepatitis B virus [8]. There is also data that some enzymes involved in fatty acid and NADPH-electron transport pathways are altered by the presence of HBV [9]. An inverse correlation between hepatitis B virus (HBV) and steady-state levels of apolipoprotein AI and CIII mRNAs was observed in two hepatoma cell lines. HBV alters infected cells despite the absence of overt cytopathogenicity [10].

Liver tests (ALT, AST, GGT) reflect liver damage caused by any disease. We have found out that hepatitis B positive patients with metabolic syndrome have higher ALT and GGT compared to patients without metabolic syndrome. In a study by Wang et al. authors report that BMI over 25 and hyperglycemia are independent predictors of ALT elevation, even in the upper half of normal range [12]. Other paper reports significantly higher ALT level in hepatitis B patients younger than 45 years [6].

The second aim of this study was to assess the influence of MS and individual components of metabolic syndrome on HBV DNA levels in

Table 4
Liver transaminase levels in chronic hepatitis B patients with and without metabolic syndrome.

	Without metabolic syndrome	Metabolic syndrome	P value
AST	0.31 ± 0.3	0.33 ± 0.32	0.918
ALT	0.23 ± 0.28	0.29 ± 0.27	<0.0001
GGT	0.4 ± 0.69	0.63 ± 0.85	<0.0001

P value measured by the Mann–Whitney U test. AST — aspartate aminotransferase, ALT — alanine aminotransferase, GGT — gamma-glutamyltransferase.

HBsAg positive patients. We proved that HBI patients with metabolic syndrome have significantly higher levels of HBV DNA. Furthermore TC and apolipoprotein B100 had a significant quadratic relationship with HBV DNA. Quadratic regression models a relationship between two variables according to a curve similar to the letter U. Patients with hypercholesterolemia as well as hypocholesterolemia had significantly higher HBV DNA load than patients with serum cholesterol in the reference range. The same is true for apolipoprotein B-100.

There is more than one possible explanation, although none has been confirmed so far. The left part of the U shaped curve means patients with low levels of total cholesterol and ApoB100 have high viral load. Variety of studies consistently reported this inverse association between these parameters [24]. In a previously cited study by Hsu et al. authors described an inverse correlation between HBV DNA and TG [6]. As discussed earlier, hepatitis B infection interferes with hepatocyte metabolism. High viral load particularly could cause decreased total cholesterol and ApoB100 synthesis. Lower levels of apolipoprotein B100 and TC could also be caused by liver fibrosis [13] and cirrhosis [22]. Serum cholesterol in general is even a good predictor of mortality in advanced liver disease [23] and lower TC levels are even associated with higher incidence of hepatocellular carcinoma in HBI patients [14]. Decreasing levels of TC and ApoB100 in patients with high HBV DNA could mark the risk of liver disease progression and could be considered an early warning flag.

The interesting observation is the right side of the U curve which depicts the relationship between TC, ApoB100 and viral load (Figs. 2 and 3). We have observed that patients with higher than normal levels of TC or apolipoprotein B100 have also higher viral load than patients with normal cholesterol or apolipoprotein B100. Our study is the first one that links viral load in HBI patients with apolipoprotein B100 so far and this observation has not been published so far, but observations from the paper by Chiang et al. support our findings. Authors report that hepatitis B patients with BMI between 23 and 24.9 have lower viral load than patients with lower or higher BMI [3].

High viral load in hepatitis B patients is a significant risk factor for liver cirrhosis and hepatocellular carcinoma, therefore this information is important in the management of these patients. Furthermore ApoB100 better reflects the metabolism of lipoproteins in the hepatocyte compared to total cholesterol, because of lower potential for confounding factors (diet, obesity, non-fasting serum).

The presence of metabolic syndrome or its components has a negative effect on the natural course on chronic hepatitis B infection. In a study from Hong Kong histological liver cirrhosis was more common among patients who had MS (38%) versus those who did not (11%, $P < 0.001$). The prevalence of cirrhosis increased with the number of present MS components [15]. Patients with chronic hepatitis B and HCC had significantly higher mean BMI and serum glucose, but lower serum lipid levels than controls ($P < 0.05$). In a subanalysis of HCC cohort authors reported significant positive correlation between BMI, insulin and homeostasis model assessment for insulin resistance (HOMA-IR) ($P < 0.01$) [15]. Data from NHANES III cohort suggest that type 2 diabetes mellitus and insulin resistance are independent mortality predictors in hepatitis B patients [16].

In the study cohort 296 (34.6%) patients had come in contact with viral hepatitis B and formed antiHBcIgG antibodies. These patients were older, had significantly lower HDL and higher TG values, as well as higher BMI and waist circumference. Total serum cholesterol and LDL levels were approximately the same in both groups. A study published by Yen et al. reports that students (mean age 19 years) who overcame acute hepatitis B infection and were antiHBc positive had 58% higher risk of MS compared to vaccinated students with protective titers of antiHBs antibodies [5]. Our data, in agreement with this study, showed a trend towards higher MS prevalence in patients who came in contact with hepatitis B virus. It is known that antiHBcIgG as well as metabolic syndrome prevalence increases with age [17], which is the probable reason behind this observation.

The presented study is one of the first reporting the relationship between MS and hepatitis B in a non-Asian population. Unfortunately we are aware of several limitations. The cross-sectional design of the study did not allow us to follow the patients prospectively. Furthermore, it included only patients younger than 45 years, but incidence of metabolic syndrome is higher in older patients. More than half of the study cohort consisted of Roma people, which is not proportional to the general Slovak population. Chronic hepatitis B was diagnosed only in 70 patients and there was no assessment of liver fibrosis either histologically, biochemically or with transient elastography. Because no biopsy data were available we were also not able to determine the presence of liver steatosis or steatohepatitis.

5. Conclusion

Patients who overcame hepatitis B (HBcIgG antibodies positive) were older and had significantly higher values of TG, BMI, and waist circumference and lower HDL than antiHBcIgG negative patients. There was a trend towards higher prevalence of MS in antiHBcIgG positive patients. Patients with hepatitis B had significantly lower levels of TC, LDL and apolipoprotein B100 compared to HBsAg negative patients. This is, to the best of our knowledge, the first study reporting lower levels of apolipoprotein B100 in hepatitis B patients. Hepatitis B patients with metabolic syndrome had significantly higher HBV DNA load than patients without MS. Patients with TC and apolipoprotein B100 in the reference range had lower HBV DNA than patients with hypocholesterolemia or hypercholesterolemia.

Learning points

- Chronic hepatitis B is associated with lower levels of total and LDL cholesterol.
- Apolipoprotein B100 is decreased in patients with chronic hepatitis B.
- HBV DNA load is higher in HBsAg positive patients with metabolic syndrome.
- HBV DNA load is higher in patients with abnormally high or low levels of total cholesterol.

Conflict of interests

Authors declare no conflict of interest.

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