

Original Article

Body mass index is closely correlated with blood lipids, blood pressure, serum uric acid and hepatic steatosis in middle-aged and elderly hypertensive health examinees

Jin Zhang¹, Li Liu²

¹Huzhou Rehabilitation Hospital, Rehabilitation Hospital of Huzhou First People's Hospital, Huzhou 313000, Zhejiang, China; ²Department of General Surgery, First People's Hospital of Huzhou, Huzhou 313000, Zhejiang, China

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Abstract: Objective: To explore the associations between body mass index (BMI) and blood lipids, blood pressure, serum uric acid, and hepatic steatosis in middle-aged and elderly hypertensive health examinees. Methods: A total of 428 middle-aged and elderly hypertensive participants were divided into underweight/normal (n=146), overweight (n=128) and obese (n=154) groups according to BMI in this retrospective analysis. General clinical data, lipid indicators, blood pressure, serum uric acid, and hepatic steatosis status were collected. Correlation analysis and multivariate regression were used to analyze the relationships between BMI and metabolic indicators and to screen independent risk factors for hepatic steatosis. Results: Compared with the underweight/normal weight group, overweight and obese participants had adverse metabolic abnormalities and a higher incidence of hepatic steatosis. BMI was positively correlated with triglyceride, low-density lipoprotein cholesterol, systolic blood pressure, diastolic blood pressure and serum uric acid, and negatively correlated with high-density lipoprotein cholesterol. Age, elevated BMI, systolic blood pressure, serum uric acid and alcoholism were independent risk factors for hepatic steatosis in hypertensive populations. Conclusion: Increased BMI is closely related to dyslipidemia, elevated blood pressure, hyperuricemia and hepatic steatosis in middle-aged and elderly hypertensive individuals. Maintaining a healthy BMI is critical for the prevention of metabolic disorders and hepatic steatosis.

Keywords: Middle-aged and elderly individuals, hypertension, body mass index, blood lipids, blood pressure, serum uric acid, hepatic steatosis

Introduction

In China, with the increase in life expectancy and the continuous improvement of living standards, the risk of hypertension has also increased [1]. Hypertension is characterized by abnormally high systolic (SBP) and diastolic blood pressure (DBP). It is an important cause of cardiovascular and cerebrovascular diseases and kidney diseases, and can induce various diseases such as dementia, stroke, chronic kidney disease, and ischemic heart disease. However, the disease can be prevented to a certain extent [2, 3]. At present, the core goal of the drug treatment plan for hypertension is to achieve ideal blood pressure (BP) control. Studies have shown that combined medication

can more effectively lower BP and reduce the occurrence of adverse reactions, especially reducing the risk of peripheral edema [4]. From a pathologic perspective, hypertension is closely related to abnormal vascular structure. The narrowing of the vascular diameter leads to increased vascular resistance, which accelerates the progression of hypertension [5]. From an epidemiologic perspective, hypertension is highly prevalent among middle-aged and elderly people. The treatment cost is high, accounting for about 16% of chronic disease medical expenditures. Moreover, this disease accounts for more than a quarter of all-cause deaths [6-8]. To reduce the mortality risk and medical expenses of middle-aged and elderly patients with hypertension, it is of great practical signifi-

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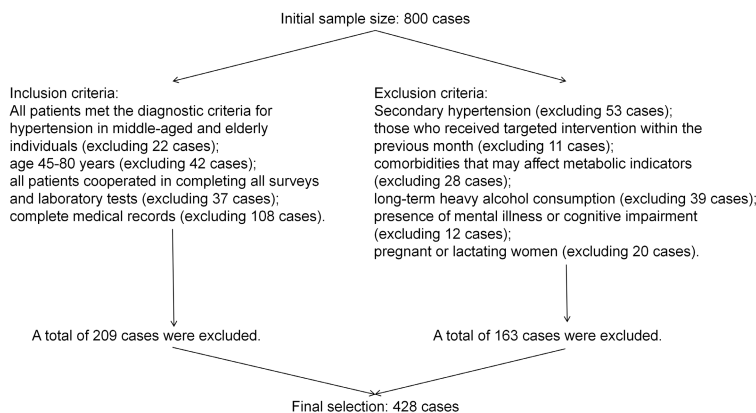


Figure 1. Flow chart of patient screening.

cance to explore the pathogenesis of hypertension, clarify its mechanism, and explore more high-quality and economical treatment options.

Obesity is a core risk factor for primary hypertension, which can induce hypertension through various pathways such as neuroendocrine activation, inflammatory response, and renal dysfunction [9]. The risk of hypertension in people with abdominal obesity is 2 to 3 times higher than that in non-obese people, and these patients are more difficult to treat, requiring more antihypertensive drugs and having a significantly higher risk of developing refractory hypertension [10, 11]. Existing studies have shown that the prevalence of obesity-related hypertension remains high in middle-aged and elderly people, and women, diabetes, dyslipidemia, and hyperuricemia were all positively correlated with obesity-related hypertension [12]. At the same time, sufficient research has confirmed that obesity is closely related to abnormally high SBP and DBP, dyslipidemia, and the development of diabetes [13, 14]. Studies by Dua et al. have shown that lifestyle interventions centered on weight management can effectively control prehypertension in overweight and obese individuals, while obesity significantly increases the risk of hypertension [15].

Currently, research in this field is relatively scarce. This study innovatively analyzed the association between body mass index (BMI) and blood lipids, BP, serum uric acid (UA), and hepatic steatosis in middle-aged and elderly individuals undergoing health checkups, aiming to provide theoretical reference for the clinical prevention and treatment of hypertension.

Materials and methods

Patient selection

Inclusion criteria: meeting the diagnostic criteria for hypertension in middle-aged and elderly individuals [16]; age 45-80 years; completion of all survey content and laboratory tests; and complete and accessible medical records.

Exclusion criteria: secondary hypertension (renal parenchymal hypertension, renovascular hypertension, primary aldosteronism, etc.);

having received targeted interventions such as antihypertensive, hypoglycemic, and lipid-lowering treatments within the past month; having diseases that may affect metabolic indicators, such as severe liver and kidney dysfunction, malignant tumors, thyroid dysfunction, or acute gout attacks; long-term heavy drinkers (men with daily alcohol consumption >40 g, women with daily alcohol consumption >20 g); mental illness or cognitive impairment; and pregnant or lactating women.

This retrospective study has been approved by the Ethics Committee of Huzhou Rehabilitation Hospital. After strict screening based on the above criteria, 428 middle-aged and elderly hypertensive patients who underwent health checkups at Huzhou Rehabilitation Hospital from January 2021 to December 2023 were ultimately included. Subjects were grouped according to their BMI: underweight/normal (BMI < 24 kg/m²) group with 146 cases, an overweight (BMI 24-28 kg/m²) group with 128 cases, and an obese (BMI > 28 kg/m²) group with 154 cases. The patient selection process is shown in **Figure 1**.

Methods

All subjects abstained from meat for 3 days prior to the examination, consuming only a vegetarian diet. The following day, all indicators were tested on an empty stomach.

Data collection

BMI, lipid profiles (triglyceride [TG], total cholesterol [TC], low-/high-density lipoprotein cholesterol [LDL-C/HDL-C]), BP (systolic and diastolic

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BP [SBP/DBP]), serum UA, and hepatic steatosis incidence were extracted and verified using a medical record retrieval system. All indicators were tested uniformly on the day of the physical examination and served as the primary endpoint.

Outcome measures

BMI was calculated according to the formula "BMI = weight (kg)/height² (m²)" after measuring the subject's height and weight. Blood lipid indicators, such as TG, TC, LDL-C, and HDL-C levels were measured using a fully automated biochemical analyzer. SBP and DBP were measured at rest using a standard mercury sphygmomanometer. Measurements were repeated twice per subject, with a 5-minute interval, and the average value was used for data analysis. UA level was detected using a fully automated biochemical analyzer.

Diagnostic criteria for the incidence of hepatic steatosis were as follows. Color Doppler ultrasound of the upper abdomen (including the liver) showed mild to moderate hepatomegaly with a smooth and regular outline, diffuse increased echogenicity of the liver parenchyma, and unclear deep liver tissue echoes under standard sensitivity.

Statistical analysis

Quantitative data conforming to a normal distribution were described using mean \pm standard deviation, and independent samples t-tests were used for comparisons between groups. Non-normally distributed data were expressed as median (interquartile range), and Mann-Whitney U tests were used for comparisons between groups. Categorical data were expressed as percentages, and chi-square tests were used for comparisons between groups. One-way ANOVA was used for comparisons among multiple groups. Pearson or Spearman correlation coefficients were used to analyze the correlation between BMI and each observed indicator. Univariate analysis and multivariate logistic regression models were used to screen for influencing factors of fatty liver. All study data were imported into SPSS 20.0 software for statistical analysis, and a *p*-value <0.05 was considered significant.

Results

Comparison of baseline characteristics among the three groups

Baseline clinical characteristics were well balanced across the study cohorts, and no significant differences were detected (all *P*>0.05). See **Table 1**.

Comparison of blood lipid levels among the three groups

TG and LDL-C levels were significantly higher in the overweight and obese groups (both *P*<0.05); and the levels of these indicators were higher in the obese group than in the overweight group (*P*<0.05). HDL-C levels showed a significant decreasing trend in the three groups (*P*<0.05). There was no significant difference in TC levels among the three groups (*P*>0.05; **Figure 2**).

Correlation between BMI and baseline blood lipid levels

Correlation analysis showed that BMI was positively correlated with TG and LDL-C in the physical examination participants (*r*=0.257, *P*<0.001; *r*=0.304, *P*<0.001), and negatively correlated with HDL-C (*r*=-0.286, *P*<0.001). There was no significant correlation between BMI and TC (*r*=0.019, *P*=0.692; **Figure 3**).

Comparison of BP levels among the three groups

SBP and DBP were highest in the obese group, followed by the overweight group, and lowest in the underweight/normal weight group (both *P*<0.05; **Figure 4**).

Association between BMI and BP

Pearson correlation analysis showed that BMI was significantly positively correlated with SBP and DBP in the physical examination participants (*r*=0.398, *P*<0.001; *r*=0.307, *P*<0.001; **Figure 5**).

Comparison of UA levels and correlation analysis

UA levels revealed a progressively significant increasing trend in the three groups (*P*<0.05).

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Table 1. Baseline data of the three groups of health examinees

Indicator	Underweight/normal group (n=146)	Overweight (n=128)	Obese (n=154)	χ^2	P
Age (years)				1.555	0.460
<58 (n=207)	70 (47.95)	57 (44.53)	80 (51.95)		
≥58 (n=221)	76 (52.05)	71 (55.47)	74 (48.05)		
Sex				0.091	0.956
Male (n=224)	75 (51.37)	68 (53.13)	81 (52.60)		
Female (n=204)	71 (48.63)	60 (46.87)	73 (47.40)		
Educational level				1.764	0.414
Below senior high school (n=278)	90 (61.64)	82 (64.06)	106 (68.83)		
Senior high school or above (n=150)	56 (38.36)	46 (35.94)	48 (31.17)		
Residence				1.260	0.533
Urban (n=327)	111 (76.03)	102 (79.69)	114 (74.03)		
Rural (n=101)	35 (23.97)	26 (20.31)	40 (25.97)		
Smoking				3.296	0.192
No (n=263)	93 (63.70)	84 (65.63)	86 (55.84)		
Yes (n=165)	53 (36.30)	44 (34.37)	68 (44.16)		
Alcoholism				0.470	0.790
No (n=295)	98 (67.12)	88 (68.75)	109 (70.78)		
Yes (n=133)	48 (32.88)	40 (31.25)	45 (29.22)		

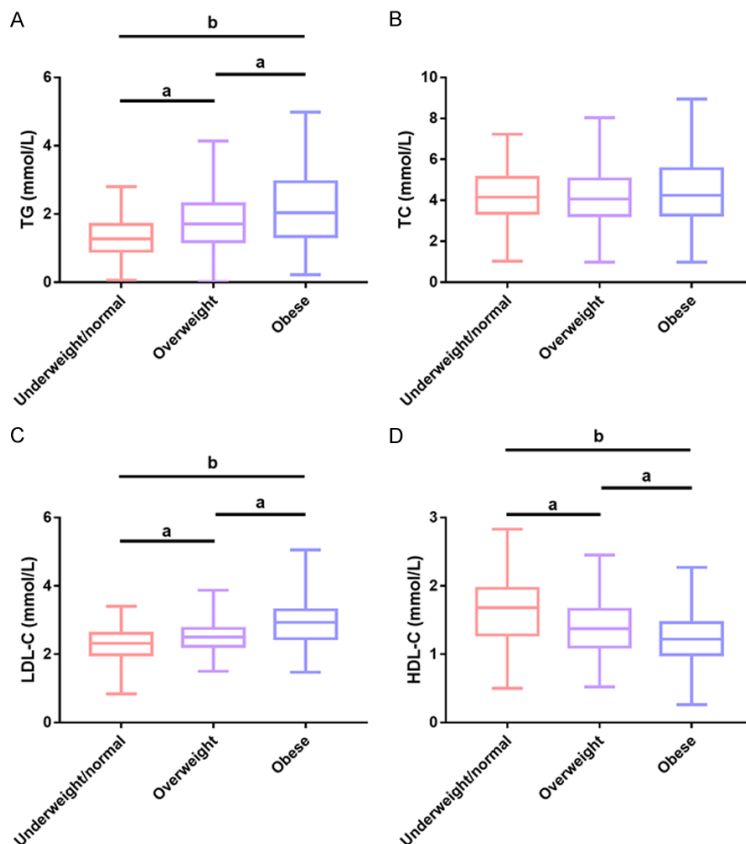


Figure 2. Comparison of blood lipid levels among the three groups. A. TG levels among the three groups. B. TC levels among the three groups. C. LDL-C levels among the three groups. D. HDL-C levels among the three groups. Note: ^aP<0.05, ^bP<0.01. TG, triglyceride; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol.

Pearson correlation analysis confirmed a positive association between BMI and UA levels in the physical examination participants ($r=0.273$, $P<0.001$; **Figure 6**).

Comparison of incidence of hepatic steatosis among the three groups

The hepatic steatosis incidence in the underweight/normal weight group (53 cases), overweight group (70 cases), and obese group (112 cases) was 36.3%, 54.7%, and 72.7%, respectively. Data showed that the incidence of hepatic steatosis was lowest in the underweight/normal weight population, followed by the overweight population, and highest in the obese population ($P<0.05$; **Figure 7**).

Analysis of influencing factors of hepatic steatosis

With hepatic steatosis as the dependent variable, and age, sex, BMI, TG, TC, LDL-C, HDL-C,

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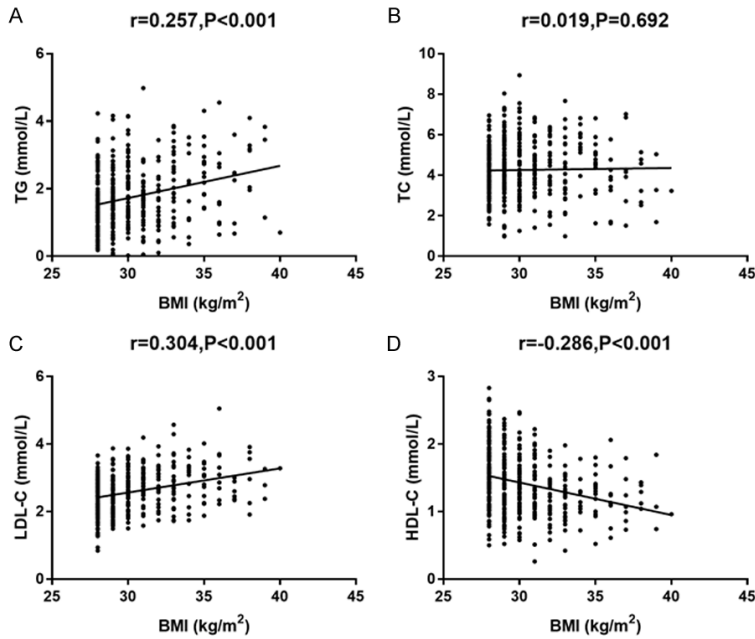


Figure 3. Correlation between BMI and blood lipid levels in the health check-up population. A. Correlation between BMI and TG. B. Correlation between BMI and TC. C. Correlation between BMI and LDL-C. D. Correlation between BMI and HDL-C. Note: BMI, body mass index; TG, triglyceride; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol.

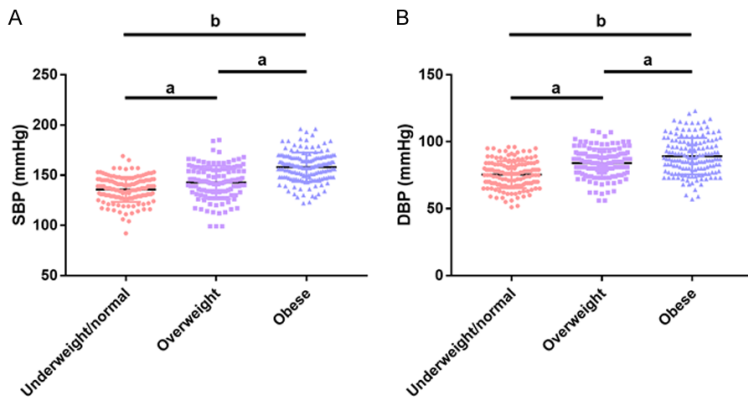


Figure 4. Comparison of Blood pressure levels among the three groups. A. SBP among the three groups. B. DBP among the three groups. Note: ^aP<0.05; ^bP<0.01. SBP, systolic blood pressure; DBP, diastolic blood pressure.

SBP, DBP, UA, education level, place of residence, smoking, and alcohol consumption as independent variables, univariate analysis was used to explore the risk factors for hepatic steatosis in middle-aged and elderly hypertensive individuals. Results indicated that BMI, TG, HDL-C, SBP, UA, and alcohol consumption were closely related to hepatic steatosis in this population (all $P<0.05$). Further multivariate regres-

sion analysis was used to screen independent influencing factors for hepatic steatosis, identifying age, BMI, SBP, UA, and alcohol consumption as independent risk factors (all $P<0.05$). Among them, obesity had the highest risk of developing the disease (OR=3.219), and the risk intensity of the other risk factors, from highest to lowest, was elevated UA (OR=2.209), long-term alcohol consumption (OR=1.786), high SBP (OR=1.646), and advanced age (OR=1.661; **Tables 2-4**).

Discussion

Hypertension affects nearly 33% of adults worldwide. Despite breakthroughs in drug treatment, the premature mortality among hypertensive patients remains irreversible [17]. Hypertensive patients are also highly susceptible to various complications, which not only increase the difficulty of treatment but also hinder the improvement of their condition [18]. The results of this study revealed that TG and LDL-C levels showed a significant progressively increasing trend in the three groups. In addition, among the middle-aged and elderly hypertensive individuals who underwent physical examinations, BMI was positively correlated with TG and LDL-C. Conversely, HDL-C levels were highest in the underweight/normal weight group, followed by the overweight group, and lowest in the obese group; BMI was negatively correlated with HDL-C in elderly and middle-aged hypertensive individuals undergoing physical examinations. The above results indicate that among the middle-aged and elderly hypertensive individuals undergoing physical examinations, the obese group generally has dyslipidemia, and the risk of developing hyperlipidemia may be increased. The study by Tang

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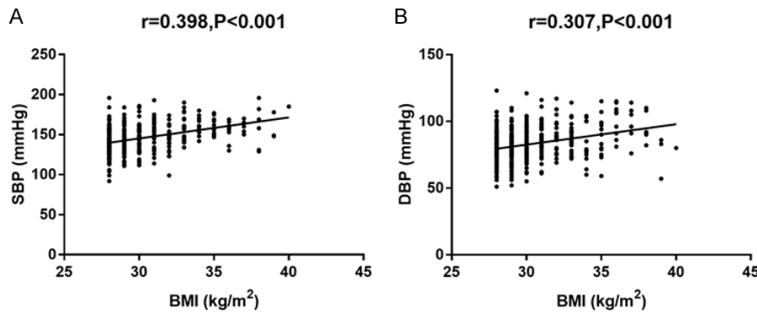


Figure 5. Association between BMI and BP in the health check-up population. A. Association between BMI and SBP. B. Association between BMI and DBP. Note: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure.

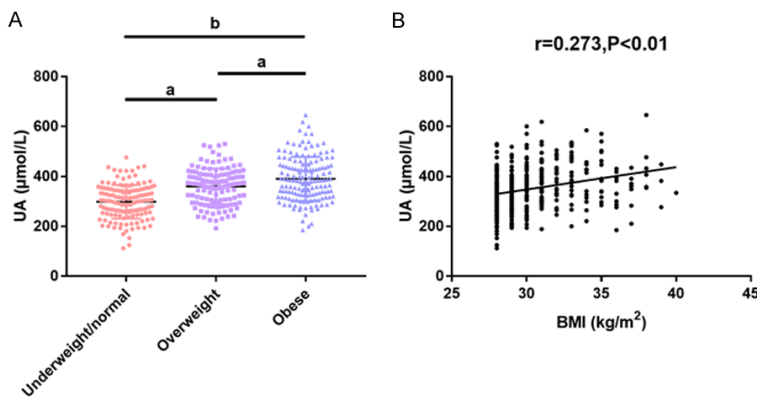


Figure 6. Comparison of UA levels and correlation analysis. A. UA levels. B. Correlation between BMI and UA in individuals undergoing health check-ups. Note: * $P < 0.05$; ^b $P < 0.01$. UA, uric acid; BMI, body mass index.

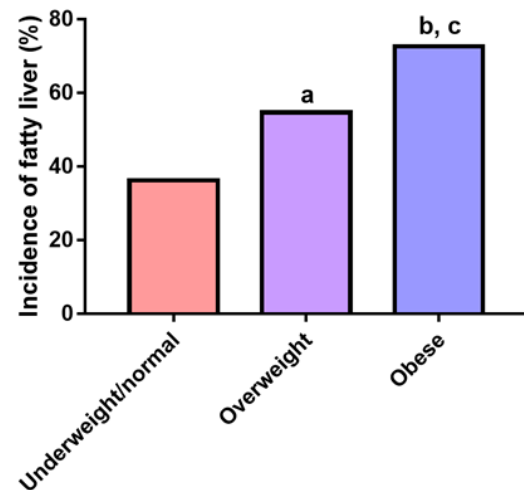


Figure 7. Comparison of hepatic steatosis incidence among the three groups. Note: * $P < 0.05$, ^b $P < 0.01$ vs. underweight/normal group; ^c $P < 0.05$ vs. overweight group.

et al. [19] confirmed that there is a significant interaction between high BMI and dyslipidemia. Low body weight is an independent protective factor against hypertension, while high BMI (overweight, obesity, etc.) and dyslipidemia are risk factors for hypertension, which is consistent with the results of this study. The superimposed interaction between dyslipidemia and high BMI may be related to the common mechanism by which the two cause BP elevation, such as both causing arterial endothelial damage, which in turn induces hypertension [20]. Additionally, dyslipidemia can also damage cell membrane structure, reduce cell membrane permeability, and damage renal microvessels, further promoting the occurrence and development of hypertension [21]. From the perspective of BP indicators, the levels of SBP and DBP showed a significant progressive increase from the underweight/normal weight group to the overweight and obese groups, and BMI

was positively correlated with both SBP and DBP. This indicates that middle-aged and elderly obese people with hypertension often have abnormally high BP. A large number of previous studies have confirmed that high BMI is closely related to hypertension [22]. From the perspective of mechanism of action, abdominal obesity interferes with the human endocrine and immune systems. Disorders of the leptin-melanocortin pathway, increased oxidative stress, changes in hemodynamics, and imbalances in vascular endothelial contraction and relaxation factors are all important contributing factors to the association between the two [22]. The study by Landi et al. [23] also reached similar conclusions, confirming that the average levels of SBP and DBP increase significantly linearly with the increase of BMI. The UA level also shows the same trend: the UA level increases significantly from the underweight/normal

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Table 2. Univariate analysis to evaluate hepatic steatosis-associated drivers in middle-aged and elderly people with hypertension

Indicator	Hepatic steatosis group (n=235)	Non-hepatic steatosis group (n=193)	χ^2	P
Age (years)			7.047	0.008
<58 (n=207)	100 (42.55)	107 (55.44)		
≥58 (n=221)	135 (57.45)	86 (44.56)		
Sex			1.859	0.173
Male (n=224)	130 (55.32)	94 (48.70)		
Female (n=204)	105 (44.68)	99 (51.30)		
BMI (kg/m ²)			30.980	<0.001
<24 (n=146)	53 (22.55)	93 (48.19)		
≥24 (n=282)	182 (77.45)	100 (51.81)		
TG (mmol/L)			4.992	0.026
<1.64 (n=214)	106 (45.11)	108 (55.96)		
≥1.64 (n=214)	129 (54.89)	85 (44.04)		
TC (mmol/L)			0.462	0.497
<4.17 (n=214)	114 (48.51)	100 (51.81)		
≥4.17 (n=214)	121 (51.49)	93 (48.19)		
LDL-C (mmol/L)			0.489	0.485
<2.54 (n=212)	120 (51.06)	92 (47.67)		
≥2.54 (n=216)	115 (48.94)	101 (52.33)		
HDL-C (mmol/L)			4.240	0.040
<1.39 (n=212)	127 (54.04)	85 (44.04)		
≥1.39 (n=216)	108 (45.96)	108 (55.96)		
SBP (mmHg)			5.209	0.023
<145.00 (n=198)	97 (41.28)	101 (52.33)		
≥145.00 (n=230)	138 (58.72)	92 (47.67)		
DBP (mmHg)			0.619	0.432
<83.00 (n=213)	121 (51.49)	92 (47.67)		
≥83.00 (n=215)	114 (48.51)	101 (52.33)		
UA (μmol/L)			14.600	<0.001
<344.00 (n=207)	94 (40.00)	113 (58.55)		
≥344.00 (n=221)	141 (60.00)	80 (41.45)		
Educational level			0.289	0.591
Below senior high school (n=278)	150 (63.83)	128 (66.32)		
Senior high school or above (n=150)	85 (36.17)	65 (33.68)		
Residence			0.625	0.429
Urban (n=327)	183 (77.87)	144 (74.61)		
Rural (n=101)	52 (22.13)	49 (25.39)		
Smoking			1.163	0.281
No (n=263)	139 (59.15)	124 (64.25)		
Yes (n=165)	96 (40.85)	69 (35.75)		
Alcoholism			6.317	0.012
No (n=295)	150 (63.83)	145 (75.13)		
Yes (n=133)	85 (36.17)	48 (24.87)		

Note: BMI, body mass index; TG, triglyceride; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; UA, uric acid.

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Table 3. Assignments

Indicator	Variable	Assignment
Age (years)	X1	<58=0, ≥58=1
BMI (kg/m ²)	X2	<24=0, ≥24=1
TG (mmol/L)	X3	<1.64=0, ≥1.64=1
HDL-C (mmol/L)	X4	<1.39=0, ≥1.39=1
SBP (mmHg)	X5	<145.00=0, ≥145.00=1
UA (μmol/L)	X6	<344.00=0, ≥344.00=1
Alcoholism	X7	No=0, yes=1
Hepatic steatosis	Y	No=0, yes=1

Note: BMI, body mass index; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; UA, uric acid.

weight group to the overweight and obese groups, and the BMI is significantly positively correlated with UA. This indicates that with an increase in weight, the risk of hyperuricemia will increase to a certain extent. The study by Zeng et al. [24] also pointed out that the increase of UA level was closely related to an increased risk of obesity, which supports the conclusion of this study. Other studies have shown that hyperuricemia in obese people is associated with hyperinsulinemia or insulin resistance to a certain extent [25]. The study found that the prevalence of hepatic steatosis in the underweight/normal weight group, overweight group, and obese group was 36.3%, 54.7%, and 72.7%, respectively. It can be seen that the risk of hepatic steatosis increases significantly with abnormally high BMI. The study by Fan et al. [26] also confirmed that high BMI is an independent dose-dependent risk factor for hepatic steatosis, suggesting that hepatic steatosis prevention and control can be carried out through weight management. A large-scale population cohort study further showed that the difference between the highest BMI in a person's lifetime and the BMI at age 20 was closely related to the increased risk of hepatic steatosis. Hepatic steatosis usually occurs after the body weight reaches its peak [27]. The study by Kuwabara et al. [28] on Japanese and American populations found that high BMI is highly correlated with the increased risk of hypertension, diabetes, dyslipidemia, and hyperuricemia. This conclusion is basically consistent with this study. This study further explored the risk factors for hepatic steatosis in middle-aged and elderly people with hypertension during physical examinations and found

that obesity is one of the strongest independent risk factors for fatty liver. The risk of hepatic steatosis in obese people is 3.22 times that of people with normal weight. At the same time, elevated serum UA (≥344.00 μmol/L) is also a key independent risk factor. The probability of hepatic steatosis in people with hyperuricemia is 2.21 times that of people with low UA. In addition, alcohol consumption, increased SBP (≥145.00 mmHg) and increasing age (≥58 years) all independently and significantly affect the occurrence of hepatic steatosis. Han et al. [29] conducted a study on healthy people undergoing physical examinations in Chengdu and listed gender, age, BMI, abnormal lipid metabolism, hypertension, hyperglycemia, hyperuricemia and metabolic syndrome as influencing factors for hepatic steatosis. This is similar to our results. Yuan et al. [30] conducted a risk factor survey on 73,566 patients with metabolic-associated fatty liver disease (MAFLD) in Beijing, China. The study confirmed that gender, urban residence, and advanced age were risk factors, while higher education was a protective factor and daily alcohol consumption was not significantly associated with the disease, which differs from the findings of this study [30]. Rieki et al. [31] investigated the influencing factors of MAFLD in overweight children aged 2-16 years and proposed that hypertriglyceridemia and decreased HDL-C were common risk factors for both men and women, effectively supplementing the findings of this study. This study has some limitations and needs to be further improved. First, this study was a cross-sectional survey, which can only clarify the correlation between BMI and various metabolic indicators, but cannot determine causality. Further prospective longitudinal studies are needed to clarify the causal relationships between variables. Second, the study did not systematically collect key lifestyle data such as dietary structure and exercise levels. Relevant information needs to be supplemented in future studies to avoid confounding bias. Third, the study lacked relevant indicators such as fasting blood glucose and insulin resistance. Supplementing these indicators will allow for a more comprehensive interpretation of the results from the perspective of metabolic syndrome.

In summary, BMI in middle-aged and elderly individuals with hypertension is significantly

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Table 4. Multivariate analysis to evaluate hepatic steatosis-associated contributors in middle-aged and elderly individuals with hypertension

Indicator	B	SE	WALD	P	OR	95% CI
Age (years)	0.507	0.213	5.672	0.017	1.661	1.094-2.521
BMI (kg/m ²)	1.169	0.224	27.235	<0.001	3.219	2.075-4.994
TG (mmol/L)	0.395	0.214	3.423	0.064	1.485	0.977-2.257
HDL-C (mmol/L)	-0.393	0.212	3.432	0.064	0.675	0.446-1.023
SBP (mmHg)	0.499	0.214	5.422	0.020	1.646	1.082-2.505
UA (μmol/L)	0.792	0.213	13.799	<0.001	2.209	1.454-3.356
Alcoholism	0.580	0.235	6.112	0.013	1.786	1.128-2.828

Note: BMI, body mass index; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; UA, uric acid; SE, Standard Error; OR, Odds Ratio; 95% CI, 95% Confidence Interval.

correlated with blood lipid levels, BP, and UA levels. The risk of hepatic steatosis increases with increasing BMI. Scientific weight management for overweight and obese hypertensive patients, adjusting their BMI to a normal range, can help prevent the occurrence of hepatic steatosis.

Disclosure of conflict of interest

None.

Address correspondence to: Li Liu, Department of General Surgery, First People's Hospital of Huzhou, Huzhou 313000, Zhejiang, China. Tel: +86-0572-2039343; E-mail: liuli_hzyy@163.com

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