Am J Transl Res 2025;17(1):200-210
www.ajtr.org /ISSN:1943-8141/AJTR0160280

Original Article

Body shape index: an index for early target organ damage in hypertension

Wangjiang Wei[1], Ping Ding[2]

[1]*Department of Cardiology, Beijing Ditan Hospital, Capital Medical University, Beijing 100015, China;* [2]*Department of Critical Care Medicine, No. 988 Hospital of The PLA Joint Logistic Support Force (PLAJLSF), Zhengzhou 450000, Henan, China*

Received September 2, 2024; Accepted December 11, 2024; Epub January 15, 2025; Published January 30, 2025

Abstract: Objective: To investigate the relationship between obesity-related indices and target organ damage in hypertension, with the goal of improving damage prevention and treatment strategies. Methods: This retrospective study included 150 hypertension cases from January 2022 to December 2023 treated at Beijing Ditan Hospital, including 72 cases of isolated hypertension, 48 with hypertension complicated by kidney damage, 47 with hypertension and left ventricular hypertrophy, and 44 with hypertension and carotid atherosclerosis. Clinical indicators and obesity-related indices were collected to analyze their correlation with target organ damage in hypertension. Results: A Body Shape Index (ABSI) (OR: 1.003 [1.001-1.913], P=0.007), BMI (OR: 1.054 [1.005-1.694], P=0.023), and BRI (OR: 0.562 [0.287-0.934], P=0.034) were independently associated with hypertensive kidney damage. Age (OR: 1.037 [1.000-1.635], P=0.034), ABSI (OR: 0.901 [0.882-0.998], P=0.034), and blood pressure grading (OR: 1.473 [1.175-1.845], P=0.002) were independent risk factors for hypertensive left ventricular hypertrophy. ABSI (OR: 1.012 [1.001-1.623], P=0.032) and smoking history (OR: 0.892 [0.781-0.998], P=0.021) were independent risk factors for hypertensive carotid artery plaque formation. Conclusion: ABSI is an independent risk factor for target organ damage in hypertension.

Keywords: Obesity-related index, target organ injury, hypertension

Introduction

Hypertension is a common chronic disease in clinical practice. Statistics show that the number of adults with hypertension in China exceeded 200 million, and with the aging population, this number is expected to rise, making prevention and treatment increasingly challenging [1]. Cardiovascular disease, the leading cause of death worldwide, has high blood pressure as one of its main risk factors [2]. Chronic hypertension can cause structural and functional damage to multiple organs such as the heart, brain, kidneys, and blood vessels. If left untreated, it may result in disability or even death, posing a significant threat to the health and well-being of hypertensive patients [3]. Numerous clinical studies have highlighted the importance of strict blood pressure control in preventing organ damage and cardiovascular events in patients with hypertension [4-7]. Subclinical target organ damage, caused by early hypertension, is reversible with timely detection and treatment [8]. Therefore, early and reliable assessment of target organ damage is crucial for clinical practice, as it can guide treatment strategies and improve the prognosis of hypertensive patients.

Overweight and obesity are among the most prevalent conditions globally, affecting about 3/4 of the global population [9]. Statistics indicate that 75% to 80% of individuals with hypertension are overweight or obese to varying degrees [10]. The Framingham Heart Study shows that 78% of primary hypertension cases in men and 65% in women are attributable to weight gain [11]. Additionally, research also indicates that obesity is a major risk factor for hypertension-related kidney disease [12].

Obesity-related indices are commonly used to assess and quantify obesity, considering fac-

https://doi.org/10.62347/FMEB5040

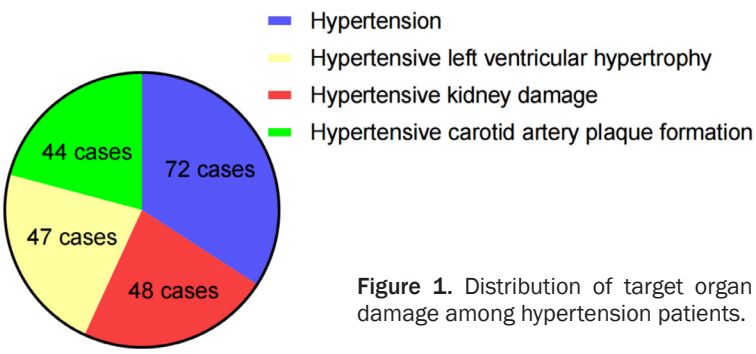

Figure 1. Distribution of target organ damage among hypertension patients.

tors such as weight, height, body composition, and sometimes additional measurements like waist circumference or skinfold thickness [13]. Some commonly used obesity-related indices include body Mass Index (BMI), waist-to-Hip Ratio (WHR), waist-to-Height Ratio (WHtR), Body Roundness Index (BRI), body fat percentage. Research has shown that WHtR is one of the predictive factors for cardiovascular events and diabetes [14]. Sato [15] suggests that ABSI has a good linear relationship with hypertension, coronary heart disease, and all-cause mortality. However, other research indicates that there is no significant difference in BMI levels between patients with hypertensive target myocardial hypertrophy and those with normal blood pressure. These inconsistent findings highlight the need for further investigation to confirm these relationships [16-18].

Currently, there is limited research on the relationship between obesity-related indices and target organ damage in hypertension. This study aims to explore the predictive value of obesity-related indices for identifying target organ damage in hypertension, with the goal of enhancing the prevention and treatment of such damage.

Methods

Case selection

A total of 150 patients with hypertension treated at Beijing Ditan Hospital from January 2022 to December 2023 were included in this study. Among them, 72 cases were diagnosed with isolated hypertension, 48 with hypertension complicated by kidney damage, 47 with hypertension and left ventricular hypertrophy, and 44 with hypertension and carotid atherosclerosis (**Figure 1**). This study was approved by the Ethics Committee of Beijing Ditan Hospital.

Inclusion criteria: (1) Patients met the diagnostic criteria outlined in the *Guidelines for the Diagnosis and Treatment of Hypertension* [19]; (2) Patients aged more than 18 years; (3) Patients with no severe hepatorenal dysfunction; (4) Complete clinical data required for the study.

Exclusion criteria: (1) Patients with neurological or mental diseases; (2) Patients with complications of coronary heart disease, myocardial infarction, heart failure, or stroke within the past 6 months; (3) Patients with acute or chronic inflammatory diseases; (4) Patients with non-hypertensive renal diseases of clear etiology, such as diabetic nephropathy or glomerulonephritis; (5) Patients diagnosed with secondary hypertension; (6) Patients with tumor or those undergoing cancer treatment.

Data collection

Hypertension: The diagnostic criteria for hypertension follow the Chinese *Guidelines for the Prevention and Treatment of Hypertension (2018 revised edition)* [19]. Patients are diagnosed with hypertension if they meet one of the following conditions: (1) In the absence of antihypertensive treatment, the systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg in three separate measurements on different days; (2) Patients with a history of hypertension who are currently taking antihypertensive medication should still be diagnosed with hypertension if their blood pressure is below 140/90 mmHg.

Hypertension with renal damage: Renal function damage was assessed using indicators such as urine creatinine, estimated glomerular filtration rate (eGFR), urine N-acetyl-β-amino glucosidase (NAG), blood creatinine, blood urea nitrogen, and urine microalbumin [20-22]. Patients are diagnosed with hypertension with renal damage if they meet one of the following criteria: 1) eGFR<59 ml/(min·1.73 m^2); 2) Elevated serum creatinine: males >115 μmol/L (1.3 mg/dL), females >107 μmol/L (1.2 mg/dL); 3) Microalbuminuria >30 mg/24 h; 4) Albumin/creatinine ratio ≥30 mg/g (3.5 mg/mmol).

Hypertension with left ventricular hypertrophy: Left ventricular hypertrophy (LVH) is diagnosed based on echocardiogram results [23, 24]. The left ventricular mass index (LVMI) is calculated using Deiereux's formula for ventricular weight correction, based on measurements of interventricular septal thickness (IVST), posterior wall thickness (PWT), and left ventricular end-diastolic diameter (LVDd) taken three times consecutively [25]. LVMI>115 g/m^2 for males and >95 g/m^2 for females is considered as left ventricular hypertrophy.

Hypertension with carotid atherosclerosis: Carotid atherosclerosis is diagnosed through carotid artery color Doppler ultrasound [26, 27]. If the ultrasound shows intima-media thickening (intima-media thickness [IMT] ≥0.9 mm) or the presence of atherosclerotic plaques in the artery, the patient can be diagnosed with carotid atherosclerosis.

Outcome measurement

Main outcomes: obesity-related indices involved in this study include waist circumference (WC), BMI, WHtR, BRI, ABSI. The formulas for these indices are as follows: BMI = weight (kg)/height $(m)^2$; WHtR = WC (cm)/height (cm); BRI = 364.2 - (365.5 × (1 - (WC/(2 × π × height)))); ABSI = WC (m)/(BMI $(kg/m^2)^{1.5}$ × Height $(m)^{0.5}$).

Secondary outcomes include general demographics, including age, gender, smoking history, alcohol consumption, past disease history, height, weight, waist circumference, and blood pressure.

Statistical analysis

SPSS 22.0 was used for statistical analysis. The measured data were expressed as mean ± standard deviation, with comparisons between two groups made using the t-test. Counted data were expressed by the number of cases/percentage (n%), and differences were analyzed using the χ^2 test. Binary logistic regression analysis was used to evaluate whether various obesity-related indices are independent risk factors for target organ injury of hypertension. Receiver Operating Characteristic (ROC) curves were employed to evaluate the discriminatory ability of obesity-related indices for predicting target organ damage in hypertension. P<0.05 was considered significant.

Result

Comparison of clinical data between hypertension and hypertensive kidney damage groups

No significant differences were observed between the hypertensive kidney damage group and the hypertension group in terms of age, smoking history, alcohol consumption, blood pressure grading, hypertension course, blood glucose, LDL, TG, TC, or Lp(a) levels (all P>0.05). However, significant differences were found between the two groups regarding gender, family history, BMI, HDL, eGFR, and UA (all P<0.05) (**Table 1**).

Comparison of obesity-related indicators between hypertension and hypertensive kidney damage groups

Figure 2 presents the mean values of obesity-related anthropometric indices. The mean values for ABSI and BRI were significantly higher in the hypertensive kidney damage group compared to those in the hypertension group. Specifically, the mean ABSI in the hypertensive kidney damage group was (0.078±0.012), compared to (0.067±0.02) in the hypertension group (P<0.001). The mean BRI was notably higher in the hypertensive kidney damage group at (3.54±0.37) compared to (3.33±0.38) in the hypertension group (P=0.034). There were no significant differences in mean WHtR or WC between the two groups (all P>0.05).

Comparison of clinical data between hypertension and hypertensive left ventricular hypertrophy groups

Significant differences were observed between the hypertension and hypertensive left ventricular hypertrophy groups in terms of age, blood pressure grading, Lp(a), eGFR, UA, and LVMI (P<0.05). However, no significant differences were found in gender, smoking history, BMI, blood glucose, LDL, HDL, TG or TC levels between the two groups (P>0.05) (**Table 2**).

Comparison of obesity-related indicators between hypertension and hypertensive left ventricular hypertrophy groups

Figure 3 presents the mean values of obesity-related anthropometric indices. The mean values for ABSI were significantly higher in the hypertensive left ventricular hypertrophy group compared to those in the hypertension group.

Table 1. Comparison of clinical data between hypertension and hypertensive kidney damage groups

Clinical data	Hypertension group (n=72)	Hypertensive kidney damage group (n=48)	P
Age	58.68±6.32	59.04±8.88	0.795
Male	14 (19.44%)	26 (54.17%)	0.000
Smoking history	14 (19.44%)	10 (20.83%)	0.852
Drinking history	10 (13.89%)	8 (16.67%)	0.676
Family History	3 (4.17%)	9 (18.75%)	0.009
Blood pressure grading			0.610
Level 1	24 (33.33%)	12 (25.00%)	
Level 2	25 (34.72%)	18 (37.50%)	
Level 3	23 (31.95%)	18 (37.50%)	
Hypertension course	6.16±2.24	6.74±2.13	0.160
BMI	24.28±2.09	23.08±1.49	0.001
Blood glucose	5.00±0.37	5.07±0.50	0.449
LDL	2.79±0.42	2.70±0.40	0.183
HDL	1.28±0.20	1.11±0.11	0.000
TG	1.58±0.51	1.74±0.58	0.109
TC	4.89±0.60	4.76±0.49	0.226
Lp(a)	174.19±52.85	162.42±61.66	0.266
eGFR	104.40±14.82	91.28±16.62	0.000
UA	378.23±51.50	417.62±72.08	0.001

Note: BMI: Body mass index; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; TG: Triglyceride; TC: Total cholesterol; Lp(a): Lipoprotein(a); eGFR: Estimated glomerular filtration rate; UA: Uric acid.

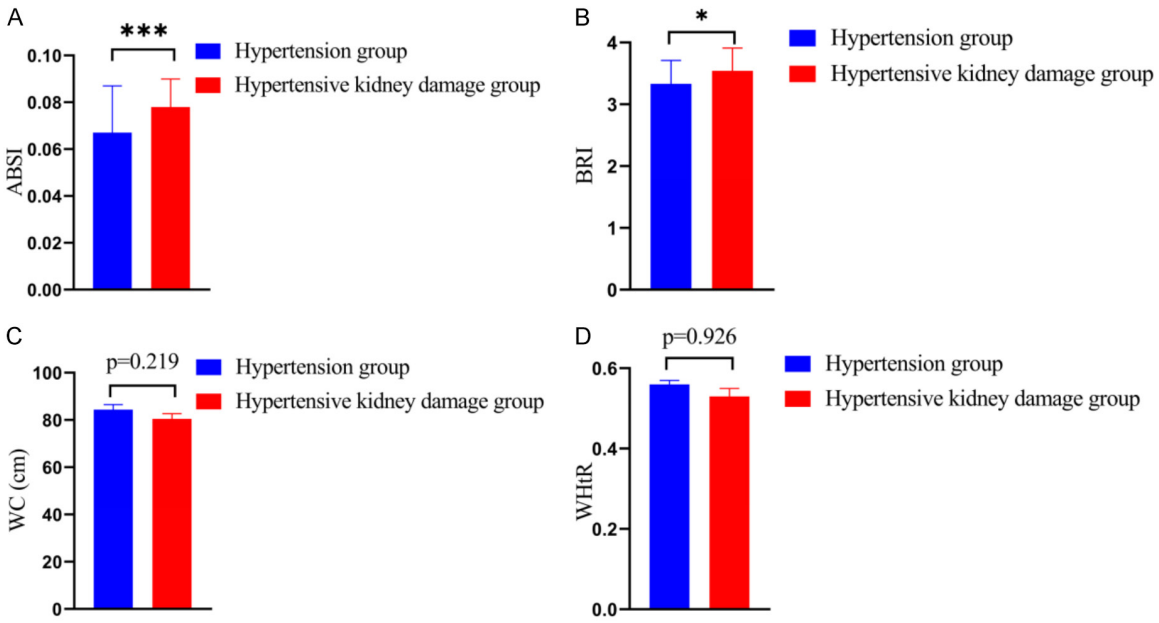

Figure 2. Comparison of obesity related indicators between hypertension and hypertensive kidney damage groups. A. ABSI: A Body Shape Index. B. BRI: Body Roundness Index. C. WC: Waist circumference. D. WHtR: Waist-to-Height Ratio. Compared to control group, *P<0.05, ****P<0.001*.

Specifically, the mean ABSI in the hypertensive left ventricular hypertrophy group was (0.081± 0.002), compared to (0.071±0.0022) in the hypertension group (P<0.001). No significant differences were found in mean WHtR, BRI, or WC between the two groups (all P>0.05).

Table 2. Comparison of clinical data between hypertension and hypertensive left ventricular hypertrophy groups

Clinical data	Hypertension group (n=72)	Hypertensive left ventricular hypertrophy group (n=47)	P
Age	58.65±10.65	62.03±12.34	0.000
Male	34 (47.22%)	25 (53.19%)	0.534
Smoking history	14 (19.44%)	9 (19.15%)	0.435
Drinking history	10 (13.89%)	6 (12.77%)	0.679
Family History	9 (12.5%)	8 (17.02%)	0.302
Blood pressure grading			0.000
Level 1	24 (33.33%)	7 (14.89%)	
Level 2	25 (34.72%)	26 (55.32%)	
Level 3	23 (31.95%)	14 (29.79%)	
Hypertension course	5.47±3.45	6.34±3.37	0.109
BMI	24.04±3.67	24.78±3.32	0.873
Blood glucose	5.09±0.67	5.17±0.67	0.342
LDL	2.78±0.75	2.71±0.76	0.673
HDL	1.28±0.32	1.23±0.30	0.346
TG	1.62±0.89	1.67±1.02	0.439
TC	4.79±0.91	4.68±0.88	0.763
Lp(a)	148.6 (86.4, 275.8)	192.3 (83.6, 357.1)	0.043
eGFR	102.39±25.01	94.23±23.02	0.002
UA	367.82±87.02	384.34±104.83	0.036
LVMI	82.03±21.98	112.97±25.84	0.000

Note: BMI: Body mass index; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; TG: Triglyceride; TC: Total cholesterol; Lp(a): Lipoprotein(a); eGFR: Estimated glomerular filtration rate; UA: Uric acid.

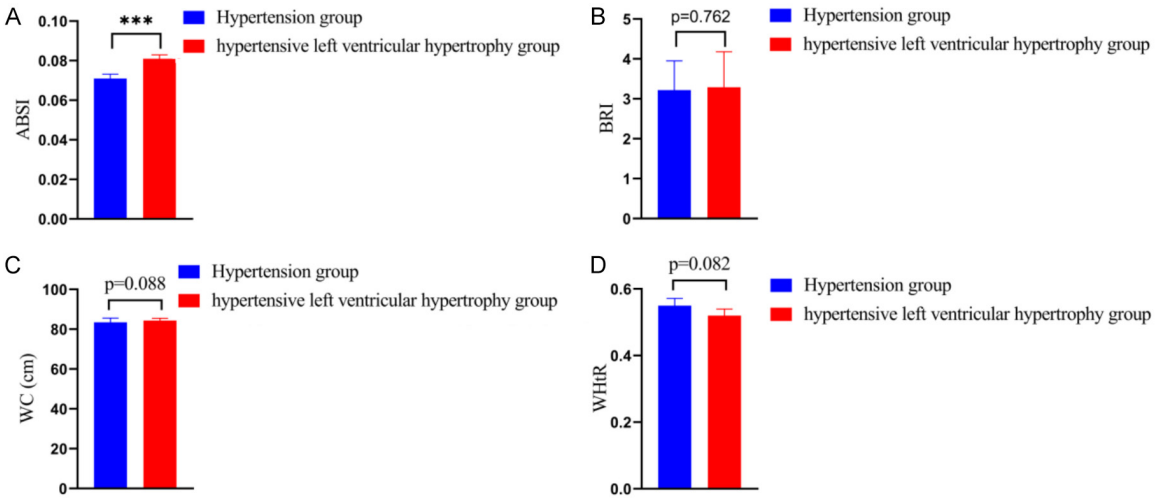

Figure 3. Comparison of obesity related indicators between hypertension and hypertensive left ventricular hypertrophy groups. A. ABSI: A Body Shape Index. B. BRI: Body Roundness Index. C. WC: Waist circumference. D. WHtR: Waist-to-Height Ratio. Compared to control group, ****P<0.001*.

Comparison of clinical data between hypertension and hypertensive carotid artery plaque formation groups

Significant differences were found between the hypertension and hypertensive carotid artery plaque formation groups in terms of gender, smoking history, family history, blood pressure grading, hypertension course, HDL, Lp(a), eGFR, and UA (all P<0.05). However, no significant differences were observed in age, BMI, blood glucose, LDL, TG and TC le-

Table 3. Comparison of clinical data between hypertension and hypertensive carotid artery plaque formation groups

Clinical data	Hypertension group (n=72)	Hypertensive carotid artery plaque formation group (n=44)	P
Age	58.65±10.65	60.21±11.36	0.134
Male	34 (47.22%)	24 (54.55%)	0.005
Smoking history	14 (19.44%)	12 (34.09%)	0.013
Drinking history	10 (13.89%)	9 (20.45%)	0.054
Family History	9 (12.5%)	10 (22.73%)	0.023
Blood pressure grading			0.012
Level 1	24 (33.33%)	9 (20.45%)	
Level 2	25 (34.72%)	21 (47.73%)	
Level 3	23 (31.95%)	14 (31.82%)	
Hypertension course	5.47±3.45	6.74±3.47	0.023
BMI	24.04±3.67	24.46±3.09	0.678
Blood glucose	5.09±0.67	5.12±0.78	0.347
LDL	2.78±0.75	2.69±0.72	0.456
HDL	1.28±0.32	1.13±0.24	0.003
TG	1.62±0.89	1.72±1.03	0.342
TC	4.79±0.91	4.61±0.91	0.265
Lp(a)	148.6 (86.4, 275.8)	182.38 (92.4, 364.6)	0.014
eGFR	102.39±25.01	97.27±20.82	0.001
UA	367.82±87.02	381.13±101.47	0.051

Note: BMI: Body mass index; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; TG: Triglyceride; TC: Total cholesterol; Lp(a): Lipoprotein(a); eGFR: Estimated glomerular filtration rate; UA: Uric acid.

vels between the two groups (P>0.05) (**Table 3**).

Comparison of obesity-related indicators between hypertension and hypertensive carotid artery plaque formation groups

Figure 4 presents the mean values of obesity-related anthropometric indices. The mean values for ABSI were significantly higher in the hypertensive carotid artery plaque formation group compared to those in the hypertension group. Specifically, the mean ABSI in the hypertensive carotid artery plaque formation group was (0.078±0.002), compared to (0.072±0.003) in the hypertension group (P<0.001). There were no significant differences in mean WHtR, BRI or WC between the groups (all P>0.05).

Multivariate logistic regression analysis of risk factors for hypertensive kidney damage

Multivariate regression analysis revealed that ABSI (OR: 1.003 [1.001-1.913], P=0.007), BMI (OR: 1.054 [1.005-1.694], P=0.023), and BRI (OR: 0.562 [0.287-0.934], P=0.034) were independent risk factors for hypertensive kidney damage (**Table 4**).

Multivariate logistic regression analysis of risk factors for hypertensive left ventricular hypertrophy

Multivariate regression analysis revealed that age (OR: 1.037 [1.000-1.635], P=0.034), ABSI (OR: 0.901 [0.882-0.998], P=0.034), and blood pressure grading (OR: 1.473 [1.175-1.845], P=0.002) were independent risk factors for hypertensive left ventricular hypertrophy (**Table 5**).

Multivariate logistic regression analysis of risk factors for hypertensive carotid artery plaque formation

Multivariate regression analysis revealed that ABSI (OR: 1.012 [1.001-1.623], P=0.032) and smoking history (OR: 0.892 [0.781-0.998], P=0.021) were independent risk factors for hypertensive carotid artery plaque formation (**Table 6**).

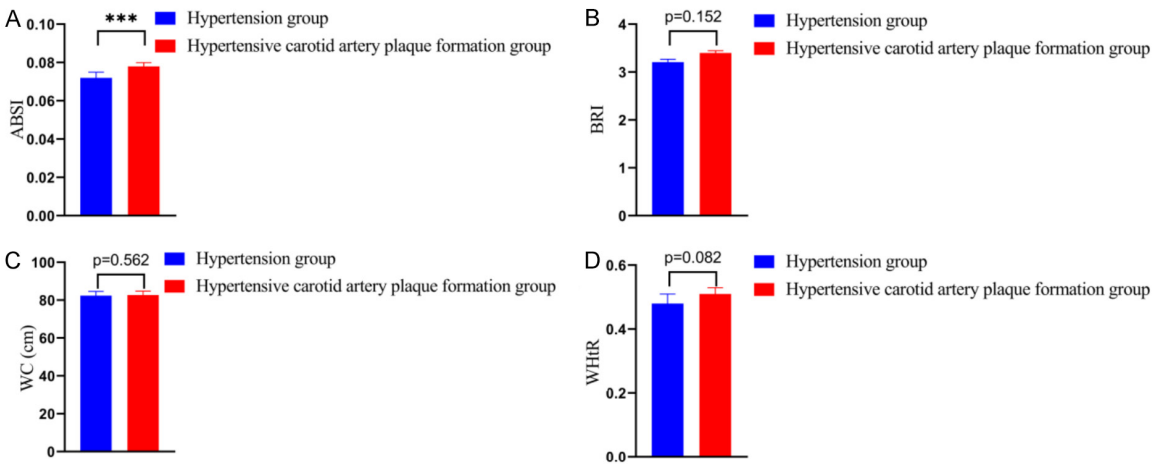

Figure 4. Comparison of obesity related indicators between hypertension and hypertensive carotid artery plaque formation groups. A. ABSI: A Body Shape Index. B. BRI: Body Roundness Index. C. WC: Waist circumference. D. WHtR: Waist-to-Height Ratio. Compared to control group, ***$P<0.001$.

Table 4. Multivariate logistic regression analysis of risk factors for hypertensive kidney damage

	β	SE	Wald	P	OR	95% CI
ABSI	3.463	1.230	7.926	0.005	31.914	2.864-355.655
BMI	-0.477	0.178	7.212	0.007	0.620	0.438-0.879
BRI	-0.653	0.238	5.396	0.034	0.562	0.287-0.934
UA	0.001	0.001	1.794	0.194	1.002	0.998-1.002
Gender	0.096	0.178	0.387	0.569	1.195	0.782-1.654
Family History	0.351	0.232	1.976	0.165	1.365	0.891-2.068

Note: ABSI: A Body Shape Index; BMI: Body mass index; BRI: Body roundness index; UA: Uric acid.

Table 5. Multivariate logistic regression analysis of risk factors for hypertensive left ventricular hypertrophy

	β	SE	Wald	P	OR	95% CI
Lp(a)	0.001	0.001	2.904	0.089	1.002	1.001-1.002
Age	0.018	0.009	4.461	0.034	1.037	1.000-1.635
UA	0.003	0.002	1.458	0.231	1.003	0.998-1.002
ABSI	-0.008	0.003	4.421	0.034	0.901	0.882-0.998
Blood pressure grading	0.382	0.124	12.034	0.002	1.473	1.175-1.845

Note: Lp(a): Lipoprotein(a); UA: Uric acid; ABSI: A Body Shape Index.

Discussion

Our study indicates that ABSI and BRI were independent risk factors for hypertensive kidney damage. Additionally, age, ABSI, and blood pressure grading were independent risk factors for hypertensive left ventricular hypertrophy. Furthermore, ABSI and smoking history were independent risk factors for hypertensive carotid artery plaque formation.

In the 48 cases of hypertensive kidney damage included in our study, we found that ABSI, BMI, and BRI were independent risk factors, which is consistent with findings from Liu et al. [28]. Research has shown that fat accumulation, particularly visceral fat, is an independent risk factor for hypertension-related kidney damage [29]. BMI is widely used for assessing obesity, with excess body fat contributing to chronic low-grade inflammation, which can damage kidney blood vessels and increase hypertension and kidney damage [30]. On the other hand, obesity can also increase activity of the sympathetic nervous system, which exacerbates

Table 6. Multivariate logistic regression analysis of risk factors for hypertensive carotid artery plaque formation

	β	SE	Wald	P	OR	95% CI
ABSI	0.001	0.001	4.576	0.032	1.012	1.001-1.623
Gender	0.078	0.198	0.154	0.678	1.109	0.782-1.673
UA	0.347	0.231	2.465	0.128	1.431	0.901-2.197
HDL	-0.542	0.276	3.654	0.056	0.587	0.337-1.076
Smoking history	-0.008	0.003	5.473	0.021	0.892	0.781-0.998
eGFR	0.021	0.013	3.452	0.067	1.024	0.998-1.048
Hypertension course	0.175	0.109	2.568	0.108	1.187	0.976-1.487
Blood pressure grading	1.732	0.109	2.568	0.198	1.187	0.965-1.489
Family History	0.367	0.234	2.389	0.124	1.476	0.981-2.257

Note: ABSI: A Body Shape Index; UA: Uric acid; HDL: High-density lipoprotein; eGFR: Estimated glomerular filtration rate.

hypertension by elevating heart rate and constricting blood vessels. Interestingly, we also found that obesity related index (ABSI and BRI) were independent risk factors for hypertensive kidney damage. ABSI, a body shape index proposed by Krakauer et al. in 2012, is based on waist circumference, height, and weight, aiming to complement BMI by offering better predictive ability for certain diseases, particularly in optimizing body shape assessment [31]. A cross-sectional study in 2016 found a strong linear relationship between ABSI and directly measured visceral fat. Compared to BMI, ABSI also has unique advantages in predicting overall mortality and diseases such as diabetes [32]. An analysis by Ji et al. [33] found that an increase in ABSI increases the risk of hypertension and cardiovascular diseases. ABSI better reflects abdominal obesity, which is closely related to metabolic syndrome and is more likely to cause cardiovascular damage. Therefore, when evaluating hypertensive patients for kidney damage, especially in obese populations, ABSI should be prioritized as a predictive tool for identifying kidney injury.

We also found that age, ABSI and blood pressure grading were independent factors predicting hypertensive left ventricular hypertrophy. As individuals age, the heart faces increased demands to pump blood throughout the body, leading to an increase in blood pressure. This chronic elevation in blood pressure can cause the walls of the left ventricle to thicken, a condition known as left ventricular hypertrophy. The thickening of the heart muscle is a compensatory mechanism to handle the increased workload; however, over time, it can impair heart function and an increase the risk of cardiovascular events [34]. Therefore, age plays a significant role in the development of left ventricular hypertrophy in hypertensive individuals. Furthermore, hypertension forces the heart to work harder, contributing to the thickening of the left ventricle known as left ventricular hypertrophy. The blood pressure, ranging from normal, prehypertension, stage 1 hypertension, to stage 2 hypertension, plays a significant role in determining the risk of developing left ventricular hypertrophy [35]. Individuals with higher blood pressure levels are more likely to develop left ventricular hypertrophy, which can further increase the risk of cardiovascular events, such as heart attacks and strokes. Interestingly, ABSI was also identified as an independent factor for hypertensive left ventricular hypertrophy. ABSI is an index assessing body fat. Excess body fat, particularly abdominal fat, increases blood pressure and places additional strain on the heart [36]. This heightened workload leads to the thickening of the left ventricle as the heart works harder to pump blood more efficiently. Over time, this can result in hypertensive left ventricular hypertrophy, further complicating cardiovascular health in hypertensive individuals.

For hypertensive carotid artery plaque formation, ABSI and smoking history were independent risk factors. In individuals with hypertension, the sustained high blood pressure puts additional stress on the arterial walls, making them more susceptible to damage. The combination of smoking and hypertension accelerates the progression of atherosclerosis in the carotid arteries, which supply blood to the brain. As plaque builds up in these arteries, it can eventually lead to blockages or the forma-

tion of blood clots, significantly raising the risk of stroke and other cardiovascular events [37]. Furthermore, smoking exacerbates hypertension by causing vasoconstriction and increasing heart rate, further elevating blood pressure levels. This synergistic effect of smoking and hypertension not only accelerates the development of atherosclerosis but also increases the overall cardiovascular risk for individuals with these risk factors [38]. Therefore, individuals with a history of smoking and hypertension should be particularly proactive in managing their blood pressure and making lifestyle changes to reduce their risk of developing carotid atherosclerosis. This may include smoking cessation, regular exercise, a healthy diet, and adherence to prescribed medication to control blood pressure and prevent further arterial damage. Also, we found that ABSI is an independent factor for hypertensive carotid artery plaque formation. Research has shown that individuals with a higher ABSI are more likely to develop hypertension and carotid atherosclerosis [39, 40]. This is because excess abdominal fat can lead to insulin resistance, inflammation, and dyslipidemia, all of which are factors that contribute to the development of hypertension and atherosclerosis [41]. Therefore, individuals with a high ABSI are at an increased risk of developing both conditions, especially when combined with other risk factors such as smoking, poor diet, and lack of physical activity.

This study has certain limitations. First, this is a retrospective study, so it cannot establish a clear causal relationship between ABSI and target organ damage in hypertension. Second, the sample size included in this study is relatively small, which may limit the generalizability of the findings. Lastly, the hypertensive population included in this study did not account for the potential influence of medication, which could have confounded the results. Therefore, further clinical and basic research, especially large-scale prospective studies, are needed to validate these findings.

In conclusion, this study demonstrates that ABSI is significantly associated with hypertensive kidney damage, left ventricular hypertrophy, and carotid atherosclerosis in hypertensive individuals. ABSI appears to be an independent risk factor for target organ damage in hypertension, suggesting its potential value in identifying individuals at higher risk for complications related to hypertension.

Disclosure of conflict of interest

None.

Address correspondence to: Ping Ding, Department of Critical Care Medicine, No. 988 Hospital of The PLA Joint Logistic Support Force (PLAJLSF), No. 602 Zhengshang Road, Zhongyuan District, Zhengzhou 450000, Henan, China. Tel: +86-15038255066; E-mail: dingping153@163.com

References

[1] Su M, Zhang Q, Bai X, Wu C, Li Y, Mossialos E, Mensah GA, Masoudi FA, Lu J, Li X, Salas-Vega S, Zhang A, Lu Y, Nasir K, Krumholz HM and Jiang L. Availability, cost, and prescription patterns of antihypertensive medications in primary health care in China: a nationwide cross-sectional survey. Lancet 2017; 390: 2559-2568.

[2] Sempere A, Assoumou L, González-Cordón A, Waters L, Rusconi S, Domingo P, Gompels M, de Wit S, Raffi F, Stephan C, Masiá M, Rockstroh J, Katlama C, Behrens GMN, Moyle G, Johnson M, Fox J, Stellbrink HJ, Guaraldi G, Florence E, Esser S, Gatell J, Pozniak A and Martínez E; NEAT 022 Study Group. Incidence of hypertension and blood pressure changes in persons with human immunodeficiency virus at high risk for cardiovascular disease switching from boosted protease inhibitors to dolutegravir: a post-hoc analysis of the 96-week randomised NEAT-022 trial. Clin Infect Dis 2023; 77: 991-1009.

[3] Guzik TJ, Nosalski R, Maffia P and Drummond GR. Immune and inflammatory mechanisms in hypertension. Nat Rev Cardiol 2024; 21: 396-416.

[4] Sun Y, Mu J, Wang DW, Ouyang N, Xing L, Guo X, Zhao C, Ren G, Ye N, Zhou Y, Wang J, Li Z, Sun G, Yang R, Chen CS and He J. A village doctor-led multifaceted intervention for blood pressure control in rural China: an open, cluster randomised trial. Lancet 2022; 399: 1964-1975.

[5] Carey RM, Moran AE and Whelton PK. Treatment of hypertension: a review. JAMA 2022; 328: 1849-1861.

[6] Tomiyama H. Vascular function: a key player in hypertension. Hypertens Res 2023; 46: 2145-2158.

[7] Bartoloni E, Alunno A and Gerli R. Hypertension as a cardiovascular risk factor in autoimmune rheumatic diseases. Nat Rev Cardiol 2018; 15: 33-44.

[8] Márquez DF, Rodríguez-Sánchez E, de la Morena JS, Ruilope LM and Ruiz-Hurtado G. Hypertension mediated kidney and cardiovascular damage and risk stratification: redefining concepts. Nefrologia (Engl Ed) 2022; 42: 519-530.
[9] Alrubaian F and Mulla Z. Governments policy measures to address obesity among adults: a scoping review of the global evidence. Lancet 2023; 402 Suppl 1: S20.
[10] Bohula EA, Wiviott SD, McGuire DK, Inzucchi SE, Kuder J, Im K, Fanola CL, Qamar A, Brown C, Budaj A, Garcia-Castillo A, Gupta M, Leiter LA, Weissman NJ, White HD, Patel T, Francis B, Miao W, Perdomo C, Dhadda S, Bonaca MP, Ruff CT, Keech AC, Smith SR, Sabatine MS and Scirica BM; CAMELLIA-TIMI 61 Steering Committee and Investigators. Cardiovascular safety of lorcaserin in overweight or obese patients. N Engl J Med 2018; 379: 1107-1117.
[11] Arnotti K, Bamber MD and Brewer V. Dietary interventions and blood pressure in overweight or obese individuals: a systematic review and meta-analysis. Clin Nutr 2022; 41: 1001-1012.
[12] Aggarwal R, Yeh RW, Joynt Maddox KE and Wadhera RK. Cardiovascular risk factor prevalence, treatment, and control in US adults aged 20 to 44 years, 2009 to March 2020. JAMA 2023; 329: 899-909.
[13] Gui J, Li Y, Liu H, Guo LL, Li J, Lei Y, Li X, Sun L, Yang L, Yuan T, Wang C, Zhang D, Li J, Liu M, Hua Y and Zhang L. Obesity- and lipid-related indices as a risk factor of hypertension in mid-aged and elderly Chinese: a cross-sectional study. BMC Geriatr 2024; 24: 77.
[14] Franek E, Pais P, Basile J, Nicolay C, Raha S, Hickey A, Ahmad NN, Konig M, Kan H and Gerstein HC. General versus central adiposity as risk factors for cardiovascular-related outcomes in a high-risk population with type 2 diabetes: a post hoc analysis of the REWIND trial. Cardiovasc Diabetol 2023; 22: 52.
[15] Sato Y, Fujimoto S, Konta T, Iseki K, Moriyama T, Yamagata K, Tsuruya K, Narita I, Kondo M, Kasahara M, Shibagaki Y, Asahi K and Watanabe T. Body shape index: sex-specific differences in predictive power for all-cause mortality in the Japanese population. PLoS One 2017; 12: e0177779.
[16] Asfar P, Meziani F, Hamel JF, Grelon F, Megarbane B, Anguel N, Mira JP, Dequin PF, Gergaud S, Weiss N, Legay F, Le Tulzo Y, Conrad M, Robert R, Gonzalez F, Guitton C, Tamion F, Tonnelier JM, Guezennec P, Van Der Linden T, Vieillard-Baron A, Mariotte E, Pradel G, Lesieur O, Ricard JD, Hervé F, du Cheyron D, Guerin C, Mercat A, Teboul JL and Radermacher P; SEPSISPAM Investigators. High versus low blood-pressure target in patients with septic shock. N Engl J Med 2014; 370: 1583-1593.
[17] Madan N, Aly D, Kathol M, Buddhavarapu A, Rieth T, Sherman A and Forsha D. Relationship between obesity and global longitudinal strain in the pediatric single ventricle Fontan population across ventricular morphologies. J Am Heart Assoc 2024; 13: e028616.
[18] Ding J, Davis-Plourde KL, Sedaghat S, Tully PJ, Wang W, Phillips C, Pase MP, Himali JJ, Gwen Windham B, Griswold M, Gottesman R, Mosley TH, White L, Guðnason V, Debette S, Beiser AS, Seshadri S, Ikram MA, Meirelles O, Tzourio C and Launer LJ. Antihypertensive medications and risk for incident dementia and Alzheimer's disease: a meta-analysis of individual participant data from prospective cohort studies. Lancet Neurol 2020; 19: 61-70.
[19] Zhang WZ. Intensified, optimized and standardized management of Chinese patients with hypertension: comments on "2018 Chinese Guidelines for Prevention and Treatment of Hypertension". J Geriatr Cardiol 2019; 16: 178-181.
[20] Petramala L, Gigante A, Sarlo F, Servello A, Circosta F, Marino L, Ciccarelli A, Cavallaro G and Letizia C. Relevance of obesity-related organ damage and metabolic syndrome classification in cardiovascular and renal risk stratification in patients with essential hypertension. Front Cardiovasc Med 2024; 11: 1369090.
[21] Zhang Y, Zhao Y, Wei C, Li Y, Aslam H, Feng Q, Huang Q, Zheng Y, Lv F, Hao W and Li J. Association of common medical comorbidities with early renal damage in the Chinese tropics with essential hypertension. BMC Nephrol 2021; 22: 366.
[22] Lin M, Li N, Heizhati M, Gan L, Zhu Q, Yao L, Li M and Yang W. Chinese visceral adiposity index is associated with incident renal damage in patients with hypertension and abnormal glucose metabolism: a longitudinal study. Front Endocrinol (Lausanne) 2022; 13: 910329.
[23] Lee HH, Lee H, Cho SMJ, Kim DW, Park S and Kim HC. On-treatment blood pressure and cardiovascular outcomes in adults with hypertension and left ventricular hypertrophy. J Am Coll Cardiol 2021; 78: 1485-1495.
[24] Chu HW, Hwang IC, Kim HM, Park J, Choi H, Choi HM, Yoon YE and Cho GY. Age-dependent implications of left ventricular hypertrophy regression in patients with hypertension. Hypertens Res 2024; 47: 1144-1156.
[25] Chuang SM, Liu SC, Leung CH, Lee YT and Chien KL. High left ventricular mass associated with increased risk of incident diabetes. Sci Rep 2024; 14: 250.
[26] Chen A, Wu W, Gong J, Han Y, Xu G and Xie L. Association of homocysteine with carotid atherosclerosis in hypertension. J Hum Hypertens 2023; 37: 227-234.

[27] Peng M, Wu Y and Jin Y. Correlations of GGT, Hcy and ABI with carotid atherosclerosis in essential hypertension patients. Cell Mol Biol (Noisy-le-grand) 2023; 69: 79-83.
[28] Liu C, Tian J, Jose MD, Dwyer T and Venn AJ. BMI trajectories from childhood to midlife are associated with subclinical kidney damage in midlife. Obesity 2021; 29: 1058-1066.
[29] Lembo M, Pacella D, Manzi MV, Morisco C, La Mura L, Mancusi C, Bardi L, Trimarco V, Trimarco B, Izzo R and Esposito G. Hypertension-mediated organ damage involving multiple sites is an independent risk factor for cardiovascular events. Eur Heart J Open 2023; 3: oead102.
[30] Jha R, Lopez-Trevino S, Kankanamalage HR and Jha JC. Diabetes and renal complications: an overview on pathophysiology, biomarkers and therapeutic interventions. Biomedicines 2024; 12: 1098.
[31] Krakauer NY and Krakauer JC. A new body shape index predicts mortality hazard independently of body mass index. PLoS One 2012; 7: e39504.
[32] Bosello O, Donataccio MP and Cuzzolaro M. Obesity or obesities? Controversies on the association between body mass index and premature mortality. Eat Weight Disord 2016; 21: 165-174.
[33] Ji M, Zhang S and An R. Effectiveness of a body shape index (ABSI) in predicting chronic diseases and mortality: a systematic review and meta-analysis. Obes Rev 2018; 19: 737-759.
[34] Butler J, Jones WS, Udell JA, Anker SD, Petrie MC, Harrington J, Mattheus M, Zwiener I, Amir O, Bahit MC, Bauersachs J, Bayes-Genis A, Chen Y, Chopra VK, Figtree G, Ge J, Goodman SG, Gotcheva N, Goto S, Gasior T, Jamal W, Januzzi JL, Jeong MH, Lopatin Y, Lopes RD, Merkely B, Parikh PB, Parkhomenko A, Ponikowski P, Rossello X, Schou M, Simic D, Steg PG, Szachniewicz J, van der Meer P, Vinereanu D, Zieroth S, Brueckmann M, Sumin M, Bhatt DL and Hernandez AF. Empagliflozin after acute myocardial infarction. N Engl J Med 2024; 390: 1455-1466.
[35] Zhu L, Chen S, Liu N, Cui Q, Ma M, Liu Z, Xing Y, Zhang Y and Wang J. Elevated plasma macrophage migration inhibitor factor is associated with hypertension and hypertensive left ventricular hypertrophy. J Hum Hypertens 2023; 37: 68-73.
[36] Chen D, Feng J, He H, Xiao W and Liu X. Classification, diagnosis, and treatment of obesity-related heart diseases. Metab Syndr Relat Disord 2024; 22: 161-169.
[37] Zou Y, Zhang X, Bu X, Zhang Z, Wu Y, Ren Y and Mu L. Analysis of carotid plaque risk factors in a population at high risk of stroke in Yubei District, Chongqing, China. J Stroke Cerebrovasc Dis 2023; 32: 107224.
[38] Borkowski P, Borkowska N, Mangeshkar S, Adal BH and Singh N. Racial and socioeconomic determinants of cardiovascular health: a comprehensive review. Cureus 2024; 16: e59497.
[39] Geraci G, Zammuto M, Gaetani R, Mattina A, D'Ignoto F, Geraci C, Noto D, Averna M, Cottone S and Mulè G. Relationship of a body shape index and body roundness index with carotid atherosclerosis in arterial hypertension. Nutr Metab Cardiovasc Dis 2019; 29: 822-829.
[40] Song P, Fang Z, Wang H, Cai Y, Rahimi K, Zhu Y, Fowkes FGR, Fowkes FJI and Rudan I. Global and regional prevalence, burden, and risk factors for carotid atherosclerosis: a systematic review, meta-analysis, and modelling study. Lancet Glob Health 2020; 8: e721-e729.
[41] Neeland IJ, Ross R, Després JP, Matsuzawa Y, Yamashita S, Shai I, Seidell J, Magni P, Santos RD, Arsenault B, Cuevas A, Hu FB, Griffin B, Zambon A, Barter P, Fruchart JC and Eckel RH; International Atherosclerosis Society; International Chair on Cardiometabolic Risk Working Group on Visceral Obesity. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. Lancet Diabetes Endocrinol 2019; 7: 715-725.