ORIGINAL RESEARCH

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Relationship between blood lipid and arterial stiffness in hypertension

Abstract

Purpose: To explore the relationship between blood lipids and pulse wave velocity (PWV).

Methods: We retrospectively selected subjects who had undergone treatment in an outpatient or inpatient clinic. The subjects were sub-grouped into four groups: Group A (no history of hypertension and dyslipidemia), Group B (a history of dyslipidemia but not hypertension), Group C (a history of hypertension but not dyslipidemia) and Group D (a history of both hypertension and dyslipidemia). We determined brachial-ankle pulse wave velocity (baPWV), atherogenic index of plasma (AIP), non-high-density lipoprotein (non-HDL-C), total cholesterol/high density lipoprotein (TC/HDL-C) and atherogenic indices (AI) for hypertension and non-hypertension and dyslipidemia and non-dyslipidemia patients.

Results: A total of 380 subjects were included. Uric acid, systolic pressure, diastolic pressure, pulse pressure and mean arterial pressure were significantly different among the groups. The prevalence of coronary arterial disease and diabetes mellitus, age, body mass index, heart rate, blood glucose, creatinine, urea, smoking history and alcohol consumption did not differ among groups. The baPWV was significantly different between both hypertension/non-hypertension and dyslipidemia/non-dyslipidemia patients. In hypertension patients (in Group C and D), partial correlation analysis showed that Δ baPWV correlated with TC, HDL-C, AI, AIP and TC/HDL-C but not LDL-C, triglyceride (TG) or non-HDL-C.

Conclusion: Hypertension and/or dyslipidemia may be risk factors for arterial stiffness. In hypertension patients, TC, HDL-C, AIP, AI and TC/HDL-C might correlate to arterial stiffness and pathological increases in these levels may indicate risk factors for arterial stiffness.

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Arterial stiffness, which is defined as a ratio of the change in pulse pressure to the change in diastolic pressure [1], is considered to be an independent predictor of cardiovascular morbidity and mortality, and a valid surrogate endpoint of cardiovascular disease [2,3]. In patients with hypertension, arterial stiffness is also regarded as a sign of early damage of target organs [4-6]. In pathology, it can be explained as the hyperplasia of fibrous tissue and increased adhesion of consecutive fibrous layer in the vascular wall [7] and regarded as the changes in the initial stages of arterial sclerosis [4,8].

Pulse wave velocity (PWV) is considered to be the “gold standard” method for the measurement of arterial stiffness. Brachial-ankle PWV (baPWV), which correlates well with invasively-measured aortic PWV, has been accepted as a conventional, reproducible and noninvasive screening tool for identifying subjects at risk of cardiovascular diseases [6].

Dyslipidemia has been considered a possible risk factor for arterial stiffness [9-14]. In the 1980s, several animal experiments proposed that a high fat diet might cause an abnormal rise in PWV in simians [9-10]. Although some evidence for positive correlation had been reported in healthy adults [11-13] and HIV-infected patients [14], the relationship between arterial stiffness and dyslipidemia, especially in hypertensions, had been debated for decades. In 2009, Cecelja et al. [15] systematically reviewed the cross-section studies of carotid-femoral PWV (cfPWV) in multivariable regression models. Total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) and triglycerides (TG) were not found to be associated with cfPWV, especially in patients with hypertension. In 2019, Choudhary et al. [16] reported a cross-section study of 615 never-treated hypertensive subjects. They found that TC, TG, LDL-C and HDL-C were not associated with PWV. Another cross-section study from Zhan et al. [17] of 14,071 hypertensive subjects showed a relationship among lipid profiles of TC, TG, HDL-C, but not LDL-C, and baPWV. A previous cross-section study from our team [18] also showed the absence of a correlation between blood lipid and PWV. These results failed to explain either the absence of correlation between PWV and blood lipid profiles or the relationship and interaction mechanism between arterial stiffness and dyslipidemia in hypertension.

In recent years, several complex profiles of blood lipids have been proposed for further evaluation of lipid metabolism. The atherogenic index (AI), which is calculated as $AI = (TC - HDL-C) / HDL-C$, was used first to estimate the status of blood lipids [20]. Subsequently, the use of AI to assess plasma (AIP; calculated as $\text{Log}_{10}(TG / HDL-C)$) [22], non-high-density lipoprotein, (non-HDL-C; calculated as $TC - HDL-C$) [19,23] and $TC / HDL-C$ was proposed. The aim

of the current study was (1) to explore the association among blood lipid and arterial stiffness in hypertension patients and (2) to estimate the diagnostic and predictive value of TC, TG, LDL-C, HDL-C, non-HDL-C, AIP, AI and $TC / HDL-C$ indices.

Methods

Subjects

We retrospectively selected 380 subjects (282 males and 98 females, with an average age of 61.82 ± 12.76 years) who had treated in the past in an outpatient clinic or inpatient clinic of the Department of Cardiology, Beijing Hospital from March 2012 to December 2018. Only subjects who had undergone routine chemical analysis, such as TC, TG, LDL-C, HDL-C, blood sugar, creatine, uric acid and baPWV, were included. The diagnostic criteria of essential hypertension were as follows: a mean systolic brachial blood pressure of at least 140 mmHg and/or a diastolic brachial blood pressure of at least 90 mmHg in two continuous measurements in the patient's previous or present history. Exclusion criteria were as follows: secondary causes of hypertension, severe heart failure (IV level for New York Heart Association heart function or a left ventricular ejection fraction of $<40\%$), severe infectious diseases, hyperthyreosis or hypothyroidism, atrial fibrillation, diseases of the immune system, stroke (including transient ischemic attack), renal insufficiency, ongoing treatment with vasoactive drugs, type 1 diabetes mellitus, diseases of the hematological system and hepatic insufficiency. In addition, subjects with either hypertension or dyslipidemia were included only if they had been received standard treatment for over one year.

Subjects were sub-grouped into four groups: Group A (no history of hypertension and dyslipidemia); Group B (a history of dyslipidemia but not hypertension); Group C (a history of hypertension but not dyslipidemia); and Group D (a history of both hypertension and dyslipidemia).

The data regarding age, gender, occupation, body mass index (BMI), heart rate, systolic and diastolic blood pressure for brachial arterial, pulse pressure index (calculated as pulse pressure/systolic pressure), history of coronary artery disease (CAD), type 2 diabetes mellitus, smoking history and alcohol consumption were collected from all subjects.

Brachial-ankle PWV measurements

The BaPWV was measured using a volume plethymographic apparatus (Colin Company, Japan). The methodology used was has been described previously [24,25].

Definition of arterial stiffness

The baPWV was employed to evaluate arterial stiffness. To calculate the theoretical reference value in patients, Hatanaka et al. [25] recommended the following formula: $\text{baPWV} = 0.21 \times \text{age}^2 (\text{years}^2) - 13.73 \times \text{age} (\text{year}) + 0.05 \times \text{MAP}^2 (\text{mmHg}^2) + 3.95 \times \text{HR} (\text{bpm}) + 36.49 \times \text{gender} (1 \text{ male}; 0 \text{ female}) + 733$. In our study, subjects with a higher practical value than the theoretical reference value were considered to be diagnosed with arterial stiffness. For every subject, we also calculated ΔbaPWV as the difference between the practical value and the theoretical reference value.

Laboratory measurement

On the morning after hospital admission, blood samples were taken prior to breakfast. Blood samples were analyzed for the following parameters: creatinine, urea, blood sugar, uric acid, TC, TG, LDL-C and HDL-C. Complex lipid indexes were calculated according to the TC, TG, LDL-C and HDL-C parameters.

Statistical analysis

Statistical analyses were performed using Microsoft Excel XP and SPSS version 19.0 for Windows (SPSS, Chicago, IL, USA). Data are expressed as the mean $\pm$ SD. After testing for normality, the differences for each variable between any two study groups (groups with or without arterial stiffness) were evaluated using Welch's t-test for continuous variables and the I2 test for categorical variables. Analysis of variance was used to evaluate the differences in variables among multiple groups. If statistical differences were observed among the groups, a subsequent pairwise comparison was performed. At the same time, we used 2 $\times$ 2 factorial analysis to test whether hypertension or dyslipidemia (or both) were factors for baPWV. Partial correlation analysis was adopted to assess the correlation between the blood lipid indexes and ΔbaPWV .

Results

Characteristics and baseline values of study subjects

Of the 380 subjects retrospectively selected, 20 subjects were sub-grouped into Group A, 55 into Group B, 101 into Group C, and 204 into Group D. Of the patients included, 253 of the patients had the complication of CAD, and 111 subjects suffered from type 2 diabetes mellitus.

The systolic pressure, diastolic pressure, and mean arterial pressure were different among the four groups ($p < 0.05$). Age, BMI, heart rate, creatine, blood sugar, and

urine, as well as the prevalence of both type 2 diabetes mellitus and CAD, and the proportion of sex, smoking history and alcohol consumption were not statistically different among groups (Table 1).

BaPWV and blood lipids among the groups

The baPWV, TG, TC, LDL-C, AI, AIP, non-HDL-C and TC/HDL-C were significantly different among the groups ($p < 0.05$); however, no differences in HDL-C were observed among the groups (Table 2).

We performed a further comparison among nondiabetics in all groups: baPWV, TG, TC, AI, AIP, non-HDL-C and TC/HDL-C were significantly different among the groups ($p < 0.05$), but not LDL-C and HDL-C (Table 3).

We also performed another comparison for patients diagnosed with CAD in all four groups: TC, AI, AIP, non-HDL-C and TC/HDL-C were significantly different among groups ($p < 0.05$), but not baPWV, TG, LDL-C and HDL-C (Table 4).

The 2 $\times$ 2 factor analysis showed that the baPWV of hypertension patients ($n=305$) was higher when compared with that of non-hypertension patients ($n=75$, $p < 0.05$). Similarly, the baPWV of dyslipidemia patients ($n=259$) was higher when compared with that of non-dyslipidemia patients ($n=121$, $p < 0.05$). In the mean contour map, the lines of hypertension and dyslipidemia did not intersect; demonstrating that hypertension and dyslipidemia were independent factors for baPWV (Figure 1).

Partial correlation analyses of blood lipid indexes and baPWV

Partial correlation analyses were performed to evaluate the correlation between the blood lipid indexes and ΔbaPWV in patients with hypertension: TC, HDL-C, AI, AIP and TC/HDL-C correlated with ΔbaPWV ($p < 0.05$). In contrast, TG, LDL-C and non-HDL-C did not show correlation with ΔbaPWV (Table 5). The correlation coefficients were -0.125, 0.167, -0.163, -0.144 and -0.144 for TC, HDL-C, AI, AIP and TC/HDL-C, respectively.

Further partial correlation analyses among nondiabetics and non-CADs were performed: HDL-C, AI, AIP and TC/HDL-C correlated with ΔbaPWV in both nondiabetics and non-CADs. The correlation coefficients were 0.171, -0.169, -0.159 and -0.169 for HDL-C, AI, AIP and TC/HDL-C, respectively, in nondiabetics and 0.238, -0.248, -0.206 and -0.248 for HDL-C, AI, AIP and TC/HDL-C, respectively, in non-CADs (Table 6, Table 7).

TABLE 1. Baseline of included subjects

Clinical parameter measured	Group A n=20	Group B n=55	Group C n=101	Group D n=204	Total n=380
Sex (M/F)	16/4	45/10	65/36	156/48	282/98
Age (year)	58.55±10.95	58.60±11.41	63.90±13.06	62.00±12.95	61.82±12.76
BMI (kg/m ²)	23.73±2.52	25.17±3.05	26.57±11.16	26.44±3.71	26.15±6.54
CAD (Yes/No)	11/9	39/16	62/39	141/63	253/127
Type 2 diabetes (Yes/No)	1/19	12/43*	32/69	68/138*	111/269
Systolic blood pressure (mmHg)	111.62±12.42	115.16±19.11	127.82±16.65†	129.33±12.93†	125.96±17.95
Mean arterial pressure (mmHg)	83.15±8.70	88.33±14.97	95.14±13.31†	97.93±13.45†	95.03±14.08
Diastolic blood pressure (mmHg)	66.35±6.52	68.58±12.33	72.25±10.18†	73.44±9.87†	72.02±10.40
Heart rate (bpm)	63.7/±8.08	65.04±9.36	67.72±10.97	65.53±8.71	65.98±9.44
Urea (μmol/L)	68.55±12.54	75.47±12.42	75.26±24.45	79.16±26.23	77.03±23.74
Uric acid (μmol/L)	311.65±88.17	353.04±93.69	342.70±109.32	367.10±95.78	355.55±99.60
Fasting blood glucose (mmol/L)	5.07±0.75	5.31±1.04	5.64±1.53	5.69±1.57	5.58±1.46
Urea (μmol/L)	4.78±1.62	4.84±1.30	5.51±2.50	5.46±2.18	5.35±2.15

Abbreviations: BMI, body mass index; CAD, coronary arterial disease; F, female; Group A, no history of hypertension and dyslipidemia; Group B, history of dyslipidemia but not hypertension; Group C, history of hypertension but not dyslipidemia; Group D, history of both hypertension and dyslipidemia; M, male.

* p<0.05, compared with Group A; † p<0.05, compared with Group B; ‡ p<0.05, compared with Group C

TABLE 2. Comparison of blood lipid indexes and baPWV of whole subjects among groups

Clinical parameter measured	Group A n=20	Group B n=55	Group C n=101	Group D n=204	Total n=380
TC (mmol/L)	3.85±1.04	4.35±0.88	4.04±0.96	4.35±1.16‡	4.24±1.08
TG (mmol/L)	1.08±0.39	1.59±1.24*	1.40±0.85	1.71±0.97‡	1.58±0.98
HDL-C (mmol/L)	1.17±0.26	1.03±0.29	1.05±0.26	1.03±0.26	1.04±0.27
LDL-C (mmol/L)	2.27±0.67	2.64±0.93	4.32±19.48	2.64±0.92*	3.07±10.09
AIP	-0.051±0.18	0.12±0.29*	0.082±0.27	0.18±0.27‡	0.14±0.28
AI	2.28±0.63	3.41±1.16*	3.02±1.14*	3.41±1.32‡	3.25±1.25
TC/HDL-C (mmol/L)	3.28±0.63	4.42±1.16*	4.02±1.14*	4.41±1.32‡	4.25±1.25
Non-HDL-C (mmol/L)	2.41±1.15	3.31±0.90*	2.99±0.88*	3.33±1.10‡	3.19±1.04
ba-PWV (cm/s)	1,283.10±358.87	1,404.51±223.97	1,592.01±310.48†	1,569.78±300.07†	15,37.14±307.73

Abbreviations: AI, atherogenic index; AIP, atherogenic index of plasma; baPWV, brachial-ankle pulse wave velocity; Group A, no history of hypertension and dyslipidemia; Group B, history of dyslipidemia but not hypertension; Group C, history of hypertension but not dyslipidemia; Group D, history of both hypertension and dyslipidemia; LDL-C, low-density lipoprotein cholesterol; non-HDL-C, non-high-density lipoprotein; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol

* p<0.05, compared with Group A; † p<0.05, compared with Group B; ‡ p<0.05, compared with Group C

FIGURE 1. Mean contour map

Discussion

Arterial stiffness has been widely accepted as damage of hypertension [4,8]. It has been reported that dyslipidemia may evoke or aggravate arterial stiffness in patients with previous hypertension [26]. The relationship between arterial stiffness and blood lipid has come to be a hot topic in the last few years. Nearly all previously studies reported either a weak correlation or the absence of a correlation between arterial stiffness and blood lipids, as estimated by classical indexes of TC, TG, LDL-C and HDL-C in hypertensions [15-18]. Moreover, these results were not well explained.

In views of previous studies, we suggest that the minimal or inconsistent correlation between arterial stiffness and blood lipid in hypertensions can be attributed to a complex relationship among three factors. First, hypertension may cause arterial stiffness due to elongation of the arterial wall in a hypertensive status. Second, dyslipidemia may act on the blood vessel endothelium and further affect the production, release and function of nitric oxide, which may be contribute to arterial stiffness [26]. Third, dyslipidemia is one of the causative factors of hypertension. Dyslipidemia may lead to hypertrophy of smooth muscle cells and the deposition of collagen, and these pathogenic changes on the arterial wall may further aggravate hypertension. In contrast, dyslipidemia may lead to injured renal capillaries, resulting in a secondary cause of hypertension [26,27]. Fourth, hypertension and arterial stiffness affect blood lipids. Patients with hypertension and/

or arterial stiffness have shown a higher excitability of sympathetic nerve, which has been proven to be effective for blood lipid metabolism [28-31]. These mechanisms result in a relationship between blood lipids and arterial stiffness that is more complex in patients with hypertension. Based on the theory that hypertension and dyslipidemia simultaneously lead to arterial stiffness, we speculated that the effect of hypertension on PWV was significantly stronger than that of dyslipidemia. This might be the reason that blood lipid was not correlated with PWV in hypertension; however, this hypothesis should be confirmed by further research.

In a study by Kanda et al. [32], the relationship between blood lipids and arterial stiffness was explored using classical indexes, as well as AI. The AI was associated with PWV but not with the classical indexes. These results were consistent with our findings.

Triglyceride, TC and LDL-C are viewed as risk factors of atherosclerosis related diseases, such as CAD [23], whereas HDL-C is found to be a protective factor of cardiovascular events [33-36]. The AIP, AI and TC/HDL-C were equal to the ratio of vascular impaired factors (i.e., TC-HDL-C, LDL-C and TC) [23] and vascular protective factors (i.e., HDL-C) [33-36]. As a result, we suggest that AIP, AI and TC/HDL-C might reflect the degree of risk of atherosclerosis-related diseases, according to abnormal proportion of blood lipids. This predictive effect had been confirmed previously in patients with atherosclerosis and CAD [33]. In our study, AI, AIP and TC/HDL-C correlated with Δ baPWV, which was consistent with another study by Kanda et al. [32]: AI, but not TG, LDL-C and HDL-C indexes, was associated with PWV. Accordingly, we hypothesize that the occurrence and development of AS, especially in early stage, may be ascribed to abnormal distribution of blood lipids.

Although the data are promising, the present study has several limitations. First, it is a cross-sectional study and the treatment, especially the anti-hypertension and anti-hyperlipidemia medication, was not referenced. Second, accepted reference values were unavailable for AIP, AI, non-HDL-C and TC/HDL-C. Therefore, subjects were not grouped based on complex indexes. We reported the correlation between lipid profiles and arterial stiffness, but did not evaluate predictive and diagnostic efficacy. Third, the sample size was not large enough especially for control group.

TABLE 3. Comparison of blood lipid indexes and baPWV in patients without type 2 diabetes mellitus among groups

Clinical parameter measured	Group A n=19	Group B n=43	Group C n=69	Group D n=138	Total n=269
TC (mmol/L)	3.78±1.01	4.39±0.91*	4.07±0.97	4.44±1.17*‡	4.29±1.09
TG (mmol/L)	1.11±0.38	1.46±0.78*	1.41±0.93	1.67±0.85*‡	1.52±0.85
HDL-C (mmol/L)	1.15±0.25	1.05±0.30	1.05±0.30	1.03±0.27	1.05±0.27
LDL-C (mmol/L)	2.28±0.69	2.76±0.97	5.26±23.55	2.73±0.93*	3.36±11.99
AIP	-0.035±0.17	0.10±0.27*	0.071±0.28	0.17±0.26*‡	0.12±0.27
AI	2.28±0.65	3.38±1.12*	3.00±1.13*	3.45±1.27*‡	3.25±1.22
TC/HDL-C (mmol/L)	3.28±0.65	4.38±1.12*	4.00±1.13*	4.45±1.27*‡	4.25±1.22
Non-HDL L-C (mmol/L)	2.34±1.14	3.34±0.92*	3.01±0.89*	3.41±1.10*‡	3.22±1.06
ba-PWV (cm/s)	1,260.63±353.96	1,447.37±217.61*	1,559.69±308.97*	1,530.77±300.04*	15,05.57±303.20

Abbreviations: AI, atherogenic index; AIP, atherogenic index of plasma; baPWV, brachial-ankle pulse wave velocity; Group A, no history of hypertension and dyslipidemia; Group B, history of dyslipidemia but not hypertension; Group C, history of hypertension but not dyslipidemia; Group D, history of both hypertension and dyslipidemia; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; non-HDL-C, non-high-density lipoprotein; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol

* p<0.05, compared with Group A; † p<0.05, compared with Group B; ‡ p<0.05, compared with Group C

TABLE 4. Comparison of blood lipid indexes and baPWV in patients of non-CADs among groups

Clinical parameter measured	Group A n=9	Group B n=16	Group C n=39	Group D n=63	Total n=127
TC (mmol/L)	3.78±1.01	4.46±0.89	4.38±0.94	4.63±1.16	4.49±1.05
TG (mmol/L)	1.12±0.40	1.49±0.79	1.51±0.89	2.02±1.26*‡	1.73±1.09
HDL-C (mmol/L)	1.20±0.28	1.10±0.33	1.12±0.28	1.00±0.26‡	1.07±0.28
LDL-C (mmol/L)	2.21±0.53	2.72±0.72	2.53±0.71	2.82±0.88*	2.67±0.88
AIP	-0.046±0.15	0.093±0.29*	0.082±0.30	0.25±0.28*‡	0.16±0.30
AI	2.43±0.63	3.27±1.21	3.08±1.11	3.79±1.39*†‡	3.41±1.30
TC/HDL-C (mmol/L)	3.43±0.63	4.27±1.21	4.08±1.11	4.79±1.39*‡	4.41±1.30
Non-HDL L-C (mmol/L)	2.56±1.23	3.36±0.89	3.26±0.85	3.63±1.11*	3.40±1.05
ba-PWV (cm/s)	1,377.67±206.51	1,426.13±277.02	1,560.10±332.42	1,486.63±294.60	1,494.94±302.02

Abbreviations: AI, atherogenic index; AIP, atherogenic index of plasma; baPWV, brachial-ankle pulse wave velocity; Group A, no history of hypertension and dyslipidemia; Group B, history of dyslipidemia but not hypertension; Group C, history of hypertension but not dyslipidemia; Group D, history of both hypertension and dyslipidemia; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; non-HDL-C, non-high-density lipoprotein; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol

* p<0.05, compared with Group A; † p<0.05, compared with Group B; ‡ p<0.05, compared with Group C

TABLE 5. Partial correlation analysis of baPWV and blood lipid indexes in hypertensions

Clinical parameter measured	Correlation coefficient	P value
TC	-0.125	0.030
TG	-0.009	0.879
HDL-C	0.167	0.004
LDL-C	-0.015	0.607
AI	-0.163	0.004
AIP	-0.144	0.012
Non-HDL-C	-0.052	0.367
TC/HDL-C	-0.144	0.012

Abbreviations: baPWV: brachial-ankle pulse wave velocity; TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; AIP: atherogenic index of plasma; AI: atherogenic index; Non-HDL-C: non-high-density lipoprotein.

Bold values indicate statistical significance; $p < 0.05$

TABLE 6. Partial correlation analysis of baPWV and blood lipid indexes in patients of nondiabetics

Clinical parameter measured	Correlation coefficient	P value
TC	0.007	0.915
TG	-0.100	0.105
HDL-C	0.171	0.003
LDL-C	-0.008	0.893
AI	-0.169	0.011
AIP	-0.159	0.011
Non-HDL-C	-0.042	0.501
TC/HDL-C	-0.169	0.001

Abbreviations: baPWV: brachial-ankle pulse wave velocity; TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; AIP: atherogenic index of plasma; AI: atherogenic index; Non-HDL-C: non-high-density lipoprotein.

Bold values indicate statistical significance; $p < 0.05$

TABLE 7. Partial correlation analysis of baPWV and blood lipid indexes in patients of non-CADs

Clinical parameter measured	Correlation coefficient	P value
TC	-0.049	0.585
TG	-0.152	0.090
HDL-C	0.238	0.007
LDL-C	-0.131	0.145
AI	-0.248	0.005
AIP	-0.206	0.020
Non-HDL-C	-0.119	0.186
TC/HDL-C	-0.248	0.005

Abbreviations: baPWV: brachial-ankle pulse wave velocity; TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; AIP: atherogenic index of plasma; AI: atherogenic index; Non-HDL-C: non-high-density lipoprotein.

Bold values indicate statistical significance; $p < 0.05$

Conclusion

We explored the relationship between blood lipids and arterial stiffness in patients with hypertension, using the following indexes: baPWV; TC; TG; LDL-C; and HDL-C, as well as the blood lipid indexes AI; AIP; TC/HDL-C; and non-HDL-C. Based on these results, we have demonstrated the following: (1) hypertension and/or dyslipidemia might represent risk factors for arterial stiffness; and (2) in patients with hypertension, TC, HDL-C, AIP, AI and TC/HDL-C might be correlated to arterial stiffness, and the pathological increase may be a risk factor for arterial stiffness. However, these conclusions should be confirmed in future studies.

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