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Descriptive study of possible relation between cardio-ankle vascular index and lipids in hypertension subjects

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Abstract

Aim: The cardio-ankle vascular index (CAVI) is a new evaluation indicator for arteriosclerosis. This study investigated the relationship between the CAVI and lipid levels in patients with hypertension in a real clinical environment.

Methods: This descriptive study enrolled 2,656 patients (male/female: 1,016/1,640) from the Outpatient Department of Vascular Medicine of Peking University Shougang Hospital and Jinding Street Community Health Service Center. CAVI was measured using a VaseraVS-1000 vascular screening system (Fukuda Denshi, Tokyo, Japan).

Results: Age, body mass index (BMI), waist circumference, hip circumference, CAVI, systolic blood pressure (SBP), diastolic blood pressure (DBP), creatinine, fasting plasma glucose (FPG), uric acid (UA), hypersensitive C-reactive protein (hs-CRP), homocysteine, HbA1c, and triglyceride (TG) were significantly higher in the hypertension group than in the non-hypertension group. The levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were significantly lower in the hypertension group than in the non-hypertension group. The CAVI value was significantly higher in patients with hypertriglyceridemia and normal LDL-C than in those with normal TG and hyper-LDL-C. Age, waist circumference, UA, FPG, HDL-C, hs-CRP, HbA1c, BMI, SBP, and DBP were independently associated with CAVI in all patients. Beta blockers were negatively correlated with CAVI (β = –0.411, P = 0.011). Sex (male) and history of hypertension and diabetes mellitus were positively correlated with CAVI (β = 0.419, P < 0.001; β = 0.247, P = 0.011; β = 0.638, P < 0.001; respectively).

Conclusions: The CAVI was significantly higher in patients with hypertension and exhibited differences based on sex. Although we did not find a significant correlation between CAVI and TG, it remains crucial to maintain blood pressure to prevent the development of arteriosclerosis.

Keywords

Cardio-ankle vascular index, hypertension, metabolic disorders, triglyceride

Introduction

Hypertension is a prevalent condition worldwide, with arteriosclerosis playing a key role in its pathogenesis. Arteriosclerosis is characterized by arterial stiffness—a predictor of future cardiovascular events [1]. The cardio-ankle vascular index (CAVI) is a new indicator for evaluating arterial stiffness [2]. CAVI has been the most sensitive surrogate marker for detecting subclinical atherosclerosis in Korean patients with diabetes mellitus and without atherosclerotic cardiovascular disease [3]. The CAVI could reflect the contractile function of arterial smooth muscle [4]. CAVI is reportedly positively correlated with homocysteine and tends to increase in patients with hypertension [5, 6].

Hyperlipidemia is one of the most common risk factors for vascular diseases and is involved in the development of arteriosclerosis. Dyslipidemia is characterized by hypertriglyceridemia, elevated levels of low-density lipoprotein cholesterol (LDL-C), and low levels of high-density lipoprotein cholesterol (HDL-C). The probability of blood lipid abnormalities is high in newly diagnosed hypertensive populations [7]. Additionally, CAVI, HDL-C, and LDL-C are closely associated with vascular diseases [6]. However, little research has been conducted on the relationship between the CAVI and lipid levels in patients with hypertension in a real clinical environment. In the present study, we investigated this relationship in patients with hypertension.

Materials and methods

Patients

The retrospective study included patients from the outpatient Department of Vascular Medicine of Peking University Shougang Hospital and Jinding Street Community Health Service Center from January 2013 to December 2020. Patients with complete biochemical results, arteriosclerosis indices (CAVI), a clear history of disease, and medication were enrolled. Patients with heart failure, liver or kidney dysfunction, systemic inflammatory diseases, peripheral arterial occlusive disease, atrial fibrillation, infectious diseases, or cancer were excluded from the study. Finally, 2,656 patients (male/female: 1,016/1,640) were enrolled.

Hypertension was defined as ≥ 140/90 mmHg three or more times on different days, current use of antihypertensive drugs, or a previous diagnosis of hypertension. Coronary artery disease was defined as coronary artery stenosis greater than 70%, as confirmed through coronary angiography or computed tomography (CT) angiography. Diabetes mellitus was defined as fasting plasma glucose (FPG) ≥ 7.0 mmol/L or plasma glucose of 2 h after meal ≥ 11.1 mmol/L or random plasma glucose ≥ 11.1 mmol/L. Stroke was defined as focal or systemic neurological dysfunction caused by brain damage resulting from bleeding or infarction, as detected on CT or magnetic resonance imaging. Hyperlipidemia was defined as LDL-C ≥ 4.10 mmol/L or triglyceride (TG) levels ≥ 1.70 mmol/L according to the Clinical Laboratory of Peking University Shougang Hospital or currently receiving antilipidemic medications. All the participants provided written informed consent.

Patient and public involvement

This retrospective, cross-sectional study was conducted with support from the Peking University Shougang Hospital fund and other projects. No intervention measures were taken, as the study relied solely on the clinical diagnosis and treatment of patients. During the initial visit, the patients were informed that their clinical data might be used for future analysis and publication of academic papers, and the consent was obtained. We are also deeply grateful for the patients' participation.

Cardio-ankle vascular index measurement

CAVI was recorded using a VaseraVS-1000 vascular screening system (Fukuda Denshi, Tokyo, Japan). The patient was in a supine position with the pillow removed, with the palms of both hands facing upwards on

either side of the body. Tie the blood pressure cuff around the upper arm and lower ankle of the limbs. The distance between the lower edge of the upper arm cuff and the transverse crease of the elbow socket is 2–3 cm, and the tightness of the cuff should be just enough to fit one finger. The distance between the lower edge of the lower limb cuff and the medial malleolus is 5 cm, and the tightness of the cuff is the same as above. Two electrodes are placed on each wrist to collect electrocardiogram signals. A miniature microphone is placed in the sternum to collect heart sound signals. Firstly, the cuff pressure is increased to 30–50 mmHg, and the instrument automatically detects the heart ankle pulse wave velocity. Then, the pressure oscillation method is used to measure the systolic and diastolic blood pressure (DBP) of both upper and lower limbs. After automatic measurements, the data obtained were analyzed using the software, and the CAVI value was automatically obtained.

Laboratory measurement

All patients were collected fasting venous blood and divided into different tubes according to laboratory examination requirements. Lipids, glucose, uric acid (UA), homocysteine, C-reactive protein, and other biomarkers were analyzed using an autoanalyzer (HITACHI-7170, Hitachi, Tokyo, Japan) at the Central Chemistry Laboratory of Peking University Shougang Hospital.

Statistical analysis

Comparisons between the two groups were performed using a t-test. Wilcoxon-Mann-Whitney test was used for non-normally distributed data (C-reactive protein, TG/HDL-C ratio, TG). Multiple groups were compared using analysis of variance. SPSS (version 26.0) was used for the statistical analysis. Proportions were analyzed employing the χ^2-test. Multiple linear regression was used to describe the dependency relationship between a dependent variable and multiple independent variables. Values are presented as mean ± SD unless otherwise stated. Statistical significance was set at $P < 0.05$ (2-tailed).

Results

Clinical characteristics of the study patients

The basic clinical characteristics of the study patients are presented in Table 1. Age, body mass index (BMI), waist circumference, hip circumference, CAVI, systolic blood pressure (SBP), DBP, creatinine, FPG, UA, TG/HDL-C ratio, hypersensitive C-reactive protein (hs-CRP), homocysteine, HbA1c, and TG levels were significantly higher in the hypertension group than in the non-hypertension group. Levels of total cholesterol (TC), LDL-C, and HDL-C were significantly lower in the hypertension group than in the non-hypertension group. In addition, the incidences of diabetes mellitus, stroke, and coronary artery disease were significantly higher in the hypertension group than in the non-hypertension group. There were significant differences in sex, smoking rate, and use of antihypertensive drugs, statins, and hypoglycemic agents between the two groups. However, it was interesting to note that none of the enrolled patients were on lower-TG drugs, such as fenofibrate, likely because their TG levels were not particularly high (e.g., not three times higher), and they were not taking any medication.

A significant difference was observed in sex composition between the hypertension and non-hypertension groups; therefore, all patients were divided into two groups according to sex (Table 1). As shown in Table 2, age, BMI, waist circumference, hip circumference, CAVI, SBP, DBP, creatinine, FPG, UA, homocysteine, and HbA1c were significantly higher in males than in females. TC, LDL-C, and HDL-C levels were significantly lower in males than in females. There was no significant difference in TG level between the male and female groups.

Next, all patients were divided into four groups according to the presence of hypertriglyceridemia and/or hyper-LDL-C: Group 1, patients with normal TG and LDL-C; Group 2, patients with normal TG and hyper-LDL-C; Group 3, patients with hypertriglyceridemia and normal LDL-C; and Group 4, patients with hypertriglyceridemia and hyper-LDL-C. As shown in Table 3, the CAVI value was significantly higher in Group 3 than in Group 2.

Table 1. Clinical characteristics in hypertension and non-hypertension groups

Characteristics	Non-hypertension (*N* = 1,347)	Hypertension (*N* = 1,309)	*P* value
Age (year)	58.73 ± 8.32	63.72 ± 9.85	< 0.001
Male (%)	34.97	41.63	< 0.001
Diabetes mellitus (%)	14.48	31.17	< 0.001
Coronary artery disease (%)	7.87	27.35	< 0.001
Smoking (%)	15.66	17.80	< 0.001
Stroke (%)	4.83	19.10	< 0.001
Calcium channel blocker (%)	0.45	44.69	< 0.001
Angiotensin-converting enzyme inhibitors (%)	0.45	7.56	< 0.001
Angiotensin II receptor blocker (%)	0.30	29.34	< 0.001
Beta blockers (%)	2.82	18.03	< 0.001
Glycosidase inhibitors (%)	6.09	11.84	< 0.001
Metformin (%)	5.64	10.85	< 0.001
Sulfonylureas (%)	2.67	5.04	0.001
Insulin (%)	2.38	3.97	0.019
Statins (%)	15.59	33.16	< 0.001
Waist circumference (cm)	83.03 ± 9.08	87.46 ± 9.35	< 0.001
Hip circumference (cm)	95.84 ± 7.11	98.47 ± 7.77	< 0.001
BMI (kg/m^2)	24.50 ± 3.32	26.00 ± 6.20	< 0.001
CAVI	8.27 ± 1.17	8.73 ± 1.42	< 0.001
SBP (mmHg)	127.93 ± 14.65	141.06 ± 17.77	< 0.001
DBP (mmHg)	80.75 ± 13.37	86.30 ± 11.82	< 0.001
Creatinine (μmol/L)	64.83 ± 14.59	70.14 ± 29.70	< 0.001
FPG (mmol/L)	5.99 ± 2.10	6.27 ± 1.88	< 0.001
UA (μmol/L)	315.64 ± 116.52	340.76 ± 176.37	< 0.001
TC (mmol/L)	5.34 ± 1.10	4.86 ± 1.17	< 0.001
HDL-C (mmol/L)	1.40 ± 0.34	1.26 ± 0.43	< 0.001
LDL-C (mmol/L)	3.03 ± 0.90	2.76 ± 0.84	< 0.001
TG/HDL-C ratio	1.36 ± 1.53	1.73 ± 5.09	< 0.001
hs-CRP (mg/L)	3.12 ± 7.10	2.08 ± 4.19	< 0.001
Homocysteine (μmol/L)	11.77 ± 6.49	13.74 ± 7.19	< 0.001
HbA1c (%)	5.74 ± 1.06	6.06 ± 1.20	< 0.001
TG (mmol/L)	1.70 ± 1.34	1.97 ± 7.22	0.005

Comparisons between the two groups were analyzed by *t*-test, and the Wilcoxon-Mann-Whitney test was used for non-normally distributed data (hs-CRP, TG/HDL-C ratio, TG). Proportions were analyzed by χ^2-test. BMI: body mass index; CAVI: cardio-ankle vascular index; DBP: diastolic blood pressure; FPG: fasting plasma glucose; HDL-C: high-density lipoprotein cholesterol; hs-CRP: hypersensitive C-reactive protein; LDL-C: low-density lipoprotein cholesterol; SBP: systolic blood pressure; TC: total cholesterol; TG: triglycerides; UA: uric acid

Multiple linear regression analysis

Multiple linear regression was used to describe the dependency relationship between CAVI and multiple independent variables such as age, BMI, SBP, DBP, creatinine, FPG, UA, TC, TG, HDL-C, LDL-C, and other variables that affected the value of CAVI. As shown in Table 4, age, waist circumference, UA, FPG, HDL-C, hs-CRP, HbA1c, BMI, SBP, and DBP were independently associated with CAVI in all patients (β = 0.445, P < 0.001; β = 0.056, P = 0.012; β = 0.047, P = 0.026; β = 0.055, P = 0.040; β = –0.054, P = 0.019; β = –0.041, P = 0.048; β = 0.103, P < 0.001; β = –0.241, P < 0.001; β = 0.115, P < 0.001; β = 0.066, P = 0.017; respectively). However, in the hypertension group, only age, BMI, and SBP were independently linked to CAVI (β = 0.391, P < 0.001; β = –0.333, P < 0.001; β = 0.145, P = 0.001; respectively, Table 5).

Binomial logistic regression analysis involving independent variables—sex, history of hypertension and diabetes mellitus, smoking, and history of drug usage—was conducted. The usage of beta blockers was negatively correlated with CAVI (β = –0.411, P = 0.011), and sex (male), as well as the history of hypertension and diabetes mellitus, was positively correlated with CAVI (β = 0.419, P <0.001; β = 0.247, P = 0.011; β = 0.638, P < 0.001; respectively, Table 6).

Table 2. Clinical characteristics in male and female groups

Characteristics	Male (*N* = 1,016)	Female (*N* = 1,640)	*P* value
Age (year)	62.00 ± 10.11	60.69 ± 9.00	0.001
Diabetes mellitus (%)	24.61	21.52	0.062
Coronary artery disease (%)	22.24	14.57	< 0.001
Hypertension (%)	53.64	46.59	< 0.001
Smoking (%)	39.67	2.50	< 0.001
Stroke (%)	16.44	9.02	< 0.001
Calcium channel blocker (%)	25.00	20.55	0.008
Angiotensin-converting enzyme inhibitors (%)	5.12	3.23	0.015
Angiotensin II receptor blocker (%)	14.47	14.70	0.877
Beta blockers (%)	12.70	8.84	0.001
Glycosidase inhibitors (%)	8.66	9.09	0.766
Metformin (%)	8.56	7.99	0.596
Sulfonylureas (%)	4.43	3.48	0.213
Insulin (%)	3.25	3.11	0.841
Statins (%)	25.98	23.23	0.106
Waist circumference (cm)	89.38 ± 8.41	82.45 ± 9.06	< 0.001
Hip circumference (cm)	98.35 ± 6.64	96.14 ± 7.79	< 0.001
BMI (kg/m^2)	25.46 ± 3.26	25.10 ± 5.83	0.042
CAVI	8.68 ± 1.41	8.38 ± 1.24	< 0.001
SBP (mmHg)	135.91 ± 16.97	133.46 ± 17.81	< 0.001
DBP (mmHg)	85.43 ± 12.70	82.27 ± 12.93	< 0.001
Creatinine (μmol/L)	78.37 ± 19.74	60.67 ± 23.00	< 0.001
FPG (mmol/L)	6.26 ± 1.91	6.05 ± 2.05	< 0.001
UA (μmol/L)	363.76 ± 141.42	306.02 ± 150.21	< 0.001
TC (mmol/L)	4.77 ± 1.13	5.31 ± 1.12	< 0.001
HDL-C (mmol/L)	1.21 ± 0.30	1.41 ± 0.42	< 0.001
LDL-C (mmol/L)	2.72 ± 0.84	3.00 ± 0.89	< 0.001
TG/HDL-C ratio	1.66 ± 1.93	1.47 ± 4.51	< 0.001
hs-CRP (mg/L)	2.79 ± 6.47	2.46 ± 5.38	0.091
Homocysteine (μmol/L)	15.50 ± 8.71	11.02 ± 4.77	< 0.001
HbA1c (%)	5.98 ± 1.22	5.84 ± 1.09	0.005
TG (mmol/L)	1.79 ± 1.51	1.86 ± 6.45	0.569

Comparisons between the two groups were analyzed by *t*-test, and the Wilcoxon-Mann-Whitney test was used for non-normally distributed data (hs-CRP, TG/HDL-C ratio, TG). Proportions were analyzed by χ^2-test. BMI: body mass index; CAVI: cardio-ankle vascular index; DBP: diastolic blood pressure; FPG: fasting plasma glucose; HDL-C: high-density lipoprotein cholesterol; hs-CRP: hypersensitive C-reactive protein; LDL-C: low-density lipoprotein cholesterol; SBP: systolic blood pressure; TC: total cholesterol; TG: triglycerides; UA: uric acid

Table 3. Clinical characteristics in different groups

Characteristics	Group 1 (*N* = 1,601)	Group 2 (*N* = 92)	Group 3 (*N* = 826)	Group 4 (*N* = 137)	*P* value
CAVI	8.49 ± 1.33	8.20 ± 1.15*	8.56 ± 1.33$^\#$	8.43 ± 1.18	0.085
Calcium channel blocker (%)	21.55	15.22	26.39	9.49	< 0.001
Angiotensin-converting enzyme inhibitors (%)	3.94	4.35	3.87	4.38	0.988
Angiotensin II receptor blocker (%)	13.30	13.04	17.92	10.95	0.011
Beta blockers (%)	9.93	4.35	12.47	5.84	0.013
Glycosidase inhibitors (%)	8.68	8.70	9.32	8.76	0.964
Metformin (%)	8.12	5.43	9.08	5.84	0.430
Sulfonylureas (%)	3.56	2.17	4.72	2.19	0.287
Insulin (%)	2.56	7.61	3.87	2.92	0.052
Statins (%)	23.30	21.74	27.97	15.33	0.004

Comparisons between multiple groups were used by analysis of variance. Proportions were analyzed by χ^2-test. * vs Group 1, $P < 0.05$; $^\#$ vs Group 2, $P < 0.05$. Group 1: subjects with normal TG and LDL-C; Group 2: subjects with normal TG and hyper-LDL-C; Group 3: subjects with hypertriglyceridemia and normal LDL-C; Group 4: subjects with hypertriglyceridemia and hyper-LDL-C. CAVI: cardio-ankle vascular index

Table 4. Relationship between CAVI and study variables among the entire study group

Characteristics	*β* coefficient	SE	*P* value	Variance inflation factor
Age (year)	0.445	0.003	< 0.001	1.081
Waist circumference (cm)	0.056	0.001	0.012	1.153
Hip circumference (cm)	0.057	0.005	0.085	2.589
Creatinine (μmol/L)	0.014	0.001	0.525	1.079
UA (μmol/L)	0.047	0.000	0.026	1.046
FPG (mmol/L)	0.055	0.014	0.040	1.668
TG (mmol/L)	0.002	0.003	0.914	1.013
HDL-C (mmol/L)	–0.054	0.060	0.019	1.241
LDL-C (mmol/L)	–0.032	0.028	0.158	1.180
hs-CRP (mg/L)	–0.041	0.011	0.048	1.022
Homocysteine (μmol/L)	0.015	0.004	0.497	1.082
HbA1c (%)	0.103	0.027	< 0.001	1.675
BMI (kg/m^2)	–0.241	0.011	< 0.001	2.613
SBP (mmHg)	0.115	0.002	< 0.001	1.830
DBP (mmHg)	0.066	0.002	0.017	1.785

Multiple linear regression analysis was used. BMI: body mass index; CAVI: cardio-ankle vascular index; DBP: diastolic blood pressure; FPG: fasting plasma glucose; HDL-C: high-density lipoprotein cholesterol; hs-CRP: hypersensitive C-reactive protein; LDL-C: low-density lipoprotein cholesterol; SBP: systolic blood pressure; TG: triglycerides; UA: uric acid

Table 5. Relationship between CAVI and study variables in the hypertension group

Characteristics	*β* coefficient	SE	*P* value	Variance inflation factor
Age (year)	0.391	0.006	< 0.001	1.083
Waist circumference (cm)	0.047	0.001	0.173	1.074
Hip circumference (cm)	0.087	0.008	0.113	2.651
Creatinine (μmol/L)	–0.002	0.001	0.957	1.062
UA (μmol/L)	0.039	0.000	0.259	1.033
FPG (mmol/L)	0.094	0.031	0.089	2.698
TG (mmol/L)	–0.011	0.004	0.737	1.017
HDL-C (mmol/L)	–0.025	0.073	0.481	1.125
LDL-C (mmol/L)	–0.054	0.049	0.134	1.133
hs-CRP (mg/L)	–0.016	0.021	0.636	1.027
Homocysteine (μmol/L)	0.030	0.006	0.389	1.095
HbA1c (%)	0.072	0.054	0.194	2.731
BMI (kg/m^2)	–0.333	0.017	< 0.001	2.630
SBP (mmHg)	0.145	0.004	0.001	1.593
DBP (mmHg)	0.050	0.004	0.242	1.593

Multiple linear regression analysis was used. BMI: body mass index; CAVI: cardio-ankle vascular index; DBP: diastolic blood pressure; FPG: fasting plasma glucose; HDL-C: high-density lipoprotein cholesterol; hs-CRP: hypersensitive C-reactive protein; LDL-C: low-density lipoprotein cholesterol; SBP: systolic blood pressure; TG: triglycerides; UA: uric acid

Table 6. Relationship between CAVI and medical history and medication usage

Characteristics	*β* coefficient	SE	*Z* value	*P* value	Exp (*β*)	95% CI for Exp (*β*)	
						Lower limit	Upper limit
Beta blockers (+: 1; –: 0)	–0.411	0.161	6.534	0.011	0.663	0.484	0.909
Gender (male: 1; female: 0)	0.419	0.094	19.941	< 0.001	1.520	1.265	1.826
Hypertension (+: 1; –: 0)	0.247	0.097	6.435	0.011	1.280	1.058	1.550
Diabetes mellitus (+: 1; –: 0)	0.638	0.107	35.342	< 0.001	1.893	1.534	2.336

Binomial logistic regression analysis was used. CAVI: cardio-ankle vascular index

Discussion

The present study showed that the CAVI was significantly higher in patients with hypertension. CAVI values differed according to sex. While we did not find a significant correlation between CAVI and TG, blood pressure should be maintained to prevent the development of arteriosclerosis.

The increase in arterial stiffness is not only a manifestation of the progression of hypertension but also an important predictor of future cardiovascular and cerebrovascular events in hypertensive populations [8]. Arterial stiffness can be represented by CAVI, which is derived from the stiffness parameter β [9]. The CAVI is a reliable indicator for evaluating arterial stiffness in patients with hypertension, diabetes, and metabolic syndrome [10]. It is a useful tool for evaluating macroangiopathy in patients with diabetes mellitus [11]. There is a relationship between the stage of diabetic retinopathy and CAVI [12]. Moreover, the CAVI is a useful parameter for identifying ischemic heart disease in patients with acute heart failure [13]. An increase in CAVI is associated with an increase in left ventricular mass and a decrease in cardiac contractile function [14]. The present study found that the CAVI was significantly higher in patients with hypertension, similar to our previous research findings [15–17]. Thus, the CAVI is an effective indicator of arterial stiffness [18].

Male and female patients with hypertension exhibit different incidence rates owing to variations in lifestyles, stress, internal environments, and other factors. Consequently, there are also differences in vascular function assessment between the sexes. There was a relationship between high CAVI and high blood pressure categories in males but not in females [19]. The study suggested that the optimal control and management strategy of hypertension was not only dependent on age, obesity, diabetes, etc., but also on sex. The LDL-C/HDL-C ratio has been positively associated with the presence of carotid plaques in male patients but not in female patients [20]. The present study showed a significant difference in sex composition between the hypertension and non-hypertension groups in terms of age, BMI, waist circumference, hip circumference, CAVI, SBP, DBP, creatinine, FPG, UA, homocysteine, HbA1c, among other factors.

Dyslipidemia—characterized by increased TG and LDL-C levels and decreased HDL-C levels—is involved in the development of coronary atherosclerosis. Dyslipidemia accounts for a large proportion of patients newly diagnosed with hypertension [7]. Blood lipids are associated with CAVI, suggesting that they are related to early vascular damage [21]. Elevated CAVI is associated with abnormal blood lipid and glucose metabolism, advanced age, increased ventricular rate, elevated mean arterial pressure, and worsening cardiovascular events in middle-aged metabolic syndrome patients [22]. The present study showed that CAVI was negatively correlated with HDL-C levels in all patients. HDL-C serves as a protective factor against vascular diseases [23]. Furthermore, an increase in CAVI and a decrease in HDL-C levels have been significantly correlated with the incidence of cardiovascular events, even after adjusting for age and sex [24]. A significant association between CAVI and metabolic syndrome components has been reported [25]. The present study showed that BMI was an independent factor associated with the CAVI in different groups, similar to the results of other studies [26]. In addition, age, waist circumference, UA, FPG, HDL-C, hs-CRP, HbA1c, BMI, SBP, and DBP were independently associated with CAVI in all patients.

We believe that patients with higher CAVI had worse lipid profiles, including elevated TC and LDL-C and decreased HDL-C. However, negative correlations between CAVI and LDL-C were observed only in the non-risk groups, including patients without diabetes who underwent a routine health checkup [27]. A community-based study involving Japanese community dwellers considered to be at low risk for atherosclerosis was based on their level of traditional CVD risk factors. These factors showed that high-sensitivity C-reactive protein was significantly positively associated with CAVI; however, no clear association was observed between CAVI and LDL-C [28]. The present study showed no significant correlation between the CAVI and risk factors, including LDL-C. Statins and other drugs may also have affected these results.

Studies have shown that angiotensin II receptor blockers such as olmesartan and calcium channel blockers including amlodipine can affect the CAVI index in patients with hypertension [29, 30]. Nicorandil administration may be effective in relieving myocardial injury and/or cardiac burden in patients with stable angina after percutaneous coronary intervention by decreasing the CAVI [31]. Significant decreases in CAVI were observed in patients after pitavastatin treatment for 12 months [32]. The CAVI significantly decreased in the statin group during the first year of the TOHO Lipid Intervention Trial Using Pitavastatin Study [33]. CAVI was significantly decreased in patients with type 2 diabetes treated with hypoglycemic

drugs, such as glimepiride [34]. The present study showed that a history of hypertension and diabetes mellitus, particularly in males, was positively correlated with CAVI. Additionally, the usage of beta blockers was negatively correlated with CAVI.

Many studies have shown a relationship between TG and arterial stiffness as evaluated using pulse wave velocity. These studies showed that TG positively correlates with pulse wave velocity [35]. There was a positive correlation between high TG and increased pulse wave velocity in the general population with LDL-C ≤ 119 mg/dL [36]. A recent study showed that TG was associated with pulse wave velocity in a Chinese population with hypertension [37]. However, little research has been conducted on CAVI and TG in patients with hypertension. In a recent study, univariate analysis showed that TG levels were positively associated with the CAVI. However, multivariate analysis showed that TG level was not an independent factor related to the CAVI [38]. However, another study showed that patients with hypertriglyceridemia had a higher adjusted CAVI than those with dyslipidemia [21]. A recent multicenter and international study showed that CAVI was significantly positively correlated with hyperglycemia and hypertension, but not significantly correlated with HDL-C and TG levels, and negatively correlated with the overweight component. This important finding may be owing to the heterogeneous effects of the metabolic syndrome components on CAVI [39]. Our study showed that there was no significant difference between CAVI and TG, and other lipids, such as LDL-C and HDL-C, were not correlated with TG, which was not the case in a previous study. However, TC and LDL-C levels were lower in patients with hypertension.

Dyslipidemia is the primary factor involved in the occurrence and progression of atherosclerosis, especially LDL-C. However, this study did not find any abnormalities that might be associated with the large number of diseases or confounding factors in the enrolled population. TG levels have shown varying results in several studies. Although previous studies found a relationship between TG levels and arteriosclerosis, no abnormalities were found in this study. This study had certain limitations. First, the selected population was complex and included outpatients from tertiary hospitals and community health service centers. Second, the patients had various accompanying diseases, and the population with vascular events, such as coronary heart disease and cerebral infarction, was different from that with a single disease. Third, many types of medications are available to the patients, such as antihypertensive, lipid-lowering, and hypoglycemic drugs. Some drugs could improve arteriosclerosis and lower LDL-C, thereby interfering with the statistical results. Fourth, in the real world, patients with hypertension, diabetes, coronary heart disease, and cerebral infarction are often encountered. Prevention and control of chronic diseases are key to preventing recurrence in such patients. Many risk factors affect blood vessels; therefore, it is important to explore vascular function and its influencing factors. Our research suggests that blood lipids were not associated with arteriosclerosis in this population, suggesting that there may be other factors, such as inflammation, and that blood lipids were only one component of the overall process of arteriosclerosis. In addition, males and females have different disease incidence rates owing to different lifestyles, pressures, internal environments, and other factors, and the heterogeneity of sex differences can also affect the results. Finally, as a retrospective cross-sectional study, it could only establish associations and not causal relationships. Therefore, a large, longitudinal prospective study should be conducted in the future.

In conclusion, CAVI was significantly higher in patients with hypertension. The CAVI values showed sex-based differences. Although we did not find a significant correlation between CAVI and TG, blood pressure should be maintained to prevent the development of arteriosclerosis.

Abbreviations

BMI: body mass index

CAVI: cardio-ankle vascular index

DBP: diastolic blood pressure

FPG: fasting plasma glucose

HDL-C: high-density lipoprotein cholesterol

hs-CRP: hypersensitive C-reactive protein

LDL-C: low-density lipoprotein cholesterol

SBP: systolic blood pressure

TC: total cholesterol

TG: triglyceride

UA: uric acid

Declarations

Author contributions

JL: Conceptualization, Writing—original draft, Writing—review & editing. HW: Conceptualization, Writing—review & editing. HL, HZ and NZ: Formal analysis, Writing—review & editing. All authors have read and agreed to the published version of the manuscript.

Conflicts of interest

No conflicts of interest, financial or otherwise, are declared by all authors.

Ethical approval

This study was approved by the ethics committee of Peking University Shougang Hospital (reference number: SGYYZ202105).

Consent to participate

During the initial visit, the participant was informed that their clinical data might be used for future analysis and publication of academic papers, and the informed consent was obtained from all individual participants included in the study. We are also deeply grateful for the patient's participation.

Consent to publication

The patients were informed that their clinical data might be used for future analysis and publication of academic papers, and the consent was obtained.

Availability of data and materials

The datasets that support the findings of this study are available from the corresponding author upon reasonable request.

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