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Relationship of a Body Shape Index and Body Roundness Index with carotid atherosclerosis in arterial hypertension

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Abstract *Background and aims:* A Body Shape Index (ABSI) and Body Roundness Index (BRI) are two new anthropometric adiposity indices that have shown to be associated better than BMI with adipose abdominal tissue, with the onset of diabetes and the risk of premature death. Little is known about the influence of ABSI and BRI on subclinical vascular damage. The study was aimed to assess the relationship between ABSI and BRI with carotid atherosclerosis damage in subjects with arterial hypertension.
Methods and results: A total of 468 patients with arterial hypertension (30–80 years old) were enrolled; adiposity indices were calculated (BMI, WC, ABSI, BRI) and carotid ultrasonographic examination was performed to detect atherosclerotic damage (IMT or atherosclerotic plaque). BRI, but not ABSI, was higher in subjects with IMT> 0.90 mm in comparison to those with a lower IMT ($p < 0.001$), whereas patients with carotid plaques showed higher values of ABSI ($p = 0.001$), as well as of BRI ($p = 0.003$). Linear regression analysis disclosed significant correlation of IMT with ABSI, BRI and BMI (all $p < 0.001$). In the multivariate analysis, BRI was independently correlated with cIMT ($p = 0.015$). On the contrary, ABSI did not show any independent association with cIMT. However, ABSI was strongly associated with carotid plaques in multiple logistic regression analysis after adjustment for potential confounding factors. When BRI or BMI replaced ABSI into the multivariate models, they did not show any independent correlation with carotid plaques.
Conclusions: ABSI may be proposed as a better correlate of carotid atherosclerosis than the traditional measures of adiposity.

Introduction

Obesity is an independent risk factor for the development of cardiovascular disease and mortality [1,2]. Several efforts have been made to identify objective and reproducible indices of obesity able to detect the adipose tissue accumulation as well as to demonstrate its correlation with cardiovascular events.

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Body Mass Index (BMI), calculated as the ratio between weight and the square of height, has long been used for its simplicity and for the established relationship with mortality and obesity-related comorbidity [1–4].

However, BMI has some important limitations. It does not allow the distinction between individuals with higher lean mass, with a better cardiovascular prognosis, and those with predominant fat mass [2,5–7]. Moreover, BMI does not provide information about district distribution of adiposity [2,5–8], which has shown to play an important role in predicting cardiovascular risk.

Although waist circumference (WC) is often advocated as a simple anthropometric marker of central obesity and associated cardiometabolic risk [2,8,9], it is not a measure without limitations. First, WC cut-off points cannot be used universally across gender or race [10,11]. Moreover, it has recently been shown that the risk of metabolic syndrome within a given WC strata is significantly higher among shorter individuals [12].

Recently, two new indices have been developed to overcome the limits of traditional obesity markers. A Body Shape Index (ABSI) introduced by Krakauer et al. [13] is based on waist circumference (WC) adjusted for height and weight. It has been shown to be associated with adipose abdominal tissue [13], with the risk of premature death [13] and with the onset of diabetes [14] better than BMI.

Similarly, in 2013 Thomas et al. have developed the Body Roundness Index (BRI), a geometric index that combines height and WC to predict the percentage of total and regional fat [15]. However, data on the relationship between these new obesity indices and the risk of cardiovascular disease are not homogeneous [16,17], and no study exists on the prognostic role that these markers have in subjects with arterial hypertension. Furthermore, no studies have been carried out to evaluate whether ABSI and BRI may be associated with subclinical vascular damage which precedes and predicts the development of cardiovascular diseases.

The aim of this study is to analyse in subjects with arterial hypertension the relationship between new anthropometric indices of adiposity (ABSI and BRI) and subclinical markers of carotid atherosclerosis.

Methods

This cross-sectional study includes a total of 468 patients with hypertension, aged between 30 and 80 years. This population was selected from Caucasians patients with essential hypertension consecutively attending our nephrology and hypertension unit. In line with the 2013 clinical guidelines issued by the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC) [18], hypertension was defined as blood pressure $\geq$140/90 mm Hg or treatment with antihypertensive drugs.

The exclusion criteria were:

- severe obesity (BMI$>$ 40 kg/m^2);
- renovascular, endocrine or malignant hypertension;
- carotid thromboendarterectomies and/or carotid percutaneous angioplasty;
- Chronic Kidney Disease in dialysis replacement treatment;
- pre-existing cardiovascular comorbidities.

Written informed consent was obtained from each subject. The study protocol, that conforms to the ethical guidelines of Helsinki declaration, was approved by local review board.

Clinical and laboratory evaluation

In all patients, careful clinical history and physical examination were performed. Subjects reported smoking cigarettes habit regularly during the past year were considered current smokers. Clinic blood pressure was recorded by a doctor by a validated automatic oscillometric device (Microlife Watch BP Office, Widnau, Switzerland) [19], following the recommendations of the 2013 ESH/ESC guidelines [18].

The anthropometric parameters were recorded as follows. Body weight and height were taken with participants barefoot and in light clothing and measured to the nearest 0.1 kg and 0.1 cm, respectively. WC was measured with an inelastic tape to the nearest 0.1 cm at a midpoint between the bottom of the rib cage and the top of the iliac crest, following exhalation. BMI was calculated as weight in kilogram divided by square of the height in meters. BSA was calculated through the Dubois et al. formula:

$$BSA = 0{,}007184 \times W^{0{,}425} \times H^{0{,}725}$$

In order to assess subclinical carotid damage, a well-trained operator, unaware of clinical data of patients, performed a B-mode and Duplex-Doppler ultrasonographic examination of carotid vasculature.

Routine biochemical parameters were determined with standard techniques using an autoanalyzer (Boehringer Mannheim for Hitachi system 911, Mannheim, Germany). Low-density lipoprotein (LDL) cholesterol was calculated by the Friedewald formula. Estimated GFR (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration equation [20].

New anthropometric adiposity indices assessment

Weight, height and waist circumference were used to calculate ABSI and BRI.

ABSI was calculated with the following formula [13]:

$$ABSI = WC / BMI^{2/3} height^{1/2}$$

To calculate the BRI we used the eccentricity index ε, which is the circular degree of an ellipse between 0 (a perfect circle) and 1 (a vertical line) [15]:

$$\varepsilon = \sqrt{1 - \left(\frac{\left(\frac{WC}{2\pi}\right)^2}{(0.5\,x\,height)^2} \right)}$$

Using the height, the waist circumference and the ε index, we obtained the BRI formula, as follows:

$$BRI = 364{,}2 - (365{,}5 \times \varepsilon)$$

As described by Thomas et al., values closer to 1 are related to leaner individuals, whereas larger values are associated with rounder individuals [15].

Carotid ultrasonography detection

The examination was performed by a single well-trained operator as previously described [21], following the recommendations of the Manheim Carotid Intima-Media Thickness Consensus [22].

Carotid IMT was not obtained in correspondence of a carotid plaque, but its measurement was shifted proximally on the plaque-free site. An average value of cIMT> 0.90 mm was considered abnormal, in agreement with the ESH/ESC 2013 hypertension guidelines [18]. We considered cP those focal structures encroaching into the arterial lumen of at least 0.5 mm or 50% of the surrounding cIMT value or demonstrating a cIMT > 1.5 mm.

A total of 6 measurements were obtained for each side, and we used the overall average cIMT value.

Statistical analyses

Statistical analyses were initially carried out in the whole study population (n = 468), and it was subsequently performed in the population divided in groups based on cIMT (≤0.90 mm or > 0.90) or on the presence or absence of carotid plaques.

Continuous variables were given as mean ± SD, except serum triglycerides that for its skewed distribution were expressed as median and interquartile range and were log-transformed. The categorical variables were presented as percentage values.

Student's *t* test for independent samples was used to compare continuous variables between 2 groups.

Proportional differences between groups were assessed by the χ^2 test, with Yates' correction (or Fisher exact test when appropriate).

In order to assess the influence of gender on the relationships of the adiposity indices with cIMT and cP, we performed a two-way analysis of variance. Interaction of the adiposity indices examined with cIMt and with CP was formally tested by assessing the significance of the multiplicative two-way interaction terms 'gender x BMI', 'gender x WC', 'gender x WtHr', 'gender x ABSI', 'gender x BRI', along with the main effects of sex and the adiposity indices.

The univariate and multivariate relationships between the variables were tested by simple and multiple linear regression analyses. The strength of the associations between the variables was expressed respectively by the Pearson correlation coefficients (*r*) and the unstandardized (B) and standardized (β) multiple regression coefficients.

Stepwise multivariate linear regression analyses first were run considering cIMT as a dependent variable and including into the model: BMI, age, sex, smoking, eGFR, SBP, DBP, uricemia, glycemia (or diabetes as dichotomous variable), LDL, HDL, triglycerides (log-transformed), statins therapy, antidiabetic therapy. Afterwards, other multivariate models were made replacing BMI alternately with WC, WtHR, ABSI and BRI. Finally, new statistical models were built by using the same BMI to enter among the covariates together with the other indices, to better assess the relationship between new anthropometric adiposity indices and cIMT independently of BMI.

Moreover, we performed logistic regression analyses to study the relationship between new anthropometric adiposity measures and carotid plaques (cP), being these latter regarded as outcome variable (0 = no plaques; 1 = plaques) and including the same covariates used to perform the above described linear regression analysis.

Moreover, the multivariate effects of sex on the relationships between adiposity and carotid damage were formally tested by adding as explanatory variables the multiplicative two-way interaction terms 'gender x BMI' or 'gender x BRI', or 'gender x ABSI' into the multiple regression models above described.

ROC curves were built and the areas under the ROC curves (AUC) were calculated to establish the ability of the new anthropometric indices to assess the vascular carotid damage (cIMT or carotid plaque), and to define the best cut-off value: values which maximize the Youden index (sensibility + specificity − 1) were defined as optimal.

Using the Hanley and McNeil method [23], we assessed the significance of the difference between the BMI diagnostic capacity and that one of the new indices. The null hypothesis was rejected at a $p < 0.05$.

Statistical analyses were carried out using the SPSS software 21.0.

Results

Clinical and demographic characteristics and the percentages of patients treated with cardiovascular drugs in the overall study population and in the population divided in subjects with cIMT values lower and greater than 0.90 mm and in patients with or without carotid plaques are shown respectively in Table 1a and in Table 1b.

The percentage of subjects with a BMI ≥30 kg/m2 was higher (77%) in the group with greater values of cIMT than in that one with a cIMT ≤ 0.90 mm (60%; $p < 0.001$). A similar trend was observed by comparing the groups divided on the basis of the presence (35%) or the absence of cP (29%), but this difference did not attain statistical significance.

Moreover, BRI, but not ABSI, was higher in subjects with cIMT> 0.90 mm in comparison to those with a lower cIMT (Fig. 1a). However, BRI as well as ABSI, were greater in subjects with cP than in those without any plaque (Fig. 1b).

Table 1a Demographic and clinical data of the overall study population and the population divided into groups based on cIMT values lower and greater than 0.90 mm and in patients with or without carotid plaques.

	Overall study population (n = 468)	Subjects with cIMT ≤ 0.90 mm (n = 230)	Subjects with cIMT > 0.9 mm (n = 238)	*p-value*	Subjects with no carotid plaques (n = 284)	Subjects with carotid plaques (n = 184)	*p-value*
Age (years)	58 ± 14	54 ± 16	61 ± 12	<0.001	54 ± 15	65 ± 10	<0.001
Males (%)	59.6	60.4	58.8	*NS*	57.0	63.6	*NS*
Smokers (%)	29.5	27.8	31.1	*NS*	26.4	34.2	*NS*
Diabetics (%)	34.4	26.5	42.0	<0.001	35.6	32.6	*NS*
Obesity (%)	23.1	23.0	39.9	<0.001	29.2	35.3	*NS*
BMI < 25 kg/m^2 (%)	25.64	29.6	21.8	*NS*	28.9	20.7	*NS*
eGFR < 60 ml/min/1.73m^2 (%)	23.1	12.6	33.2	<0.001	19.0	29.3	0.013
Height (cm)	166 ± 10	167 ± 9	165 ± 10	0.037	167 ± 10	165 ± 10	0.017
Weight (Kg)	77.7 ± 15.8	76.7 ± 16.2	78.8 ± 15.5	*NS*	77.5 ± 16.5	78.0 ± 14.8	*NS*
BMI (kg/m^2)	28.1 ± 4.7	27.4 ± 4.6	28.8 ± 4.7	0.001	27.7 ± 4.8	28.7 ± 4.4	0.023
WC (cm)	94 ± 14	92 ± 13	96 ± 14	0.003	92 ± 14	96 ± 12	0.012
WHtR	0.57 ± 0.09	0.55 ± 0.08	0.58 ± 0.09	<0.001	0.56 ± 0.09	0.58 ± 0.09	0.001
BSA (m^2)	1.85 ± 0.22	1.85 ± 0.21	1.86 ± 0.22	*NS*	1.86 ± 0.22	1.85 ± 0.20	*NS*
ABSI (m$^{11/6}$kg$^{-2/3}$)	0.788 ± 0.048	0.783 ± 0.051	0.792 ± 0.044	*NS*	0.782 ± 0.046	0.797 ± 0.049	0.001
BRI	4.857 ± 2.071	4.518 ± 1.878	5.184 ± 2.196	<0.001	4.628 ± 2.090	5.210 ± 1.997	0.003
cIMT (mm)	0.91 ± 0.23	0.73 ± 0.14	1.09 ± 0.13	<0.001	0.89 ± 0.24	0.95 ± 0.20	0.003
cIMT > 0.90 mm (%)	50.9	0.0	100.0	<0.001	48.6	54.3	*NS*
Carotid plaques (%)	39.3	36.5	42.0	*NS*	0.0	100.0	<0.001
Clinic SBP (mmHg)	134 ± 17	131 ± 17	137 ± 16	<0.001	134 ± 15	135 ± 18	*NS*
Clinic DBP (mmHg)	80 ± 10	79 ± 10	80 ± 11	*NS*	81 ± 10	77 ± 11	<0.001
Clinic PP (mmHg)	55 ± 15	52 ± 14	57 ± 14	<0.001	52 ± 14	58 ± 15	<0.001
Heart rate (bpm)	71 ± 11	72 ± 12	71 ± 10	*NS*	72 ± 9	71 ± 13	*NS*
Serum glucose (mg/dl)	109 ± 42	106 ± 35	113 ± 47	*NS*	101 ± 37	122 ± 45	<0.001
Serum creatinine (mg/dl)	1.17 ± 0.78	1.02 ± 0.59	1.31 ± 0.91	<0.001	1.14 ± 0.78	1.21 ± 0.80	0.412
eGFR (ml/min/1.73 m^2)	76.6 ± 27.4	85.1 ± 24.9	68.4 ± 27.3	<0.001	80.4 ± 28.3	70.6 ± 25.0	<0.001
Serum cholesterol (mg/dl)	182 ± 40	181 ± 38	184 ± 42	*NS*	189 ± 38	172 ± 41	<0.001
c-LDL (mg/dl)	109 ± 34	107 ± 32	111 ± 36	*NS*	115 ± 33	100 ± 35	<0.001
c-HDL (mg/dl)	48 ± 13	50 ± 14	45 ± 11	<0.001	48 ± 13	47 ± 14	*NS*
Serum uric acid (mg/dl)	6.06 ± 1.71	5.74 ± 1.56	6.36 ± 1.79	<0.001	5.97 ± 1.70	6.20 ± 1.73	*NS*
Serum TRG (mg/dl)	118 (88–159)	110 (84–149)	124 (93–166)	0.001	116 (87–164)	118 (89–152)	*NS*

eGFR: estimated glomerular filtration rate; BMI: body mass index; WC: waist circumference; WHtR: Waist-to-height ratio; BSA: body surface area; ABSI: A Body Shape Index; BRI:Body Roundness Index; cIMT: carotid intima media thickness; SBP: systolic blood pressure; DBP: diastolic blood pressure; PP: pulse pressure; LDL: low density lipoprotein; HDL: high density lipoprotein; TRG: triglycerides.

Table 1b Percentage of patients treated with cardiovascular drugs in the overall study population and the population divided into groups based on cIMT values lower and greater than 0.90 mm and in patients with or without carotid plaques.

	Overall study population (n = 468)	cIMT ≤ 0.90 mm (n = 230)	cIMT ≥ 0.90 mm (n = 238)	*p-value*	No carotid plaques (n = 284)	Carotid plaques (n = 184)	*p-value*
Angiotensin II receptor antagonists (%)	27.1	26.1	28.2	*NS*	25.7	29.3	*NS*
ACE inhibitors (%)	42.5	37.8	47.1	0.050	45.4	38.0	*NS*
Diuretics (%)	30.1	28.3	31.9	*NS*	31.0	28.8	*NS*
Calcium channel blockers (%)	28.0	23.9	31.9	*NS*	32.7	20.7	0.006
Alfa blockers (%)	41.9	32.6	50.8	<0.001	45.4	36.4	*NS*
Alfa beta blockers (%)	1.5	1.7	1.3	*NS*	1.8	1.1	*NS*
Beta blockers (%)	28.2	33.5	23.1	0.014	18.3	43.5	<0.001
Central antiadrenergic drugs (%)	8.1	6.1	10.1	*NS*	8.8	7.1	*NS*
Nitrates (%)	5.8	4.8	6.7	*NS*	2.5	10.9	*NS*
Xanthine oxidase inhibitors (%)	9.0	5.7	12.2	*NS*	11.3	5.4	*NS*
Statins (%)	21.8	26.1	17.6	0.033	9.5	40.8	<0.001
Antidiabetic drugs (%)	31.8	25.7	37.8	0.002	35.2	26.6	*NS*
Antiplatelet drugs (%)		37.0	38.2	*NS*	29.9	49.5	<0.001

NS = p > 0.05; BMI: Body Mass Index; ACE: Angiotensin-Converting Enzyme.

Two-way ANOVA showed that none of the interaction terms 'gender x BMI', 'gender x WC', 'gender x WtHr', 'gender x ABSI', 'gender x BRI', used to assess the influence of gender on the relationships of the adiposity indices with cIMT and cP, attained the statistical significance (all p > 0.10).

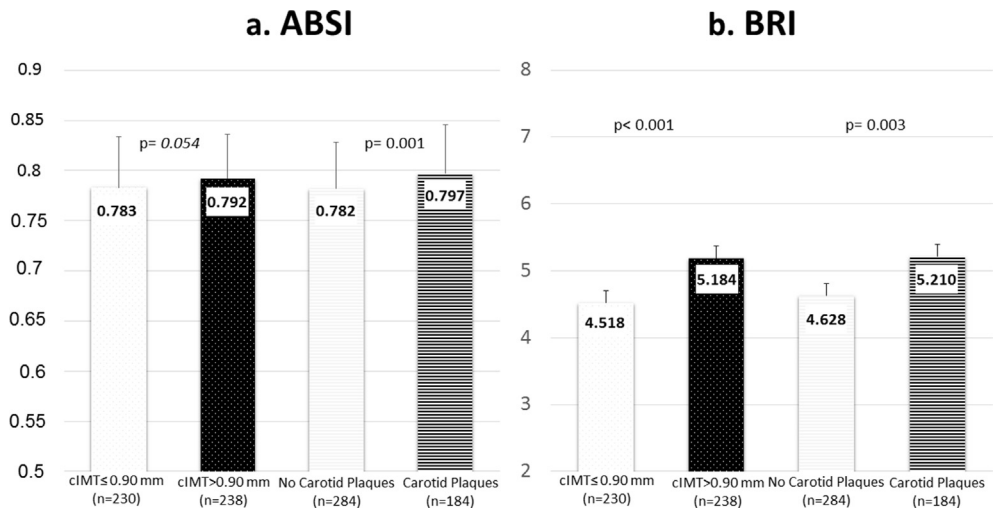

Figure 1 ABSI and BRI average levels in groups divided according to cIMT values and the presence or absence of carotid atherosclerotic plaques.

The univariate correlations between cIMT, anthropometric adiposity indices and other variables in the overall study population are shown in the Table 2.

The results of multivariate linear regression analysis, where cIMT was considered as a dependent variable, and including in the model, alternatively, BMI, BRI or ABSI among the covariates are summarised in Table 3.

BMI was independently correlated with cIMT ($p = 0.011$) (Table 3—A), and similar results were obtained with BRI (0.015) (Table 3—B). On the contrary, ABSI did not show any independent association with cIMT when added in the model along with other covariates (Table 3—C).

When we ran again the multivariate model including both BMI and BRI, only the former remained independently associated with cIMT ($p = 0.001$).

The logistic regression analyses conducted in order to assess the independent correlates of carotid plaque disclosed a significant association of cP with ABSI ($p = 0.007$), but not with BRI or BMI.

When we tested the multivariate effects of sex on the relationships between adiposity and carotid damage by adding as explanatory variables the multiplicative two-way interaction terms 'gender x BMI'or 'gender x BRI', or 'gender x ABSI' into the linear and logistic multiple

Table 2 Correlations between cIMT, fat anthropometric indices and other variables in the overall study population.

	cIMT	BMI	WC	WHtR	ABSI	BRI
	r	r	r	r	r	r
Age (years)	0.382***	0.055 NS	0.081 NS	0.165***	0.203***	0.151**
Height (cm)	−0.142**	−0.027 NS	0.001 NS	−0.384***	−0.428***	−0.391***
Weight (Kg)	0.060NS	0.795***	0.754***	0.470***	0.100*	0.453***
BMI (kg/m^2)	0.177***	/	0.942***	0.881***	0.465***	0.870***
WC (cm)	0.165***	0.942***	/	0.920***	0.685***	0.904***
WHtR	0.206***	0.881***	0.920***	/	0.793***	0.992***
BSA (m^2)	−0.004 NS	0.573***	0.559***	0.205***	−0.079 NS	0.187***
ABSI ($m^{11/6}kg^{-2/3}$)	0.130**	0.465***	0.685***	0.793***	/	0.775***
BRI	0.195***	0.870***	0.904***	0.922***	0.775***	/
Clinic SBP (mmHg)	0.229***	0.067 NS	0.034 NS	0.051 NS	−0.021 NS	0.050NS
Clinic DBP (mmHg)	0.044 NS	0.007NS	0.029 NS	−0.028 NS	−0.010 NS	−0.027 NS
Clinic PP (mmHg)	0.228***	0.070 NS	0.017 NS	0.078 NS	−0.016 NS	0.076 NS
Heart rate (bpm)	−0.095*	0.048 NS	0.067 NS	0.065 NS	0.079NS	0.073*
Serum glucose (mg/dl)	0.100*	0.177***	0.175***	0.155**	0.093*	0.143**
Serum creatinine (mg/dl)	0.230***	0.029 NS	0.033 NS	0.017 NS	0.004 NS	0.016 NS
eGFR (ml/min/1.73m^2)	−0.362***	−0.066 NS	−0.070 NS	−0.128**	−0.119**	−0.126**
Serum cholesterol (mg/dl)	0.030 NS	0.006 NS	0.017 NS	0.033 NS	0.077NS	0.020 NS
c-LDL (mg/dl)	0.031 NS	0.022 NS	0.038 NS	0.046 NS	0.081NS	0.036 NS
c-HDL (mg/dl)	−0.161***	−0.199***	−0.208***	−0.102*	−0.020 NS	−0.095*
Serum uric acid (mg/dl)	0.224***	0.220***	0.214***	0.161***	0.068 NS	0.160**
Serum TRG (mg/dl)	0.140**	0.180***	0.186***	0.112*	0.066 NS	0.092*

BMI: body mass index; WC: waist circumference; WHtR: Waist-to-height ratio; BSA: body surface area; ABSI: A Body Shape Index; BRI:Body Roundness Index; SBP: systolic blood pressure; DBP: diastolic blood pressure; PP: pulse pressure; LDL: low density lipoprotein; HDL: high density lipoprotein; TRG: triglycerides.

Table 3 Multivariate regression analyses performed considering cIMT as outcome variable and including alternatively, BMI [A], BRI [B] or ABSI [C] along with other confounding factors. The variables included in the multiple regression models are showed in the *statistical section*.

Outcome variable: cIMT	Regression coefficients			
	Not standardized		Standardized	
	B	SE	β	p
[A] Model (R^2 = 0.286)				
Age	0.005	0.001	0.306	<0.001
Clinic SBP	0.002	0.001	0.152	<0.001
eGFR	−0.001	0.000	−0.153	0.001
Antidiabetic drugs	0.061	0.020	0.124	0.002
Statins	−0.067	0.023	−0.120	0.004
BMI	0.005	0.002	0.105	0.011
HDL-c	−0.002	0.001	−0.102	0.017
Serum uric acid	0.012	0.006	0.089	0.038
[B] Model (R^2 = 0.286)				
Age	0.005	0.001	0.297	<0.001
Clinic SBP	0.002	0.001	0.155	<0.001
eGFR	−0.001	0.000	−0.149	0.002
Serum uric acid	0.013	0.006	0.095	0.025
Antidiabetic drugs	0.061	0.020	0.124	0.002
Statins	−0.066	0.023	−0.118	0.005
BRI	0.011	0.004	0.099	0.015
HDL-c	−0.002	0.001	−0.112	0.008
[C]Model (R^2 = 0.276)				
Age	0.005	0.001	0.309	<0.001
Clinic SBP	0.002	0.001	0.157	<0.001
eGFR	−0.001	0.000	−0.152	0.002
Antidiabetic drugs	0.060	0.020	0.121	0.002
HDL-c	−0.002	0.001	−0.121	0.004
Statins	−0.065	0.023	−0.116	0.006
Serum uric acid	0.014	0.006	0.107	0.012

SBP: Systolic Blood pressure; eGFR: estimated Glomerular Filtration Rate; BMI: Body Mass Index; HDL-c: High density lipoprotein cholesterol; BRI: Body Roundness Index.

regression models above described, we found that all these interaction terms were not statistically significant (all $p > 0.23$).

To assess the ability of new anthropometric adiposity indices (BRI and ABSI) to identify a carotid vascular damage, and to compare with that one of BMI, ROC curves were built and the areas under the curves (AUC) were estimated considering as dependent variables alternatively cIMT $\geq$ 0.90 mm or the presence of carotid plaques (Fig. 2). BMI, BRI and ABSI did not differ significantly in the capacity to identify a cIMT$\geq$0.90 mm, whereas ABSI showed a greater ability to identify the presence of cP compared to the BRI ($p = 0.027$).

Discussion

WHO recommends BMI and WC as valid indicators of fatness and this assumption has been supported by many studies concerning their associations with health risk [1,2]. On the other hand, there are data questioning BMI and WC reliability and indicating that they provide a false diagnosis of body fatness [2,5,6,10–12].

Recently, two new indices have been suggested to overcome the limitations and inaccuracies of traditional obesity markers: ABSI and BRI [13–15]. In the present paper we assessed their relationships with BMI and WC and with well recognised markers of subclinical carotid damage.

The most relevant finding of our study was the association between the new and the traditional anthropometric indices analysed and the atherosclerotic vascular damage in a wide group of patients with arterial hypertension, and this may represent one of the pathogenetic mechanisms that may help to explain the enhanced risk of cardiovascular events and mortality related to the body fat increase.

Moreover, our study showed that BMI, ABSI and BRI were associated to different types of vascular damage. Indeed, in our population, cIMT significantly correlated with BMI, ABSI and BRI in bivariate analyses, but only BMI and BRI showed to be independently related to cIMT once the effect of potential confounding factors was taken into account in multivariate analyses.

Conversely, ABSI was the only anthropometric index independently associated to the presence of carotid plaques.

It is likely that different obesity indices have different prognostic value, because they identify different subclinical organ damage pattern. Moreover, one can speculate

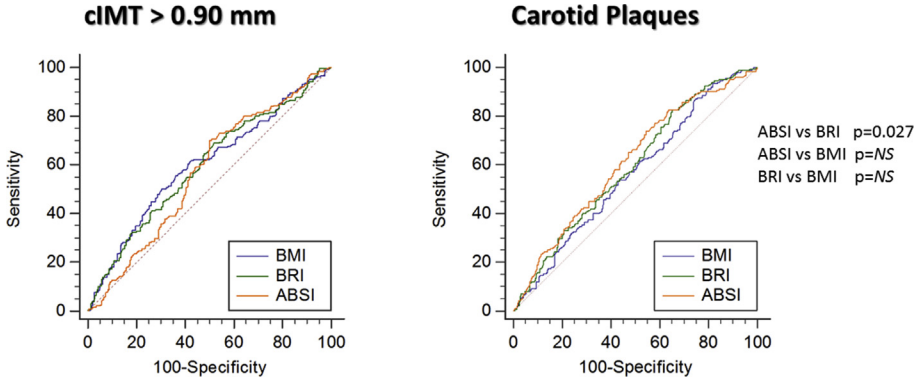

Figure 2 Comparison of ROC curves built to assess the values of BMI, BRI and ABSI able to identify a cIMT > 0.90 mm or the presence of carotid plaques.

that ABSI is linked to unknown atherogenic factors which are not related to the other adiposity markers.

A Body Shape Index (ABSI) was specifically developed as a transformation of waist circumference (WC), statistically independent of BMI to better evaluate the relative contribution of WC to central obesity and clinical outcomes [13]. Subsequent studies also found that ABSI was a better predictor of blood pressure than BMI or WC in adolescents [24] and could more accurately depict the variability in circulating insulin and lipoproteins than BMI in young, healthy male subjects [25]. However, some studies reported that ABSI seemed to be a weaker index for identifying CV risk factors than BMI [16,26–29]. Moreover, in a retrospective study conducted on 6081 Caucasian adults, the joint use of BMI and ABSI was more strongly associated with visceral abdominal fat thickness (VAT), measured by ultrasound, than BMI alone [30].

In the Diabetes Epidemiology: Collaborative analysis Of Diagnostic criteria in Europe (DECODE) study including 40 investigations from 14 European countries dealing with diabetes and its risk factors positive linear relationships of ABSI with CV and total mortality were observed, but ABSI was not superior to BMI in predicting CV disease mortality [17].

On the other hand, a recent meta-analysis including 24 retrospective cohort studies and 14 cross-sectional reports found that a standard deviation increase in ABSI was associated with an increase in the odds of hypertension by 13% and type 2 diabetes by 35% and an increase in cardiovascular disease risk by 21% and all-cause mortality risk by 55%. ABSI outperformed BMI and WC in predicting all-cause mortality but underperformed in predicting chronic diseases [31].

Thomas et al. first developed the BRI to predict body fat, the percentage of visceral adipose tissue, and establish an initial impression of an individual's physical health [15]. Up to now, only one study has used the BRI to predict disease and showed that the BRI was capable of identifying both CVD and CVD risk factors [16]. Additionally, the adjusted OR of the BRI was higher than those of the BMI and WC, although the differences were not statistically significant [16].

Recent studies have shown that BRI could be used as an adipose indicator for determining the presence of left ventricular hypertrophy [28], hyperuricaemia [29], CVD and diabetes [28].

As suggested by Thomas et al., although BRI improves quantification of body shape and provides a more accurate estimate of total %body fat and %VAT, the calculation of BRI is so complicated that it sacrifices simplicity in comparison to BMI [15]. This complication may influence its clinical application.

There are some potential limitations of our study.

The cross-sectional design of the present investigation precludes establishing causal relationships between the variables tested. Furthermore, it is worth noting that our results were obtained in a selected population of Caucasian middle-aged patients with mild-to-moderate essential hypertension, without renal insufficiency, free of CV diseases. Therefore, the conclusions of our study cannot be extrapolated to non-White populations, and caution is needed when applying the results of our investigation to different clinical settings.

On the other hand, the greatest strength of this study is that it was performed at a single clinical research centre in a large population of subjects with strictly standardized methods.

Conclusions

In summary, our study highlights the relationship between the new anthropometric adiposity indices and carotid atherosclerotic damage; BRI, along with BMI, were independently related to a cIMT >0.90 mm, and ABSI was the only anthropometric adiposity index independently associated to the presence of carotid atherosclerotic plaque.

However, the cross-sectional nature of our study does not allow us to explain the reasons of these relationships. Further studies with a different design are needed to better clarify this issue.

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