The Relationship between Body Composition and Renal Resistive Index in Newly diagnosed Hypertensive Patients

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ABSTRACT

Objective: *Obesity is a major modifiable risk factor for atherosclerosis. Thus, early risk markers are needed to identify obese subjects. The objective of this study was to examine the relationship between body composition and arterial stiffness documented by Doppler-derived renal resistive index (RRI).*

Subjects and methods: *We enrolled 120 newly-diagnosed hypertensive patients (mean age 45 ± 8 years) who were admitted to our Nephrology Clinic. Body fat percentage (BFP) was measured by bioelectrical impedance (BIA). Doppler examinations were performed and RRI was calculated for all participants.*

Results: *The female patients had higher RRI than male patients (0.69 vs 0.65,* $p \leq 0.05$*). The study patients were divided into three groups according to their BFP defined by BIA. Group three patients, who exhibited higher body fat, had significantly higher body mass index [BMI] (*$p < 0.05$*), total leukocyte count (*$p < 0.05$*), C-reactive protein [CRP] (*$p < 0.05$*), triglyceride (*$p < 0.05$*), and female predominance. Group 3 patients were statistically older than Group 1 pa-tients (46.2 vs 40.6 years,* $p < 0.05$*). Additionally, RRI levels were higher in Group 3 than in Group 1 [0.69 vs 0.65,* $p < 0.05$*] (Table 3). In logistic regression analysis, independent factors affecting RRI were age, gender, BFP and CRP levels (all p-values were < 0.05).*

Conclusions: *Body fat percentage was associated with higher RRI, in hypertensive patients. Altered renal haemodynamic profile is involved in the long-term renal risk associated with body fat distribution.*

Keywords: Body composition, hypertension, renal resistive index

Relación Entre Composición Corporal E Índice de Resistividad Renal en Pacientes Hipertensos Recién Diagnosticados

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RESUMEN

Objetivo: *La obesidad es un importante factor de riesgo modificable de la aterosclerosis. Por lo tanto, se necesitan marcadores tempranos de riesgo para identificar sujetos obesos. El objetivo de este estudio fue examinar la relación entre composición corporal y rigidez arterial documentado por el índice resistivo renal (IRR) medido por Doppler.*

Sujetos y Métodos: *Enrolamos 120 pacientes hipertensos recién diagnosticados (edad promedio 45 ± 8 años) que ingresaron a la Clínica de Nefrología. El porcentaje de grasa corporal (PGC) fue medido por impedancia bioeléctrica (AIB). Se realizaron exámenes Doppler y el IRR fue calculado para todos los participantes*

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Resultados: *Las pacientes tenían mayor IRR que los pacientes masculinos (0.69* vs *0.65,* $p \leq 0.05$*). Los pacientes del estudio fueron divididos en tres grupos según su PGC definido por AIB. Los pacientes del Grupo 3, quienes mostraron poseer una mayor grasa corporal, tuvieron significativamente mayor índice de masa corporal [IMC] (*$p < 0.05$*), recuento de leucocitos totales (*$p < 0.05$*), proteína C reactiva [PCR] (*$p < 0.05$*), triglicéridos (*$p < 0.05$*) y predominio femenino. El grupo 3 de pacientes fue estadísticamente mayor que los pacientes del Grupo 1 (46.2 vs 40.6 años,* $p < 0.05$*). Además, los niveles de IRR fueron más altos en el Grupo 3 que en el Grupo 1 [0.69 vs 0.65,* $p < 0.05$ *(cuadro 3). En el análisis de regresión logística, los factores independientes que afectaban el IRR fueron la edad, el género, los niveles de PGC y PCR (todos los valores de p fueron < 0.05).*

Conclusiones: *El porcentaje de grasa corporal se asoció con mayor IRR en pacientes hipertensos. El perfil de alteración hemodinámica renal participa en el riesgo renal a largo plazo asociado con la distribución de la grasa corporal.*

Palabras claves: Composición corporal, hipertensión, índice resistivo renal

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INTRODUCTION

Cardiovascular disease (CVD) is considered a public health burden and is the most common cause of mortality all over the world (1). Obesity is a major modifiable risk factor for CVD that goes along with atherosclerosis (2). The latency time for developing CVD may be several decades (3). Thus, early risk markers are needed to identify obese subjects at risk.

Increasing arterial stiffness is accepted as an intermediate endpoint for cardiovascular (CV) events and a surrogate marker for subclinical CVD (4, 5). Studies have shown that arterial stiffness had independent predictive value for CV mortalities in patients with uncomplicated essential hypertension (6, 7). Ultrasound and doppler imaging has been used for years in a variety of clinical settings such as detection of renal artery stenosis and arterial stiffness (7, 8). Recent clinical evidence indicates that an increased Doppler-derived renal resistive index (RRI) in patients with primary hypertension not only reflects changes in intrarenal perfusion, but it is also associated with systemic haemodynamics and atherosclerosis (9, 10). This study will give us additional information about CVD risk in hypertension beyond traditional risk factors.

The objective of this study was to examine the relationship between body composition and arterial stiffness documented by RRI. This article will briefly review the main clinical applications of RRI and its' prognostic usefulness in the management of obese hypertensive patients.

SUBJECTS AND METHODS

One hundred and twenty newly-diagnosed hypertensive patients (mean age 45 ± 8 years, 60 males) who attended our Nephrology Clinic were enrolled in the study. Study exclusion criteria were antihypertensive (present or past) drug use, diabetes mellitus (current use of anti-diabetic medications, a fasting blood glucose level of more than 126 mg/dL, and/or a random blood glucose level of more than 200 mg/dL or haemoglobin $A_{1c} > 6.2\%$), severe or secondary hypertension (≥ 210/130 mmHg), all endocrinological disorders (hyperparathyroidism, Addison's disease *etc*), renal failure (serum creatinine > 1.5 mg/dL), heart failure, peripheral or cerebral vascular disease, cancer and hepatic disease. After at least five minutes of rest in a sitting position, office BP was measured for all patients for bilateral arms using a sphygmomanometer with the appro- priate cuff size.

Three blood pressure (BPs) measurements were taken at least five minutes apart and the mean BP was used for analysis. Weight, height and waist circumference were determined for each subject; waist circumference was measured at the narrowest diameter between the costal margin and the iliac crest. Height (m) and weight (kg) were measured with the patient dressed in light clothing and without shoes. Body mass index (BMI) was calculated by the weight in kilograms divided by the square of the height in metres. Blood samples were obtained after nocturnal fasting the same week as when 24-hour ambulatory blood pressure monitoring (ABPM) was initiated. Blood chemistry was determined

by standard methods. The study was approved by the local scientific ethics committee. The patients were enrolled after providing their informed consent.

All the patients were analysed by ambulatory blood pressure monitoring ABPM recorded using the Oscar (Sun Tech Medical). The data were considered adequate when a minimum of 70 valid records were obtained in 24 hours, with at least two records per hour during the night-time. The following parameters were evaluated: average 24-hour, daytime and night-time systolic BP (SBP) and diastolic BP (DBP). Body fat percentage (BFP) was measured by bioelectrical impedance (BIA). All participants underwent at least one single-frequency BIA assessment (average of two measurements). Body composi- tion and BIA data were obtained using the Metron BioScan 916 v3 analyzer. The first pair of electrodes was placed in the hand with the inner electrode attached to the dorsum of the wrist and the outer electrode attached to the dorsal surface of the third metacarpal bone. The second pair of electrodes was placed in the ipsilateral foot, with the electrodes placed on the anterior surface of the ankle and the dorsal surface of the third metatarsal bone. A single frequency, low-amplitude imperceptible current (0.7 mA at 50 kHz) was introduced *via* the outer electrodes and the voltage reduction was detected at the wrist and ankle.

Doppler examinations were performed by Sonoline Elegra Cx 5-2 multi-Darray transducer (Siemens Medical Solutions). Doppler measurements were performed by the same sonographer. Renal resistive index was calculated by system software at an interlobar artery level according to the equations: RI = (V max – V min)/V max. V max is the maximum systolic velocity, V min is the minimum diastolic velocity, and V mean is the time-averaged mean velocity. Equations were evaluated for the threshold for an increased RI of the discriminatory level of ≥ 0.70 (11).

Statistical analysis

Descriptive statistics are presented as mean ± SD. Continuous variables were tested to detect substantial deviations from normality by computing the Kolmogorov-Smirnov Z. The assumption of satisfactory normal distribution was met for all of the examined variables. Pearson correlation coefficients were used to explore the bivariate associations between examined variables and partial Pearson correlation coefficients were used when appropriate. Stepwise multiple linear regression models were constructed using important covariates from correlation analyses to elucidate independent determinants of RI. For all of the analyses, p levels < 0.05 were considered statistically significant. The data were analysed using SPSS for Windows (version 16.0; SPSS Inc, Chicago, IL, USA).

RESULTS

A total of 120 newly-diagnosed hypertensive patients (mean age 45 ± 8 years, 60 males) were included in the study. The demographic and clinical characteristics of the recipients are presented in Table 1.

Table 1: Demographic and laboratory features of study patients according to gender

Parameters	Female (n = 60)	Male (n = 60)	*p*-value
Demographic characteristics			
Age (yr)	44 ± 9	43 ± 10	ns
BMI (kg/m^2)	28.4 ± 1.8	26.3 ± 3.6	$p < 0.05$
WHR	88 ± 10	82 ± 9	$p < 0.05$
BFP %	38.8 ±11.8	32.6 ± 12.0	$p < 0.05$
SBP(mm Hg)	142.8 ± 12.8	136.4 ± 10.8	$p < 0.05$
DBP (mm Hg)	88.5 ± 10.0	83.9 ± 11.1	$p < 0.05$
Blood analysis			
Glukoz (mg/DL)	97.6 ± 9.5	99.5 ± 10.3	ns
Creatinin (mg/DL)	0.86 ± 0.14	0.86 ± 0.13	ns
LDL (mg/DL)	125.7 ± 32.3	118.5 ± 36.2	ns
Trigliserit (mg/L)	164 ± 92	151 ± 111	ns
CRP (mg/DL)	3.1 ± 2.1	2.3 ± 1.7	ns
Hb (g/DL)	13.2 ± 0.9	14.3 ± 1.3	$p < 0.05$
Hct (%)	40.7 ± 3.8	42.2 ± 3.8	$p < 0.05$
Leukocyte count (per μL)	6.1 ± 1.9	5.9 ± 2.0	ns
PLT (per ml)	248.6 ± 60.0	272.8 ± 65.0	ns
RRI	0.69 ± 0.02	0.65 ± 0.01	$p < 0.05$

BMI: Body mass index, WHR: waist-to-hip ratio, BFP: Body fat percentage, SBP: systolic blood pressure, DBP: diastolic blood pressure LDL: Low density lipoprotein CRP: C-reactive protein Hb: haemogloblin, Hct: Haematocrite, PLT: platelet RRI: Doppler-derived renal resistive index.

We investigated the relationship between body fat, inflammation and laboratory parameters separately for males and females. Mean values of WHR, BMI, BFP, SBP, DBP were significantly higher for females than males (all p-values < 0. 05). The female patients had higher RRI than male patients (0.69 *vs* 0.65, $p \leq 0.05$).

The patients were categorized into three Groups according to their BMI; BMI I (BMI: < 24.9 kg/m^2; n = 74); BMI II (BMI: 25 to 29.9 kg/m^2; n = 27) and BMI III (BMI: ≥ 30.0 kg/m^2; n = 19). The BMI tertiles revealed that BMI III patients who exhibited BMI ≥ 30 kg/m^2 had significantly higher age (p = 0.03), BFP (p = 0.04), waiste-to-hip (WHR) (p = 0.02), triglyceride (p = 0.02) and CRP (p = 0.02) than BMI I. Additionally, RRI was higher in BMI III than BMI I [0.69 *vs* 0.66, respectively, p = 0.02] (Table 2).

Table 2: Comparison of clinical and laboratory values of patients according to body mass index

Parameters	BMI I < 24.9 (n = 74)	BMI II = 25-29.9 (n = 27)	BMI III > 30.0 (n = 19)	*p*
Age	42 ± 8	43 ± 7	44 ± 9	$p < 0.05$
WHR	83 ± 7	86 ± 5	89 ± 6	$p < 0.05$
BFP	33.7 ± 11.6	35.3 ± 10.1	39.0 ± 10.8	$p < 0.05$
Triglyceride (mg/dL)	152 ± 102	156 ± 110	159 ± 114	$p < 0.05$
CRP (mg/L)	3.1 ± 1.8	3.9 ± 2.0	5.9 ± 3.1	$p < 0.05$
RRI	0.66 ± 0.1	0.67 ± 0.1	0.69 ± 0.2	$p < 0.05$

BMI: Body mass index, WHR: waist-to-hip ratio, BFP: body fat percentage, CRP: C-reactive protein, RRI: Doppler-derived renal resistive index

The study patients were divided into three Groups according to their BFP defined by BIA (Table 3). Group 1 (BFP: < 26%, n = 30) comprised the patients in the lowest tertile of low BFP; Group 2 (BFP = 26% to 48%, n = 60), patients in the middle tertile; and Group 3 (BFP > 48%, n = 30), patients in the upper tertile. Group 3 patients, who exhibited ≥ 48% of body fat, had significantly higher BMI ($p < 0.05$), total leukocyte count ($p < 0.05$), CRP ($p < 0.05$), triglyceride ($p < 0.05$) and female predominance. Group 3 patients were statistically older than Group 1 patients (46.2 *vs* 40.6 years, $p < 0.05$). Additionally, RRI levels were higher in Group 3 than Group 1 [0.69 *vs* 0.65, $p < 0.05$] (Table 3).

In logistic regression analysis, independent factors affecting RRI were: age, gender, BFP and CRP levels (all *p*-values were < 0.05).

Table 3: Comparison of clinical and laboratory values of patients according to body fat percentage tertiles

Parameters	Group 1 BFP ≤ 26 (n = 30)	Group 2 BFP = 26-48 (n = 60)	Group 3 BFP ≥ 48 (n = 30)	*p*
Age	42.6 ± 4.2	44.4 ± 5.1	46.2 ± 3.1	$p < 0.05$
BMI	25.9 ± 2.1	27.1 ± 1.2	29.1 ± 1.6	$p < 0.05$
Triglyceride (mg/dL)	156 ± 101	157 ± 100	159 ± 112	$p < 0.05$
Leukocyte (per μL)	5.7 ± 2.1	5.9 ± 1.9	6.0 ± 2.0	$p < 0.05$
CRP	3.2 ± 1.7	4.2 ± 2.0	6.7 ± 2.1	$p < 0.05$
RRI	0.65 ± 0.1	0.66 ± 0.1	0.69 ± 0.2	$p < 0.05$

BMI: Body Mass Index, WHR: waist-to-hip ratio, BFP: Body fat percentage, CRP: C-reactive protein, RRI: Doppler-derived renal resistive index.

DISCUSSION

In our study, we investigated the influence of body fat composition on CV risk parameters in a group of hypertensive patients. Body fat distribution is associated with increased long-term mortality and morbidity risk, as shown in several recent studies (12, 13). In recent years, investigators tried to find clinical tools for body composition analysis in all patients. Bioelectrical impedance is an adequate and easily accessible clinical tool for monitoring nutritional status in patients. We observed a direct correlation between bioimpedance measures and the traditional anthropometric and laboratory markers of cardiovascular risks in newly diagnosed hypertension patients.

Body fat percentage and BMI were significantly higher for females than males in our study (Table 1). The most important and drastic gender differences in BFP are related to reproductive functions that change with age (14). In distinction to the course of reproductive functions in females, the rapid decline in gender hormones is expressed by the cessation of menses in most women, whereas men experience hormonal changes in a slow and continuous manner of decline. This decline in endocrine functions in female patients is directly or indirectly associated with changes in fat distribution (14). This hormonally driven shift in body fat distribution from peripheral to abdominal in females may increase their RRI parameters (14).

We found that higher BMI and BFP were associated with higher RRI which was a parameter of unfavourable renal haemodynamic profile, in study populations of non-diabetic patients. These data are in line with epidemiologic studies showing that central body fat distribution is an independent risk factor for renal haemodynamic factors could be involved in increased renal and cardiovascular disease. High BMI likely reflects high BFP rather than high muscle mass in the female patients, whereas men exhibited both increase in muscle mass and fat percentages in parallel to increase BMI. Decreased muscle mass has been associated with arterial stiffness documented by RRI in female patients with female hypertension (15). Skeletal muscle is the main site for in-

sulin mediated glucose disposal, some investigators hypothesized that low muscle mass may be associated with insulin resistance and cardiovascular risks (16).

The associations of BFP, WHR, triglyceride, CRP and RRI with BMI were demonstrated by the multivariate analysis. Relation between body fat distribution and CVD is in line with our prior findings in haemodialysis persons (17). Furthermore, we demonstrated that this association is present in a population of hypertensive, non-diabetic patients. Several studies have reported that WHR was associated with albuminuria and BP both irrespective of BMI (18, 19). In the current study, however, we have not found a robust association between WHR and albuminuria, possibly because of the very-low albuminuria excretion rates, which reflect the strict inclusion criteria of these newly diagnosed hypertensive patients.

Our data showed that the higher BFP group were older, had high BMI and poor inflammatory profile in terms of higher levels of CRP and leukocyte count. Body fat percentage was another factor that contributed to inflammation and vascular complications. An increase in adipose tissue results in the infiltration of macrophages and enhanced inflammation (20). Moreover, it is associated with increased proinflammatory cytokines (IL-6, TNF-a) that contribute to atherosclerosis and CVD in high-fat composition populations (20). C-reactive protein itself may play a role in promoting or propagating vascular injury through complement recruitment (21). Thus, inflammation play a role in classically initiated vascular injury, while at the same time, fat mass may independently initiate or propagate inflammation and vascular injury (22). In this study, BMI and BFP increased together and cardiovascular risks were higher in high-BMI and high-BFP groups. On the contrary, increase in muscle mass could be protective against inflammation.

Several mechanisms may play a role in the deleterious effects of central fat accumulation on atherosclerosis, such as oxidative stress and inflammation by upregulation of pro-inflammatory adipokines and cytokines (23). Furthermore, central fat is associated with dyslipidaemia and inflammatory parameters most likely in mutual interaction (24). The combination higher WHR and BFP indicate a higher post-glomerular efferent arteriolar tone that can affect renal sodium handling by altering peritubular Starling forces, hampering sodium excretion, and hence contributing to sodium-sensitive hypertension (25). The latter is also supported by data on the renal effects of the renin-angiotensin-aldosterone system blockade in relation to BMI (25). Whether this also applies to the renal haemodynamic changes in relation to body fat distribution remains to be explored in further studies.

This study has several limitations. First, because of its cross-sectional design, this study cannot assess causality. Second, no detailed data on metabolic status (other than the exclusion of diabetes) were available, this is a relevant subject for future studies.

In conclusion, BFP was associated with higher RRI, in hypertensive persons. Of note, this association was dependent of BMI. These data suggest the possibility that an altered renal haemodynamic profile is involved in the long-term renal risk associated with body fat distribution, as seen in epidemiologic studies.

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