



Effect of Obesity on Blood Pressure and Arterial Stiffness in Middle-Aged Korean Women

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Objectives: Our study aims to provide basic scientific data on the importance of obesity management in middle-aged Korean women by analyzing its effects on blood pressure and arterial stiffness. In addition, we examined the correlations of these two parameters.

Methods: The study participants were 40 middle-aged female volunteers, who were classified into obesity group (n = 20) and normal weight group (n = 20). Statistical analysis was performed using independent *t*-test and the Pearson correlation coefficient was used to correlate blood pressure and arterial stiffness.

Results: This study evaluated the systolic blood pressure, diastolic blood pressure, and pulse wave velocity (PWV). These results were higher in the obesity group than the normal weight group. Furthermore, blood pressure and arterial stiffness (PWV, augmentation pressure) were static correlated.

Conclusion: Obesity is closely related to blood pressure and arterial stiffness. Therefore, indices for blood pressure and arterial stiffness may play a vital role in predicting and preventing obesity and its sequelae.

Key Words: arterial stiffness, blood pressure, hypertension, middle aged women, obesity, pulse wave velocity

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INTRODUCTION

Obesity is a serious global health problem, and increases the risk of premature death. There has been a rapid increase in the obese population to >1.5 billion people, representing 20% of the total world population [1].

In addition, the obese population in Korea is increasing; it has increased from 31.8% men and 27.4% women in 2001 to 36.3% men and 28.0% women in 2012. It is estimated that > 30% of the adult population in Korea is overweight or obese. In particular, obesity in the female population rapidly increases after middle age, around the time of menopause, with 17.9% of 30–39-year-old women, 35.7% of 40–49-year-old women, 33.7% of 50–59-year-old women, and 42.7% of 60–69-year-old women considered obese [2].

Various explanations have been provided for the increased prevalence of obesity in middle-aged women, among which the major causes are reduced basal metabolic rate due to lack of physical activity, decrease in the growth hormone and estrogen, and overeating [3].

Lack of physical activity is reported to be associated with hypertension, and body mass index is observed to be high in pre-hypertensive and hypertensive groups, demonstrating a significant



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correlation between body weight and blood pressure [4,5].

The incidence of hypertension in Korean women older than 30 years has increased from 21.8% in 2007 to 25.4% in 2012, indicating a continual increase in the incidence of both hypertension and obesity in Korean women [2].

Furthermore, obesity is associated with an increase in arterial stiffness, due to negative changes in the vascular structure. Obesity reduces vascular elasticity due to an increase in intravascular inflammation and changes in endothelial function, with an increase in the arterial intima-media thickness and a decrease in the arterial lumen diameter [6–8].

Arterial stiffness raises the systolic blood pressure (SBP) while lowering the diastolic blood pressure (DBP); the increased pulse pressure increases the load on the left ventricle, resulting in a higher risk of myocardial infarction and other coronary heart diseases (CHDs) [9].

Thus, the level of arterial stiffness is a useful index for the prediction of CHDs, since increased ventricular load and decreased diastolic function impair coronary blood supply [9].

Therefore, obesity is a risk factor for hypertension and arterial stiffness. Our study aims to provide basic scientific data on the importance of obesity management in middle-aged Korean women by analyzing its effects on blood pressure and arterial stiffness; in addition, we examine the correlations of these two parameters.

MATERIALS AND METHODS

1. Subjects

Our study subjects were randomly selected 40 middle-aged Korean women (aged 40–50 years) who were recruited from Busan, Korea. They were divided into two groups: the obesity group (OG, $n = 20$; $33.98 \pm 2.81\%$ body fat) and the normal weight group (NG, $n = 20$; $26.02 \pm 4.01\%$ body fat).

All participants were asymptomatic of illness or disease and free from acute or chronic injury, as confirmed by the American College of Sports Medicine participant activity readiness Par-Q health questionnaire. All participants provided written informed consent approved by the Institutional Human Research Committee of Pusan National University (PNU IRB/2016 99). A complete description of demographic characteristics of the study participants are presented in Table 1.

2. Body composition

Height was measured to the nearest 0.1 cm without shoes. Body weight was measured to the nearest 0.1 kg, with the participant wearing light clothes. Body fat (%) was determined using

a bioelectrical impedance analysis method (X-SCAN PLUS II; JAWON Medical, Seoul, Korea).

3. Pulse wave velocity (PWV)

A commercially available applanation tonometer (SphygmoCor; AtCor Medical Ltd., Sydney, Australia) was used along with the analysis software (version 8.0, SphygmoCor Cardiovascular Management Suite) for the measurement of PWV (m/s), an indicator of arterial stiffness, as previously described [10].

4. Augmentation pressure (AP), augmentation index (AIx)

AP and AIx are predictors of major adverse cardiovascular outcomes. Hazard ratios and 95% confidence intervals were calculated for each 10-mmHg increase in AP (adjusted for pulse pressure) or 10% increase in AIx. Both AP and AIx were adjusted for mean aortic pressure, heart rate, and ejection fraction in all models. The central pressure waveform was measured using applanation tonometry. The height of the late systolic peak pressure above the inflection defines the augmented pressure. AIx was defined as the ratio between the local carotid PP and the AP of the reflected wave, and was expressed as a percentage. Two measurements with 10-s intervals were averaged [11].

5. Data analysis

Data are presented as mean \pm standard deviation. Statistical analyses were performed using independent *t*-test, using the SPSS Statistics software, ver. 20.0 (IBM Co., Armonk, NY, USA), and the Pearson correlation was used to correlate blood pressure and arterial stiffness. All statistical tests used an alpha level set at $p < 0.05$.

A repeated test number (sample size) of 40 was derived from a priori sample size estimation using a commercial software (G*Power, version 3.0.1; Universitat, Kiel, Germany), based on repeated measures ANOVA F-statistic at $p < 0.05$, $\beta = 0.2$, and an effect size of 0.5.

Table 1. Descriptive characteristics of study participants

Variable	Group	
	NG (n = 20)	OG (n = 20)
Age (y)	54.65 \pm 3.70	53.25 \pm 4.09
Weight (kg)	61.84 \pm 7.76	55.02 \pm 4.80
Height (cm)	158.70 \pm 5.11	159.45 \pm 5.24
BMI (kg/m ²)	24.38 \pm 1.79	21.64 \pm 1.55
% Body fat (%)	26.02 \pm 4.01	33.98 \pm 2.81

Values are presented as mean \pm standard deviation. NG, normal weight group; OG, obesity group; BMI, body mass index.

RESULTS

1. Blood pressure

Table 2 summarizes the differences between blood pressures of two groups. We observed that differences between SBP (NG vs. OG: 122.25 ± 8.62 mmHg vs. 135.80 ± 11.91 mmHg) and DBP (77.35 ± 7.34 mmHg vs. 84.70 ± 7.44 mmHg) were significantly higher in OG compared to those in the NG ($p < 0.01$, $p < 0.05$).

2. PWV

The difference in PWV was higher in the OG than in the NG (NG vs. OG: 6.92 ± 0.90 m/s vs. 7.74 ± 0.94 m/s, $p < 0.05$) (Table 2).

3. AP, AIx

Table 2 summarized the differences in AP and AIx. AP (NG vs. OG: 10.60 ± 5.01 mmHg vs. 13.35 ± 4.39 mmHg) and AIx ($30.10 \pm 11.69\%$ vs. $13.35 \pm 4.39\%$) did not demonstrate statistically significant differences between the OG and NG.

4. Correlation between blood pressure and arterial stiffness

Table 3 presents the correlation between blood pressure and arterial stiffness. We observed that SBP was static associated with PWV ($r = 0.554$, $p < 0.001$) and AP ($r = 0.492$, $p < 0.01$). DBP was static associated with PWV ($r = 0.379$, $p < 0.05$).

DISCUSSION

With its increasing prevalence worldwide, obesity is rec-

ognized as a major disease in the modern society, and there is ongoing research on its elimination. While obesity is known as a major risk factor for cardiovascular disease, severe obesity is associated with a greater risk of CHDs, and persistent efforts are being made to prevent and anticipate CHDs [12,13]. Moreover, measures are being taken to predict CHDs by measuring blood pressure and blood glucose, as well as screening for abnormal vascular structure [14].

One important factor in the prevention of CHDs is the correction of structural and functional changes in blood vessels, and this can be achieved by lowering blood pressure and reducing vascular stiffness [7,14]. Abnormal changes in blood pressure cause CHDs via atherosclerosis, resulting in decreased vascular elasticity and increased peripheral resistance [13]. Arterial stiffness is known to increase the risk of CHDs, as it elevates central blood pressure and causes left ventricular hypertrophy and reduced coronary artery perfusion [9].

Middle age causes the onset of the gradual decline of physical development; physical stamina noticeably declines in the late 40s, and women become vulnerable to obesity, hypertension, and cardiac disease [15]. The body secretes less estrogen in preparation for menopause, which promotes fat accumulation leading to obesity [16]. Lack of physical activity and unhealthy dietary habits lead to dyslipidemia and increase the risk of coronary artery disease [17].

Our study demonstrated higher SBP and DBP in obese, middle-aged women than in normal weight, middle-aged women; in addition, the OG had a higher PWV. These results are in accordance with those of preceding studies, which demonstrate that increase in body fat percentage and triglycerides in obesity leads to an elevated blood lipid concentration, which affects vascular stiffness, and in turn causes an increase in blood flow velocity [18].

We observed a static correlation between SBP and DBP and arterial stiffness.

Increased arterial stiffness causes elevated pulse pressure, increasing the afterload on the heart, and reduces coronary artery

Table 2. Difference between blood pressure and arterial stiffness in each group

Variable	Group		t-value
	NG (n = 20)	OG (n = 20)	
SBP (mmHg)	122.25 ± 8.62	135.80 ± 11.91	-3.946**
DBP (mmHg)	77.35 ± 7.34	84.70 ± 7.44	-2.461*
PWV (m/s)	6.92 ± 0.90	7.74 ± 0.94	-2.858*
AP (mmHg)	10.60 ± 5.01	13.35 ± 4.39	-1.386
AIx (%)	30.10 ± 11.69	13.35 ± 4.39	-0.381

Values are presented as mean \pm standard deviation.

NG, normal weight group; OG, obesity group; SBP, systolic blood pressure; DBP, diastolic blood pressure; PWV, pulse wave velocity; AP, augmentation pressure; AIx, augmentation index.

* $p < 0.05$, ** $p < 0.01$.

Table 3. Correlation of blood pressure and arterial stiffness

Variable	Group	
	SBP	DBP
PWV	0.554***	0.379*
AP	0.492**	0.273
AIx	0.187	0.239

SBP, systolic blood pressure; DBP, diastolic blood pressure; PWV, pulse wave velocity; AP, augmentation pressure; AIx, augmentation index.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

perfusion; furthermore, it increases the risk of CHDs in females than in males [8]. Although our study does not clarify the exact mechanisms of the correlation between blood pressure and arterial stiffness, it demonstrates an increase in blood pressure due to obesity and a static correlation between blood pressure and arterial stiffness, with reduced coronary artery perfusion. This suggests that the most important factors for the prevention of obesity-related diseases are lifestyle changes, such as regular exercise, which can reduce cardiac disease risk, and proper dietary habits, which can prevent dyslipidemia and alleviate hyperlipidemia.

In conclusion, obesity is closely related to blood pressure and arterial stiffness. Therefore, indices for blood pressure and arterial stiffness may play a vital role in predicting and preventing

obesity and its sequelae.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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