ARAŞTIRMA/ORIGINAL ARTICLE

Gülhane Tıp Derg 2015;57: 26 - 30

doi: 10.5455/gulhane. 163839

Investigation of the aortic pulse wave velocity in hypertensive and normotensive obese subjects

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ÖZET

Hipertansif ve normotansif obez olgularda aortik nabız dalga hızının incelenmesi

Arteryel sertliğin halihazırda "altın standart" ölçümü aortik (karotis-femoral) nabız dalga hızıdır.Obezite genellikle hipertansiyon ve hiperglisemi gibi kardiyovasküler risk faktörleri ile birliktelik gösteren bir hastalıktır. Biz hipertansif ve normotansif obez olgularda, Aortik nabız dalga hızı ve vücut kitle indeksi arasındaki ilişkiyi araştırmayı amaçladık.Kan basıncı ve vücut kitle indeksine göre iki grup olmak üzere toplam 109 olgu alındı. Vücut kitle indeksine göre olgular 30.0-34.9 kg/m2 olanlar Clas I, ≥35 kg/m2 olanlar Clas II olarak tanımlandı. Sonrasında Clas I ve II grupları kan basıncına göre iki gruba normotansif-Clas I, II ve hipertansif-Clas I, II olarak ayrıldı. Tüm grupta (n:109) nabız dalga hızı, yaş, sistolik kan basıncı ve diyastolik kan basıncı ile korelasyon göstermekteydi (sırasıyla, r=0.229; p=0.017, r=0.301; p=0.001 ve r=0.323; p=0.001). Nabız dalga hızının Clas I ve Clas II grupta vücut kitle indeksi ile korelasyonu yoktu. Bu verilere göre, vücut kitle indeksi 30 kg/m2 üzerindeki olgularda nabız dalga hızının artması vücut kitle indeksi ile ilişkili değildir, fakat yaş, sistolik ve diyastolik kan basıncı ile ilişkili olabilir.

Anahtar Kelimeler: *Nabız dalga hızı, Obezite, Vücut kitle indeksi, Hipertansiyon*

SUMMARY

Aortic pulse wave velocity is currently the'gold standard'measure of arterial stiffness. In this study we aimed to investigatethe relationships betweenAortic pulse wave velocity and body mass index in hypertensive andnormotensiveobesesubjects.A total of109 obese subjects were divided into two groups according to arterial blood pressures andbody mass index. Subjects with a body mass index of 30.0-34.9 kg/m2 were defined as Class I, and ≥35.0 kg/m2 as Class II. Then Class I and II groups were divided into two groups according to blood pressures as normotensive-Class I, II and hypertensive-Class I, II.In the whole group (n:109) pulse wave velocity was significantly correlated with age, systolic blood pressure and diastolic blood pressure (r=0.229; p=0.017, r=0.301; p=0.001 and r=0.323; p=0.001, respectively). Pulse wave velocity was not significantly correlated with body mass index neither in the whole group nor in the Class I and Class II groups.These results suggest that in the patients with a body mass index of ≥30.0 kg/m2, increase ofpulse wave velocity is not associated with body mass index, but can be related with age, systolic and diastolic blood pressure.

Key words: *Pulse wave velocity, Obesity, Body mass index, Hypertension*

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Makalenin Geliş Tarihi: Nov 13, 2014 • **Kabul Tarihi:** Jan 07, 2015 • **Çevrim İçi Basım Tarihi:** 15 Şubat 2015

Introduction

Obesity, which is an important health problemin all over the world, is often associated with cardiovascular risk factors, such as hypertension, hyperglycemia and dyslipidemia, and poses a greater risk for cardiovascular events(1,2).Data from the Framingham Heart Study have implicated obesity as a contributory factor in 60-70% of essential hypertensive cases(3), and obese individuals have a 3.5-fold increase in thelikelihood of suffering from hypertension (4).

Aortic pulse wave velocity (PWV) is currently the 'gold standard' measure of arterial stiffness, which is an important independent predictor of risk to developing a cardiovascular event (5). This concept defines the viscoelastic properties of the vessel wall. Increased arterial stiffness is associated with hypertension, age, diabetes, coronary artery disease, chronic kidney disease, tobacco use, dyslipidemia, cerebrovascular disease, peripheral arterial disease, etc. (6,7).In this study we aimed to investigate the relationship between PWV and body mass index (BMI) in hypertensive (HT) and normotensive (NT) obese subjects.

Materials And Methods

Our retrospective cross-sectional study was carried out on a total of 109 cases which includes 57 hypertensive-obese and 52 normotensive-obese patients, between January 2011-June 2011 in Department of Internal Medicine. Patients with obesity and hypertension undergoing arterial stiffness measurement between18-65 years old were included into the study.

Height (cm) and weight (kg) of cases were recorded, body mass index was calculated (kg/m2) as recommended by the World Health Organization, and obesity was diagnosed in those over 30 kg/m2(3). Patients with a systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg defined as HT, with a SBP <140 mmHg and DBP <90 mmHg defined as NT.Patients with blood pressure measurements, provided that after 15 minutes of rest, without smoking or caffeinated beverages and repeated measurements of 140/90 mmHg or above or receiving antihypertensive drugs were considered to be hypertensive. Obese subjects were divided into two groups according to the arterial blood pressures and BMI. Subjects with a BMI of 30.0-34.9 kg/m2 were defined as Class I, and ≥35.0 kg/m2 as Class II. Four subjects' BMI were above ≥40.0 kg/m2 in the Class II group. Then Class I and Class II groups were divided into two groups according to the blood pressures as NT-Class I, NT-Class II and HT-Class I, HT-Class II.

Blood samples were taken in the morning after a fasting period of 12 hours for biochemical analyses.The complete

Table 1. Comparison of blood pressure and arterial stiffness measurements, demographic, and laboratory data of hypertensive obese and normotensive obese groups

	Hypertensive obese	Normotensive obese	P value
Sex (n, F/M)	57 (35/22)	52 (28/24)	.425*
Age (years)	50.7±12.8	48.7±12.9	.60
BMI (kg/m2)	34.0±2.8	33.0±2.9	.13
Systolic BP (mm Hg)	153.7±13.8	126.3±8.5	<001
Diastolic BP (mm Hg)	88.7±9.3	76.9±6.7	<001
Heart Rate (beats/minute)	81.5±11.9	73.8±10.9	.001
Urea (mg/dL)	28.0±6.3	29.0±10.8	.56
Creatinine (mg/dL)	0.85±0.12	0.92±0.15	.02
HDL (mg/dL)	47.0±13.2	48.7±11.4	.50
LDL (mg/dL)	132,5±31.0	127.7±37.8	.48
Triglyceride (mg/dL)	179.4±74.8	143.9±66.2	.01
Hemoglobin (g/dL)	13.9±1.4	13.8±1.3	.79
Thrombocyte (103/mm3)	276±76	261±70	.28
WBC (/mm3)	6.8±1.5	6.8±1.8	1.0
PWV (m/s)	10.0±2.0	8.5±1.9	<001

Abbreviations: BMI ; bodymass index, BP– blood pressure; HDL– high-density lipoprotein cholesterol; LDL – low-density lipoprotein cholesterol; WBC – white blood cell; PWV – pulse wave velocity;
*Chi-square test, otherwise independent samples t test. Values are given as mean ± standard deviation.

blood counts, blood chemistry including serum lipid parameters, blood pressure measurements, BMI and arterial stiffness measurements of all patients were recorded.Patients with an additional disease (diabetes mellitus, chronic renal failure, cerebrovascular disease, malignancy, etc.)except for obesity and hypertension were not included in the study. The study was approved by the local ethics committee.

Measurement of Arterial Stiffness

TensioMed measurement of arterial stiffness (TensioMed Ltd., Budapest, Hungary) device was used. Systolic and diastolic blood pressures, PWV measurements were recorded. TensioMed brand arteriography device automatically calculated the measurements. Measurements were made after 5-10 minutes rest of the patients, non-smoking or taking caffeinated beverages for at least the last 30 minutes, in a reserved quiet room with a stable room temperature, in the supine position, and away from external stimulus. The distance between the jugular notch and the symphysis pubis was measured in a straight line carefully while the patients were layingand the data were recorded on the device. During the measurement period, brachial artery occlusion was made (only 8-20 sec.) and the blood flow was ceased as a part of the process (5,8,9). Then the systolic blood pressure, diastolic blood pressure, mean arterial blood pressure, pulse rate, pulse pressure, PWV were derived from the device. The pressure waves were recorded along with the PWV values.

Statistical Analysis

For statistical analysis, SPSS (Statistical Package for the Social Sciences ver. 15.0, SPSS Inc, Chicago, IL, USA)computer program was used. Quantitative variables were expressed as mean ± standard deviation. In comparison of the groups, Student t test was used. Categorical variables were compared by chi-square test. Pearson correlation analysis was used to evaluate the relationship between variables. Regression analysis was used to determine the relationships between variables and the PWV. P <0.05 was considered as statistically significant.

Results

The mean ages of the HT obese and NT obese groups were 50.7±12.8 and 48.7±12.9 years old, respectively. Baseline demographic and laboratory data for patients are presented in Table 1. PWV, heart rate (HR), SBP and DBP measurements were significantly different between the two groups, as expected (10.0±2.0, 8.5±1.9 m/s; p<0.001, 81.5±11.9, 73.8±10.9 b/m; p=0.001, 153.7±13.8, 126.3±8.5 mmHg; p<0.001 and 88.7±9.3, 76.9±6.7 mmHg; p<0.001, respectively). PWV was significantly correlated with SBP, DBP and age (r=0.301; p=0.001, r=0.323; p=0.001 and r=0.229; p=0.017, respectively), but had no significant correlation with BMI (r=0.004; p=0.964)in the whole group(Table 2).

Table 2. Correlations between age, systolic and diastolic blood pressure with pulse wave velocity

	Systolic blood pressure	Diastolic blood pressure	Age	BMI
Puls wave velocity (m/s)	r=0.301 p=0.001	r=0.323 p=0.001	r=0.229 p=0.017	r=0.004 p=0.964

Table 3. Comparison of the whole group according to the body mass index

	Class I (n=79)	Class II (n=30)	P value*
Age (years)	51.1±12.4	45.2±13.4	0.043
SBP (mmHg)	139.2±18.8	144.6±15.3	0.058
DBP (mmHg)	84.0±11.1	80.9±6.2	0.197
HR (beats/minute)	76.8±12.0	80.6±12.2	0.139
BMI (kg/m2)	32.1±1.5	37.4±2.3	<0.001
PWV (m/s)	9.3±2.2	9.2±2.0	0.828

Abrevations:Class I; 30.0-34.9 kg/m2, Class II; ≥35.0 kg/m2 , SBP; Systolic blood pressure, DBP; Diastolic blood pressure, HR; Heart rate, BMI; body mass index, PWV; Pulse wave velocity.

*Independent samples t test. Values are given as mean ± standard deviation.

A linear regression analysis was performed using PWV as dependent variable and age, SBP and DBP as predictors.The final equation of the estimated PWV was formulated below.

PWVestim=1.881 + (0.029*Age + 0.02*SBP + 0.038*DBP) (F=6.108; p=0.001)

In the Class I group PWV had a significant correlation with SBP and DBP (r=0.298; p=0.008 and r=0.366; p=0.001, respectively), but had no significant correlation with BMI (p=0.301). PWV had not significantly correlated with age, SBP, DBP, HR and BMI in the Class II group.

When the groups were compared according to the category of BMI, age was higher in the Class I group (p=0.043) than the Class II group. Between the two groups PWV was not significantly different (p=0.828) (Table 3).

PWV showed a significant correlation with DBP in the NT-Class I group (r=0.325; p=0.036), although there was not a significant correlation with age, SBP, HR and BMI. There was a significant correlation between PWV and age in the NT-Class II group (r=0.743; p=0.014).

In the hypertensive groups PWV had no significant correlations with age, SBP, DBP, HR and BMI.

When the groups were compared according to the category of BMI, age and DBP were higher in the HT-Class I group (p=0.010 and p<0.001, respectively) than the HT-Class II group. PWV was not significantly different in these groups (p=0.871 and p=0.553, respectively) (Table 4).

Discussion

We found that, in obese population age and blood pressure had effects on arterial stiffness, but BMI did not have such an effect.

Endothelial dysfunction is one of the basic mechanisms of the atherosclerotic process. Increased aortic stiffness or reduced compliance is commonly indicative of atherosclerotic involvement of the vascular system. As a non-invasive method of measurement of arterial stiffness; it has a decisive importance for cardiovascular diseases, especially for atherosclerosis and related diseases (10).Arterial stiffness is a well recognized predictor of cardiovascular morbidity and mortality(11-14). Arterial stiffness and its consequences represent a great challenge for the twenty-first century affluent countries, and ''de-stiffening'' will be the goal of the next decades (15).

Several studies have shown a close correlation between PWV and obesity (16). Urbina et al. demonstrated increased arterial stiffness in young obese participants (17).A studyofobese childrenin Turkey reported increased PWV(18), but we could not reacha similar studyin adults. Obesity is associated with arterial stiffness and increased PWV independently from age, arterial blood pressure, and ethnicity. However, the association is stronger for waist circumference and visceral adiposity, rather than global obesity measured by BMI (19,20).

In the Complior study by Asmar Rand colleagues, age and SBP were shown to be major factors in determining the PWV (21).In Turkey,a study on overweight HT and obese HT patients (with no-significant difference between the systolic and diastolicblood pressures,p=0.26), there was no significant difference among the PWV measurements (22).Although BMI's were different, PWV measurements were similar in similar blood pressures.The lack of correlation between BMI and PWV in our study is similar with the results of these studies. Thus, it can be concluded that BMI did not influence the PWV or affect it minimally in subjects with a BMI of ≥30 kg/m2.

Impaired arterial elasticity in young patients with white-coat hypertension has also been shown by Longo and his colleagues (23). In HARVEST study it is shown that arterial distensibility may already be impaired in the early stage of hypertension. The hypertensive participants also showed a higher PWV (24). PWV was increased in obese hypertensive patients in our study, and had a significant correlation with systolic and diastolic blood pressures. Thus, with the addition of a hypertensive complication on obesity, we can say that cardiovascular morbidity, mortality, and atherosclerotic process are further accelerated. Gardner and Parker observed a progressive increase in compliance with aging in both large and small arteries (25). In Europe, PWV determined normal and reference values and its was showed thatincreases as an associated with age and blood pressure(9).In our study, we found a positive correlation between age and PWV similar withthese studies. Measures of arterial stiffness also showed a progressive increase with age.

Sympathetic nervous system activation such as congestive heart failure, renal failure, andobstructive sleep apnea are

Table 4. Comparison of the normotensive and hypertensive groups according to the body mass index

	NT- Class I (n=42)	NT-Class II (n=10)	P value*
Age (years)	48.6±13.7	49.6±9.6	0.981
SBP (mmHg)	126.2±8.8	127.3±7.8	0.745
DBP (mmHg)	76.7±7.0	77.9±5.3	0.592
HR (beats/minute)	73.6±10.7	74.8±12.6	0.748
BMI (kg/m2)	31.9±1.5	**38.1±2.1**	**<0.001**
PWV (m/s)	**8.6±2.0**	**8.3±1.8**	**0.871**
	HT-Class I (n=37)	**HT-Class II (n=20)**	**P value***
Age (years)	**53.9±10.0**	**43.1±14.7**	**0.010**
SBP (mmHg)	**154.1±15.8**	**153.3±9.5**	**0.732**
DBP (mmHg)	**92.2±9.0**	**82.4±6.1**	**<0.001**
HR (beats/minute)	**80.5±12.3**	**83.6±11.2**	**0.370**
BMI (kg/m2)	**32.3±1.4**	**37.0±2.3**	**<0.001**
PWV (m/s)	**10.2±2.1**	**9.7±2.1**	**0.553**

associated with arterial stiffness. Furthermore, cigarette smoking is associated with impairment of large artery function and increased aortic stiffness (26). There is a growing evidence that tachycardia -reliable marker of high sympathetic tone and cadiovascular risk (27)- is an important determinant of arterial function and PWV (28). Sympathetic overactivity might contribute to development and progression of hypertension and its complications (15). In our study,we found that obese hypertensive patients had significantly increased pulse rates. Our results showed that obese hypertensive patients had increased sympathetic activation and this supported the contribution to the in crease in cardiovascular risks.

Several mechanisms are included in the pathophysiology of obesity associated hypertension (29). The pathophysiology of obesity-related hypertension is complex and involves the renin-angiotensin-aldosterone system, sympathetic nervous system, oxidative stress, endothelial dysfunction and adipokine dysregulation. Perivascular adipose tissue damage in obesity may be a contributing factor to the development of hypertension in obesity, and rescuing its function may offer therapeutic potential (30-33).

Limitations

Our study has some limitations. Firstly, if available, waist circumference(WC) of the patients data could reveal the difference between WC and BMI and could strengthen the work. Secondly, if the overweight group as a control group and cigarette smoking history were existed it would be better., Thirdly, this is a small retrospective study to draw definitive conclusions. Although our small scale confers some clues for this approach, it is obvious that large scale and more detailed investigations are needed.Finally, a prospectively designed study of leptin, insulin, aldosterone, oxidative stress, and inflammatory cytokines could explain the contribution of obesity-related hypertension.

Conclusion

As a result;in patients with a BMI of ≥30.0 kg/m2, increase of pulse wave velocity is not associated with BMI, but can be related with age, SBP and DBP.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

1. Wormser D, Kaptoge S, Di Angelantonio E, et al. Separate and combined associations of body-mass index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58 prospective studies. Lancet. 2011 Mar 26;377(9771):1085–95.
2. WHO. Obesity and overweight. [Internet]. 2013 [cited 2013 Aug 6]. Available from: http://www.who.int/mediacentre/factsheets/fs311/en.
3. Henry SL, Barzel B, Wood-Bradley RJ, Burke SL, Head GA, Armitage JA. Developmental origins of obesity-related hypertension. Clinical and experimental pharmacology & physiology. 2012 Sep;39(9):799–806.
4. Kotchen TA. Obesity-related hypertension: epidemiology, pathophysiology, and clinical management. American journal of hypertension. 2010 Nov;23(11):1170–1178.
5. Laurent S, Cockcroft J, Van Bortel L, et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications.Eur Heart J. 2006 Nov;27(21):2588-605.
6. Kostis JB, Lawrence-Nelson J, Ranjan R, Wilson AC, Kostis WJ, Lacy CR. Association of increased pulse pressure with the development of heart failure in SHEP. Systolic Hypertension in the Elderly (SHEP) Cooperative Research Group. American journal of hypertension. 2001 Aug;14(8 Pt 1):798–803.
7. Heitzer T, Schlinzig T, Krohn K, Meinertz T, Münzel T. Endothelial dysfunction, oxidative stress, and risk of cardiovascular events in patients with coronary artery disease. Circulation. 2001 Nov 27;104(22):2673–8.

8. Van Bortel LM, Laurent S, Boutouyrie P, et al. Expert consensus document on the measurement of aortic stiffness in daily practice using carotid-femoral pulse wave velocity.J Hypertens. 2012; 30:445–448.
9. Mattace-Raso F, Hofman A, Verwoert GC, et al. Determinants of pulse wave velocity in healthy people and in the presence of cardiovascular risk factors: 'establishing normal and reference values'. Eur Heart J. 2010 Oct;31(19):2338-50.
10. Qureshi G, Brown R, Salciccioli L, et al. Relationship between aortic atherosclerosis and non-invasive measures of arterial stiffness. Atherosclerosis. 2007 Dec;195(2):e190–194.
11. Inoue N, Maeda R, Kawakami H, et al. Aortic pulse wave velocity predicts cardiovascular mortality in middle-aged and elderly Japanese men. Circ J. 2009 Mar;73(3):549–553.
12. Shokawa T, Imazu M, Yamamoto H, et al. Pulse wave velocity predicts cardiovascular mortality: findings from the Hawaii-Los Angeles-Hiroshima study. Circ J. 2005 Mar;69(3):259–264.
13. Laurent S, Boutouyrie P, Asmar R, et al. Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. Hypertension. 2001 May;37(5):1236–1241.
14. Mitchell GF, Hwang S-J, Vasan RS, et al. Arterial stiffness and cardiovascular events: the Framingham Heart Study. Circulation. 2010 Feb 2;121(4):505–511.
15. Palatini P, Casiglia E, Gąsowski J, et al. Arterial stiffness, central hemodynamics, and cardiovascular risk in hypertension. Vasc Health Risk Manag. 2011 Jan;7:725–739.
16. Zebekakis PE, Nawrot T, Thijs L, et al. Obesity is associated with increased arterial stiffness from adolescence until old age. J Hypertens. 2005 Oct;23(10):1839–1846.
17. Urbina EM, Kimball TR, Khoury PR, Daniels SR, Dolan LM. Increased arterial stiffness is found in adolescents with obesity or obesity-related type 2 diabetes mellitus. J Hypertens. 2010 Aug;28(8):1692–1698.
18. Celik A, Ozçetin M, Yerli Y, et al. Increased aortic pulse wave velocity in obese children. Turk Kardiyol Dern Ars. 2011 Oct;39(7):557–562.
19. Sutton-Tyrrell K, Newman A, Simonsick EM, et al. Aortic stiffness is associated with visceral adiposity in older adults enrolled in the study of health, aging, and body composition. Hypertension. 2001 Sep;38(3):429–33.
20. Safar ME, Czernichow S, Blacher J. Obesity, arterial stiffness, and cardiovascular risk. J Am Soc Nephrol. 2006 Apr;17(4 Suppl 2):S109–111.
21. Asmar R, Topouchian J, Potocka AC, Pannier B, Rudnichi A, Safar M. [Evaluation of the effect of an antihypertensive treatment on arterial distensibility in essential arterial hypertension. The Complior study. A preliminary analysis of data at inclusion]. Arch Mal Coeur Vaiss. 1999 Aug;92(8):949–955.
22. Yıldız M, İnnice M, Aykut P, Gül Ç, Vardar S, Kürüm T. Assessment of Carotid-Femoral Pulse Wave Velocity in Patients with Hypertension and Different Levels of Body-Mass Index. Medical Network Kardiyoloji. 2004;11(2):105–9.
23. Longo D, Zaetta V, Perkovic D, et al. Impaired arterial elasticity in young patients with white-coat hypertension. Blood Press Monit. 2006 Oct;11(5):243–249.
24. Saladini F, Santonastaso M, Mos L, et al. Isolated systolic hypertension of young-to-middle-age individuals implies a relatively low risk of developing hypertension needing treatment when central blood pressure is low. J Hypertens. 2011 Jul;29(7):1311–1319.
25. Gardner AW, Parker DE. Association between arterial compliance and age inparticipants 9 to 77 years old. Angiology. 2010 Feb-Mar;61(1):37–41.
26. Jatoi NA, Jerrard-Dunne P, Feely J, Mahmud A. Impact of smoking and smoking cessation on arterial stiffness and aortic wave reflection in hypertension. Hypertension. 2007 May;49(5):981–5.
27. Palatini P, Benetos A, Grassi G, et al. Identification and management of the hypertensive patient with elevated heart rate: statement of a European Society of Hypertension Consensus Meeting. J Hypertens. 2006 Apr;24(4):603–610.
28. Willum-Hansen T, Staessen JA, Torp-Pedersen C, et al. Prognostic value of aortic pulse wave velocity as index of arterial stiffness in the general population. Circulation. 2006 Feb 7;113(5):664-70.
29. Weisbrod RM, Shiang T, Al Sayah L, et al. Arterial Stiffening Precedes Systolic Hypertension in Diet-Induced Obesity. Hypertension 2013 Dec;62(6):1105-1110.
30. Arslan E, Çakar M, Şarlak H, et al.Investigation of the aortic pulse wave velocity in patients with Gilbert's syndrome. Clin Exp Hypertens. 2013;35(7):512-5.
31. Selcuk A, Bulucu F, Kalafat F, et al.Skinfold thickness as a predictor of arterial stiffness: obesity and fatness linked to higher stiffness measurements in hypertensive patients. Clin Exp Hypertens. 2013;35(6):459-64.
32. Cakar M, Bulucu F, Karaman M, et al. Asymmetric dimethylarginine and augmentation index in newly diagnosed patients with hypertension.Angiology. 2015 Jan;66(1):43-8.
33. Aghamohammadzadeh R, Heagerty AM. Obesity-related hypertension: epidemiology, pathophysiology, treatments, and the contribution of perivascular adipose tissue. Ann Med. 2012 Jun;44 Suppl 1:S74–84.