Original Article

Morning blood pressure surge and dipping profile in non-hypertensive obese subjects.

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Summary:

Background: Obesity is an evolving major health problem in both developed and developing countries. Non-hypertensive obese may have an elevated Morning Blood pressure surge (MBPS), which is associated with increased risk for cardiac events (CE) independently of office and ambulatory blood pressure (BP). Non-hypertensive obese also may have a blunted nocturnal decrease in BP during the night, while healthy normotensive non-obese individuals have a 10%–20% nocturnal decrease in blood pressure (BP) during the night or dipping. Thus, 24-hour ambulatory blood pressure monitor (ABPM) is the gold standard to evaluate MBPS and dipping profile in non-hypertensive obese individuals.

Objectives: to measure and evaluate morning blood pressure surge and dipping profile for normotensive obese subjects by using 24-hour Ambulatory blood pressure monitor (ABPM).

Methods: A total of 86 asymptomatic obese individuals (54 males, 32 females) were recruited from Obesity Unit in Alkindy College of Medicine/ University of Baghdad. Ambulatory blood pressure monitoring was done in addition to 42 healthy non-obese subjects of either sex served as controls. Fasting blood glucose level was considered for all study subjects in addition to anthropometric measurements.

Results: Obese subjects had a significantly higher morning blood pressure surge (MBPS), BMI is positively correlated with MBPS with significance (P=0.0001) with Pearson's correlation coefficients (r= 0.92). Obese subjects had a significantly higher mean 24-hour, daytime and night systolic/diastolic blood pressure with P value less than 0.0001. The dipping profile of obese subjects revealed a significantly less dipping percent as compared with normal weight control subjects in both systolic and diastolic BP with p = 0.02 and 0.04 for systolic and diastolic blood pressures respectively.

Conclusion: BMI is positively correlated with MBPS. In regard to dipping profile, obese subjects had decreased or blunted nocturnal fall in blood pressure. In addition, obese subjects had increased ambulatory blood pressures (systolic, diastolic, daytime and nighttime pressures), emphasizing the strong association between obesity and pathogenesis of hypertension.

Keywords: morning blood pressure surge, dipping profile, obesity.

Introduction:

It has been clearly established that hypertension prevalence is increased in overweight and obese subjects. Mechanisms leading to increased blood pressure in obesity include sympathetic over-activity, insulin resistance and sodium retention (1) .Clues to the basic mechanisms involved in the link between obesity and hypertension first appeared in the 1940s and 1950s with the important observations by Vague. Until the 1980s there was no cogent explanation for the documented association between weight and BP. Trivial attributions, such as small cuff/large arm artifact, or excessive salt intake, have been excluded as a cause for this association. Similarly, a purely hemodynamic explanation, based on increased plasma volume and increased cardiac output, are not sufficient explanations since the latter do not account for the increase in peripheral resistance noted in obese hypertensive patients when compared with normotensive obese patients. (2)Based on observations made in his own obesity practice, Vague noted that the cardiovascular (CV) and metabolic complications of obesity were more common in patients with the upper body obesity phenotype, which he called "android," as compared with lower body obesity, which he referred to as "gynoid." These prescient observations attracted little attention until the 1980s when population-based studies in Scandinavia, using waist to hip ratio as a quantifiable surrogate for the upper body phenotype, demonstrated significant CV risk (hypertension, myocardial infarction, and type 2 diabetes mellitus) in association with a high waist to hip ratio (3). Parallel lines of research showed that insulin resistance was also associated with obesity(4), and many subsequent clinical and population-based studies showed an association of insulin levels and/or insulin resistance

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with hypertension in both obese and non-obese people (5). Thus, insulin resistance, hypertension; and obesity are tracked together in population-based and clinical studies. These observations formed the basis for understanding the pathophysiology of obesity-related hypertension (6). Pathogenetic factors are reviewed here since they provide the basis for a rational therapeutic strategy. Insulin and Sympathetic Activity; the relationship of insulin to BP, although controversial at first, has a plausible explanation, and insulin is now generally acknowledged to play a role in the pathophysiology of obesity related hypertension(7). Since insulin stimulates the sympathetic nervous system (SNS) (8), and since obese patients have increased SNS activity, (9) a role for insulin-mediated SNS stimulation seems a likely factor in the pathogenesis of high BP in obese subjects. This is supported by studies demonstrating concomitant decreases in BP and SNS activity when insulin is lowered by low energy diets in obese patients (10) Insulin also has a direct action on the kidney to stimulate sodium retention(11).Renin-Angiotensin-Aldosterone System; the renin-angiotensin-aldosterone system (RAAS) is activated in obesity, Aldosterone levels may be increased out of proportion to the increase in renin activity . Several mechanisms have been thought to underlie RAAS activation, including SNS stimulation of renin release with the generation of angiotensin II; angiotensinogen production in adipose tissue, especially intra-abdominal adipocytes with the generation of angiotensin II and aldosterone; and effects of free fatty acids, along with other poorly defined factors, on aldosterone production and release (12). Sodium Excretion, Pressure Natriuresis, and Salt Sensitivity; obesity predisposes the kidney to reabsorb sodium by neural (SNS), hormonal (aldosterone and insulin), and renovascular (angiotensin II) mechanisms (13). This enhanced sodium avidity shifts the pressure natriuresis curve to the right (14), thereby necessitating higher arterial pressure to excrete the day's salt intake and maintain sodium balance and volume homeostasis. This is the basis for the documented salt sensitivity of obesity-related hypertension (15)and underlines the need for diuretics in the therapeutic regimen.Healthy, normotensive individuals have a 10%–20% nocturnal decrease in blood pressure (BP) during the night. This decrease is under the influence of psychosocial, behavioral, and neurohumoral (sympathetic nervous system, renin–angiotensin system) factors (16). Obese may have blunted nocturnal decrease, or even increase, in blood pressure during sleep. This has been reported to increase the risk cardiovascular diseases. It is well known that there is a tendency for increased occurrence of cardiovascular events between 6 and 9 am, just after waking from sleep. (17)This is usually a result of increased morning blood pressure surge (MBPS) and 24-hour ambulatory blood pressure monitoring (ABPM) is the gold standard to evaluate these nocturnal changes and MBPS in obese individuals (18).

Patients and Methods:

A total of 86 asymptomatic obese individuals (54 males, 32 females) were recruited from Obesity Unit in Alkindy College of Medicine/ University of Baghdad, none of the them had any history of hypertension, diabetes, cardiovascular disease, or stroke and none was taking any medications for these clinical conditions. In addition, 42 healthy non-obese subjects of either sex served as controls. Subjects with an office BP measurement ≥ 140/90 mmHg were diagnosed as hypertensive according to seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure Criteria. (19). An Oscar Ambulatory Blood Pressure Monitor (SunTech Medical, Model Oscar 2B/ 222B, USA) was used for the ABPM. BP measurement were done at 30-minute periods between 9 am and 9 pm and at 30-minute periods between 9 pm and 9 am. In case of insufficient measurement or technical problems, the ABPM was repeated. The resultant ABPM reports were evaluated to define dipper versus non-dipper status (i.e., whether patients BP did or did not, respectively, decrease during the night). Individuals with a decrease in the night/sleep time mean blood pressure (systolic and/or diastolic) 10% compared with daytime mean blood pressure were considered dippers and all others were considered non-dippers (20). MBPS was calculated by subtracting the mean SBP value of the hour that the lowest SBP was measured during sleep from the second hour SBP following waking (21). Office, daytime, night, and 24-hour mean SBP and DBP values were also recorded. Anthropometric measurements included height and weight. Body mass index was calculated and recorded for each subjects.

Statistics: p value was considered to be significant at p less than 0.05, the significance of difference in the means was assessed using Student's t test (2 sample test), and Chi-Square Goodness of Fit Test. Data were analyzed using MINITAB Release 16.1 of MINITAB statistical software.

Results:

Anthropometric characteristics and ABPM data of the subjects were summarized in table 1.

Table 1: Anthropometric and Ambulatory blood pressure monitor (ABPM) data of the studied groups.

Parameter	obese	control	P value
Number of participants	86	42	
men/women (n)	(51 /34)	(24/18)	
Age, mean ± SD (years)	43.04 ± 9.07	45.19 ± 9.09	0.214
BMI, mean ± SD (Kg/m2)	33.84 ± 3.22	25.46 ± 2.80	0.0001
MBPS, mean ± SD (mmHg)	45.7 ± 10.3	30.3 ± 10.8	0.0001
Office SBP, mean ± SD (mmHg)	132.08 ± 5.65	130.24 ± 5.9	0.09
Office DBP, mean ± SD (mmHg)	81.56 ± 6.33	78.21 ± 7.5	0.01
ABPM 24-hour SBP, mean ± SD (mmHg)	134.48 ± 7.54	122.24 ± 4.8	0.0001
ABPM 24-hour DBP, mean ± SD (mmHg)	81.95 ± 6.09	76.33 ± 3.89	0.0001
ABPM daytime SBP, mean ± SD (mmHg)	135.73 ± 7.23	127.00 ± 5.12	0.0001
ABPM daytime DBP, mean ± SD (mmHg)	83.55 ± 5.45	79.81 ± 4.58	0.0001
ABPM night SBP, mean ± SD (mmHg)	131.84 ± 11.63	113.05 ± 5.73	0.0001
ABPM night DBP, mean ± SD (mmHg)	79.41 ± 9.23	69.14 ± 3.76	0.0001

BMI=body mass index; ABPM= ambulatory blood pressure monitor; SBP= systolic blood pressure; DBP= diastolic blood pressure.

There was no significant differences in the mean age between obese and control groups (p= 0.21), while they differs significantly in BMI (p= 0.0001). Mean morning blood pressure surge (MBPS) was significantly higher in obese subjects in comparison to control subjects (p= 0.0001). In addition, There was significantly higher mean office diastolic blood pressure measurements in obese subjects (p= 0.01). Whereas the difference in mean office systolic blood pressure measurements between obese and control groups was not significant (p= 0.09), though obese subjects had a higher mean office systolic measurements. The means of 24-hour, daytime and night systolic and diastolic records were significantly higher in obese subjects (p= 0.0001). Table 2 shows a positive and significant correlation between BMI and MBPS (r= 0.75, 0.92), (p= 0.0001) in obese subjects, though this correlation was not significant for control subjects (r= 0.19, 0.24), (p= 0.12, 0.21).

Table 2: Correlation between BMI and MBPS in obese and control subjects

	BMI (obese)	
p-value	r	P
MBPS	0.29	0.0001
	BMI (control)	
p-value	r	P
MBPS	0.24	0.12

Table 3 showed the dipping percent in systolic and diastolic pressure measurements between daytime and night records for both study groups,

Table 3: The dipping percent in SBP and DBP.

	obese	control	obese	control
variable	Mean SBP ± SD (mmHg)	Mean SBP ± SD (mmHg)	Mean DBP ± SD (mmHg)	Mean DBP ± SD (mmHg)
day	135.73 ± 7.23	127.00 ± 5.12	83.55 ± 5.45	79.81 ± 4.58
night	131.84 ± 11.63	113.05 ± 5.73	79.41 ± 9.23	69.14 ± 3.76
% of dipping	2.86	10.98	4.95	13.36
p-value	0.02		0.04	

there was a significantly higher dipping percent in obese subjects as compared with control subjects (p=0.02) for systolic records and (p=0.04) for diastolic records.

Discussion:

The normal rise in blood pressure (BP) in the morning has less intensely studied, despite speculation it may underlie the higher incidence of cardiac events (CE) in the morning. The morning period has been recognized as the highest risk period of the day for cardiovascular events, particularly stroke and is also associated with a rapid surge in blood pressure (22). The present data showed that obese have a significantly higher mean BP surge (MBPS) even they were normotensive, so non-hypertensive obese subjects share the same risk of developing an increase incidence of morning CE as normal weight hypertensive patients. In addition, MBPS is positively correlated with BMI in both study subjects, however, the correlation in obese subjects is significant. These findings are consistent with a study by A Amici et al, which stated that in normotensives as well as in well-controlled hypertensives, a higher MBPS is associated with CE risk independently of office and ambulatory BP, so

reduction of the MBPS could thus be a therapeutic target for preventing CE in non-hypertensive subjects (23).This study showed a slightly higher office systolic blood pressure in obese subjects than control subjects, the difference is statistically not significant. However, obese subjects had a significantly higher office diastolic blood pressure, studies have shown that office blood pressure closely correlates with BMI, results that are in accordance with our results(24) , as increased adiposity could be a key determinant for the development of prehypertension in susceptible individuals as obesity has been reported to be associated with increased sympathetic and decreased parasympathetic activity(25) .Ambulatory blood pressure records revealed a significant higher systolic and diastolic mean 24-hour BP, higher daytime, night systolic and diastolic BP in obese subjects. These findings are consistent with Kotsis et al(26), risk estimates from the Framingham Heart study suggest that nearly 78% of hypertension in men and 65% in women can be directly attributed to excess body weight(27).Obese subjects had an impaired nighttime decrease in blood pressure as they exhibited a blunted nocturnal fall in BP (non-dipping pattern). The current data have showed that obese subjects had a significantly lower dipping in BP. This findings also has been reported in previous analysis and was detected in small groups of obese adults and children (28). it has been previously demonstrated that blunted nocturnal BP fall is linked to a worse cardiovascular prognosis (29).

Conclusion:

BMI is positively correlated with MBPS. In regard to dipping profile, obese subjects had decreased or blunted nocturnal fall in blood pressure. Obese subjects had increased ambulatory blood pressures (systolic, diastolic, daytime and nighttime pressures), emphasizing the strong association between obesity and pathogenesis of hypertension.

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