Do Obese Individuals With Hypertension Have More Difficult-to-Control Blood Pressure and End Organ Damage Than Their Nonobese Counterparts?

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The authors assessed whether individuals with elevated body mass index (BMI) and hypertension had more difficult-to-control blood pressure (BP) and more evidence of end organ damage using data collected prospectively over 11 years from a secondary care hypertension clinic. A total of 1114 individuals were divided by BMI criteria into normal (n=207), overweight (n=440), and obese (n=467). Mean daytime, nighttime, and 24-hour systolic BP and diastolic BP were similar in all groups. There was less nocturnal dip in obese compared with overweight groups (P=.025). Individuals with a normal BMI were taking fewer antihypertensive medications than those in the obese group (P=.01). Individuals classified as obese had a higher left ventricular mass index than those with a normal BMI (female, P=.028; male, P<.001); this relationship remained after multivariate linear regression. Obese individuals with hypertension required more medication to achieve similar mean ambulatory BP values, had less nocturnal dip in BP, and had a higher prevalence of left ventricular hypertrophy. As such, obese patients are at potentially increased risk of cardiovascular events. *J Clin Hypertens (Greenwich).* 2015;17:466–472.

The prevalence of obesity is rising, bringing with it an increased recognition of its impact on many health conditions.[1] Excess body weight has been estimated to be the sixth most important health risk factor, contributing to the overall burden of disease.[2] This includes several conditions traditionally seen as signs of end organ damage including left ventricular hypertrophy (LVH) and chronic kidney disease (CKD).

The link between increasing body weight and hypertension is well established.[3,4] However, the mechanisms are complex and remain an issue of scientific debate. The relationship has been described as a "two-way street" rather than one exerting a causative effect on the other.[5] Both increasing body mass and hypertension can lead to end organ damage.

Obesity causes several cardiovascular and hemodynamic changes including inducing a state of chronic volume overload as a result of the increased requirements to circulate blood through large and relatively low resistance adipose tissue.[6] This may result in structural changes to the heart, including increased myocardial mass.[7]

There is increasing evidence of obesity contributing to the development and progression of CKD.[8,9] The development of CKD is often multifactorial in nature, resulting from an interaction between risk factors. Obesity may influence CKD through structural changes in the glomeruli (so-called obesity-related glomerulopathy), alterations to renal hemodynamics, activation of cascades including the renin-angiotensin-aldosterone system, and chronic inflammatory changes.[10] These changes may result in deterioration of renal function and development of albuminuria. The concept of obesity as a modifiable risk factor has been demonstrated by the improvement in renal function in individuals after weight loss.[11–13] Factors such as obstructive sleep apnea, more common in overweight individuals, has been shown to have an adverse effect on BP control and renal function.[14]

Obesity in young- to middle-aged patients with mild hypertension exhibit more end organ damage (LVH and microalbuminuria) than their counterparts with normal body mass index (BMI).[15] However, evidence is sparse in individuals with more severe hypertension. We investigated whether the perceived wisdom about obesity and hypertension held true in this population.

Therefore, the aims of this study were to assess whether individuals with raised BMI and hypertension had more difficult-to-control BP and if they were more likely to have evidence of end organ damage in the form of LVH and renal impairment.

PATIENTS AND METHODS

Data were collected prospectively from all individuals attending a teaching hypertension hospital clinic in Birmingham, United Kingdom, for an 11-year period (1998–2011) and recorded electronically. The main indication for referral to the clinic was difficulty in controlling hypertension. Other indications were multiple antihypertensive drug intolerances and suspected white-coat effect. We subsequently accessed these data retrospectively using a modified database where patient-identifiable characteristics had been removed.

All patients referred to the clinic had BP measured with a validated BP monitor using Omron M5-I automated BP machines (Omron Healthcare, Inc, Lake

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Forest, IL) according to European Hypertension Society International protocol validation.[16,17] An average of three BP readings with patients in the sitting position after 5 minutes of rest was taken as clinic BP.[18] Twenty-four–hour ambulatory BP monitoring (ABPM), M-mode echocardiography, electrocardiography, urine analysis, and blood tests (including urea and electrolytes) were routinely performed.

ABPM was performed using a Spacelabs 90207 device (Spacelabs Healthcare, Snoqualmie, WA) according to British Hypertension Society (BHS) validation grade B/B and using 90217 ABPM monitors (Spacelabs Healthcare) according to BHS validation grade A/A in accordance with national guidelines.[19] Machines were regularly calibrated. Average 24-hour, daytime (7 AM to 11 PM), and nighttime (from 11 PM to 7 AM) BP (both systolic BP [SBP] and diastolic BP [DBP]) were recorded. Nocturnal dip was calculated as average daytime SBP minus nighttime SBP divided by daytime SBP and then expressed as a percentage.

Height was measured to the nearest 1 cm, and weight was measured to the nearest 0.1 kg to enable the BMI to be calculated (weight divided by the square of height in meters).

Clinical data obtained during the initial clinical consultation included age, ethnicity (self-reported), history of cardiovascular disease (angina pectoris, myocardial infarction, and stroke), diabetes mellitus, use of antihypertensive medication, and smoking status. Renal impairment was assessed using serum creatinine and urinary albumin creatinine ratio (ACR) measurements to quantify albuminuria. The Modification of Diet in Renal Disease equation was used to convert serum creatinine to estimated glomerular filtration rate (eGFR, mL/min/1.73 m^2) as per the prevalent practice in the hospital at the time of the study.[20] Left ventricular hypertrophy was assessed by M-mode echocardiography carried out by experienced technicians.

Left ventricular mass (LVM) was calculated using the Penn Convention equation:[21]

$$\begin{aligned}\text{LVM(g)} &= 1.04 \times ([\text{left ventricular end-diastolic diameter} \\ &\quad + \text{interventricular septum end diastolic diameter}]^3 \\ &\quad - [\text{left ventricular end-diastolic diameter}]^3) - 13.6\end{aligned}$$

LVM was normalized for body surface area (BSA) to calculate LVM index (LVMI) using the formula

$$\text{LVMI} = \text{LVM}/\text{BSA}$$

and BSA calculated by the Mosteller formula:[22]

$$\text{BSA} = (\text{height(cm)} \times \text{weight(kg)}/3600)^{0.5}$$

Individuals with a known diagnosis of diabetes mellitus (DM) were excluded in order to eliminate the confounding effect of DM on target organ damage, as were those with a BMI <18.5 kg/m^2. All identifiable characteristics were removed to anonymize data before analysis.

Statistical Analysis

Analyses were performed with BMI divided into internationally accepted categories of normal (BMI 18.5–24.9 kg/m^2), overweight (BMI 25–29.9 kg/m^2), and obese (BMI >30 kg/m^2, n=467) groups. Statistical analysis was performed using SPSS for windows (PASW Statistics 20; SPSS Inc, Chicago, IL). For all analyses, a *P* value below .05 was considered statistically significant.

Parametric (BP, serum creatinine) data were assessed using one-way analysis of variance; subsequent post-hoc Bonferroni analyses were carried out to identify which groups demonstrated statistically significant differences and to avoid type I error. Nonparametric data (LVMI, ACR) were assessed using Kruskal-Wallis test for three-way analysis, and Mann-Whitney *U* test was performed on paired comparisons if statistical significance was demonstrated.

Categorical variables were analyzed using chi-square contingency tables (sex, ethnicity, smoking status).

LVMI was analyzed separately for both sexes in view of different reference ranges.

Where significance was found, multivariate analyses was performed using linear regression models, incorporating other variables into the model if their *P* value was <.1. A "backward-stepwise" model was utilized. Data are presented as correlation coefficient with 95% confidence interval and *P* value. Goodness-of-fit is indicated by R^2 value, which provides an estimate of how much of the dependent variable can be explained by the independent variables in the model.

While data for most variables were nearly complete, not all individuals had recorded ACR or LVMI. Percentage completeness of data are indicated below.

RESULTS

The original database included 1253 patients. Individuals were excluded if they were underweight (BMI <18.5 kg/m^2; n=9) or had a formal diagnosis of diabetes (n=130). The 1114 individuals who fulfilled the inclusion criteria were divided into normal weight (n=207), overweight (n=440), and obese (n=467) groups. Median BMI for the complete cohort was 29.1 kg/m^2 (interquartile range, 25.8–32.0 kg/m^2).

The majority of patients (776 of 1114, 69.7%) were of white European ethnicity. South Asians (n=143) and those of African or Caribbean origin (n=82) made up 12.8% and 7.4% of the study group, respectively. Nine percent (100 of 1114) had no ethnicity information. The

three groups were well matched for sex, age, and ethnicity. Table I shows demographic data comparing the weight groups.

Smoking status was available in 96.1% (1070 of 1114) of patients. More individuals in the normal weight group were current smokers, compared with the other two groups (*P*=.011). However, similar percentages were found for all groups when current and past smokers were combined (*P*=.951).

No statistically significant difference was seen in reported prevalence of cerebrovascular disease (*P*=.291). There was a trend towards a higher prevalence of coronary artery disease in the overweight and obese groups although this did not reach statistical significance (*P*=.055).

There were no differences in rates of resistant hypertension, defined as an office BP >140/90 mm Hg and ABPM >135/85 mm Hg and taking three antihypertensive agents (*P*=.215), or white-coat hypertension, defined as office BP >140/90 mm Hg and ABPM <135/85 mm Hg (*P*=.091), between the groups.

BP control was assessed by comparing mean ambulatory BP between the groups. All participants had adequate number of daytime BP recordings from 24-hour ABPM, allowing for calculation of mean daytime BP. Mean daytime SBP and DBP were similar for all groups (Table II). Daytime and nighttime readings were obtained in 1061 of 1114 (95.2%) individuals. While mean nighttime SBP and DBP were similar for all groups, less nocturnal dip was observed in the obese BMI group compared with the normal and overweight groups (*P*=.013). Post-hoc Bonferroni analysis identified a significant difference between the obese and overweight groups in nocturnal dip (*P*=.025).

There were statistically significant differences in the mean number of antihypertensive medications used in each weight group (normal 1.6, overweight 1.8, obese 2.0, *P*=.001; Table II). Post-hoc Bonferroni analysis identified a significant difference in medication levels between individuals of normal weight and their obese counterparts (*P*=.001). Similar percentages of all BMI groups were taking four or more antihypertensives.

Echocardiography-derived LVMI results were available for 858 (77.0%) participants. There were significant differences in mean LVMI between the groups (Table III). For women, LVMI was higher in obese vs normal (*P*=.028) and obese vs overweight (*P*=.01) groups. For men, LVMI was higher in overweight vs normal (*P*=.007) and obese vs normal (*P*<.001) groups.

To ascertain whether the association between BMI and LVMI remained significant in multivariate analyses, linear regression was performed with LVMI (log-transformed due to distribution) as the dependent variable, split by sex. BMI was analyzed as a continuous variable. Because of high correlations between BP readings, mean SBP and DBP daytime ABPM readings and nocturnal dip were the BP variables included in the model.

Linear regression demonstrated that elevated BMI was a significant predictor of higher LVMI (Table IV) for both male and female participants. Other variables predicting a higher LVMI were daytime SBP from ABPM (both sexes), black ethnicity (male sex), South Asian ethnicity (male sex associated with lower LVMI), nocturnal dip (male sex), and lower eGFR (female sex). This model explains 26.6% of the LVMI variation for male participants (adjusted r^2 0.266) and 18.9% (adjusted r^2 0.189) for women.

Creatinine and eGFR results were available for 1072 individuals (96.2%) and ACR in 953 (85.5%). Analysis

TABLE I. Baseline Demographic and Vascular Comorbidity Characteristics

	Complete Cohort (N=1114)	Normal (n=207)	Overweight (n=440)	Obese (n=467)	*P* Value
Sex, No. (%)					
Female	589 (52.9)	117 (56.5)	213 (48.4)	259 (55.5)	.53
Male	525 (47.1)	90 (53.8)	227 (51.6)	208 (44.5)	
Age, y					
Mean (standard deviation)	53.1 (16.9)	53.8 (18.1)	54.2 (17.6)	51.8 (15.7)	.087
Ethnicity, No. (%)					
White	776 (69.7)	138 (66.7)	306 (69.5)	332 (71.1)	.674
Black	82 (7.4)	14 (6.8)	29 (6.6)	39 (8.4)	
Asian	143 (12.8)	32 (15.5)	60 (13.6)	51 (10.9)	
Other	13 (1.2)	2 (1.0)	7 (1.6)	4 (0.9)	
Not stated	100 (9.0)	21 (10.1)	38 (8.6)	41 (8.8)	
Smoking status, No. (%)					
Current	214 (19.2)	58 (28.0)	78 (17.7)	78 (16.7)	.011
Past	332 (29.8)	45 (21.7)	139 (31.6)	148 (31.7)	
Never	524 (47.0)	97 (46.9)	207 (47.0)	220 (47.1)	
Not stated	44 (3.9)	7 (3.4)	16 (3.6)	21 (4.5)	
Coronary artery disease, %[a]	8.2	4	9.4	9	.055
Cerebrovascular disease, %[b]	6.6	8.6	7	5.4	.291

Data completeness: [a]96.9% and [b]94.8%.

TABLE II. Blood Pressure Parameters and Antihypertensive Medication Divided by BMI Group

	Complete Cohort (N=1114)	Normal (n=207)	Overweight (n=440)	Obese (n=467)	*P* Value	Data Completeness, %
BP parameters (mean±SD), mm Hg						
Clinic SBP	156.9 (26.3)	159.7 (28.0)	157.7 (43.8)	154.9 (26.3)	.068	98.8
24-hour ambulatory SBP	135.2 (18.0)	136.5 (19.0)	134.3 (17.1)	135.4 (18.5)	.352	97.4
Daytime ambulatory SBP	139.3 (18.0)	140.0 (18.4)	138.6 (17.4)	139.6 (18.4)	.56	100
Nighttime ambulatory SBP	126.1 (20.8)	125.9 (21.2)	124.7 (20.1)	127.7 (21.2)	.101	95.2
Clinic DBP	94.3 (14.0)	94.5 (14.1)	93.7 (13.8)	94.8 (14.2)	.488	98.7
24-hour ambulatory DBP	79.8 (11.8)	81.2 (11.2)	79.2 (12.0)	79.8 (11.8)	.143	97.7
Daytime ambulatory DBP	83.1 (12.1)	84.7 (11.8)	82.4 (12.3)	83.0 (12.1)	.07	99.9
Nighttime ambulatory DBP	72.5 (12.6)	73.6 (12.6)	71.7 (12.6)	72.7 (12.6)	.205	95.2
Nocturnal dip (% of SBP), mean (SD)	9.4 (8.8)	10.1 (9.2)	10.0 (8.3)	8.4 (9.0)	.01[a]	95.2
No. of antihypertensives, mean (SD)	1.9 (1.3)	1.6 (1.3)	1.8 (1.3)	2.0 (1.3)	.001[b]	100
≥4 agents, %	11.3	11.1	11.6	11.1	.97	100
Resistant hypertension, %	8.3	7.8	6.9	10.1	.215	98.5
White-coat hypertension, %	9.1	7.3	11.4	7.7	.091	98.5

Abbreviations: BMI, body mass index; BP, blood pressure; DBP, diastolic blood pressure; SBP, systolic blood pressure; SD, standard deviation. [a]Post-hoc analysis: overweight to obese *P*=.02. [b]Post-hoc analysis: normal to obese *P*=.001.

TABLE III. Markers of Cardiac and Renal End Organ Damage

	Complete Cohort	Normal	Overweight	Obese	*P* Value	Data Completeness, %
LVMI — Female Median (IQR)	117.8 (100.4–140.6)	112.3 (97.8–138.3)	116.0 (93.3–134.4)	124.6 (105.3–146.1)	.023	75
LVMI — Male Median (IQR)	143.2 (112.2–169.8)	121.3 (100.3–162.9)	140.7 (113.0–168.9)	148.3 (120.3–183.2)	.002	78.9
Creatinine, μmol/L Mean (SD)	95.7 (54.0)	93.8 (34.4)	95.5 (26.1)	96.7 (54.0)	.694	96.2
eGFR, mL/min Mean (SD)	72.5 (20.8)	73.9 (21.8)	72.6 (21.0)	71.8 (20.2)	.497	95.8
ACR, mg/mmol Median (IQR)	2.5 (0.8–6.0)	3.2 (0.8–6.8)	2 (0.7–5.5)	2.9 (0.8–6.4)	.811	85.5

Abbreviations: ACR, albumin creatinine ratio; eGFR, estimate glomerular filtration rate; IQR, interquartile range; LVMI, left ventricular mass index; SD, standard deviation.

of renal function using these measurements did not demonstrate any significant differences between the groups (*P*=.497 for eGFR and *P*=.81 for ACR). Similarly, no difference was found for the proportion of each group with an eGFR <60 mL/min (*P*=.691) or ACR >3.5 mg/mmol (*P*=.190).

The analyses for evidence of end organ damage are detailed in Table III.

DISCUSSION

In this study, overweight and obese individuals required more antihypertensive agents to achieve similar ambulatory BP control and had less nocturnal dip on ambulatory BP measurement. Both the overweight and obese groups had higher prevalence of LVH. There was no difference in the prevalence of CKD between obese-hypertensive people and their nonobese counterparts.

Obese patients required more antihypertensive medication to achieve similar ambulatory BP control as those with lower BMI. This finding is consistent with a recent population-based study showing that not only is hypertension much more prevalent in the obese population, but they also demonstrate poorer BP control.[23] Possible reasons for this include increased salt and fluid retention, activation of the sympathetic nervous system and stimulation of the renin-angiotensin-aldosterone system associated with obesity.[24]

The circadian rhythm of BP control is an important parameter measured in 24-hour ambulatory BP recordings. Nighttime ambulatory BP has been shown to be a major determinant of survival.[25,26] Similarly, absence of a nocturnal dip is associated with increased cardiovascular morbidity and mortality and renal impairment.[27,28] While no difference in nighttime SBP or DBP was found, this study supports previous findings that normal nocturnal BP reduction is significantly decreased in obese individuals.[29,30] Daytime and nighttime readings were divided by fixed cut points (7 AM and 11 PM) rather than by patient-reported day and night intervals. Studies have shown that this may underestimate the nocturnal BP drop.[31,32] Hermida and colleagues[33] observed that taking one antihypertensive

TABLE IV. Multivariate Linear Regression Model for LVMI (Log-Transformed) as the Dependent Variable[a]

	Men				Women			
Variable	Coefficient	Lower CI	Upper CI	*P* Value	Coefficient	Lower CI	Upper CI	*P* Value
BMI (log-transformed)	0.304	0.134	0.475	.001	0.164	0.026	0.301	.020
Age (per 10 y)	0.017	−0.003	0.036	.101	0.033	0.016	0.050	<.001
Ethnicity								
White		ref				ref		
Black	0.145	0.025	0.265	.018	0.070	−0.020	0.160	.126
South Asian	−0.107	−0.191	−0.023	.013	−0.064	−0.143	0.016	.116
Other	0.145	−0.093	0.383	.231	−0.005	−0.200	0.190	.960
Not known	0.020	−0.098	0.138	.740	0.066	−0.024	0.157	.152
Day SBP (per 5-mm HG rise)	0.031	0.023	0.039	<.001	0.024	0.017	0.032	<.001
Nocturnal dip (per % increase)	−0.007	−0.010	−0.003	<.001	−0.002	−0.005	0.001	.107
eGFR (per 5-mL/min increase)	−0.009	−0.017	−0.001	.021	−0.007	−0.014	0.000	.038

Abbreviations: BMI, body mass index; BP, blood pressure; CI, confidence interval; eGFR, estimated glomerular filtration rate; LVMI, left ventricular mass index; SBP, systolic blood pressure. [a]Regression analysis performed separately for men and women because of different reference ranges.

agent at night resulted in reduced loss of nocturnal dip and cardiovascular risk. This study involved patients with CKD and it is not clear whether the results can be extrapolated to hypertensive patients without CKD. More work is required to investigate this further.

When the mean clinic BP was compared between the three groups there was a trend toward a progressively lower mean SBP from the normal to the obese group. Perhaps this did not reach statistical significance because of large standard deviations in all three groups. However, in this study we assessed BP control primarily by comparing mean ambulatory BP values, which is considered to be the gold standard in the context of hypertension management.[34]

LVH, as measured by LVMI, was higher for both sexes as BMI increased, in agreement with past evidence of the additive effect of obesity in hypertensive individuals.[35] This relationship between elevated BMI and higher LVMI remained in a multivariate linear regression model demonstrating that the association is independent of BP, nocturnal dip, history of coronary artery disease or cardiovascular disease (excluded in backward-regression model), and renal function. A previous study looking at excess adiposity, as measured by waist circumference or skin-fold thickness, found that LVMI was higher in hypertensive individuals with central obesity compared with those with a normal waste circumference, and postulated that this effect may be mediated by leptin.[36] Unfortunately, at the time of data collection other anthropometric measures of adiposity were not routinely collected. Further research is needed to evaluate whether it is increased BMI or these other measures that confer a higher risk.

Interestingly, differences in LVMI were shown in black (associated with higher LVMI) and South Asian (associated with lower LVMI) male participants. The relationship between LVMI and ethnicity has previously been explored, with BP and nocturnal dip being associated with LVMI in black but not white individuals.[37] However, Mayet and colleagues, while stating there were no significant baseline differences with BMI between white and black groups, do not appear to have included BMI in their analyses. Therefore, we feel the relationship between LVMI, BMI, and ethnicity is worthy of further investigation.

We were unable to detect any difference in renal function when measured by serum creatinine, eGFR, or ACR. Similar and acceptable BP control in all three groups producing similar renal protection may account for this. Another possible reason may be that the study looked at a single point in time, namely an individual's first visit to the hypertension clinic. We therefore do not know whether difference in renal function would have become apparent with time.

The overweight and obese individuals in this study had a higher prevalence of LVH and lack of nocturnal dip. Both of these are known to be independently associated with increased risk of end organ damage and cardiovascular disease.[18] We therefore suggest that despite similar BP control, the individuals with higher BMI are at a higher risk of developing complications. Indeed, the overweight and obese individuals in this study had a higher prevalence of coronary artery disease than those with normal weight although this did not reach statistical significance (P=.055).

Furthermore, much of the previous research looked at the additional effect of obesity on individuals with *mild* hypertension. Individuals with moderate to severe hypertension may have already begun to develop end organ damage as a result of their persistently elevated BP regardless of their BMI status.

STUDY STRENGTHS AND LIMITATIONS

The strengths of this study are that it included a prospectively collected, ethnically diverse cohort of patients referred to the hypertension clinic. BP control was assessed using 24-hour ambulatory BP readings in line with British Hypertension Society guidelines. LVH was assessed from LVMI calculated from echocardiography-derived data. Diabetic patients were excluded to

remove the confounding factor of the metabolic syndrome:[38] indeed, it is the metabolic syndrome that has been the focus of previous research and consequently our study provides an alternative insight.[39,40]

The main limitation of this study is its cross-sectional nature. We looked at an individual's first visit to the hypertension clinic, with no follow-up, to assess whether evidence of end organ damage progressed. The majority of patients were referred for difficulty in controlling BP, as reflected by their elevated clinic BP readings (Table II). However, when assessed by 24-hour monitoring, BP control was better in all three groups of patients.

The average number of antihypertensive medications was lower than expected, with roughly 11% of patients taking four or more agents. This may reflect the fact that a large number of individuals were referred before the use of ABPM became widespread in the United Kingdom, thereby not accounting for issues such as white-coat hypertension possibly resulting in incorrectly elevated readings warranting referral.[41] Indeed, the prevalence of white-coat hypertension was 9.1% with no significant difference between BMI groups. Additionally, issues such as incorrectly sized BP cuffs in primary care may have falsely elevated readings.[42] We were not able to assess adherence with antihypertensive medication. Finally, some data were not complete, although all parameters except LVMI (77.0%) and ACR (85.5%) had at least 90% data entry.

CONCLUSIONS

Our findings suggest that individuals in the obese BMI category are more likely to take more antihypertensive medication to achieve similar BP control and have less nocturnal dip compared with people in the normal weight and overweight groups, supporting the prevailing view that they have more difficult-to-control BP compared with their leaner counterparts. They are also more likely to have LVH despite similar overall BP control. The lower nocturnal dip and higher prevalence of LVH suggest that these patients are at higher risk for developing cardiovascular complications. No significant difference was found in renal function between groups. Longitudinal follow-up of these individuals will enable further evaluation regarding development of cardiovascular complications and CKD.

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