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Weight loss facilitates reduction of left ventricular mass in obese hypertensive patients: The Campania Salute Network

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Abstract *Background and aims:* Reduction of left ventricular mass index (LVMi) during antihypertensive treatment is less likely to occur in obese subjects. The aim of the study was to assess whether weight loss influences reduction of LVMi in treated, obese, hypertensive patients.
Methods and results: From the Campania Salute Network registry, we identified 1546 obese hypertensive patients (50 ± 9 years, 43% women) with more than 12 months follow-up. Echocardiographic reduction of LVMi was considered as achievement of normal values (<47 g/m$^{2.7}$ in women or <50 g/m$^{2.7}$ in men) or a reduction of $\geq10\%$ during follow-up. Weight loss was considered as $\geq5\%$ reduction in body weight, and occurred in 403 patients (26%) during a median follow-up of 50 months (IQrange:31–93). Median weight loss was 8.6% (IQrange:6.5–12). Patients with weight loss had higher baseline body mass index ($p < 0.05$), while there was no difference in age, sex, duration of hypertension, prevalence of diabetes, metabolic syndrome and average blood pressure during follow-up. During follow-up, 152 patients (9.8%) exhibited reduction of LVMi. Reduction of LVMi was more frequent (12.9% vs 9.1%, $p < 0.030$) in patients losing weight than in those who did not. In logistic regression analysis, weight loss was associated with reduction of left ventricular mass index (OR 1.51 [95%CI 1.02–2.23], $p = 0.039$), independent of significant associations with younger age, lower average systolic blood pressure during follow-up, longer follow-up time and higher LVMi at baseline.
Conclusion: In treated obese hypertensive patients, weight loss during follow-up promotes significant reduction of LVMi, independent of baseline characteristics and blood pressure control.

Introduction

In hypertension, obesity is a main contributor for development and maintenance of left ventricular (LV) hypertrophy (LVH) [1–3], influencing LV mass through a combination of hemodynamic and non-hemodynamic stimuli [4]. Increased LV mass index (LVMi) is reported in obesity in the absence of hypertension, even in the presence of relative fat-free mass deficiency (sarcopenia) [5]. Recently, we demonstrated that decrease in LVMi in

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treated hypertensive patients is less likely to occur in obese subjects, independent of blood pressure (BP) control, both in an unselected and in a high-risk hypertensive population [2,3,6,7]. Obesity also increases the risk of incident LVH, despite aggressive antihypertensive treatment [8].

The impact of weight loss on reduction of LVMi or regression of LVH has been previously explored only in interventional studies [9–11] and in morbidly obese subjects with substantial weight loss after bariatric surgery [12,13]. However, the results are coherent, and suggest that weight reduction promotes reduction of LVMi in obesity even in the absence of arterial hypertension. In clinical practice, life style counseling for weight reduction is currently highly recommended in obese, hypertensive patients, even though the impact of weight reduction during antihypertensive therapy in daily clinical practice is not further elucidated. Whether a general counseling on weight reduction may facilitate regression of hypertensive LVH/reduction of LVMi independently of BP control in obese hypertensive patients is not yet confirmed in observational studies on epidemiological scale in the real world of outpatient clinics.

Accordingly, we studied whether spontaneous weight loss following standard lifestyle recommendations facilitates reduction of LVMi in treated young to middle-age obese hypertensive participants of a large hypertensive outpatient registry.

Methods

Patient population

The Campania Salute Network registry is an open outpatient registry collecting information from 60 general practitioners and 23 community hospitals in the Campania Region in the Southern Italy, networked by the Hypertension Research Center of the Federico II University Hospital in Naples, Italy, previously described in detail [3,14]. In short, all participants were referred to and examined at the Hypertension Research Center, including clinical assessment, laboratory testing and cardiovascular imaging. They were provided with a smart card as a utility for further follow-up by their primary physician. For this longitudinal, observational study, we identified 1546 hypertensive patients, 18-to-65 year-old without prevalent cardiovascular disease, with body mass index (BMI) $\geq$ 30 and follow-up time $\geq$ 12 months (Fig. 1). All participants had signed informed consent that health information could be used for scientific purposes; the Federico II University Hospital Ethic Committee approved the database generation and the study was performed in line with the Helsinki declaration.

Clinical and laboratory examinations

Obesity was identified as BMI $\geq$30 kg/m^2. All patients were given general dietary recommendations following the indications of the Dietary Approaches to Stop Hypertension (DASH) [15], without specific assignment to hypocaloric diets or specific programs to control adherence to diet. Weight loss was defined as a reduction in body weight $\geq$5% at the final available visit, compared to initial value.

Systolic and diastolic BP were measured in the sitting position by a calibrated aneroid sphygmomanometer after 5 min of rest. Consistent with the current guidelines [16], BP was measured 3 times at 2 min intervals and the average of the last 2 BP was taken as the BP. Isolated systolic hypertension was defined as baseline systolic BP $\geq$ 140 mmHg and baseline diastolic BP < 90 mmHg. For this analysis, given the possible fluctuation of BP at the single control visit, BP was considered controlled when the average BP of all control visits during follow-up was <140/90 mmHg.

Fasting lipid and glucose profile were measured by standard methods in external quality-controlled laboratories. Metabolic syndrome (MetS) was defined according to the modified ATPIII criteria, substituting waist circumference with BMI $\geq$30 kg/m^2, as previously done when waist circumference was not available [17].

Glomerular filtration rate (GFR) was estimated using the CKD-EPI formula [18]. The antihypertensive treatment during follow-up was taken as the type of antihypertensive medications prescribed in more than 50% of the follow-up visits in the individual patients [3].

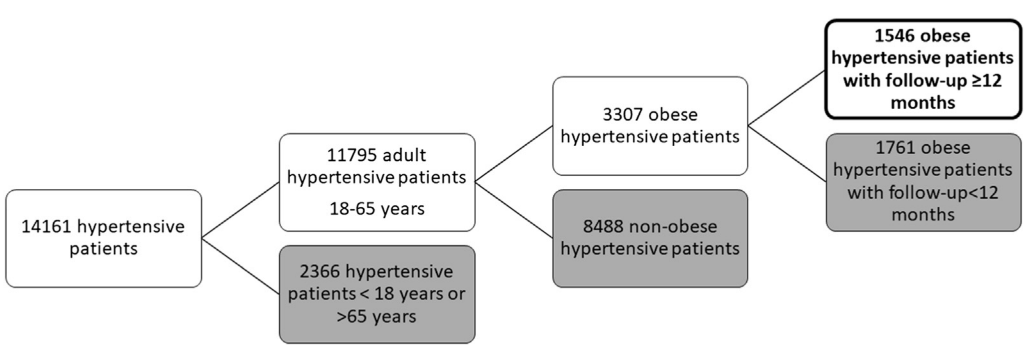

Figure 1 Flow chart demonstrating the final study population in black.

Echocardiography

Echocardiograms were performed following a standardized protocol at baseline and during follow-up visits (at least once a year) using available commercial machines, and measurements were made according to the joint American Society of Echocardiography/European Association of Echocardiography recommendations [19]. As previously reported [20], echocardiograms were recorded on videotapes, stored digitally and read offline on dedicated workstations (MediMatic, Genova, Italy) by one expert reader, under the supervision of a senior faculty member.

LV mass (LVM) was calculated from a necropsy-validated formula [21], and indexed for height in meters to the power of 2.7 (LVMi) [22]. LVH was adjudicated when LVMi was $\geq$50 g/m$^{2.7}$ in males or $\geq$47 g/m$^{2.7}$ in females [16,23]. We considered a reduction of LVMi to be clinical significant if $\geq$ 10% referred to baseline value or if normal values were achieved at the follow-up visit.

Statistics

Data were analyzed using SPSS (version 22.0, SPSS, Chicago, IL, USA) and continuous variables were reported as median and interquartile range or mean $\pm$ 1 SD, while categorical variables were reported as numbers and percentages. Descriptive statistics comparing patients with and without weight reduction was performed using unpaired t-test or chi-squared statistics as appropriate in normally distributed parameters, and Mann–Whitney U test when the parameters was not normally distributed. Univariate and forward stepwise binary logistic regression analysis was used to verify whether weight loss, contributes to explain the outcome variable, i.e variation of LVMi, independently of known confounders including age, sex, BP control during follow-up, GFR, diabetes, LVMi at baseline, follow-up time and number or class of antihypertensive drugs. $P < 0.05$ was considered statistically significant.

Results

Weight reduction occurred in 403 patients (26%) during a median follow-up of 50 months (interquartile range: 31–93), with a median weight loss of 8.6% (interquartile range: 6.5–12). Patients with weight reduction had higher baseline and lower follow-up BMI than patients without weight reduction (all $p < 0.0001$), while there was no significant difference in age, sex, prevalence of diabetes, metabolic syndrome and isolated systolic hypertension, heart rate, GFR, lipid and smoking status or initial BP (Table 1). There were no differences between groups in follow-up time, average BP during follow-up, proportion of patients with BP control during follow-up or number and class of antihypertensive drugs (Table 1).

Table 1 Patients characteristics and antihypertensive treatment in groups without and with significant weight reduction at follow-up.

	No weight reduction (n = 1143)	Weight reduction (n = 403)	p-value
Age (years)	51 ± 9	50 ± 10	0.317
Female sex (%)	43	44	0.673
Duration of hypertension (years)	5.0 (2.0–10.0)	5.0 (2.0–11.0)	0.291
BMI (kg/m^2) baseline	33.1 ± 3.0	34.1 ± 4.5	<0.001
BMI (kg/m^2) follow-up	33.7 ± 3.5	30.6 ± 4.2	<0.001
Diabetes (%)	12	15	0.151
Metabolic syndrome (%)	57	59	0.711
Isolated systolic hypertension (%)	17	15	0.352
Smoking (%)	18	14	0.131
SBP baseline (mmHg)	144 ± 19	143 ± 17	0.458
DBP baseline (mmHg)	91 ± 11	91 ± 11	0.827
Heart rate (bpm)	76 ± 11	76 ± 12	0.270
Total cholesterol (mg/dL)	204 ± 39	208 ± 41	0.138
LDL-cholesterol (mg/dL)	128 ± 36	130 ± 39	0.256
Triglycerids (mg/dL)	149 ± 81	149 ± 80	0.975
GFR_{EPI} (mL/min/1.73 m^2)	83 ± 15	83 ± 16	0.549
Follow-up time (months)	49 (30–89)	55 (32–100)	0.077
Average follow-up SBP (mmHg)	138 ± 13	138 ± 11	0.312
Average follow-up DBP (mmHg)	86 ± 7	85 ± 7	0.112
BP control at follow-up (%)	73	77	0.194
Number of antihypertensive drugs during follow-up	1.8 ± 1.0	1.7 ± 1.0	0.164
Anti-RAS (%) during follow-up	84	82	0.382
B-blockers (%) during follow-up	26	27	0.662
Ca-channel blockers (%) during follow-up	27	24	0.176
Diuretics (%) during follow-up	51	49	0.429
Oral antidiabetic medication (%) during follow-up	10	12	0.156

BMI: Body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; LDL-cholesterol: Low density lipoprotein-cholesterol; GFR: Glomerular filtration rate; BP: Blood pressure; Anti-RAS: renin angiotensin blockers.

Table 2 Baseline echocardiographic characteristics in groups without or with weight reduction at follow-up.

	No weight reduction (N = 1143)	Weight reduction (N = 403)	P-Value
LVMi baseline (g/$m^{2.7}$)	51.3 ± 9.6	50.8 ± 8.7	0.369
LVH baseline (%)	56	57	0.779
LV ejection fraction baseline (%)	66 ± 4	66 ± 4	0.069
Reduction of LVMi (%)	9	13	0.03

LVMi: Left ventricular mass index; LVH: Left ventricular hypertrophy; LV: Left ventricular.

No significant differences were found in baseline LVMi, prevalence of LVH or LV ejection fraction (all p = ns, Table 2). At the end of follow-up, 152 patients (9.8%) exhibited reduction of LVMi. Reduction in LVMi during follow-up was significantly more likely in patients with weight reduction then in patients who maintained or increased their body weight (OR = 1.49 [95%CI 1.04–2.13] $p < 0.030$).

In multivariable logistic regression analysis, a weight loss of ≥5% during follow-up increased the chance of reduction of LVMi by 51% ($p = 0.039$), independent of younger age ($p = 0.039$), lower average systolic BP during follow-up, higher LVMi at baseline and longer follow-up time (all $p < 0.01$, Table 3). The results remained unchanged even after adjusting the multivariable model for number or class of antihypertensive drugs used during follow-up.

Discussion

This retrospective study demonstrates that a moderate weight reduction in obese hypertensive patients contributes to the reduction of LVMi, independently of BP control. The results are in line with previous findings, where weight reduction was obtained by vigorous lifestyle intervention combining dietary regimes and exercise or bariatric surgery [10,12]. However, this is the first time that an independent relationship between weight reduction and reduction of LVMi is demonstrated within a real world context, including patients from a large hypertensive outpatient registry, in which weight loss was the result of general counseling on life style in line with common clinical practice, and not driven by a specific dietary regimen and/or program of physical exercise or bariatric surgery.

LVMi reduction during antihypertensive treatment is associated with a significant reduction in cardiovascular risk [24,25]. However, we have recently demonstrated that in a real world context, significant reduction of LVMi is difficult to achieve, in particularly in obese hypertensive patients, a failure that is also independent of optimal BP control [2,3]. The need for combining antihypertensive treatment with weight reduction is in line with the suggested multifactorial etiology of LVH in obesity. Increase in LVMi in hypertensive patients with concomitant obesity is certainly attributable to cardiomyocyte hypertrophy in response to combined hemodynamic pressure and volume overload induced by the high BP itself, as well as the characteristic increased circulating blood volume in obesity. However, in the obese patient, non-hemodynamic factors mostly related to body composition and fat distribution play an important role to explain the increase in LVMi. These factors include increased epicardial fat accumulation, direct fat infiltration in the myocardium, accumulation of triglycerides in the contractile elements, chronic obesity-related inflammation with edema, and reactive fibrosis in the myocardium [23].

Weight reduction has previously also been demonstrated to be associated with a reduction in BP; however, a recent Cochrane analysis indicates that though weight reduction diets reduces body weight and BP, the magnitude of the effects remains uncertain [26]. Our present analysis in the Campania Salute Network including hypertensive patients treated even before presenting at the tertiary care outpatient clinic, indicates that weight reduction per sé did not impact BP control during follow-up. Thus, the reduction in LVMi during follow-up, independent of BP control, suggests a potential added effect of weight-loss on reduction of LVMi in treated obese hypertensive patients.

Table 3 Independent predictors of reduction in LV Mass index during follow-up. Univariable and multivariable forward stepwise binary logistic regression model.

Variables	Wald	OR	95%CI	p-value	Wald	OR	95% CI	p-value
	Unadjusted				Adjusted			
≥5% weight loss	4.7	1.49	1.04–2.13	0.030	4.3	1.51	1.02–2.23	0.039
Age (years)	0.5	0.99	0.98–1.01	0.463	4.3	0.98	0.96–0.99	0.039
Female sex	0.6	1.14	0.81–1.61	0.448				
Average SBP follow-up (mmHg)	3.8	0.99	0.97–1.00	0.052	18.7	0.96	0.95–0.98	<0.001
GFR_{EPI} (mL/min/1.73 m^2)	0.002	1.00	0.99–1.01	0.964				
Diabetes	0.8	0.78	0.45–1.34	0.371				
LVMi baseline (g/$m^{2.7}$)	61.9	1.07	1.05–1.09	<0.001	76.0	1.09	1.07–1.11	<0.001
Follow-up time (years)	8.7	1.01	1.00–1.01	0.003	8.2	1.01	1.00–1.01	0.004

SBP: Systolic blood pressure; GFR: Glomerular filtration rate; LVMi: Left ventricular mass index.

The effect of weight reduction on LVH regression has previously been demonstrated in morbid obese subjects undergoing substantial weight loss due to bariatric surgery [12]. In a meta-analysis by Cuspidi C et al. including 23 studies and 1022 patients, exploring the effect of bariatric surgery on LV structure and function, LVH regression was reported to be a major finding, in addition to improvement in diastolic function [27].

Previous studies and meta-analyses confirmed that LVH, a recognized risk predictor in hypertension, is particularly common in obese hypertensive patients, and is present even in the absence of elevated BP [1,28]. This is in-line with the high prevalence (56%) of LVH detected in our population of obese hypertensive patients. In the current guidelines for hypertension management, obese hypertensive patients are classified as a high-risk population, prone to target organ damage and subsequent cardiovascular events [16]. Zhang et al. identified overweight and obesity as independent covariates of changes in LV mass in a meta-analysis including 28 randomized trials, comprising 2403 hypertensive patients [29]. Our results underline that weight reduction is difficult to achieve and maintain in a real-world context, only with generic counseling and without specific intervention; only 26% of the obese hypertensive patients in the registry had a weight reduction $\geq$5%. This proportion is in line with the self-reported weight reduction of at least 5% is reported by 36.6% of the participants in the National Health and Nutrition Examination Survey [30]. There is definitely a need for better weight reduction programs in obese hypertensive patients with increased LVMi.

In clinical practice, patients with combined obesity and hypertension are particularly challenging to treat and BP often remain sub-optimally controlled [2,3,7]. Our results strongly indicate that in the presence of obesity, antihypertensive treatment should always be combined with programs of weight reduction, to improve BP control and to favor reduction of LVMi. Compared with the general guideline recommendation of aggressive treatment of all cardiovascular risk factors in patients with hypertension [16], a specific warning for the association of hypertension with obesity might be worthy.

Limitations

There are limitations in our study. Due to its observational and retrospective nature, our study cannot identify or even infer causal mechanisms for the observed association between weight reduction and reduction in LVMi. The study population is relatively unselected and not matched and no information is available on possible comorbidities explaining weight loss. In spite of the robust consistency between our findings and previous pathophysiologic, epidemiologic and interventional studies, our results underline the need of prospectively controlled interventional studies on an epidemiological scale, where weight reduction is achieved by previously specified interventions (dietary changes, increased physical activity or both).

In conclusions, in young to middle-age treated obese hypertensive patients, weight reduction during follow-up promotes significant reduction of LVMi, independent of baseline characteristics and BP control. These findings confirm the multifactorial origin of increased LVMi in the presence of obesity and strongly suggest that in hypertensive patients with concomitant obesity, weight loss strategies in addition to antihypertensive treatment should be systematically considered to promote reduction in LVMi.

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Conflicts of interest

None.

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