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The correlation of ABMP and RV dysfunction detected by speckle tracing in obese hypertensive patients

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***Abstract*---Background:** Obesity significantly raises the risk of high blood pressure as well as heart conditions. Ambulatory blood pressure monitoring (ABPM) is a reliable way to diagnose and manage hypertension, providing more accurate predictions for cardiovascular results than standard office readings. Right ventricular (RV) dysfunction, evaluated using speckle tracking echocardiography, is becoming a new indicator of cardiovascular risk. **Objective:** To investigate the connection between ABPM parameters and right ventricular dysfunction in patients who have elevated blood pressure with excess body weight. **Methods:** Cross-sectional research involved 346 hypertensive patients, categorized into three groups based upon their BMI and waist circumference. ABPM was performed, and RV function was assessed using speckle tracking echocardiography. Data analysis was conducted based on the mean of the absolute changes among consecutive measurements as well as usage to compute the short-term blood pressure changes as well as the individuals' wake-up intervals. **Results:** Obese individuals exhibited significantly higher systolic blood pressure between 1 and 6 hours before waking compared to those of normal weight. Moreover, both diastolic and systolic blood pressure variations raised with elevated BMI. Lastly, strain imaging revealed that obese hypertensive patients had more right ventricular impairments compared to their normal-weight counterparts. **Conclusion:** As hypertensive individuals progressed from normal weight to obesity, our study found that blood pressure (BP) variability rose frequently, two-dimensional right ventricular (RV) deformation decreased, and RV enlargement gradually increased.

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Keywords---ABMP, RV Dysfunction, Speckle Tracing, Obesity, Hypertension.

Introduction

Obesity contributes directly to 75% of occurrences of hypertension, according to the American Heart Association. As obesity rates increase, so does the prevalence of hypertension, with these conditions frequently coexisting. Obesity can cause hypertension through multiple mechanisms, reinforcing these epidemiological observations [1,2].

Blood pressure variability (BPV) refers to fluctuations in blood pressure among different time periods: short-term (minutes to hours), mid-term (days to weeks), and long-term (months to years). Home measures, ambulatory blood pressure monitoring (ABPM), or repeated clinical visits (visit-to-visit BPV) can all be used to evaluate it [3].

A raised likelihood of cardiovascular events, such as nonfatal heart attacks, heart failure, and strokes, as well as all-cause mortality, was linked to higher visit-to-visit fluctuation in systolic blood pressure, according to a large study involving 25,814 individuals who participated in the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) [4].

There is little data on blood pressure (BP) fluctuation in people who are overweight or obese, even if they do not have arterial hypertension. High nighttime blood pressure variability dramatically raised the mortality risk linked to obesity, according to a large study [5].

Even after controlling left ventricular (LV) metrics, the Multi-Ethnic Study of Atherosclerosis, which recruited 4,127 attendees, observed the fact that obesity as well as being overweight was linked to right ventricular (RV) remodeling [6]. The relationship between right ventricular remodeling, blood pressure (BP) variation, as well as being overweight or obese among those having arterial hypertension has, however, yet to be thoroughly studied.

24-hour ambulatory blood pressure monitoring (ABPM), in contrast to office readings, offers comprehensive information about blood pressure profiles and their fluctuations. Compared to office blood pressure readings, ABPM parameters—such as dipping status, standard deviation, and the systolic and diastolic blood pressure (SBP and DBP) during the day and at night— possess a stronger correlation with greater likelihood of cardiovascular diseases and deaths [7]. A BPM evaluates short-term arterial pressure variability, as substantially predicts organ damage, heart attack, and fatalities [8].

Given a link between poorer cardiovascular outcomes and blood pressure fluctuation, obesity, and right ventricular remodeling, our goal was to analyze the link between BP fluctuations and RV function in hypertensive individuals of different weight categories (normal-weight, overweight, and obese). Knowing this

link could help explain why obese hypertensive patients experience such bad results.

We postulated that in hypertension individuals of all body shapes and sizes, from normal weight to obese, blood pressure (BP) fluctuation indices are associated with right ventricular (RV) functionality and deformation.

Method

Between December 2019 and January 2021, 349 hypertension participants in the study were patients who were seen at Suez Canal University Hospital's outpatient clinic. The population's general characteristics, such as age, sex, waist circumference, BMI categories, and median values of both office and ambulatory arterial pressure data, are shown in Table 1.

Whether treated or under anti-hypertensive medication, arterial hypertension is diagnosed using European Society of Cardiology recommendations once the office's diastolic blood pressure (DBP) is 90 mmHg or more, and the systolic blood pressure (SBP) is 140 mmHg or more [(9)].

Every patient in the study had anthropometric measurements—height, weight, and waist circumference among other things. A tape measure was used to measure the subject's waist circumference while they were standing, at midpoint from the iliac crest to last rib. Patients were split into two groups based on their waist circumference: those with normal measures and those with abnormal measurements. ≥102 cm in men and ≥88 cm in women were the cutoff thresholds, per the National Cholesterol Education Program Adult Treatment Panel III recommendations [(10)].

Through Quetelet's equation, body mass (kg) over height (m) squared, Body mass index (BMI) determined. Three categories of patients were created using the World Health Organization's (WHO) BMI standards [(11)]:

- Group 1: (BMI < 25 kg/m²) normal weight.
- Group 2: (25 ≤ BMI < 30 kg/m²) overweight.
- Group 3: (30 ≤ BMI < 35 kg/m²) first stage obesity.

Right Ventricular and Right Atrial assessment:

Subcostal view was used to measure the thickness of the right ventricle (RV), while the parasternal long-axis image was used to measure its internal diameter. The maximal volume of the right atrium (RA) was determined and linked with body surface area from the four-chamber view at ventricular end-systole [(12)].

Using pulsed-wave Doppler in the apical four-chamber view, tricuspid flow velocities at end-expiration during quiet breathing were measured to determine the early diastolic peak flow velocity (Et). Using the apical four-chamber window of Doppler tissue imaging, right ventricular (RV) cardiac velocities were measured. During systole (s't) and early diastole (e't), the sample volume was situated precisely above the lateral part of tricuspid annulus. The tricuspid (E/e't) ratio was obtained through the previously established Doppler values [(12)].

The right ventricular (RV) systolic blood pressure (PASP) was calculated using the formula PASP = (tricuspid regurgitation velocity)2 + right atrial pressure using the most recent guidelines. The right atrium was assessed by measuring the diameter of the inferior vena cava and documenting changes throughout respiration. If the inferior vena cava diameter is less than 2.1 cm and collapses more than 50%, the RA pressure is normal at 3 mm Hg (range 0–5 mm Hg); if the diameter is greater than 2.1 cm and collapses less than 50%, the RA pressure is high at 15 mm Hg (range 10–20 mm Hg) [12].

RV Strain and Strain Rate was conducted through three consecutive cardiac cycles of two-dimensional pictures from the apical four-chamber window. Vivid IQ was dedicated to 2D strain analysis. Three segments from the interventricular septum and three segments from RV free wall (proximal, medial, and apical segments for each wall) were among the six segments for which the software produced longitudinal strain and strain rate values. Software was used to generate strain rates and global longitudinal RV strain, which were then computed as the mean of the six segments [12].

24-Hour Ambulatory Blood Pressure Monitoring and Clinic Blood Pressure Measurement: Participants' blood pressure was continuously monitored for 24 hours. An aneroid manometer was used in the morning to take the clinic's blood pressure (BP). Three successive readings, taken in a seated position five to ten minutes apart after five minutes of rest, were averaged and recorded. The nondominant arm was subjected to noninvasively determine 24-hour ambulatory blood pressure monitoring using CONTEC ABPM50. The device took blood pressure measurements each 20 minutes between 7 AM and 11 PM and once each 30 minutes between 11 PM till 7 AM.

During blood pressure (BP) checks, patients were told to stay still but to continue with their regular daily activities. Additionally, they were instructed to keep a record of their everyday routine, including when they woke up and went to bed. Daytime blood pressure was defined as the average blood pressure readings obtained for the remainder of the day, and the nocturnal one was defined as the average blood pressure readings obtained at the time patients went to bed until they woke up. From the recorded data, 24-hour, daytime, as well as nocturnal averages of heart rate, diastolic blood pressure, systolic blood pressure, and mean arterial pressure were computed. A recording was considered valid and satisfactory if it represented more than 70% from all the measurements planned for the testing period. Arterial hypertension has been defined according to the most recent guidelines. Blood pressure fluctuation was measured using the standard deviation (SD) between the mean BP readings throughout the day, at night, and across a period of 24 hours.

Statistical Analysis

Equal variance analysis was used to compare continuous variables that represented in mean ± SD, due to their normal distribution. The different groups were compared using the Bonferroni post hoc technique. Proportional differences were assessed through chi-square testing. Correlation analysis has been applied to find the connections across blood pressure variability indices and a number of

clinical and echocardiographic indicators. A P-value below0.05 was regarded statistically significant.

Results

The age and gender distribution of the groups under study did not differ significantly (Table 1). Hypertensive individuals who were overweight or obese showed greater clinic diastolic and systolic blood pressures. (Table 1). The clinic heart rates of the various groups were the same. Obese patients had higher levels of glycosylated hemoglobin (HbA1c) than normal-weight patients (Despite that difference was none statistically significant); Obese subjects exhibited lower levels of HDL-c than normal-weight subjects, and obese patients had greater levels of total cholesterol, triglycerides as well as LDL-c than both normal-weight and overweight subjects. Serum creatinine levels in each group appeared similar (Table 1).

Table 1: Baseline features for the studied group

Variable	**HTN Plus Normal BMI (n= 114)**	**HTN Plus BMI 25-30 (n=117)**	**HTN Plus BMI≥30 (n=115)**	***P* Value**
Age	46 ± 5	47 ± 6	49 ± 7	0.186
Women	(46%)	(43%)	35%	0.578
BMI	23.26 ± 1.5	27.56 ± 1.9	32.46 ± 2.4	-
Waiste circumference	85.3 ± 6.2	94.7 ± 7.5	108.4 ± 8.9	<0.001
BSA	1.86 ± 0.18	2.01 ± 0.23	2.19 ± 0.21	-
Clinic SBP	147 ± 8	153 ± 9	159 ± 12	‹ .001
Clinic DBP	92 ± 5	96 ± 8	98 ± 8	.003
Heart rate	66 ± 7	68 ± 7	69 ± 8	.381
S. Creatinine	0.91 ± 0.11	1.01 ± 0.13	1.13 ± 0.18	.176
HbA1c	5.3 ± 0.2	5.4 ± 0.21	5.5 ± 0.22	0.214
LDL Cholesterol	112 ± 13.9	117 ± 14.8	129 ± 15.7	0.004
HDL cholesterol	46 ± 2.1	44 ± 2.3	43 ± 2.4	0.015
Triglyceride	198 ± 16.3	201 ± 15.8	205 ± 16.1	0.125
Total cholesterol	214 ± 5.8	223 ± 7.3	231 ±8.9	0.023

Table 2 Measures of ambulatory blood pressure as well as heart rate

Variable	HTN plus Normal BMI (n= 38)	HTN plus BMI 25-30 (n=37)	HTN plus BMI≥30 (n=35)	*P* Value
24 h				
SBP (mmHg)	137 ± 8	143 ± 11	148 ± 13	<.001
DBP (mmHg)	82 ± 7	85 ± 8	89 ± 10	<.001
MAP (mmHg)	99 ± 8	104 ± 8	107 ± 10	<.001
Heart Rate (beats/minutes)	70 ± 7	72 ± 8	71± 9	.432
Daytime				
SBP (mmHg)	143 ± 9	147 ± 11	150 ± 12	0.11
DBP (mmHg)	85 ± 7	87 ± 8	91 ± 9	.001
MAP (mmHg)	104 ± 9	108 ± 10	111 ± 12	.009
Heart Rate (beats/minutes)	72 ± 9	74 ± 11	75 ±10	.512
Nighttime				
SBP (mmHg)	115 ± 8	127 ± 11	136 ± 13	<.001
DBP (mmHg)	68 ± 8	75 ± 8	81 ± 9	<.001
MAP (mmHg)	86 ± 8	92 ± 9	99 ± 11	<.001
Heart Rate (beats/minutes)	64 ± 6	64 ± 8	63 ± 7	0.392
Nocturnal reduction rate (%)				
SBP	19.1 ± 3.1	14.2 ± 2.9	9.1 ± 2.2	<.001
DBP	17.4 ± 3.5	15.8 ± 3.2	12.3 ± 3.6	<.001
MAP	17.5 ± 3.7	15.6 ± 3.5	10.9 ± 2.9	<.001
Heart Rate	12.8 ± 3.1	14.9 ± 3.2	15.8 ± 4.2	0.385
SD				
24-h SBP	17.6 ± 4.9	18.7 ± 5.4	21.4 ± 7	.008
24-h DBP	14.6 ± 3.8	16.1 ± 4.2	17.2 ± 4.6	.021
Daytime SBP	15.7 ± 4.9	18.2 ± 5.3	20.5 ± 6.2	.001
Daytime DBP	13.3 ± 3.6	15.4 ± 4.1	17.1 ± 4.7	<.001
Nighttime SBP	12.2 ± 3.1	14.6 ± 3.5	17.3 ± 4.1	<.001
Nighttime DBP	9.9 ± 2.5	11.7 ± 3.1	13.9 ± 3.7	<.001

DBP, diastolic BP; MAP, mean arterial pressure; SBP, systolic BP. Data are expressed as mean ± SD.
*P < .05 for all comparisons.

Ambulatory BP Monitoring

Obese people had higher blood pressures (BPs) throughout the day than people of normal weight, and over the night, BPs progressively rose from individuals who were lean to those who were fat. Obese patients had considerably greater blood pressure throughout a 24-hour period than did lean and overweight patients (Table 2). Patients with hypertension who were normal weight to obese experienced a steady decline in their systolic, diastolic, and mean blood pressure lowering rates at night. All groups' 24-hour, daytime, and nocturnal heart rates

were comparable. Similar patterns were seen in BP variability indices: obesity was associated with larger 24-hour and daytime standard deviations (SDs) than normal weight and lean to obese patients saw a gradual increase in SDs at night (Table 2).

Table 3 Echocardiographic Variables for RV structure as well as function in the study sample

Variable	HTN with Normal BMI (n= 38)	HTN with BMI 25-30 (n=37)	HTN With BMI≥30 (n=35)	*P* Value
RV diameter (mm)	24.3 ± 2.9	25.4 ± 3.3	26.1 ± 3.6	.124
RV thickness (mm)	4.3 ± 0.6	4.7 ± 0.8	5.3 ± 1.1	<.001
TAPSE	20 ± 1.8	19 ± 1.9	18 ±2.1	.123
FAC	48 ± 2.1	47 ± 1.9	46 ±1.88	0.053
E/e' of the Rt side	7.9 ± 2.3	9.2 ± 2.7	11.1± 3	<.001
PASP (mm Hg)	26 ± 5	28± 6	32 ± 5	<.001
RV GLS	-24.2 ± 3.1	-23.1± 3.3	-20.5 ± 3.1	.003
RV FWLS	-27.4 ± 3.5	-26.5 ± 3.4	-25.2 ± 3.2	.019

FWLS: Free Wall Strain, FAC: Fractional Area Change, GLS: Global Longitudinal Strain, RV: Right Ventricular, PASP: Pulmonary Artery systolic Pressure, TAPSE: Tricuspid Plane Systolic Excursion.
*P < .05 for all comparisons.

Table 3 showed that the observed groups' RV diameters were comparable. Compared to lean and overweight patients, obese patients had thicker RV walls. Compared to lean and overweight participants, obese subjects had a considerably greater RA volume index. Obese participants with hypertension had the highest tricuspid E/e'. Compared to participants of normal weight, PASP was higher in those who were overweight or obese. Regarding RV GLS along with RV FWLS strain patterns and speckle tracking, the groups differed statistically significantly, but not when it came to the evaluation of RV systolic function using the standard 2D echocardiographic parameters.

Table 4 Correlations across 2D structural and mechanical parameters and BP variability indices during the day and at night

	Variability in Daytime BP		Variability in Nighttime BP	
	SD SBP	SD DPP	SD SBP	SD DPP
Variables	**R**	**r**	**r**	**R**
BMI	0.08	0.13	0.15	0.18
E/e'	0.19	0.15	0.16	0.15
RV thickness	0.12	0.15	0.25	0.13
PASP	0.13	0.10	0.18	0.09
RV GLS	-0.32	-0.21	-0.38	-0.20
RVFWLS	-0.34	-0.23	-0.22	-0.19

BMI: Body Mass Index, FWLS: Free Wall Strain, GLS: Global Longitudinal Strain, RV: Right Ventricular, PASP: Pulmonary Artery systolic Pressure, TAPSE: Tricuspid Plane Systolic Excursion.
*P < .05 for all comparisons.

Several associations between distinct clinical and echocardiographic characteristics and different blood pressure variability parameters (SDs) during the day and at night are shown in Table 4. The diastolic and systolic blood pressure SDs for each night revealed the best connections with these measures, but the SD of systolic blood pressure had the strongest correlations among the daily blood pressure variability parameters, with a range of clinical and echocardiographic markers.

Discussion

In comparison to non-obese hypertensives, our study revealed that obese hypertension patients had greater mean arterial pressure during the day and greater rise in diastolic and systolic blood pressure measurement and night fluctuation

Tadic and associates categorized 127 untreated hypertension patients based on their BMI and Conducted ABPM upon them. In accordance to their conclusions, blood pressure gradually rose. from lean to obese participants during night (as evaluated from bedtime until waking). Obese people also had greater daytime blood pressure (measured throughout the day) than normal weight. Additionally, people who were obese (BMI > 30) had higher blood pressure variability (standard deviation) throughout the day than those who were normal weight, and participants who were lean to obese saw a progressive rise in blood pressure variability at night [13].

Middle-aged people without known hypertension or type 2 diabetes mellitus (T2DM) who displayed an ambulatory blood pressure monitoring trend that did not dip proved more inclined to have metabolic syndrome (P < 0.001), according to a study by Ukkola et al. Furthermore, in multivariate models, impaired glucose tolerance (IGT) and ABPM non-dipping status proved to be independent predictors of IGT [14].

Additionally, Palatini and colleagues discovered a relationship between the variability in systolic as well as diastolic during the night and the BMI of subjects with untreated hypertension [15].

On the other hand, after bariatric surgery, Głuszewska and associates detected a significant decline in the variability of blood pressure among people with hypertension with a BMI of 40 kg/m² or greater. The normotensive group did not experience this decrease. Furthermore, six months after surgery, hypertension was remitted to 41.7% of patients [16].

When compared to those with hypertension of normal weight, our study revealed that obese patients exhibited progressive RV impairment and deteriorating 2D right ventricular (RV) deformation

Because of enhanced sympathetic as well as renin-angiotensin-aldosterone activity, in addition to their close relationship, hypertensive individuals, including those who are lean, overweight, and obese, have a correlation between variations in right ventricular (RV) and blood pressure (BP) [17]. Decreased physical activity, diminished arterial and cardiopulmonary reflexes, and increased inflammatory indicators could be additional contributing reasons. According to our research, obese hypertension people who have raised pulmonary pressure have higher RV afterload, which causes RV dilatation and a subsequent drop within RV ejection fraction (EF). The decrease in RV function in arterial hypertension may also be caused by the two ventricles' interdependence [18].

Therefore, a stressed left ventricle could change the right ventricle's volume-pressure characteristics and have a detrimental effect on RV function. This movement is mediated by the spiral and circular muscle fibers that surround both ventricles and the interventricular septum, which is formed of a thicker left muscle mass and a thinner right muscle mass. RV dysfunction results from elevated left ventricular (LV) end-diastolic pressure in hypertensive individuals with obesity, which is then carried backwards to the right ventricle [19].

The belief that simple arterial hypertension has no effect on the right ventricle is out of date. In spite of early aberrant rises in arterial pressure, there is a significant change in right ventricular (RV) pressures, volumes, and ejection fraction, which supports the idea that the left and right ventricles work as one unit. More research is necessary to fully understand the complex origins of the right-heart discrepancies observed in essential hypertension [19].

Limitation

There were various restrictions on the current investigation. The quality of echocardiographic pictures has a major impact on the assessment of RV function and structure. Furthermore, due to the cross-sectional format of the investigation, no causal connection between obesity, RV remodeling, and blood pressure fluctuation could be identified.

Conclusion

Our study observed that blood pressure (BP) variability steadily increased, two-dimensional right ventricular (RV) deformation deteriorated, and RV enlargement gradually increased as hypertensive individuals went from normal weight to obesity. Changes in RV morphology were linked to variations in blood pressure during the day and at night. To determine if BP variability may forecast RV remodeling in subjects with hypertension who are overweight or obese, longer-term research is required. In comparison with office-based blood pressure examinations, ABPM offers more accurate predictive insights for cardiac events and is essential for evaluating blood pressure fluctuation in obese patients.

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