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MEDICINE

The Association Between Morning Blood Pressure Surge and Cardiovascular Disease in Normotensive Type 2 Diabetic Patients: Observational Analytical Study in the Form of a Cross-Sectional Study

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Abstract

Morning increase and nighttime decrease in blood pressure (BP) have been associated with cardiovascular (CV) morbidity and mortality in general and diabetic subjects. We aimed to study the association between morning blood pressure surge (MBPS) and left ventricular hypertrophy (LVH) in normotensive type 2 diabetes mellitus (T2DM) patients. This comparative cross-sectional study included 90 normotensive T2DM patients divided according to the presence of diastolic function into two groups. Normotensive T2DM patients were subjected to ambulatory blood pressure monitoring (ABPM) to detect the MBPS and echocardiography to assess left ventricular mass index (LVMI) and diastolic function. Data were analyzed using SPSS and stepwise multiple regression and binary logistic regression analysis was performed to detect the associated independent variables for increase in LVMI. Finally, a receiver operating characteristic (ROC) curve was performed to assess a cut-off value for MBPS. A total of 31% of patients had diastolic dysfunction (DD). There was a strong positive correlation between LVMI and MBPS (r = 0.609, p-value < 0.001). Stepwise multiple regression analysis showed that MBPS was the first independent predictor associated with increased LVMI. Binary logistic regression analysis for the status of diastolic function showed an odds ratio (OR) of 1.26 and 1.11 at 95% confidence interval (CI) for MBPS and age respectively. ROC curve for prediction of DD based on the degree of MBPS showed a fair area under curve and a cut-off value of 25.5 mmHg with 85.7% sensitivity and 61.3% specificity. Higher levels of MBPS are associated with increased LVMI and DD in normotensive T2DM patients. This has potential implication as it might be a significant predictor for discovery of subclinical hypertension in early stages of diabetes mellitus and can help in the prevention of heart failure development.

Keywords Morning blood pressure surge · Left ventricular hypertrophy · Normotensive · Type 2 diabetes

Introduction

Blood pressure (BP) decreases in the night [1] and then increases in the morning as the subject awakes and starts daily activities [2]. Unfavorable outcomes were related to changes that happen beyond physiological range. Morning increase and nighttime decrease in BP [3–5] have been associated with CV morbidity and mortality in general and diabetic subjects [6, 7]. It has been increasingly recognized that the early MBPS provides a significant predictor of cardiovascular disease (CVD) risk [8].

The slight fluctuations in BP homeostasis even in the normotensive range appear to be associated with more accelerated microvascular complications in T2DM [9]. A few studies

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with contrasting findings have assessed MBPS as a CV risk predictor in T2DM [10, 11], so a better interpretation of the mechanisms linking MBPS to CV events is in need to understand if MBPS accelerates vascular injury eventually leading to CV events [12].

Objective

We aimed to study the association between morning blood pressure surge (MBPS) and left ventricular hypertrophy (LVH) in normotensive type 2 diabetes mellitus (T2DM) patients.

Methods

Out of 122 patients who underwent ABPM, 7 patients were excluded due to incomplete ABPM readings, 9 patients were excluded due to confirmation of masked hypertension, and 18 patients had inadequate quality imaging. The remaining 90 patients were enrolled into the study. Our comparative cross-sectional study included 90 normotensive type 2 diabetic patients divided into two groups according to presence of diastolic function into two groups: *group I* (without diastolic dysfunction) 62 (68.8%) patients and *group II* (with diastolic dysfunction) 28 (31.1%) patients. The mean age of the study group was (49 ± 6) years. The group had 47 males (52.2%) and 43 females (47.8%). Further descriptive analysis of the demographic data, ABPM parameters, and relevant echocardiographic measurements is demonstrated in (Tables 7, 8, and 9) respectively.

Our study was conducted from August 2018 to September 2019 at our Cardiology Department at Zagazig University Hospital, Egypt.

The protocol was approved by our Zagazig University Institutional Review Board (ZU-IRB), Egypt which confirmed that all methods were performed in accordance with the relevant guidelines and regulations and informed consent was obtained from all participants.

Our patients subjected to measurement of ABPM data for assessment of MBPS and its association with cardiovascular complications.

The sample size is calculated through the following equation: $n = \frac{2\left(Z_{\partial}+Z_{1-\beta}\right)^{2\sigma 2,}}{\Delta^2}$ and sample size with confidence level = 95% and confidence interval: 10.33 and so the sample size needed was 90 patients.

Diagnosis of T2DM was done according to diagnostic criteria of the "American Diabetes Association (ADA): a fasting plasma glucose level of ≥126 mg/dL, a 2-hour plasma glucose level of ≥ 200 mg/dL during a 75-g oral glucose tolerance test (OGTT) and a random plasma glucose of ≥ 200 mg/dL" [13]. The patients were normotensive according to the definition by the "European Society of Cardiology and European Society of Hypertension (ESC/ESH) guidelines: office SBP < 140 mmHg and DBP < 90 mmHg, ABPM mean day SBP < 135 mmHg and DBP < 85 mmHg, ABPM mean night SBP < 120 mmHg and DBP < 70 mmHg and ABPM mean 24 hours SBP < 130 mmHg and DBP < 80 mmHg" [14].

Exclusion Criteria

Patients with one or more of the following criteria were excluded from the study: ischemic heart disease, current or history of antihypertensive medications, renal failure, acute or chronic infectious disease, arrhythmias (including atrial fibrillation), valvular heart disease, congestive heart failure, pregnancy or lactation, and prior or current cancer.

All patients were subjected to the following:

1. Complete history taking.
2. General and local examination includes calculating body mass index (BMI) and body surface area (BSA), clinic blood pressure (BP) measurement confirming normal BP according to the previously mentioned criteria, examination of pulse, lower limbs, and neck veins, and local examination of the chest and heart.
3. Electrocardiography (ECG)
4. ABPM: All patients underwent ABPM using Riester Ricardio ambulatory BP monitor approved according to the protocol of ESH. Intervals between measurements were 30 min. Patients with missing recordings of more than 2 h were excluded or had a repeated ABPM.

Patients defined daytime and sleep-time by filling a diary during the test.

Sleep-trough method was used to define MBPS as "the difference between mean SBP in the first two hours after awakening and the mean of three readings centred on the maximum asleep SBP dip" [15].

5. *Echocardiographic* assessment was done to all subjects using Siemens ACUSON X300 ultrasound machine.

   Left ventricular ejection fraction (LVEF) was assessed by modified Simpson's method in apical four chamber (A4C) and apical two chamber (A2C) views [16].

   LVH was assessed by M-mode in parasternal long axis (PLAX) view for measurement of interventricular septum thickness (IVST) and posterior wall thickness (PWT) if more than 11 mm was considered that patient has LVH [16].

   LV mass was assessed by M-mode in the PLAX view using the following formula:

$$\mathrm{LVM} = 0.8 \times \left[1.04\left[(\mathrm{LVIDd} + \mathrm{PWd} + \mathrm{IVSd})^3 - (\mathrm{LVID})^3\right]\right] + 0.6\ \mathrm{g}$$

Table 1 Spearman's correlation of LVMI with MBPS and average 2-h awakening SBP

Spearman's correlation		MBPS	Average awakening SBP
LVMI for males	Correlation coefficient	0.7	0.5
	p-value	< 0.001 (HS)	< 0.001 (HS)
LVMI for females	Correlation coefficient	0.7	0.6
	p-value	< 0.001 (HS)	< 0.001 (HS)

LVMI left ventricular mass index, *MBPS* morning blood pressure surge

where LVIDd is the LV internal diameter; PWd is the posterior wall thickness; IVSd is the interventricular septum thickness all measured in end diastole. LVMI was then calculated by dividing LVM over BSA [16].

For assessment of diastolic function, pulsed wave (PW) Doppler imaging of trans-mitral flow was done in A4C view for assessment of E wave velocity, A wave velocity, and E/A ratio. Tissue Doppler imaging (TDI) of septal and lateral e′ was done in A4C view. Assessment of left atrial volume (LAV) was done using area length method in A2C and A4C views and left atrial volume index (LAVI) was then calculated by dividing LAV over BSA. Continuous wave (CW) Doppler for assessment of peak tricuspid regurgitation jet velocity was also done in A4C view. Evaluation of diastolic function was done according to the 2016 recommendations posted by the American Society of Echocardiography and European Association of Cardiovascular Imaging (ASE/EACVI) [17] who classify diastolic function into three classes (normal, indeterminate, and diastolic dysfunction) based on four key variables. Prevalence studies showed that 8-15% of patients are classified as indeterminate [18, 19].

Statistics

Analysis of the collected data was performed using statistical package for the social sciences (SPSS) version 23.

The correlation between LVMI and other numerical variables was assessed by Spearman's rank correlation (Table 1) and (Fig. 1). Stepwise multiple regression analysis was performed to detect the significant predictor of increase in LVMI (Tables 2 and 3). The presence of diastolic dysfunction (DD) was tested with other categorical data using Chi-square (X^2) test (Table 4). Differences between subjects with and without DD regarding numerical variables were assessed using the Mann-Whitney *U* test (Tables 5 and 6 and Fig. 2). Then, odds ratios for the statistically significant independent variables were assessed using binary logistic regression analysis. A

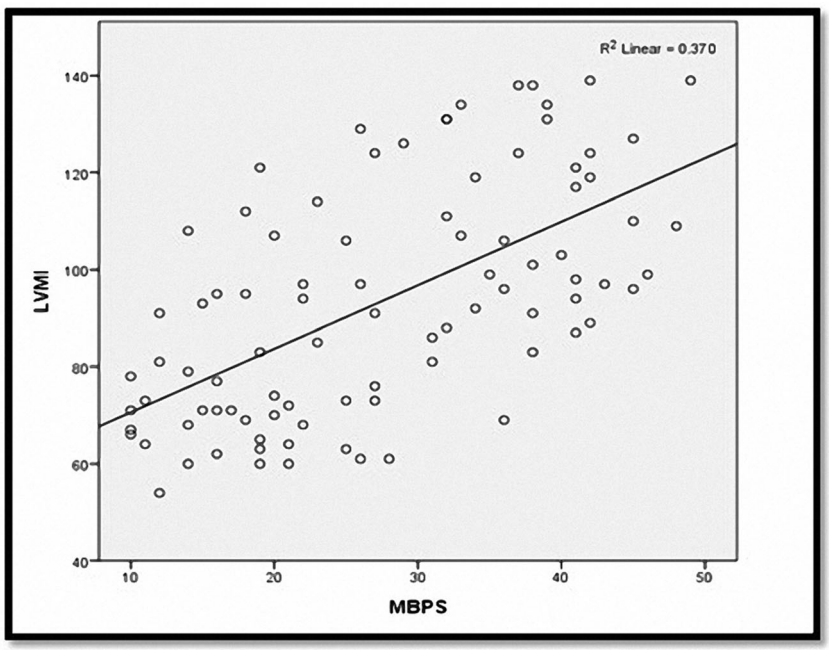

Fig. 1 Scatterplot showing strong positive correlation between LVMI and MBPS

Table 2 Stepwise multiple regression models for LVMI predictors in males

Model	*R*	*R* square	Adjusted *R* square	*p*-value
1. MBPS	0.7	0.5	0.5	< 0.001 (HS)
2. MBPS and average asleep SBP	0.7	0.5	0.5	0.04

LVMI left ventricular mass index, *MBPS* morning blood pressure surge, *SBP* systolic blood pressure

receiver operating characteristic (ROC) curve (Fig. 3) was performed to assess a cut-off value for MBPS to predict DD with the best available sensitivity and specificity. All tests of significance were two-tailed and a p-value < 0.05 was considered statistically significant, a p-value < 0.001 was considered highly statistically significant, and a p-value ≥0.05 was considered non-statistically significant.

Spearman's rank correlation showed a highly statistically significant strong positive correlation between LVMI with MBPS and average 2-h awakening SBP with correlation coefficient ($r = 0.609$), p-value <0.001, and ($r = 0.458$) p-value < 0.001 respectively (Table 1 and Fig. 1).

Stepwise multiple regression analysis generated three models explaining the variance in LVMI. The first model showed that MBPS predicts the variance in LVMI with adjusted R^2 (0.363) and standardized coefficient (beta) (0.608), p-value < 0.001. The second model showed that MBPS combined with the average asleep SBP predict the variance in LVMI with adjusted R^2 (0.404) and standardized coefficient (beta) (0.218), p-value 0.009. The third model showed that MBPS combined with the average asleep SBP and average 2-h awakening SBP predict the variance in LVMI with adjusted R^2 (0.445) and standardized coefficient (beta) (0.435), p-value 0.008. Variables excluded by the stepwise process were age, BMI, office DBP, DM duration, HbA1c, average day SBP, average day DBP, average asleep DBP, average 24-h SBP, average 24-h DBP, 2-h awakening SBP, maximum night dip SBP, and EF.

Results

This comparative cross-sectional study included 90 normotensive T2DM divided according to presence of diastolic function into two groups: *group I* (without diastolic dysfunction) 62 (68.8 %) patients and *group II* (with diastolic dysfunction) 28 (31.1 %) patients (Tables 7, 8, and 9).

Chi-square test showed no statistically significant difference between the two groups regarding gender, smoking, and dipping status with p-values 0.530, 0.634, and 0.871 respectively.

Mann-Whitney U test showed statistically significant increase of age and average 2-h awakening SBP in group II than group I (p-values = 0.023 and 0.046 respectively), and a highly statistically significant increase of MBPS in group II than group I (p-value <0.001) as shown in Table 6.

Binary logistic regression analysis revealed that age (OR 1.119, 95% confidence interval (CI) 1.101-1.239, p-value 0.031) and MBPS (OR 1.264, 95% CI 1.121-1.425, p-value <0.001) can independently predict abnormal diastolic function.

Finally, an ROC curve (Fig. 3) was performed to assess a cut-off value for MBPS showing a fair area under curve (AUC) of 0.745 at 95% CI (0.644-0.845) at p-value <0.001. The curve showed that the most appropriate cut-off value of MBPS for prediction of DD was 25.5 mmHg with sensitivity of 85.7% and specificity of 61.3%.

Table 3 Stepwise multiple regression models for LVMI predictors in females

Model	*R*	*R* square	Adjusted *R* square	*p*-value
1. MBPS	0.7	0.5	0.5	< 0.001 (HS)
2. MBPS and age	0.7	0.6	0.5	0.04

LVMI left ventricular mass index, *MBPS* morning blood pressure surge

Discussion

Increased morning rise and nighttime fall were reported to be associated with CV morbidity and mortality in both general [3, 5] and diabetic populations [6, 7]. Even slight alterations in BP pattern seem to be a risk factor for microvascular injury in T2DM [9].

The aim of this study was to find the effect of MBPS on LVH in normotensive type 2 diabetic patients.

In this study, we found that MBPS is an independent predictor for LVMI in normotensive type 2 diabetic patients independent of dipping status. This result is coincident with that of Kaneda et al. who concluded that exaggerated MBPS is associated with LVH independent of ambulatory BP level in 120 Japanese subjects [20].

Moreover, this association was also demonstrated in normotensives and well-controlled hypertensive patients. A study on normotensive subjects with clinic BPs < 140/90 mmHg and 24-h BPs < 130/80 mmHg concluded that sleep-trough MBPS was significantly correlated with increased LVMI [21]. Another study featuring well-

Table 4 Comparison between subjects with normal and abnormal diastolic function

Variable	Category	Normal function (n = 62)	Diastolic dysfunction (n = 28)	X^2 value	p-value
Gender	Males	31 (50%)	16 (57.1%)	0.3	0.5 (NS)
	Females	31 (50%)	12 (42.9%)		
Smoking	Yes	19 (30.6%)	10 (35.7%)	0.3	0.6 (NS)
	No	43 (69.3%)	18 (64.2%)		
Dipping status	Normal	29 (46.7%)	14 (50%)	0.7	0.9 (NS)
	Extreme	5 (8%)	2 (7.1%)		
	Reduced	22 (35.4%)	8 (28.5%)		
	Non-dip	6 (9.6%)	4 (14.2%)		

X^2 Chi-square test

controlled hypertensive patients with 24-h BPs < 130/80 mmHg also concluded that sleep-trough MBPS was significantly correlated with increased LVMI [22].

Also, Nakano et al. concluded that 24-h BP levels predict vascular injury in type 2 diabetic patients independent of dipping status [23]. On the other hand, Eguchi et al. observed that variability in nighttime BP was an independent predictor of CVD in type 2 diabetic patients [10]. This contradiction in the results of these studies was explained by Kaneda et al. who suggested that quality of sleeping is an important factor for the changes occurring during nighttime which can consequently affect the dipping status in different studies [20].

Our study showed a 31% prevalence of DD. In line with our data, Fontes-Carvalho et al. reported 36.6% DD in T2DM with metabolic syndrome [24].

However, the percentage of DD in this study was lower than that recorded by Yadava et al. and by Patil et al. who reported 49% and 54% respectively [25, 26]. This may be due to longer duration of T2DM in their subjects (less than 5-15 years) while in this study, the duration is ≤ 5 years, so they reported significant relation between DD and longer durations of diabetes, while there is no significant difference between patients with normal and abnormal diastolic function regarding the duration of DM in the current study.

Patil et al. showed that higher HbA1c was correlated to the prevalence of DD [26], while this study did not report significant difference in HbA1c between subjects with normal and abnormal diastolic function. However, Exiara et al. found that 63% of normotensive well-controlled type 2 diabetics (HbA1c < 6.5) had DD [27] and Yadava et al. concluded no significant association between HbA1c and DD [25]. This contradiction

Table 5 The difference between subjects with normal and abnormal diastolic function regarding personal and laboratory data

Variable	Normal diastolic function (n = 62)	Diastolic dysfunction (n = 28)	Mann-Whitney U test	p-value
Age	48.2 ± 6.1	51.1 ± 5.5	608.0	0.02 (S)
	49.00 (36-63)	51 (38-59)		
BMI	27.5 ± 2.4	27.2 ± 2.9	808.0	0.6 (NS)
	27.15 (23-32)	26.85 (23-33)		
Office SBP	116.5 ± 7.7	116.1 ± 8.2	828.0	0.7 (NS)
	120 (95-125)	117.5 (100-130)		
Office DBP	73.2 ± 6.1	73.5 ± 6.9	832.0	0.7 (NS)
	72.50 (60-80)	75 (60-85)		
DM duration	3.1 ± 1.1	3 ± 1.2766	730.0	0.2 (NS)
	3 (1-5)	3 (1-5)		
HbA1c	7.8 ± 0.7	7.8 ± 0.6	833.5	0.7 (NS)
	7.8 (6.7-8.8)	7 (6.8-8.8)		
Serum creatinine	0.9 ± 0.1	0.9 ± 0.1	855.5	0.9 (NS)
	0.9 (0.6-1.1)	0.9 (0.6-1.1)		
UACR	46.9 ± 32.0	58.2 ± 48.2	720.0	0.2 (NS)
	30 (15-164)	28 (16-196)		

BMI body mass index, *BSA* body surface area, *DM* diabetes mellitus, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *UACR* urine albumin creatinine ratio

Table 6 The difference between subjects with normal and abnormal diastolic function regarding ABPM data

Variable	Normal diastolic function (n = 62)	Diastolic dysfunction (n = 28)	Mann-Whitney U test	p-value
Age	48.2 ± 6.1 49.00 (36-63)	51.1 ± 5.6 51 (38-59)	608.0	0.02 (S)
BMI	27.5 ± 2.4 27.2 (23-32)	27.3 ± 2.9 26.9 (23-33)	808.0	0.6 (NS)
Office SBP	116.5 ± 7.7 120 (95-125)	116.07 ± 8.205 117.5 (100-130)	828.0	0.7 (NS)
Office DBP	73.2 ± 6.1 72.5 (60-80)	73.57 ± 6.920 75 (60-85)	832.0	0.7 (NS)
DM duration	3.1 ± 1.1 3 (1-5)	3 ± 1.3 3 (1-5)	730.0	0.2 (NS)
HbA1c	7.8 ± 0.7 7.8 (6.7-8.8)	7.9 ± 0.6 7 (6.8-8.8)	833.5	0.8 (NS)
Average day SBP	117.3 ± 9.2 116 (100-131)	118.8 ± 8.4 118.5 (100-133)	789.0	0.5 (NS)
Average day DBP	77.2 ± 4.3 77.5 (69-82)	77.8 ± 4.5 78.5 (71-84)	857.0	0.9 (NS)
Average asleep SBP	106.3 ± 7.3 105.5 (96-118)	107.1 ± 7.9 108 (95-117)	828.5	0.7 (NS)
Average asleep DBP	63.9 ± 3.6 64 (58-69)	63.2 ± 3.1 62 (56-69)	834.0	0.8 (NS)
Average 24-h SBP	114.5 ± 8.5 114 (100-128)	112.9 ± 8.6 113 (98-127)	779.5	0.4 (NS)
Average 24-h DBP	71.9 ± 4.1 72 (65-79)	71.2 ± 4.5 70 (63-78)	831.5	0.8 (NS)
Average 2-h awake SBP	127.1 ± 13.1 125 (106-163)	131.9 ± 9.6 132 (111-151)	639.0	0.05 (S)
Maximum night dip SBP	101.9 ± 4.6 102 (95-110)	100.7 ± 3.7 101.5 (95-108)	746.5	0.3 (NS)
MBPS	24.5 ± 11.0 21 (10-45)	34 ± 8.2 34.5 (18-49)	443.0	< .001 (HS)

BMI body mass index, *DM* diabetes mellitus, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *MBPS* morning blood pressure surge

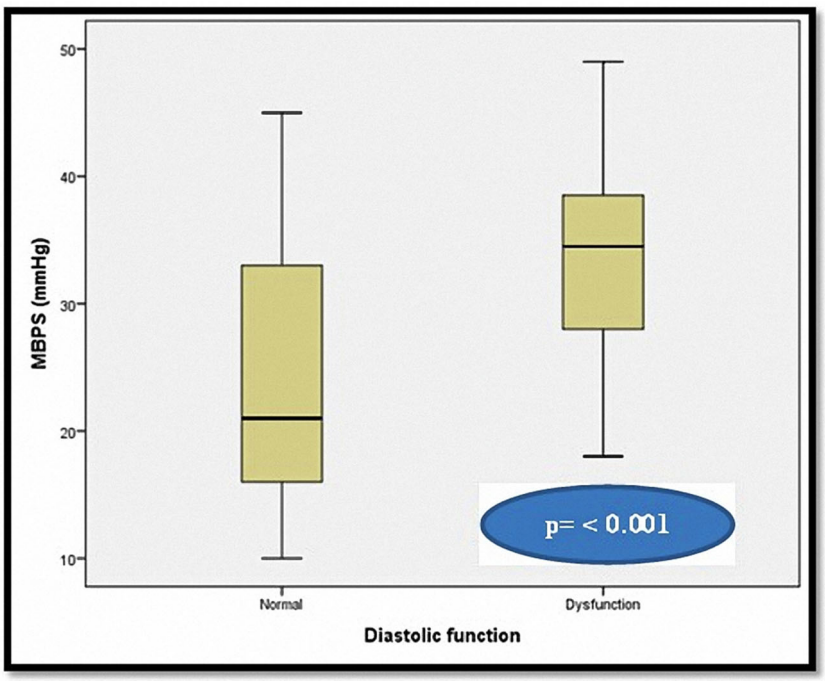

Fig. 2 Box plot showing the highly significant difference in MBPS between subjects with normal (left) and abnormal (right) diastolic function, being significantly higher in subjects with diastolic dysfunction (p value < 0.001)

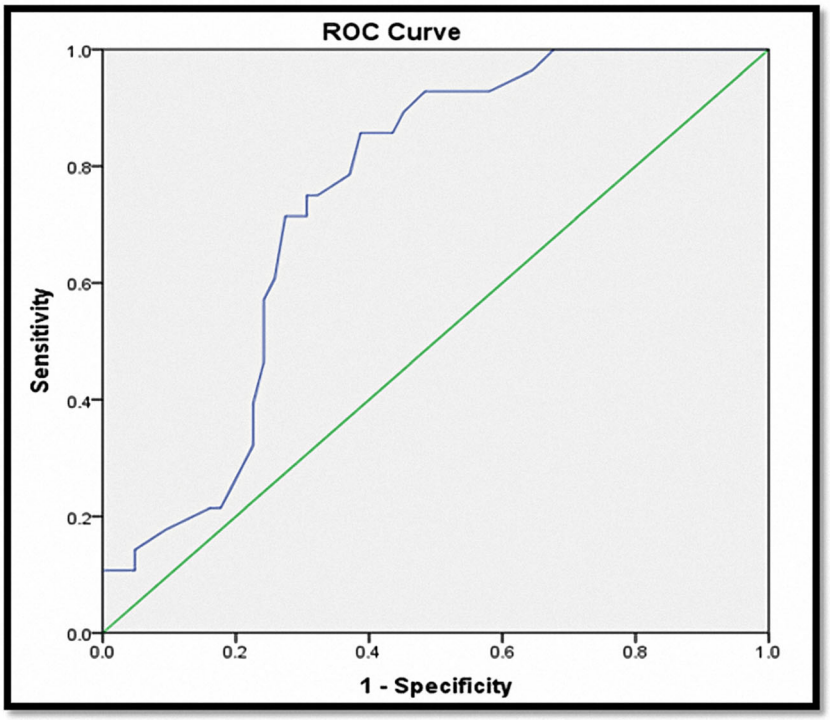

Fig. 3 ROC curve of MBPS prediction of diastolic dysfunction. ROC curve performed to assess a cut-off value for MBPS showed fair area under curve (AUC) of 0.745 at 95% CI (0.644-0.845) at *p*-value < 0.001. Coordinates of the curve showed that the most appropriate cut-off value of MBPS for prediction of diastolic dysfunction was 25.5 mmHg with sensitivity of 85.7% and specificity of 61.3%

may be explained by Fontes-Carvalho et al. who reported that diastolic function changes preceded the onset of T2DM, being principally associated with insulin resistance state and not only to persistent hyperglycemia and may be also explained by the longer T2DM duration in these studies [24].

Actually, the wide variety of results among researchers may be due to the different thresholds and methods of MBPS calculation [28]. In addition, ethnicity [29], sex, age, BMI [29, 30], hypertension [30], use of β-blockers [30, 31], a history of CVD [30, 32], duration of diabetes mellitus [29], renal dysfunction, a sedentary lifestyle [33], and socioeconomic position [32] all are factors that can affect the measured parameters. This proposes that the association between MBPS and CVD is more complex than using a single threshold and is unsurprising given that analysis of subject's continuous predictors on scale is more accurate and is less liable to error than dichotomization [28].

Table 7 Descriptive analysis for patient characteristics and relevant laboratory tests

Variable		Description
Age		49.07 ± 6.1 years
Gender	Males (%)	47 (52.2%)
	Females (%)	43 (47.8%)
BMI		27 (23-33.2) kg/m^2
BSA		1.8 ± 0.2 m^2
Smoking	Smokers (%)	29 (32.2%)
	Non-smokers (%)	61 (67.8)
DM duration		3 (1-5) years
Office SBP		116.4 ± 7.8 mmHg
Office DBP		73.3 ± 6.3 mmHg
HbA1c		7.1 (6.6-7.9) %
Serum creatinine		0.9 (0.6-1.1) mg/dL

BMI body mass index, *BSA* body surface area, *DM* diabetes mellitus, *SBP* systolic blood pressure, *DBP* diastolic blood pressure

Table 8 Descriptive analysis for ABPM and MBPS data

Variable		Description
Average day SBP		117.7 ± 8.9 mmHg
Average day DBP		77.8 ± 4.4 mmHg
Average asleep SBP		106.6 ± 7.5 mmHg
Average asleep DBP		63.9 ± 3.5 mmHg
Average 24-h SBP		114.00 ± 8.52 mmHg
Average 24-h DBP		71.8 ± 4.2 mmHg
2-h awakening SBP		128.6 ± 12.3 mmHg
Maximum night dip SBP		101.6 ± 4.4 mmHg
Dipping status	Normal (%)	43 (47.8%)
	Extreme (%)	7 (7.8%)
	Reduced (%)	30 (33.3%)
	Non-dipping (%)	10 (11.1%)
MBPS		27.5 ± 11.1 mmHg

SBP systolic blood pressure, *DBP* diastolic blood pressure, *MBPS* morning blood pressure surge

Table 9 Descriptive analysis for relevant echocardiography parameters

Variable		Description
Septal thickness		9 (7-12) mm
Posterior wall thickness		8 (7-12) mm
LVMI	Male	108 (71.4-139.8) g/m^2
	Female	87.3 (62.7-116.2) g/m^2
LVEF		61 (54-69) %
E wave velocity		68 (51-120) cm/s
A wave velocity		61.42 (35-97) cm/s
Lateral e′		13.6 (8.4-18.9) cm/s
Septal e′		10.550 (5.6-15.1) cm/s
Average septal and lateral e′		12.3 (7.1-16.5) cm/s
Average E/e′		6.028 (3.5-16.8)
TR jet velocity		2.8 (1.9-3.7) m/s
LAVI		29.5 (20.9-40.2) mL/m^2
Diastolic function	Normal (%)	62 (68.9%)
	Abnormal (%)	28 (31.1%)

LVMI left ventricular mass index, *LVEF* left ventricular ejection fraction, *TR* tricuspid regurgitation, *LAVI* left atrial volume index

Limitations of the Study

1. Small study size.
2. The use of a single day ambulatory blood pressure monitoring (ABPM) may not be sufficient to characterize the MBPS profile and clinical prognosis in individual patients, due to the low reproducibility [34], and the various factors that could increase MBPS, including aging, hypertension, glucose abnormality, alcohol intake, smoking, poor sleep quality, psychological stress, and physical stress [35, 36]
3. We recommend further studies should include information about clinical status of the employer as well as medication use, as antihypertensive or hypoglycemic drugs that could influence the results of the study.

Conclusion

Higher levels of MBPS are associated with increased LVMI and DD in normotensive type 2 diabetic patients independent of office and average ambulatory BP values and dipping status in our study.

Clinical Implication and Recommendation

Given its incremental predictive value, morning blood pressure surge associated with increased left ventricular mass index and diastolic dysfunction may have a role in reducing the progress hypertension and evident heart failure in diabetic patients; we supposed that early identification of MBPS as an early marker of subclinical hypertension and heart failure in type II DM might be of value for risk of patients with DM.

For that, it might be a significant predictor for discovery of subclinical hypertension in early stages of diabetes mellitus and can help in the prevention of heart failure development.

It is recommended to do a larger study, with long follow-up and meticulous medication history to determine the relation between DM, HTN, and medications.

Abbreviations BP, blood pressure; CV, cardiovascular; MBPS, morning blood pressure surge; LVH, left ventricular hypertrophy; T2DM, type 2 diabetes; ABPM, ambulatory blood pressure monitoring; LVMI, left ventricular mass index; ROC, receiver operating curve; DD, diastolic dysfunction; OR, odds ratio; CI, confidence interval; CVD, cardiovascular disease; ADA, American Diabetes Association; OGTT, oral glucose tolerance test; BMI, body mass index; BSA, body surface area; ECG, electrocardiogram; LVEF, left ventricular ejection fraction; A4C, apical four chambers; A2C, apical two chambers; LVH, left ventricular hypertrophy; PLAX, parasternal long axis; IVST, interventricular septum thickness; PWT, posterior wall thickness; HTN, hypertension

Availability of Data and Materials Our observational analytical study in the form of cross-sectional study data used to support the findings of this study is available from the corresponding author upon request.

Author's Contribution II drafted the article with the final revision, prepared the figures, and checked the language and grammar. AA collected the data and acquisition analysis and was the major contributor in writing the manuscript. RM put the concept and design of the work. IS analyzed and interpreted the patient data regarding MBPS in type 2 DM and critical revision of the article. All authors read and approved the final manuscript.

Declarations

Ethics Approval and Consent to Participate Our study was approved by the Zagazig University Institutional Review Board (ZU-IRB), Egypt. Trial registration: ZU-IRB#4751-11-07-2018 registered 11 July 2018, email: IRB_123@medicine.zu.edu.eg. Informed and written consent was obtained from all participants in our research.

Consent for Publication Not applicable.

Competing Interests The authors declare no competing interests.

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