Impact of abnormal circadian blood pressure profile on left atrial function assessed by 2D speckle tracking echocardiography and its effect on the functional capacity of hypertensive patients

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Background The left atrial (LA) function has an important role in hypertension as it is strongly predictive of adverse cardiac events and death, assessment of the circadian blood pressure (BP) profile is more predictive than assessment of the office BP reading in estimating the cardiovascular risk. The 2D speckle tracking echocardiography (2D-STE) provides more insight into early hypertension-induced LA dysfunction.

Aim The aim of this work is to assess the impact of abnormal circadian BP profile on LA function using 2D-STE and its effect on functional capacity in hypertensive patient with preserved ejection fraction assessed by conventional echocardiography.

Patients and methods This work included 60 consecutive hypertensive patients with preserved ejection fraction by conventional echocardiography [mean age 48±5 years and body surface area (BSA) 1.9±1.1] they were classified into two groups according to the data derived from 24 h ambulatory BP, group 1: dipper group that included 28 patients (mean age 48±8 years and BSA 1.9±0.1) and group 2: nondipper group that included 32 patients (mean age 50±6 years and BSA 1.9±0.09). All patients were evaluated by comprehensive 2D and Doppler echocardiographic techniques, TDI and 2D-STE, ambulatory BP, and stress. Myocardial perfusion imaging using treadmill exercise test was done to exclude coronary artery disease (CAD) and to assess the functional capacity.

Results The results showed a statistically high significant decrease in the average peak left atrial global longitudinal strain in group 2 (the nondipper group) (group 1=26±4 vs. 20.4±3.5 in group 2, $P<0.00$), and a statistically significant decrease in the functional capacity parameter using treadmill metabolic equivalents (METs) in group 2 (group 1=7.3±1.4 vs. 6.2±0.8 in group 2, $P<0.01$) in comparison with group 1. Also we found a significant positive correlation between the average peak atrial longitudinal strain and the functional capacity assessed by treadmill stress test ($r=0.424$, $P=0.05$). There were no other significant differences between the two groups with respect to other LA parameters by conventional echo Doppler, TDI, and 2D-STE.

Conclusion Abnormal circadian BP profile add more deleterious effect on LA function in hypertensive patients as detected by 2D-STE that denotes more decrease in functional capacity and worse cardiac events.

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Introduction

Hypertension (HTN) is a heterogeneous multifactorial disorder. It is a major health problem throughout the world because of its high prevalence and its association with increased risk of cardiovascular disease. Inadequate diagnosis, decreased orientation, and suboptimal control of blood pressure (BP) in diagnosed patients is a major driver of cardiovascular morbidity and mortality with an increased burden to health care resources [1].

Assessing the circadian BP profile is more predictive than assessing the office BP readings in estimating the cardiovascular risk [2].

Nondippers are known to have more increased levels of cardiac natriuretic hormones that are correlated with increased left ventricular (LV) and left atrial (LA) abnormalities in treated hypertensive patients [3].

The LA structural remodeling and/or functional impairment might play a part in the pathogenesis and development of heart failure with or without preserved ejection fraction (EF) [4]. The 2D speckle tracking echocardiography (2D-STE) enables detection of early LA dysfunction before LA morphological changes. LA dysfunction determined by 2D-STE can provide additional prognostic information to conventional echocardiographic parameters in patients with cardiovascular disease. Hence 2D-STE appears to be a promising technique for diagnosis and therapeutic decision making [5]. The 2D-STE strain imaging of the LA might be useful in the assessment of target organ

damage and in the initiation of antihypertensive treatment on several conditions. Also there is a strong association between exercise intolerance by exercise testing and LA function. Exercise intolerance and impaired LA function are powerful predictors of future cardiovascular events [6].

Aim

The aim of this work was to assess the impact of abnormal circadian BP profile on LA function by 2D-STE and its effect on functional capacity in hypertensive patient with preserved EF by conventional echocardiography.

Patients and methods

Study cohort

Our study included 60 hypertensive patients (18 men and 42 women) the mean age was 48±5 years, who attended Al-Azhar University Hospital, Cairo, Egypt (most patients in Al-Azhar University Hospital were females); during the period from August 2016 to June 2017 were on antihypertensive medications. The studied patients were divided into two groups according to their ambulatory BP results:

(1) Group I: included 32 patients with nondipper HTN.
(2) Group II: included 28 patients with dipper HTN.

Oral consent had been obtained from the patients and the study was approved by FMG ethical committee. Excluded from this study were patients who had one or more of the following criteria:

LV-EF less than 55%, rhythm other than sinus rhythm, moderate to severe valvular heart disease, any evidence of myocardial ischemia either detected by resting LV segmental wall motion abnormality during conventional echocardiographic examination or by stress myocardial effusion [myocardial perfusion imaging (MPI)], also diabetic patients, patients with cerebrovascular, or peripheral vascular disease were excluded from this study.

Methods

All patients included in this study were subjected to the following:

(1) Thorough medical history taking and clinical examination, history of HTN, duration of antihypertensive medications used and the details of the medications, baseline measurements of weight, height were recorded, and the body surface area (BSA) was calculated according to the Mosteller formula:([height (cm)×weight (kg)]/3600)½.
(2) Office BP measurement methodology:
BP was measured during office visit according to the recommendation of the American heart association, and also by using 24-h ambulatory BP monitoring. Patients were considered to be hypertensive if systolic blood pressure (SBP) more than 139 mmHg and diastolic blood pressure (DBP) more than 89 mmHg according to the WHO [7].
(3) Standard 12 leads surface ECG: to assess any abnormal findings.
(4) Exercise MPI:
Exercise ECG:
It is used to estimate the functional capacity of the individuals and for assessment of myocardial ischemia, patients with positive test were excluded from this study. The test was carried out using (GE T 2100 treadmill, GE Medical Systems Information Technologies Inc., 8200 West Tower Avenue, Milwaukee, WI, USA) Bruce protocol that involved 3 min stages to achieve a steady state before workload is increased. Workload was estimated in METs, which reflect the resting volume of oxygen consumption per minute, for a 70 kg 40-year-old man MET is equivalent to 3.5 ml/kg/min [8].
MPI
All subjects underwent a gated-SPECT MPI according to a two-day protocol. Supine images were acquired with a dual-head (Philips) camera with a low-energy, high-resolution collimators. All radionuclide images and associated data were processed according to the standard protocols. The ^{99m}Tc sesta MIBI was used in all patients (the routine tracer used in our laboratory). Myocardial perfusion was calculated as the relative percent tracer uptake in each of the 17 segments according to the standard model.
Experienced nuclear cardiology specialists used this data quantitatively and qualitatively to interpret each MPI study. Readers assigned a score (0–4) to each segment; (0) for normal uptake; (1, 2, and 3) for mild, moderate, and severe reduction of uptake respectively; and (4) for absent uptake. Sum stress score (SSS), sum rest score (SRS), and sum difference score (SDS) were reported. Other markers of high risk perfusion scans were reported separately (e.g. increased LHR, TID, abnormal regional, and global wall motion abnormalities). Negative SPECT study was defined as SSS less than 4 and SDS=0 [9].
(5) The 24-h ambulatory BP monitoring:

The machine used was TONOPORTO GE with software cardiosoft version. The cuff used was applied on the nondominant arm and its size was selected according to the measured arm circumference standards. Every patient was instructed to avoid strenuous physical activities and to keep his/her forearm extended during each ABPM measurement. The protocol of BP measurements were taken at every 20-min interval during daytime (i.e. between 7 a.m. and10 p.m.) and at 1-h period during night time (between 10 p.m. and 07 a.m.). If 20% or more of the measurements could not be taken, those patients were excluded from the study or the procedure was repeated. The patients were instructed to perform their normal daily activities during the day and go to bed not later than 10:00 p.m. They were also instructed to stay in bed until 6: 00 a.m. A test reading was measured after the patient had wearied the ambulatory BP to guarantee that there was no consistent difference of greater than or equal to 10 mmHg in the measured BP between ABPM devices and manual standard sphygmomanometer. Patients were given a diary to report his other activity and the time of wake up and sleep.

The following parameters of the ambulatory BP monitoring were obtained:

Average daytime/nighttime

The patients were considered as having normal awake BP if the corresponding value was less than 135 mmHg SBP and less than 85 mmHg DBP. Normal sleep BP was considered less than 120/70 mmHg. The overall normal 24 h, BP was less than 130/80 mmHg [10].

Blood pressure variance

Patients with both SBP and DBP decrease of 10% or more during night time were accepted as presenting the dipper status, whereas patients were classified as nondipper if the BP decrease during the night was less than 10%, either of a systolic or diastolic nature. Nocturnal BP decrease (the so-called 'dipping' phenomenon) was calculated by using the following equation [11]:

$$\frac{(\text{Awake mean BP} - \text{sleep mean BP})}{\text{Awake mean BP}} \times 100.$$

Transthoracic echocardiography

Conventional transthoracic echo Doppler examination was performed for all patients in both supine and left lateral position using Vivid-7GE system with tissue Doppler imaging capability. All cases were examined using multi frequency (2.5–3.5 MHz) matrix probe M3S with simultaneous ECG physiosignal displayed with all recorded echo images and loops.

All parameters were taken on the basis of the American Society of Echocardiography Standards [12].

Conventional echocardiography

Transthoracic echocardiography data was obtained from standard views (parasternal long, short axis, and apical views) by using different modalities (2D, M-mode, and Doppler study) for the assessment of the following parameters:

(1) Assessment of LV Function by M-mode and 2D echocardiography:
   (a) Left ventricular end diastolic dimension (LVEDD), left ventricular end systolic dimension (LVESD), interventricular septal diameter (IVSD), left ventricular posterior wall diameter (LVPWD), left ventricular EF, left ventricular fractional shortening (FS).
   (b) 2D-echocardiography was used for assessment of segmental wall motion abnormalities, evaluation of any associated valvular lesions, and estimation of LV EF using modified Simpson's method.
   (c) Left ventricular mass and mass index were calculated using linear measurements derived from

   2D targeted M-mode by application of modified Devereauxs formula:

$$0.8 \times (1.04 \times (\text{LVEDd} + \text{PWTd} + \text{SWTd})3 - (\text{LVEDd})3) + 0.6/\text{body surface area}.$$

   Normal range (49–115g/m^2 in males, 43–95g/m^2 in females) [13].

(2) Assessment of left atrium (LA):
   (a) LA dimension was estimated in parasternal long axis view from 2D targeted M-mode, normal range 2.0–4.0 cm.
   (b) LA volume was estimated by the biplane area length method, using apical 4 and 2 chamber views.

   Maximal LA volume was measured just before mitral valve opening at end systole (vol_{max}). Minimal LA volume was measured at end diastole when mitral valve closed (vol_{min}). Precontractile LA volume was measured at P wave onset on ECG just before atrial contraction, if in sinus rhythm [pre-A LA volume (vol-P)].

(c) LA volume index was calculated as LA volume divided by BSA [14]. Indexed to BSA, the normal maximal LA volume is 22±5–6 ml/m^2; minimal LA volume is 11±4 ml/m^2; and pre-A LA volume is 15±5 ml/m^2 [15].

(d) LA functions were calculated from the following formulae [16]:

$$\text{LA reservoir function was calculated as LA expansion} = \frac{(\text{LA max} - \text{LA min})}{\text{LA min}} \times 100,$$

$$\text{LA conduit function as LA passive emptying fraction} = \frac{(\text{LA max} - \text{LA pre A})}{\text{LA max}} \times 100,$$

$$\text{LA contractile function as LA active emptying fraction} = \frac{(\text{LA pre A} - \text{LA min})}{\text{LA pre A}100\%}.$$

(e) LA function examination using Tissue Doppler including the following parameters:
For the TDI velocity and strain we used the two apical views (4, 2) chambers views. For data acquisition, three complete cardiac cycles were collected and stored in a cine-loop format. The image sector width was set as narrow as possible to allow frame rate acquisition greater than 80 frames/s. Special attentions was paid to the color Doppler velocity range setting to avoid any aliasing within the image.The TDI sample volume was placed at mid atrial segment of interest, usually about 2 mm for measuring velocity and preferably not more than 12 mm of length for strain because of its thin-walled structure [17]. Offline analysis of the digitally stored loops was done by trace profile displacement of the velocity and strain to obtain, the average peak LA systolic myocardial velocity (SMV), the average peak LA early diastolic myocardial velocity (Em velocity), the average peak LA late diastolic myocardial velocity (Am velocity) and average LA longitudinal strain measured at end systole, normal range (42±4) [18].

(f) The 2D STE:
LA longitudinal strain was assessed using 2D speckle-tracking analysis with QRS onset as the reference point, applying a commercially available LV strain software package to the left atrium (GE EchoPAC Software v.110.1.2, GE Healthcare, Milwaukee, WI, USA). The region of interest was adjusted to include the LA myocardium in both 4 and 2 chambers views. The left atrium manual correction was performed to optimize tracking results if needed.
QRS-timed analysis to obtain peak atrial longitudinal strain (PALS), which is the first positive peak measured at the end of the reservoir phase, and peak atrial contraction strain (PACS), measured just before the start of the active atrial contractile phase, which is the second positive peak are calculated by averaging values observed in all LA segments (global PALS and PACS) [19]. The analysis of the LA strain curve was done using QRS onset as the reference point to measure:
Peak atrial strain during ventricular systole, (εS) measured just before mitral valve opening and it is surrogate of the reservoir function and late peak strain just before the active atrial contractile phase (εCT) begins, at the onset of the P wave on the electrocardiogram, surrogate of the contractile functionThe average LA strain was obtained after averaging the six segments in each view reference values of global, 4-chamber, and 2-chamber PALS [19].

Statistical analysis of data

Numerical variable was expressed as mean and SD, the following statistical tests were used for analysis of data by SPSS version 19 (SPSS Inc., Chicago, Illinois, USA):

(1) Independent *t*-test for testing statistical significant difference between means of the two groups in each classification.
(2) Pearson's correlation test with the determination of the correlation coefficient (*r*) to test a positive or negative relationship between two variables.

P value less than 0.05 was considered statistically significant.

Results

In this study; 60 hypertensive patients (18 men and 42 women) their mean age (48±5 years)with mean disease duration (5.4±1.8 years) on medical treatment, their left ventricular function was proved to be preserved as measured by conventional echocardiography (63.2 ±4.2) they were classified into two groups dippers (group 1, *n*=18) and nondippers (group 2, *n*=32). There were no significant differences regarding the demographic data between the two groups and clinic office BP measurements (mean value of the office SBP reading was 151.6±10.8 and the DBP reading was 92.3 ±6.2).

A significant difference was found regarding the functional capacity parameters measured in METs between the studied groups ($P<0.01$) being lower in the nondippers group.

Medications used by patients are shown in Table 1 except for beta blockers; there were no significant differences regarding antihypertensive drugs distribution. Hence, 24 (40%) patients were on monotherapy, 34 (57%) were on dual antihypertensive therapy, and only two (3%) patients were on triple antihypertensive therapy as shows in Table 1

Comparison between the two groups is demonstrated in Table 2, Figs 1, 2, which showed comparable results regarding the conventional echo parameters of the LV in the whole cohort except the LV mass index that was higher in nondippers than dippers (105.9±26.4 vs., 78.3±11.8, respectively) with a highly significant difference ($P<0.01$). Similarly; the conventional echo parameters of the LA volumes showed no significant difference between the two groups but we could observe nonsignificant increased LA reservoir function in nondipper group and significantly decreased LA conduit function and highly significant increase in the active LA contractile function of the same group. Tissue Doppler study showed significant difference between the two groups regarding the E velocity ($P<0.01$).

Table 1 Demographic data of the studied groups (Dippers and nondippers)

	Dippers (*N*=28)	Nondippers (*N*=32)	*P* value
Age	49.36±6.416	49.44±8.310	0.977
Men [*n* (%)]	8 (28.57)	10 (31.25)	0.596
BSA	1.86±0.11	1.92±0.125	0.174
Duration	5.64±1.82	5.19±1.72	0.488
METs	7.3±1.4	6.2±0.8	< 0.011
Clinic SBP	155.71±9.37	152.50±9.31	0.355
Clinic DBP	92.14±5.79	92.50±6.83	0.982
Ambulatory BP			
Systolic daytime	145.86±13.74	145.87±11.21	0.997
Diastolic daytime	87.57±5.87	87.62±6.66	0.982
Systolic nocturnal	135.36±13.34	130.31±8.71	0.225
Diastolic nocturnal	83.36±10.55	83.87±8.64	0.884
Medications [*n* (%)]			
ACE inhibitors	20 (71.43)	24 (75)	0.574
BBs	16 (57.14)	6 (18.75)	0.035
CCBs	2 (7.14)	2 (6.25)	0.724
Diuretics	8 (28.57)	18 (56.25)	0.123

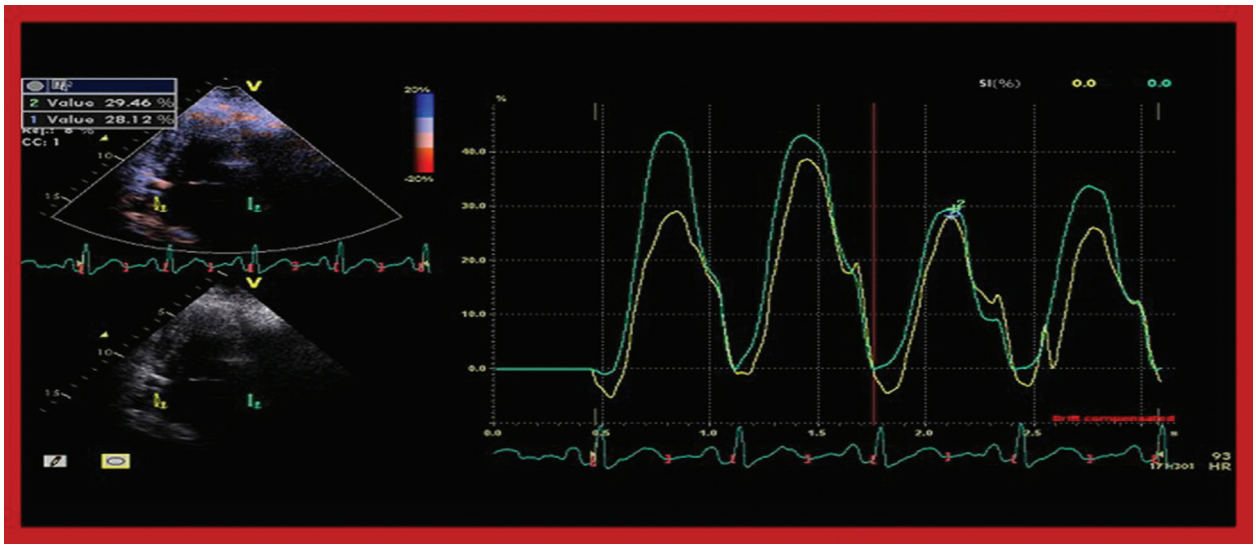
ACEI, angiotensen converting enzyme inhibitors; BBs, beta blockers; BP, blood pressure; BSA, body surface area; CCB, calcium channel blockers; DBP, diastolic blood pressure; SBP, systolic blood pressure.

Table 2 Echocardiographic data in the studied groups

	Dippers (*N*=28)	Nondippers (*N*=32)	*P* value
LV			
LVEDD (cm)	50.2±3.5	50.4±4.8	NS
LVESD (cm)	31±3	30.4±3.6	NS
LV mass index	78.3±11.8	105.9±26.4	<0.001
EF by M-mode	67.5±4.5	69.3±5.6	NS
EF by 2D	63±4.4	63.3±4.1	NS
LV Doppler echo			
E velocity	73±12.4	68.3±14.8	NS
A velocity	72.8±9	78.2±18.9	NS
E/A ratio	0.98±0.20	0.86±0.36	NS
E velocity	73±12.4	68.3±14.8	NS
DT	229.4±49.6	226.9±30.9	NS
LA			
LA AP (cm)	36±2	37.5±2	NS
LA max volume	26.05±6.9	27.5±6.7	NS
LA min volume	10.4±4.2	10.3±4.7	NS
LA pre cont. volume	16.7±5.1	19.7±6.9	NS
LA reservoir function	175±96	203±114	NS
LA conduit function	36±17.3	27.6±15.8	0.05
LA contractile function	38.9±14.3	46.4±15.4	0.00
LA strain			
Average S	5.7±1.4	5.5±1.1	NS
Average E	−6.8±1.7	−5.4±1.6	0.00
Average A	−7.8±0.8	−7.6±1.8	NS
Average strain of LA	18.8±7.1	19.8±12.5	NS
Average longitudinal strain	26±4	20.4±3.5	0.00
Average atrial contractile strain	10±3	9.5±2.4	NS

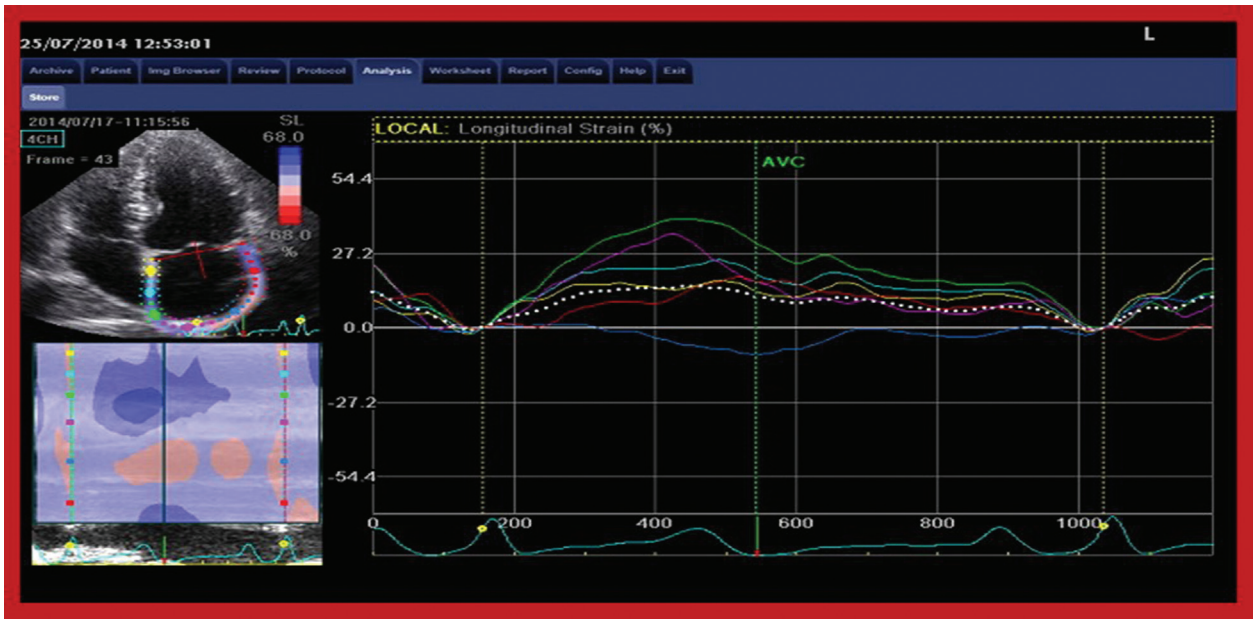
EF, ejection fraction; LA, left atrium; LV, left ventricle; LVEDD, left ventricular end diastolic dimension; LVESD, left ventricular end systolic dimension.

Figure 1



Offline strain analysis for assessment of left atrial function at anterior and inferior wall shows peak systolic strain.

Figure 2



Offline analysis of 2D speckle tracking echocardiography of left atrial at apical four chambers view.

Strain study by 2D speckle tracking echo modality showed an impaired average longitudinal strain in the nondippers group in comparison to the dippers group (20.4±3.5 vs. 26±4, respectively) with a high significant difference ($P<0.01$). Both groups were similar in terms of other LA 2D strain parameters.

In the studied nondipper patients; two variables were found to be related to PALS; the LV mass index that showed a strongly negative correlation with PALS ($r=-0.612$, $P<0.00$), it was found to be the only independent predictor for PALS in this group (odds ratio=0.111, 95% confidence interval=0.023–0.198), and METs value, which is the measurement for the functional capacity of those patients assessed by treadmill stress test and had a positive correlation with PALS ($r=-0.424$, $P<0.05$), as shown in Table 3 and Fig. 3.

Discussion

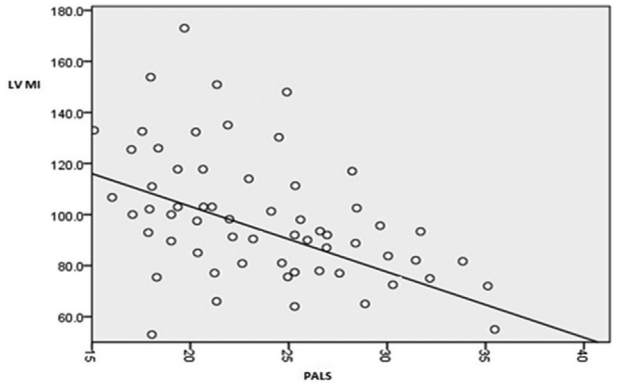
Nondipper BP pattern is defined as BP decrease in night time, is less than 10%. Studies have shown that nondipper BP pattern may be associated with increased

Table 3 Factors correlated with the peak atrial longitudinal strain

PALS	*r*	*P* value
LV mass index	–0.612	<0.00
METs	0.424	<0.05

PALS, peak atrial longitudinal strain; LV, left ventricle.

Figure 3



Showed positive correlation between peak atrial longitudinal strain and left ventricular mass index.

left ventricular mass, therefore it carries a higher risk for cardiovascular events [20].

Numerous studies have shown an increased LV mass index impaired diastolic function, and higher diastolic LV filling pressure in nondippers than in dippers. [21–23]. In this study, we showed increased LV MI in nondippers too. However; Cesare *et al.* [24] claimed that in treated essential hypertensive with or without BP control the extent of nocturnal BP decrease is not associated with an increase in LV mass or LVH prevalence.

Previous studies had confirmed the strong correlation between the LA function and the functional capacity, and stated the LA function as an important determinant of exercise capacity in hypertensive patients. Moreover; exercise intolerance and impaired LA function are powerful predictors of future cardiovascular events according to Arruda *et al.* [25].

In our study, the functional capacity obtained from treadmill exercise ECG (METs) was 6.8±1 and the calculated % predicted METs was 93%±0.01 while Kenya *et al.* [26] who studied 486 patients (male=306) with preserved LVEF and negative exercise echocardiography for ischemia found that METs=9.26±2.5 and % predicted METs=105±24, which were different from our results. But they agreed with our results in confirming a significant positive correlation between the functional capacity (METs) and the LA systolic function assessed by 2D-STE strain (PALS) ($P<0.05$), also they reported significant positive correlation between METs and the LA volume, the E/Em which we did not find in our work, these differences could be attributed to the small sample size we used, different sex distribution, and demographic data of our patients [26].

Also our results showed highly significant decrease in the functional capacity (METs) in the nondipper group versus dipper group ($P=0.007$).

During early diastole, the passive emptying volume reduces on account of increased LV stiffness and deteriorated diastolic relaxation. The impairment of LA passive emptying volume also contributes to a larger residual LA volume before its active contraction. The increased volume of passive LA emptying indicates a larger residual volume of LA before its active pumping.

The increase in presystolic LA volume and fiber length results in augmented LA contraction forces (Frank–Starling mechanism). LA systolic functions play a pivotal role during LV filling as suggested by the increased LA active emptying volume and emptying fraction in nondipper hypertensive. Our results agreed with Necip *et al.* [27]; who could clarify an increased LA systolic function in 52 nondipper hypertensive when compared to 40 dipping hypertensive that was confirmed by real time 3D echocardiography (RT3-DE) measurements, comparable results could be obtained from our nondipper patients who had an impaired LA conduit function as a result of impaired LV stiffness, with consequent increased reservoir function and subsequent significant increased LA active emptying contractile function but without any significant difference between the studied groups with respect to the LA volumes [27].Assessment of global LA longitudinal strain by 2D-STE in our study showed paramount high significant decrease in nondipper patients compared to dipper patients(P =0.00) that was consistent to what have been reported by Goksel *et al.* [28] who studied 78 treated hypertensive patients divided into dipper and nondipper and reported the same results.

Our study showed nonsignificant increase in LA strain by TDI in nondipper hypertensive patients, not consistent to what have been reported by Chan *et al.* [29] who studied 40 nontreated hypertensive patients divided into dipper and nondipper and found significant increase in LA strain by TDI in nondipper hypertensive patients, this may be due to different patients selection in both the studies.

Also our study showed nonsignificant decrease in LA systolic velocity and nonsignificant increased E/Em in nondipper patients that was discordant with Goksel *et al.* [28] who showed significant decrease in LA systolic velocity and significant increased E/Em in nondipper patients.

Our results showed that nonsignificant increase in LA volumes in non-dipper patients, which is discordant with the results of Goksel *et al.* [28] and Chan *et al.* [29] who reported that the LA volumes were significantly increased in nondipper patients. This may be explained by the small number of the patients in our study

This result was consistent with the results of Noha [30] who found decrease in PALS in hypertensive patients with LVH. However, she studied large number of 150 hypertensive patients with LVH and 50 patients without LVH but did not compare them.

Finally, the global peak LA longitudinal strain by 2D-STE might be used as an early marker of end organ damage in hypertensive patients, especially in the nondipper hypertensive patients and in those with increased LV mass index, also the LA longitudinal strain could be used as a good predictor of the functional capacity in hypertensive patients.

Clinical implication

HTN is associated with impaired LA function as assessed by STE strain imaging technique, even before LA enlargement. Close BP monitoring is of great importance for detection of nondipper hypertensive patients who are at increased risk of myocardial remolding. The global peak LA longitudinal strain assessed by 2D-STE might be used as an early marker of end organ damage in hypertensive patients especially in the nondipper HTN and in those with increased LV mass index, it could also be used as a good predictor of the functional capacity in hypertensive patients.

Limitations

The study was applied on a relatively small number of patients because the challenging selection of patients in absence of co-morbidities of cardiac history.

Conclusion

The current study on the treated hypertensive patients with different clinic BP control profiles indicate that the lack of nocturnal decline in BP is associated with the deleterious effect on LA function in hypertensive patients as detected by 2D-STE that lead to more decrease in functional capacity and worse cardiac events.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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