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Clinical Investigation

Postpartum Detection of Diastolic Dysfunction and Nondipping Blood Pressure Profile in Women With Preeclampsia

Marwa Sayed, MD[1]; Mariam Rashed, MSc[1]; Ahmed M. Abbas, MD[2]; Amr Youssef, MD[1]; Mohamed Abdel Ghany, MD[1]

[1]Department of Cardiology, Faculty of Medicine, Assiut University, Assiut, Egypt
[2]Department of Obstetrics and Gynecology, Faculty of Medicine, Assiut University, Assiut, Egypt

Abstract

Background: Left ventricular diastolic dysfunction and nocturnal "nondipping" of blood pressure detected via ambulatory blood pressure monitoring are predictors of increased cardiovascular morbidity.

Methods: A prospective cohort study including normotensive women with a history of preeclampsia in their current pregnancy was conducted. All cases were subjected to 24-hour ambulatory blood pressure monitoring and 2-dimensional transthoracic echocardiography 3 months after delivery.

Results: This study included 128 women with a mean (SD) age of 28.6 (5.1) years and a mean (SD) basal blood pressure of 123.1 (6.4)/74.6 (5.9) mm Hg. Among the participants, 90 (70.3%) exhibited an ambulatory blood pressure monitoring profile illustrating nocturnal blood pressure "dipping" (the mean night to day time blood pressure ratio ≤ 0.9), whereas 38 (29.7%) were nondippers. Diastolic dysfunction (impaired left ventricular relaxation) was present in 28 nondippers (73.7%), whereas none of the dippers exhibited diastolic dysfunction. Women with severe preeclampsia were more frequently nondippers (35.5% vs 24.2%; $P = .02$) and experienced diastolic dysfunction (29% vs 15%; $P = .01$) than were those with mild preeclampsia. Severe preeclampsia (odds ratio [OR], 1.08; 95% CI, 1.05-10.56; $P < .001$) and history of recurrent preeclampsia (OR, 1.36; 95% CI, 1.3-4.26; $P \leq .001$) were significant predictors for nondipping status and diastolic dysfunction (OR, 1.55; 95% CI, 1.1-2.2; and OR, 1.23; 95% CI, 1.2-2.2, respectively; $P < .05$).

Conclusion: Women with a history of preeclampsia were at higher risk for developing late cardiovascular events. The severity and recurrence of preeclampsia were significant predictors of both nondipping profile and diastolic dysfunction.

Keywords: Pre-eclampsia; blood pressure monitoring, ambulatory; echocardiography; ventricular dysfunction

Introduction

Preeclampsia (PE) is a disorder characterized by new-onset hypertension and proteinuria during pregnancy, complicating 2% to 8% of all pregnancies.[1]

Excessive inflammatory reaction and endothelial damage, accompanied by a wide range of multiorgan dysfunctions, are the main manifestations of PE.[2] Multiple studies have been conducted to investigate the influence of PE on maternal cardiovascular (CV) function and structure. Some reports have shown that the impact of PE recovered a few weeks postpartum,[3] although women with a history of PE are still at increased risk of future CV events,[4] which suggests the need for preventive strategies among these women.[5,6]

Better targeting of the implementation of these strategies might be achieved by improving the process of identifying the women at highest risk. A few studies have reported cardiac remodeling and nocturnal hypertension in some patients with previous PE, which are correlated with CV morbidity and mortality.[4,7-11]

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Corresponding author: Marwa Sayed, MD, Department of Cardiovascular Medicine, Assiut University, PO Box 71515, Assiut, Egypt (marwakh_aya@yahoo.com)

Tissue Doppler imaging (TDI) can better detect left ventricular (LV) myocardial remodeling than can conventional Doppler examination; moreover, TDI has been strongly correlated with invasive indices of myocardial filling pressures and long-term CV risk.[12] Ambulatory blood pressure monitoring (ABPM) has been closely correlated with target organ damage and future CV events.[13-16] Therefore, the current study assessed the cardiac function and blood pressure (BP) pattern in postpartum women with PE to identify possible predictors of future CV risk at 12 weeks postpartum.

Abbreviations and Acronyms

2D	2-dimensional
ABPM	ambulatory blood pressure monitoring
Amax	maximum atrial filling
AUC	area under the curve
BP	blood pressure
CV	cardiovascular
DBP	diastolic blood pressure
EF	ejection fraction
Emax	maximum elastance
LV	left ventricle
OR	odds ratio
PE	preeclampsia
SBP	systolic blood pressure
TDI	tissue Doppler imaging

Patients and Methods

Study Type, Setting, and Duration

This prospective cohort study was performed at a tertiary university hospital between May 2017 and June 2019. The institutional review board approved the study protocol. The study was conducted in accordance with the ethical guidelines for human studies. After receiving an explanation regarding the nature of the procedure, all study participants provided informed consent.

Study Participants

Women between ages 18 and 40 years with a history of PE during their current pregnancy were included in the study. All women were enrolled within their first week postpartum. We excluded patients who had chronic hypertension, were currently pregnant, or had chronic kidney insufficiency with a glomerular filtration rate less than 60 mL/min/1.73 m^2 for 3 months or more, irrespective of the cause. Aside from patients with any disease requiring anti-inflammatory medication, those with diabetes or any other endocrine disorders, such as hyperthyroidism or any associated severe comorbidities or CV risk factors (eg, family history of CV diseases), and women with obesity (body mass index ≥35) were excluded. A full history of each patient was taken, and full physical examinations were performed. Severe PE was defined as the presence of any of the following parameters: (1) markedly elevated BP measurements (≥160 mm Hg systolic BP [SBP] or ≥110 mm Hg diastolic BP [DBP]) measured at least 6 hours while the patient is relaxed and lying on a bed, (2) proteinuria (≥5 g/24 h or ≥3+ on 2 random samples taken 4 hours apart); or (3) detection of manifestations of the end-organ disease oliguria (<500 mL in 24 hours) after 20 weeks' gestation associated with multiorgan involvement.[17]

Ambulatory Blood Pressure Monitoring

Mean SBP and DBP, mean arterial pressure, and BP load values were obtained for a full 24 hours via ABPM. Dipping status was defined as the percentage of reduction of nocturnal BP as recommended by the European Society of Hypertension practice guidelines for ABPM.[18]

Accordingly, those with normal dipping status were those whose nocturnal BP shows at least a 10% decline compared with their daytime BP (mean nighttime to daytime BP ratio ≤0.9); nondippers were those whose nocturnal BP declines less than 10% compared with the level of their daytime BP (mean nighttime to daytime BP ratio >0.9); extreme dippers are those whose nocturnal BP has an exaggerated decrease of BP that is greater than 15% compared with their daytime BP; reversed dippers are those whose average nighttime BP is higher than their average daytime BP. The night to day ratio was calculated by expressing the mean nighttime DBP as a percentage of the mean daytime DBP.[19]

Two-Dimensional Echocardiography

Transthoracic 2-dimensional (2D) echocardiography was performed on all participants using a VIVID S5 transducer (GE Medical Systems). Standard views were acquired in the left lateral decubitus position.

Left ventricle end-systolic and end-diastolic volumes were recorded in the apical 4- and 2-chamber views to calculate ejection fraction (EF) derived from Simpson's modified biplane method. The M-mode was applied to assess

LV cavity dimensions, wall motion, and thickness at the level of the papillary muscle in the parasternal short-axis view. The LV internal dimension measurements in diastole included interventricular septum, posterior wall, LV end-diastole, and LV end-systole dimension. Left ventricular diastolic parameters, including maximum elastance (Emax), maximum atrial filling (Amax), E/A ratio, E/e′ ratio, deceleration time, and isovolumetric relaxation time, were recorded using pulsed-wave Doppler and TDI for the transmittal inflow in the apical 4-chamber view.

Diastolic function was estimated according to the recommendations of the European Association of Echocardiography and the American Society of Echocardiography. Left ventricular diastolic dysfunction is defined as impaired LV relaxation with or without impaired restoring forces (and early diastolic suction), with higher LV chamber stiffness, which increase cardiac filling pressures and decrease the mitral E/A ratio and e′ velocity (pulsed-wave velocity across lateral or septal mitral annulus).[19]

Based on clinical data, the patients were divided according to PE severity (mild, moderate, and severe PE) and BP profile (dippers and nondippers).

Statistical Analysis

All statistical analyses were performed using the SPSS Statistics version 20 (IBM). The Shapiro–Wilk test was used to assess the normality of continuous data. All continuous variables in the study were normally distributed; thus, they are expressed as mean (SD) and compared with a Student *t* test. Nominal data were expressed as frequency (percentage). The χ^2 test was used to compare nominal data. Multivariate logistic regression analysis was used to identify the predictors of nondipping and diastolic dysfunction in the study cohort. The diagnostic accuracy of the initial SBP and DBP to identify nondippers and those with diastolic dysfunction was determined using receiver operating characteristic curve analysis. The level of confidence was 95%, and $P < .05$ was considered significant.

Results

This study initially surveyed 500 patients with PE who were admitted while the study was conducted. Only 186 of them agreed to participate in this study. Moreover, 52 of these patients were excluded because of a previous diagnosis of hypertension, and 6 others were excluded because of rheumatic heart disease with significant valve lesions. After obtaining Assiut University Review Board approval, 128 women who fulfilled the inclusion criteria were enrolled. Based on 24-hour ABPM while 3 months postpartum, 90 of 128 (70.3%) women studied were dippers, whereas only 38 (29.7%) were nondippers.

Baseline Patient Characteristics

The baseline characteristics of the studied patients are shown in Table I. Dippers and nondippers had mean (SD) ages of 27.97 (5.18) and 29.26 (5.10) years, re-

TABLE I. Baseline Characteristics of Patients (N = 128)

Characteristics	Dippers (n = 90)	Nondippers (n = 38)	*P* value[a]
Age, mean (SD), y	27.97 (5.18)	29.26 (5.10)	.36
Nulliparity, No. (%)	22 (24.4)	8 (21)	.43
Gestational age, mean (SD), wk	35.77 (3.24)	36.20 (2.88)	.66
Mode of termination, No. (%)			.73
Cesarean delivery	48 (53.3)	20 (52.6)	
Vaginal delivery	42 (46.7)	18 (47.4)	
History of preeclampsia, No. (%)	42 (46.7)	22 (73.3)	.03
Severe preeclampsia, No. (%)	40 (44.4)	22 (73.3)	.01
SBP, mean (SD), mm Hg	155.88 (19.80)	158.42 (16.07)	.62
DBP, mean (SD), mm Hg	99.88 (15.82)	99.47 (11.65)	.91

DBP, diastolic blood pressure; SBP, systolic blood pressure.

[a] $P < .05$ was considered statistically significant.

spectively, with the mean (SD) ages of gestation at termination being 35.77 (3.24) vs 36.20 (2.88) weeks, respectively. We noticed that severe PE presented in 40 of 90 (44.4%) and 22 of 38 (73.3%) dippers and nondippers, respectively. In addition, nondippers had a higher frequency of previous PE diagnosis and severe PE ($P < .05$).

Echocardiographic Characteristics

There was no significant difference in the diastolic parameters, namely LV end-diastolic diameter, LV end-systolic diameter, interventricular septum, posterior wall as well as Emax, LV end-systolic volume, and LV end-diastolic volume. Ejection fraction was observed between dippers and nondippers either by M-mode or Simpson's method ($P > .05$). Dippers had a significantly higher E/A ratio, e′ septal, and e′ lateral and a significantly lower Amax, E/e′ septal, E/e′ lateral, deceleration time, isovolumetric relaxation time, and left atrial

TABLE II. Echocardiographic Characteristics in Both Groups[a]

Echocardiographic characteristics	Dippers (n = 90)	Nondippers (n = 38)	*P* value[b]
Systolic function			
EF, %			
by M-mode measurement	62.33 (4.17)	61.89 (4.08)	.69
by Simpson method	61.62 (4.20)	61.21 (4.41	.72
LV end-diastolic dimension, mm	43.60 (4.55)	43.21 (6.22)	.87
LV end-systolic dimension, mm	28.84 (3.78)	29.05 (4.52)	.85
LV end-diastolic volume, mL	78.62 (14.39)	80.42 (18.78)	.67
LV end-systolic volume, mL	29.84 (5.71)	30.42 (4.27)	.69
Interventricular septum dimension in diastole, mm	9.01 (1.31)	9.63 (1.25)	.08
Posterior wall dimension in diastole, mm	9.31 (1.32)	9.31 (0.88)	.98
Diastolic function			
Emax, mL/s	0.86 (0.15)	0.76 (0.29)	.07
Amax, mL/s	0.56 (0.14)	0.82 (0.24)	<.001
E/A ratio	1.58 (0.25)	0.99 (0.50)	<.001
e′ lateral, m/s	0.16 (0.02)	0.10 (0.03)	<.001
e′ septal, m/s	0.12 (0.02)	0.08 (0.03)	<.001
E/e′ lateral, m/s	5.28 (0.98)	7.54 (2.64)	<.001
E/e′ septal, m/s	6.87 (1.36)	9.74 (3.54)	<.001
Deceleration time, ms	152.86 (14.88)	211.47 (45.04)	<.001
Isovolumetric relaxation time, ms	65.75 (8.93)	102.36 (28.26)	<.001
Left atrium volume, mL/m^2	15.33 (1.81)	22.84 (5.37)	<.001
Diastolic dysfunction, No. (%)	0	28 (73.7)	<.001

A, mitral late diastolic velocity because of atrial contraction; Amax, maximum atrial filling; E, mitral early diastolic velocity; e′, pulsed-wave velocity across lateral or septal mitral annulus; EF, ejection fraction; Emax, maximum elastance; LV, left ventricle.

[a] Data are expressed as mean (SD), unless otherwise indicated.

[b] $P < .05$ was considered statistically significant.

volume than those of nondippers ($P < .001$). Diastolic dysfunction was present in 28 (73.7%) nondippers and none of the dippers ($P < .001$; Table II).

Dipping Profile in the Studied Groups

The total average SBP and DBP and the night average as well as day/night BP reduction were significantly lower in dippers than in nondippers ($P < .001$). Daytime DBP showed a tendency to be higher in dippers (mean [SD], 79.52 [6.96] vs 78.02 [6.31] mm Hg); however, this difference was not statistically significant (Table III).

Effect of PE Severity

A total of 62 (48.4%) women included in the study had severe PE, whereas the remaining 66 women (51.6%) had mild to moderate PE. Moreover, no significant differences in EF were observed between the groups. Nevertheless, women with severe PE were more frequently nondippers (35.5% vs 24.2%; $P = .02$) and were more likely to have diastolic dysfunction (29% vs 15%; $P = .01$) than were those with mild to moderate PE (Table IV).

Assessment of Diastolic Dysfunction

Among the women studied, 28 (21.9%) had diastolic dysfunction, whereas 100 (78.1%) had no diastolic dysfunction. Furthermore, no significant differences in DBP, SBP, or gestational age were observed among the groups, whereas all women with diastolic dysfunction were nondippers (diastolic dysfunction was present in 28 nondippers [73.7%]). Of the women who had no diastolic dysfunction, none were nondippers (Table V).

Predictors of Nondipping

Logistic regression analysis revealed that severe PE (OR, 1.08; 95% CI, 1.05-10.56; $P < .001$) and recurrent PE (OR, 1.36; 95% CI, 1.30-4.26; $P < .001$) were

TABLE III. Dipper Profiles in Studied Groups

BP profile	Dippers, mean (SD), mm Hg (n = 90)	Nondippers, mean (SD), mm Hg (n = 38)	*P* value[a]
Total average			
Systolic	119.72 (7.06)	126.55 (5.66)	<.001
Diastolic	72.44 (6.17)	76.81 (5.57)	<.001
Day average			
Systolic	129.06 (6.61)	129.94 (6.22)	.26
Diastolic	78.02 (6.31)	79.52 (6.96)	.40
Night average			
Systolic	110.64 (7.45)	123.15 (5.95)	<.001
Diastolic	67.06 (6.46)	74.10 (4.96)	<.001
Day/night reduction			
Systolic reduction	14.29 (3.29)	5.15 (3.29)	<.001
Diastolic reduction	14.04 (3.87)	6.41 (2.97)	<.001

BP, blood pressure.

[a] $P < .05$ was considered statistically significant.

TABLE IV. Echocardiographic Findings Based on Severity of Preeclampsia

Variables	Mild to moderate preeclampsia (n = 66)	Severe preeclampsia (n = 62)	*P* value[a]
Ejection fraction, mean (SD), %			
By M-mode measurements	119.72 (7.06)	126.55 (5.66)	<.001
By Simpson method	72.44 (6.17)	76.81 (5.57)	<.001
Diastolic dysfunction, No. (%)	10 (15)	18 (29)	.01
Nondippers, No. (%)	16 (24.2)	22 (35.5)	.02

[a] $P < .05$ was considered statistically significant.

TABLE V. Blood Pressure, Dipper Profile, and Gestational Age Based on the Development of Diastolic Dysfunction

Variables	Diastolic dysfunction (n = 28)	No diastolic dysfunction (n = 100)	*P* value[a]
Blood pressure, mean (SD), mm Hg			
SBP	162.85 (16.01)	154.90 (19.15)	.66
DBP	101.42 (12.92)	99.30 (15.15)	.16
Gestational age, mean (SD), wk	36.25 (3.25)	35.79 (3.11)	.63
Nondippers, No. (%)	28 (100)	0	<.001

DBP, diastolic blood pressure; SBP, systolic blood pressure.

[a] $P < .05$ was considered statistically significant.

TABLE VI. Predictors of Nondipper Status

Variables	Odds ratio	95% CI	*P* value[a]
Age, y	0.95	0.75-1.21	.71
Nulliparity	0.45	0.40-1.04	.99
Gestation age, wk	0.81	0.57-1.16	.26
Recurrent preeclampsia	1.36	1.30-4.26	<.001
Severe preeclampsia	1.08	1.05-10.56	<.001
Basic blood pressure, mm Hg[b]	0.88	0.72-1.08	.25

[a] $P < .05$ was considered statistically significant.
[b] Basic blood pressure is the woman's blood pressure in the evaluation setting 3 months after preeclampsia condition.

predictors for nondipping in such patients (Table VI). We found that an initial SBP cutoff level of more than 150 mm Hg had a sensitivity and specificity of 54.2% and 64.4%, respectively, for predicting nondipping, with an area under the curve (AUC) of 0.59 ($P = .01$). A DBP cutoff level of more than 90 mm Hg had a sensitivity and specificity of 68.4% and 44.4%, respectively, for predicting nondipping, with an AUC of 0.53 ($P = .03$).

Predictors of Diastolic Dysfunction

The findings showed that severe PE, previous PE, nondipping, average nighttime DBP, and DBP night reduction were predictors for diastolic dysfunction ($P < .05$; Table VII). An initial SBP cutoff level of more than 140 mm Hg had a sensitivity and specificity of 100% and 32%, respectively, for predicting diastolic dysfunction, with an AUC of 0.69 ($P < .001$). In addition, we found that DBP at a cutoff level of more than 95 mm Hg had a sensitivity and specificity of 71.7% and 50%, respectively, for predicting diastolic dysfunction, with an AUC of 0.60 ($P < .001$).

Discussion

The hemodynamic changes associated with PE mainly result from endothelial dysfunction and increased the sensitivity of blood vessels to angiotensin II, leading to peripheral vasoconstriction and increasing total vascular resistance.[20-22]

Although the office BP may return to its normal values in the postpartum period, there are no sufficient data regarding the reversibility of the BP pattern after delivery among patients with a history of PE. Therefore, a history of PE is still considered a risk factor for CV events later in life.[4,23]

The current study attempted to investigate the circadian BP pattern and explore its association with subclinical CV effects using 2D echocardiography in women with PE 3 months postpartum. The findings showed a positive relationship between PE and a nondipping profile. Moreover, we compared the dipping profiles among those with mild to moderate and severe PE. Accordingly, we found that circadian rhythm abnormality (ie, blunted nocturnal dipping profile) was more frequent among those with previously documented severe PE than among those with a previous diagnosis of mild to

TABLE VII. Predictors of Diastolic Dysfunction

Variables	Odds ratio	95% CI	*P* value[a]
Previous preeclampsia	1.23	1.22-2.22	.03
Severe preeclampsia	1.55	1.11-2.20	.01
Nondipping	3.43	1.99-5.56	<.001
Average nighttime DBP, mm Hg	1.61	1.24-3.94	<.001
DBP night reduction, mm Hg	1.70	1.11-2.73	<.001

DBP, diastolic blood pressure.

[a] $P < .05$ was considered statistically significant.

moderate PE. Moreover, a blunted nocturnal BP profile was more prevalent in the women with recurrent PE. Similarly, Ditisheim et al[24] described a positive association between PE and a nondipping profile. The postpartum persistence of high levels of angiotensin II,[25] abnormal nitric oxide synthesis and metabolism,[26] and the altered systemic vascular, which may persist for a prolonged period,[27,28] are the main mechanisms that induce abnormal circadian rhythm in PE.

Studies have established that nocturnal hypertension exacerbates the endothelial damage in PE.[29] Evidence has also found that the deterioration of maternal hemodynamics is more common among pregnant women with hypertension and nondipping BP patterns.[30] The fast reduction in the LV pressure at the end of systole and early diastole is an energy-dependent process. That is what makes the process of myocardial relaxation vulnerable in relation to various CV disorders.[12,31] Moreover, we detected that an increase in the afterload was associated with an increase in the LV mass, both of which were associated with an increase in the LV filling pressure, a reduction in LV compliance, and diastolic dysfunction in patients with PE.[32]

Analysis of diastolic function using TDI in this cohort revealed that abnormal hemodynamic alterations induced a reversal in myocardial remodeling, which was more frequently observed among nondippers. These findings show that most of the women with a nondipping profile had diastolic dysfunction, an issue that was not observed among dippers.

Diastolic dysfunction is generally antecedent to systolic dysfunction in the context of hypertensive or ischemic heart diseases and carries prognostic value in the prediction of long-term CV morbidity.[33] According to Frank Starling and Laplace laws, both elevated after load and peripheral vascular resistance are associated with adaptive response mechanisms to reduced LV wall stress and maintenance of normal LV contractility, which could promote equilibrium between myocardial oxygen supply and demand. Among these mechanisms are increased LV mass and even LV hypertrophy,[34-36] which have been found in a portion of patients with severe PE.[37,38]

Left ventricular concentric hypertrophy is known to be accompanied by subendocardial fibrosis. Duman et al[39] described the coexistence of longitudinal myofibril dysfunction, which is mainly distributed in the subendocardium of patients with PE. This implies subendocardial ischemia and damage, which was validated by the autopsy findings of severe PE with adverse outcomes in Duman et al's study.[39]

Therefore, 2D ventricular EF was used to examine systolic function in the study cohort. Surprisingly, no pathological reduction in LVEF was found in either mild to moderate or severe PE during the postpartum period.

Studies have shown that there are 3 types of myofibril arrangement in the LV, mainly longitudinal and oblique in the subendocardial and subepicardial layers and circumferential between the layers. Contraction of the longitudinal and oblique myofibrils occurs at the early systole, followed by contraction of the circumferential myofibrils responsible for the LV ejection. The subendocardial fibers are more vulnerable to the effects of ischemia and/or pressure load.[40]

Standard parameters to estimate LV contractility, such as EF, are volume- and heart rate–dependent.[41,42] Both are increased as compensatory hemodynamic changes during pregnancy.[20,22] This could explain the preserved EF in this study's cohort.

Tyldum et al[43] and Tatapudi et al[44] also reported preserved LVEF in women with PE. Interestingly, Ilic et al[30] and Valensise et al[45] reported systolic dysfunction

and reduced EF among nondippers, which were absent among dippers. However, they studied pregnant women without proteinuria who had gestational hypertension and a higher average BP value than that of the patients in this study.

Uno et al[46] also found cardiac remodeling concerning LV end-diastolic and end-systolic dimensions among patients with PE, although the EF was still preserved. These changes were more prominent in those with severe PE than in those with mild PE and were not reversed completely 1 month after delivery.

Recent studies estimating the longitudinal LV systolic function using 2D speckle-tracking echocardiography have found that the global strain value in women with severe PE had decreased, which indicates LV systolic dysfunction in women with severe PE.[47,48] In addition, severe and recurrent PE were noted as significant predictors for both a nondipping profile and diastolic dysfunction. Valensise et al[45] detected that cardiac dysfunction in the nonpregnant state was more frequent among patients with recurrent PE. However, Tatapudi et al[44] detected that cardiac dysfunction was more prominent among those with severe PE.

Limitations

The main limitation of this study was the relatively small sample-size population in a single-center experience with all inherited constraints on which the results are based. This leads to higher variance and increases the potential for bias. In addition, the contribution of healthy pregnant women to the prospective cohort study was, for some participants, not convenient, which made it more difficult to recruit participants for the study. Participants did not have prepregnancy echocardiographic data because they were part of a relatively healthy population group.

Conclusion

Despite returning the office BP to its normal level, the abnormal BP profile and cardiac remodeling could persist 3 months after the end of pregnancy. Severe and recurrent PE are both important predictors for a nondipping BP profile and diastolic dysfunction. The findings of this study supported use of TDI and ABPM as easily available, noninvasive, and easily applicable methods for screening populations at high risk.

Recommendations

We recommend the analysis of echocardiographic findings before pregnancy with a simultaneous evaluation of the longitudinal LV systolic function using strain rate imaging and longer follow-up times to determine whether diastolic dysfunction and nondipping status are predictors of sustained hypertension in this cohort. This would certainly guide prevention and intervention strategies for patients with PE and reduce the risk of heart failure and other future CV morbidities in subsequent pregnancies and among younger mothers.

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