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ORIGINAL RESEARCH

CARDIOOBSTETRICS

Echocardiographic Differences in Women Across Subtypes of Hypertensive Disorders of Pregnancy

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ABSTRACT

BACKGROUND Hypertensive disorders of pregnancy (HDP) can be classified into gestational hypertension, preeclampsia (PRE), and chronic hypertension with superimposed preeclampsia (SPE).

OBJECTIVES The purpose of this study was to retrospectively examine the echocardiographic differences in biventricular structure and function in 3 HDP groups of women in comparison to normotensive pregnant controls.

METHODS Women with an echocardiogram during or within the first year of pregnancy were identified within our integrated health network. Exclusion criteria included age <18 years, diagnosis of pulmonary embolism, malignancy, autoimmune disease, and structural heart disease.

RESULTS We identified a total of 706 subjects (cases: n = 427, normotensive controls: n = 279). Cases were divided into 3 groups: gestational hypertension (n = 57), PRE (n = 291), and SPE (n = 79). In adjusted analyses, echocardiographic parameters demonstrated a graded difference in left ventricular (LV) mass index, relative wall thickness, mitral inflow E, mitral inflow A, septal e', lateral e', E/e', left atrial volume index, tricuspid velocity, and lateral e' velocities with the most profound findings noted in the SPE group. Specifically, adjusted LV mass index (adjusted $\beta = 14.45$, 95% CI: 9.00-19.90) and E/e' (adjusted $\beta = 2.97$, 95% CI: 2.27-3.68) was highest in the SPE group in comparison to controls ($P < 0.001$).

CONCLUSIONS LV remodeling and diastolic filling abnormalities are more common in HDP and are most evident in SPE and PRE. Echocardiography during or immediately after pregnancy may be useful in these high-risk women to identify these abnormalities. The long-term implications of these echocardiographic abnormalities require further study. (JACC Adv 2024;3:100725) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

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ABBREVIATIONS AND ACRONYMS

E/A = early to atrial filling velocity ratio

LAVi = left atrial volume index

LVEDD = left ventricular end-diastolic diameter

LVEDV = left ventricular end-diastolic volume

LVESD = left ventricular end-systolic diameter

LVESV = left ventricular end-systolic volume

LVMi = left ventricular mass index

mitral A = mitral inflow velocity of late diastolic filling

mitral E = mitral inflow velocity of early diastolic filling

TAPSE = tricuspid annular plane systolic excursion

Hypertensive disorders of pregnancy (HDP) remain one of the leading causes of maternal morbidity and mortality globally.[1,2] HDP have consistently been associated with a higher long-term risk of cardiovascular disease and are seen in up to 10% of pregnancies.[3-5] HDP has been classified into 3 distinct groups by the International Society for the Study of Hypertension in Pregnancy: gestational hypertension (GHTN), preeclampsia (PRE), and chronic hypertension with superimposed preeclampsia (SPE) based on the timing of onset of hypertension (before or after 20 weeks of gestation), proteinuria, and evidence of end-organ injury.[6] Differences in maternal and fetal as well as long-term cardiovascular outcomes have been reported between these 3 subtypes.[7-10]

HDP-related changes in maternal cardiac geometry and function are more pronounced than the typical cardiovascular physiological changes that occur with pregnancy.[11] Systemic vascular resistance remains elevated 6 months postpartum in patients with PRE, and cardiac maladaptation in pregnancy can take up to a year to completely resolve.[12,13] There is a paucity of literature looking at differences in echocardiographic structure and function between 3 subtypes of HDP. Despite the accumulated evidence, echocardiography is not endorsed by current guidelines for risk stratification of women with HDP. Identifying higher-risk groups that manifest the greatest abnormalities in cardiac structure and function can lead to the targeted use of echocardiography in women with HDP. We sought to examine the echocardiographic changes in these 3 groups of women in comparison to nonhypertensive pregnant controls.

METHODS

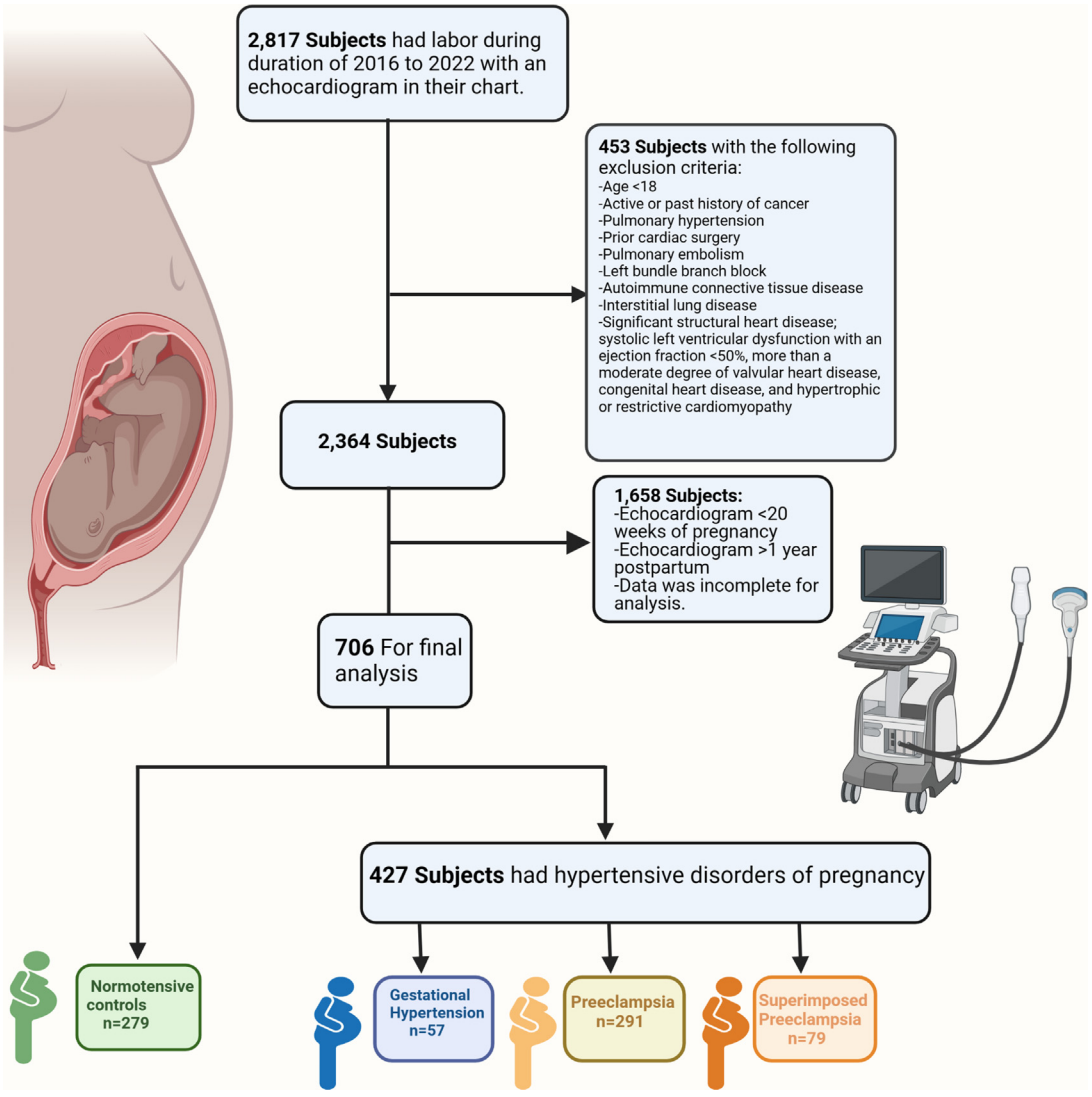
STUDY DESIGN AND SUBJECTS. We performed a retrospective study utilizing electronic medical records within an integrated health-care system, identifying all patients who underwent an echocardiogram during or immediately after pregnancy and within our study period, January 2016 to February 2022. Hypertensive pregnant women who had echocardiography from 20 weeks gestation up to 1 year after delivery were identified as cases. Cases were individually reviewed and further classified into 3 main groups: GHTN: blood pressure (BP) ≥140/90 mmHg that occurs after the first 20 weeks of pregnancy, is not associated with proteinuria or systemic signs and symptoms; PRE: BP ≥140/90 mmHg that occurs after the first 20 weeks of pregnancy and is associated with proteinuria or end-organ dysfunction such as thrombocytopenia, liver failure, or rise in creatinine; and SPE: prior diagnosis of HTN or BP ≥140/90 mmHg before the first 20 weeks of pregnancy associated with new or worsening proteinuria or end-organ dysfunction as with PRE. Patients with chronic hypertension were also included in the SPE group in the absence of proteinuria, if they had BP elevation with thrombocytopenia, abnormal liver function, worsening renal dysfunction, pulmonary edema, or new-onset visual disturbances.[6,14]

Nonhypertensive pregnant women with an echocardiogram during the same gestational to postpartum period were assigned to a normotensive control group. Most of these patients had cardiac symptoms such as palpitations or dyspnea during pregnancy but without any prior cardiac history. Noncardiac diagnoses that could lead to cardiac echocardiographic abnormalities were excluded. Exclusion criteria included age <18 years, active or past history of cancer, pulmonary hypertension, prior cardiac surgery, pulmonary embolism, autoimmune connective tissue disease, antiphospholipid syndrome, interstitial lung disease, left bundle branch block, and significant structural heart disease including left ventricular systolic dysfunction with an ejection fraction <50%, more than a moderate degree of valvular heart disease, congenital heart disease, and hypertrophic or restrictive cardiomyopathy. **Figure 1** represents a patient selection algorithm.

Demographics, comorbidities, pregnancy, and echocardiographic data were collected. Both body mass index (BMI) and body surface area (BSA) were calculated from height and body weight closest to the echocardiogram. For those with SPE and PRE, we examined whether they developed severe features based on systolic BP ≥160 mm Hg, diastolic BP ≥110 mm Hg, or evidence of end-organ dysfunction such as elevated liver function tests, thrombocytopenia, neurologic symptoms, pulmonary edema, or worsening renal function.[15] The Institutional Review Board of Allegheny Health Network approved the study protocol.

TRANSTHORACIC ECHOCARDIOGRAPHY. Standard 2-dimensional and Doppler echocardiograms were performed per the American Society of Echocardiography guidelines.[16] Measurements of left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter, interventricular septal thickness (IVSd), and left ventricular (LV) posterior wall

FIGURE 1 Patient Selection Algorithm[15]



thickness (PWT) were performed by 2D echocardiography. LVEDV and LVESV were measured using a modified Simpson method, and LV ejection fraction (LVEF) was calculated.[17] LV mass was calculated using the American Society of Echocardiography methodology and indexed to BSA to obtain left ventricular mass index (LVMi).[17] Relative wall thickness (RWT) was calculated using the formula (2 × PWT)/LVEDD.[18] The presence of pericardial effusion was reported as trace, mild, moderate, or large based on visual assessment on 2D echocardiography. The right ventricular size was measured at the base of the ventricle in the apical 4-chamber view.

Using LVMi and RWT, we classified LV geometry into normal geometry (LVMi ≤95 g/m^2 and RWT ≤0.42), concentric remodeling (LVMi ≤95 g/m^2 and RWT >0.42), concentric hypertrophy (LVMi >95 g/m^2 and RWT >0.42), and eccentric hypertrophy (LVMi >95 g/m^2 and RWT ≤0.42).[18] In the 4-chamber view, the TAPSE was obtained from M-mode recording. Parameters of diastolic function included LAVi, MV E and A velocities and the ratio (E/A), peak tricuspid regurgitation (TR) velocity, deceleration time (DT), and septal and lateral tissue Doppler e' and a' velocities and the ratio MV E/septal e'. All measurements were obtained from the electronic medical record, and when individual measurements were not available, they were independently measured on the images by study physicians trained in echocardiography.

TABLE 1 Baseline Characteristics by Subtype

	Controls (n = 279)	Gestational Hypertension (n = 57)	Preeclampsia (n = 291)	Superimposed Preeclampsia (n = 79)
Age, y[a]	29.67 ± 5.16	30.58 ± 5.92	30.72 ± 5.74	33.22 ± 5.24
Body mass index, kg/m^2[a]	29.86 ± 6.74	33.1 ± 9.46	33.51 ± 9.31	35.96 ± 8.17
Race				
Asian	8 (2.9%)	0 (0%)	3 (1.0%)	3 (3.8%)
Black[a]	33 (11.8%)	7 (12.3%)	61 (21.0%)	19 (24.0%)
Caucasian	233 (83.5%)	49 (86.0%)	226 (77.7%)	57 (72.2%)
Other	5 (1.8%)	1 (1.8%)	1 (0.3%)	0 (0%)
Hispanic ethnicity	4 (1.4%)	2 (3.5%)	4 (1.4%)	1 (1.27%)
Insurance type				
Self	169 (60.6%)	34 (59.7%)	164 (56.4%)	40 (50.6%)
Medicaid	26 (9.3%)	2 (3.5%)	26 (8.9%)	7 (8.9%)
Medicare	0 (0%)	0 (0%)	2 (0.7%)	1 (1.3%)
Commercial	84 (30.1%)	21 (36.8%)	99 (34.0%)	31 (39.2%)
Household median income for zip code, $[a]	66,346 ± 23,009	61,879 ± 20,113	60,791 ± 18,713	60,061 ± 18,247
Tobacco use				
Never	212 (76.0%)	44 (77.2%)	202 (69.4%)	50 (63.3%)
Current	21 (7.5%)	4 (7.0%)	37 (12.7%)	12 (15.2%)
Former	46 (16.5%)	9 (15.8%)	52 (17.9%)	16 (20.3%)
Obstructive sleep apnea[a]	0 (0%)	0 (0%)	14 (4.8%)	5 (6.3%)
Atrial fibrillation	4 (1.4%)	2 (3.5%)	0 (0%)	2 (2.5%)
Chronic kidney disease stage III/IV	17 (6.1%)	0 (0%)	1 (0.3%)	1 (1.3%)
Thyroid disease				
Hypothyroidism	31 (11.1%)	9 (15.8%)	35 (12.0%)	10 (12.7%)
Hyperthyroidism	3 (1.1%)	0 (0%)	3 (1.0%)	1 (1.3%)
Diabetes mellitus				
Gestational	10 (10.9%)	4 (7.3%)	40 (13.8%)	17 (21.5%)
Pre-gestational[a]	16 (20.3%)	34 (11.7%)	4 (7.0%)	10 (3.6%)
Polycystic ovarian syndrome	17 (6.1%)	6 (10.5%)	25 (8.6%)	9 (11.4%)
Gestational age, wk[a]	33.29 ± 5.88	35.58 ± 3.91	34.65 ± 3.94	32.16 ± 4.2
Multiple gestation	8 (2.9%)	2 (3.5%)	16 (5.5%)	3 (3.8%)
Presence of severe features	NA	NA	239 (82.4%)	71 (89.9%)

Values are mean ± SD or n (%). [a]Statistically significant difference between hypertensive disorders of pregnancy and control group.

STATISTICAL ANALYSIS. Data were reported as mean ± SD for continuous variables, and as frequencies (percentages) for categorical variables. All echocardiographic parameters conformed approximately to normal distributions based on the visual assessment of frequency histograms. Linear regression models were developed to assess the association between each echocardiographic parameter and HDP subtype. Multivariable linear regression models adjusted for age, BMI, race, smoking status, gestational diabetes, and nongestational diabetes. Echocardiographic parameters already indexed to BSA were not adjusted for BMI. The nonhypertensive control group served as the reference group in all models; thus, all reported regression coefficients compare the HDP subtype to the control group. All regression coefficients are interpreted as the mean difference in the echocardiographic parameter in the HDP subtype vs controls. Two *P* values for each HDP subtype-echocardiographic parameter comparison are reported: one associated with testing for any differences across all groups; and a second associated with testing for trends across groups (ie, whether the echocardiographic parameter is increasing/decreasing across HDP groups). Trends across HDP groups were assessed by modeling the HDP group as an ordinal, integer variable (1, 2, 3, 4) in the linear regression models.

For an elevated LVMi (>95 g/m^2), logistic regression models were developed to assess its association with the HDP subtype. Odds ratios and 95% CIs are reported with the nonhypertensive control group serving as the reference. Multivariable logistic regression models adjusted for age, race, smoking status, gestational diabetes, and nongestational diabetes. Again, 2 *P* values are reported as described earlier. A *P* value

TABLE 2 Linear Regression Models Assessing the Association Between Hypertensive Subtype and Echocardiographic Remodeling Parameters

	Control (n = 279)	Gestational Hypertension (n = 57)	Preeclampsia (n = 291)	Superimposed Preeclampsia (n = 79)	*P* Value[a]	Trend *P* Value[b]
LVEDD, cm	4.59 ± 0.46	4.58 ± 0.44	4.70 ± 0.53	4.67 ± 0.62	0.06	0.02
Adjusted β[c]	0.0 (—)	−0.07 (−0.21, 0.08)	0.04 (−0.04, 0.13)	−0.02 (−0.15, 0.11)	0.41	0.59
LVESD, cm	3.10 ± 0.41	3.06 ± 0.40	3.15 ± 0.48	3.08 ± 0.51	0.33	0.43
Adjusted β[c]	0.0 (—)	−0.08 (−0.21, 0.05)	0.01 (−0.07, 0.09)	−0.08 (−0.20, 0.04)	0.30	0.64
LVEDV, ml	92.53 ± 24.05	93.93 ± 30.71	101.33 ± 26.84	103.89 ± 35.79	0.001	<0.001
Adjusted β[c]	0.0 (—)	−5.02 (−14.92, 4.87)	5.39 (0.13, 10.64)	7.33 (−0.39, 15.04)	0.03	0.01
LVESV, ml	35.39 ± 12.09	36.08 ± 14.18	38.21 ± 13.85	40.55 ± 16.74	0.04	0.004
Adjusted β[c]	0.0 (—)	−2.24 (−7.26, 2.78)	1.18 (−1.48, 3.85)	2.09 (−1.83, 6.01)	0.39	0.21
LVEDVi, ml/m^2	48.88 ± 12.25	45.77 ± 12.61	50.84 ± 11.92	48.61 ± 14.29	0.11	0.36
Adjusted β[d]	0.0 (—)	−3.01 (−7.70, 1.68)	2.22 (−0.28, 4.71)	0.48 (−3.15, 4.11)	0.09	0.20
LVESVi, ml/m^2	18.73 ± 6.44	17.58 ± 5.84	19.03 ± 6.44	18.73 ± 6.23	0.62	0.66
Adjusted β[d]	0.0 (—)	−1.23 (−3.61, 1.16)	0.38 (−0.89, 1.64)	−0.09 (−1.95, 1.77)	0.60	0.71
LVMi, g/m^2	69.19 ± 15.40	69.94 ± 18.48	80.54 ± 21.64	85.26 ± 29.81	<0.001	<0.001
Adjusted β[c]	0.0 (—)	0.51 (−6.03, 7.05)	10.54 (6.93, 14.15)	14.45 (9.00, 19.90)	<0.001	<0.001
PWT, mm	0.89 ± 0.16	0.94 ± 0.20	1.00 ± 0.18	1.10 ± 0.23	<0.001	<0.001
Adjusted β[d]	0.0 (—)	0.04 (−0.02, 0.09)	0.09 (0.06, 0.12)	0.17 (0.12, 0.21)	<0.001	<0.001
IVSD, mm	0.85 ± 0.15	0.93 ± 0.19	0.99 ± 0.19	1.13 ± 0.27	<0.001	<0.001
Adjusted β[d]	0.0 (—)	0.05 (0.00, 0.11)	0.11 (0.08, 0.14)	0.22 (0.18, 0.27)	<0.001	<0.001
RWT	0.39 ± 0.09	0.42 ± 0.11	0.43 ± 0.10	0.49 ± 0.13	<0.001	<0.001
Adjusted β[d]	0.0 (—)	0.02 (−0.01, 0.05)	0.04 (0.02, 0.05)	0.08 (0.06, 0.11)	<0.001	<0.001
RV Size, cm	3.08 ± 0.55	2.87 ± 0.50	2.89 ± 0.57	2.85 ± 0.44	0.001	<0.001
Adjusted β[c]	0.0 (—)	−0.29 (−0.49, −0.10)	−0.26 (−0.37, −0.15)	−0.34 (−0.49, −0.18)	<0.001	<0.001

Values are mean ± SD unless otherwise indicated. Two *P* values for each comparison are reported: *P* value is associated with testing for any differences across all groups; and trend *P* value is associated with a test for trend across groups. [a]Testing for overall differences across the 4 groups. [b]Testing for trend across the 4 groups. [c]β = regression coefficient from a linear regression model adjusted for age, body mass index, race, smoking status, gestational diabetes, and nongestational diabetes. [d]β = regression coefficient from a linear regression model adjusted for age, race, smoking status, gestational diabetes, and nongestational diabetes.

IVSD = interventricular septal thickness; LAVi = left atrial volume index; LVEDD = left ventricular end-diastolic diameter; LVEDV = LV end-diastolic volume; LVEDVi = left ventricular end-diastolic volume index; LVESD = LV end-systolic diameter; LVESV = LV end-systolic volume; LVESVi = left ventricular end-systolic volume index; LVMi = left ventricular mass index; PWT = posterior wall thickness; RV = right ventricle; RWT = Relative left ventricular wall thickness.

<0.05 was considered significant. All analyses were performed by a biostatistician using SAS v9.4.

RESULTS

BASELINE CHARACTERISTICS. A total of 706 women who had an echocardiogram from 20 weeks of gestation to 1 year postpartum were included in the final analysis, and participants were categorized into 2 groups: normotensive (n = 279) and women with HDP (n = 427). Among those with HDP, 57 (13.3%) had GH, 291 (68.1%) had PRE, and 79 (18.5%) had SPE. In our total population, 381 (53.4%) patients had an echocardiogram intrapartum, 205 (29.0%) patients had it within 1 month postpartum, and 120 (17.0%) from 1 month to 1 year postpartum. There were 32 women with postpartum PRE. Compared to the normotensive participants, the HDP women were more likely to be older, Black, have higher BMI, and have lower household income. HDP women had more comorbidities such as sleep apnea and were less likely to have a diagnosis of pregestational diabetes mellitus (Table 1). There was no difference in the number of multiple gestation pregnancies between the HDP and non-HDP groups. The majority of PRE and SPE groups had severe features, 82.4% (n = 239) and 89.9% (n = 71), respectively. The high proportion of women with severe features in our study population likely reflects the indication for echocardiography.

CHANGES IN CARDIAC GEOMETRY. Women with HDP had significantly higher mean LVEDd, LVEDV, LVESV, LVMi, RWT, IVSd, and PWT than the normotensive group (Supplemental Table 1). There was a graded increase in PWT, IVSd, and RWT, with SPE having the highest value as depicted by our unadjusted linear regression model (Table 2). When adjusted for possible confounders, the GHTN group was no longer statistically different from the controls. LVEDV, LVESV, and LVMi were higher in PRE and SPE, but only LVMi remained statistically significant. In unadjusted logistic regression analysis, PRE (OR: 6.47, 95% CI: 3.31-12.65) and SPE (OR: 12.27, 95% CI: 5.71-26.38) were independently associated with

TABLE 3 Left Ventricular Geometry by Subgroup

Type of Remodeling	Control	Gestational Hypertension	Preeclampsia	Superimposed Preeclampsia	*P* Value
Normal	169 (69.0%)	27 (61.4%)	105 (40.5%)	20 (26.7%)	<0.001
Concentric remodeling	65 (26.5%)	15 (34.1%)	95 (36.7%)	28 (37.3%)	0.08
Concentric hypertrophy	3 (1.2%)	2 (4.6%)	41 (15.8%)	22 (29.3%)	<0.001
Eccentric hypertrophy	8 (3.3%)	0 (0%)	18 (7.0%)	5 (6.7%)	0.09

Values are n (%).

LVMi >95 g/m^2 (P < 0.001 and a trend P value of <0.001). However, GHTN was not associated with an LVMi >95 g/m^2 (OR: 0.99, 95% CI: 0.21-4.62). When adjusted for age, race, smoking status, gestational diabetes, and nongestational diabetes, HDP was significantly associated with LVMi >95 g/m^2: GHTN (OR: 0.92, 95% CI: 0.20-4.36), PRE (OR: 5.78, 95% CI: 2.93-11.40), and SPE (OR: 9.54, 95% CI: 4.31-21.14) with a P value across all groups of <0.001 and a trend P value of <0.001.

Controls were less likely to have LV remodeling changes (Table 3). Concentric hypertrophy was more prevalent in HDP subtypes than in controls and was most noted in the SPE group (Table 4, Central Illustration). On the other hand, eccentric hypertrophy with an increase in LV wall thickness and cavity size was noted most in PRE. The left atrial size was larger in HDP groups than in controls although the LAVI remained in the normal range (Table 4).

LEFT VENTRICULAR FUNCTIONAL CHANGES. No difference in LVEF was noted between HDP subtypes and controls with all study subjects having LVEF >55%. Diastolic parameters such as LAVi, mitral E, mitral A, E/A, DT, Sep E', lateral e', E/e', and TR velocity were significantly abnormal in HDP women compared to those in normotensive women (Supplemental Table 1). Linear regression demonstrated an increase in mitral E, mitral A, and TR velocity in those with PRE and SPE when compared to controls with the largest changes noted in the SPE group (Table 3). DT and mitral annular septal and lateral e' velocities had a graded decrease when comparing PRE and SPE groups with controls in our unadjusted and adjusted regression models, with the SPE group having the lowest annular velocities and shortest DT, all reflective of a greater decrease in diastolic compliance of the LV. There was a graded increase across the HDP subtypes for E/e' in both the unadjusted and adjusted linear regression models, with the highest E/e' in the SPE group.

RELATIONSHIP WITH THE SEVERITY OF HDP. Out of 370 patients with SPE and PRE, 310 (84%) had severe features and demonstrated higher LVMi (97.99 ± 22.45 vs 90.93 ± 19.67, P = 0.031) and mitral E wave velocity (82.83 ± 23.02 vs 75.52 ± 26.99, P = 0.041) than those without severe features.

PERICARDIAL EFFUSION. We also observed that HDP patients were more likely to develop pericardial effusion than controls (14.99% vs 5.04%, P < 0.001). Both GHTN (1.75% vs 0%, P = 0.027) and PRE (4.46% vs 0%, P < 0.001) were more likely than controls to have more than just trace pericardial effusion.

ECHOCARDIOGRAPHIC PARAMETERS IN POSTPARTUM HDP. We performed a subset analysis of the women that underwent echocardiography only in the postpartum period and compared HDP to controls (Supplemental Table 2). The postpartum HDP cohort (n = 245) also had unfavorable remodeling and LV diastolic function indices compared to controls (n = 80). These results may demonstrate that the changes noted intrapartum are sustained for the first few months after delivery. Formal interaction tests supported the hypothesis that the associations between the HDP subtype and echocardiographic parameters were similar according to the timing of echocardiography (intrapartum vs postpartum). Our study did not examine the persistence of these changes beyond the first postpartum year.

ECHOCARDIOGRAPHIC DIFFERENCES IN BLACK AND WHITE WOMEN. Among women with HDP, we examined the racial impact on LV diastolic and remodeling changes by examining differences between self-identified non-Hispanic White and Black women. Our cohort included 87 Black women and 332 White Women with HDP. No significant differences in Doppler parameters were noted by race. LVMi (85.08 ± 26.39 vs 79.16 ± 22.28, P = 0.02) and PWT (1.08 ± 0.22 vs 1.00 ± 0.18, P = 0.002) were higher in Black women, with only PWT remaining significant after adjustment (Supplemental Table 3).

DISCUSSION

We found that women with HDP have a higher degree of LV remodeling and LV diastolic filling abnormalities than controls with an ordinal increase across the

TABLE 4 Linear Regression Models Assessing the Association Between Hypertensive Subtype and Echocardiographic Diastolic Function Parameters

	Control (n = 279)	Gestational Hypertension (n = 57)	Preeclampsia (n = 291)	Superimposed Preeclampsia (n = 79)	*P* Value[a]	Trend *P* Value[b]
LAVi, ml/m^2	22.51 ± 6.25	25.04 ± 6.85	25.40 ± 8.41	25.17 ± 8.24	0.002	<0.001
Adjusted β[c]	0.0 (—)	2.54 (−0.21, 5.29)	2.62 (1.01, 4.22)	2.01 (−0.34, 4.36)	0.01	0.004
Mitral E, cm/s	87.77 ± 20.75	91.91 ± 22.27	96.45 ± 22.30	98.43 ± 21.59	<0.001	<0.001
Adjusted β[d]	0.0 (—)	1.95 (−4.49, 8.39)	6.22 (2.51, 9.93)	7.01 (1.31, 12.72)	0.006	<0.001
Mitral A, cm/s	62.00 ± 16.33	67.46 ± 22.51	70.85 ± 21.14	77.29 ± 21.69	<0.001	<0.001
Adjusted β[d]	0.0 (—)	2.62 (−3.33, 8.57)	6.36 (2.97, 9.74)	9.91 (4.70, 15.12)	<0.001	<0.001
E/A	1.51 ± 0.51	1.45 ± 0.43	1.44 ± 0.48	1.33 ± 0.41	0.06	0.01
Adjusted β[d]	0.0 (—)	−0.03 (−0.18, 0.12)	−0.04 (−0.12, 0.04)	−0.09 (−0.22, 0.04)	0.54	0.17
DT, s	0.20 ± 0.05	0.18 ± 0.07	0.19 ± 0.06	0.17 ± 0.05	0.01	0.005
Adjusted β[d]	0.0 (—)	−0.01 (−0.03, 0.00)	−0.01 (−0.02, 0.00)	−0.02 (−0.04, −0.01)	0.02	0.01
Septal e', cm/s	11.16 ± 2.67	10.88 ± 2.55	10.23 ± 2.48	9.03 ± 2.11	<0.001	<0.001
Adjusted β[d]	0.0 (—)	−0.08 (−0.95, 0.80)	−0.74 (−1.23, −0.25)	−1.63 (−2.35, −0.91)	<0.001	<0.001
Lateral e', cm/s	15.64 ± 3.61	14.58 ± 3.56	13.19 ± 3.25	11.19 ± 3.20	<0.001	<0.001
Adjusted β[d]	0.0 (—)	−0.74 (−1.91, 0.42)	−2.12 (−2.79, −1.45)	−3.66 (−4.63, −2.69)	<0.001	<0.001
E/e'	6.69 ± 1.92	8.06 ± 2.61	8.66 ± 2.68	10.40 ± 3.06	<0.001	<0.001
Adjusted β[d]	0.0 (—)	1.01 (0.17, 1.85)	1.60 (1.11, 2.09)	2.97 (2.27, 3.68)	<0.001	<0.001
TR Velocity, cm/s	208.04 ± 43.12	221.41 ± 52.71	228.31 ± 43.87	229.76 ± 43.02	<0.001	<0.001
Adjusted β[c]	0.0 (—)	10.30 (−6.04, 26.64)	14.32 (4.78, 23.85)	11.59 (−3.59, 26.77)	0.03	0.01
TR max gradient	18.32 ± 6.41	20.72 ± 8.27	21.95 ± 8.00	22.21 ± 7.88	<0.001	<0.001
Adjusted β[c]	0.0 (—)	1.88 (−0.73, 4.49)	2.42 (0.85, 3.99)	1.83 (−0.64, 4.31)	0.02	0.01
TAPSE, mm	23.60 ± 4.43	25.43 ± 5.05	25.47 ± 5.12	25.65 ± 5.06	0.001	<0.001
Adjusted β[c]	0.0 (—)	1.50 (−0.48, 3.48)	1.67 (0.60, 2.73)	1.70 (0.16, 3.25)	0.01	0.002

Values are mean ± SD unless otherwise indicated. Two *P* values for each comparison are reported: *P* value is associated with testing for any differences across all groups; Trend *P* value is associated with a test for trend across groups. [a]Testing for overall differences across the 4 groups. [b]Testing for trend across the 4 groups. [c]β = regression coefficient from a linear regression model adjusted for age, race, smoking status, gestational diabetes, and nongestational diabetes. [d]β = regression coefficient from a linear regression model adjusted for age, body mass index, race, smoking status, gestational diabetes, and nongestational diabetes.

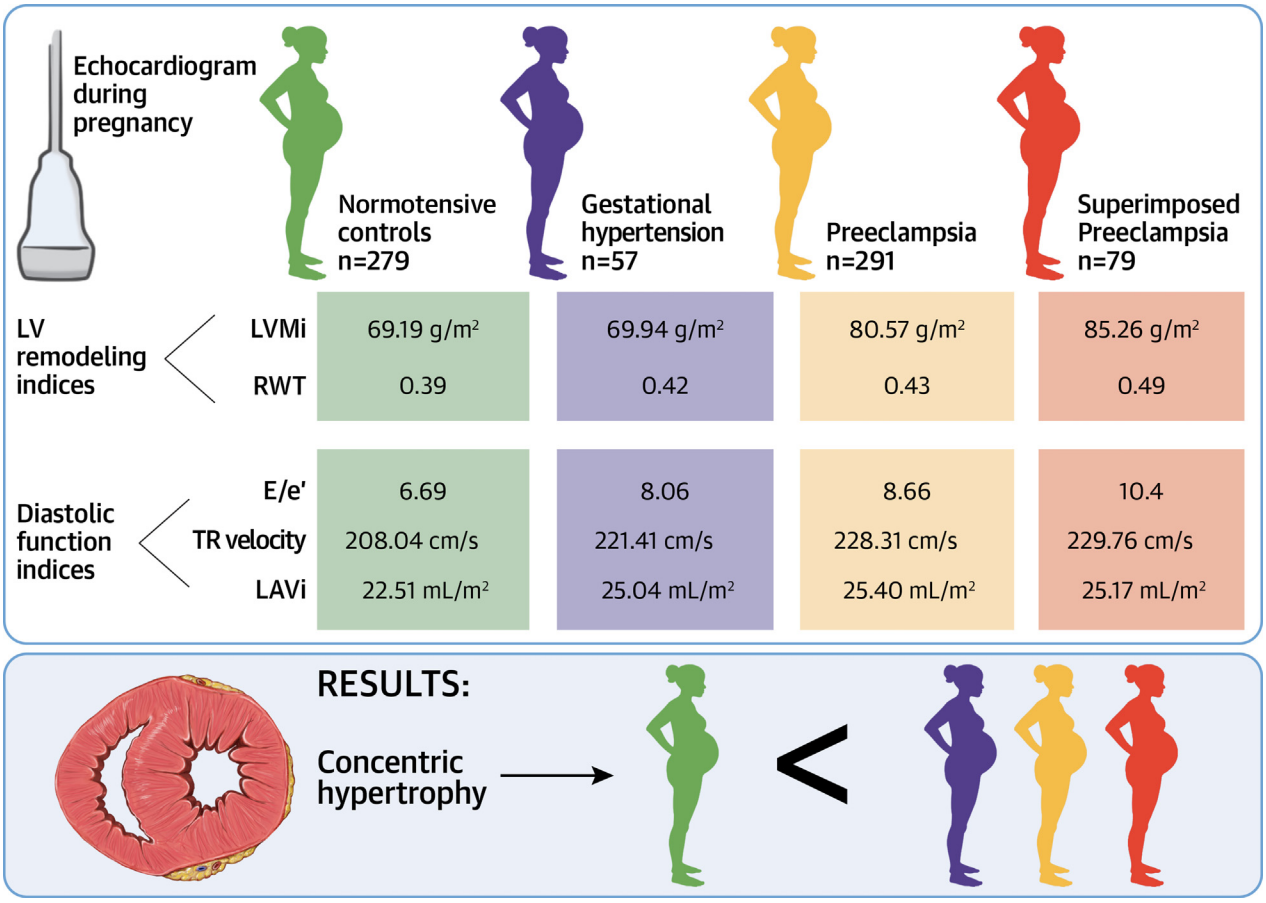
DT = deceleration time; e' = tissue Doppler mitral annular velocity; Lateral e' = lateral tissue Doppler velocity; Septal e' = septal tissue Doppler velocity; TR Vel = Tricuspid velocity.

subtypes of HDP. SPE patients had more significant changes than GHTN and PRE patients. This is the largest single-center study to evaluate intrapartum and early postpartum echocardiographic changes in HDP and bring forth the graded differences in LV remodeling across the 3 groups of women with HDP. Moreover, these echocardiographic changes can also be identified in the early postpartum period, the "fourth trimester," suggesting the persistence of these abnormalities even after delivery.

Echocardiography in HDP has traditionally been reserved for concerns of peripartum cardiomyopathy or research purposes. There is a need to risk stratify women with HDP: Chronic HTN with SPE and PE with severe features are both associated with higher maternal and perinatal mortality, and this study demonstrates a higher degree of LV remodeling in these 2 groups of women. Women with SPE manifesting echocardiographic abnormalities may require closer monitoring and optimization of cardiovascular health to prevent adverse pregnancy outcomes in future pregnancies and improve overall cardiovascular outcomes.[19] Our results are consistent with the literature, showing that maladaptive changes are more likely to occur in HDP patients and further extend these findings as the degree of change varies by the subtype of HDP.[20-22] These changes are more distinctive than the normal physiological adaptations of pregnancy, the latter manifesting as mild LV dilation without an increase in LV wall thickness and filling pressures. Normal controls in this study did not demonstrate any echocardiographic abnormalities, and GHTN did not significantly differ compared to controls. While 40% of PRE also did not have adverse LV remodeling, eccentric LV remodeling was more often observed in this group, suggesting that an increased plasma volume may be a driver of the pathological changes.[23-25]

A systematic review of 745 women with GHTN and 815 with PRE suggested that PRE has a greater impact on LV remodeling and function than GHTN, consistent with our findings.[26] This has been explained by the finding of higher systemic vascular resistance in PRE patients than in GHTN patients.[27,28] Few studies have reported on echocardiographic changes in SPE.[26,29,30] SPE complicates about 20% of pregnancies in women with chronic hypertension and is associated with higher maternal and perinatal morbidity

CENTRAL ILLUSTRATION **Echocardiographic Changes in Women Across Different Subtypes of Hypertensive Disorders of Pregnancy**[15]



Alhuneafat L, et al. JACC Adv. 2024;3(1):100725.

LAVi = left atrial volume index; LV = left ventricular; LVMi = left ventricular mass index; RWT = relative wall thickness; TR = tricuspid regurgitation.

than PRE alone.[9,31,32] We report a higher severity of echocardiographic abnormalities such as concentric hypertrophy and higher LV filling pressure in SPE. Although the current study did not examine women with chronic hypertension without SPE, a prior study did not describe significant LV remodeling in this group.[20] Hence, echocardiography may be most useful in those with PRE or SPE. Our population had a higher proportion of those with severe features as echocardiograms are often ordered in this patient population in our institution. Both SPE and PRE with severe features showed higher LVMi and mitral inflow E velocities than SPE and PRE without severe features. While similar echocardiographic changes have been described in previous studies of echocardiography in severe PE, our study demonstrates that the presence of severe features further accentuates the cardiac structural and functional abnormalities even in those with SPE and PE.[15,22,33,34] Because this is an observational study, the timing of echocardiographic findings in relation to clinical alterations in platelets, liver function, or renal function could not be determined. Nevertheless, in the quest for the identification of higher-risk women who may be at risk of peripartum morbidity, echocardiography could play a role in risk stratification.

Cardiovascular effects of HDP do not resolve in the postpartum period as the increased systemic vascular resistance in PRE can persist for several months postpartum.[13] Giorgione et al[35] who prospectively evaluated 30 HDP patients found that delivery did not improve postpartum echocardiographic indices.

Almost half of our HDP patients and controls had their echocardiogram in the postpartum period, but differences in LV remodeling and LV diastolic function persist between HDP patients and controls.

Various proportions of volume and pressure overload cause different types of LV remodeling.[36] The most common form of LV remodeling in our group was concentric hypertrophy, consistent with the published literature.[20,23,37,38] In a population-based sample without cardiovascular disease, concentric hypertrophy had the worst prognosis out of different types of LV remodeling.[39] In our regression model, we identified that PRE and SPE were associated with an elevated LVMi (>95 g/m^2) after adjusting for multiple potential confounders.

Black women are at higher risk of hypertension in the general population and during pregnancy than White women.[40-43] Black women with PRE also experience worse hospital outcomes when compared to White women.[44] In an attempt to assess if echocardiographic changes may explain differences in outcomes, our subtype-adjusted analysis highlighted some remodeling changes such as increased PWT in Black women. A prior study revealed that in a general hypertensive population, Black patients were more likely to have greater PWT, LV mass, and RWT than White patients.[45]

Pericardial effusions are common during pregnancy and are often asymptomatic and trace or mild in size.[46] We found patients with HDP were more likely to develop pericardial effusion, and GHTN and PRE had more than just trace pericardial effusion, albeit not large or complicated by tamponade, similar to prior reports.[47]

Our observations from a large sample size uniquely highlight the differences between various subtypes of HDP and the persistence of the echocardiographic changes postpartum and differentiate the patterns of LV remodeling in women with HDP. We identify women with SPE as the highest risk group, which parallels the adverse prognostic data in these women. The presence of severe features, reflective of multiorgan involvement, is also associated with the worst cardiac abnormalities, potentially identifying short-term and/or long-term cardiovascular risk. Multiple studies have established HDP as a predictor of cardiovascular disease later in life.[8,48] Echocardiography has proven to have an important prognostic role in the general population in predicting adverse outcomes such as using LVMi and E/e' to predict ischemic heart disease and heart failure in chronic hypertensive patients.[48-50] The findings of our study can potentially influence future guidance on the surveillance and management of this high-risk obstetric population with HDP in the peripartum period.

STUDY LIMITATIONS. Despite the strengths of this study noted earlier, the findings of this study should be interpreted with the following limitations due to the retrospective study design and inherent data limitations. Echocardiograms were performed based on clinician discretion and may have only included those with cardiac symptoms or other suspected cardiac abnormalities, thereby adding some selection bias. However, the consistency of our results with prior studies underscores the applicability of these results to all women with HDP. We combined intrapartum and postpartum echocardiograms in the analysis which does not help determine the best timing for echocardiographic assessment. However, a comparative subset analysis, as well as interaction analysis, found that regardless of the timing of echocardiography, differences reported in LV diastolic function and remodeling persisted. Future studies examining serial changes in echocardiography in the long term can further clarify these questions. Moreover, our control group did not include those with chronic hypertension without SPE, which can potentially provide insight into how SPE alters cardiac geometry and diastolic function in a population with chronic hypertension. Finally, the role of medication use and BP control during pregnancy and thereafter could impact the echocardiographic changes, but this information was not consistently available and therefore not included in the analysis.

CONCLUSIONS

Left ventricular remodeling and diastolic filling abnormalities occur in HDP and are most evident in SPE and PRE. Echocardiography during or immediately after pregnancy may be useful in these high-risk women to identify these abnormalities. The long-term implications of these echocardiographic abnormalities on cardiovascular outcomes require further study.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: The current study identifies distinctive echocardiographic abnormalities among subtypes of HDP, persistent postpartum changes, and varying LV remodeling patterns in affected women. Superimposed preeclampsia has the highest degree of remodeling, mirroring adverse prognostic data.

COMPETENCY IN PATIENT CARE: Abnormalities in established prognostic indicators such as E/e' and LV mass index are often seen in women with HDP. Echocardiography can play a pivotal role in early identification of LV remodeling changes in women with HDP, especially among those with preeclampsia and chronic hypertension with superimposed preeclampsia.

TRANSLATIONAL OUTLOOK: While the current study provides valuable short-term insights into echocardiographic changes in HDP, longer-term follow-up is essential to fully understand how these are reflected on future cardiovascular outcomes.

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KEY WORDS **echocardiography, gestational, hypertension, preeclampsia, pregnancy, remodeling**

APPENDIX **For supplemental tables, please see the online version of this paper.**