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Early cardiovascular changes in hypertensive pregnancies: insights from left atrial strain and compliance

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Abstract

Background Hypertensive disorders of pregnancy (HDP), which include gestational hypertension and preeclampsia, are associated with adverse maternal and fetal outcomes and may result in subclinical cardiac dysfunction. Traditional echocardiographic parameters, including the ejection fraction and Doppler indices, often fail to detect early myocardial and atrial abnormalities.

Objective To evaluate early cardiovascular changes in pregnant women with hypertensive disorders, with an explicit focus on left atrial (LA) strain and compliance assessed through speckle-tracking echocardiography.

Methods A prospective observational study was conducted at a tertiary care center, enrolling 73 pregnant women from 20 weeks of gestation onward: 38 with HDPs and 35 with normotensive pregnancies. Transthoracic echocardiography, including two-dimensional speckle tracking, was used to assess global longitudinal strain (GLS), LA strain (reservoir, conduit, contraction), and LA compliance (LASr/E/e'). Standard Doppler and tissue Doppler indices were also evaluated. Logistic regression was used to identify independent predictors of HDP.

Results Compared with controls, women with HDPs presented significantly lower LA reservoir strain (31.00% vs. 35.00%, $p = 0.002$), conduit strain (18.00% vs. 24.00%, $p < 0.001$), and LA compliance (3.87 vs. 5.57, $p < 0.001$), despite having a preserved ejection fraction. GLS also decreased (19.00% vs. 21.00%, $p < 0.001$). In the multivariate analysis, decreased LA compliance (OR 0.50, 95% CI 0.33–0.76, $p = 0.001$) and GLS (OR 1.49, 95% CI 1.08–2.03, $p = 0.014$) were identified as independent predictors of HDP.

Conclusion Pregnant women with hypertensive disorders exhibit early subclinical cardiac dysfunction characterized by impaired left atrial (LA) strain and compliance. These advanced echocardiographic parameters may serve as sensitive markers for early cardiovascular changes, providing superior diagnostic value compared with conventional metrics. Routine evaluation of LA strain and compliance could improve risk stratification and guide management in hypertensive pregnancies.

Keywords Preeclampsia, Hypertensive disorders of pregnancy, Left atrial strain, LA compliance, Speckle-tracking echocardiography, Diastolic dysfunction

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Introduction

Physiological cardiovascular adaptations in pregnancy

Normal pregnancy induces significant cardiovascular adaptations to support the metabolic and circulatory demands of the growing fetus. Cardiac output increases by approximately 30–50% due to elevated stroke volume and heart rate. Concurrently, systemic vascular resistance decreases under the influence of pregnancy-related vasodilators, leading to a mid-pregnancy decline in blood pressure [1]. These changes include mild cardiac chamber dilation and concentric hypertrophy, which are typically well tolerated in healthy pregnancies [2]. However, they reflect the considerable hemodynamic burden placed on the maternal heart and vasculature during gestation [3].

Hypertensive disorders of pregnancy and associated risks

Hypertensive disorders of pregnancy (HDP), which include gestational hypertension and preeclampsia, affect approximately 5–10% of pregnancies [4]. Among HDP, preeclampsia occurs in an estimated 2–8% of cases and is a leading cause of maternal and fetal morbidity [5–7]. Clinically, these disorders range from mild blood pressure elevation to severe multiorgan dysfunction and pose considerable risks to both mother and fetus. HDP account for a significant proportion of maternal deaths globally, with estimates suggesting over 70,000 maternal and 500,000 fetal deaths annually [7].

Beyond the acute perinatal period, HDP have important long-term cardiovascular implications. Women with a history of preeclampsia or gestational hypertension face increased risks of chronic hypertension, ischemic heart disease, stroke, and heart failure later in life [8]. These observations have contributed to the growing recognition of HDP as an early and sex-specific risk factor for future cardiovascular disease, underscoring the importance of timely identification and follow-up in this population.

Limitations of conventional echocardiographic markers

Echocardiography is the primary imaging tool for assessing maternal cardiac function during and after pregnancy. However, traditional echocardiographic measures such as left ventricular ejection fraction (LVEF) and Doppler indices may fail to detect subtle myocardial or atrial dysfunction associated with HDP. LVEF often remains preserved in women with gestational hypertension or preeclampsia, potentially masking early systolic impairment [2]. Diastolic function indicators (e.g., E/A ratio, e' velocities) can suggest elevated filling pressures but are relatively insensitive to early-stage changes. Structural alterations such as mild LA enlargement or LV hypertrophy may occur but typically appear later in disease progression and lack specificity [9, 10]. Therefore, conventional echocardiographic evaluation may

underestimate the presence of subclinical cardiovascular dysfunction in HDP.

Advanced echocardiographic techniques (strain analysis and LA function)

Speckle-tracking echocardiography (STE) provides a more sensitive assessment of myocardial mechanics by measuring myocardial deformation. Global longitudinal strain (GLS) of the left ventricle has been shown to decline in preeclampsia despite preserved LVEF, signaling early systolic dysfunction [11, 12]. STE can also be used to evaluate left atrial (LA) function across its reservoir, conduit, and contraction phases. The LA reservoir strain (LASr) reflects LA expansion during ventricular systole and is a sensitive marker of LA compliance. Reduced LASr and LASr/E/ e' ratios—used to estimate LA compliance—have been associated with elevated LV filling pressures and diastolic dysfunction in hypertensive pregnancies [13]. These advanced parameters may detect cardiac changes earlier than traditional echocardiographic metrics, allowing for earlier identification of patients at risk.

Rationale and objective

Given the hemodynamic demands of pregnancy and the cardiovascular risks associated with HDP, there is a need for more sensitive tools to detect early cardiac dysfunction. LA strain and compliance assessment through speckle-tracking echocardiography may unmask subclinical atrial and myocardial changes not captured by conventional imaging. The present study aims to evaluate left atrial mechanics—including reservoir strain, conduit strain, and compliance (LASr/E/ e')—in pregnant women with HDP compared to normotensive controls. Additionally, we aim to assess whether these parameters can serve as early indicators of cardiovascular involvement and contribute to improved risk stratification in pregnancies complicated by hypertension.

Materials and methods

Study design and ethical approval

This was a prospective observational case-control study conducted at Vilnius University Hospital Santaros Clinics between December 2021 and November 2023. The study was approved by the Biomedical Research Ethics Committee of the Vilnius Region (Approval No. 2020/11-1282-763, dated November 24, 2020). Written informed consent was obtained from all participants before enrollment.

Study population

Pregnant women from the 20th gestational week onward were enrolled and assigned to one of two groups:

1. The hypertensive disorders of pregnancy (HDP) group included women diagnosed with gestational hypertension, preeclampsia, or eclampsia.
2. The control group consisted of women with normotensive, uncomplicated pregnancies.

This case-control design enabled direct comparison between women with and without HDP. While participants were not individually matched, the groups were assessed for baseline comparability, including maternal age, height, weight, and body mass index (BMI). Where differences were observed, they were statistically adjusted in subsequent analyses.

Exclusion criteria included maternal age under 18 years, preexisting renal or cardiovascular disease, chronic hypertension, multiple pregnancies, and known fetal anomalies.

Preeclampsia was diagnosed in accordance with the criteria of the American College of Obstetricians and Gynecologists (ACOG): systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg on two separate occasions at least 4 h apart in a previously normotensive woman, in combination with proteinuria ≥ 0.3 g/24 h.

Participants were enrolled from 20 to 38 weeks of gestation, depending on clinical presentation. Mean gestational age at the time of echocardiographic assessment was reported for both groups in the Results section. Given the physiological changes in cardiac structure and function that occur throughout pregnancy, the variability in gestational age at assessment was acknowledged and is discussed as a limitation of the study.

In the HDP group, echocardiographic evaluations were performed either prior to the initiation of antihypertensive therapy or within the first 24 h following the start of treatment. This timing was recorded to minimize the influence of medication on echocardiographic parameters, although the potential impact of early treatment cannot be fully excluded and is noted as a study limitation.

Blood pressure measurement

Blood pressure (BP) was measured via a validated automated oscillometric device (Omron) in accordance with international guidelines for the management of hypertension during pregnancy. All measurements were taken while the participants were in a seated position after at least 5 min of rest in a quiet environment, with the arm supported at heart level and an appropriately sized cuff placed on the upper arm. Two consecutive measurements were recorded at 1-minute intervals, and the average was used for analysis. A diagnosis of gestational hypertension or preeclampsia was made if the SBP was ≥ 140 mmHg and/or the DBP was ≥ 90 mmHg on two measurements taken at least 4 h apart.

Echocardiographic assessment

Transthoracic echocardiography was performed via a GE Vivid E95 ultrasound system with an M5Sc-D phased-array transducer. Examinations were conducted in accordance with the guidelines of the American Society of Echocardiography (ASE) and the European Association of Cardiovascular Imaging (EACVI), utilizing participants in the left lateral decubitus position. All scans were performed by an experienced cardiologist blinded to the clinical and laboratory data.

Standard two-dimensional (2D), M-mode, pulsed-wave Doppler, and tissue Doppler imaging data were obtained. Left ventricular (LV) diastolic function was evaluated via mitral inflow velocities (E and A), the E/A ratio, deceleration time (DT), and early diastolic myocardial velocities (septal and lateral e'). The average E/ e' ratio was calculated to estimate the LV filling pressure.

Left ventricular end-diastolic pressure (LVEDP) was estimated using the Nagueh formula: $LVEDP = 1.9 + (1.24 \times E/e')$. This formula has been previously validated in clinical studies [14].

Assessment of the left atrial strain and compliance

Two-dimensional speckle-tracking echocardiography (2D-STE) was utilized to assess left atrial (LA) deformation. Left atrial reservoir strain (LASr), conduit strain (LAScd), and contraction strain (LASct) were quantified in the apical four-chamber view, following EACVI recommendations. LA compliance was determined as the ratio of LASr to E/ e' (i.e., $LASr/E/e'$).

The following reference values were used on the basis of prior normative data:

- LVEF $> 51.7\%$
- LASr $> 31\%$
- LA compliance ($LASr/E/e'$) > 3

Early systolic dysfunction was defined by global longitudinal strain (GLS) $< 17.8\%$. Mild diastolic dysfunction was defined as follows:

- Septal $e' < 7$ cm/s.
- Lateral $e' < 10$ cm/s.

These parameters were employed to identify subclinical cardiac dysfunction in women experiencing hypertensive disorders during pregnancy.

Statistical analysis

The normality of the data distribution was tested via the Shapiro–Wilk test. Continuous variables are reported as either medians and interquartile ranges (IQRs) or means \pm standard deviations (SDs), depending on their distribution. Group comparisons were made via the

Mann–Whitney U test for nonnormally distributed variables and the independent-samples t test for normally distributed data. Categorical variables were compared via Fisher's exact test.

Correlation analysis was performed via Spearman's rank correlation for nonparametric data and Pearson's correlation for parametric data. Binary logistic regression was utilized to identify independent predictors of hypertensive disorders. A two-tailed p value of <0.05 was considered statistically significant. Statistical analyses were conducted via IBM SPSS Statistics, version 30.0.

Results

Baseline characteristics of the study population

The baseline characteristics of the study participants are presented in Table 1. A total of 73 pregnant women were enrolled in the study and divided into two groups: the control group (K), consisting of 35 women with uncomplicated pregnancies, and the study group (HDP), composed of 38 women diagnosed with hypertensive disorders of pregnancy, such as gestational hypertension, preeclampsia, or eclampsia.

Initially, the HDP group was divided into two subgroups: women with gestational hypertension (H group, $n=7$) and women with preeclampsia or eclampsia (P group, $n=31$). However, as no statistically significant differences were found between these subgroups for most parameters, they were combined into a single HDP group for further analysis.

A statistically significant difference in maternal age was observed between the groups ($p=0.040$), with women in the HDP group being older:

- Control group: 30.0 years (26.50–32.00).
- HDP group: 32.50 years (29.00–35.00).

Significant differences in blood pressure values were observed between the groups for systolic, diastolic, and mean arterial pressure (all $p<0.001$), with notably greater values in the HDP group. Additionally, the heart rate was significantly lower in the HDP group ($p=0.021$).

Statistical comparisons were performed via the Mann–Whitney U test for continuous variables.

Echocardiographic parameters

Significant differences in echocardiographic parameters were observed between the groups, as presented in Table 2. Compared with those in the control group, women in the HDP group had a significantly greater left ventricular mass index (LVMI) and lower cardiac output. The left atrial area, assessed in both two- and four-chamber views, was significantly larger in the HDP group ($p<0.001$ for both).

Although conventional Doppler parameters such as E, A, the E/A ratio, and deceleration time did not differ significantly between groups, clear distinctions were evident in the tissue Doppler indices. The HDP group presented significantly lower e' septal and e' lateral values, as well as higher E/ e' ratios ($p<0.001$), indicating impaired diastolic function. Additionally, the estimated pulmonary capillary wedge pressure (PCWP) was significantly elevated in the HDP group.

A significant reduction in left ventricular global longitudinal strain (GLS) was observed in the HDP group despite no difference in ejection fraction. With respect to left atrial strain, women with hypertensive disorders presented significantly lower conduit and reservoir strain values, reflecting early alterations in atrial function. The maximum left atrial volume (LAVmax) was also significantly greater in the HDP group.

Statistical comparisons were performed via the Mann–Whitney U test.

Analysis of LA compliance and diastolic dysfunction

A statistically significant reduction in left atrial compliance (LASr/E/ e') was observed in the HDP group compared with the control group, indicating that impaired atrial mechanical function was linked to elevated filling pressures.

Although the prevalence of reduced left ventricular ejection fraction (LVEF $<51.7\%$) was very low and not significantly different between groups, subclinical atrial dysfunction was more common among women with hypertensive disorders. In the HDP group, 47.4% of the samples had a reduced LA reservoir strain (LASr $<31\%$), whereas 22.9% of the samples in the control group had a reduced LA reservoir strain ($p=0.026$). Similarly, a

Table 1 Baseline characteristics of the study population

Parameter	Control group ($n=35$)	HDP group ($n=38$)	p value
Age, years	30.0 [26.50–32.00]	32.50 [29.00–35.00]	0.040
Systolic blood pressure, mmHg (sBP)	115.0 [103.50–120.00]	150.0 [141.00–161.00]	<0.001
Diastolic blood pressure, mmHg (dBP)	70.0 [65.50–76.50]	92.0 [83.00–100.00]	<0.001
Mean arterial pressure, mmHg (MAP)	84.0 [76.50–91.00]	101.0 [97.00–107.00]	<0.001
Heart rate, bpm (HR)	80.0 [70.00–87.50]	70.0 [62.00–78.00]	0.021

All values are expressed as medians [interquartile ranges]

Abbreviations: sBP systolic blood pressure, dBP diastolic blood pressure, MAP mean arterial pressure, HR heart rate

Table 2 Echocardiographic parameters and their comparison between groups

Parameter	Control group (n = 35)	HDP group (n = 38)	p value
LV mass index (g/m ²)	66.20 [62.95–75.15]	76.40 [68.20–78.55]	0.029
Cardiac output (L/min)	5.20 [4.45–5.65]	4.45 [4.03–4.99]	0.012
LVEF (%)	60.00 [58.00–62.50]	60.25 [58.00–62.00]	0.568
LA area, 2-chamber (cm ²)	19.00 [17.05–20.25]	21.50 [19.10–22.80]	< 0.001
LA area, 4-chamber (cm ²)	18.90 [17.50–21.00]	21.95 [19.30–23.40]	< 0.001
E velocity (m/s)	0.87 [0.76–0.98]	0.855 [0.73–0.98]	0.497
A velocity (m/s)	0.63 [0.55–0.70]	0.61 [0.56–0.69]	0.864
E/A ratio	1.37 [1.25–1.59]	1.34 [1.14–1.61]	0.500
Deceleration time (ms)	197.00 [168.50–216.00]	193.50 [169.00–235.00]	0.821
e' septal (cm/s)	11.00 [10.00–12.00]	10.00 [8.00–11.00]	< 0.001
e' lateral (cm/s)	17.00 [15.50–18.00]	12.00 [11.00–14.00]	< 0.001
E/e' lateral	5.11 [4.50–5.78]	6.57 [5.38–8.50]	< 0.001
E/e' septal	7.90 [6.82–9.00]	9.20 [7.50–10.80]	0.017
E/e' average	6.26 [5.38–7.09]	7.62 [6.47–9.61]	< 0.001
PCWP (Nagueh, mmHg)	8.37 [7.62–9.73]	10.04 [8.92–12.44]	< 0.001
LV global longitudinal strain (%)	21.00 [19.00–21.00]	19.00 [18.00–20.50]	< 0.001
LV stroke volume (mL)	68.00 [62.00–75.00]	65.75 [60.00–71.00]	0.532
LV end-diastolic volume (mL)	108.00 [98.50–119.00]	108.00 [98.00–118.00]	0.799
LV end-systolic volume (mL)	40.00 [39.00–45.50]	43.00 [37.00–48.00]	0.736
LA conduit strain (%)	24.00 [20.00–26.00]	18.00 [15.00–20.00]	< 0.001
LA contraction strain (%)	12.00 [9.50–13.50]	13.00 [10.00–15.00]	0.220
LA reservoir strain (%)	35.00 [31.00–39.00]	31.00 [29.00–33.00]	0.002
LA volume (LAVmax, BIP, mL)	49.25 [45.50–54.50]	59.25 [50.00–68.00]	0.008

Strain parameters: conduit strain – passive LA emptying; contraction strain – active LA emptying; reservoir strain – LA filling during ventricular systole. All values are expressed as medians [interquartile ranges]

Abbreviations: LV left ventricle, LA left atrium, LVEF left ventricular ejection fraction, MMI myocardial mass index, CO cardiac output, E early mitral inflow velocity, A late mitral inflow velocity e' early diastolic tissue velocity, PCWP pulmonary capillary wedge pressure (estimated by the Nagueh formula), LAVmax maximum left atrial volume, BIP biplane method

Table 3 Diastolic dysfunction and left atrial compliance parameters

Parameter	Control group (n = 35)	HDP group (n = 38)	p value
LA compliance (LASr/E/e')	5.57 [5.07–6.74]	3.87 [3.09–5.10]	< 0.001
LVEF < 51.7%	1 (2.9%)	1 (2.6%)	0.732
LASr < 31%	8 (22.9%)	18 (47.4%)	0.026
LA compliance < 3.0	1 (2.9%)	9 (23.7%)	0.010
Septal e' < 7 cm/s	0 (0%)	1 (2.6%)	0.521
Lateral e' < 10 cm/s	0 (0%)	2 (5.3%)	0.268

Abbreviations: LASr left atrial reservoir strain, E/e' estimated LV filling pressure, LVEF left ventricular ejection fraction, e' early diastolic tissue velocity

significantly greater proportion of participants in the HDP group had low LA compliance (< 3.0) (23.7% vs. 2.9%, $p = 0.010$).

Impaired early diastolic tissue velocities (e') were rare in both groups, with only isolated cases noted in the HDP group and no statistically significant differences observed. These findings suggest that LA compliance and strain parameters may serve as sensitive markers of early diastolic dysfunction in pregnant women with hypertensive conditions, even in the absence of overt systolic impairment or abnormal Doppler velocities.

The data are presented in Table 3. Statistical comparisons were conducted via the Mann–Whitney U test and Fisher's exact test.

Logistic regression analysis

According to a stepwise logistic regression model, reduced left atrial compliance (LASr/E/e') was independently associated with hypertensive disorders during pregnancy (OR = 0.50, 95% CI: 0.33–0.76, $p = 0.001$), indicating that lower compliance was significantly linked to increased odds of HDP. Additionally, global longitudinal strain (GLS) was also an independent predictor (OR = 1.49, 95% CI: 1.08–2.03, $p = 0.014$), suggesting that both parameters reflect early subclinical myocardial changes in hypertensive pregnancies.

The data are presented in Table 4. Statistical significance was determined via binary logistic regression analysis (stepwise method).

Discussion

LA strain and subclinical diastolic dysfunction in HDP

Our study demonstrated that pregnant women with hypertensive disorders of pregnancy (HDP) present significantly lower left atrial (LA) reservoir strain (LASr)

Table 4 Logistic regression analysis for predicting hypertensive disorders during pregnancy

Variable	B	SE	Wald	p value	OR (Exp(B))	95% CI for OR
LA compliance (LASr/E/e')	−0.685	0.211	10.560	0.001	0.504	0.333–0.762
GLS (Global Longitudinal Strain)	0.395	0.160	6.077	0.014	1.485	1.084–2.033
Constant	11.322	3.371	11.279	< 0.001	82654.820	–

Abbreviations: B regression coefficient, SE standard error, Wald Wald chi-square test, Exp(B) odds ratio (OR), CI confidence interval, GLS global longitudinal strain, LA left atrium, LASr left atrial reservoir strain, E/e' estimated LV filling pressure

and conduit strain than normotensive controls do. This reduction in LA strain parameters, along with preserved ejection fraction, suggests the presence of subclinical diastolic dysfunction. LA reservoir strain serves as a sensitive indicator of left ventricular (LV) filling pressure and compliance, and a decrease in LASr often reflects increased LA stiffness or elevated LV end-diastolic pressure [15, 16]. In the context of HDP, a lower LASr implies that the left atrium (LA) is less able to expand during ventricular systole, which is consistent with higher filling pressures despite the absence of overt heart failure symptoms. The significantly reduced LA compliance (assessed as LASr/E/e') in the HDP group further supports this interpretation. LA compliance reflects the ability of the left atrium (LA) to accommodate blood (strain) for a given filling pressure; a decrease in this ratio serves as an early sign of diastolic dysfunction. Our findings align with this pathophysiology: even though conventional Doppler measures (E/A ratio, E/e') may still fall within normal ranges for many HDP patients, the depressed LA strain and compliance indicate that the myocardium is already under strain from elevated pressures. This is a hallmark of “heart failure with preserved ejection fraction (HFpEF)-like” changes occurring at a subclinical stage. Practically, HDP appears to induce early myocardial remodeling, wherein the LA becomes stiffer and less efficient as a reservoir, reflecting the hemodynamic burden of hypertension during pregnancy. This subclinical diastolic dysfunction has been noted in prior studies of preeclampsia, where the severity of diastolic dysfunction correlated with disease severity [17]. Our results reinforce that HDP, even before clinically evident cardiac dysfunction, is associated with measurable impairments in cardiac function. This underscores the need for vigilant cardiovascular evaluation in these patients, as subtle diastolic dysfunction may progress if unrecognized.

In addition, recent findings suggest that left atrial mechanics may also be altered in normotensive pregnancies that later develop fetal growth restriction (FGR). Vasapollo et al. demonstrated that left atrial function was reduced at mid-gestation in women who subsequently developed FGR, even in the absence of hypertension [18]. This observation reinforces the notion that advanced echocardiographic parameters may unmask early subclinical dysfunction with higher sensitivity and specificity than conventional measures, not only in HDP but also

in pregnancies considered normotensive at the time of evaluation.

Comparison with previous studies on LA mechanics in HDP

Our observations align with emerging evidence that hypertensive pregnancy disorders negatively impact left atrial (LA) function. A recent systematic review and meta-analysis by Sonaglioni et al. examined eight studies (566 HDP patients vs. 420 controls) and revealed that the LA reservoir strain was significantly lower in women with hypertensive disorders during pregnancy (mean ~ 34% vs. ~ 43% in controls) [17]. Similarly, the LA conduit strain was reduced (~ 23% vs. ~ 32% in controls) [19]. Notably, a meta-analysis revealed that LA contractile strain (the active booster pump function) was not significantly different between HDP and normotensive pregnancies [19]. This finding suggests that in hypertensive pregnancies, the passive phases of LA filling and emptying (reservoir and conduit) are primarily impaired, whereas active contraction (which is often augmented as a compensatory mechanism) may be relatively preserved. Our study also did not find a significant change in LA contractile function, in line with these findings.

In addition to these aggregate findings, Sonaglioni et al. also demonstrated in a separate prospective study that reduced global LA peak reservoir strain (LASr) in women with new-onset gestational hypertension was independently associated with the persistence of hypertension at one-year postpartum [20]. This highlights the prognostic value of LASr, suggesting it not only reflects acute hemodynamic impairment but may also identify patients at risk of long-term cardiovascular morbidity. These results align with our observations and reinforce the role of LA strain as both a diagnostic and prognostic marker in HDP.

Several individual studies support our findings. Li et al. investigated LA strain in preeclampsia patients and reported significantly decreased LASr and LA conduit strain in PE patients compared with healthy pregnant controls [13]. Importantly, they also reported a significant reduction in the LA compliance index (LASr/E/e') in preeclampsia [13], which aligns with our results. Cong et al. similarly reported decreased LA strain in women with preeclampsia, along with increased LV mass, indicating concentric remodeling in response to hypertension [21]. These studies collectively reinforce that LA functional

impairment is a consistent feature of HDP, even if standard echocardiographic measures appear normal.

With respect to left ventricular (LV) function, our finding of lower global longitudinal strain (GLS) in the hypertensive disorders of pregnancy (HDP) group, despite preserved ejection fraction, is well supported by the literature. Numerous investigations have shown that LV GLS is significantly reduced in preeclampsia and gestational hypertension, reflecting subtle systolic dysfunction. In a systematic review of speckle-tracking echocardiography in hypertensive pregnancies, 12 of 13 studies reported decreased GLS in women with HDP compared with normotensive controls [22]. For example, one study reported that women with preeclampsia presented significantly worse longitudinal, radial, and circumferential strain than normotensive pregnant women did despite no difference in LVEF [23]. These decreases in GLS reflect our data and highlight the notion that HDP can induce diffuse myocardial dysfunction that is not apparent in conventional measures such as EF. Moreover, some of these impairments may continue after pregnancy: women with a history of severe or early-onset preeclampsia have been found to have persistent reductions in strain indices from months to years postpartum [22]. O'Driscoll et al. recently demonstrated that, at six months postpartum, women who experienced preterm preeclampsia still presented significantly reduced left atrial (LA) reservoir and conduit strain, as well as increased LA stiffness, compared with those of postpartum controls [15]. Interestingly, those LA abnormalities were observed even in women without postpartum hypertension or overt ventricular dysfunction [15]. This lasting impact highlights that the cardiac changes associated with HDPs are not just acute effects of pregnancy but may indicate an underlying myocardial phenotype that presents long-term risk.

Overall, our findings align with the broader context of HDP-related cardiac remodeling: a pattern of subclinical diastolic dysfunction (impaired left atrial function, elevated filling pressures) combined with subtle systolic dysfunction (decreased global longitudinal strain, GLS) has been consistently reported in the literature [22, 23]. We contribute to this body of evidence by demonstrating that these changes occur early in the progression of HDP and can be detected in vivo via advanced echocardiographic techniques.

Diagnostic value of LA strain and compliance vs. conventional markers

The notable changes in left atrial (LA) strain and compliance in hypertensive disorders of pregnancy (HDP) underscore the diagnostic superiority of these metrics over traditional echocardiographic markers for detecting early cardiac dysfunction. Conventional Doppler indices

of diastolic function (E/A ratio, deceleration time, E/e') and even LA size often remain within normal ranges during pregnancy, or alterations may be too subtle to indicate an abnormality. In our cohort, standard measures suggested "preserved" systolic and diastolic function according to guideline criteria, yet strain analysis revealed significant functional impairment. This phenomenon is well documented: strain imaging can reveal cardiac dysfunction that may be overlooked by ejection fraction or Doppler measures [23]. Shahul et al. demonstrated that women with preeclampsia exhibited significantly reduced LV strain despite having a normal EF, indicating that speckle-tracking strain is more sensitive than EF for the early detection of systolic dysfunction [23]. Similarly, early-stage diastolic dysfunction may not meet formal diagnostic thresholds; however, LA strain can still be abnormal despite "normal" filling pressure estimates. Jarasūnas et al. demonstrated that hypertensive patients, even those with intermittent atrial fibrillation, exhibited reduced left atrial (LA) reservoir and conduit strain, even in the absence of any Doppler evidence of diastolic dysfunction. Furthermore, worsening diastolic function correlated with progressively lower LA strain [16]. These findings suggest that LA strain is an earlier marker of impaired relaxation and increased chamber stiffness than conventional Doppler parameters.

In our study, multivariate logistic regression identified LA compliance (LASr/E/e') and LV GLS as independent predictors of HDP, whereas traditional markers did not exhibit such robust associations. This highlights that LA compliance and GLS provide additive diagnostic information. LA compliance appears to capture the interplay between LA function and filling pressure in a single measurement. Recent research suggests that LA compliance may outperform LASr alone in the early detection of cardiac alterations in conditions such as preeclampsia. Sun et al. reported that in preeclamptic women with otherwise "normal" echocardiograms (normal EF and no gradable diastolic dysfunction), the proportion with abnormal LASr/E/e' was significantly greater than that with abnormal LASr alone [13]. In other words, the LASr/E/e' ratio indicated subtle dysfunction earlier than LASr alone, suggesting that a mild elevation in filling pressure (increasing E/e') combined with a mild reduction in strain can create an abnormal compliance index, even when each component is individually borderline. This finding aligns with our results: many HDP patients may have an E/e' in the upper "normal" range and a moderately reduced LASr; this combination yields a low compliance index, identifying patients with "occult" diastolic dysfunction. Thus, the LA strain and compliance serve as early warning signs. They integrate information about myocardial deformation and filling pressure that single conventional metrics cannot provide. In practical terms,

these advanced indices could enable clinicians to detect cardiac involvement in HDPs more reliably and sooner than relying on, for example, an E/e' cutoff or the presence of symptoms.

Notably, LA strain and compliance can potentially be used to track disease severity. Li et al. reported that LA compliance ($LA_{Sr}/E/e'$) progressively worsened with increasing diastolic dysfunction grade in preeclampsia patients, and very low compliance ($LA_{Sr}/E/e' < 3.4$) was an independent predictor of acute cardiac complications in these patients [13]. This finding indicates a role not only in diagnosis but also in risk stratification: patients with the most depressed left atrial (LA) function are more likely to experience pulmonary edema, severe hypertensive crises, or other cardiac events during hypertensive disorders of pregnancy (HDP). Conventional markers alone (e.g., blood pressure values or proteinuria levels) may not capture that risk as directly as a targeted cardiac functional measure does. Taken together, these findings emphasize that incorporating LA strain and compliance into echocardiographic assessments offers a more comprehensive evaluation of cardiovascular health during pregnancy. These findings enhance our diagnostic toolkit, revealing subclinical myocardial changes that hold prognostic significance. By detecting HDP-related cardiac dysfunction earlier and more accurately, LA strain and global longitudinal strain (GLS) could enable timely interventions (e.g., intensifying blood pressure control and adjusting delivery timing) to prevent progression to overt heart failure or other complications.

Clinical implications for early screening and monitoring

The observed reductions in left atrial (LA) strain, LA compliance, and global longitudinal strain (GLS) in women with hypertensive disorders of pregnancy (HDP) have significant clinical implications for managing pregnancies complicated by hypertension. First, these parameters could be utilized for early screening of cardiovascular involvement in pregnant women at risk. For example, in a woman with new-onset gestational hypertension or mild preeclampsia, a targeted echocardiogram assessing LA strain and GLS may reveal subclinical dysfunction. Identifying such changes could lead to closer monitoring and proactive management. Patients who show significantly impaired LA strain or compliance might require more aggressive antihypertensive treatment or more frequent maternal–fetal assessments, as they could be at greater risk for worsening cardiac stress or pulmonary edema. As Sonaglioni et al. reported, strain echocardiography can aid in identifying HDP patients who would benefit from tighter blood pressure control and closer follow-up to mitigate adverse outcomes [19]. This

personalized approach could enhance maternal safety by detecting early signs of cardiac decompensation.

Moreover, these findings strengthen the argument for integrating cardiovascular surveillance into routine care for women with HDPs. Current obstetric practices focus on blood pressure control and symptom monitoring; however, our data (and other data) suggest that adding an echocardiographic evaluation of heart function can reveal important risk information. In an era in which preeclampsia is recognized as a predictor of future heart disease, pregnancy can be viewed as a “stress test” for the mother’s heart. Abnormal LA strain or GLS during pregnancy might not only predict immediate peripartum complications but also identify women who require long-term cardiometabolic follow-up after delivery. For example, persistent LA strain reduction postpartum, as observed by O’Driscoll et al. [15], might suggest a persistent vulnerability that could develop into heart failure or chronic hypertension years later. Indeed, epidemiological studies indicate that women with a history of preeclampsia face a significantly increased risk of cardiovascular disease postpartum, including a fourfold greater risk of heart failure later in life [24]. By incorporating LA strain and compliance measures into post-HDP evaluations, clinicians may be able to identify which women are at greatest risk and require preventive interventions, such as lifestyle modifications, cardiology referrals, or closer blood pressure monitoring. It is conceivable that one day, LA strain could be utilized similarly to how obstetricians currently apply certain biomarkers as part of a risk score for maternal cardiovascular health outcomes.

Another clinical implication is guiding the timing of delivery or escalation of care. In cases of severe preeclampsia, if echocardiography reveals severely depressed left atrial (LA) compliance or evolving systolic dysfunction (despite maximal medical therapy), this may support a decision for early delivery to prevent maternal cardiac decompensation. Conversely, reassuring strain values could instill confidence in continuing the pregnancy with careful observation. Furthermore, these parameters can inform anesthetic management; for example, anesthesiologists may choose specific techniques if they are aware that the patient has diastolic dysfunction to avoid fluid overload. In summary, the use of LA strain and global longitudinal strain (GLS) enhances our ability to holistically manage hypertensive pregnancies, extending beyond mere blood pressure treatment. Early detection of cardiac changes enables obstetricians and cardiologists to collaborate more effectively, implement interventions to reduce cardiac workload, and plan

safe delivery and postpartum strategies tailored to the mother's cardiovascular status.

Conclusions

This study demonstrated that pregnant women with hypertensive disorders exhibit subclinical cardiac dysfunction, which is characterized primarily by reduced left atrial (LA) compliance and impaired myocardial deformation. Although conventional echocardiographic parameters such as ejection fraction and Doppler indices remained within normal limits, LA reservoir strain (LASr), LA compliance (LASr/E/e'), and global longitudinal strain (GLS) were significantly altered in the hypertensive group.

Stepwise logistic regression analysis revealed that both reduced LA compliance and impaired GLS were independent predictors of hypertensive disorders during pregnancy. These findings suggest that speckle-tracking echocardiography may serve as a sensitive tool for the early detection of cardiovascular alterations in this population, even before overt systolic or diastolic dysfunction becomes apparent.

Advanced echocardiographic parameters, particularly LA compliance and strain, may offer added clinical value for early risk stratification and individualized management of women at risk for preeclampsia and related complications.

In conclusion, our study contributes to a growing body of evidence that hypertensive disorders during pregnancy are associated with early, subclinical cardiovascular changes. LA strain and compliance have emerged as powerful tools for detecting and quantifying subclinical cardiac involvement, offering advantages over conventional echocardiographic markers. These findings not only deepen our understanding of the cardiac effects of HDP, supporting the concept of pregnancy as a stress test for the maternal heart but also have tangible implications for improving maternal care through early detection, personalized management, and postpregnancy follow-up to reduce future cardiovascular risk.

Limitations

This study has several limitations, including the small size of some subgroups, particularly those with gestational hypertension ($n=7$), and the limited subgroup analysis. The fact that the study was conducted at a single center may reduce the generalizability of the findings. The observational, cross-sectional design prevents conclusions about the causality or progression of cardiac changes. The lack of postnatal follow-up or perinatal outcomes limits the assessment of the predictive value of echocardiographic findings for maternal or neonatal prognosis.

Additionally, participants were enrolled across a broad gestational age range (20–38 weeks), which may have influenced left atrial size and function due to physiological changes occurring throughout pregnancy. Although mean gestational age was reported and groups were statistically compared, this variability should be considered when interpreting the findings. Furthermore, although echocardiographic evaluations in the HDP group were performed prior to or shortly after the initiation of antihypertensive treatment, early pharmacological intervention may have influenced strain or compliance values and should be considered when interpreting the results.

Despite these limitations, this study offers crucial insights into early cardiovascular changes in pregnant women with hypertensive disorders and emphasizes the potential role of LA compliance and myocardial strain in clinical assessment.

Abbreviations

HDP	Hypertensive disorders of pregnancy
PE	Preeclampsia
BP	Blood pressure
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
MAP	Mean arterial pressure
HR	Heart rate
LV	Left ventricle
LA	Left atrium
LVEF	Left ventricular ejection fraction
GLS	Global longitudinal strain
LASr	Left atrial reservoir strain
LAScd	Left atrial conduit strain
LASct	Left atrial contraction strain
PCWP	Pulmonary capillary wedge pressure
CO	Cardiac output
LVMI	Left ventricular mass index
LAVmax	Left atrial maximum volume
2D	Two-dimensional
STE	Speckle-tracking echocardiography
EF	Ejection fraction
DT	Deceleration time
E/e'	Ratio of early mitral inflow velocity to early diastolic tissue velocity
ASE	American Society of Echocardiography
EACVI	European Association of Cardiovascular Imaging
ACOG	American College of Obstetricians and Gynecologists
IQR	Interquartile range
SD	Standard deviation
OR	Odds ratio
CI	Confidence interval

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Authors' contributions

All authors contributed to the study's conception and design. Data collection and analysis were performed by the main author, and the manuscript was drafted and revised with input from all co-authors. All authors read and approved the final manuscript.

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Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This study was approved by the Biomedical Research Ethics Committee of the Vilnius Region (Approval No. 2020/11-1282-763, dated November 24, 2020). Written informed consent was obtained from all participants before enrollment.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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References

1. Kopley JM, Bates K, Mohiuddin SS. Physiology, maternal changes. In: StatPearls. Treasure Island: StatPearls Publishing; 2025.
2. Curtis SL, Belham M, Bennett S, James R, Harkness A, Gamlin W, Thilaganathan B, Giorgione V, Douglas H, Carroll A, et al. Transthoracic echocardiographic assessment of the heart in pregnancy—a position statement on behalf of the British society of echocardiography and the United Kingdom maternal cardiology society. *Echo Res Pract*. 2023. <https://doi.org/10.1186/s44156-023-00019-8>.
3. Afari HA, Davis EF, Sarma AA. Echocardiography for the pregnant heart. *Curr Treat Options Cardiovasc Med*. 2021;23: 55. <https://doi.org/10.1007/s11936-021-00930-5>.
4. Khedagi AM, Bello NA. Hypertensive disorders of pregnancy. *Cardiol Clin*. 2021;39:77–90. <https://doi.org/10.1016/j.ccl.2020.09.005>.
5. Karrar SA, Martingano DJ, Hong PL. Preeclampsia. In: StatPearls. Treasure Island: StatPearls Publishing; 2025.
6. Traub A, Sharma A, Gongora MC. Hypertensive disorders of pregnancy: a literature review—pathophysiology, current management, future perspectives, and healthcare disparities. 2023.
7. Rana S, Lemoine E, Granger JP, Karumanchi SA. Preeclampsia. *Circ Res*. 2019;124:1094–112. <https://doi.org/10.1161/CIRCRESAHA.118.313276>.
8. Wu P, Haththotuwa R, Kwok CS, Babu A, Kotronias RA, Rushton C, Zaman A, Fryer AA, Kadam U, Chew-Graham CA, et al. Preeclampsia and future cardiovascular health. *Circ Cardiovasc Qual Outcomes*. 2017;10: e003497. <https://doi.org/10.1161/CIRCOUTCOMES.116.003497>.
9. Mattioli AV, Pennella S, Demaria F, Farinetti A. Atrial remodeling in pregnant hypertensive women: comparison between chronic and gestational hypertension. 2012;6. <https://doi.org/10.2174/1874192401206010009>.
10. Castleman JS, Ganapathy R, Taki F, Lip GYH, Steeds RP, Kotecha D. Echocardiographic structure and function in hypertensive disorders of pregnancy. *Circ Cardiovasc Imaging*. 2016;9: e004888. <https://doi.org/10.1161/CIRCIMAGING.116.004888>.
11. Reddy M, Wright L, Rolnik DL, Li W, Mol BW, La Gerche A, da SilvaCosta F, Wallace EM, Palmer K. Evaluation of cardiac function in women with a history of preeclampsia: a systematic review and meta-analysis. *J Am Heart Assoc*. 2019;8: e013545. <https://doi.org/10.1161/JAHA.119.013545>.
12. Chow YY, Chapman M, Mahadevan D, Dekker G, Arstall M. P1778 preeclampsia is associated with reduced myocardial work efficiency during pregnancy. *Eur Heart J*. 2020;21: jez3191134. <https://doi.org/10.1093/ehjci/jez319.1134>.
13. Li R, Sun F, Piao S, He X, Li R, Xu L, Song G, Cong J. Left atrial strain and compliance correlate with diastolic dysfunction grades and complications during pre-eclampsia: a speckle-tracking echocardiography study. *Ultrasound Med Biol*. 2021;47:3411–9. <https://doi.org/10.1016/j.ultrasmedbio.2021.08.003>.
14. Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quiñones MA. Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. *J Am Coll Cardiol*. 1997;30:1527–33. [https://doi.org/10.1016/S0735-1097\(97\)00344-6](https://doi.org/10.1016/S0735-1097(97)00344-6).
15. O'Driscoll JM, McCarthy FP, Giorgione V, Jalaludeen N, Seed PT, Gill C, Sparkes J, Poston L, Marber M, Shennan AH, et al. Left atrial mechanics following preeclamptic pregnancy. *Hypertension*. 2024;81:1644–54. <https://doi.org/10.1161/HYPERTENSIONAHA.123.22577>.
16. Jarasunas J, Aidietis A, Aidietiene S. Left atrial strain - an early marker of left ventricular diastolic dysfunction in patients with hypertension and paroxysmal atrial fibrillation. *Cardiovasc Ultrasound*. 2018. <https://doi.org/10.1186/s12947-018-0147-6>.
17. Melchiorre K, Sutherland GR, Baltabaeva A, Liberati M, Thilaganathan B. Maternal cardiac dysfunction and remodeling in women with preeclampsia at term. *Hypertension*. 2011;57:85–93. <https://doi.org/10.1161/HYPERTENSIONAHA.110.162321>.
18. Vasapollo B, Novelli GP, Maellaro F, Gagliardi G, Pais M, Silvestrini M, Pometti F, Farsetti D, Valensise H. Maternal cardiovascular profile is altered in the pre-clinical phase of normotensive early and late intrauterine growth restriction. *Am J Obstet Gynecol*. 2025;232:312e1. 312.e21.
19. Sonaglioni A, Pusca I, Casieri F, Dell'Anna R, Luigi Nicolosi G, Bianchi S, Lombardo M. Echocardiographic assessment of left atrial mechanics in women with hypertensive disorders of pregnancy: a systematic review and meta-analysis. *Eur J Obstet Gynecol Reproductive Biology*. 2024;299:62–70. <https://doi.org/10.1016/j.ejogrb.2024.05.044>.
20. Sonaglioni A, Lonati C, Lombardo M, Rigamonti E, Binda G, Vincenti A, Nicolosi GL, Bianchi S, Harari S, Anzà C. Incremental prognostic value of global left atrial peak strain in women with new-onset gestational hypertension. *J Hypertens*. 2019;37:1668–75. <https://doi.org/10.1097/HJH.0000000000002086>.
21. Company Calabuig AM, Nunez E, Georgiopoulos G, Nicolaides KH, Charakida M, De Paco matalana C. Three-dimensional echocardiography and strain cardiac imaging in women with pre-eclampsia with follow-up to 6 months postpartum. *Ultrasound Obstet Gynecol*. 2023;62:852–9. <https://doi.org/10.1002/uog.27442>.
22. Moors S, van Oostrum NHM, Rabotti C, Long X, Westerhuis MEMH, Kemps HMC, Oei SG, van Laar JOEH. Speckle tracking echocardiography in hypertensive pregnancy disorders: a systematic review. *Obstet Gynecol Surv*. 2020;75:497–509. <https://doi.org/10.1097/OGX.0000000000000811>.
23. Shahul S, Rhee J, Hacker MR, Gulati G, Mitchell JD, Hess P, Mahmood F, Arany Z, Rana S, Talmor D. Subclinical left ventricular dysfunction in preeclamptic women with preserved left ventricular ejection fraction: a 2D speckle tracking imaging study. *Circ Cardiovasc Imaging*. 2012. <https://doi.org/10.1161/CIRCIMAGING.112.973818>.
24. Mantel Å, Sandström A, Faxén J, Andersson DC, Razaz N, Cnattingius S, Stephansson O. Pregnancy-induced hypertensive disorder and risks of future ischemic and nonischemic heart failure. *JACC Heart Fail*. 2023;11:1216–28. <https://doi.org/10.1016/j.jchf.2023.03.021>.

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