Preeclampsia Is Associated With Persistent Postpartum Cardiovascular Impairment

Karen Melchiorre, George Ross Sutherland, Marco Liberati, Basky Thilaganathan

Abstract—Preeclampsia is associated with asymptomatic global left ventricular abnormal function and geometry during the acute phase of the disorder. These subclinical abnormalities in cardiac findings are known to be important in cardiovascular risk stratification for nonpregnant patients. Furthermore, epidemiological studies have also demonstrated a relationship between preeclampsia and cardiac morbidity and mortality later in life. The aim of this study was to evaluate the postpartum natural history and clinical significance of asymptomatic left ventricular impairment known to occur with acute preeclampsia. This was a prospective longitudinal case-control study of 64 subjects with preeclampsia and 78 matched controls. There were 3 time point assessments, pregnancy and 1 and 2 years postpartum. The assessments included a medical and family history, blood pressure profile, echocardiography, and 12-lead ECG. At 1 year postpartum, asymptomatic left ventricular moderate-severe dysfunction/hypertrophy was significantly higher in preterm preeclampsia (56%) compared with term preeclampsia (14%) or matched controls (8%; \( P \) values <0.001). The risk of developing essential hypertension within 2 years was significantly higher in both preterm preeclamptic women and those with persistent left ventricular moderate-severe abnormal function/geometry. The cardiovascular implications of preeclampsia do not end with the birth of the infant and placenta. The majority of preterm preeclamptic women have stage B asymptomatic heart failure postpartum, and 40% develop essential hypertension within 1 to 2 years after pregnancy. Women with a history of preterm preeclampsia may benefit from formal cardiovascular risk assessment in the 1 to 2 years after delivery to identify those who would benefit from targeted therapeutic intervention. (Hypertension. 2011;58:709-715.)

Key Words: preeclampsia ■ remodeling ■ heart failure ■ echocardiography ■ hypertension ■ tissue Doppler ■ strain rate

Acute preeclampsia is associated with significantly higher prevalence of asymptomatic global left ventricular (LV) abnormal function/geometry and myocardial injury than uneventful pregnancy.1 Preeclampsia is also associated with significantly higher risk of subsequent heart failure, ischemic and hypertensive heart diseases, and related mortality compared with uneventful pregnancy.2,3 There is an increasing understanding that cardiovascular diseases are generally progressive disorders that proceed through asymptomatic to symptomatic stages.4–6 One of the principal manifestations of this progression is the change in the geometry and function of the left ventricle.4–6 Therapeutic intervention during the asymptomatic phase of cardiac impairment can improve the long-term prognosis more effectively than when commenced at a symptomatic stage.4–6 Notwithstanding these recent insights, there is a paucity of studies that assess postnatal cardiac chamber function/geometry and myocardial status in asymptomatic women who have had a pregnancy complicated by preeclampsia. The aim of this study was to evaluate the postpartum natural history and clinical significance of asymptomatic LV abnormalities seen in preeclampsia.

Methods

This was a prospective longitudinal case-control study carried out over a 3-year period from January 2008. Women with singleton pregnancy and preeclampsia recruited in pregnancy and their matched controls were asked to come back at 1 and 2 years postpartum. Preeclampsia was classified according to the International Society for the Study of Hypertension in Pregnancy guidelines.7 Severity of preeclampsia and preeclampsia-related maternal complications were defined accordingly to the National High Blood Pressure Education Program Working Group guidelines.8 Cases were prospectively differentiated as preterm preeclampsia (requiring delivery before 37 weeks’ gestation for severe preeclampsia) and term preeclampsia (delivered at term gestation). All of the echocardiographic assessments were performed within 24 hours of the diagnosis of preeclampsia and before commencing any medication. Controls were normotensive women with singleton pregnancy matched for gestational age at echocardiography, age, and ethnicity. Only cases and controls not pregnant at 1-year follow-up were included in the study. All of the participants provided informed written consent for

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the study, which was approved by a local institutional review board. This study was approved by the Wandsworth Local Research Ethics Committee (ref No. 01.78.5).

The 1-year postpartum assessment included a repeat medical and family history, body measurement assessment, blood pressure profile,9 echocardiography, and 12-lead ECG. This assessment, excluding echocardiography and ECG, was repeated at 2 years postpartum. Further investigations in the case of abnormal cardiovascular findings were arranged as appropriate. All of the subjects were studied by standard 2D and Doppler transthoracic echocardiography, tissue Doppler, strain, and strain rate analysis, as described previously in detail and outlined in brief below.

**Left-Sided Cardiovascular System Assessment**

Global LV diastolic function, left heart chamber filling pressures, and geometry were assessed and graded using standard diagnostic algorithms with the recommended adjustments reflecting the concomitant systolic function and age.10 Ejection fraction (EF) was evaluated by biplane Simpson method, and LV global radial systolic dysfunction was defined as EF <55%.11 Global longitudinal systolic dysfunction was defined as average peak systolic velocity at the level of the left and septal sites of mitral valve annulus (Av S1) index 2 SDs below the expected mean for age. LV global systo-diastolic dysfunction was defined as LV global diastolic dysfunction in the presence of reduced EF (EF: <55%). The severity of LV dysfunction and hypertrophy was graded according to the European Association and American Society of Echocardiography guidelines.10,11

**Right-Sided Cardiovascular System Assessment**

Right heart function and remodeling were assessed integrating conventional echocardiographic indices and tissue Doppler velocity and deformation indices following recently published guidelines. Right ventricle (RV) hypertrophy, RV enlargement, right atrial enlargement, RV longitudinal systolic dysfunction, and RV global diastolic dysfunction were defined as present according to the European Association and American Society of Echocardiography 2010 guidelines.15

**Strain and Strain Rate Assessment**

Strain and strain rate indices were investigated as described previously.1,13,14 Regional peak systolic strain rate index was considered abnormal if it was 2 SDs below the expected mean for age.13 This abnormality was defined as segmental myocardial impaired contractility. Regional diastolic dysfunction was defined as early to late strain rate ratio ≤1.1 This abnormality was defined as segmental impaired myocardial relaxation.16,17 High amplitude postsystolic shortening was defined as a postsystolic shortening index ≥25%.1

**Correction of Indices**

All of the conventional echocardiographic indices were adjusted for body surface area11 and all of the tissue Doppler velocity and deformation indices to the end-diastolic LV or RV long axis length.18

**Statistical Analysis**

Data were analyzed in preterm and term cohorts as defined a priori using SPSS 15 software (SPSS Inc, Chicago, IL). Variables were compared using Mann–Whitney U or χ2 tests, as appropriate. Paired-group comparisons were only undertaken if Kruskal-Wallis testing indicated significant differences. A value of P<0.05 was considered statistically significant, and all tests were 2-sided. Repetitability, reproducibility, sample size, and power calculation were reported previously.1

**Results**

Seventy-seven women whose pregnancies were complicated by preeclampsia (27 preterm and 50 at term; please see Table S1 in the online Data Supplement at http://hyper.ahajournals.org/) were recruited during pregnancy and requested to attend for postpartum follow-up. At 1 year postpartum, all of the preterm and 37 term preeclampsia cases were available for assessment after exclusions for another pregnancy (n=5) and loss to follow-up (n=8). All of the study participants, including 78 matched controls, attended follow-up at 1 and 2 years postpartum. The baseline patient characteristics and blood pressure indices of the study population are shown in Table 1.

**Left-Sided Cardiovascular System**

**LV Diastolic Function**

Global LV diastolic dysfunction, left atrial remodeling, high amplitude postsystolic shortening, and impaired segmental myocardial relaxation seen at acute disease in pregnancy persisted significantly in preterm preeclampsia at 1 year postpartum (Tables 2 and 3 and Figure 1; please see Table S2). Approximately half of preterm preeclampsia women still met the criteria for the diagnosis of global LV diastolic dysfunction grade 1a (29.6%) and grade 2 (22.2%) at postpartum follow-up. In contrast, diastolic dysfunction recovered in term preeclampsia women, although there was persistence in segmental impairment of myocardial relaxation (Tables 2 and 3; please see Table S3).

**LV Geometry**

Global LV altered geometry seen in pregnancy regressed significantly in both the term and preterm preeclampsia groups, as well as in controls at 1 year postpartum follow-up (Tables 2 and 3 and Figure 2; please see Table S4). However, 41% of preterm preeclampsia women still presented abnormal LV geometric pattern (concentric remodeling: n=4, eccentric hypertrophy: n=4, concentric hypertrophy: n=3) at postpartum follow-up.
LV Systolic Function

Global systolic dysfunction and impaired myocardial contractility seen in preterm preeclampsia during pregnancy persisted at 1 year postpartum in these women (Tables 2 and 3). One fifth of them still presented LV mild radial systolic dysfunction (EF: 45% to 54%) at postpartum follow-up, whereas all of the other cases and controls had normal LV systolic function throughout the whole study period (Tables 2 and 3; please see Table S4). LV systolic dysfunction was associated with moderate-severe LV diastolic dysfunction/hypertrophy in all of the cases.

Table 2. One-year Postpartum Cardiovascular Changes in Preterm and Term Preeclamptic Cases and Controls

<table>
<thead>
<tr>
<th>Group</th>
<th>Term Preeclampsia (n = 37)</th>
<th>Preterm Preeclampsia (n = 27)</th>
<th>Controls (n = 78)</th>
<th>Term Preeclampsia vs Control</th>
<th>Preterm Preeclampsia vs Control</th>
<th>Term vs Preterm Preeclampsia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemodynamic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP, mm Hg</td>
<td>93 (83 to 101)</td>
<td>88 (80 to 97)</td>
<td>77 (18)</td>
<td>0.002</td>
<td>0.003</td>
<td>0.6</td>
</tr>
<tr>
<td>LVM/CWI, g/mm Hg · L · min</td>
<td>0.26 (0.20 to 0.33)</td>
<td>0.36 (0.21 to 0.41)</td>
<td>0.27 (0.22–0.30)</td>
<td>0.8</td>
<td>0.002</td>
<td>0.02</td>
</tr>
<tr>
<td>Wall stress index, dyne/cm²/m²</td>
<td>43 (32 to 60)</td>
<td>57 (42 to 67)</td>
<td>44 (40–48)</td>
<td>0.1</td>
<td>0.018</td>
<td>0.02</td>
</tr>
<tr>
<td>LV global cardiac findings</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
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<tr>
<td>Altered geometry</td>
<td>7 (18.9)</td>
<td>11 (40.7)</td>
<td>5 (6.4)</td>
<td>0.09</td>
<td>0.0001</td>
<td>0.1</td>
</tr>
<tr>
<td>Diastolic dysfunction</td>
<td>6 (16.2)</td>
<td>14 (51.9)</td>
<td>6 (7.7)</td>
<td>0.28</td>
<td>0.0001</td>
<td>0.006</td>
</tr>
<tr>
<td>Radial systolic dysfunction</td>
<td>2 (5.4)</td>
<td>5 (19)</td>
<td>1 (1.3)</td>
<td>0.5</td>
<td>0.004</td>
<td>0.2</td>
</tr>
<tr>
<td>LV moderate-severe abnormalities</td>
<td>5 (13.5)</td>
<td>15 (55.6)</td>
<td>6 (7.7)</td>
<td>0.5</td>
<td>0.0001</td>
<td>0.0001</td>
</tr>
<tr>
<td>Stage B (asymptomatic) heart failure</td>
<td>9 (24)</td>
<td>19 (70)</td>
<td>8 (10)</td>
<td>0.088</td>
<td>0.0001</td>
<td>0.02</td>
</tr>
<tr>
<td>RV global cardiac findings</td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Hypertrophy</td>
<td>2 (5.4)</td>
<td>5 (19)</td>
<td>1 (1.3)</td>
<td>0.19</td>
<td>0.004</td>
<td>0.21</td>
</tr>
<tr>
<td>Diastolic dysfunction</td>
<td>0</td>
<td>5 (19)</td>
<td>0</td>
<td>0.0001</td>
<td>0.024</td>
<td></td>
</tr>
<tr>
<td>Systolic dysfunction</td>
<td>2 (5.4)</td>
<td>5 (18.5)</td>
<td>1 (1.3)</td>
<td>0.5</td>
<td>0.004</td>
<td>0.2</td>
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<tr>
<td>Myocardial function</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Impaired LV relaxation*</td>
<td>17 (45.9)</td>
<td>20 (74)</td>
<td>10 (12.8)</td>
<td>0.0001</td>
<td>0.0001</td>
<td>0.046</td>
</tr>
<tr>
<td>Impaired LV contractility*</td>
<td>6 (16.2)</td>
<td>14 (51.9)</td>
<td>3 (3.9)</td>
<td>0.056</td>
<td>0.0001</td>
<td>0.025</td>
</tr>
<tr>
<td>Impaired RV relaxation</td>
<td>5 (13.5)</td>
<td>5 (18.5)</td>
<td>0</td>
<td>0.005</td>
<td>0.0001</td>
<td>0.35</td>
</tr>
<tr>
<td>Impaired RV contractility</td>
<td>1 (2.7)</td>
<td>2 (7.4)</td>
<td>0</td>
<td>0.70</td>
<td>0.0001</td>
<td>0.19</td>
</tr>
</tbody>
</table>

Values are given as median (interquartile range) when indices are provided or as No. (percentage). MAP indicates mean arterial pressure; LV, left ventricular; RV, right ventricular; LVM, left ventricular mass index; CWI, cardiac work index.

*Data show the No. of women with ≥1 segment affected among investigated segments.

Table 3. Longitudinal Cardiovascular Changes in Preterm and Term Preeclampsia Cases From Pregnancy to 1-y Postpartum

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Hemodynamic</td>
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<tr>
<td>MAP, mm Hg</td>
<td>114 (107 to 119)</td>
<td>93 (83 to 101)</td>
<td>0.0001</td>
<td>117 (112 to 120)</td>
<td>88 (80 to 97)</td>
<td>0.0001</td>
</tr>
<tr>
<td>LVM/CWI, g/mm Hg · L · min</td>
<td>0.26 (0.19 to 0.29)</td>
<td>0.26 (0.20 to 0.33)</td>
<td>0.4</td>
<td>0.25 (0.21 to 0.33)</td>
<td>0.36 (0.21 to 0.41)</td>
<td>0.002</td>
</tr>
<tr>
<td>Wall stress index, dyne/cm²/m²</td>
<td>42 (32 to 63)</td>
<td>43 (32 to 60)</td>
<td>0.5</td>
<td>49 (34 to 64)</td>
<td>57 (42 to 67)</td>
<td>0.3</td>
</tr>
<tr>
<td>LV global cardiac findings</td>
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<td></td>
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</tr>
<tr>
<td>Altered geometry</td>
<td>36 (72)</td>
<td>7 (18.9)</td>
<td>0.019</td>
<td>22 (81)</td>
<td>11 (40.7)</td>
<td>0.005</td>
</tr>
<tr>
<td>Diastolic dysfunction</td>
<td>20 (40)</td>
<td>6 (16.2)</td>
<td>0.0001</td>
<td>14 (51.8)</td>
<td>14 (51.8)</td>
<td>0.8</td>
</tr>
<tr>
<td>Radial systolic dysfunction</td>
<td>2 (4)</td>
<td>1 (2.7)</td>
<td>0.9</td>
<td>7 (25.9)</td>
<td>5 (19)</td>
<td>0.7</td>
</tr>
<tr>
<td>RV global cardiac findings</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertrophy</td>
<td>16 (32)</td>
<td>2 (5.4)</td>
<td>0.006</td>
<td>13 (48.2)</td>
<td>5 (18.5)</td>
<td>0.043</td>
</tr>
<tr>
<td>Diastolic dysfunction</td>
<td>8 (16)</td>
<td>0</td>
<td>0.029</td>
<td>5 (18.5)</td>
<td>5 (18.5)</td>
<td>0.7</td>
</tr>
<tr>
<td>Systolic dysfunction</td>
<td>2 (4)</td>
<td>2 (5.4)</td>
<td>0.8</td>
<td>8 (29.6)</td>
<td>5 (18.5)</td>
<td>0.73</td>
</tr>
<tr>
<td>Myocardial function</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impaired LV relaxation*</td>
<td>32 (64)</td>
<td>17 (45.9)</td>
<td>0.1</td>
<td>23 (85.2)</td>
<td>20 (74)</td>
<td>0.5</td>
</tr>
<tr>
<td>Impaired LV contractility*</td>
<td>21 (42)</td>
<td>6 (16.2)</td>
<td>0.020</td>
<td>16 (59.3)</td>
<td>14 (51.9)</td>
<td>0.78</td>
</tr>
<tr>
<td>Impaired RV relaxation</td>
<td>10 (20)</td>
<td>5 (13.5)</td>
<td>0.6</td>
<td>7 (25.9)</td>
<td>5 (18.5)</td>
<td>0.78</td>
</tr>
<tr>
<td>Impaired RV contractility</td>
<td>5 (10)</td>
<td>1 (2.7)</td>
<td>0.4</td>
<td>4 (14.8)</td>
<td>2 (7.4)</td>
<td>0.70</td>
</tr>
</tbody>
</table>

MAP indicates mean arterial pressure; LV, left ventricular; RV, right ventricular; LVM, left ventricular mass index; CWI, cardiac work index; wall stress index, left ventricle end-systolic meridian wall stress index. Values are given as median (interquartile range) when indices are provided or as No. (percentage).

*Number of women with at least one segment affected among investigated segments.
Hemodynamic
At 1 year postpartum, total vascular resistance index decreased to baseline values in both preeclampsia groups. Blood pressure measurements (mean arterial pressures and systolic and diastolic blood pressures), although returned within normal reference ranges, were still significantly higher in both preeclampsia groups versus control (Tables 2 and 3; please see Table S4). Postpartum LV mass:cardiac work ratio and LV wall stress were significantly increased in preterm preeclampsia but not term preeclampsia versus controls (Tables 2 and 3).

Right-Sided Cardiovascular System
Preterm preeclampsia women demonstrated persistence of global RV systo-diastolic dysfunction and basal impaired myocardial relaxation at 1 year postpartum, whereas RV hypertrophy significantly regressed without returning to the baseline (Tables 2 and 3). RV dysfunction/hypertrophy was only seen in preterm preeclampsia women with advanced LV global diastolic dysfunction. In contrast, term preeclampsia women recovered from RV global diastolic dysfunction/hypertrophy but still had basal impaired myocardial relaxation (Tables 2 and 3).

Development of Subsequent Cardiovascular Complications
The American Heart Association/American College of Cardiologists define asymptomatic stage B heart failure as any patient exhibiting LV hypertrophy or LV systolic or diastolic dysfunction. The prevalence of asymptomatic stage B heart failure at 1 year postpartum follow-up (Table 2) was significantly higher in preterm versus term preeclampsia and controls but not in term preeclampsia versus controls. Similarly, moderate-severe LV abnormalities were significantly more prevalent in postnatal preterm preeclampsia women compared with those with term preeclampsia and controls (Table 2 and Figure 3).

At 2 years postpartum, 16 (25.0%) of the 64 women who had preeclampsia and 1 (1.3%) of the 78 controls developed essential hypertension (Figure 4; P<0.001). Women with moderate-severe LV abnormalities at 1 year postpartum were more likely to develop hypertension at 2 years postpartum (13 of 26 [50.0%]) compared with those with normal LV function/geometry or mild LV abnormalities (4 of 116 [3.5%]; relative risk of essential hypertension in women with LV
Discussion

This longitudinal prospective study of cardiac function and geometry in preeclampsia demonstrates a significant incidence of asymptomatic LV abnormalities, high prevalence of hypertension, and increased cardiovascular risk status within 2 years of delivery in preeclamptic women. These findings are also more marked in women who developed preterm compared with term preeclampsia and normal pregnancy.

Left-Sided Cardiac Chamber Function and Geometry

The postpartum persistence of LV global systo-diastolic dysfunction in approximately half of the preterm preeclampsia women in contrast to significant resolution to normal in term preeclampsia suggests an exhaustion of cardiac reserve in preterm preeclampsia women only. LV remodeling/hypertrophy in acute preeclampsia may have been an adaptive response to minimize wall stress with the sudden development of hypertension in pregnancy.1–19 However, residual postpartum LV altered geometry does not appear to be a compensatory phenomenon. Abnormal geometry in postpartum preterm preeclampsia women is likely to progress, because increased wall stress is known to sustain and exacerbate the remodeling process.5

Neither of the 2 previous studies that assessed cardiovascular longitudinal changes in preeclampsia into the postpartum period graded global diastolic dysfunction.19,20 Furthermore, postpartum follow-up in the latter studies was limited to 6 months after delivery, and the studies did not distinguish between preterm and term preeclampsia cases.19,20 Regardless of these limitations, in agreement with our findings, both studies similarly found that changes in LV geometry are rapidly reversible in control women, but resolution remains incomplete in preeclampsia women.19,20 Importantly, the only study to include preterm preeclampsia cases demonstrated impaired LV systolic function,20 whereas Simmons et al19 found that LV systolic function was unchanged in mainly term preeclampsia. Two other studies assessed LV function and structure in postpartum preeclampsia women only.21,22 In agreement with our study, they demonstrated the presence of a significant altered LV geometry and impaired LV function in postpartum preeclampsia.21,22

Right-Sided Cardiac Chamber Function and Geometry

The postpartum persistence of RV global systo-diastolic dysfunction in ≈20% of preterm preeclampsia women in contrast to significant resolution to normal in term preeclampsia is likely to be related to the corresponding findings in the left-sided cardiac system. In fact, persistently higher diastolic filling pressures in a poorly compliant or failing LV may have led to secondary increased pulmonary resistance in these cases. RV dysfunction may also be related to the impaired function and structure of the interventricular septum, mainly seen in preterm preeclampsia, because the septum affects ejection and filling of the right, as well as the left, ventricles. These hypotheses are supported by the finding that RV dysfunction and hypertrophy are only seen in women with advanced LV global diastolic dysfunction.
Myocardial Function

Our findings of impaired myocardial relaxation and high amplitude post-systolic shortening in preterm, as well as term, preeclampsia demonstrates that, even when cardiac chamber function appeared normal in preeclampsia women, there is a status of reduced myocardial performance. The relevance of this assessment is that strain rate indices are preload independent and better correlated to invasive indices of intrinsic myocardial status than conventional echo indices. Under normal loading conditions, impaired myocardial contractility/relaxation precedes the development of overt chamber systolic-diastolic dysfunction. Several studies have also shown the association of high amplitude longitudinal post-systolic shortening and abnormal strain rate indices with subendocardial ischemia and fibrosis. Myocardial impairment was again more severe and widespread in postpartum preterm than term preeclampsia. These observations are consistent with data demonstrating that both preterm and term preeclampsia have a higher risk of subsequent development of congestive heart failure and ischemic heart diseases compared with women with uneventful pregnancy, and this risk is higher in preterm versus term condition.

Cardiovascular Outcome

Our study demonstrates that preterm preeclampsia is strongly associated with the persistence of LV dysfunction/hypertrophy at 1 year postpartum follow-up and the risk of development of essential hypertension within 2 years. Recent guidelines have highlighted the importance of asymptomatic LV dysfunction/hypertrophy in the risk stratification for cardiac disease defining this condition as stage B for the development of heart failure. The progression from stage B (asymptomatic) to stage C (symptomatic) heart failure is associated with a 5-fold increase in risk of death, which can be ameliorated by early therapeutic intervention. Stage B heart failure was more prevalent in preterm than in term preeclampsia subjects or controls, consistent with the long-term outcome studies demonstrating that women with preterm preeclampsia have a higher risk of subsequent congestive heart failure and ischemic cardiac diseases compared with women with term preeclampsia or normal pregnancy. It should be noted that the prevalence of stage B heart failure found in the preterm preeclampsia group with an average age of 31 years is 70% and that the population prevalence estimates for stage B heart failure in adults aged >45 years is 34%.

Clinical Perspective

The cardiovascular implications of preeclampsia do not cease with the birth of the infant and placenta. This study demonstrates that women with preterm preeclampsia, and to a lesser extent term preeclampsia, are at significantly increased risk of asymptomatic LV dysfunction/hypertrophy and essential hypertension within 1 to 2 years after delivery. Many conventional risk factors for subsequent cardiovascular morbidity are linked with aging, and their effects only become apparent at advanced stages when intervention is less efficacious. Our findings provide an opportunity to identify these high-risk women before other risk factors or symptoms become clinically apparent. Such assessment may serve to reduce the sexual discrepancy in outcomes of cardiovascular disease. A strategy of echocardiographic assessment to screen and treat those women at highest risk of subsequent cardiovascular morbidity needs to be evaluated in a larger prospective interventional trial.

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Disclosures

None.

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