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Clinical Course, Associated Factors, and Blood Pressure Profile of Delayed-Onset Postpartum Preeclampsia

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Abstract

Objective: To identify clinical risk factors associated with development of delayed-onset postpartum preeclampsia, and to characterize management and subsequent risk of cardiovascular disease.

Methods: This is a case-control study of women admitted to the hospital with delayed-onset postpartum preeclampsia (defined as a new diagnosis of preeclampsia presenting between 48 hours and 6 weeks postpartum) compared to women with full-term, uncomplicated pregnancies without a hypertensive diagnosis or diabetes. Included women delivered between January 2014 and June 2018 at a single tertiary-care center. Women with an antenatal diagnosis of preeclampsia or chronic hypertension were excluded. Univariate analysis was used to identify risk factors associated with delayed-onset postpartum preeclampsia and to compare rates of hypertension and anti-hypertensive medication use with follow-up beyond three months postpartum among a subset of controls matched 2:1 with cases. Multivariable logistic regression was performed and included co-variates identified in a backward stepwise approach.

Results: Compared to controls (N=26,936), women with delayed-onset postpartum preeclampsia (N=121) were significantly more likely to be of non-Hispanic black race (31.4% vs. 18.0%), obese (39.7% vs. 20.1%), and deliver by cesarean (40.5% vs. 25.8%), all $p<0.01$. For women diagnosed with delayed-onset postpartum preeclampsia, the median postpartum day of presentation was 7.0 (IQR 5.0–9.0) with 93.4% presenting secondary to symptoms, most commonly a headache. A majority (73.6%) underwent imaging studies and 49.6% received intravenous anti-hypertensive agents. A total of 86 (71.0%) women with delayed-onset postpartum preeclampsia and 169

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(72.8%) controls had longer term information available, with median follow-up time 1.5 years (IQR 0.8–2.8). Delayed-onset postpartum preeclampsia was associated with higher blood pressures at ≥3 months postpartum (median systolic 130 mmHg vs. 112 mmHg and median diastolic 80 mmHg vs. 70 mmHg, p<0.001).

Conclusion: Delayed-onset postpartum preeclampsia is associated with variable management strategies. There is substantial overlap between the clinical risk factors for delayed-onset postpartum preeclampsia and antepartum preeclampsia. Our findings suggest that delayed-onset postpartum preeclampsia is also associated with an increased risk of progression to chronic hypertension.

PRECIS:

Delayed-onset postpartum preeclampsia is variably managed, and is associated with substantial maternal morbidity and an increased risk of subsequent chronic hypertension.

INTRODUCTION

Hypertension and preeclampsia are leading causes of maternal morbidity and mortality. The incidence of preeclampsia continues to increase with the majority of women diagnosed during the antepartum period and immediately postpartum.[1] Delayed-onset or late postpartum preeclampsia is defined as a new diagnosis of preeclampsia presenting between 48 hours and 6 weeks postpartum.[2–6] The prevalence of de novo postpartum hypertensive disorders is not well understood, with reported rates ranging from 0.3% to 27.5%.[7] The mechanisms underlying its development are also poorly characterized, but prior studies have shown an overlap of risk factors with antepartum preeclampsia, including obesity, older maternal age, black race and cesarean delivery.[2,8–11]

The current evidence describing the clinical course of delayed-onset postpartum preeclampsia is limited and generally includes women with antepartum hypertensive disorders of pregnancy.[4–6] There are no standardized guidelines concerning postpartum management in this population. Recent assessment of postpartum readmissions for hypertension demonstrates a greater risk of maternal morbidity associated with delayed-onset postpartum preeclampsia compared to antepartum hypertensive disorders.[11] Furthermore, while antepartum preeclampsia is associated with future cardiovascular disease, long-term cardiovascular risk for women with delayed-onset postpartum preeclampsia is unclear.[12–17]

We sought to identify clinical risk factors, describe clinical management, and characterize follow-up blood pressure profiles in women readmitted with delayed-onset postpartum preeclampsia without prior preeclampsia or hypertensive disorders.

METHODS

We conducted a retrospective case-control study of women readmitted with new delayed-onset postpartum preeclampsia compared to controls with uncomplicated pregnancies without readmissions for postpartum complications. Delayed-onset postpartum preeclampsia was defined as a new diagnosis of preeclampsia from 48 hours until 6 weeks postpartum.

Preeclampsia was defined by the American College of Obstetricians and Gynecologists (ACOG) criteria as blood pressure ≥ 140 mmHg systolic or 90 mmHg diastolic on two or more occasions more than 6 hours apart accompanied by proteinuria or end organ dysfunction or blood pressure ≥ 160 mmHg systolic or 110 mmHg diastolic.[1] We included women diagnosed with new delayed-onset postpartum preeclampsia who were readmitted to Magee-Womens Hospital of University of Pittsburgh Medical Center (Pittsburgh, PA) from January 2014 to May 2018. As the focus of this study is on women with no antenatal history of hypertension, we excluded women with a pre-pregnancy history of chronic hypertension or an antenatal diagnosis of preeclampsia. Women were also excluded if they had a history of pre-gestational diabetes.

Data were collected using hospital readmission data, the Magee Obstetric Maternal and Infant (MOMI) database and chart review. The MOMI database was established in 1995 from all women who have delivered at Magee-Womens Hospital through the present. It includes information on over 300 variables for maternal, fetal and neonatal characteristics obtained from admitting services, International Classification of Diseases, Ninth Revision (ICD-9) codes, electronic medical record abstraction, electronic birth records and ultrasound. It is populated in real time as a research database, with dedicated data administrators who review, validate and store the data collected.[18] Delayed-onset postpartum preeclampsia cases were identified from readmission data, which is tracked prospectively at our institution in compliance with the Centers for Medicare and Medicaid Services (CMS) requirements. Controls were identified from the MOMI database and included women with a full-term, uncomplicated pregnancy without a diagnosis of chronic hypertension, any hypertensive disorder of pregnancy or pre-existing diabetes. After identification of cases and controls, a thorough chart review of the cases and a subset of the controls was performed. The charts for review were randomly distributed to two physician-authors (ER and AH). Each chart was reviewed, and data abstracted into pre-specified data collection forms in REDCAP. Ten percent of the records were randomly selected for re-review by the other physician. If there was any disagreement, then the subject was discussed and adjudicated by the three physician authors (AJ, AH, ER). The University of Pittsburgh Institutional Review Board approved this project (Institutional Review Board number PRO15060439).

Statistical analyses were completed using Stata IC 15 software package (StataCorp LP, College Station, TX). Baseline characteristics were compared between controls and cases to identify risk factors for delayed-onset postpartum preeclampsia. Continuous variables were compared using Student’s t-tests and Wilcoxon-Mann Whitney tests as appropriate. Categorical variables were analyzed using Chi-square or Fisher’s exact, where appropriate. With the presence or absence of delayed-onset postpartum preeclampsia as the outcome, a multivariable logistic regression model for each patient characteristic was built. Characteristics with a probability value of <0.1 in individual models were evaluated with the use of forward, backward and stepwise multivariable logistic regression models to identify variables that were independently associated with the outcome. Results are presented as unadjusted or adjusted odds ratios with corresponding 95% confidence intervals, and a p-value <0.05 was considered statistically significant.

In order to characterize follow-up blood pressure and maternal cardiovascular outcomes following delayed-onset postpartum preeclampsia, detailed medical record review was performed on a subset of controls, matched 2:1 by delivery date to cases. If there were more than two control subjects available for inclusion on a given date of delivery, we selected the first two based on delivery time. In the event that we were unable to identify an appropriate control meeting our inclusion criteria on a specific delivery date, there were less than two controls per case (n=10 cases matched to one control). In women for whom follow-up blood pressure (≥3 months postpartum) was available through the medical record, we characterized antihypertensive medication use, clinical diagnosis of chronic hypertension and diagnosis of diabetes mellitus. With the presence or absence of chronic hypertension as the primary outcome, a multivariable logistic regression model for each was built. Characteristics with a probability value of <0.1 in individual models were evaluated with the use of forward, backward and stepwise multivariable logistic regression models to identify variables that were independently associated with the outcome. Results are presented as adjusted odds ratios with corresponding 95% confidence intervals, and a p-value <0.05 was considered statistically significant.

RESULTS

We included 121 women with delayed-onset postpartum preeclampsia and 26,936 controls with uncomplicated pregnancies. Compared to controls, women with delayed-onset postpartum preeclampsia were older, and more likely to be of non-Hispanic black race and were also more likely to have a higher pre-pregnancy body mass index (BMI), Increasing category of BMI was associated with an increasing risk of delayed-onset postpartum preeclampsia). They were also more likely to deliver by cesarean (Table 1). Eighteen women with delayed-onset postpartum preeclampsia (14.9%) had a history of preeclampsia during a prior pregnancy. In our multivariable logistic regression model, after controlling for each of the other factors, maternal age ≥35 years, pre-pregnancy BMI ≥30 kg/m^2, cesarean section and non-Hispanic black race remained significantly associated with delayed-onset postpartum preeclampsia (Table 2).

For the 121 women diagnosed with delayed-onset postpartum preeclampsia, the median postpartum day of presentation was 7.0 [IQR 5.0–9.0] (Table 3). There were no cases beyond 19 days post-delivery. Almost all women presented with symptoms. The most common symptom was headache, followed by shortness of breath and swelling. The few women who were asymptomatic had been noted to have incidentally elevated blood pressures had presented to the hospital for further care. Three women presented with eclampsia. Median peak systolic and diastolic blood pressures were 175 mmHg [IQR 166–183] and 102 mmHg [IQR 94–110], respectively, and 28 women (23.1%) had abnormal laboratory values, including serum creatinine, platelets or liver function tests. Twenty-one women (17.4%) had an abnormal urine protein to creatinine ratio (≥0.3 mg/dL). The median length of re-hospitalization was 2.0 days [IQR 1.0–6.0] and 10.7% were admitted to the Intensive Care Unit. A majority underwent imaging studies and received magnesium for seizure prophylaxis. Diuretics were administered to 23.1% of women and one-half received an intravenous (IV) anti-hypertensive agent (Table 4).

To characterize follow-up blood pressure and maternal cardiovascular outcomes, we matched a subset of our controls 2:1 by delivery date to cases. Demographic and delivery characteristics of this subset are similar to the overall cohort (Appendix 1, available online at http://links.lww.com/xxx). A total of 86 women with delayed-onset postpartum preeclampsia and 169 controls had follow-up data available (≥3 months postpartum). Women with follow-up data available did not significantly differ in demographic or delivery characteristics from women without available follow-up data (Appendixes 2 and 3, available online at http://links.lww.com/xxx). The median follow-up time was 1.5 years [IQR 0.8–2.8]. A total of 51.2% of cases (44/86) had been discharged on an anti-hypertensive medication, with most women (3¼4; 70.5%) remaining on medication at 6 weeks postpartum. There were 18 women (20.9%) who required medication more than 3 months postpartum. Compared to controls, delayed-onset postpartum preeclampsia was associated with higher systolic and diastolic blood pressures at both the postpartum visit and longer term (Table 5). It was also associated with an odds ratio of 2.83 (95% CI 1.48–5.42) of developing chronic hypertension after adjustment for BMI and time postpartum.

DISCUSSION

Our findings identify several clinical risk factors for development of delayed-onset postpartum preeclampsia in women without an antenatal diagnosis of preeclampsia or chronic hypertension. Non-Hispanic black race, obesity and delivery by cesarean are all significantly associated with delayed-onset postpartum preeclampsia, consistent with prior smaller studies.[2,8,10] Notably, there is substantial overlap with known risk factors for antepartum preeclampsia, which may shed light on the mechanisms of the disease.[19,20] We also demonstrate variable management strategies and utilization of hospital resources by women presenting with delayed-onset postpartum preeclampsia. Few prior studies have examined the clinical course of delayed-onset postpartum preeclampsia. One small case series found a median time to postpartum presentation of five days, with most women also presenting with headaches and elevated blood pressures.[6]

There are very limited data on the association between delayed-onset postpartum preeclampsia and future cardiovascular disease. In our study, women with delayed-onset postpartum preeclampsia had significantly higher blood pressures at both the postpartum visit and ≥3 months postpartum. They were also more likely to progress to chronic hypertension, with 45% of women with delayed-onset postpartum preeclampsia developing stage 1 or stage 2 hypertension compared to 16% of controls within the follow-up time (median 1.1 years). This is similar to previously reported rates of chronic hypertension within the first-year postpartum following preeclampsia with antepartum onset.[21–23] Given the overlap of clinical risk factors and prevalence of subsequent chronic hypertension, our findings suggest a relationship between postpartum preeclampsia and antepartum preeclampsia. Additional studies are needed to further elucidate the pathophysiology of delayed-onset postpartum preeclampsia and define its place in the spectrum of hypertensive disorders of pregnancy.

This study is not without limitations. All of the women presented to and were treated at a single tertiary-care hospital and thus our results may not be generalizable to other

populations. Given the retrospective nature of this study, the data analyzed were limited to that available for chart review in the hospital records. In particular, it is likely that women for whom longer term follow-up data were available differed from women who did not have available follow-up data, however, our results suggest that they do not differ significantly in terms of baseline demographic or delivery characteristics (Appendix 3, http://links.lww.com/xxx). It is unlikely that the data evaluated to compare cases and controls were more accurate or complete for one group versus the other, which would have had a larger impact on our analysis. We may not have identified all women who developed delayed-onset postpartum preeclampsia because less severe cases may not have presented to the hospital for care or have been readmitted, while some women likely sought care at an outside institution. For this reason, we are hesitant to provide an exact incidence in our cohort as it is very likely that this would be an underestimate and might falsely reassure the clinician. Prior studies have noted an overall incidence between 0.3% to 27.5%.[7] Nonetheless, this study is one of the largest case-control studies examining the presentation and initial management of delayed-onset postpartum preeclampsia among women without an antenatal diagnosis of chronic hypertension or preeclampsia.

Delayed-onset postpartum preeclampsia is associated with substantial maternal morbidity with varying management strategies apparentLonger term, affected women demonstrate increased risk of progression to chronic hypertension compared to controls. There is also substantial overlap between the clinical risk factors for delayed-onset postpartum preeclampsia and antepartum preeclampsia, and thus it is very plausible that the two disorders and their mechanisms are closely related. There is growing awareness of the importance of continued maternal health care through the fourth trimester, the time period from childbirth through 12 weeks postpartum.[24] This study recognizes several common risk factors for the development of delayed-onset postpartum preeclampsia and its associated morbidity. Given the overall rarity of this condition, and the frequency of clinically identified risk factors, recommendations for additional surveillance or earlier follow up for women with identified risk factors would likely not be clinically efficient or practical. However, it is crucial that health care providers continue to counsel women on the signs and symptoms of preeclampsia even after delivery. Larger studies are needed to confirm our findings and to assist in the development of evidence-based protocols for prevention and management in this understudied population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1.

Demographics and delivery characteristics Data are n (%) or median [interquartile range]

	Controls N=26,936	Cases N=121	OR (95%CI)	p-value
Age (years)	30 [26–33]	31 [27–35]	--	0.02
Race				
Caucasian	19,332 (71.8)	74 (61.2)	Ref.	
Non-Hispanic Black	4,846 (18.0)	38 (31.4)	2.00 (1.34–2.96)	0.003
Asian or Pacific Islander	1873 (7.0)	5 (4.1)	0.70 (0.28–1.72)	
Other or Unspecified	885 (3.3)	4 (3.3)	1.18 (0.43–3.24)	
Pre-pregnancy BMI (kg/m^2)†	24.3 [21.6–28.6]	28.3 [24.0–34.0]	--	<0.001
BMI Category†				
Underweight (<18.5 kg/m^2)	974 (4.0)	0	--	
Normal (18.5–24.9 kg/m^2)	12,564 (51.3)	36 (29.8)	Ref.	
Overweight (25.0–29.9 kg/m^2)	6,036 (24.6)	37 (30.6)	2.14 (1.35–3.39)	<0.001
Obese Class I (30.0–34.9 kg/m^2)	2,908 (11.9)	22 (18.2)	2.64 (1.55–4.50)	
Obese Class II (35.0–39.9 kg/m^2)	1,302 (5.3)	10 (8.3)	2.68 (1.33–5.41)	
Obese Class III (≥40.0 kg/m^2)	723 (3.0)	16 (13.2)	7.72 (4.27–13.98)	
Nulliparous	10,857 (40.3)	45 (37.2)	Ref.	0.5
Multiparous	16,079 (59.7)	76 (62.8)	0.88 (0.61–1.27)	
Gestational diabetes	1,452 (5.4)	12 (9.9)	1.93 (1.06–3.52)	0.03
Gestational age at delivery (weeks)	39.4 [39.0–40.3]	39.0 [37.7–39.9]	--	<0.001
Birthweight (grams)	3426 [3170–3720]	3260 [2878–3614]	--	<0.001
Cesarean delivery	6,938 (25.8)	49 (40.5)	2.11 (1.46–3.05)	<0.001

† N=24,507 controls (BMI data missing)

Table 2.

Adjusted odds of delayed-onset postpartum preeclampsia according to clinical characteristics

Characteristic	Controls N=26,936	Cases N=121	Adjusted OR*	p-value
Age ≥35 years	4,740 (17.6%)	32 (26.4%)	1.67 (1.09–2.57)	0.02
Non-Hispanic Black race	4,846 (18.0%)	38 (31.4%)	1.98 (1.32–2.97)	0.001
Pre-pregnancy BMI ≥30 kg/m^2†	4,933 (20.1%)	48 (39.7%)	2.32 (1.59–3.40)	<0.001
Cesarean delivery	6,938 (25.8%)	49 (40.5%)	1.96 (1.35–2.86)	<0.001

*Adjusted OR (odds ratio): odds of delayed-onset postpartum preeclampsia determined using multivariable logistic regression, adjusted for maternal age ≥ 35 years, non-Hispanic black race, pre-pregnancy BMI ≥30 kg/m^2 and Cesarean delivery

†N=24,507 controls (BMI data missing)

Table 3.

Clinical presentation of delayed-onset postpartum preeclampsia

	N=121
Days postpartum	7.0 [5.0–9.0]
Reason for presentation	
Elevated BP at physician's office	25 (20.7)
Elevated BP at home / non-medical location	29 (24.0)
Symptoms	113 (93.4)
Symptoms at presentation	
None	8 (6.6)
Eclampsia	3 (2.5)
Headache	83 (68.6)
Shortness of breath	26 (21.5)
Abdominal pain	17 (14.0)
Peripheral edema	22 (18.2)
Highest systolic BP (mmHg)	175 [166–183]
Highest diastolic BP (mmHg)	102 [94–110]
Abnormal serum lab values *	28 (23.1)
Admitted to Intensive Care Unit	13 (10.7)
Adverse outcomes	
Cerebrovascular accident	1 (0.8)
Mechanical ventilation	3 (2.5)

Data are n (%) or median [interquartile range]

* serum lab values include creatinine, platelets and liver function tests

Table 4.

Clinical management of delayed-onset postpartum preeclampsia

	N=121
Underwent any radiologic study	89 (73.6)
Chest X-Ray	40 (33.1)
Chest CT	17 (14.0)
Head CT	44 (36.4)
Brain MRI	26 (21.5)
Echocardiogram	27 (22.3)
Other	14 (11.6)
Intravenous anti-hypertensive agents	60 (49.6)
Magnesium prophylaxis	100 (82.6)
Oral anti-hypertensive agents	74 (61.2)
Diuretic treatment	28 (23.1)
Length of hospitalization (days)	2.0 [1.0–6.0]
Discharged on anti-hypertensive agent	69 (57.0)

Data are n (%) or median [interquartile range]

Table 5.

Postpartum and follow-up outcomes

	Controls N=169	Cases N=86	p-value
Systolic BP at postpartum visit (mmHg)	112 [104–120]	122 [114–130]	<0.001
Diastolic BP at postpartum visit (mmHg)	70 [64–78]	78 [70–82]	<0.001
Anti-hypertensive use at postpartum visit	0	31 (36.0)	<0.001
Follow-up time (years)	1.6 [0.9–3.2]	1.1 [0.5–2.2]	0.004
Systolic BP at follow-up (mmHg)	112 [106–120]	130 [120–140]	<0.001
Diastolic BP at follow-up (mmHg)	70 [65–78]	80 [72–88]	<0.001
Diagnosis of chronic hypertension	27 (16.0)	39 (45.3)	<0.001
Diagnosis of diabetes mellitus type 2	0	3 (3.5)	0.04
Anti-hypertensive use at follow-up	2 (1.2)	18 (20.9)	<0.001

Data are n (%) or median [interquartile range] unless otherwise specified