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Clinical morbidities, trends, and demographics of eclampsia: a population-based study

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Abstract

OBJECTIVE—We sought to identify trends, demographics, and pre-pregnancy and peripartum morbidities of eclampsia in California.

STUDY DESIGN—We identified cases of eclampsia by International Classification of Diseases, Ninth Revision code using California health discharge data from 2001 through 2007. Cases with missing race/ethnicity as well as age <15 years or >55 years were excluded. Among the remaining cases, patients with eclampsia (n = 1888) were compared against those without (n = 2,768,983). Adjustments were performed for potential confounding variables using logistic regression. Significance was set at $P < .05$.

RESULTS—The incidence of eclampsia decreased over time, from 8.0 cases per 10,000 deliveries in 2001, to 5.6 cases per 10,000 deliveries in 2007 ($P < .001$). There was a bimodal distribution in age-related risk, with the highest risks at the extremes of age. Non-Hispanic blacks were associated with the highest risk of eclampsia while Asians had the lowest risk. Several antepartum morbidities had increased associations with eclampsia, including preexisting cardiac disease (adjusted odds ratio [OR], 6.84; 95% confidence interval [CI], 5.40–8.66), lupus erythematosus (adjusted OR, 3.68; 95% CI, 1.53–8.86), and twin gestations (adjusted OR, 3.28; 95% CI, 2.70–3.99). Peripartum complications increased in eclampsia included cerebrovascular hemorrhage/disorders (adjusted OR, 112.15; 95% CI, 77.47–162.35), peripartum cardiomyopathy (adjusted OR, 12.88; 95% CI, 6.08–27.25), amniotic fluid embolism (adjusted OR, 11.94; 95% CI, 3.63–39.21), and venous thromboembolism (adjusted OR, 10.71; 95% CI, 5.14–22.32).

CONCLUSION—This large population database confirms that there is a decline in eclampsia over time. However, there are extremely morbid complications associated with eclampsia, emphasizing the need for its close monitoring and prevention.

Keywords

demographics; eclampsia; morbidities; trends

Eclampsia is defined as the onset of seizures in the setting of preeclampsia and without another neurologic cause or condition.¹ The spectrum of hypertensive disease in pregnancy, which includes preeclampsia-eclampsia, is one of the leading causes of maternal morbidity

and mortality both in the United States and worldwide, resulting in 10–15% of maternal deaths.^{2–4} Eclampsia is further associated with increased morbidity due to risks of hypoxic-ischemic brain damage and intracranial hemorrhage from seizure activity.⁵ Of eclampsia cases, 2–20% are complicated by perinatal loss, while 1–20% are complicated by maternal fatality, with the highest rates of morbidity and mortality in developing countries.^{4,6–8} The risk of eclamptic seizures is approximately 2% in patients with untreated severe preeclampsia, while it is roughly 0.5% in patients with mild preeclampsia.⁹ Eclampsia may occur antenatally, intrapartum, or postnatally.

The incidence of eclampsia is cited to be on the order of 2–8 cases per 10,000 deliveries in developed countries and up to 16–69 cases per 10,000 in developing countries.^{6,10} Several recent studies from Canada and Ireland have demonstrated a decline in eclampsia incidence over time. Liu et al⁶ found a decline from 12.4 per 10,000 to 5.9 from 2003 through 2009 in Canada, while O'Connor et al⁴ found a decline from 5.4 per 10,000 to 3.5 over a span of 30 years in Ireland. It is believed that the use of magnesium sulfate prophylactically has been associated with a decrease in incidence of eclampsia.^{4,11}

The objective of our study was to use a large California cohort to assess the incidence of eclampsia and its trends over time. We also sought to identify antenatal risk factors and adverse outcomes associated with eclampsia.

Materials and Methods

This is a retrospective study using California health discharge data from 2001 through 2007. The database, provided by the California Office of Statewide Health Planning and Development (OSHPD), is a publicly available dataset comprising cases where a patient is treated in a licensed general acute care hospital in California.¹² It contains information regarding demographics, hospital of treatment, diagnoses, specific procedures undergone, and details regarding the patient's stay such as source of funding and length of stay. The dataset has undergone prior validation studies specifically in pregnancy-related conditions and has been used in prior obstetrical publications.^{13–15} The local institutional review board granted exempt approval because of the deidentified, retrospective design.

Of 3,556,567 deliveries extracted from inpatient California discharge data using delivery codes, we identified cases of eclampsia using *International Classification of Diseases*, Ninth Revision (ICD-9) coding for “eclampsia complicating pregnancy childbirth or the puerperium” (642.60, 642.61, 642.62, 642.63, 642.64). Other maternal conditions and procedures (eg, gestational diabetes, cesarean delivery) were identified using ICD-9 coding as well, with certain antepartum conditions utilizing ICD-9 codes as needed from outside of pregnancy in addition to those during pregnancy (ie, ICD-9 codes 630–677, “complications of pregnancy, childbirth, and the puerperium”). For example, thyroid disease was coded using ICD-9 codes 240.x–246.x in addition to the pregnancy-related 648.1x code. Other conditions such as hypercoagulable state only had codes available from outside of pregnancy (289.81, “primary hypercoagulable state” and 289.82, “secondary hypercoagulable state”). For peripartum cardiomyopathy, data were analyzed only from years 2003 through 2007, since its ICD-9 code (674.5x) was only available starting in 2003.

The study group of subjects with eclampsia was compared to those without eclampsia. To improve ascertainment to only reproductive-age women, we eliminated patients <15 years of age (the lower limit of reproductive age) as defined by the World Health Organization.¹⁶ We also eliminated patients >55 years of age to allow for morbidity analysis in the high-risk very advanced maternal age population (age >45 years) while still minimizing age-related

coding error.¹⁷ We also eliminated cases missing age or race/ethnicity to minimize bias from omitted data in our logistic regression adjustments.

Student *t* test was used for calculation of continuous variables. Cochran-Armitage test was used for trends over time. Fisher exact or χ^2 test was used for comparison between discrete variables. We used a multivariable logistic regression analysis for adjustment of covariates. For antepartum morbidities, we adjusted for all factors that showed significant differences between the 2 groups: age, race/ethnicity, insurance type, and year of delivery. For peripartum morbidities, in addition to the aforementioned factors, we also adjusted for obesity, diabetes, cardiac disease, asthma, renal disease, urinary tract infection, and multiple gestations. This was done to try to establish an independent relation between eclampsia and each peripartum outcome. We also performed 2 subanalyses: an analysis of race/ethnicity risk for eclampsia using the above logistic regression adjustment, as well as an analysis of associated antepartum/peripartum factors for maternal death. Results were expressed in odds ratios (ORs) and 95% confidence intervals (CIs). A statistical software package (SPSS 20.0; IBM Corp, Armonk, NY) was used for analysis.

Results

There were 2534 cases of eclampsia of 3,556,567 deliveries, giving an incidence of 7.12 cases per 10,000 deliveries. We eliminated 3617 patients >55 or <15 years of age. In all, 782,079 cases were removed due to missing race/ethnicity. A total of 2,770,871 cases remained, composed of 1888 cases of eclampsia and 2,768,983 cases without eclampsia.

Figure 1 demonstrates the incidence of eclampsia during the study period (2001 through 2007). There was a significant decrease in incidence over time ($P < .001$). The year 2001 had the highest rate at 8.0 cases per 10,000 deliveries, decreasing to the lowest rate at 5.6 cases per 10,000 deliveries in 2007. The fastest rate of decline occurred from 2005 through 2007, when the incidence declined by nearly 25%.

The characteristics of the study population with and without eclampsia are shown in Table 1. There were differences in age, race/ethnicity, and insurance status between the 2 groups. There was a trend toward a lower percentage of subjects with advanced maternal age in the eclamptic group. The length of hospital stay for eclamptics was significantly longer, on average, by 2 days (4.54 ± 3.65 vs 2.50 ± 2.18 days, mean $\pm$ SD, $P < .001$).

Figure 2 demonstrates the prevalence of eclampsia displayed by 5-year increments of age. There was a clear bimodal distribution in age-related risk, with peaks in the youngest and oldest age groups. The eclampsia risk in subjects age <19 years was 15.71 cases per 10,000 and was 14.54 cases per 10,000 in subjects age >45 years. The nadir of age-related risk was at 4.80 cases per 10,000 in subjects aged 30–34 years.

The risk of eclampsia when compared among different races/ethnicities is demonstrated in Table 2, after adjustment for age, insurance type, year of delivery, obesity, diabetes, cardiac disease, asthma, renal disease, urinary tract infection, multiple gestations, and tobacco use. Using Caucasians as a referent group, non-Hispanic blacks were almost twice as likely to develop eclampsia (adjusted OR, 1.81; 95% CI, 1.51–2.17) while Hispanics were about a fourth more likely (adjusted OR, 1.27; 95% CI, 1.14–1.42).

Several antepartum obstetrical and medical complications, after adjustment for age, race/ethnicity, insurance type, and year of delivery, were associated with an increased rate of eclampsia (Table 3). Preexisting cardiac disease had the highest association with an adjusted OR of 6.84 (95% CI, 5.40–8.66) followed by lupus erythematosus (adjusted OR, 3.68; 95% CI, 1.53–8.86), urinary tract infection (adjusted OR, 2.82; 95% CI, 2.23–3.55), renal disease

(adjusted OR, 2.44; 95% CI, 1.09–5.44), pregestational diabetes (adjusted OR, 2.73; 95% CI, 1.98–3.76), obesity (adjusted OR, 2.32; 95% CI, 1.78–3.02), and asthma (adjusted OR, 2.23; 95% CI, 1.73–2.88). Both twins and higher-order multiple gestations had >3-fold higher associations with eclampsia as well.

Morbidities occurring in the peripartum period are listed in Table 4. These were adjusted for obesity, diabetes, cardiac disease, asthma, renal disease, urinary tract infection, and multiple gestations, in addition to age, race/ethnicity, insurance type, and year of delivery. Several outcomes, whether as consequence or cause, were increased in cases of eclampsia. Cerebrovascular hemorrhage/disorders had the highest association with an adjusted OR of 112.15 (95% CI, 77.47–162.35), followed by peripartum cardiomyopathy (adjusted OR, 12.88; 95% CI, 6.08–27.25), amniotic fluid embolism (adjusted OR, 11.94; 95% CI, 3.63–39.21), venous thromboembolism (VTE) (adjusted OR, 10.71; 95% CI, 5.14–22.32), uterine rupture (adjusted OR, 5.53; 95% CI, 2.47–12.39), cesarean delivery (adjusted OR, 4.01; 95% CI, 3.64–4.41), fetal distress (adjusted OR, 3.45; 95% CI, 2.54–4.68), and intrauterine fetal demise (adjusted OR, 3.04; 95% CI, 2.06–4.49).

Using the unedited cohort, 16 maternal deaths occurred of 2534 cases of eclampsia, giving a case fatality rate of 6.31 cases per 1000. Compared to the noneclamptic population, eclamptics had a >16-fold odds of death (adjusted OR, 16.69; 95% CI, 8.06–34.57). Of these 16 deaths, 9 had identifiable race/ethnicity data; 2 deaths (22%) occurred in Caucasians, 5 (56%) in Hispanics, and 2 (22%) in non-Hispanic blacks. Associated antepartum morbidities found to be significantly associated with death (calculated using Fisher exact test) included amniotic fluid embolism (OR, 55.89; 95% CI, 5.50–568.36), cerebrovascular hemorrhage/disorders (OR, 39.19; 95% CI, 14.03–109.49), VTE (OR, 33.51; 95% CI, 3.69–304.28), preexisting cardiac disease (OR, 24.96; 95% CI, 9.18–67.90), lupus erythematosus (OR, 23.91; 95% CI, 2.77–206.51), and intrauterine fetal demise (OR, 20.52; 95% CI, 5.54–76.02).

Comment

Our large population-based study demonstrates several interesting aspects of eclampsia using a contemporary large California population. The overall incidence was 7.1 per 10,000 deliveries. There was a notable decline during the study period, which is the first time to our knowledge that this finding has been demonstrated in a US population. A decreasing incidence trend has also been validated by other recent studies, but these were performed in Canada and Ireland.^{4,6} Some potential reasons for this decline are improved access to prenatal care, as well as increased recognition of the importance of critical care management in obstetrics.^{18,19} Another possible reason is an increased use of magnesium sulfate, which has been used for seizure prophylaxis in the United States since the mid 20th century.²⁰ The role of magnesium sulfate was confirmed by the Magpie trial, a landmark multicenter randomized controlled study published in 2002 demonstrating that magnesium sulfate cut the risk of eclampsia by 50% when used in patients with preeclampsia.¹¹ Several publications have cited the trial as a reason for the increased mainstream use of magnesium sulfate both in the developed and developing world.^{4,6} Unfortunately, use of magnesium sulfate is not coded via ICD-9, so it is not possible to corroborate this increased use specifically in our population.

Many antepartum factors such as cardiac disease, lupus erythematosus, and multiple gestations were associated with an increased risk of eclampsia. Several of these associations have also been demonstrated in a recent retrospective study by Liu et al⁶ using a large population cohort in Canada.

Our data demonstrate that the risk of VTE is >10-fold higher in subjects with eclampsia. Recent recommendations by organizations such as the American College of Chest Physicians and Royal College of Obstetrics and Gynecology have cited preeclampsia-eclampsia as a high-risk condition for thrombosis and recommend that these patients should be treated with postpartum thromboprophylaxis.^{21,22} Given that VTE prevention is a major hospital quality measure, our findings also suggest that eclamptics are an extremely high-risk group for developing VTE, and should be strongly considered for thromboprophylaxis.

Eclampsia also appears to have a disproportionately high association with other catastrophic outcomes (cerebrovascular disorders/hemorrhage, peripartum cardiomyopathy, amniotic fluid embolism). Awareness of an increased association of these conditions with eclampsia is important, since immediate attention is required if a patient develops clinical signs of these conditions. They also pose an interesting question as to how eclampsia is pathophysiologically linked to these conditions—whether it is solely due to hypertensive sequelae, or if there is another explanation. Our study did find associations between several conditions (lupus erythematosus, preexisting cardiac disease, intrauterine fetal demise, and others) with maternal death in our eclampsia cohort. Future research should identify why and how these associations occur, as this could help target future treatment/prevention endeavors.

Our study was limited to events during each admission for delivery. While preeclampsia and eclampsia both have increased risk of both cerebrovascular and cardiovascular risks at the time of the affected pregnancy, there is increasing evidence that preeclampsia may also be a risk factor for future cardiovascular and cerebrovascular events. A large population-based prospective cohort study with long-term follow-up conducted in northern Finland demonstrated that even isolated gestational hypertension alone had a stronger association with subsequent cardiovascular diseases.²³ Two large metaanalyses found that in later life, subjects with preeclampsia/eclampsia had 2 times higher risk of ischemic heart disease and stroke compared to their nonpreeclamptic counterparts.^{24,25} Hence, appropriate prevention, identification, and treatment of eclampsia in pregnancy is of utmost importance, not just for the patient's immediate health but for her long-term health as well.

The demographic findings in our study have been reflected elsewhere in the literature as well. A study by Gong et al²⁶ using a New York birth cohort found similar differences in ethnicity and eclampsia risk. The publication by Gong et al²⁶ further stratified its patients into country of origin, but the overall ethnic risks were congruent with ours.

In using such a large database for analysis, we acknowledge there are some limitations. The California OSHPD maintains unique patient records/confidentiality by masking certain variables—this resulted in a large portion of our original cohort being eliminated simply due to missing age or race/ethnicity data. Fortunately, due to size of the dataset, >2.7 million cases (including 1888 cases of eclampsia) still remained, precluding any concern of adequate sample size.

Large population datasets are inherently susceptible to inaccuracies in ICD-9 coding. Romano et al¹³ found in a validation study of OSHPD data from 1992 through 1993 that there was very high sensitivity (>90%) in the coding of delivery types (cesarean, forceps) as well as certain peripartum conditions. There was moderate-high accuracy in coding of conditions such as chorioamnionitis, diabetes, premature labor, and preeclampsia, but poor sensitivity (<60%) for other conditions such as anemia, asthma, and obesity. On the other hand, a 2005 article reviewing the history of ICD coding by O'Malley et al²⁷ suggests that accuracy of ICD coding has continued to improve over time. This lends hope to the

possibility that the coding accuracy has since improved in our data, since our dataset is much more recent (2001 through 2007) than the data used in the study of Romano et al.¹³

It has been suggested that in studies using large population-based cohorts, the actual strength of association may be overestimated by the calculated OR.²⁸ Thus, ORs in the lower range (eg, OR 1–2) should be interpreted with caution, as they may not be clinically significant. Care must also be made in assuming an actual biological link of an independent variable to the outcome. For example, although our study demonstrated a significant association between eclampsia and uterine rupture, there is not a clear pathophysiological association between the 2 conditions. Such observations could be due to chance and, as in most large retrospective datasets, only relationships of association can be established, rather than causality. Other reasons such as coding/misclassification bias are possible, although they should be less likely with sentinel events such as eclampsia and uterine rupture.

The dataset did not include certain information such as birthweight, neonatal outcomes, or gestational age at delivery. We were also unable to identify some cases of eclampsia due to the sheer nature in ICD-9 coding. In the final dataset, 6945 cases of ICD-9 code 642.7x, “preeclampsia or eclampsia superimposed on preexisting hypertension,” were not included as eclampsia cases, due to the fact that we could not discern which were specific for eclampsia. Consequently, we may have under-reported eclampsia incidence. However, assuming a <1% seizure risk in patients with preeclampsia as reported by the Magpie trial, this would mean that we may have underreported approximately 69 cases.¹¹ Given the 1888 cases we had in our analysis, absence of this small number of eclampsia cases should likely not affect our morbidity and risk factor calculations, and may actually strengthen our associations. Finally, our incidence of 7.1 in 100,000 is still similar to that reported in other studies, including those that used newer, more specific ICD, 10th Revision coding.^{6,8,10,29}

In conclusion, our large population-based study highlights certain aspects of eclampsia in this diverse California population. Eclampsia incidence appears to be decreasing. Several antepartum risk factors as well as highly morbid outcomes appear to be increased in eclampsia. Future studies should aim to characterize any pathophysiological connections between eclampsia and these antepartum and peripartum associations. This may help identify potential ways of preventing this extremely high-risk condition in mothers.

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FIGURE 1.
Incidence of eclampsia from 2001 through 2007

FIGURE 2.
Prevalence of eclampsia vs age

TABLE 1

Baseline characteristics between women with eclampsia and those without

Baseline characteristic	Subjects with eclampsia (n = 1888)	Subjects without eclampsia (n = 2,768,983)	P value
	n (prevalence)	n (prevalence)	
Age, y			< .001
15–19	352 (18.6%)	229,407 (8.3%)	
20–24	521 (27.6%)	647,623 (23.4%)	
25–29	397 (21.0%)	732,882 (26.5%)	
30–34	318 (16.8%)	682,994 (24.7%)	
35–39	229 (12.1%)	388,867 (14.0%)	
40–44	63 (3.3%)	82,628 (3.0%)	
45	8 (0.4%)	4582 (0.2%)	
Advanced maternal age (35 y)	300 (15.9%)	476,077 (17.2%)	.071
Race/ethnicity			< .001
Caucasian	596 (31.6%)	1,056,824 (38.2%)	
Black	164 (7.6%)	120,853 (3.9%)	
Native American/Eskimo/Aleut	2 (0.1%)	3685 (0.1%)	
Asian/Pacific Islander	96 (4.4%)	241,486 (7.7%)	
Hispanic	1030 (54.6%)	1,346,135 (48.6%)	
Private insurance	785 (41.6%)	1,352,917 (48.9%)	< .001
Length of stay, d	4.54 ± 3.65	2.50 ± 2.18	< .001

Expressed as mean ± SD. Calculations done via χ^2 or *t* test, where appropriate.

TABLE 2**Racial/ethnic risks of eclampsia**

Variable	Non-Hispanic Caucasian	Non-Hispanic black	Asian	Hispanic	Native American
Odds of eclampsia	1	1.81 (1.51–2.17) $P < .001$	0.83 (0.67–1.04)	1.27 (1.14–1.42) $P < .001$	0.73 (0.18–2.95), $P = .66$

All results expressed as odds ratio (95% confidence interval). Non-Hispanic Caucasians used as referent group. Adjusted for age, insurance type, year of delivery, obesity, diabetes, cardiac disease, asthma, renal disease, urinary tract infection, multiple gestations, and tobacco use.

TABLE 3

Associations between subjects with eclampsia and antepartum obstetrical and medical complications

Obstetrical and medical complication	<u>Rate of eclampsia per 10,000 (no. of cases)</u>		Unadjusted OR (95% CI)	P value	Adjusted OR (95% CI)	P value
	Condition present	Condition absent				
Pregestational diabetes	17.25 (39)	6.73 (1849)	2.57 (1.87–3.530)	< .001	2.73 (1.98–3.76)	< .001
Gestational diabetes	8.43 (124)	6.72 (1764)	1.25 (1.05–1.51)	.016	1.47 (1.22–1.78)	< .001
Obesity	14.68 (57)	6.70 (1831)	2.19 (1.68–2.85)	< .001	2.32 (1.78–3.02)	< .001
Asthma	15.10 (62)	6.69 (1826)	2.26 (1.75–2.91)	< .001	2.23 (1.73–2.88)	< .001
Thyroid dysfunction	10.85 (36)	6.76 (1852)	1.61 (1.15–2.23)	.006	1.97 (1.41–2.75)	< .001
Renal disease	16.63 (6)	6.80 (1882)	2.45 (1.10–5.46)	.039	2.44 (1.09–5.44)	.029
Lupus erythematosus	23.65 (5)	6.80 (1883)	3.48 (1.45–8.39)	.016	3.68 (1.53–8.86)	.004
Urinary tract infection/ asymptomatic bacteriuria	20.72 (75)	6.63 (1813)	3.13 (2.48–3.94)	< .001	2.82 (2.23–3.55)	< .001
Drug dependence	15.68 (6)	6.80 (1882)	2.31 (1.03–5.15)	.049	2.15 (0.96–4.81)	.062
Preexisting cardiac disease	40.61 (73)	6.59 (1815)	6.18 (4.89–7.81)	< .001	6.84 (5.40–8.66)	< .001
Twin gestation	20.30 (109)	6.54 (1779)	3.11 (2.56–3.77)	< .001	3.28 (2.70–3.99)	< .001
Higher-order multiple gestation (triplets)	19.67 (6)	6.80 (1882)	2.90 (1.30–6.46)	.019	3.27 (1.46–7.31)	.004

CI, confidence interval; *OR*, odds ratio.

TABLE 4

Differences in peripartum morbidity between subjects with eclampsia compared to those without

Peripartum morbidity	Rate per 100 of morbidity unless otherwise specified (no. of cases)		Unadjusted OR (95% CI)	P value	Adjusted OR (95% CI)	P value
	Eclampsia present (n = 1888)	Eclampsia absent (n = 2,768,983)				
Fetal distress (per 1000)	22.78 (43)	5.93 (16,423)	3.91 (2.89–5.29)	< .001	3.45 (2.54–4.68)	< .001
Failed induction	4.71 (89)	1.54 (42,545)	3.17 (2.56–3.92)	< .001	2.94 (2.37–3.64)	< .001
Abnormal fetal heart rate	19.28 (364)	12.1 (335,427)	1.73 (1.55–1.94)	< .001	1.67 (1.49–1.87)	< .001
Intrauterine fetal demise (per 1000)	13.77 (26)	3.90 (10,804)	3.57 (2.42–5.25)	< .001	3.04 (2.06–4.49)	< .001
Uterine rupture (per 10,000)	31.78 (6)	5.3 (1477)	5.97 (2.68–13.33)	.001	5.53 (2.47–12.39)	< .001
Cesarean delivery	61.86 (1168)	29.3 (811,453)	3.91 (3.57–4.29)	< .001	4.01 (3.64–4.41)	< .001
Postpartum hemorrhage	6.41 (121)	2.6 (72,987)	2.53 (2.10–3.04)	< .001	2.33 (1.94–2.80)	< .001
Venous thromboembolism (per 10,000)	42.37 (8)	1.77 (492)	23.95 (11.89–48.22)	< .001	10.71 (5.14–22.32)	< .001
Amniotic fluid embolism (per 100,000)	158.90 (3)	4.69 (130)	33.90 (10.78–106.57)	< .001	11.94 (3.63–39.21)	< .001
Cerebrovascular hemorrhage/disorders (per 10,000)	195.97 (37)	1.21 (335)	165.20 (117.28–232.70)	< .001	112.15 (77.47–162.35)	< .001
Peripartum cardiomyopathy (per 10,000) ^a	347.49 (9)	6.54 (1307)	55.04 (28.25–107.26)	< .001	12.88 (6.08–27.25)	< .001
Death (per 100,000)	476.69 (9)	7.87 (218)	60.83 (31.19–118.67)	< .001	16.69 (8.06–34.57)	< .001

CI, confidence interval; OR, odds ratio.

^a Calculated for years 2003 through 2007 only (eclampsia present = 1319; eclampsia absent = 1,998,567). Not significant: dilation and curettage postdelivery, manual extraction of placenta, retained placenta, bladder repair, hematoma evacuation, cervical laceration repair, failed operative vaginal delivery, hysterectomy.