Risk of postpartum readmission for depression in relation to ischaemic placental disease: a population-based study

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Summary

Background There are limited data on postpartum readmissions for depression in the United States (US). Specifically, the extent to which ischaemic placental disease (IPD) during pregnancy predisposes patients to develop postpartum depression remains poorly understood. We investigated whether IPD is associated with postpartum readmission for new-onset depression in the first year after delivery.

Methods In this population-based study, the 2010–2018 Nationwide Readmissions Database was utilised to evaluate rates of postpartum readmission for depression within the calendar year of delivery hospitalisation among patients with and without IPD. IPD was defined as preeclampsia, placental abruption, or small for gestational age (SGA) birth. We expressed associations between IPD and depression readmission based on a confounder-adjusted hazards ratio (HR) with a 95% confidence interval (CI).

Findings Of 33.3 million delivery hospitalisations, 3,027,084 (9.1%) had IPD. The total follow-up among those with and without IPD were 17,855,830 and 180,100,532 person-months, respectively, with a median follow-up of 5.8 months for both groups. Rates of depression readmission were 95.7 (n = 17,095) and 37.5 (n = 67,536) per 100,000 readmissions among patients with and without an IPD, respectively (HR, 2.39; 95% CI, 2.32–2.47); this risk was the highest for preeclampsia with severe features (HR, 3.14; 95% CI, 3.00–3.29). Patients had a greater risk of readmission if they had any two forms of IPD (HR, 3.02; 95% CI, 2.75–3.33), and those with a concurrent diagnosis of preeclampsia and abruption posed the highest risk (HR, 3.23; 95% CI, 2.71–3.86).

Interpretation These findings suggested that patients with IPD are at a substantially increased risk of readmission for depression within a year following delivery. This study underscores the need for increased surveillance, improved detection, and faster treatment of depression in this vulnerable population.

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Keywords: Depression; Ischaemic placental disease; Preeclampsia; Placental abruption; Small for gestational age

Introduction

Postpartum depression accounts for significant morbidity and mortality with an estimated prevalence of 6% to more than 20% worldwide.[1–4] The peripartum period is especially vulnerable to suicide, which comprises about 20% of postpartum deaths in the United States.[5] These numbers may be underestimated with over 70% of women possibly undiagnosed with postpartum depression[6] due to variations in screening modalities or rates, cultural norms, and mental health stigma; differences in screening are further based on geography, ethnicity, or socioeconomic status as well as

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Research in context

Evidence before this study

Postpartum depression accounts for significant morbidity and mortality with an estimated prevalence of 6% to more than 20% worldwide. We searched PubMed and Google Scholar databases with the search terms that included key words and combinations of phrases "postpartum depression," "isch[a]emic placental disease," "preeclampsia," "placental abruption," and "small for gestational age infant" for articles published in English from database inception to 5th January, 2023. While some literature exists to support an association between postpartum depression and preeclampsia, the extent to which ischaemic placental disease (IPD) during pregnancy predisposes patients to develop postpartum depression remains poorly understood. Data additionally remains limited regarding postpartum readmissions for depression in the United States (US).

Added value of this study

This large population-based retrospective cohort study of 33.3 million delivery hospitalisations demonstrates that IPD including preeclampsia, placental abruption, and SGA infants are key risk factors for postpartum depression. Patients are more likely to be readmitted postpartum for depression within a year following delivery when they have IPD during pregnancy with the highest risk for those who have any form of preeclampsia. Specifically, those with preeclampsia with severe features are at the greatest risk of postpartum depression readmission. Patients having two forms of IPD have an over three-fold increased hazard of readmission for depression in the postpartum period.

Implications of all the available evidence

Our findings underscore the link between IPD and postpartum depression with data showing increased evidence of readmission for those with IPD. Given the numerous adverse consequences associated with postpartum depression, it is imperative to identify clinical predictors for optimal early detection, treatment, and prevention of negative outcomes, and our findings focus on the severity of the increased risk in this population. It is important to develop improved screening and detection modalities for those at high risk of IPD. Patients should be screened more vigilantly upon discharge who have pregnancies complicated by IPD to identify those at risk and direct them into care immediately.

obstetrical complications that augment risks such as the need for cesarean delivery, gestational diabetes, and preterm birth.[7,8] It is well established that postpartum depression adversely impacts maternal health, family life, and infant development leading to behavioural, emotional, and cognitive impairments along the life course and is implicated in poor mother-infant bonding.[4,9]

While there is no established pathophysiology for postpartum depression, evidence points to numerous risk factors. A history of depression predisposes one to develop postpartum depression.[10] Childbirth alone may be considered a traumatising experience that alters personality or causes anxiety and post-traumatic stress.[4] Prior studies highlight biological associations such as hormonal sensitivity or role of steroid and stress hormones,[11] genetics,[12] hypothalamic-pituitary-adrenal (HPA) axis dysfunction,[13] changes in neurotransmitters,[14] oxidative stress or inflammation,[15] and immune-mediated pathways.[15–17] Recent studies have purported a possible increased risk of postpartum depression among patients that are diagnosed with preeclampsia.[18,19] Preeclampsia, a common complication affecting about 5% of all pregnancies, a likely consequence of uteroplacental ischaemia, has pathophysiological mechanisms that are common to both placental abruption and small for gestational age (SGA) births.[20] These pathologies, in turn, increase the burden of oxidative stress and inflammatory factors.[21] This can contribute to postpartum depression in addition to the risk of an obstetrical complication which can itself compound the stress of childbirth. This association is grounded in studies suggesting hypertension as a risk factor for depression.[22]

While some literature exists to support an association between postpartum depression and preeclampsia,[7,23–25] the risks of postpartum depression in relation to IPD, in general, and to abruption and SGA births, remain largely unknown. Further, due to conflicting data and great morbidity at stake, it is imperative to improve our understanding of this association to potentially underscore a key clinical predictor—IPD—of new postpartum psychiatric disease and thus improve early recognition and prevention or intervention for those at highest risk. Given that population-based postpartum readmission rates for depression in those who had IPD during pregnancy have not been well characterised, the purpose of this study was to evaluate the risk of readmissions for depression within the calendar year of delivery complicated by preeclampsia, placental abruption, and SGA births, all markers for IPD.

Methods

Study design

We performed a population-based retrospective cohort study in which eligible patients with pregnancy and delivery were identified and followed up for one-year hospital readmission during each individual year of the US Nationwide Readmissions Database (NRD) between 2010 and 2018. The NRD is an all-payer database that consists of hospital inpatient stays as part of the

Health Care Utilization Project (HCUP) by the Agency for Healthcare Research and Quality (AHRQ).[26] This database includes information collected on a state level to allow for tracking of patients across hospital admissions within a state. A total of 30 individual state inpatient databases contributed to the 2018 NRD, and population weights provided by the database allow researchers to determine discharge estimates that are nationally representative. Investigators had training and followed the data use agreements with the HCUP. Since the NRD is a publicly available deidentified database, this study was exempted by the Rutgers University Institutional Review Board and informed consent was not needed. This manuscript was formatted according to the STROBE guidelines.[27]

Ischaemic placental disease

The key exposure, IPD, was defined as a syndrome of preeclampsia, placental abruption, or SGA birth.[28,29] These conditions were identified based on The *International Classification of Diseases, Ninth and Tenth Revisions, Clinical Modification* (*ICD-9-CM* and *ICD-10-CM*) codes. The codes for preeclampsia, placental abruption, and SGA births are shown in eTable S1 in the supplement.

Postpartum depression

We identified patients admitted for postpartum depression based on *ICD-9-CM* and *ICD-10-CM* (see eTable S1 in the supplement). We included codes to focus only on postpartum depression, rather than including other major psychiatric illnesses, given its prevalence and the ability to perform targeted impactful interventions in advance if identified. To avoid temporal bias regarding historical versus new diagnoses, women with diagnoses of any psychiatric diseases before or during the index delivery hospitalisation were excluded. This additionally allowed us to exclude those with a prior diagnosis of depression, which could also impact pregnancy and risk for postpartum depression.

Patient characteristics including maternal age, insurance payer, and median ZIP code income quartile were analysed. Hospital factors including hospital teaching status, hospital type, and hospital bed size were also studied. The primary outcome was one-year hospital readmission. Readmission was defined as readmission to the same or different hospital within one calendar year of discharge from the index hospitalisation. Hospital transfers were not included as readmissions, and only the first readmission was included in the analysis to account for multiple readmissions. Secondary outcomes included the analysis of the contribution of combined IPD factors on depression readmission.

Statistical analysis

We estimated rates of hospital readmission for depression within the calendar year following delivery between patients that were diagnosed with and without IPD. We fit Cox proportional hazards regression to estimate the associations between IPD and hospital readmission for depression. We established risk sets with the date of delivery being the entry time, and date of readmission as the 'event.' Patients that were never hospitalised within the calendar year (i.e., by 31 December of the given delivery year) or those that died (regardless of the cause of death) were censored. Since the HCUP data only provides the month and year of delivery and hospitalisation, but not the day, we imputed '15' as the day. We derived the person-month of follow-up for every patient.[30]

From the Cox models, we derived the unadjusted and confounder-adjusted hazards ratio (HR) and 95% confidence interval (CI) and the effect measure. We adjusted for maternal age, insurance payer, median ZIP code income quartile, hospital teaching status, hospital type, and hospital bed size as potential confounders. All analyses were weighted by NRD sampling weights to enable the generalisation of associations to the American Hospital Association (AHA) Annual Survey of Hospitals in the US.

Given the potential for the associations to be affected by unmeasured confounding, we calculated the E-value.[31,32] The E-value provides an assessment of what the minimum value of the unmeasured confounder be (on the HR scale), over and above the observed confounders, to nullify the observed HR and to move the lower limit of the 95% CI to overlap with the null.[31,32]

All statistical analyses were performed using SAS (version 9.4; SAS Institute Inc., Cary, NC, USA).

Role of the funding source

There was no funding source for this study. HLG and CVA take responsibility for the integrity of the data, the accuracy of the statistical analysis, and verifying the underlying data. All authors were responsible for the decision to submit the manuscript.

Results

The NRD identified about 33.3 million (weighted) delivery hospitalisations from 2010 to 2018 that met inclusion criteria (Fig. 1). We identified a total of 3,027,084 deliveries (9.1%) with a recorded diagnosis of IPD. Patient demographics and characteristics of these two groups are described in Table 1.

Of all readmissions for depression, 17,095 (20.2%) had any IPD during pregnancy and 67,536 (79.8%) did not. The median follow-up, or time between delivery hospitalisation and readmission, among those with IPD and without IPD was 5.8 months for both groups. Specifically, of those with an ischaemic placental disease, 366,257 (1.1%) had a placental abruption, 1,899,920 (5.7%) had any type of preeclampsia, 1,004,369 (3.0%) with preeclampsia without severe

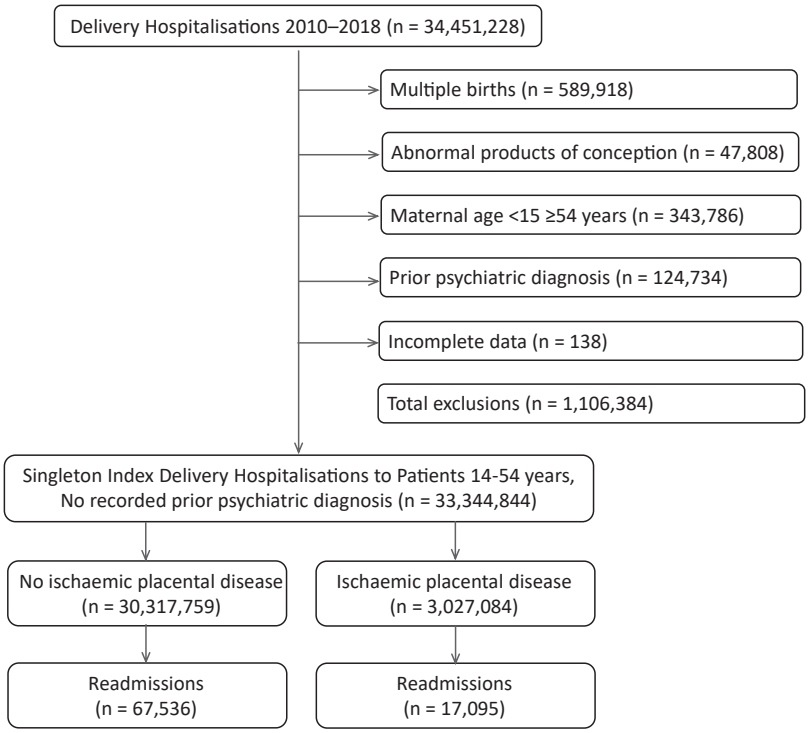

Fig. 1: Flow chart of the patient selection process.

features and 895,551 (2.7%) with preeclampsia with severe features, and 961,758 (2.9%) had an SGA delivery.

Rates of readmission for depression in relation to IPD are shown in Table 2. Patients with pregnancies complicated by any IPD were at over two-fold increased hazards of readmission for depression. Patients with preeclampsia with severe features had the highest risk of readmission, followed by patients with preeclampsia without severe features, placental abruption, and then patients with small for gestational age neonates.

Assessment of one versus two or three forms of IPD to determine compounded risk was performed (Table 3) and showed that having two or three forms of the disease strongly increased the overall readmission rate for depression (two: HR, 3.02; 95% CI, 2.75–3.33; three: HR, 2.53; 95% CI, 1.52–4.22) in contrast to having just one form of IPD (HR, 2.35; 95% CI, 2.28–2.43). A combination of any preeclampsia with placental abruption or any preeclampsia with SGA posed the greatest risk (HR, 3.23; 95% CI, 2.71–3.86 and HR, 3.11; 95% CI, 2.76–3.49, respectively) (Table 4).

We assessed the extent to which unmeasured confounding may have affected the reported associations between IPD and readmission for depression (eTable S2 in the Supplement). The high E-values relative to the confounder-adjusted HRs and the lower limit of the observed 95% CI suggest it is very unlikely that the reported associations are biased by unmeasured confounders.

Discussion

Postpartum depression continues to have a widespread devastating impact. This study aimed to evaluate the influence of IPD, specifically preeclampsia, placental abruption, and SGA births, on readmission to the hospital within the first year after delivery for depression. To date, to the best of our knowledge, this is the largest population-based study to assess postpartum depression readmission. This analysis demonstrated that women are more likely to be readmitted postpartum for depression within a year following delivery when they have IPD during pregnancy with the highest risk for those who have any form of preeclampsia. Moreover, those having two forms of IPD have over three-fold increased hazard of readmission for depression in the postpartum period. These findings underscore the association between IPD and depression, but also highlight the need for increased surveillance as well as early recognition and interventions in this population.

Our research findings exemplify the link between IPD and postpartum depression, consistent with results from previous studies that suggest an association between preeclampsia and postpartum depression. While we recognise that IPD may be associated with the risk of

	Ischaemic placental disease (n = 3,027,084)	No ischaemic placental disease (n = 30,317,759)
Ischaemic placental disease		
Any ischaemic placental disease	3,027,084 (9.1)[a]	
Any preeclampsia	1,899,920 (5.7)[a]	
Preeclampsia without severe features	1,004,369 (3.0)[a]	
Preeclampsia with severe features	895,551 (2.7)[a]	
Placental abruption	366,257 (1.1)[a]	
Small for gestational age birth	961,758 (2.9)[a]	
Patient factors	**Number (%)**	**Number (%)**
Maternal age (years)		
<20	267,587 (8.8)	2,085,536 (6.9)
20–24	315,460 (22.8)	3,056,910 (21.8)
25–29	806,641 (26.7)	8,758,034 (28.9)
30–34	728,598 (24.1)	8,102,140 (26.7)
35–39	410,431 (13.6)	3,855,178 (12.7)
40–44	113,911 (3.8)	825,542 (2.7)
45–49	9768 (0.3)	89,201 (0.3)
Payer		
Medicare	32,909 (1.1)	208,572 (0.7)
Medicaid	1,430,331 (47.3)	12,758,852 (42.1)
Private	1,425,184 (47.1)	15,846,450 (52.3)
Self-pay	42,797 (1.4)	476,635 (1.6)
Other	89,081 (2.9)	963,216 (3.2)
Missing values	6784 (0.2)	64,034 (0.2)
Median ZIP code income quartile		
Lowest income quartile	993,026 (32.8)	8,320,720 (27.5)
2nd lowest quartile	769,859 (25.4)	7,597,549 (25.1)
2nd highest quartile	702,910 (23.2)	7,550,573 (24.9)
Highest income quartile	531,891 (17.6)	6,571,757 (21.7)
Missing values	29,399 (1.0)	277,160 (0.9)
Hospital factors		
Hospital teaching status		
Metropolitan non-teaching	783,279 (25.9)	9,448,389 (31.2)
Metropolitan teaching	1,970,801 (65.1)	17,701,917 (58.4)
Non-metropolitan	273,005 (9.0)	3,167,452 (10.5)
Hospital type		
Private, not for profit	2,286,876 (75.6)	22,943,481 (75.7)
Private, invest-own	348,240 (11.5)	3,819,743 (12.6)
Government, non-federal	391,968 (13.0)	3,554,536 (11.7)
Hospital bed size		
Small	375,409 (12.4)	4,123,170 (13.6)
Medium	799,183 (26.4)	8,355,160 (27.6)
Large	1,852,493 (61.2)	17,839,429 (58.8)
Year of delivery hospitalisation		
2010	300,094 (9.9)	3,448,343 (11.4)
2011	296,863 (9.8)	3,409,731 (11.3)
2012	304,996 (10.1)	3,388,941 (11.2)
2013	311,357 (10.3)	3,387,403 (11.2)
2014	323,743 (10.7)	3,403,446 (11.2)
2015	341,161 (11.3)	3,391,407 (11.2)
2016	347,910 (11.5)	3,401,051 (11.2)
2017	388,031 (12.8)	3,286,595 (10.8)
2018	412,928 (13.6)	3,200,843 (10.6)

[a]The prevalence rates correspond to the total population of delivery hospitalisations.

Table 1: **Patient demographic and hospital characteristics for those with and without ischaemic placental disease: Nationwide Readmissions Data, 2010–2018.**

	Total delivery hospitalisations (weighted)	Person-months	Number of depression readmission cases (Rate per 100,000)	Hazards ratio (95% confidence interval)	
				Unadjusted	Adjusted[a]
No ischaemic placental disease	30,317,759	180,100,532	67,536 (37.5)	1.00 (Reference)	1.00 (Reference)
Any ischaemic placental disease	3,027,084	17,855,830	17,095 (95.7)	2.49 (2.41–2.56)	2.39 (2.32–2.47)
Any preeclampsia	1,899,920	11,221,556	12,526 (111.6)	2.89 (2.79–2.99)	2.76 (2.66–2.85)
Preeclampsia without severe features	1,004,369	5,973,144	5639 (94.4)	2.41 (2.30–2.52)	2.34 (2.24–2.45)
Preeclampsia with severe features	895,551	5,248,411	6887 (131.2)	3.35 (3.20–3.51)	3.14 (3.00–3.29)
Placental abruption	366,257	2,169,116	1907 (87.9)	2.30 (2.13–2.49)	2.25 (2.08–2.43)
Small for gestational age birth	961,758	5,644,768	4103 (72.7)	1.92 (1.81–2.03)	1.85 (1.74–1.96)

[a]Hazards ratios were adjusted for confounding effects of maternal age, insurance status, income quartile, hospital bed size, hospital type, hospital teaching status, and year of delivery hospitalisation from Cox proportional hazards regression models.

Table 2: **Rates postpartum readmission for depression in relation to ischaemic placental disease: Nationwide Readmissions Data, 2010–2018.**

postpartum psychiatric illness, no study to date has assessed the readmission to the hospital, which may underscore the gravity of the situation. A recent meta-analysis[23] showed that three studies focused on preeclampsia as a risk factor for postpartum depression. Specifically, Blom et al.[7] demonstrated a 2.58 times increased risk for depression up to two months postpartum in those with preeclampsia, Bergink et al.[24] highlighted the increased risk up to 3 months postpartum with increased risk of preeclampsia by 2.85 times, and Meltzer-Brody et al.[25] showed that preeclampsia increased the risk of depression in the first year by 1.45 times. Additional evidence supported a worsened severity of depression symptoms in those who had preeclampsia when compared to those who did not.[23,25,33,34] Further, an association between preeclampsia and other postpartum psychiatric diseases such as psychosis has been demonstrated.[34] Despite this evidence, there are studies with conflicting data that do not suggest an association between preeclampsia and depression.[35] Furthermore, while there are a few studies suggesting an association between psychiatric disease and placental abruption,[36] there is very limited data regarding postpartum depression after a delivery complicated by placental abruption.

Ischaemic placental disease	Hazards ratio (95% confidence interval)	
	Unadjusted	Adjusted[a]
No ischaemic placental disease	1.00 (Reference)	1.00 (Reference)
One ischaemic placental disease	2.43 (2.36–2.51)	2.35 (2.28–2.43)
Two ischaemic placental disease	3.27 (2.97–3.59)	3.02 (2.75–3.33)
Three ischaemic placental disease	2.66 (1.59–4.44)	2.53 (1.52–4.22)

[a]Hazards ratios were adjusted for confounding effects of maternal age, insurance status, income quartile, hospital bed size, hospital type, hospital teaching status, and year of delivery hospitalisation from Cox proportional hazards regression models.

Table 3: **Risk of postpartum readmission for depression based on the number of ischaemic placental disease conditions: Nationwide Readmissions Data, 2010–2018.**

There is a psychological impact of preeclampsia, placental abruption, and SGA delivery on the mother. Evidence shows increased susceptibility to postpartum depression, anxiety, acute stress, and PTSD-like symptoms in these patients.[36] Moreover, there are shared risk factors between IPD and postpartum depression that may establish a more united link in pathophysiology. Both disorder types have their initial onset more associated with a primary pregnancy and augmented risk occurs with longer intervals between subsequent pregnancies. Also, a history of a prior episode is the strongest risk factor for subsequent pregnancies.[37,38]

Further, there is biological plausibility to explain the relationship between IPD and postpartum depression. Preeclampsia has been the focus of different studies as it is associated with other obstetrical outcomes including fetal growth restriction and placental abruption[39] and is tied to increased risk of depression. Specifically, increased vascular inflammation is purported to associate IPD and postpartum psychiatric illness. With preeclampsia, there is vascular remodeling of uterine spiral arterioles leading to poor placental perfusion and impairment in function. This results in an ischaemic process of excess oxidative stress and increased inflammatory factors in the blood suggesting a mode of inflammation after exaggerated endothelial activation causing changes in brain function as a late-stage impact of preeclampsia. This altered immunologic response can be implicated in pathogenesis. Similarly, inflammation and oxidative stress are related to the start of depressive symptoms in unipolar depression although evidence correlating these biomarkers to mood symptoms is limited.[40] While this may elucidate the link between IPD and postpartum depression, there is a dearth of investigations specifically focused on placental abruption or SGA with the majority of research highlighting preeclampsia.

Ischaemic placental disease	Hazards ratio (95% confidence interval)	
	Unadjusted	Adjusted[a]
No ischaemic placental disease	1.00 (Reference)	1.00 (Reference)
Preeclampsia (any) only	2.84 (2.73–2.94)	2.72 (2.62–2.82)
Placental abruption only	2.17 (1.98–2.37)	2.13 (1.95–2.33)
Small for gestational age birth only	1.67 (1.56–1.79)	1.63 (1.52–1.74)
Preeclampsia and placental abruption	3.48 (2.92–4.14)	3.23 (2.71–3.86)
Preeclampsia and small for gestational age birth	3.38 (3.01–3.79)	3.11 (2.76–3.49)
Placental abruption and small for gestational age birth	1.85 (1.59–2.64)	1.79 (1.25–2.56)
All three present	2.66 (1.59–4.44)	2.53 (1.52–4.22)

[a]Hazards ratios were adjusted for confounding effects of maternal age, insurance status, income quartile, hospital bed size, hospital type, hospital teaching status, and year of delivery hospitalisation from Cox proportional hazards regression models.

Table 4: **Associations between ischaemic placental disease and risk of postpartum readmission for depression: Nationwide Readmissions Data, 2010–2018.**

Women with preeclampsia have been shown to have increased proinflammatory cytokine levels in serum produced by macrophages, natural killer (NK) cells, and Th1 cells with higher levels of IL-6, IL-8, IL-1β, TNF-α, and C-reactive protein.[38,41,42] Such cytokines like IL-6 have been additionally shown to promote the development of depression and psychosis in young adulthood.[43] Lastly, endothelial dysfunction combined with inflammation that occurs in preeclampsia can promote blood-brain barrier permeability and affect brain neurotransmitter function, neuroendocrine function, synaptic plasticity, and neural circuits to affect mood.[7,44]

Additionally, multiple genetic factors and changes in gene expression could help explain the link between preeclampsia and postpartum depression. Li et al.[45] found that genetic polymorphisms in 5,10-methylenetetrahydrofolate reductase (MTHFR) were independent risk factors for the development of postpartum depression. A meta-analysis of 54 studies showed that changes in the MTHFR genotype are also a risk factor for preeclampsia.[46] Additional abnormal expression of common genes like 5-hydroxytryptamine transporter (5-HTT) and estrogen receptor (ESR) has been implicated in augmenting one's susceptibility to both preeclampsia and postpartum depression.[14]

Experts have advocated for closer monitoring of patients for postpartum depression specifically with screening tools like the Edinburgh Postnatal Depression Scale.[24,47] Despite an abundance of literature on postpartum depression and our evidence highlighting increased readmission for those with IPD, the psychiatric disease is still often missed or diagnosed late with lack of or delays in treatment.[48,49] Given the numerous adverse consequences associated with postpartum depression, it is crucial to identify clinical predictors for optimal early detection, treatment, and prevention of negative outcomes.[50] While overall depression readmission rates for those with IPD are low, our findings focus on the severity of the increased risk in this population. Screening of all patients with IPD, in general, including those without depression readmission is beneficial. Thus, it is important to develop improved screening and detection modalities for those at high risk with IPD. Clinical implications of this study include that it may be reasonable to screen women more vigilantly upon discharge who have pregnancies complicated by IPD to identify those at risk and direct them into care immediately.

This is the largest study to date examining the relationship between IPD and postpartum readmission for depression. The results were from a large, nationally representative database over several years, making this study generalisable. Further, given the strong association between IPD and depression, the results are likely generalisable to other countries. While misclassification and under-ascertainment are always potential issues with the utilisation of administrative diagnosis codes, the analysis demonstrated appropriate rates of disease and readmission.

This study also had limitations that should be addressed. The NRD does not collect or have complete data for certain variables (i.e., race, obesity, outpatient data) which limits a broad overview of population-based risk without potential important clinical details. There is bias implicit in the limitation of the medical record and possible coding errors. The study cannot account for clinical management during delivery hospitalisation such as psychiatric consultation or the start of antidepressant medication at that time. We also cannot account for outpatient management of depression or visits to the emergency department without admission. Another limitation inherent to this database is that patients cannot be tracked across calendar years and given that the database is state-based, a patient may have delivered in a state different from where the readmission occurred and hospitalisations were not linked to being included in the study.

This study provides evidence that IPD including preeclampsia, placental abruption, and SGA infants are key risk factors for postpartum depression. Specifically,

women with preeclampsia with severe features are at the greatest risk of postpartum depression readmission. Our study underscores the need to raise awareness of the potential risk of depression after IPD in pregnancy. Both providers and patients with IPD should be educated on early signs and symptoms of postpartum depression, and these patients should be more closely monitored and screened to allow for improved earlier detection and intervention to improve quality of life, mental health, and potentially life-threatening consequences.

Contributors

HLG and CVA take responsibility for the integrity of the data, the accuracy of the statistical analysis, and verifying the underlying data. All authors were responsible for the decision to submit the manuscript.

Study concept and design: JCF and CVA were responsible for study concept and design.

Acquisition, analysis, or interpretation of data: All authors were responsible for acquisition, analysis, or interpretation of data.

Drafting of the manuscript: JCF was responsible for drafting of the manuscript.

Critical revision of the manuscript for important intellectual content: All authors critically revised the manuscript.

Statistical analysis: HLG and CVA were responsible for statistical analysis.

Obtained funding: No funding is applicable.

Administrative, technical, or material support: CVA provided administrative, technical, or material support.

Study supervision: CVA was responsible for study supervision.

Data sharing statement

All data utilised in this study can be accessed from the Healthcare Cost and Utilization Project (https://www.hcup-us.ahrq.gov/).

Declaration of interests

All authors declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; and no other relationships or activities that could appear to have influenced the submitted work.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.eclinm.2023.102011.

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