Published in final edited form as:

Epilepsy Behav. 2009 November ; 16(3): 426–430. doi:10.1016/j.yebeh.2009.07.009.

Postpartum depression in women with epilepsy: Influence of antiepileptic drugs in a prospective study

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Abstract

Patients with epilepsy are at high risk for major depressive disorder (MDD) and, according to one report, postpartum depression (PPD) as well. The study described here sought to determine the prevalence and risk factors for PPD among women with epilepsy. Fifty-six women with epilepsy participating in a prospective study of perinatal antiepileptic drug (AED) pharmacokinetics were included. Participants completed the Beck Depression Inventory (BDI) during pregnancy and the postpartum period. Fourteen participants (25.0%) had a postnatal BDI score ≥ 12 indicative of PPD. Logistic regression indicated that significant risk factors for PPD among women with epilepsy included multiparity (odds ratio = 12.5) and AED polytherapy (odds ratio = 9.3). The rate of PPD was unaffected by the use of specific AEDs. In conclusion, PPD rates are higher among women with epilepsy than the general population, particularly those who are multiparous or receiving AED polytherapy, and there is no evidence that AED selection modifies this risk.

Keywords

Antiepileptic drugs; Epilepsy; Polytherapy; Postpartum depression; Pregnancy

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Conflict of interest statement

Ms. Galanti has received research support from the National Institutes of Health (NIH).

Dr. Newport has received research support from Eli Lilly, Glaxo-SmithKline, Janssen, and Wyeth as well as NARSAD and NIH; served on advisory boards for GlaxoSmithKline; and served on speaker's bureaus and/or received speaker's honoraria from Astra-Zeneca, Eli Lilly, GlaxoSmithKline, and Pfizer.

Dr. Pennell has received research support from GlaxoSmith-Kline, UCB Pharma, NIH, and Centers for Disease Control and Prevention. She has served on the expert panel for the Keppra Pregnancy Registry.

Ms. Newman has received research support from GlaxoSmith-Kline, Marinus Pharmaceuticals, NIH, and UCB Pharma.

Ms. Titchner has received research support from NIH.

Ms. Knight has received research support from Bristol-Myers Squibb, Cyberonics, Eli Lilly, Forest, Janssen, NIH, Novartis, and Wyeth.

Dr. Stowe has received research support from GlaxoSmithKline, NIH, and Wyeth; served on advisory boards for Wyeth, Bristol-Myers Squibb, and GlaxoSmithKline; and received speakers' honoraria from Eli Lilly, GlaxoSmithKline, Pfizer, and Wyeth.

1. Introduction

Psychiatric comorbidity is common in patients with epilepsy, with 46.1% experiencing at least one comorbid psychiatric condition [1]. Furthermore, patients with epilepsy report depressive symptoms at much higher frequencies than those without epilepsy [2–9] and are four times more likely to be hospitalized for major depressive disorder (MDD) [10].

Pregnancy and the postpartum period may be windows of increased vulnerability to MDD, and this susceptibility may be further inflated in women with epilepsy. In the general population, 7–13% of all pregnant women experience major depressive episodes [4], and in the postpartum period the rate rises to 10–15% [11]. A recent investigation of 35 women with epilepsy and 35 matched controls demonstrated higher rates of postpartum depression (PPD) in those with epilepsy (29%) compared with those without epilepsy (11%) [12]. In this investigation, the severity of postpartum depressive symptoms was not predicted by type of epilepsy, seizure frequency, or monotherapy versus polytherapy antiepileptic drug (AED) regimen.

Previous investigations in non-postpartum patients have indicated that AED treatment reduces both seizure frequency and severity of psychological symptoms [13–15]. Recognizing that numerous AEDs are used in the treatment of mood disorders, it is not surprising that therapy with certain AEDs might improve mood disturbance in some patients with epilepsy. For example, valproate and carbamazepine are not only effective in seizure management, but are also used to manage manic and mixed episodes associated with bipolar disorder [16]. However, lamotrigine (LTG) is the only AED shown to provide prophylaxis against depression in those with bipolar disorder [17]. Several investigations indicate that patients with epilepsy treated with LTG experience greater improvement in mood than those treated with other AEDs [18–23].

The current investigation is a natural extension of these existing studies regarding the risk for depressive illness in association with epilepsy and AED therapy, assessing the risk for PPD in women with epilepsy as well as the potential efficacy of continuous AED treatment in the prophylaxis against PPD. In particular, we sought to confirm and extend results of the previous cross-sectional investigation of PPD in women with epilepsy [12] with a prospective sample using serial longitudinal monitoring of mood, seizure activity, and pharmacotherapy, determining risk factors for PPD as well as the impact of LTG, in particular, on the risk for PPD symptoms.

Our *a priori* hypothesis is that women with epilepsy would be at greatest risk for depression following childbirth if they have (1) significant psychosocial stressors (e.g., unplanned pregnancy, unmarried, obstetrical complications); (2) a history of depressive illness prior to conception, and (3) poor or difficult seizure management during the postpartum period. Furthermore, we hypothesize that women treated with LTG are less likely to experience PPD.

2. Methods

Women presenting to the Emory Women's Epilepsy Program (WEP), a tertiary referral center specializing in the management of perinatal epilepsy, were enrolled during pregnancy or preconception and followed longitudinally in a prospective observational study of the perinatal course of neuropsychiatric illness and the pharmacokinetics of neuropsychiatric medications during pregnancy and lactation (SCOR P50 MH-68036). Details of this study have been previously described [24]. Briefly, at study entry, baseline demographic (race, marital status, age, etc.) and clinical (pregnancy planning, gravidity, type of epilepsy, psychiatric history) information was recorded. At serial follow-up visits across pregnancy

and the postpartum period, subjects completed the self-rated Beck Depression Inventory (BDI), a diary of between-visit interval seizure activity, and a clinician-administered week-by-week determination of exposure to medications and other substances. Inclusion criteria for the present analysis included a psychiatric diagnostic assessment by a board-certified psychiatrist (D.J.N., Z.N.S.) and completion of the BDI during the final trimester of pregnancy and the first 12 weeks postpartum. Written informed consent was obtained prior to study enrollment. The study was approved by the Emory University institutional review board.

The BDI is a 21-item self-rated depression scale widely used to screen for depression in community samples and medical populations [25]. By convention, the presence of clinically significant depressive symptoms was defined as a BDI score ≥ 12 , based on data from the Emory Women's Mental Health Program comparing depression rating scales with the Structured Clinical Interview for Diagnosis (SCID) Mood Module criteria for an episode of major depression (unpublished observation).

The data analysis was conducted in three stages. First, a descriptive analysis of the study subjects was performed. In the second stage of the analysis, univariate tests (χ^2 , t test) were conducted to assess the association of various putative predictors with the occurrence of PPD. Finally, multivariate logistic regression modeling was performed in the third stage of analysis to complete the assessment of PPD predictors. Those predictors found to have a significant association ($P \leq 0.10$) with PPD in the univariate analyses were included as candidate predictors in the initial multivariate logistic model. Backward elimination was performed to determine the final reduced logistic model.

3. Results

A total of 121 women with epilepsy were enrolled in the primary investigation of the pharmacokinetics of AEDs in pregnancy. Fifty-six pregnant women with epilepsy (46% of the available sample) completed all components necessary for inclusion in the current analysis. The mean age of the participants was 27.9 ± 5.8 years. The racial composition of the sample was 64.2% ($n = 36$) white/Caucasian, 25.0% ($n = 14$) black/African-American, and 10.7% ($n = 6$) other. The mean level of education was 14.9 ± 2.5 years. Thirty-seven (66.1%) of the participants were married at conception.

With respect to obstetrical history, 49.1% ($n = 27$) of the participants were primigravidas, and 65.5% ($n = 36$) were primipas. The current pregnancy was unplanned for 35.2% ($n = 19$, missing data $n = 2$) of the participants.

Pharmacological treatment data for the first 12 postpartum weeks were available for all 56 participants, identifying 78.6% ($n = 44$) patients taking only one AED, and 21.4% ($n = 12$) on two AEDs. The following are the frequencies for AED use: lamotrigine, 58.9% ($n = 33$); levetiracetam, 14.3% ($n = 8$); topiramate, 14.3% ($n = 8$); carbamazepine, 8.9% ($n = 5$); divalproex, 8.9% ($n = 5$); phenytoin, 8.9% ($n = 5$); oxcarbazepine, 5.4% ($n = 3$); and zonisamide, 1.8% ($n = 1$). Three (5.4%) women were taking antidepressants at the time of delivery.

The types of epilepsy represented among the participant group included localization-related epilepsy ($n = 35$, 62.5%), juvenile myoclonic epilepsy ($n = 10$, 17.9%), primary generalized epilepsy—unspecified ($n = 7$, 12.5%), and epilepsy unclear whether focal or generalized ($n = 4$, 7.1%).

Thirty-nine (69.6%) participants were seizure free during the 12-week postpartum follow-up period. Among those who did experience seizure activity during the postpartum follow-up,

the number of seizures ranged from 1 to 105 (mean: 19.6 ± 26.1) over the 12-week period. Only 4 (7.1%) participants experienced a tonic-clonic seizure during the first 12 weeks of the postpartum period. During pregnancy, 27 (48.2%) participants experienced one or more seizures. Six (10.7%) participants experienced a tonic-clonic seizure during pregnancy.

PPD (defined as a BDI score ≥ 12) was endorsed by 14 (25.0%) of the 56 participants. Univariate analyses of demographic/obstetrical (cf. Table 1) predictors revealed that multiparous women ($P < 0.03$) and those who experienced tonic-clonic seizures during the postpartum period ($P < 0.05$) were more likely to experience PPD. In addition, there were statistical trends ($0.05 < P < 0.10$) to suggest that PPD was more common among those who (1) were ≥ 35 years old ($P < 0.06$), (2) were receiving AED polytherapy during the postpartum period ($P < 0.06$), and (3) experienced seizures of any type during the first 12 weeks after delivery ($P < 0.10$).

The univariate analysis provided no evidence to indicate that LTG afforded greater protection from PPD than other AEDs. Similarly, there was no evidence that any of the other AEDs (data not shown) were associated with the incidence of PPD. Contrary to our hypothesis, PPD rates appeared higher in women treated with LTG than in those taking other AEDs (30.3% vs 17.4%, $P < 0.36$), and were consistently higher whether they had a prior lifetime history of MDD (44.4% vs 28.9%, $P < .64$) or not (15.8% vs 8.3%, $P = 1.00$), though none of these differences were statistically significant. There was no evidence to indicate that a past history of MDD factored into the selection of LTG for perinatal seizure management. Whereas 56.3% (9/16) of the participants with a lifetime history of MDD were treated with LTG, 63.6% (21/33) of those with no history of MDD were treated with LTG.

Logistic regression was performed incorporating those predictors that demonstrated evidence of an association with PPD into the univariate analyses (i.e., age, parity, PP seizure activity, AED polytherapy) as candidate predictors in the initial model. In addition, recognizing their historical associations with altered risk of recurrence of depression, LTG therapy and lifetime history of MDD were included in the initial model. The reduced model identified multiparity (OR = 12.5 [95% CI: 1.9–82.7]) and AED polytherapy (OR = 9.3 [95% CI: 1.5–58.0]) as significant predictors of PPD. The log likelihood of the main effects model was 52.19 ($\chi^2(2) = 16.55$, $P < 0.0003$). The Hosmer and Lemeshow goodness-of-fit statistic was 0.16 ($df = 2$, $P < 0.93$) indicating that the logistic model for PPD fit the data adequately.

4. Discussion

The rate of PPD in this sample of women with epilepsy was 25.0%. Although this rate approximates the 29% rate reported by Turner and colleagues [12], it is important to note that the earlier study did not include women with a history of MDD. In the present study, the rate of PPD among subjects without a MDD history was 15.2%. Several factors may account for the nearly twofold higher PPD rate (among women with epilepsy without a prior history of MDD) in the Turner study in comparison to the present investigation: (1) different depression rating scales were used (the earlier study used the Edinburgh Postnatal Depression Scale); (2) the prospective longitudinal design of the current study resulted in more office contacts (a pharmacological PPD prophylaxis trial indicated a potential benefit from contact during pregnancy) [26]; and (3) AED treatment was objectively assessed via repeated laboratory assay during the perinatal period and the maternal daily dose was adjusted for optimal seizure control in the current study.

With respect to predictors of PPD in women with epilepsy, the greatest effect in the present study was seen for multiparity (OR = 12.5). Mayberry et al. demonstrated a similar finding

relating to higher rates of PPD in multiparous women [27]. The multiparity finding in the present study is likely related to the trend for a higher PPD rate among women with advanced maternal age (≥ 35 years old at delivery). Specifically, older women are more likely to have had multiple pregnancies (71.4% vs 30.6%, $P < 0.04$ in the current study). Consequently, a high PPD rate in one factor seems to predict a high rate in the other.

AED polytherapy (OR = 9.3) was also significantly associated with PPD. This was not observed in the previous study of epilepsy and PPD [12], although others have postulated an association between complex AED regimens and nonpuerperal depression in patients with epilepsy. The statistical association of PPD with postpartum tonic-clonic seizure activity and the statistical trend associating PPD with any postpartum seizure activity may be due in part to an interaction with AED polytherapy. Women receiving AED polytherapy were not more likely to experience seizure activity in the postpartum period (33.3% vs 29.6%, $P = 1.00$); however, those with postpartum seizure activity were more likely to develop PPD if they were receiving AED polytherapy rather than monotherapy (100.0% vs 23.1%, $P < 0.02$). This polytherapy versus monotherapy finding was not present for those who were seizure free during the postpartum period (25.0% vs 16.1%, $P < 0.62$). Similarly, all three of the participants who experienced a postpartum tonic-clonic seizure despite AED polytherapy also experienced PPD; however, the one participant who experienced a postpartum tonic-clonic seizure while receiving AED monotherapy was not depressed during the postpartum period. Overall, these findings indicate that treatment-resistant epilepsy (as evidenced by active seizure activity despite AED polytherapy) carries a higher risk for PPD.

Contrary to our hypotheses, there was no statistical evidence of an association between the occurrence of PPD and (1) a lifetime history of MDD, (2) unplanned pregnancy, or (3) LTG therapy throughout the perinatal period. Despite the lack of a statistically significant association between PPD and lifetime history of MDD in the current study, the pattern of results was consistent with previous investigations in women without epilepsy [28] demonstrating a higher rate of PPD among those with a history of MDD (37.5%) than among those with no prior history of depression (15.2%).

Neither was unplanned pregnancy a significant predictor of PPD in the current study. Warner et al. [29] reported that women with unplanned pregnancies have a significantly higher PPD rate (38.0%), and one would expect to see a similar rate in our sample of women with epilepsy. The reasons for this discrepancy are unclear, though again, it is possible that the clinical support provided during the course of participation in this longitudinal observational study accommodated (at least in part) for the deleterious consequences of psychosocial stressors such as unplanned pregnancy.

Despite previous findings on the potential benefits of LTG for mood disturbance and depression in patients with epilepsy [18–23], LTG was not superior to other AEDs in reducing the risk for PPD. Although prescription bias could play a role, that is, patients with epilepsy and depressive symptoms may be preferentially prescribed LTG over alternative agents, this was not a contributing factor in the current study as those with a prior history of MDD were not more likely to have been treated with LTG. During the window of enrollment for this study, LTG was a recommended first-line treatment option for women with epilepsy during childbearing years. As such, a large proportion of the study patients were treated with LTG with a view to maximizing AED reproductive safety rather than any effort to manage psychiatric comorbidity with AED selection.

There are several potential limitations in generalizing the current results to a community-derived sample of women with epilepsy, including: (1) all subjects included in the analyses

were compliant with participation in a high-burden clinical research study of the pharmacokinetics of medications in pregnancy; (2) treatment was monitored by clinicians highly experienced in the management of neuropsychiatric illnesses during the perinatal period; (3) there was ongoing laboratory assessment of AED concentrations, thyroid function, and drugs of abuse. Although not specifically addressed in the current study, the contribution of other psychosocial factors, such as socioeconomic status and social support, is potentially relevant in expanding these data to other populations of women with epilepsy.

Despite these potential limitations, the current study is consistent with previous research demonstrating a high rate of PPD in women. The findings of this study should alert clinicians (neurologists, obstetricians, primary care physicians) treating women with epilepsy during pregnancy and the postpartum period that they should screen their patients for PPD, especially patients requiring AED polytherapy for treatment-resistant epilepsy.

Acknowledgments

This work was supported by NIH Specialized Center of Research MH-68036 (to Z.N.S.).

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Table 1

Univariate analyses of demographic and clinical predictors of postpartum depression in women with epilepsy ($N = 56$).

Predictor	BDI 12 depressed ($n = 14$)	BDI 11 not depressed ($n = 42$)	Fisher's exact χ^2
<i>Demographic</i>			
Age			$P < 0.06$
35 years	4/7 (57.1%)	3/7 (42.9%)	
<35 years	10/49 (20.4%)	39/49 (79.6%)	
Race			$\chi^2 = 2.86, P < 0.24$
White/Caucasian	9/36 (25.0%)	27/36 (75.0%)	
Black/African-American	2/14 (14.3%)	12/14 (85.7%)	
Other race	3/6 (50.0%)	3/6 (50.0%)	
Education			$P < 0.31$
No college	2/16 (12.5%)	14/16 (87.5%)	
Attended college	11/38 (28.9%)	27/38 (71.1%)	
Marital status			$P < 0.34$
Unmarried	3/19 (15.8%)	16/19 (84.2%)	
Married	11/37 (29.8%)	26/37 (70.3%)	
<i>Obstetrical</i>			
Parity			$P < 0.03$
Multipara	9/20 (45.0%)	11/20 (55.0%)	
Primipara	5/36 (13.9%)	31/36 (86.1%)	
Pregnancy planned?			$P = 1.00$
No	4/19 (21.1%)	15/19 (78.9%)	
Yes	9/35 (25.7%)	26/35 (74.3%)	
Delivery			$P < 0.35$
Preterm (<37 weeks)	3/7 (42.9%)	4/7 (57.1%)	
Term (≥ 37 weeks)	11/49 (22.4%)	38/49 (77.6%)	
<i>Psychiatric</i>			
Lifetime history of major depressive disorder			$P < 0.15$
Yes	6/16 (37.5%)	10/16 (62.5%)	
No	5/33 (15.2%)	28/33 (84.8%)	
Third-trimester depression			$P < 0.33$
Yes	3/6 (50.0%)	3/6 (50.0%)	
No	9/37 (24.3%)	28/37 (75.7%)	
Antidepressant at delivery			$P < 0.16$
Yes	2/3 (66.7%)	1/3 (33.3%)	
No	12/53 (22.6%)	41/53 (77.4%)	
<i>Neurological</i>			
Type of epilepsy			$\chi^2 = 34.3, P < 0.34$
Localization-related	7/35 (20.0%)	28/35 (80.0%)	
Juvenile myoclonic	4/10 (40.0%)	6/10 (60.0%)	

Predictor	BDI 12 depressed (<i>n</i> = 14)	BDI 11 not depressed (<i>n</i> = 42)	Fisher's exact χ^2
Primary generalized—unspecified	1/7 (14.3%)	6/7 (85.7%)	<i>P</i> < 0.06
Unclear if focal or generalized	2/2 (50.0%)	2/2 (50.0%)	
AED therapy			
Polytherapy	6/12 (50.0%)	6/12 (50.0%)	<i>P</i> < 0.36
Monotherapy	8/44 (18.2%)	36/44 (81.8%)	
AED used			
Lamotrigine	10/33 (30.3%)	23/33 (69.7%)	<i>P</i> < 0.10
Other	4/23 (17.4%)	19/23 (82.6%)	
Any seizures in postpartum period			
Yes	7/17 (41.2%)	10/17 (58.8%)	<i>P</i> < 0.05
No	7/39 (17.9%)	32/39 (82.1%)	
Any tonic-clonic seizures in postpartum period			
Yes	3/4 (75.0%)	1/4 (25.0%)	<i>P</i> < 0.55
No	11/52 (21.2%)	41/52 (78.8%)	
Any seizures during pregnancy			
Yes	8/27 (29.6%)	19/27 (70.4%)	<i>P</i> < 0.64
No	6/29 (20.7%)	23/29 (79.3%)	
Any tonic-clonic seizures during pregnancy			
Yes	2/6 (33.3%)	4/6 (66.7%)	
No	12/50 (24.0%)	38/50 (76.0%)	