Research Square

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Perinatal depression symptoms impact on the Computerized Cardiotocography parameters: a prospective study

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Research Article

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Abstract

Objective: The present study aims to investigate the possible computerized cardiotocography (cCTG) parameters alteration in patients with a high risk of Perinatal depression (PND), evaluated in the third trimester of pregnancy through the Edinburgh Postnatal Depression Scale (EPDS).

Methods: We conducted a prospective observational study from 1st august 2024 to 20th august 2025. All pregnant women (after 30 gestational weeks) were included and performed cCTG and EPDS evaluation. On EPDS score, pregnant were divided into low-risk (score <10) or high-risk (score ≥10) groups. For all included pregnant demographic characteristics and cCTG parameters were recorded. The baseline characteristics and cCTG parameters were compared between groups.

Results: 205 Pregnant were enrolled, and no statistical differences in demographic, obstetric, or fetal characteristics were found between the groups. There were no significant differences between high- and low-EPDS groups for any cCTG parameters including baseline fetal heart rate (p=0.39), short term variability (p=0.56), long-term variability (p=0.39), accelerations (p=0.69), and fetal movements (p=0.41). The correlation analyses showed that there was no linear association of EPDS scores to cCTG indices.

Conclusions: Maternal depressive symptoms, in late gestation, did not impact cCTG-derived parameters. These results indicated that cCTG fetal surveillance was effective for pregnancies complicated by maternal depressive symptoms and suggested that maternal psychological symptoms and fetal monitoring are distinct, yet complementary aspects of perinatal care.

Introduction

Perinatal period is a critical period for mental diseases, especially for perinatal depression (PND). Diagnostic and Statistical Manual of Mental Disorders-5th edition (DSM-5) defined the PND as the manifestation of a major depressive episode during peripartum period [1]. PND differs from common maternity blues, which can cause crying and irritability in 70% of mothers and resolve spontaneously within two weeks [2]. The post-partum depression ranges around 12–16% of women [3]. These represent a conservative estimation, with a rate of underestimated or underdiagnosed condition [4]. To screen PND, Edinburgh Postnatal Depression Scale (EPDS) was adopted [5, 6]. EPDS consists of 10 items which evaluate the mental state of the patient. All the answers correspond to a score, and the total score indicates if a psychiatric evaluation is necessary [6]. The pathogenesis of PND is multifactorial and not yet fully understood and involves environmental as well as biological factors [7, 8]. Previous studies showed a link between PND and lower educational level, poor economic status of families, history of mental illness, domestic violence, perinatal smoking or drinking, and multiparity [9]. Considering the biological factors, such as vitamin D, Brain-derived neurotrophic factor (BDNF) and IL-6, were associated with an increased risk of PND [10, 11]. In the postpartum period, maternal depression represents a risk factor for the child, including behavioral problems and cognitive development disorders [12]. Even in the prenatal period, maternal stress, anxiety, and depression affect the development of the offspring with a significant risk of psychopathology [13]. Several studies investigated the fetal well-being using

cardiotocography (CTG) [14]. CTG recorded the fetal heart rate (FHR) and the uterine contractile activity and is used for the antenatal and intrapartum fetal well-being surveillance [14, 15]. To reduce the intra and inter-operator CTG analysis, computerized cardiotocography (cCTG) was introduced [16]. This instrument gave an objective analysis of the FHR pattern and added specific analysis, as short term variation (STV) necessary to the fetal growth restriction (FGR) management [17]. Maternal stress caused alterations in cortisol and catecholamine status [18]. This effect could impact FHR variability, accelerations, and cCTG pattern [19]. In this context, maternal psychological state could impact the cCTG variables and the biological plausibility for this link is supported by data. No prospective studies have specifically focused on antepartum cCTG pattern in the late third trimester and its possible association with maternal psychosocial status, evaluated with the EPDS scores. Our study aimed to investigate this gap and to explore cCTG variables differences between the high risk PND pregnant versus the low risk PND.

Material and Methods

A prospective observational study carried out at the University of Campania “Luigi Vanvitelli”, Naples, Italy, from 1st August 2024 to 20 august 2025. Maternal psychological condition assessed by the EPDS and cCTG in the late pregnancy were studied. We followed STROBE Statement to report observational studies [20]. All consecutive pregnant women who performed outpatient cCTG to our hospital were enrolled after a written and oral consent. Eligibility criteria were single pregnancy, gestational age ≥ 30 weeks. Exclusion criteria: multiple pregnancies, structural or aneuploidy fetal malformations, previous psychiatric disorders receiving medical treatment and use of drugs with reported impact on FHR variability such as β-blocker. Maternal psychological distress was measured with the EPDS. The EPDS was completed before the cCTG test in a silent, private room. EPDS cutoff score was ≥ 10 and indicate a high risk of depressive symptoms. Fetal evaluation was carried out with a Sonicaid Team 3 (Huntleigh Healthcare Ltd, Cardiff, United Kingdom). Each cCTG duration was not less than 20 min with a semi-reclining position. All the cCTG parameters were recorded: FHR-baseline, number of accelerations and decelerations, fetal movements per hour (FM/h), high and low episodes, short-term variability (STV), long-term variability (LTV), cCTG duration and signal loss. The main objective was to study the fetal heart rate patterns obtained through computerized CTG in the high-risk PND group.

Continuous variables were reported as median and interquartile ranges (IQRs). Categorical variables are reported as percentages. Inter-group analysis (EPDS category < 10 versus EPDS ≥ 10) were performed with Wilcoxon rank sum test for continuous variables and Fisher's exact test for categorical variables

To measure the linear association between continuous variables, Pearson’s correlation test was used if variables had a normal distribution. Otherwise, Spearman’s rank correlation test was used. P-values < 0.05 were considered statistically significant. All the statistical analyses were performed with R Studio Statistical software version 4.1.3. Sample size estimation was based on a significance test for Pearson’s correlation between EPDS scale and continuous cCTG parameters, assuming a two-tailed alpha level of 0.05 and a statistical power of 80%. With a total sample size of at least of 194 participants, the study is

powered to detect a minimum effect size of |r|0.20, which corresponds to a small-to-moderate effect according to Cohen's conventions. The study was conducted in accordance with the ethical guidelines of the Declaration of Helsinki and was approved by the Ethics Committee of University of Campania "Luigi Vanvitelli" (protocol number 98 of February 28, 2019).

Results

205 pregnant women were included and subsequently we divided the patients into two groups based on EPDS scores: the first group (147) with an EPDS < 10 and the second (58) with EPDS ≥ 10. Demographic and clinical features between the two groups were comparable (Table 1 and Table 2). Maternal age (median 31.0 years, IQR, 28.00–35.00, versus 30.0 years, IQR, 26.25–34.00, p value 0.39), gestational age in assessment (38.14 weeks, 37.29–39.00, versus 38.50 weeks, 37.29–39.25, p value 0.55), pre-pregnancy BMI (24.39, 21.56–28.37, versus 24.71, 22.07–29.14, p value 0.38), or maternal weight (73.0 kg, 68.0–87.0 versus 75.0 kg, 70.0–90.0, p-value 0.18). Distribution of rates of pregnancy by ethnicity, mode of conception, fetal sex, smoking and gestational weight gain did not significantly differ among the EPDS strata (all p > 0.05). Analyzing the cCTG parameters in the two groups, EPDS < 10 and EPDS ≥ 10 (Table 3), no one statistical difference was found. cCTG parameters showed an average recorded time required of 40.0 min in both groups, with comparable signal loss (0.30, 0.00–1.00 versus 0.35, 0.00–0.80, p value 0.98). Fetal movement per hour showed a median of 30.0, 16.5–57.7 versus 24.7, 13.3–46.9, with a p value of 0.41), and accelerations numbers was 10.0 versus 9.5, p-value 0.69.

Table 1 – Baseline clinical characteristics of patients stratified for EPDS class

	EPDS		
Characteristic	**< 10**, N = 147[1]	**≥ 10**, N = 58[1]	**Overall**, N = 205
Ethnicity			
Caucasian	124.0 (84.4%)	51.0 (87.9%)	175.0 (85.4%)
Other	23.0 (15.6%)	7.0 (12.1%)	30.0 (14.6%)
Age	31.00 (28.00, 35.00)	30.00 (26.25, 34.00)	31.00 (27.00, 35.0
Gestational age	38.14 (37.29, 39.00)	38.50 (37.29, 39.25)	38.14 (37.29, 39.1
Onset pregnancy			
IVF	6.0 (4.1%)	0.0 (0.0%)	6.0 (2.9%)
Spontaneous	141.0 (95.9%)	58.0 (100.0%)	199.0 (97.1%)
Maternal weight	73.00 (68.00, 87.00)	75.00 (70.00, 90.00)	74.00 (68.00, 88.0
Pre-Pregnancy BMI	24.39 (21.56, 28.37)	24.71 (22.07, 29.14)	24.44 (21.64, 28.6
Weight gain	10.00 (8.00, 13.00)	11.00 (9.00, 14.00)	10.00 (8.00, 13.0
Fetal sex			
Female	75.0 (51.0%)	27.0 (46.6%)	102.0 (49.8%)
Male	68.0 (46.3%)	28.0 (48.3%)	96.0 (46.8%)
Unknown	4.0 (2.7%)	3.0 (5.2%)	7.0 (3.4%)
Smoker			
No	132.0 (89.8%)	49.0 (84.5%)	181.0 (88.3%)
Yes	15.0 (10.2%)	9.0 (15.5%)	24.0 (11.7%)
Comorbidities[2]			
GDM	21 (14.2%)	9 (15.5%)	30 (14.6%)
Pre-GDM	4 (2.7%)	0 (0.0%)	4 (1.9%)
GH	4 (2.7%)	1 (1.7%)	5 (2.4%)
Pre-GH	2 (1.3%)	1 (1.7%)	3 (1.4%)
FGR	2 (1.3%)	2 (3.4%)	4 (1.9%)

[1]Median (IQR) or Frequency (%); [2]Total number and (Frequency, %); GDM, gestational diabetes; GH, Gestational hypertension; FGR, fetal growth restriction;

Also, the numbers of decelerations were similar, p value 0.87, high or low episodes of variability with a p value 0.21 and 0.76, respectively. LTV (46.0 versus 48.6, p-value 0.39), or STV (8.7 versus 9.3, p value 0.56). Figure 1 shows the scatterplot matrix about the EPDS score and the cCTG parameters.

All the variables were evaluated including FHR-baseline, number of accelerations and decelerations, FM/h, high and low episodes, STV, LTV, cCTG duration and signal loss. No statistical linear association emerges between EPDS scores and the cCTG parameters (Fig. 1).

Table 2 – Baseline clinical characteristics of patients stratified for EPDS class with statistical association testing

	EPDS		
Characteristic	**< 10**, N = 147[1]	**≥ 10**, N = 58[1]	**p-value**[2]
Ethnicity			0.66
Caucasian	124.0 (84.4%)	51.0 (87.9%)	
Other	23.0 (15.6%)	7.0 (12.1%)	
Age	31.00 (28.00, 35.00)	30.00 (26.25, 34.00)	0.39
Gestational age	38.14 (37.29, 39.00)	38.50 (37.29, 39.25)	0.55
Onset pregnancy			0.19
IVF	6.0 (4.1%)	0.0 (0.0%)	
Spontaneous	141.0 (95.9%)	58.0 (100.0%)	
Maternal weight	73.00 (68.00, 87.00)	75.00 (70.00, 90.00)	0.18
Pre-pregnancy BMI	24.39 (21.56, 28.37)	24.71 (22.07, 29.14)	0.38
Weight gain	10.00 (8.00, 13.00)	11.00 (9.00, 14.00)	0.45
Fetal sex			0.56
Female	75.0 (51.0%)	27.0 (46.6%)	
Male	68.0 (46.3%)	28.0 (48.3%)	
Unknown	4.0 (2.7%)	3.0 (5.2%)	
Smoker			0.34
No	132.0 (89.8%)	49.0 (84.5%)	
Yes	15.0 (10.2%)	9.0 (15.5%)	
Comorbidities[2]			
GDM	21 (14.2%)	9 (15.5%)	0.82
Pre-GDM	4 (2.7%)	0 (0.0%)	0.20
GH	4 (2.7%)	1 (1.7%)	0.67
Pre-GH	2 (1.3%)	1 (1.7%)	0.67
FGR	2 (1.3%)	2 (3.4%)	0.33

[1]Median (IQR) or Frequency (%)

[2]Fisher's exact test; Wilcoxon rank sum test

Also, the numbers of decelerations were similar, p value 0.87, high or low episodes of variability with a p value 0.21 and 0.76, respectively. LTV (46.0 versus 48.6, p-value 0.39), or STV (8.7 versus 9.3, p value 0.56). Figure 1 shows the scatterplot matrix about the EPDS score and the cCTG parameters.

Table 3 – cCTG parameter of patients stratified for EPDS class with statistical association testing

	EPDS		
Characteristic	**< 10**, N = 147[1]	**≥ 10**, N = 58[1]	**p-value**[2]
Duration	40.00 (32.00, 60.00)	40.00 (33.25, 53.00)	0.96
Signal loss	0.30 (0.00, 1.00)	0.35 (0.00, 0.80)	0.98
MAF per hour	30.00 (16.45, 57.70)	24.65 (13.32, 46.92)	0.41
FHR,baseline	137.00 (130.00, 144.00)	136.50 (129.25, 143.00)	0.65
Accelerations	10.00 (7.00, 13.00)	9.50 (8.00, 13.50)	0.69
Decelerations	0.00 (0.00, 1.00)	0.50 (0.00, 1.00)	0.87
High episodes	20.00 (14.00, 27.50)	24.00 (14.25, 30.75)	0.21
Low episodes	0.00 (0.00, 10.50)	0.00 (0.00, 9.00)	0.76
LTV	46.00 (39.15, 56.85)	48.60 (39.15, 57.95)	0.39
STV	8.70 (7.50, 10.88)	9.30 (7.53, 10.78)	0.56

[1]Median (IQR) or Frequency (%)

[2]Wilcoxon rank sum test; Fisher's exact test

All the variables were evaluated including FHR-baseline, number of accelerations and decelerations, FM/h, high and low episodes, STV, LTV, cCTG duration and signal loss. No statistical linear association emerges between EPDS scores and the cCTG parameters (Figure 1).

Discussion

Our prospective study did not evidence statistical cCTG difference, such as fetal heart rate baseline, accelerations, decelerations, fetal movements, short- and long-term variability, or signal loss between high and low risk for post-partum depression, evaluated with EPDS scores of ≥ 10 in late pregnancy. This finding indicates that, in late gestation, the maternal depressive symptom did not impact the fetal heart rate pattern evaluated by cCTG. Different studies try to link maternal psychological distress and fetal autonomic regulation. Maternal depression and stress could dysregulate the hypothalamic–pituitary–adrenal axis, with an increased secretion of cortisol, and changes in the sympathetic–parasympathetic balance, all these mechanisms could impact fetal heart regulation and fetal neurodevelopment [21]. Different experimental and observational studies explored the prenatal depressive symptoms and their association with decreased FHR variability, supporting the idea that maternal affective alteration may reflect intrauterine fetal autonomic regulation [22]. In addition, fetal heart rate variability appeared related to fetal neurobehavioral development and postnatal heart self-regulation [23]. The lack of differences in computerized cardiotocography parameters evidenced in our cohort appeared discordant with this

evidence. However, methodological and conceptual differences are present. The FHR correlation with maternal depressive symptoms, was explored through fetal external stimuli [22]. These studies typically investigated beat-to-beat heart rate variability, with rapid autonomic modification, more sensitive to subtle alterations than average parameters obtained from cCTG [23]. In comparison, cCTG parameters reduce the intra-operator and intra-operator difference and permitted a clinical evaluation of the fetal well-being [24]. In addition, we evaluated the FHR in the third trimester of pregnancy, a period with fetal late autonomic maturation and greater stability of FHR [25]. At this stage of pregnancy, fetal adaptive and compensatory mechanisms improved and could mitigate the maternal psychological distress impact. Severity and clinical depressive symptoms also need consideration. In our cohort, PND risk was evaluated using EPDS screening, without a clinical diagnostic interview. EPDS quantify the PND risk and capture a wide range of depressive symptom severity [26]. Maternal abnormalities in autonomic states, especially maternal heart variability, were already explored in PND conditions [27]. Different studies investigated maternal heart rate variability associations with postpartum depressive and anxiety symptoms [28]. A scoping review highlights considerable heterogeneity within the studies investigating the association between perinatal depression and heart rate variability, varying in the timing, measurement procedures, and population characteristics, which limited synthesis and interpretation of findings [27]. All these articles did not explore the maternal impact on the clinical evaluation of the FHR. cCTG is applied for objective evaluation of fetal well-being, particularly for fetus with fetal growth restriction (FGR) [29]. Our findings imply that maternal depressive symptomatology did not impact the interpretation of antepartum cCTG. This finding is comforting for clinicians' management of pregnancies with PND, as it reinforces the validity of standard fetal surveillance in these conditions. The absence of detectable FHR alteration through cCTG analysis supports no different antenatal surveillance for perinatal depression. The literature shows that PND is linked to different maternal, neonatal, and children's outcomes, including the reduction in mother–infant interactions and recent research suggested an integrated psychological, neuroendocrine, and physiological factors to fully grasp the influence on maternal–fetal relationships [31, 32]. Clinically, cCTG was not impacted by maternal depressive symptoms. Although screening for PND is necessary to establish links with maternal morbidity, adverse obstetric outcomes and long-term offspring development [33]. These results reinforce the idea that maternal psychological well-being and fetal surveillance during pregnancy require separate management and that they are complementary but distinct elements of perinatal care. Our study presents several strong aspects. Firstly, the study the study design. Second, EPDS represents a validated and internationally accepted screening [34]. Third, FHR was evaluated through cCTG parameters, representing an innovative and objective analysis. This study presents also several limitations that should be considered. First, after the EPDS evaluation no data is present from the psychiatric interview. As a result, a sub analysis of the post-partum depression cases was lacking. Second, EPDS scores relate to symptoms present at a single point in the patient's clinical history and do not assess all pregnancy duration or variations during pregnancy [35]. Third, cCTG may not be sensitive enough to detect small changes in fetal reactivity, which have been demonstrated in experimental studies using vibroacoustic or vocal stimulation [36]. At least no cortisol levels or other biochemical markers were evaluated, factors

potentially involved in these conditions. Future studies that combine psychological assessments with biological stress markers and longitudinal maternal follow-up will be performed to improve our findings

Conclusions

In summary, our prospective study shows that maternal depressive symptoms measured with EPDS are not associated with computerized cardiotocography parameters changes. The null results are strong and clinically meaningful and highlight the complexity between maternal psychological health and fetal autonomic regulation. Additional studies are necessary to confirm this data also in patients with a diagnosis of post-partum depression.

Declarations

Author Contribution

Conceptualization, M.L.V.; methodology, L.P., C.B. and D.P.; validation, M.T. and M.L.; formal analysis, M.F.; investigation and data curation, R.M. and A.C.; writing—original draft preparation, F.C. and M.L.V.; writing—review and editing, M.L.V.; visualization, D.R.B..; supervision, M.T. and M.L..; All authors have read and agreed to the published version of the manuscript. All authors read and approved the final manuscript.

Data Availability

Data are not publicly available due to privacy or ethical restrictions. Data will be made available on reasonable request. Further enquiries can be directed to the corresponding author.

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Figures

Figure 1 – Scatter-plot matrix of EPDS scale and continuous cCTG parameters with statistical association testing

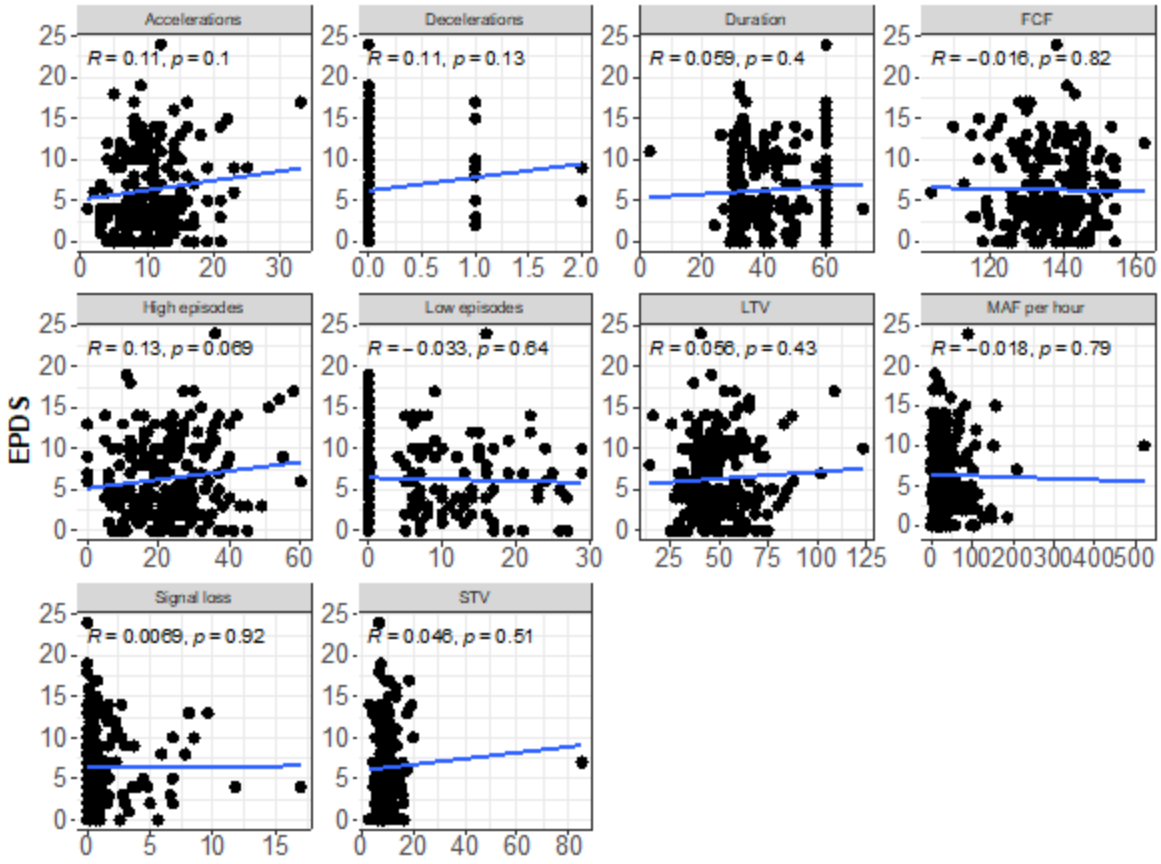


Figure 1

See image above for figure legend.