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Original Article

Placental Changes and Perinatal Outcomes among Women with Preeclampsia/Eclampsia and Normotensive Women: A Comparative Study

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Abstract

Objective: The study objective was to evaluate placental changes and the perinatal outcomes among women with preeclampsia/eclampsia and compare to normotensive pregnant women. **Materials and Methods:** This was a comparative (prospective) study, participants were 146 pregnant women; 73 preeclamptic/eclamptic (study group) and 73 normotensive (control group) at 28–40-week gestation selected by purposive sampling. The primary outcome measure was the placenta characteristics, while the secondary outcome was the perinatal outcomes. Statistical analysis was done using SPSS version 23.0, and statistical significance was set at $P \leq 0.05$. **Results:** The mean placental weight for study group was significantly lower than controls (556.82 g ± 169.72 vs. 649.93 g ± 116.38; $P \leq 0.001$); 12 (16%) placentae in the study group had gross placental infarction compared to none (0%) among controls. Study group placentae showed 11 types of microscopic placental changes compared to four among controls. Decidual vasculopathy ($P = 0.049$), incomplete vascular modeling ($P = 0.019$), accelerated villi maturity ($P = 0.049$), acute chorioamnionitis ($P = 0.048$), and microcalcifications ($P = 0.040$) were significantly associated with low APGAR scores in the study group. The 1st and 5th min APGAR scores were lower in the study group ($P \leq 0.001$, 49.3% vs. 8.2%) and ($P = 0.002$, 11% vs. 0%), respectively, while all the eight perinatal mortality recorded were in the study group. **Conclusion:** Preeclampsia/eclampsia is associated with abnormal gross and microscopic placental changes which predisposes to increased adverse perinatal outcome. Antenatal surveillance for preeclampsia/eclampsia should prioritize Doppler studies to characterize the placenta and appropriately plan the delivery.

Keywords: Clinical correlates, gross lesions, microscopic lesions, placental lesions, preeclampsia

Introduction

Hypertensive disorders of pregnancy are a leading global cause of maternal, fetal, and neonatal morbidity and mortality with a prevalence of 2%–17% in Nigeria.[1-3] Preeclampsia/eclampsia is a multiorgan spectrum of diseases characterized by onset of hypertension (systolic ≥140 mmHg and/or diastolic ≥90 mmHg) after 20-week gestation and significant proteinuria (>0.3 g per 24 h or ≥2+ proteinuria, detected by urine dipstick).[1] Eclampsia is a complication of severe preeclampsia characterized by convulsions with its attendant increased complications.[2] Theories of pathogenesis of preeclampsia include abnormal placentation and reduced placental perfusion leading to widespread dysfunction of the maternal vascular endothelium and accompanying hypertension.[4-6] This includes defective invasion of the spiral arteries by the cytotrophoblast and defective spiral artery remodeling.[7,8] Preeclampsia can range from mild to severe and the clinical presentation may range from asymptomatic mild cases to multiorgan failure.

Researchers have reported both gross as well as microscopic pathological findings in the placentae of babies born to women

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with preeclampsia/eclampsia.[9,10] However, such reports are limited in low-income countries especially sub-Saharan Africa partly due to the social, cultural, and religious beliefs associated with the placenta in these areas such that most families do not readily release the placenta for research purposes. This study aimed to evaluate placental changes and the perinatal outcomes among women with preeclampsia/eclampsia and compare to normotensive pregnant women in a low-income country.

Materials and Methods

Study setting

The study was conducted at the University of Ilorin Teaching hospital which is a tertiary health facility in North central Nigeria with an average number of 43 deliveries/week.

Study period

The study period spanned from January 1, 2018 to November 30, 2018.

Study design

The study design was a comparative study.

Study participants

Participants were pregnant women aged 18–40 years at 28–41 weeks of gestation who were admitted for delivery categorized into study group (women with preeclampsia/eclampsia) and control group (normotensive women) after matching for maternal age and gestational age.

Inclusion criteria

Pregnant women at 28–41 weeks who were admitted for delivery and delivered at the study center during the study period who consented to participate in the study.

Exclusion criteria

Women whose pregnancy ended before the 28th week or beyond the 41st week of gestation were excluded from the study.

Sample size determination

The sample size was calculated using the formula[11]

Where: N = sample size

z = standard normal deviation (a constant which is 1.96 at 95% confidence interval)

P = prevalence of preeclampsia at the study site i.e., 0.05 (5%)[12]

d = observed difference at 0.05 (5%) level of significance.

$q = 1 - P = 1–0.05 = 0.95$

$$n = \frac{1.96^2 \times 0.05 \times 0.95}{(0.05)^2} = 73$$

Thus, a minimum sample size of 73 was recruited into each arm of the study to give a total number of 146 participants.

Sampling method

Purposive nonprobability sampling was employed.

Study procedure

Participants were screened and educated about the study at the delivery, antenatal, and emergency wards of the Obstetrics and Gynaecology Department; eligible and consenting women were recruited into the study, and a written informed consent was obtained from all participants. All participants were monitored, and labor was managed with partograph with routine intrapartum management; at delivery, the birth weight, and APGAR scores were noted, blood clots were removed from the placenta, they were weighed within 1 h after delivery after examination for completeness, gross abnormalities in the umbilical cord, fetal membranes, and the placenta disc. Full thickness sections were taken from the central areas of each placenta and a section of the membranes (membrane roll) and additional sections were taken from grossly abnormal areas when present. The sections from the fresh placentae were fixed and processed for paraffin sections; staining of the sections was with H and E. Special (histochemical) stains used included Masson's Trichrome to demonstrate the presence of fibrosis, and Periodic Acid Schiff to demonstrate the basement membranes of placental/villous vessels. The mothers and their newborn received routine postpartum care as indicated for each mother–baby pair.

Primary outcome measure

The primary outcome measure was placental characteristics, i.e., placental weight, gross, and microscopic abnormalities.

Secondary outcome measure

The secondary outcome measure was the perinatal outcome.

Data management

The study data were collected using a data collection sheet designed for the study and entered into the computer. Descriptive statistics were presented as frequency, percentages, and mean. Chi-square was used to compare categorical variables. *T*-test was used to compare continuous variables and Yates corrected Chi-square (y) was used where applicable. Analysis was performed using SPPSS version 23.0 (IBM, Armonk, NY, USA) and $P < 0.05$ was termed significant at 95% confidence interval.

Ethics

Ethical approval was obtained from the ethical review committee of the University of Ilorin Teaching Hospital (Approval number: NHREC/02/05/2010. Date: January 11, 2017), and written informed consent was obtained from all participants.

Results

There were 1575 deliveries during the period of the study, 81 women had preeclampsia/eclampsia giving a prevalence of 5.14%; 25 (34%) had mild preeclampsia, 32 (44%) severe preeclampsia, and 16 (22%) eclampsia. Table 1 shows that the study group had significantly higher preterm delivery (36 vs. 20; $P = 0.029$), lower mean gestational age at delivery (37.04 ± 2.32 vs. 38.48 ± 1.28; $P \leq 0.001$),

higher caesarean delivery (30 vs. 2; $P \leq 0.001$), smaller mean placenta weight (556.82 ± 16 vs. 649.93 ± 116.38; $P \leq 0.001$), higher gross placenta infarction (12 vs. 0), low first (36 vs. 6; $P \leq 0.001$), and fifth (15 vs. 0; $P \leq 0.001$) min APGAR scores as well as neonatal mortality (8 vs. 0; $P = 0.004$) compared to control group.

Table 2 shows that 11 microscopic placental changes were identified in the placentae of the control group compared to 4 in the study group, while all the microscopic placenta changes except acute chorioamnionitis were significantly higher in the study group. Among babies born to mothers in the study group, microscopic changes such

Table 1: Obstetric and neonatal parameters

Variables	Study group, *n* (%)	Control, *n* (%)	χ^2/*t*-test	*P*
Gravidity				
Mean±SD	2.86±1.66	2.68±1.41	0.698	0.486
Range	1-8	1-7		
Parity				
Mean±SD	1.40±1.47	1.44±1.23	−0.184	0.855
Range	0-5	0-5		
Gestational age at delivery				
28-31	4 (5.5)	0	7.077[y]	0.029
32-36	32 (43.8)	20 (27.4)		
37-41	37 (50.70)	53 (72.6)		
Mean±SD	37.04±2.32	38.48±1.28	−4.640	<0.001
Mode of delivery				
SVD	43 (58.9)	71 (97.3)	31.337	<0.001
Caesarean section	30 (41.1)	2 (2.7)		
Placenta parameters				
Placenta weight	556.82±169.72	649.93±116.38	−3.866	<0.001
Gross infarction	12 (16.4)	0		
1st min APGAR				
Low (0-4)	36 (49.3)	6 (8.2)	30.082	<0.001
Normal (5-10)	37 (50.7)	67 (91.8)		
Mean±SD	5.45±2.39	7.38±0.74	−6.577	<0.001
5th min APGAR				
Poor (0-3)	8 (11)	0	12.764[y]	0.002
Abnormal (4-6)	7 (9.6)	0		
Reassuring (7-10)	58 (79.5)	73 (100.0)		
Mean±SD	7.07±2.75	8.71 ± 0.48	−5.020	<0.001
Foetal outcome				
Alive	65 (89.0)	73 (100.0)	8.464	0.004
Dead	8 (11)	0		

[y]Yates corrected Chi-square result. SVD: Spontaneous vertex delivery, SD: Standard deviation

Table 2: Presence of microscopic placental changes among study participants and the effect on 5th min APGAR scores of study group

Microscopic parameters	Study group, *n* (%)	Control group, *n* (%)	χ^2	*P*	5' APGAR score of study group			χ^2	*P*
					Poor	Abnormal	Reassuring		
Decidual vasculopathy	11 (15.1)	0	11.896	0.001	4 (36.4)	1 (9.1)	6 (54.5)	6.032	0.049
Infarction	22 (30.1)	0	25.903	<0.001	4 (18.2)	4 (18.2)	14 (63.6)	4.915	0.086
Increased syncytial knots	48 (65.8)	0	71.510	<0.001	4 (8.3)	7 (14.6)	37 (77.1)	4.626	0.099
Incomplete vascular remodelling	9 (12.3)	0	9.591	0.002	4 (44.4)	0	5 (55.6)	7.916y	0.019
Chorangiosis	7 (9.6)	2 (2.7)	2.960	0.085	0	0	7 (100.0)	0.326[y]	0.849
Terminal villous hypoplasia	4 (5.5)	0	4.113	0.043	0	0	4 (100.0)	0.081[y]	0.960
Accelerated villi maturity	11 (15.1)	0	11.896	0.001	4 (36.4)	1 (9.1)	6 (54.5)	6.039	0.049
Villi immaturity	11 (15.1)	3 (4.1)	5.056	0.025	2 (18.2)	2 (18.2)	7 (63.6)	2.002	0.364
Stromal fibrosis	50 (68.5)	0	76.042	<0.001	6 (12.6)	5 (10.0)	39 (78.0)	0.227	0.893
Acute chorioamnionitis	3 (4.1)	4 (5.5)	0.150	0.698	2 (66.7)	1 (33.3)	0	6.067	0.048
Microcalcifications	54 (74.0)	24 (32.9)	24.774	<0.001	3 (5.6)	6 (11.1)	45 (83.3)	6.422	0.040

χ2: Chi Square test y= Yates Corrected chi square result P<0.05

as decidual vasculopathy (P = 0.049), incomplete vascular remodeling (P = 0.019), accelerated villi maturity (P = 0.049), acute chorioamnionitis (P = 0.048), and microcalcifications were significantly associated with abnormalities in the APGAR scores at birth.

Figure 1 shows that the microscopic placental abnormalities increased with the severity of preeclampsia being higher for severe compared to mild preeclampsia.

Figure 2 shows that the highest number of perinatal mortality (6) was reported among babies whose placentas showed stromal fibrosis (6/50); incomplete vascular remodeling was associated with four deaths (4/9), while all babies (3) with features of acute chorioamnionitis on their placentas died (3/3).

Discussion

The incidence of preeclampsia/eclampsia in this study was comparable to a report of 5% in a previous study at the study center;[12] it was however higher than 1.2% from Calabar[13] and lower 8.8% from Jos[3] all in Nigeria. This further confirms the variations in the incidence of preeclampsia across different regions in the same country.

The overall mean placental weight in women with preeclampsia/eclampsia was significantly lower than those of the normotensive women. This is similar to reports from Southeast Nigeria,[14] Croatia[15] and India;[10,16] this may be attributed to the occurrence of placental abnormalities and reduced function and eventual effect on placental growth and function.

Gross placenta infarcts were reported in the placentae of women with preeclampsia/eclampsia unlike normotensive women in this study. A previous study in Nigeria reported 59% incidence of gross infarcts in the placentae of preeclamptic mothers,[14] while 66% was reported among women with preeclampsia compared to 8% among normotensive controls in a study from India.[18] The lower incidence of gross infarcts observed in this study may be attributable to the study design which allows examination of fresh placentae unlike studies which examined formalin preserved samples because fixation in formalin makes it easier to quantify the extent of infarction.[19,20] Other reports showed that gross placental infarcts are common and are not necessarily pathologic except in cases of multiple or extensive infarcts occupying more than 5% of the placental surface.[14,17]

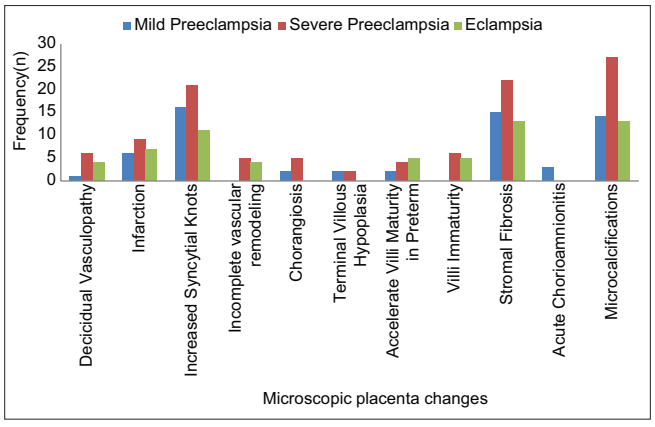

Figure 1: Microscopic placental parameters and disease severity

The report of many microscopic placental lesions among women with preeclampsia/eclampsia in this study included stromal fibrosis (68.5%), increased syncytial knotting (65.8%), infarction (30.1%), decidual vasculopathy (15.1%), accelerated villi maturity (15.1%), and incomplete vascular remodeling (12.3%). This compares to the observation of Moldenhauer *et al*. who reported that decidual arteriolopathy, villi hypermaturity, intervillous thrombi, and central infarction were significantly higher in the placentae of women with preeclampsia compared to normotensive women.[21] Similarly, a systematic review of histopathological changes in preeclampsia done in the UK reported villous lesions in 48.2% of pregnancies with preeclampsia and 11.6% of normotensive pregnancies, while vascular lesions were recorded in 37.3% of preeclampsia relative to 8.1% of normal pregnancies.[22]

There were observed relationship between disease severity and microscopic findings of incomplete vascular remodeling, villi immaturity, and calcifications in the placentae, especially among women with severe preeclampsia/eclampsia in this study. The absence of incomplete vascular remodeling in the placentas of normotensive women and those with mild diseases affirms its association with the severe spectrum of preeclampsia. Decidual vascular changes have been reported as the most specific lesions and the possible underlying causes of other pathologies such as infarction and abruption in women with maternal hypertensive disorders.[23] Although only three women had microscopic features of acute chorioamnionitis in this study, all were in women with preeclampsia/eclampsia and all resulted in perinatal mortality This may be due to the attendant fetal inflammatory response syndrome associated with the condition.[22,23]

Eight (11%) perinatal mortality was recorded in this study and all were in women with preeclampsia/eclampsia; this

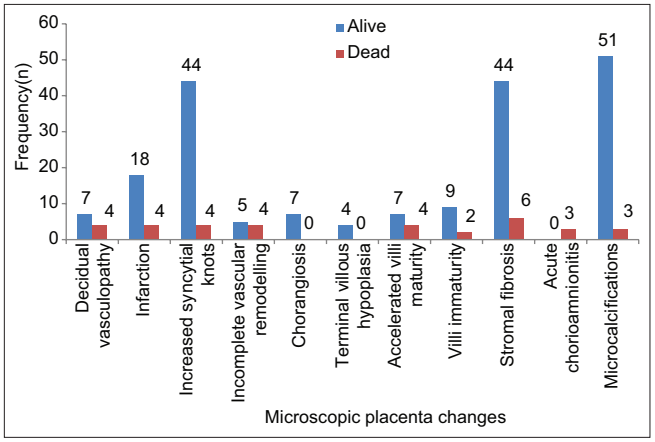

Figure 2: Relationship between microscopic placenta parameters and perinatal mortality

corroborates a similar report from Madagascar which reported a perinatal mortality rate of 11.34% attributable to placental insufficiency.[24]

It is worthy of note that many newborns with microscopic placental abnormalities had good APGAR scores at birth and survived. While this may be related to the promptness of delivery and available neonatal resuscitation of the affected women,[25,26] it further suggests that the presence of placental microscopic abnormalities is not an absolute factor for adverse perinatal outcomes. Therefore, future research should explore the role of the number, types, and grade of the microscopic pathologic abnormalities, maternal disease severity, and individual fetal and neonatal factors on the perinatal outcomes.

Conclusion

This study concludes that both gross and microscopic placental changes are associated with pregnancies complicated by preeclampsia/eclampsia with attendant increased risk for adverse perinatal outcomes. Therefore, antepartum fetal surveillance should be escalated to prioritize the characterization of placenta features using Doppler ultrasonography in the fetuses of such women in order to appropriately plan the delivery.

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Conflicts of interest

There are no conflicts of interest.

References

1. Singh S, Ahmed EB, Egondu SC, Ikechukwu NE. Hypertensive disorders in pregnancy among pregnant women in a Nigerian Teaching Hospital. Niger Med J 2014;55:384-8.
2. Mammaro A, Carrara S, Cavaliere A, Ermito S, Dinatale A, Pappalardo EM, *et al.* Hypertensive disorders of pregnancy. J Prenat Med 2009;3:1-5.
3. Musa J, Mohammed C, Ocheke A, Kahansim M, Pam V, Daru P. Incidence and risk factors for pre-eclampsia in Jos Nigeria. Afr Health Sci 2018;18:584-95.
4. Palei AC, Spradley FT, Warrington JP, George EM, Granger JP. Pathophysiology of hypertension in pre-eclampsia: A lesson in integrative physiology. Acta Physiol (Oxf) 2013;208:224-33.
5. Warrington JP, George EM, Palei AC, Spradley FT, Granger JP. Recent advances in the understanding of the pathophysiology of preeclampsia. Hypertension 2013;62:666-73.
6. Xiong X, Xu H, Wei SQ. Impact of preeclampsia on placental weight and fetal growth: Preterm versus term births. J Maternal Fetal Neonatal Med 2010;31:201-4.
7. Uzan J, Carbonnel M, Piconne O, Asmar R, Ayoubi JM. Pre-eclampsia: Pathophysiology, diagnosis, and management. Vasc Health Risk Manag 2011;7:467-74.
8. Burton GJ, Redman CW, Roberts JM, Moffett A. Pre-eclampsia: Pathophysiology and clinical implications. BMJ 2019;366:12381.
9. Sunil J. Gross examination of the placenta and its importance in evaluating an unexplained intrauterine fetal demise. J Fetal Med 2015;2:113-20.
10. Kambale T, Iqbal B, Ramraje S, Swaimul K, Salve S. Placental morphology and fetal implications in pregnancies complicated by pregnancy-induced hypertension Med J DY Patil 2016;9:341-7.
11. Araoye MO. Sample size determination. In: Araoye MO, editor. Research Methodology with Statistics for Health and Social Sciences. Ilorin: Nathadex Publishers; 2004. p. 115-8.
12. Adedoyin MA, Adetoro O. Pregnancy and its outcome among teenage mothers in Ilorin, Nigeria. East Afr Med J 1989;66:448-52.
13. Koofreh ME, Ekott M, Ekpoudom DO. The prevalence of pre-eclampsia among pregnant women in the University of Calabar Teaching Hospital, Calabar. Saudi J Health Sci 2014;3:133-6.
14. Ezeigwe CO, Okafor CI, Eleje GU, Udigwe GO, Anyiam DC. Placental peripartum pathologies in women with preeclampsia and eclampsia. Obstet Gynecol Int 2018;2018:1-9.
15. Kos M, Czernobilsky B, Hlupic L, Kunjko K. Pathological changes in placentas from pregnancies with preeclampsia and eclampsia with emphasis on persistence of 99 endovascular trophoblastic plugs. Croat Med J 2005;46:404-9.
16. Sankar KD, Bhanu PS, Ramalingam K, Kiran S, Ramakrishna BA. Histomorphological and morphometrical changes of placental terminal villi of normotensive and preeclamptic mothers. Anat Cell Biol 2013;46:285-90.
17. Roberts DJ. Placental pathology, a survival guide. Arch Pathol Lab Med 2008;132:641-51.
18. Shevade S, Arole V, Bharambe V, Paranjape V. Placental morphology and fetal outcome in preeclampsia and normotensive pregnancies. IOSR J Dent Med Sci Ver IX 2015;14:2279-861.
19. Benirschke K, Kaufmann P, Baergen RN. Pathology of the Human Placenta. 5th ed. 233 Spring Street, New York, USA: Springer Science+Business Media, Inc.,; 2006.
20. Onyiriuka AN, Okolo AA. Perinatal outcome in patients with pre-eclampsia in Benin City, Nigeria. Trop J Obstet Gynaecol 2005;21:148-52.
21. Moldenhauer JS, Stanek J, Warshak C, Khoury J, Sibai B. The frequency and severity of placental findings in women with preeclampsia are gestational age dependent. Am J Obstet Gynecol 2003;189:1173-7.
22. Falco ML, Sivanathan J, Laoreti A, Thilaganathan B, Khalil A. Placental histopathology associated with pre-eclampsia: Systematic review and meta-analysis. Ultrasound Obstet Gynaecol 2017;50:295-301.
23. Baergen RN. Manual of Pathology of the Human Placenta. 2nd ed. Boston, MA: Springer US; 2011. p. 335-48.
24. Randriamahavonjy R, Tsifiregna RL, Andrianirina ZZ, Andrianampanalinarivo HR. Materno-fetal outcomes in pre eclampsia in a rural hospital of Antananarivo Madagascar. IJRMS 2018;6:1064-7.
25. Jepson HA, Talashek ML, Tichy AM. The Apgar score: Evolution, limitations, and scoring guidelines. Birth 1991;18:83-92.
26. Montgomery KS. Apgar scores: Examining the long-term significance. J Perinat Educ 2000;9:5-9.

ملخص المقال باللغة العربية

تغييرات المشيمة ونتائج الفترة المحيطة بالولادة بين النساء الاتي يعانين من التسمم ما قبل وخلال الحمل والنساء اللواتي يعانين من ضغط دم طبيعي: دراسة مقارنة

المؤلفون:

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الهدف: كان هدف الدراسة هو تقييم التغيرات المشيمية والنتائج في الفترة المحيطة بالولادة بين النساء المصابات بتسمم ما قبل الحمل (مقدمات الارتعاج) / وتسمم الحمل (الارتعاج)، ومقارنتها بالنساء الحوامل ذوات ضغط الدم العادي.

المواد والطرق: كانت هذه دراسة مقارنة (مستقبلية)، كان عدد المشاركات فيها 146 امرأة حامل: 73 منهن كانوا يعانين من التسمم ما قبل وخلال الحمل (مجموعة الدراسة) و 73 ذوات ضغط دم معتدل (مجموعة المراقبة) وذلك عند 28-40 أسبوعًا من الحمل، تم اختيارهن بطريقة أخذ العينات الهادف. كان القياس الأولي المستهدف هو خصائص المشيمة، بينما كان الهدف الثانوي هو نتائج الفترة المحيطة بالولادة. تم إجراء التحليل الإحصائي باستخدام SPSS الإصدار 23.0، وتم تعيين الدلالة الإحصائية عند$P < 0.05$.

النتائج: كان متوسط وزن المشيمة لمجموعة الدراسة أقل بكثير من المجموعة المراقبة (556.82± 169.72 جرام مقابل 649.93 ±116.38 جرام بدلالة إحصائية ($P<0.001$). كان لدى 12 (16٪) في مجموعة الدراسة احتشاء إجمالي في المشيمة مقارنة مع عدم وجود احتشاء في المشيمة (0٪) بين المجموعة المراقبة. كما أظهرت مجموعة الدراسة 11 نوعاً من التغيرات المشيمية المجهرية مقارنة بأربعة بين مجموعة المراقبة شملت اعتلال الأوعية الدموية الساقطي ($P<0.049$) نموذجة الأوعية الدموية غير المكتملة ($P<0.019$) الزغب المتسارع النضج ($P<0.049$)، التهاب المشيمة والسلى الحاد ($P<0.048$)، والتكلسات الدقيقة ($P<0.040$)، جميعها ارتبطت ارتباط ذو دلالة إحصائية مع درجات أبغار (APGAR scores) لحديثي الولادة المنخفضة في مجموعة الدراسة. كانت درجات أبغار لحديثي الولادة في الدقائق الأولى والخامسة أقل في مجموعة الدراسة مقارنة بمجموعة المراقبة بدلالة إحصائية تساوي ($P<0.001$)، 49.3٪ مقابل 8.2٪ ($P<0.020$) و11٪ مقابل 0٪ ($P<0.001$) على التوالي، في حين كانت جميع وفيات الفترة المحيطة بالولادة الثمانية المسجلة في مجموعة الدراسة.

الخلاصة: تسمم ما قبل الحمل (مقدمات الارتعاج) / وتسمم الحمل (الارتعاج) يرتبطان بالتغيرات المشيمية الجسيمة والمجهرية غير الطبيعية التي تؤدي إلى زيادة النتائج العكسية في الفترة المحيطة بالولادة. يجب أن يعطي الترصد السابق للولادة بتسمم ما قبل الحمل وتسمم الحمل الأولوية لدراسات دوبلر (Doppler studies) لتوصيف المشيمة والتخطيط المناسب للولادة.

الكلمات المفتاحية: الارتباطات السريرية، الآفات الجسيمة، الآفات المجهرية، آفات المشيمة، تسمم الحمل.