International Journal of Clinical Obstetrics and Gynaecology

ISSN (P): 2522-6614
ISSN (E): 2522-6622
Indexing: Embase
Impact Factor (RJIF): 6.71

www.gynaecologyjournal.com
2026; 10(1): 54-57
Received: 15-10-2025
Accepted: 14-11-2025

Heba Salah Mahmoud Ghonim
Specialist Obstetric and Gynecologist, Prime Hospital Dubai, Dubai, United Arab Emirates

Nermeen Kamal Mohamed Emam
Specialist Obstetric and Gynecologist, Prime Hospital Dubai, United Arab Emirates

Ahmed Menshawy Mohamed
Specialty Registrar, Prime Hospital Dubai, United Arab Emirates

Heba Abu Saleem
Assistant Consultant King Abdullah Bin Abdulaziz University Hospital, Riyadh, Saudi Arabia

Corresponding Author:
Heba Salah Mahmoud Ghonim
Specialist Obstetric and Gynecologist, Prime Hospital Dubai, Dubai, United Arab Emirates

Impact of fetal growth restriction as a diagnostic criterion for preeclampsia on maternal disease severity: A retrospective analytical study

Heba Salah Mahmoud Ghonim, Nermeen Kamal Mohamed Emam, Ahmed Menshawy Mohamed and Heba Abu Saleem

DOI: https://www.doi.org/10.33545/gynae.2026.v10.i1a.1855

Abstract
Background: Preeclampsia (PE), which significantly impacts maternal and neonatal morbidity and mortality, is a multisystem illness of pregnancy characterized by the start of hypertension and signs of maternal organ dysfunction occurring after 20 weeks of gestation. Fetal growth restriction (FGR) is a primary cause of stillbirth and neonatal mortality. The aim of our study is to assess whether including FGR as a diagnostic criterion for preeclampsia is associated with greater maternal disease severity.
Methods: A retrospective analytical study was conducted among 101 singleton pregnancies diagnosed with preeclampsia before 37 weeks' gestation. Cases were classified as Group A (PE with FGR, N=24) and Group B (PE without FGR, N=77). Baseline demographics, maternal outcomes (severe hypertension, HELLP, ICU admission), and perinatal outcomes (birthweight, gestational age, NICU admission) were collected.
Results: Group A patients presented and delivered earlier (32.4±4.8 vs 36.6±3.3 weeks, $p<0.01$) and had significantly lower neonatal birthweights (1520±610 g vs 2590±655 g, $p<0.01$). Maternal morbidity did not differ between groups: severe hypertension (70.8 vs 61.0%, P=0.38), intravenous antihypertensives (41.7 vs 45.5%, P=0.76), and composite complications (20.8 vs 22.1%, P=0.89). NICU admission (66.7 vs 27.3%, $p<0.01$) and perinatal death (12.5 vs 3.9%, $p<0.05$) were significantly higher in Group A.
Conclusions: FGR as a diagnostic criterion signifies a placentally mediated, early-onset preeclampsia linked to adverse neonatal outcomes while exhibiting comparable maternal morbidity.

Keywords: Uteroplacental dysfunction, preeclampsia, fetal growth restriction, hypertensive disorders of pregnancy, maternal morbidity, neonatal outcome

Introduction

Preeclampsia (PE) is a multisystem illness of pregnancy characterized by the start of hypertension and signs of maternal organ or uteroplacental dysfunction occurring after 20 weeks of gestation. Affecting 3-8% of pregnancies globally, it significantly impacts maternal and neonatal morbidity and mortality [1, 2]. Fetal growth restriction (FGR), sometimes due to chronic uteroplacental insufficiency, complicates 5-10% of pregnancies and is a primary cause of stillbirth and neonatal mortality [3].

PE and FGR arise from compromised spiral-artery remodeling and endothelial dysfunction, resulting in placental hypoperfusion and the production of anti-angiogenic factors, including soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin [4-6]. These common mechanisms have led to discussions on whether FGR should be included in the diagnostic range of PE.

Prior to 2013, most diagnostic paradigms regarded FGR as indicative of serious PE. Later, the ACOG Task Force took FGR off the "severe features" list since the data did not show that it made maternal morbidity worse [7]. The 2018 ISSHP classification, on the other hand, brought back uteroplacental dysfunction, including FGR, as a valid way to diagnose PE, even if there is no proteinuria [8]. This discrepancy highlights the uncertainty about the maternal implications of FGR.

The aim of our study is to assess whether including FGR as a diagnostic criterion for preeclampsia is associated with greater maternal disease severity.

Patients and Methods

This retrospective cohort study included all singleton pregnancies complicated by preeclampsia that resulted in delivery between January 2020 and December 2024 in United Arab Emirates. The diagnosis of preeclampsia was established according to the International Society for the Study of Hypertension in Pregnancy (ISSHP, 2018) and American College of Obstetricians and Gynecologists (ACOG, 2020) criteria, requiring new-onset hypertension after 20 weeks of gestation (systolic blood pressure ≥ 140 mmHg or diastolic ≥ 90 mmHg on two occasions at least four hours apart) accompanied by either proteinuria (≥ 300 mg per 24 hours or ≥ 1+ dipstick) or signs of maternal organ dysfunction. Cases fulfilling the ISSHP definition of preeclampsia on the basis of uteroplacental dysfunction manifested as FGR or abnormal uterine or umbilical artery Doppler flow were included.

FGR was defined as an estimated fetal weight below the 10th percentile for gestational age combined with evidence of placental insufficiency such as abnormal Doppler velocimetry, in line with the international Delphi consensus proposed by Gordijn *et al*. [9]. Eligible women were classified into two groups according to their diagnostic presentation. Group A comprised preeclamptic patients whose diagnosis was based on the presence of FGR with no evidence of maternal organ dysfunction, whereas Group B included patients diagnosed through maternal organ dysfunction in the absence of FGR. Pregnancies complicated by both FGR and maternal organ dysfunction at diagnosis were excluded to preserve phenotypic distinction. Additional exclusion criteria included multiple gestations, major fetal anomalies, pre-existing chronic hypertension, renal disease, or incomplete medical documentation.

Demographic, clinical, and laboratory data were obtained from electronic medical records, including maternal age, parity, Body Mass Index (BMI), obstetric history, gestational age at diagnosis, and laboratory indices such as platelet count, liver enzymes, and serum creatinine. Blood pressure measurements were taken using automated sphygmomanometers following standardized protocols. Fetal biometry and Doppler assessments were performed by experienced sonographers using uniform equipment and reference charts. The management of preeclampsia followed institutional and ISSHP guidelines, including antihypertensive therapy with labetalol or hydralazine, magnesium sulfate for seizure prophylaxis, and corticosteroid administration for fetal lung maturation when delivery before 34 weeks was anticipated.

The primary maternal outcomes evaluated were the incidence of severe hypertension (≥ 160/110 mmHg), HELLP syndrome, eclampsia, pulmonary edema, renal impairment, and the need for intensive care unit (ICU) admission. Secondary outcomes included gestational age at delivery, mode of delivery, birthweight, Apgar score at five minutes, umbilical artery pH, neonatal intensive care unit (NICU) admission, and perinatal death. The latency period from diagnosis to delivery was calculated to assess disease progression.

Statistical analysis

Statistical analysis was done by SPSS v26 (IBM Inc., Armonk, NY, USA). Shapiro-Wilks test and histograms were used to evaluate the normality of the distribution of data. Quantitative data were presented as mean and standard deviation (SD) and were analyzed by unpaired student t-test. Qualitative data were presented as frequency and percentage (%) and were analyzed using Chi-square test or Fisher's exact test when appropriate. A two tailed p-value ≤ 0.05 was considered statistically significant.

Results

There were no significant differences in maternal age, parity, body mass index, or pre-existing medical conditions. A significant difference was observed in the gestational age at diagnosis, which occurred earlier in Group A compared with Group B (p<0.001), Table 1.

Table 1: Baseline Demographics

	Group A (N=24)	Group B (N=77)	P-Value
Maternal age (years)	34.2±4.5	35.5±5.1	0.266
Nulliparity n (%)	17 (70.8)	55 (71.4)	0.955
Pre-pregnancy BMI (kg/m²)	27.1±3.9	27.5±4.0	0.668
Chronic hypertension n (%)	2 (8.3)	6 (7.8)	1.000
Diabetes mellitus n (%)	1 (4.2)	4 (5.2)	1.000
Gestational age at diagnosis (weeks)	32.4±4.8	36.6±3.3	< 0.001*

Although Group A had slightly higher severe-hypertension rates, this difference was not significant. Also, the rates of use of intravenous antihypertensives, magnesium sulfate administration, and composite maternal complications did not differ significantly. Similarly, the incidence of HELLP syndrome, eclampsia, pulmonary edema, and ICU admission was low and showed no statistical variation. Table 2

Table 2: Maternal morbidity and management

	Group A (N=24)	Group B (N=77)	P-Value
Severe hypertension n (%)	17 (70.8)	47 (61.0)	0.385
IV antihypertensives n (%)	10 (41.7)	35 (45.5)	0.744
Magnesium sulfate n (%)	15 (62.5)	41 (53.2)	0.426
HELLP syndrome n (%)	3 (12.5)	9 (11.7)	1.000
Eclampsia n (%)	1 (4.2)	2 (2.6)	0.561
Pulmonary edema n (%)	0 (0)	2 (2.6)	1.000
ICU admission n (%)	1 (4.2)	4 (5.2)	1.000
Composite maternal complications n (%)	5 (20.8)	17 (22.1)	0.897

Pregnancies complicated by FGR (Group A) delivered significantly earlier and had markedly lower birthweights than those without FGR (Group B) (p<0.001). More than half of infants in Group A were small for gestational age. Although 5-minute Apgar scores were comparable, the rate of NICU admission was significantly higher among Group A infants (P=0.001). Perinatal mortality was higher in the FGR group but did not reach statistical significance. Table 3

Table 3: Perinatal and Neonatal Outcomes

	Group A (N=24)	Group B (N=77)	P-Value
Gestational age at delivery (weeks)	33.3±4.2	37.4±3.1	< 0.001*
Birthweight (g)	1520±610	2590±655	< 0.001*
Small-for-gestational-age n (%)	13 (54.2)	3 (3.9)	< 0.001*
Apgar score (5 min)	8.4±1.3	8.7±1.0	0.236
NICU admission n (%)	16 (66.7)	21 (27.3)	0.001*
Perinatal death n (%)	3 (12.5)	3 (3.9)	0.144

Discussion

Our results showed that women with preeclampsia who have FGR delivered significantly earlier and had markedly lower birthweights with worse outcomes, but the severity of difficulties for the mother is the same as for those without FGR.

This trend strengthens the view that FGR signifies the placental rather than the maternal systemic aspect of preeclampsia (PE). The lack of differences in rates of severe hypertension, HELLP syndrome, or eclampsia between groups corroborates earlier studies by Kasuya *et al.* [10] and Mitchell *et al.* [2], which indicated that the incorporation of FGR as a diagnostic criterion does not amplify maternal disease burden but rather identifies a subgroup marked by early onset and heightened neonatal morbidity.

Obata *et al.* [6] also discovered that fetuses presenting with preeclampsia characterized by FGR exhibited prolonged latency periods and a slower rate of maternal deterioration in contrast to organ dysfunction-based preeclampsia. This corresponds with our observation that the time of delivery in the FGR group was predominantly influenced by foetal rather than maternal indicators. Obata *et al.* [6] showed that there were overlapping placental transcriptome signatures between PE with and without FGR. This suggests that both are variations of the same clinical spectrum instead of being separate things. These findings resonate with the notion posited by Roberts and Post [3] and Redman and Sargent [11], suggesting that preeclampsia and FGR are dual outcomes of a same placental injury, presenting distinctively in the maternal and fetal compartments.

Mitchell *et al.* [2] discovered that incorporating FGR into diagnostic criteria elevated infant death from 4.6% to 16.4%, although maternal morbidity rates remained constant. These results align with the numerical trends identified in our cohort, indicating that neonatal mortality and NICU admissions were considerably elevated among FGR cases, but maternal ICU admission, magnesium sulfate utilization, and HELLP syndrome rates were comparable between groups. Takahashi *et al.* [1] similarly found that cases where uteroplacental dysfunction, evidenced by aberrant Doppler flow or FGR, was the early indication of preeclampsia had a more gradual clinical progression, yet were linked to acidosis at birth and negative neonatal outcomes. Their findings, along with ours, suggest that FGR at presentation delineates a more placenta-centric phenotype of preeclampsia that endangers fetal survival without necessarily increasing maternal risk.

Previous research, including Odegård *et al.* [12], indicated that preeclampsia exacerbated by FGR was associated with more severe maternal hypertension, proteinuria, and biochemical abnormalities. Nonetheless, these investigations occurred before contemporary mainstream standards and frequently confounded small-for-gestational-age fetuses with pathological FGR. According to the Delphi-derived definition by Gordijn *et al.* [9], it is evident that genuine FGR signifies objectively aberrant growth kinetics and Doppler indications of placental insufficiency, rather than inherent smallness. When this standardization is implemented, the perceived disparity in maternal severity significantly diminishes.

Our findings pathophysiologically corroborate the "two-stage model" of preeclampsia posited by Roberts and Hubel [13], wherein aberrant placentation and resultant placental ischemia precede the maternal systemic inflammatory and endothelial response. FGR is the fetal manifestation of the initial stage, whereas maternal organ dysfunction aligns with the latter stage. When the illness is limited to the placenta, the fetus has growth restriction; conversely, when the maternal endothelium undergoes diffuse activation, clinical preeclampsia develops. Thus, the simultaneous occurrence of both symptoms indicates timing and individual vulnerability rather than an inherently greater maternal severity. Verlohren *et al.* [14] likewise validated that these biomarkers increase concurrently, highlighting their same etiological route.

Placental histology in both illnesses displays comparable abnormalities, including decidual vasculopathy, fibrinoid necrosis, and villous infarction, as described by Roberts and Post [3] and illustrated in the case series by Calagna *et al.* [15]. In Calagna's report of early-onset FGR with severe preeclampsia, meticulous Doppler monitoring facilitated the extension of pregnancy until maternal instability necessitated cesarean section, an approach reflected in the TRUFFLE trials [16, 17], which demonstrated that postponing delivery until the late deterioration of the ductus venosus enhances neurodevelopmental outcomes without compromising maternal health. The similarity between our findings and those from prospective trials highlights that the management of FGR-related PE is mostly dependent on the equilibrium between fetal maturity and placental insufficiency, rather than maternal complication risk.

Conversely, Unterscheider *et al.* [18] enhanced the criteria for intrauterine growth restriction, highlighting that numerous fetuses are inherently normal and not at elevated perinatal risk. Our findings corroborate this distinction: the notable newborn morbidity in Group A likely indicates authentic placental insufficiency rather than inherent smallness.

From a clinical standpoint, our data indicated that FGR should continue to be recognized as a valid diagnostic criterion for preeclampsia within the ISSHP framework. This is not due to its association with a poorer maternal prognosis, but rather because it delineates a placentally driven subtype that necessitates meticulous fetal monitoring and delivery planning. Integrating FGR improves diagnostic sensitivity for early placental illness, enabling prompt referral to tertiary care and rapid corticosteroid therapy. However, doctors should not depend on its presence as a sign that the mother is getting worse, as biochemical and hemodynamic indices are still the best ways to tell.

Kahramanoglu *et al.* [19] underscored the worldwide prevalence of placental diseases and promote phenotype-based classifications, differentiating between “maternal” and “placental” preeclampsia instead of “mild” and “severe.” Our findings align with this model, as FGR-associated PE in our group adhered to the placental phenotype, manifesting early and jeopardizing the fetus without increasing maternal morbidity. These observations bolster a conceptual transition towards individualized management informed by the underlying pathophysiology instead of standardized severity criteria.

Our study was limited by its retrospective design and modest sample size, precluding multivariate adjustment for confounders such as gestational age at diagnosis.

Conclusions

FGR as a diagnostic criterion signifies a placentally mediated, early-onset preeclampsia linked to adverse neonatal outcomes while exhibiting comparable maternal morbidity.

Financial support and sponsorship: Nil

Conflict of Interest: Nil

References

1. Takahashi M, Makino S, Oguma K, Imai H, Takamizu A, Koizumi A, *et al.* Fetal growth restriction as the initial finding of preeclampsia is a clinical predictor of maternal and neonatal prognoses: A single-center retrospective study. BMC Pregnancy Childbirth. 2021;21:678.
2. Mitchell C, Becker D, Steele R, Wang M, Biggio J, Harper

L. Impact of inclusion of fetal growth restriction in severe preeclampsia diagnosis. American Journal of Obstetrics & Gynecology. 2017;216:S140.
3. Roberts DJ, Post MD. The placenta in pre-eclampsia and intrauterine growth restriction. J Clin Pathol. 2008;61:1254-60.
4. Maynard SE, Min JY, Merchan J, Lim KH, Li J, Mondal S, *et al*. Excess placental soluble FMS-like tyrosine kinase 1 (sFlt1) may contribute to endothelial dysfunction, hypertension, and proteinuria in preeclampsia. J Clin Invest. 2003;111:649-58.
5. Levine RJ, Lam C, Qian C, Yu KF, Maynard SE, Sachs BP, *et al*. Soluble endoglin and other circulating antiangiogenic factors in preeclampsia. N Engl J Med. 2006;355:992-1005.
6. Obata S, Toda M, Tochio A, Hoshino A, Miyagi E, Aoki S. Fetal growth restriction as a diagnostic criterion for preeclampsia. Pregnancy Hypertens. 2020;21:58-62.
7. American College of Obstetricians and Gynecologists. Hypertension in pregnancy. Report of the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy. Obstet Gynecol. 2013;122:1122-31.
8. Brown MA, Magee LA, Kenny LC, Karumanchi SA, McCarthy FP, Saito S, *et al*. Hypertensive disorders of pregnancy: ISSHP classification, diagnosis, and management recommendations for international practice. Hypertension. 2018;72:24-43.
9. Gordijn SJ, Beune IM, Thilaganathan B, Papageorghiou A, Baschat AA, Baker PN, *et al*. Consensus definition of fetal growth restriction: A Delphi procedure. Ultrasound Obstet Gynecol. 2016;48:333-9.
10. Kasuya M, Akiba N, Iriyama T, Sayama S, Kubota K, Toshimitsu M, *et al*. The impact of fetal growth restriction in diagnosing preeclampsia on the severity of maternal features. J Obstet Gynaecol Res. 2022;48:912-9.
11. Redman CW, Sargent IL. Placental stress and pre-eclampsia: A revised view. Placenta. 2009;30 Suppl A:S38-42.
12. Odegård RA, Vatten LJ, Nilsen ST, Salvesen KA, Austgulen R. Preeclampsia and fetal growth. Obstet Gynecol. 2000;96:950-5.
13. Roberts JM, Hubel CA. The two stage model of preeclampsia: Variations on the theme. Placenta. 2009;30 Suppl A:S32-7.
14. Verlohren S, Herraiz I, Lapaire O, Schlembach D, Moertl M, Zeisler H, *et al*. The sFlt-1/PlGF ratio in different types of hypertensive pregnancy disorders and its prognostic potential in preeclamptic patients. Am J Obstet Gynecol. 2012;206:58.e1-8.
15. Calagna G, Picciotto F, Guiglia R, Messina L, Bisanti A, Schiattarella A, *et al*. Early-onset fetal growth restriction with severe preeclampsia: Our experience of a challenging topic. Italian Journal of Gynaecology & Obstetrics. 2022;34:310-6.
16. Lees C, Marlow N, Arabin B, Bilardo CM, Brezinka C, Derks JB, *et al*. Perinatal morbidity and mortality in early-onset fetal growth restriction: cohort outcomes of the trial of randomized umbilical and fetal flow in Europe (TRUFFLE). Ultrasound Obstet Gynecol. 2013;42:400-8.
17. Frusca T, Todros T, Lees C, Bilardo CM, Hecher K, Visser GHA, *et al*. Outcome in early-onset fetal growth restriction is best combining computerized fetal heart rate analysis with ductus venosus Doppler: Insights from the Trial of Umbilical and Fetal Flow in Europe. American Journal of Obstetrics & Gynecology. 2018;218:S783-S9.
18. Unterscheider J, Daly S, Geary MP, Kennelly MM, McAuliffe FM, O'Donoghue K, *et al*. Optimizing the definition of intrauterine growth restriction: The multicenter prospective PORTO Study. Am J Obstet Gynecol. 2013;208:290.e1-6.
19. Kahramanoglu Ö, Schiattarella A, Demirci O, Sisti G, Ammaturo FP, Trotta C, *et al*. Preeclampsia: state of art and future perspectives. A special focus on possible preventions. J Obstet Gynaecol. 2022;42:766-77.

How to Cite This Article
Ghonim HSM, Emam NKM, Mohamed AM, Saleem HA. Impact of fetal growth restriction as a diagnostic criterion for preeclampsia on maternal disease severity: A retrospective analytical study. International Journal of Clinical Obstetrics and Gynaecology. 2026;10(1):54-57.