



Clinical Characteristic and Maternal-Perinatal Outcome of Early-Onset vs Late-Onset Preeclampsia: A Systematic Review

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ARTICLE INFO	ABSTRACT
Published Online: 20 May 2026	Introduction: Preeclampsia is not a single disease entity. Based on pathophysiological trajectories, Early-Onset Preeclampsia (EO-PE, <34 weeks) is highly associated with primary defective placentation and failed spiral artery remodeling, whereas Late-Onset Preeclampsia (LO-PE, ≥34 weeks) correlates more with maternal cardiometabolic decompensation. This review aims to evaluate the clinical significance of these pathophysiological differences on pregnancy outcomes. Method: This systematic review evaluated 12 primary observational studies. The primary focus of the review was to compare the severity of maternal and perinatal outcomes between patient groups with EO-PE and LO-PE. Results and Discussion: The literature synthesis demonstrated substantial differences in clinical profiles. EO-PE patients had a significantly lower gestational age at termination and lower mean birth weight due to the urgency of iatrogenic delivery. The EO-PE group consistently dominated the incidence of severe systemic maternal complications, including HELLP syndrome and placental abruption. Regarding perinatal outcomes, infants of EO-PE mothers bore massive morbidities, including increased risks of extreme prematurity, fetal growth restriction, low Apgar scores, and a high need for Perinatal Intensive Care Unit (NICU) admission. Conversely, although lethal complications were generally lower in LO-PE, the literature revealed a paradoxical outcome where the incidence of Postpartum Hemorrhage (PPH) was reported to be higher in this late-onset group. Conclusion: EO-PE consistently presents a substantially more lethal spectrum of maternal and perinatal complications compared to LO-PE. This worsening clinical profile underscores the urgency of implementing early detection strategies, specific risk stratification based on the time of onset, and proactive mobilization of advanced NICU facilities in cases of early-onset preeclampsia.
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I. INTRODUCTION

Preeclampsia is a multisystem complication of pregnancy, defined as newly-onset hypertension and proteinuria, or signs of organ dysfunction that occurred after 20 weeks of gestation in a previously normotensive woman.¹ This syndrome is a major contributor to maternal and fetal morbidity and mortality worldwide.² Preeclampsia exhibit with various clinical manifestations, many of them are life-threatening. Maternal complications are ranging from HELLP syndrome, renal failure, and eclampsia, while fetal

complication included asphyxia, intrauterine growth restriction (IUGR), and fetal death.^{3,4}

Given its distinct manifestations, modern classification divides preeclampsia based on gestational age at diagnosis into two main entities: Early-Onset Preeclampsia (EO-PE) diagnosed before 34 weeks of gestation, and Late-Onset Preeclampsia (LO-PE) at 34 weeks of gestation or more.⁵ EO-PE is primarily driven by uteroplacental abnormalities resulting from inadequate placentation, which can lead to fetal hypoxia. In contrast, LO-PE is strongly associated with maternal cardiometabolic risk factors, leading to impaired

uteroplacental perfusion despite the development of placental still preserved.⁶

Although EO-PE is generally considered to have a worse outcome profile, recent studies have reported inconsistent findings. This variability may make it difficult for clinicians to accurately predict clinical outcomes. Therefore, this systematic review was conducted to comprehensively synthesize evidence comparing Early -Onset and Late-Onset Preeclampsia in maternal and perinatal outcomes.

II. METHOD

A. Study Design

This research is a systematic review evaluating the relevant literature related to preeclampsia outcomes. The review protocol was developed and reported according to Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA).⁷ This review focused on the qualitative evidence synthesis from observational studies comparing EO-PE and LO-PE on maternal and perinatal outcomes.

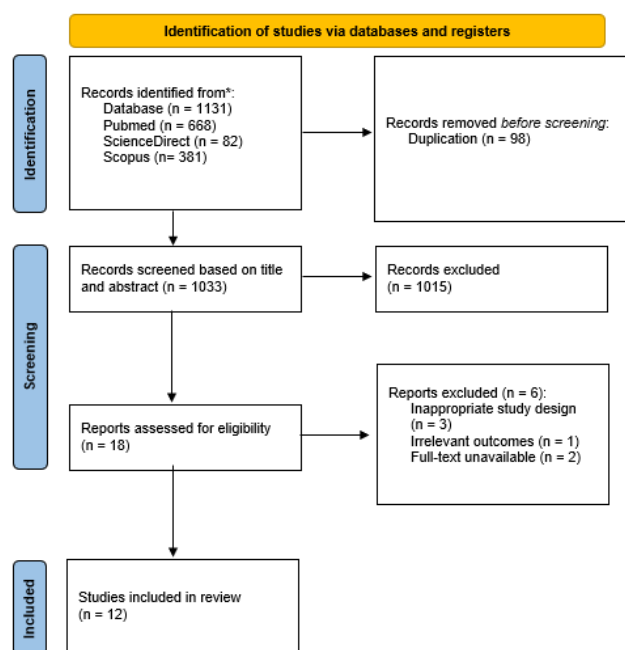


Figure 1. PRISMA Flowchart

B. Eligibility Criteria

The eligibility criteria in this review were developed based on the PECO framework (Population, Exposure, Comparator, Outcome). Included studies are primary observational studies (such as prospective cohorts, retrospective cohorts, and cross-sectional) in a population of pregnant women with preeclampsia that specifically compared exposure between EO-PE and LO-PE. This review evaluated maternal outcome parameters (HELLP syndrome, eclampsia, placental abruption, organ dysfunction, intensive care needs, and mortality) and perinatal outcomes (birth weight, fetal growth restriction/IUGR, APGAR score, NICU care needs,

asphyxia, and perinatal mortality). Case reports, case series, narrative reviews, meta-analyses, editorials, animal studies, and irrelevant studies are excluded.

C. Search Strategy

A systematic and comprehensive literature search was conducted to identify relevant studies on the preeclampsia complication, both EO-PE and LO-PE. The evidence search used a combination of specific keywords structured based on the study population and outcomes. Key terms used in the search process included: “preeclampsia”, “early-onset preeclampsia”, “late-onset preeclampsia”, “maternal outcome”, and “perinatal outcome”. These keywords are combined using Boolean operators such as “AND” and “OR.” This approach enables the inclusion of a broad spectrum of medical terminology, thereby reducing the risk of unintentional exclusion of relevant literature.

D. Study Selection and Data Extraction

The study selection process was conducted in a staged manner to minimize selection bias. All search results were exported to Mendeley (version 1.19.8) for duplicate removal and subsequently uploaded to the Rayyan.ai platform for systematic screening. Three reviewers independently assessed titles and abstracts, followed by a full-text review to determine study eligibility according to predefined criteria.

Data from eligible studies were extracted using a standardized format, including the name of the primary author, year of publication, country of study, study design, sample size, and study quality. Data related to main outcome were also collected, categorized into maternal and perinatal outcomes. This extraction procedure was conducted objectively without any manipulation of the primary investigator's original reporting.

E. Study Quality Assessment

To evaluate methodological quality and risk of bias, each included study was independently assessed by three authors using *Joanna Briggs Institute (JBI) Critical Appraisal Tools*.

F. Data Synthesis

The approach used to combine the results of various studies in this review was narrative synthesis. Findings from each literature were compared and synthesized descriptively based on the direction of association between complications, the consistency of results across studies, and the patterns in the severity of maternal and perinatal outcomes between EO-PE and LO-PE. Conclusions were drawn based on the overall qualitative evidence from the available extracted data. An easy way to comply with the conference paper formatting requirements is to use this document as a template and simply type your text into it. Wherever Times is specified, Times Roman or Times New Roman may be used.

III. RESULT

A. Study Characteristics

A total of 12 studies is included in this systematic review that met the eligibility criteria. The included studies represented a broad geographic distribution, encompassing Indonesia, Pakistan, Taiwan, China, India, the Netherlands, Denmark, France, Turkey, Poland, and Ethiopia. Most of included studies are retrospective cohort study, with some prospective cohort and cross-sectional studies.^{1,3,8-17}

Sample sizes across the studies are vary, from tens to thousands of patients based on national populations. In general, the population distribution shows a preference for LO-PE over EO-PE. This phenomenon is clearly seen in large-scale observational studies, such as Bernardes et al. (2020) study that involving 851 cases of EO-PE compared to 5,519 cases of LO-PE, and Iacobelli et al. (2017) reporting 464 cases of EO-PE compared to 933 cases of LO-PE.^{5,11}

Based on the methodological quality assessment using the JBI instrument, included studies exhibited various risk of bias profiles, range from very low to moderate. Six cohort studies achieved the maximum score, indicating very high methodological quality with a very low risk of bias. One cohort study achieved a score of 10/11, while three cohort studies scored 9/11. On the other hand, two cross-sectional studies evaluated in this review achieved a feasibility score of 6/8. Details regarding the characteristics of all selected studies are presented in **Table 1**.

B. Maternal Outcomes

Maternal complications outcomes show a variable pattern with specific trends based on the onset. The high rate of HELLP syndrome in EO-PE is supported by Bernardes et al. (2020) and Li et al. (2025) studies.^{11,16} However, a study by Wadhwani et al. (2020) reported the contrast finding, with HELLP syndrome slightly higher in the LO-PE group, at 24.0% compared to 19.3% in EO-PE.¹

Regarding eclampsia complications, several studies stated a higher incidence in the EO-PE group, as reported by Simsek et al. (2018) at 20.9% compared to 8.5% and Akbar et al. (2021) at 16.3% compared to 9.8%.^{10,17} In contrast, other literature shows a relatively equivalent or slightly higher

prevalence in the LO-PE group, for example in the study by Teka et al. (2023) with a proportion of 15.3% compared to 15.0%.⁸

The incidence of placental abruption has been consistently reported to be higher in patients with EO-PE.^{9,16} However, a contrasting pattern was observed for postpartum hemorrhage (PPH), which consistently had a higher incidence in the LO-PE group.^{1,5} Finally, regarding mortality rates (maternal death and near-misses), Simsek et al. (2018) documented a significantly higher risk in EO-PE, while Teka et al. (2023) reported equal mortality rates between the two groups.^{8,17}

C. Perinatal Outcomes

Across the included studies, EO-PE consistently demonstrated significantly worse perinatal outcomes compared with LO-PE. A defining feature of EO-PE is the high incidence of preterm delivery, which directly contributes to low birth weight (LBW); infants born to mothers with EO-PE typically weigh approximately 1,200–1,500 g, compared with 2,400–3,000 g in LO-PE.^{1,13,16}

The incidence of IUGR or SGA was also reported more frequent in the EO-PE group. Wadhwani et al. (2020) found the proportion of SGA reached 53.2% in EO-PE compared to 38.2% in LO-PE.¹ However, there was an exception in the study by Iacobelli et al. (2017) who noted a slightly higher proportion of SGA in the LO-PE group (23.3%) compared to EO-PE (21.1%).⁵

Furthermore, neonates born to mothers with EO-PE exhibit significantly impaired respiratory adaptation at birth, as reflected by lower Apgar scores and a markedly increased need for neonatal intensive care unit (NICU) admission compared with those in the LO-PE group.^{13,16} Ultimately, the most fatal complications, including IUFD and perinatal mortality, were substantially higher and more pronounced in the EO-PE group.¹¹

Table 1Study Characteristics

Author (Year)	Country & Research Period	Study Design	EOPE/LOPE (N)	Average Age (Years) (EOPE vs LOPE)	JBI Score
Akbar et al. (2021)	Indonesia; January 2016 – December 2016	Cross-sectional	47/55	not reported	6/8
Bernardes et al. (2020)	Netherlands; January 2000 – December 2007	Population-based prospective cohort	851/5,519	28 (25–31) vs 29 (26–31) (median (IQR))	11/11
Gohar et al. (2021)	Pakistan; March–September 2016	Cross-sectional	82/172	24.35 ± 3.13 vs 26.60 ± 4.23	6/8

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				(mean ± SD)	
Hauge et al. (2024)	Denmark; May 2019–March 2020	Prospective clinical cohort	139/772	32.0 ± 4.8 vs 31.9 ± 4.6 (mean ± SD)	11/11
Hung et al. (2018)	Taiwan; January 2001 – December 2016	Cohort retrospective	83/511	not reported	11/11
Iacobelli et al. (2017)	France; January 2001 – December 2015	Cohort retrospective	464/933	29.2 ± 6.8 vs 28.5 ± 7.0 (mean ± SD)	10/11
Li et al. (2016)	China; January 2010 – January 2011	Cohort retrospective	63/114	28 (20–41) vs 29 (20–41) (median (range))	9/11
Li et al. (2025)	China; January 2016 – December 2021	Cohort retrospective	525/713	31.2 ± 5.5 vs 30.8 ± 5.5 (mean ± SD)	11/11
Simsek et al. (2018)	Türkiye; ten-year period	Cohort retrospective	235/281	not reported	11/11
Teka et al. (2023)	Ethiopia; January 2015 – December 2021	Cohort retrospective	253/681	28.0 ± 6.7 vs 27.2 ± 6.2 (mean ± SD)	11/11
Wadhwani et al. (2020)	India; July 2016 – October 2017	Prospective cohort	150/150	27.21 ± 4.40 vs 26.43 ± 4.04 (mean ± SD)	9/11
Wójtowicz et al. (2019)	Poland; 2013–2017	Cohort retrospective	113/101	30.7 ± 5.5 vs 30.1 ± 5.3 (mean ± SD)	9/11

JB: Joanna Briggs Institute, EOPE: Early-onset Preeclampsia, LOPE: Late-onset Preeclampsia

Table 2Maternal Outcomes

Author (Year)	Eclampsia	HELLP	Placental abruption	Postpartum Hemorrhage	ICU Care	Maternal Death
Akbar et al. (2021)	7 (16.3%) vs 5 (9.8%)	13 (30.2%) vs 8 (15.7%)	not reported	not reported	not reported	1 (2.3%) vs 3 (5.9%)
Bernardes et al. (2020)	not reported	127 (14.9%) vs 280 (5.1%)	18 (2.1%) vs 20 (0.4%)	not reported	not reported	not reported
Gohar & Syed (2021)	not reported	not reported	not reported	not reported	not reported	not reported
Hauge et al. (2024)	not reported	7 (5.0%) vs 40 (5.2%)	not reported	not reported	not reported	not reported
Hung et al. (2018)	not reported	not reported	not reported	0 vs 1 (0.2%)	not reported	not reported
Iacobelli et al. (2017)	12 (2.6%) vs 31 (3.3%)	40 (8.6%) vs 23 (2.5%)	not reported	5 (1.5%) vs 40 (5.2%)	not reported	not reported
Li et al. (2016)	not reported	1 (1.5%) vs 1 (0.8%)	3 (4.7%) vs 1 (0.8%)	1 (1.5%) vs 1 (0.8%)	not reported	not reported
Li et al. (2025)	11 (2.1%) vs 6 (0.8%)	34 (6.5%) vs 10 (1.4%)	40 (7.6%) vs 11 (1.5%)	4 (0.8%) vs 12 (1.7%)	60 (11.4%) vs 31 (4.4%)	0 vs 0
Simsek et al. (2018)	49 (20.9%) vs 24 (8.5%)	45 (19.1%) vs 29 (10.3%)	not reported	not reported	not reported	91 (38.7%) vs 63 (22.4%) (mortality & near-miss)
Teka et al. (2023)	38 (15.0%) vs 104 (15.3%)	Hemolysis: 34 (13.4%) vs 66 (9.7%)	17 (6.7%) vs 41 (6.0%)	11 (4.3%) vs 39 (5.7%)	14 (5.5%) vs 26 (3.8%)	6 (2.4%) vs 19 (2.8%)

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Wadhwani et al. (2020)	26 (17.3%) vs 20 (13.3%)	29 (19.3%) vs 36 (24.0%)	24 (16.0%) vs 11 (7.3%)	2 (1.3%) vs 6 (4.0%)	3 vs 1	0 vs 0
Wójtowicz et al. (2019)	17 (15.0%) vs 9 (9.0%)	5 (4.4%) vs 4 (4.0%)	19 (16.8%) vs 4 (4.0%)	3 (2.6%) vs 1 (1.0%)	22 (19.4%) vs 6 (6.0%)	1 (0.8%) vs 0 (0.0%)

Table 3Perinatal Outcomes

Author (Year)	Preterm Birth	Birth Weight	SGA or IUGR	Low Apgar Score	NICU Care	IUFD or Stillbirth	Perinatal Death
Akbar et al. (2021)	41 (97.6%) vs 19 (38%)	1,525±763 vs 2,650±650 g	IUGR: 5 (11.9%) vs 4 (8%)	Apgar-1 min : 5±5 vs 7±2; Apgar-5 min : 7±5 vs 8±2	not reported	IUFD: 6 (14.3%) vs 5 (10%)	1 (2.4%) vs 0 (0%)
Bernardes et al. (2020)	not reported	not reported	SGA <5th percentile: 66 (7.8%) vs 679 (12.3%)	not reported	not reported	Stillbirth: 77 (9.0%) vs 25 (0.5%)	44 (5.2%) vs 12 (0.2%)
Gohar & Syed (2021)	49 (59.75%) vs 60 (34.88%)	not reported	SGA: 24 (29.2%) vs 51 (29.6%)	not reported	50 (60.92%) vs 72 (41.82%)	not reported	8 (9.7%) vs 22 (12.7%)
Hauge et al. (2024)	not reported	1257±517 vs 3091±660 g	SGA: 88 (64.2%) vs 77 (10.0%)	not reported	not reported	not reported	not reported
Hung et al. (2018)	not reported	1392.1±443.2 vs 2836.1±620.4 g	SGA: 42 (50.6%) vs 128 (25.0%)	Apgar <7 (1 min): 30 (36.1%) vs 17 (3.3%)	69 (83.1%) vs 44 (8.6%)	not reported	3 (3.6%) vs 0 (0.0%)
Iacobelli et al. (2017)	not reported	1228±436 vs 2617±601 g	SGA: 98 (21.1%) vs 217 (23.3%)	Apgar ≤3 (1 min): 111 (27.1%) vs 93 (10.1%)	not reported	IUFD: 31 (6.7%) vs 12 (1.3%)	13 (2.8%) vs 0 (0.0%)
Li et al. (2016)	not reported	1560 (1010-2980) vs 2400 (1410-4440) g	FGR: 10 (15.8%) vs 7 (6.1%)	not reported	not reported	Stillbirth: 22 (35%) vs 7 (6.1%)	not reported
Li et al. (2025)	411 (78.3%) vs 284 (39.8%)	1339.1±452.3 vs 2798.5±721.4 g	IUGR: 72 (13.7%) vs 53 (7.4%)	Apgar 1 min (median): 6 vs 10; Apgar 5 min (median): 8.5 vs 10	318 (60.6%) vs 173 (24.3%)	IUFD: 26 (5.0%) vs 5 (0.7%); Stillbirth: 10 (1.9%) vs 0 (0.0%)	0 vs 0
Simsek et al. (2018)	not reported	1280±459 vs 2541±748 g	not reported in its entirety	not reported	not reported	Stillbirth: 52 (22.1%) vs 18 (6.4%)	24 (10.5%) vs 6 (2.1%)
Teka et al. (2023)	not reported	BBL <2.5 kg: 215 (88.1%) vs 214 (32.0%)	IUGR: 43 (17.0%) vs 70 (10.3%)	Apgar <7 (1 min): 83 (37.9%) vs 83 (12.8%)	not reported	Stillbirth: 68 (26.9%) vs 54 (7.9%)	14 (12.8%) vs 14 (3.9%)
Wadhwani et al. (2020)	not reported	1502±0.58 vs 2416±0.59 kg	SGA: 83 (53.2%) vs 60 (38.2%)	Apgar <7 (5 min): 10 (6.4%) vs 5 (3.2%)	34 (21.8%) vs 4 (2.5%)	Stillbirth: 33 (21.2%) vs 3 (1.9%)	8 (5.1%) vs 4 (2.5%)
Wójtowicz et al. (2019)	not reported	1358±497 vs 2511±689 g	FGR <10th percentile: 80	Apgar <7 (1 min): 51	not reported	IUFD: 1 (0.9%) vs 0 (0.0%)	13 (11.5%)

	(70.7%) vs 49 (49.0%)	(45.0%) vs 4 (4.0%)	vs 0 (0.0%)
SGA: <i>Small Gestational Age</i> , IUGR: <i>Intrauterine Growth Restriction</i> , IUFD: <i>Intrauterine Fetal Death</i> , FGR: <i>Fetal Growth Restriction</i>			

IV. DISCUSSION

This systematic review exhibits significant differences in complication profiles between EO-PE and LO-PE. EO-PE consistently associated with more severe maternal and perinatal outcomes. EO-PE is triggered by failure of extra-villous trophoblast invasion and incomplete spiral artery remodelling in the first half of pregnancy.⁹ This failure of vascular remodelling put the pregnancy into a chronic placental hypoxic state that triggers a toxic cascade into the maternal systemic circulation in the early gestational age.¹⁸ Conversely, in the LO-PE group, the uteroplacental often develops normally, but maternal metabolic predisposition factors such as high body mass index or glucose intolerance fail to adapt to the physiological and cardiovascular burdens towards the end of gestation.^{6,19}

The severity of maternal target organ damage is greatly determined by the degree of toxicity from the ischemic placenta. In EO-PE, the release of antiangiogenic factors and pro-inflammatory cytokines occurs massively, triggering persistent systemic endothelial dysfunction. This endothelial damage become the underlying mechanism of fatal complications, such as HELLP syndrome and eclampsia, occur more frequent in the EO-PE group than in the LO-PE group.^{18,20}

We highlighted an interesting finding in postpartum hemorrhage outcomes. Although EO-PE are linked to most of life-threatening systemic complications, the incidence of PPH was reported higher in the LO-PE group. Li et al. (2025) stated that low potassium levels during pregnancy acts as a significant risk factor, which increase the vulnerability of postpartum hemorrhage.²¹

The high rates of preterm birth and low birth weight (LBW) in EO-PE should be primarily understood as iatrogenic consequences.^{10,22} Because the only definitive intervention to halt the progression of life-threatening maternal multiorgan dysfunction is termination of pregnancy, delivery is often undertaken before the fetus reaches physiological maturity. Furthermore, clinical deterioration that necessitates early delivery commonly includes uncontrolled hypertension, abnormal laboratory parameters, oligohydramnios, and severe intrauterine growth restriction (IUGR), all of which pose significant risks to fetal survival.¹⁰

The high incidence of IUGR or SGA, as well as low APGAR scores in EO-PE, is not purely due to premature birth, but an extent impact of chronic placental perfusion failure. Fetuses in the EO-PE group survive in a chronic hypoxic and nutritionally deprived intrauterine environment,

leading to markedly reduced physiological reserves when exposed to the stress of labor.²³

We acknowledge some limitations in this review that require critical interpretation. There is considerable heterogeneity in study designs, which combine retrospective, medical record-based cohort to a prospective cohort study. Furthermore, sample size across studies was vary. Differences in diagnostic criteria used by the included literature also exposed this review into a potential bias in the classification of disease severity.

V. CONCLUSIONS

According to the synthesis of evidence from included studies, this systematic review concludes that EO-PE and LO-PE have distinct clinical characteristics and complication outcomes. EO-PE is consistently associated with a significantly higher risk of maternal and perinatal complications. EO-PE patients are more susceptible to HELLP syndrome, severe organ dysfunction, and placental abruption. Infants of EO-PE mothers were posed to high morbidity burden, including preterm birth, low birth weight (LBW), and more NICU admission. These findings highlighting the importance of early detection strategies, risk stratification, and intensive clinical monitoring. Multidisciplinary treatment strategies are fundamental to reducing morbidity and mortality.

REFERENCES

1. Wadhwani P, Saha PK, Kalra JK, Gainer S, Sundaram V. A study to compare maternal and perinatal outcome in early vs. late onset preeclampsia. *Obstet Gynecol Sci* [Internet]. 2020;63(3):270–7. Available from: <https://www.scopus.com/inward/record.uri?eid=2-s2.0-85086372015&doi=10.5468%2FOGS.2020.63.3.270&partnerID=40&md5=0b21d30739f4587688ebdf5395203de7>
2. Duhig K, Vandermolen B, Shennan A. Recent advances in the diagnosis and management of pre-eclampsia. 2018;7:1–8.
3. Gohar S, Syed W. Comparison of adverse fetomaternal outcome in early and late onset preeclampsia. *J Postgrad Med Inst* [Internet]. 2021;35(1):15–8. Available from: <https://www.scopus.com/inward/record.uri?eid=2-s2.0-85116764837&doi=10.54079%2Fjpmi.35.1.2732&p>

- artnerID=40&md5=0f1284fa4d8b1f93e6e2b3e194b39b34
4. Ives CW, Sinkey R, Rajapreyar I, Tita AT., Oparil S. Preeclampsia — Pathophysiology and Clinical Presentations. 2020;76(14).
5. Iacobelli S, Bonsante F, Robillard PY. Comparison of risk factors and perinatal outcomes in early onset and late onset preeclampsia: A cohort based study in Reunion Island. *J Reprod Immunol* [Internet]. 2017;123:12–6. Available from: <https://www.scopus.com/inward/record.uri?eid=2-s2.0-85028299664&doi=10.1016%2Fj.jri.2017.08.005&partnerID=40&md5=be78a6c5f4a7cc297706841ec9319d58>
6. Staff CA. The two-stage placental model of preeclampsia : An update. 2019;135(June):1–10.
7. Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., Shamseer, L., Tetzlaff, J. M., Akl, E. A., Brennan, S. E., Chou, R., Glanville, J., Grimshaw, J. M., Hróbjartsson, A., Lalu, M. M., Li, T., Loder, E. W., Mayo-Wilson D. The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ Publ Gr*. 2021;372.
8. Teka H, Yemane A, Abraha HE, Berhe E, Tadesse H, Gebre F, et al. Clinical presentation, maternal-fetal, and perinatal outcomes of early-onset versus late onset preeclampsia-eclampsia syndrome in a teaching hospital in a low-resource setting: A retrospective cohort study. *PLoS One* [Internet]. 2023;18(2 February). Available from: <https://www.scopus.com/inward/record.uri?eid=2-s2.0-85148974052&doi=10.1371%2Fjournal.pone.0281952&partnerID=40&md5=59e43d68efad0a9d3fbaa7743127e1e>
9. Wójtowicz A, Zembala-Szczerba M, Babczyk D, Kołodziejczyk-Pietruszka M, Lewaczyńska O, Huras H. Early- and Late-Onset Preeclampsia: A Comprehensive Cohort Study of Laboratory and Clinical Findings according to the New ISHHP Criteria. *Int J Hypertens*. 2019;2019:4108271.
10. Akbar MIA, Kinanti H, Ernawati EE, Lestari P. Maternal and Perinatal Outcomes of Early-onset and Late-onset Preeclampsia at a Tertiary Center Hospital. *J South Asian Fed Obstet Gynaecol* [Internet]. 2021;13(5):338–42. Available from: <https://www.scopus.com/inward/record.uri?eid=2-s2.0-85139495374&doi=10.5005%2FJJP-JOURNALS-10006-1915&partnerID=40&md5=eea35784ee9ba4e13966e10a135bd5a0>
11. Bernardes TP, Mol BW, Ravelli ACJ, van den Berg P, Boezen HM, Groen H. Early and late onset preeclampsia and small for gestational age risk in subsequent pregnancies. *PLoS One*. 2020;15(3):e0230483.
12. Hauge MG, Linde JJ, Kofoed KF, Ersbøll AS, Johansen M, Sigvardsen PE, et al. Early-onset vs late-onset preeclampsia and risk of coronary atherosclerosis later in life: a clinical follow-up study. *Am J Obstet Gynecol MFM*. 2024 May;6(5):101371.
13. Hung TH, Hsieh TT, Chen SF. Risk of abnormal fetal growth in women with early- and late-onset preeclampsia. *Pregnancy Hypertens* [Internet]. 2018;12:201–6. Available from: <https://www.sciencedirect.com/science/article/pii/S21077891730123X>
14. Iacobelli S, Bonsante F, Robillard PY. Preeclampsia and preterm birth in Reunion Island: a 13 years cohort-based study. Comparison with international data. *J Matern perinatal Med Off J Eur Assoc Perinat Med Fed Asia Ocean Perinat Soc Int Soc Perinat Obstet*. 2016 Sep;29(18):3035–40.
15. Li XL, Guo PL, Xue Y, Gou WL, Tong M, Chen Q. An analysis of the differences between early and late preeclampsia with severe hypertension. *Pregnancy Hypertens*. 2016 Jan;6(1):47–52.
16. Li X, Kang F, Li X, Du X, Yang Y. Comparison of Characteristics Between Early-Onset and Late-Onset Severe Preeclampsia: A Retrospective Cohort Study from a Tertiary Hospital in China. *Reprod Sci* [Internet]. 2025;32(1):139–49. Available from: <https://www.scopus.com/inward/record.uri?eid=2-s2.0-85201275059&doi=10.1007%2Fs43032-024-01674-w&partnerID=40&md5=7f9d833cca9ec206ceb44e12c938dbab>
17. Simsek A, Uludag S, Tuten A, Acikgoz AS, Uludag S. Maternal and perinatal outcomes in early onset and late onset preeclampsia. 2018;
18. Pinheiro MB, Gomes KB, Ronda CRSC, Guimarães GG, Freitas LG, Teixeira-Carvalho A, et al. Severe preeclampsia: Association of genes polymorphisms and maternal cytokines production in Brazilian population. *Cytokine* [Internet]. 2015;71(2):232–7. Available from: <https://www.sciencedirect.com/science/article/pii/S1043466614005651>
19. Burton GJ, Jauniaux E. Pathophysiology of placental-derived fetal growth restriction. *Am J Obstet Gynecol* [Internet]. 2018;218(2, Supplement):S745–61. Available from: <https://www.sciencedirect.com/science/article/pii/S000293781732344X>

20. Gathiram P, Moodley J. Review Articles Preeclampsia: its pathogenesis and pathophysiology. 2016;27(2):71–8.
21. Privitera AA, M F, Valenti G, Raniolo S. The role of serum potassium and sodium levels in the development of postpartum hemorrhage. A retrospective study. Ital J Gynaecol Obstet 32(2)126-135. 2020;
22. Ni Y, Cheng W. Comparison of indications of pregnancy termination and prognosis of mothers and neonates in early- and late-onset preeclampsia. Hypertens pregnancy. 2016 Aug;35(3):315–22.
23. Akbar M, Sari IM, Ernawati, Aditiawarman. Plasma Level of Umbilical Cord Hemeoxygenase-1 (HO-1) and Perinatal Outcome in Early Onset and Late Onset Severe Preeclampsia. 2019;3(1).