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ORIGINAL ARTICLE

Comparison of maternal and perinatal outcomes between early-and late-onset preeclampsia

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ABSTRACT

Objective: Preeclampsia is a major cause of maternal and fetal-neonatal morbidity and mortality worldwide. The timing of onset–whether early (<34 weeks) or late (≥34 weeks)–may influence both maternal and perinatal outcomes. This retrospective study aimed to compare the clinical and perinatal outcomes of early- versus late-onset preeclampsia in singleton pregnancies.

Material and Methods: Medical records of 193 women with singleton pregnancies complicated by preeclampsia were retrospectively analyzed at a tertiary referral center between January 2013 and January 2014. Patients were categorized into early-onset (24–34 weeks) and late-onset (≥34 weeks) groups. Maternal demographic and clinical characteristics, laboratory parameters, obstetric complications, and neonatal outcomes (birth weight, Apgar scores, NICU admission, cord blood pH) were compared.

Results: Early-onset preeclampsia was associated with significantly higher AST, ALT, LDH, proteinuria, hypoalbuminemia, hypoproteinemia, magnesium sulfate therapy, cesarean delivery, oligohydramnios, fetal growth restriction, maternal complications, and NICU admissions. Compared with neonates in the late-onset preeclampsia group, those born to mothers with early-onset disease had significantly lower birth weights, reduced Apgar scores at 1 and 5 minutes, and more acidotic cord blood gases.

Conclusion: Early-onset preeclampsia represents a more severe form of the disease, characterized by higher maternal morbidity and adverse neonatal outcomes. The 34-week threshold appears to be a critical determinant of prognosis, with longer gestation positively influencing neonatal survival and health. Early detection, close monitoring, and timely delivery remain key strategies for improving maternal and perinatal outcomes.

Keywords: Early-onset preeclampsia, late-onset preeclampsia, preeclampsia.

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INTRODUCTION

Preeclampsia is defined as new-onset hypertension and proteinuria or end-organ dysfunction after the 20th week of pregnancy.[1] Preeclampsia occurs in 2–3% of pregnancies.[2] The occurrence of generalized tonic-clonic seizures in a preeclamptic woman, in the absence of other neurological conditions that could cause convulsions, is defined as eclampsia. Each year, more than 4 million women worldwide develop preeclampsia, and eclamptic convulsions occur in about 100,000 of these cases.[3]

Regarding the pathophysiology of preeclampsia, maternal, fetal, and placental factors are involved.[4] Abnormalities in the development of placental vascularization have been identified weeks to months before the onset of the disease, particularly in early pregnancy.[5,6] These abnormalities lead to placental hypoperfusion, hypoxia, and ischemia. Hypoperfusion and ischemia trigger the release of antiangiogenic factors (such as soluble fms-like tyrosine kinase-1 [sFlt-1] and soluble endoglin [sEng]) into the maternal circulation, which can cause widespread endothelial dysfunction. This results in hypertension, proteinuria, and other clinical manifestations of preeclampsia.[7] Although the severity of the disease is primarily influenced by maternal and pregnancy-specific factors, paternal and environmental factors may also play a role.[8]

Preeclampsia is a condition that can cause significant morbidity and mortality for both the mother and the fetus. Despite the marked reduction in maternal mortality in developed countries, it remains one of the leading causes of pregnancy-related deaths.[9] The most effective treatment for improving maternal and fetal prognosis is still the timely termination of pregnancy.

Uteroplacental insufficiency due to vasospasm may endanger fetal life, while cardiovascular, renal, pulmonary, and cerebral complications that develop during eclamptic seizures can threaten maternal life.

In many women, these findings become more prominent, especially after the 34th gestational week (late-onset preeclampsia). Late-onset preeclampsia (LO-PE) (≥34 weeks) is more common than early-onset preeclampsia (EO-PE) (<34 weeks).[10] Approximately 10% of women develop preeclampsia before 34 gestational weeks (EO-PE). Our study aims to compare fetal and maternal outcomes in cases of EO-PE and LO-PE. The study included all cases diagnosed with superimposed preeclampsia, preeclampsia with or without severe features, eclampsia, and HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome.

Women with preeclampsia are at increased risk for life-threatening complications such as placental abruption, acute renal failure, cerebral hemorrhage, hepatic failure or rupture, pulmonary edema, disseminated intravascular coagulation, and eclampsia. Globally, 10–15% of maternal deaths are attributed to preeclampsia.[11] In the United States, preeclampsia is one of the leading causes of maternal mortality.[12–14] Maternal death due to preeclampsia occurs in approximately 1 out of every 10,000 live births.[15,16]

Due to the increased risk of fetal growth restriction and preterm birth in affected pregnancies, fetal and neonatal morbidity and mortality are elevated.[17]

Risk factors:[8] History of preeclampsia in previous pregnancies, nulliparity, family history of preeclampsia, existing medical problems (diabetes, chronic hypertension, antiphospholipid antibodies, body mass index ≥26, chronic kidney disease), multiple pregnancies, advanced maternal age.

The diagnostic criteria according to international guidelines:[18–21]

• Preeclampsia Without Severe Features: Systolic blood pressure detected for the first time after the twentieth week of gestation ≥140 mmHg, diastolic blood pressure ≥90 mmHg, and proteinuria defined as a dipstick result of ≥1+ on a spot urine sample or a protein excretion of ≥300 mg/day in a 24-hour urine collection.

• Preeclampsia With Severe Features: Systolic blood pressure≥160 mmHg and diastolic blood pressure ≥110 mmHg, detected for the first time after the twentieth week of gestation and/or proteinuria, elevated serum creatinine, thrombocytopenia, microangiopathic hemolysis, elevated ALT or AST, persistent headache or other cerebral or visual symptoms, persistent epigastric pain, pulmonary edema, or oliguria.

• Eclampsia: Defined as the occurrence of grand mal seizures in a woman in the absence of other neurological conditions that may cause seizures.

• HELLP Syndrome: Hemolysis, elevation in liver function tests, and thrombocytopenia.

• Chronic Hypertension: Defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg before the 20th week of pregnancy or lasting longer than the 12th postpartum week.

• Superimposed Preeclampsia: Defined as new-onset proteinuria and/or end-organ damage in a chronically hypertensive woman after the 20th week of pregnancy. The aggravation of hypertension may also characterize the condition during the second half of pregnancy, particularly when acute and resistant, or by the emergence of clinical features indicating severe disease.

• Gestational Hypertension: Defined as hypertension occurring after the 20th week of pregnancy, without signs and symptoms of preeclampsia and proteinuria. Hypertension is expected to resolve by the 12th postpartum week.

Post-diagnostic laboratory/imaging evaluations should include:

1. Complete blood count
2. Serum creatinine
3. Serum AST (aspartate aminotransferase) and ALT (alanine aminotransferase) levels
4. Obstetric ultrasound (estimated fetal weight, amniotic fluid volume)
5. Fetal evaluation (fetal biophysical profile or non-stress test)

MATERIAL AND METHODS

This study was carried out by retrospectively examining the files of 193 pregnant women diagnosed with preeclampsia who gave birth in a single tertiary center between January 2013 and January 2014 to compare their maternal and perinatal outcomes, with the approval of the ethics committee. The study was conducted in accordance

with the principles of the Declaration of Helsinki. Approval was obtained from the İstanbul Kartal Research Hospital (approval No: 89513307/1009/416-24, date: 10.02.2015). Informed consent was obtained from all participants.

Pregnant women with a single live fetus diagnosed with preeclampsia were included in the study. The results were compared before and after 34 weeks (EO-PE and LO-PE). Cases of preeclampsia with or without severe features, superimposed preeclampsia, eclampsia, and HELLP syndrome were included in both groups. Multiple pregnancies and in-utero fetal loss were excluded from the study.

Blood pressure measurements for diagnosis were recorded when the patient was found to have elevated values at least twice, at an interval of 6 hours, while resting.

Each patient was evaluated according to demographic and clinical characteristics, including maternal age, parity, gestational age at diagnosis, and type of hypertensive disorder (preeclampsia with or without severe features, eclampsia, HELLP syndrome, or superimposed preeclampsia). Obstetric outcomes, including mode of delivery, indications for cesarean delivery, and administration of magnesium sulfate therapy, were recorded. The presence of concomitant medical conditions (chronic hypertension, diabetes mellitus, pulmonary disease, thyroid disorders, cardiac disease, deep vein thrombosis, gastritis/gastroesophageal reflux, renal disease, or cholelithiasis) was also documented.

Laboratory investigations included hemoglobin concentration, platelet count, liver function tests (AST, ALT), renal function markers (uric acid, BUN, creatinine), and proteinuria assessment (proteinuria assessed by dipstick testing in spot urine samples and quantified by 24-hour urinary protein excretion), along with albumin and total protein levels.

Fetal and maternal complications were analyzed, including fetal growth restriction, oligohydramnios, and maternal morbidities such as blood transfusion requirement, eclampsia, HELLP syndrome, placental abruption, pulmonary embolism, and uterine rupture. Neonatal outcomes were assessed by birth weight, 1- and 5-minute Apgar scores, need for neonatal intensive care unit (NICU) admission, and umbilical cord blood pH values.

Statistical Analysis

Statistical analyses were performed using SPSS software (version 16.0). Categorical parameters were presented with frequency and percentage tables. Normality testing was performed to determine whether parametric or non-parametric tests would be used in comparison tests (for parameters where mean values were analyzed). The Kolmogorov-Smirnov test (Shapiro-Wilk when the sample size was below 50) was used for normality testing.

If the p-value obtained in the Kolmogorov-Smirnov test was greater than 0.05 in all subgroups, the distribution was considered normal, indicating no excessive deviation from normality. In cases where the normality assumption was not met, non-parametric tests (Mann-Whitney-U, Kruskal-Wallis, etc.) were used as an alternative to parametric tests (t-test, analysis of variance, etc.).

In the normality test, two independent sample t-tests were used to compare normally distributed dependent variables according to independent variables with two subgroups. The Mann-Whitney-U test was used to compare dependent variables without normal distribution according to independent variables with two subgroups. The Chi-square test was used to compare categorical variables. The significance level was determined as $p<0.05$.

RESULTS

The mean maternal age of the EO-PE group (30.36±7.54) and the LO-PE group (30.34±6.47) did not differ significantly.

<36.1% of the EO-PE group were primiparous and 63.9% were multiparous. >35.7% of the LO-PE group were primiparous and 64.3% were multiparous. There was no statistically significant difference in parity between the two groups.

In the EO-PE group, among those with comorbidities, 5.6% had hypertension, 2.8% hyperlipidemia and lung disease, 5.6% hyperlipidemia and diabetes, 2.8% goiter and kidney disease, 2.8% hyperlipidemia and goiter, 2.8% diabetes, and 2.8% deep vein thrombosis. In the LO-PE group, among those with comorbidities, 5.1% had hypertension, 1.9% hyperlipidemia and diabetes, 0.6% goiter and kidney disease, 1.3% hyperlipidemia and goiter, 0.6% hyperlipidemia and kidney disease, 0.6% diabetes and lung disease, 0.6% hyperlipidemia and diabetes and lung disease, 1.3% diabetes and goiter, 8.3% diabetes, 1.9% cardiac disease, 4.5% goiter, 1.3% lung disease, and 0.6% renal disease.

<83.3% of the EO-PE group did not have preeclampsia in their previous pregnancy, and 16.7% had preeclampsia. >89.8% of the LO-PE group did not have preeclampsia in their previous pregnancy, and 10.2% had preeclampsia. There was no statistically significant difference in the history of preeclampsia between the two groups.

Among all women included in the study, 40.4% had preeclampsia without severe features, 49.7% had preeclampsia with severe features, 2.1% had eclampsia, 5.2% had HELLP syndrome, and 2.6% had superimposed preeclampsia. In the EO-PE group, 5.6% had preeclampsia without severe features, 72.2% had preeclampsia with severe features, 5.6% had eclampsia, and 16.7% had HELLP syndrome. In the LO-PE group, 48.4% had preeclampsia without severe features, 44.6% had preeclampsia with severe features, 1.3% had eclampsia, 2.5% had HELLP syndrome, and 3.2% had superimposed preeclampsia.

As shown in Table 1, uric acid and creatinine levels did not differ significantly between the two groups. Proteinuria assessed by dipstick testing of spot urine samples and quantified as total protein excretion (mg/day) in 24-hour urine collections, blood urea nitrogen (BUN), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and lactate dehydrogenase (LDH) levels were significantly elevated in EO-PE. In contrast, the 1st and 5th minute Apgar scores, umbilical cord blood pH, and birth weight were significantly higher in LO-PE. No significant differences were found between EO-PE and LO-PE for maternal hemoglobin levels, platelet counts, albumin concentrations, and total protein levels.

According to Table 2, comorbidity rates did not differ significantly between the EO-PE (27.8%) and LO-PE (29.9%) groups. Magnesium sulfate use was significantly higher in the EO-PE group (30.6%) compared to the LO-PE group (7%).

Table 1: Comparison of parameters EO-PE and LO-PE- Mann-Whitney U Test results

Parameters	EO-PE			LO-PE			U	p
	n		SD	n		SD		
Uric acid	36	5.59	1.73	157	5.25	1.35	2661.00	0.584
Spot urine protein excretion- dipstick	36	**2.39**	1.20	156	1.67	1.21	1914.00	**0.002**
24 hours urine protein excretion (miligrams/24 hours)	17	**3973.4**	3961.5	46	2302.1	3765.3	248.00	**0.027**
Blood creatinine	36	0.64	0.18	157	0.65	0.37	2542.50	0.348
BUN	36	**12.19**	5.14	157	9.60	5.08	1850.00	**0.001**
ALT	36	**25.33**	29.59	157	18.22	23.80	2072.00	**0.012**
AST	36	**33.78**	33.49	157	28.36	40.05	2184.00	**0.033**
LDH	36	**527.00**	249.34	156	449.88	314.50	2140.00	**0.026**
Apgar score1	36	5.67	2.65	157	**8.46**	0.94	896.00	**0.000**
Apgar score5	36	7.78	2.10	157	**9.64**	0.66	1032.00	**0.000**
Cord blood-pH	36	7.27	0.10	157	**7.36**	0.15	1182.00	**0.000**

ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; BUN: Blood urea nitrogen; EO-PE: Early-onset preeclampsia; LDH: Lactate dehydrogenase; LO-PE: Late-onset preeclampsia; n: Number; p: p-value; SD: Standard deviation; U: Mann–Whitney U Test.

Table 2: Chi-Square Analysis Results of Comparison of EO-PE and LO-PE according to their comorbidity status

Gestational age	Additional medical conditions				Total	χ^2	p
	No		Yes				
	n	%	n	%			
EO-PE	26	72.2	10	27.8	36	0.066	0.798
LO-PE	110	70.1	47	29.9	157		
Total	136	70.5	57	29.5	193		

EO-PE: Early-onset preeclampsia; LO-PE: Late-onset preeclampsia; n: Number; p: p-value; %: Percentage; χ^2: Chi-Square Test.

In the EO-PE group, 5.6% delivered vaginally and 94.4% by cesarean section, whereas in the LO-PE group, 25.5% delivered vaginally and 74.5% by cesarean section. The rate of cesarean delivery was significantly higher in EO-PE compared to LO-PE.

According to Table 3, fetal growth restriction was significantly more frequent in the EO-PE group (80.6%) compared with the LO-PE group (36.3%).

According to Table 4, there was no significant difference between the two groups in terms of oligohydramnios.

According to Table 5, all neonates in the EO-PE group required NICU admission, whereas only 23.6% of the LO-PE group did ($p<0.001$). Maternal complications were more diverse and frequent in EO-PE, including blood transfusion (2.8%), eclampsia crisis (2.8%), placental abruption (5.6%), eclampsia (2.8%), HELLP syndrome (11.1%), pulmonary embolism (2.8%), and uterine rupture (2.8%). In the LO-PE group, complications included blood transfusion (3.2%), placental abruption (0.6%), eclampsia (1.3%), and HELLP syndrome (1.9%).

DISCUSSION

This study analyzed demographic and clinical variables in women with preeclampsia, focusing on whether maternal and neonatal outcomes varied significantly when 34 weeks of gestation was applied as the dividing line between early- and late-onset disease. Overall, our findings suggest that 34 weeks represents a critical threshold for maternal well-being, whereas for the fetus, each additional week of gestation is associated with a more favorable prognosis.

Particularly in cases of severe preeclampsia and HELLP syndrome, elevations in AST, ALT, and LDH levels are well recognized. In our study, these parameters were found to be significantly different between early- and late-onset preeclampsia,

Table 3: Chi-Square Analysis Results of Comparison EO-PE and LO-PE According to Fetal Growth Retardation Status

Gestational age	No		Yes		Total	χ^2	p
	n	%	n	%			
EO-PE	7	19.4	29	80.6	36	23.211	**0.000**
LO-PE	100	63.7	57	36.3	157		
Total	107	55.4	86	44.6	193		

EO-PE: Early-onset preeclampsia; LO-PE: Late-onset preeclampsia; n: Number; p: p-value; %: Percentage; χ^2: Chi-Square Test.

Table 4: Chi-Square analysis results of comparison of EO-PE and LO-PE according to oligohydramniosis status

Gestational age	Oligohidramniosis				Total	χ^2	p
	No		Yes				
	n	%	n	%			
EO-PE	19	52.8	17	47.2	36	2.297	0.130
LO-PE	104	66.2	53	33.8	157		
Total	123	63.7	70	36.3	193		

EO-PE: Early-onset preeclampsia; LO-PE: Late-onset preeclampsia; n: Number; p: p-value; %: Percentage; χ^2: Chi-Square Test.

Table 5: Chi-Square analysis results of comparison of EO-PE and LO-PE according to the NICU admission

Gestational age	No		Yes		Total	χ^2	p
	n	%	n	%			
EO-PE	0	0.0	36	100.0	36	72.748	**0.000**
LO-PE	120	76.4	37	23.6	157		
Total	120	62.2	73	37.8	193		

NICU: Neonatal intensive care unit; EO-PE: Early-onset preeclampsia; LO-PE: Late-onset preeclampsia; n: Number; p: p-value; %: Percentage; χ^2: Chi-Square Test.

with higher values observed in the early-onset group. These results suggest that early-onset preeclampsia (EO-PE) is associated with a more severe clinical course than late-onset preeclampsia (LO-PE).

The frequency of magnesium sulfate use for the prevention of eclampsia was markedly higher among patients with EO-PE, consistent with its association with more severe disease.

The rate of admission to neonatal intensive care units was greater among newborns from the EO-PE group. This is an expected finding, given that EO-PE is generally associated with a more severe disease course, a higher incidence of fetal growth restriction causing fetal stress, and preterm delivery performed due to maternal or fetal indications.

The increased frequency of cesarean delivery in the EO-PE group may be explained by multiple factors, including the lower birth weight of premature neonates—prompting cesarean section to reduce the risk of birth trauma–and the frequent absence of sufficient cervical ripening in cases requiring urgent delivery.[22]

Neonatal birth weight and the 1st and 5th minute Apgar scores were also lower in the EO-PE group. Cord blood samples obtained from the umbilical vein demonstrated that infants of mothers with EO-PE were more acidotic at birth.

One of the earliest responses to impaired fetal nutrition is the development of oligohydramnios. Prolonged and more severe nutritional compromise may subsequently lead to fetal growth restriction (FGR). In our study, no significant difference was observed between EO-PE and LO-PE in terms of oligohydramnios; however, FGR was found to be more frequent in the early-onset group.

In EO-PE, both the earlier onset of the disease and its more severe course contributed to oligohydramnios and FGR. In LO-PE, the later onset and the relatively short interval between disease onset

and delivery likely limited the duration of fetal exposure to stress. As a result, although amniotic fluid volume was affected, it did not progress to FGR.

The presence or absence of additional medical conditions did not appear to make a significant difference. This may be explained by the relatively young mean age of the study population, the generally low prevalence of chronic diseases, and the likelihood that any existing chronic conditions in these patients were well controlled under medical follow-up.

Greater disease severity is associated with an increased risk of additional complications.[23] Accordingly, in the EO-PE group—where the disease follows a more severe course—the probability of developing further complications is understandably higher. Conditions such as anemia requiring blood transfusion, placental abruption, eclampsia, and HELLP syndrome were observed more frequently in the EO-PE group.

An increase in serum uric acid has been suggested as a potential marker for predicting preeclampsia. In our study, higher uric acid levels were associated with increased proteinuria, hypoalbuminemia, and elevated creatinine, reflecting renal involvement. However, previous reports suggest that hyperuricemia alone may not be a reliable predictor.[23,24]

Our findings demonstrated that greater protein loss in spot urine or 24-hour collections was linked to higher BUN, creatinine, AST, and ALT values, in addition to reductions in serum albumin, total protein, Apgar scores at both 1 and 5 minutes, and infant birth weight. This supports the concept that the magnitude of proteinuria may be indicative of preeclampsia severity.

A 2014 U.S. study of 670,120 singleton deliveries reported severe maternal morbidity in 15.5% of EO-PE and 12% of LO-PE, with higher rates of cardiovascular, renal, hepatic, and transfusion-related complications in EO-PE.[10] Consistent with this, our study also found increased blood transfusion requirements, elevated liver enzymes, and hypoproteinemia in EO-PE. However, no maternal deaths were observed, likely due to the smaller sample size. Elevated liver function tests and hypoproteinemia were more frequently observed in the EO-PE group.

A 2009 Korean study of 212 patients, using 32 weeks as the cutoff, reported higher rates of severe preeclampsia, elevated liver enzymes, pulmonary edema, fetal death, lower Apgar scores, and increased perinatal mortality in EO-PE.[25] Similarly, in our study using 34 weeks as the cutoff, no differences were observed regarding maternal age, parity, or preeclampsia history, but EO-PE was associated with greater proteinuria, more severe disease, and lower Apgar scores.

A 2013 U.S. study of 456,668 pregnancies reported a preeclampsia prevalence of 3.1%, with EO-PE at 0.38% and LO-PE at 2.72%. EO-PE was associated with African-American ethnicity, chronic hypertension, and congenital anomalies, while LO-PE was more common in younger, nulliparous, and diabetic women.[26] Perinatal death or severe neonatal morbidity occurred in 16.4% of EO-PE versus 2% of LO-PE. In our study, maternal age and parity did not differ between groups. Although comprehensive data on neonatal morbidity were unavailable, neonates born to mothers with EO-PE had significantly lower birth weights and Apgar scores, as well as more acidotic umbilical cord blood gases.

In a study conducted in Türkiye in 2014, preeclamptic patients were divided into early- and late-onset groups using 34 weeks as the cutoff, and maternal-fetal morbidity and mortality were compared, incorporating uterine artery Doppler values as well.[27] Similar to our findings, perinatal outcomes were reported to be worse in the early-onset group. Additionally, impaired uterine artery Doppler values were identified in EO-PE, supporting the conclusion that abnormal placentation is the primary etiological factor in EO-PE.

CONCLUSION

The impact of preeclampsia on maternal and perinatal health is profound, contributing to increased morbidity and mortality. Even in regions where maternal mortality has significantly declined, such as developed countries, preeclampsia continues to be recognized as one of the predominant causes of pregnancy-associated fatal outcomes.

In assessing the severity of preeclampsia, maternal clinical findings and biochemical markers are typically prioritized. Nevertheless, the etiological mechanisms of early-onset and late-onset forms are fundamentally distinct. The pathogenesis of EO-PE is largely driven by abnormal placentation, whereas LO-PE tends to be influenced predominantly by maternal factors.

To conclude, replacing the traditional categorization of preeclampsia based on the presence or absence of severe features with the division into EO-PE and LO-PE appears more suitable for outcome prediction. While early-onset disease carries a higher risk of maternal complications, prolongation of pregnancy has been shown to positively influence neonatal survival and health.

Statement

Ethics Committee Approval: The study was approved by İstanbul Kartal Research Hospital Ethics Committee granted approval for this study (date: 10.02.2015, number: 89513307/1009/416-24).

Informed Consent: Informed consent was obtained from all participants.

Conflict of Interest: The authors have no conflict of interest to declare.

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