

# APRI, FIB-4, and FIB-5 Scores and Their Association with Late-Onset Preeclampsia

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## ABSTRACT

**OBJECTIVES:** Late-onset preeclampsia (LO-PE) is a major cause of maternal–perinatal morbidity. Noninvasive liver fibrosis indices, the AST-to-platelet ratio index (APRI), Fibrosis-4 (FIB-4), and Fibrosis-5 (FIB-5), may capture subclinical hepatic injury in preeclampsia. We assessed the association of these indices with LO-PE and their diagnostic performance.

**STUDY DESIGN:** In this single-center, retrospective, case-control study, we compared pregnant women with LO-PE (defined as preeclampsia onset at or after 34 weeks of gestation, with no chronic hypertension; n=84) with healthy pregnant controls matched by gestational age (n=84). Demographics, obstetric features, and laboratory parameters (AST, ALT, ALP, platelet count, and albumin) were obtained from medical records. APRI, FIB-4, and FIB-5 indices were calculated using standard formulas. Differences between groups were assessed using statistical tests. For normally distributed data, parametric tests (such as the independent-samples t-test) were used. For nonnormally distributed data, we used non-parametric tests (such as the Mann-Whitney U test). The ability of each index to distinguish between cases and controls was evaluated using ROC curve analysis. ROC analysis plots sensitivity versus 1-specificity to assess diagnostic performance.

**RESULTS:** ALT, AST, ALP, FIB-4, and APRI levels were significantly higher, whereas FIB-5, platelet, and albumin levels were significantly lower in patients with preeclampsia than in controls (p<0.05). APRI, FIB-4, and FIB-5 values were found to be important parameters affecting disease status (p<0.05). When APRI and FIB-4 values increase by 1 unit, the risk of disease will increase 23.683 and 59.402 times, respectively. In contrast, for each additional unit increase in the FIB-5 score, the risk of disease decreases by 13.3%.

**CONCLUSION:** APRI and FIB-4 are independent predictors of LO-PE and provide robust discrimination between affected and unaffected patients. While FIB-5 offers limited diagnostic accuracy, APRI and FIB-4, available from routine antenatal labs, can be leveraged for timely risk stratification and early detection of LO-PE. However, prospective validation and clear thresholds are needed to enable effective clinical implementation.

**Keywords:** APRI; FIB-4; FIB-5; Hepatic fibrosis; Late-onset preeclampsia; Non-invasive liver markers

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## Introduction

Preeclampsia is a multisystem hypertensive disorder of pregnancy defined by new-onset hypertension after 20 weeks of gestation (1), accompanied by proteinuria and/or maternal end-organ dysfunction. It is a significant disease that can cause complications for both mother and fetus, remaining a leading cause of perinatal and maternal mortality worldwide (2). Although abnormal placentation leading to systemic inflammation and endothelial dysfunction is central to its development, pro-inflammatory and pro-thrombotic mechanisms may further contribute to end-organ injury and hypoperfusion; however, the exact etiology remains incompletely understood (3-5). Preeclampsia can involve the liver through endothelial dysfunction, microvascular injury, and impaired hepatic perfusion, leading to biochemical abnormalities and, in severe cases, overt hepatic complications. Most patients exhibit only

mild-to-moderate liver enzyme abnormalities, while marked aminotransferase elevations are predominant in severe forms, such as HELLP syndrome (6).

APRI, FIB-4, and FIB-5 are noninvasive indices calculated from routine laboratory parameters. While they were originally developed for hepatology, they may also be relevant in preeclampsia, given the associated changes in transaminases, platelet count, and albumin. These indices may serve as exploratory markers of hepatic stress or disease burden in preeclampsia, though they have not been validated in pregnant populations (7).

This retrospective study aimed to compare prediagnostic APRI, FIB-4, and FIB-5 values between women with late-onset preeclampsia and normotensive pregnant controls, and to examine their association with case status within the study cohort.

## Material and Method

**Study Population:** This retrospective, case-control study was conducted from December 2022 to December 2024 in the antenatal clinic of a tertiary hospital in Ankara, Türkiye. We included 84 patients diagnosed with preeclampsia and 84 healthy controls. The study received approval from the Ethics Committee of the Ankara Atatürk Sanatoryum Training and Research Hospital (Approval No: 2024/220; Date: 12.02.2025) and adhered to the principles of the Declaration of Helsinki. Patients were included in the preeclampsia group if they were diagnosed after 34 weeks of pregnancy without prior chronic hypertension (n=84). Late-onset preeclampsia was defined by newly elevated blood pressure ( $\geq 140/90$  mmHg) after 34 weeks of pregnancy. Diagnosis also required either significant proteinuria ( $\geq 300$  mg/24h) or clinical features indicating systemic compromise, such as low platelet count, hepatic enzyme elevation, renal impairment, pulmonary congestion, or cerebral/visual disturbances (8). The control group was composed of age- and gestational age-matched healthy pregnant women. These women attended outpatient antepartum follow-up, had singleton live pregnancies, and no preeclampsia (n=84). Exclusion criteria were age less than 20 or over 40 years, less than 34 weeks' gestation, other liver conditions such as viral hepatitis or drug-induced liver injury, comorbidities (pre-existing hypertension, viral liver infections, gestational diabetes, coagulation disorders, increased hepatic enzyme levels, and thrombocytopenia), systemic infections (including urinary tract infections), multiple pregnancies, or cases without retrievable records.

**Study Parameters:** Data for the preeclampsia and control groups were retrieved from the hospital's database. Demographic characteristics (such as age, education status, and body mass index), gestational information (gestational age, gravida, and parity), systolic and diastolic blood pressure,

and laboratory values (AST, ALT, ALP, platelet count, and albumin) were included.

Timing of laboratory measurements and definition of gestational age.

To ensure a clear temporal relationship between laboratory indices and disease onset, APRI, FIB-4, and FIB-5 were calculated using only prediagnostic laboratory results. For the preeclampsia group, the index sample was the most recent routine antenatal blood test before clinical diagnosis. If multiple eligible samples were available, the closest to diagnosis was chosen. For controls, laboratory values were taken from routine third-trimester antenatal visits. These were selected to match the gestational age of the corresponding patient as closely as possible. All laboratory values were retrieved from hospital records and analyzed using standard laboratory procedures. In this manuscript, 'gestational week' refers to the gestational age at the time of the index sample.

**FIB-4, FIB-5 Indexes and APRI Score:** FIB-4, FIB-5, and APRI are widely used noninvasive indices for assessing liver fibrosis. To calculate these scores, we obtained pre-diagnostic laboratory parameters (ALT, AST, and platelet counts) from routine antenatal blood tests performed before the clinical diagnosis of preeclampsia. The formulas are as follows:  $FIB-4 = (Age \times AST [U/L]) / (Platelet\ count [10^9/L] \times \sqrt{ALT [U/L]})$ ;  $FIB-5 = [albumin \times 0.3 + platelet\ count \times 0.05] - [ALP \times 0.014 + AST/ALT\ ratio \times 6 + 14]$ ;  $APRI = (AST\ level / Upper\ Limit\ of\ Normal\ AST) \times 100 \div Platelet\ count [10^9/L]$ .

### Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 27. Frequency tables and descriptive statistics were used to summarize the data. For normally distributed variables, parametric methods were used. In accordance with parametric methods, the Independent-samples t-test was used to compare measurement values between two independent groups. Nonparametric methods were used for non-normally distributed variables. In accordance with nonparametric methods, the Mann-Whitney U test was used to compare measurement values between two independent groups. To examine the relationships between two qualitative variables, Pearson's chi-square test was performed. To determine the APRI, FIB-4, and FIB-5 values that discriminate disease status, the ROC analysis was used. In statistical analyses, a p-value of  $< 0.05$  was considered statistically significant.

## Results

The groups were similar in terms of sociodemographic characteristics, including age, educational level, average monthly income, and gestational history, as shown in Table I ( $p > 0.05$ ).

**Table I:** Comparison of socio-demographic and maternity characteristics of the groups

Variable	Preeclampsia group (n=84)		Control Group (n=84)		Statistical analysis* Probability
Variable	29.50±5.78	Median [IQR]	28.23±5.34	Median [IQR]	Statistical analysis* Probability
Age (years)	36.42±1.87	30.0 [10.8]	38.56±1.33	27.0 [8.0]	Z=-1.434 p=0.152
Week of gestation	2.90±1.81	36.0 [3.0]	2.63±1.40	39.0 [2.0]	Z=-7.074 <b>p&lt;0.001</b>
Gravida	1.38±1.45	2.5 [3.0]	1.33±1.19	3.0 [1.8]	Z=-0.546 p=0.585
Parity	29.59±5.33	1.0 [2.0]	27.94±4.49	1.0 [2.0]	Z=-0.232 p=0.817
BMI (kg/m <sup>2</sup> )	n	29.0 [7.9]	N	28.0 [6.0]	t=2.153 <b>p=0.033</b>
	28	%	27	%	
<b>Employment status</b>	56		57		
Yes		33.3		32.1	$\chi^2=0.027$
No	17	66.7	18	67.9	p=0.869
<b>Level of education</b>	53		51		
Primary/secondary school	14	20.2	15	21.4	$\chi^2=0.102$
High school		63.1		60.7	p=0.951
University	6	16.7	8	17.9	
<b>Spouse education level</b>	60		59		
Primary/secondary school	18	7.2	17	9.6	$\chi^2=0.323$
High school		71.4		70.2	p=0.851
University	12	21.4	12	20.2	
<b>Average monthly income</b>	45		51		
Below minimum wage	27	14.3	21	14.3	$\chi^2=1.125$
Minimum wage		53.6		60.7	p=0.570
Above minimum wage		32.1		25.0	

\*"Mann-Whitney U" test (Z-table value) statistics were used to compare the measurement values of two independent groups in data that do not have a normal distribution. "Pearson- $\chi^2$ " cross-tabulations were used to examine the relationships of two qualitative variables.

Patients with preeclampsia showed significantly higher ALT, AST, ALP, FIB-4, and APRI values, along with significantly lower FIB-5 scores, platelet counts, and albumin levels compared with the control group ( $p<0.05$ ) (Table II).

The APRI cut-off value for distinguishing patients was  $\geq 0.25$ , with 83.3% sensitivity and 65.5% specificity (AUC=0.827;  $p<0.001$ ). The FIB-4 cut-off value for distinguishing patients was  $\geq 0.665$ , with 82.1% sensitivity and 59.5% specificity (AUC - 0.776;  $p < 0.001$ ). The FIB-5 cut-off value for distinguishing patients was  $\leq 41.773$ , with 56.0% sensitivity and 71.4% specificity (AUC=0.669;  $p<0.001$ ). (Table III).

## Discussion

In this retrospective cohort, APRI and FIB-4 values were higher, and FIB-5 values were lower among women with late-onset preeclampsia compared with normotensive pregnant controls. (Table II) Because these indices were calculated from routine antenatal tests obtained before the clinical diag-

nosis (prediagnostic index sample), our findings should be interpreted as an association rather than as diagnostic or causal evidence. In addition, although ROC analyses suggested that APRI and FIB-4 discriminated case status within this dataset, these cut-offs should not be considered validated screening thresholds without prospective external validation. (Table III)

Given the maternal and fetal morbidity associated with preeclampsia, identifying clinical and biochemical patterns linked to disease presence remains of clinical interest. Preeclampsia is among the major pregnancy-specific disorders associated with abnormal liver function tests and hepatic impairment, alongside hyperemesis gravidarum, HELLP syndrome, intrahepatic cholestasis of pregnancy, and acute fatty liver of pregnancy (6). Liver function may be significantly altered, particularly in women with severe preeclampsia. Consistent with prior literature, hepatic involvement in preeclampsia is variable; while mild to moderate aminotransferase elevations are relatively common, pronounced elevations are generally confined to severe cases, particularly those complicated by HELLP syndrome or acute hepatic injury. In

**Table II:** Comparison of biochemical findings of the groups

Variable	Preeclampsia Group (n=84)		Control Group (n=84)		Statistical Analysis* Probability
Variable	0.38±0.16	Median [IQR]	0.23±0.07	Median [IQR]	Statistical Analysis* Probability
APRI	0.96±0.39	0.3 [0.1]	0.66±0.22	0.2 [0.1]	Z=-7.671 <b>p&lt;0.001</b>
FIB-4	41.86±4.41	0.9 [0.5]	44.02±3.38	0.6 [0.3]	Z=-6.170 <b>p&lt;0.001</b>
FIB-5	24.86±6.95	41.5 [5.2]	17.71±3.46	43.9 [4.9]	Z=-3.788 <b>p&lt;0.001</b>
AST (U/L)	16.31±5.77	24.0 [9.0]	12.69±4.23	17.5 [5.0]	Z=-7.446 <b>p&lt;0.001</b>
ALT (U/L)	201.75±51.32	16.0 [8.8]	228.73±49.38	12.0 [6.0]	Z=-4.100 <b>p&lt;0.001</b>
PLT (×10 <sup>9</sup> /L)	108.12±32.52	197.0 [58.0]	52.39±11.79	225.5 [67.8]	Z=-3.217 <b>p&lt;0.001</b>
ALP (U/L)	32.25±3.44	96.0 [47.3]	34.34±2.36	52.0 [17.8]	Z=-11.099 <b>p&lt;0.001</b>
Albumin (g/dL)		32.0 [5.8]		34.0 [2.0]	Z=-4.288 <b>p&lt;0.001</b>

\*For data without a normal distribution, "Mann-Whitney U" test (Z-table value) statistics were used to compare measurement values between two independent groups

**Table III.** ROC curve for APRI, FIB-4, and FIB-5 levels by disease status

Variable	Area	Standard Error	Probability	AUC 95% G.A. Under	AUC 95% G.A. Top	Cut-off
APRI	0.827	0.031	<0.001	0.765	0.888	≥0.25
FIB-4	0.776	0.035	<0.001	0.706	0.845	≥0.665
FIB-5	0.669	0.042	<0.001	0.588	0.751	≤41.773

their review, Joshi et al. (2010) stated that abnormal liver function tests are due to vasoconstriction in the hepatic vascular bed and are seen in 20-30% of patients, with values up to 10 times the upper limit of normal in preeclamptic patients (9). In their study, Zhang et al. (2022) investigated the association between high-normal liver enzymes and preeclampsia, including 5,685 pregnant women between 9 and 13 weeks of gestation without pre-existing liver disease. They reported that elevated ALT, AST, ALP, and GGT levels were early biochemical markers associated with the subsequent development of preeclampsia (10). Hazari et al. (2014) conducted a similar study with 80 patients (40 preeclamptic and 40 normotensive) and reported that total bilirubin, AST, ALT, ALP, GGT, and LDH levels were significantly increased in mild and severe preeclampsia, whereas total protein and albumin levels were decreased (11). As described in earlier studies, patients with preeclampsia in our cohort had higher transaminase and ALP levels, whereas serum albumin concentrations were notably lower than those of healthy controls (Table II).

In preeclamptic patients, in addition to elevated transaminases, right upper quadrant pain, nausea, vomiting, and,

rarely, hepatic rupture or subcapsular hemorrhage may be observed. Histopathological changes underlying these clinical findings include sinusoidal fibrin deposition, microvesicular fat accumulation, periportal hemorrhage, and, in severe cases, infarction. Various studies have shown that in severe cases, the condition can lead to liver fibrosis. In their prospective study, Frank Wolf et al. (2015) included 32 patients who were diagnosed with preeclampsia between 24 and 41 weeks' gestation and 16 normotensive patients. FibroScan measurements performed by a hepatologist between 1 and 7 days postpartum revealed a statistically significant increase in liver fibrosis in preeclamptic patients (12). Similarly, Ammon et al. (2018) studied liver stiffness and its association with complications during pregnancy. They enrolled 537 pregnant women and concluded that an elevated liver stiffness, especially in the last trimester, appears to be an independent predictor of preeclampsia (13). Although liver fibrosis is typically evaluated by elastography or histopathologic assessment, composite blood-based indices such as APRI, FIB-4, and FIB-5 may remain of exploratory interest in preeclampsia because they incorporate routine laboratory parameters

that are altered in this syndrome. In our cohort, APRI and FIB-4 were higher, and FIB-5 was lower in women with late-onset preeclampsia (Table II). However, these differences should not be interpreted as evidence of specific hepatic fibrosis. Rather, they may reflect hepatic involvement and the broader systemic burden of disease. Accordingly, these indices should be regarded as adjunctive, hypothesis-generating markers rather than diagnostic biomarkers for preeclampsia. In their study, Şaşmaz et al. (2020) investigated the role of APRI score in the diagnosis of HELLP syndrome, and concluded that APRI was a better predictor than AST score in predicting HELLP. The study found that when the APRI cut-off was set at 0.339, the sensitivity was 82.6% and the specificity was 87.6% (14). In their study, Zhaoqi et al. (2024) determined the markers that can be used to predict the progression of gestational hypertension to preeclampsia when complicated by HELLP syndrome, and concluded that APRI may be one of the markers associated with progression to a more severe hypertensive phenotype (15).

In their study, Gezer et al. (2025) investigated the role of non-invasive liver fibrosis markers in predicting adverse maternal and perinatal outcomes in patients with hypertensive disorders of pregnancy and found significantly higher FIB-4 and APRI scores in affected patients. They concluded that although these indices cannot serve as direct markers of histological fibrosis, they may be useful for detecting early and subclinical hepatic changes (16).

**Limitations:** Our study is limited by its retrospective design, relatively small sample size, and the inclusion of a socioeconomically homogeneous single-center cohort. This may limit the generalizability of the findings to populations with different demographic and socioeconomic profiles. However, the observed associations support further prospective evaluation of APRI, FIB-4, and FIB-5 as adjunctive markers of hepatic involvement in late-onset preeclampsia.

Although most socio-demographic and obstetric characteristics were comparable between groups, a statistically significant difference in body mass index (BMI) was observed. Given that BMI is a known risk factor for preeclampsia and may also influence metabolic and inflammatory parameters, this imbalance introduces potential residual confounding. Additionally, despite matching efforts, gestational age differed significantly between groups, which may have affected laboratory and clinical comparisons due to physiological changes across late pregnancy. These differences should be considered when interpreting group comparisons and subsequent analyses.

## Conclusion

In conclusion, APRI, FIB-4, and FIB-5 were associated with late-onset preeclampsia in our retrospective cohort. Given that these indices are inexpensive and readily obtain-

able, they may serve as adjunct hypothesis-generating markers to characterize hepatic involvement in preeclampsia and inform future risk models. Prospective, large-scale studies with external validation are required before these indices can be recommended for screening or clinical prediction.

### Declarations

*Ethics approval and consent to participate:* This study was approved by the Ministry of Health of the Republic of Türkiye, Sağlık Bilimleri University, Ankara Atatürk Sanatoryum Training and Research Hospital Approval and Ethics committee, under approval number (Approval No: 2024/220; Date:12.02.2025). Written informed consent was obtained from all participants.

*Consent for publication:* Written informed consent for publication of the data included in this study was obtained from all participants.

*Competing interests:* The authors declare that they have no competing interests.

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## References

1. Tyas BD, Lestari P, Aldika Akbar MI. Maternal perinatal outcomes related to advanced maternal age in preeclampsia pregnant women. *J Family Reprod Health.* 2019;13(4): 191-200. PMID: 32518569, PMCID: PMC7264866.
2. Zhang H, Li X, Zhang T, Zhou Q, Zhang C. Establishment and validation of a predictive model of preeclampsia based on transcriptional signatures of 43 genes in decidua basalis and peripheral blood. *BMC Bioinformatics.* 2022;23(1):527. Doi: 10.1186/s12859-022-05086-y. PMID: 36476092, PMCID: PMC9730617.
3. Mancia G, Hall JE. Introduction to a compendium on the pathophysiology and treatment of hypertension. *Circ Res.* 2019;124(7):967-8. Doi: 10.1161/CIRCRESAHA.119.314953. PMID: 30920925.
4. Melchiorre K, Giorgione V, Thilaganathan B. The placenta and preeclampsia: villain or victim? *Am J Obstet*

- Gynecol. 2022;226(2S):S954-S962. Doi: 10.1016/j.ajog.2020.10.024. PMID: 33771361.
5. Jung E, Romero R, Yeo L, Gomez-Lopez N, Chaemsaitong P, Jaovisidha A, et al. The etiology of preeclampsia. *Am J Obstet Gynecol.* 2022;226(2S):S844-S866. Doi: 10.1016/j.ajog.2021.11.1356. PMID: 35177222, PMCID: PMC8988238.
  6. Lee NM, Brady CW. Liver disease in pregnancy. *World J Gastroenterol.* 2009;15(8):897-906. Doi: 10.3748/wjg.15.897. PMID: 19248187, PMCID: PMC2653411.
  7. Deng H, Qi X, Guo X. Diagnostic Accuracy of APRI, AAR, FIB-4, FI, King, Lok, Forns, and FibroIndex scores in predicting the presence of esophageal varices in liver cirrhosis: a systematic review and meta-analysis. *Medicine (Baltimore).* 2015;94(42):e1795. Doi: 10.1097/MD.0000000000001795. Erratum in: *Medicine (Baltimore).* 2016;95(5):1. Erratum in: *Medicine (Baltimore).* 2016;95(5):e615b. Doi: 10.1097/01.md.0000480464.27661.5b. PMID: 26496312; PMCID: PMC4620760.
  8. Gestational hypertension and preeclampsia: ACOG Practice Bulletin, Number 222. *Obstet Gynecol.* 2020;135(6):e237-e260. Doi: 10.1097/AOG.00000000000003891. PMID: 32443079.
  9. Joshi D, James A, Quaglia A, Westbrook RH, Heneghan MA. Liver disease in pregnancy. *Lancet.* 2010;375(9714):594-605. Doi: 10.1016/S0140-6736(09)61495-1. PMID: 20159293.
  10. Zhang Y, Sheng C, Wang D, Chen X, Jiang Y, Dou Y, et al. High-normal liver enzyme levels in early pregnancy predispose the risk of gestational hypertension and preeclampsia: A prospective cohort study. *Front Cardiovasc Med.* 2022;9:963957. Doi: 10.3389/fcvm.2022.963957. PMID: 36172586, PMCID: PMC9510982.
  11. Hazari NR, Hatolkar VS, Munde SM. Study of serum hepatic enzymes in preeclampsia. *International Journal of Current Medical and Applied Sciences.* 2014;2(1):01-08.
  12. Frank Wolf M, Peleg D, Kariv Silberstein N, Assy N, Djibre A, Ben-Shachar I. Correlation between changes in liver stiffness and preeclampsia as shown by transient elastography. *Hypertens Pregnancy.* 2016;35(4):536-41. Doi: 10.1080/10641955.2016.1197934. PMID: 27391875.
  13. Ammon FJ, Kohlhaas A, Elshaarawy O, Mueller J, Bruckner T, Sohn C, et al. Liver stiffness reversibly increases during pregnancy and independently predicts preeclampsia. *World J Gastroenterol.* 2018;24(38):4393-4402. Doi: 10.3748/wjg.v24.i38.4393. PMID: 30344423, PMCID: PMC6189842.
  14. Şaşmaz Mİ, Ayvaz MA, Dülger AC, Kuday Kaykısız EK, Güven R. Aspartate-aminotransferase to platelet ratio index score for predicting HELLP syndrome. *Am J Emerg Med.* 2020;38(3):459-62. Doi: 10.1016/j.ajem.2019.02.014. PMID: 30777375.
  15. Li Z, Dai Y, Yun L, Guo W. A prediction model for the progression from gestational hypertension to pre-eclampsia complicated with HELLP syndrome. *Int J Gynaecol Obstet.* 2024;165(3):1002-12. Doi: 10.1002/ijgo.15274. PMID: 38018274.
  16. Gezer M, Taşdemir Ü, Özdemir ME, Yiğit S, Cambaztepe B, Demirci O. Liver fibrosis markers as predictors of adverse outcomes in pregnancy-related hypertensive disorders. *J Perinat Med.* 2025;53(9):1230-7. Doi: 10.1515/jpm-2025-0190. PMID: 40967788.