International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Preciado ES et al. Int J Reprod Contracept Obstet Gynecol. 2026 Dec;15(12):xxx-xxx
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20264237

Original Research Article

Predictive and prognostic performance of sFlt-1/PlGF ratio for preeclampsia in women with singleton pregnancy in a tertiary care institution in Mexico

Etna Sahagún Preciado*, Alfonso López Lerma, Diana L. Olmedo Mendez

Department of Gynecology and Obstetrics, Instituto Mexicano del Seguro Social, Centro Médico Nacional de Occidente, Guadalajara, Jalisco, Mexico

Received: 13 October 2026
Revised: 01 November 2026
Accepted: 02 November 2026

***Correspondence:**
Dr. Etna Sahagún Preciado,
E-mail: etnasahagun@icloud.com

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ABSTRACT

Background: Preeclampsia is a complex hypertensive disorder of pregnancy characterized by placental dysfunction and maternal multiorgan injury. Circulating angiogenic and antiangiogenic biomarkers may improve risk stratification and prediction of adverse outcomes.
Methods: This retrospective cohort study evaluated the predictive and prognostic performance of the soluble fms-like tyrosine kinase-1 (sFlt-1)/placental growth factor (PlGF) ratio in patients with singleton pregnancy from 24 to 36 + 6 weeks who had clinical or biochemical suspicion of preeclampsia. The sFlt-1/PlGF ratio was calculated from maternal serum concentrations measurements of both biomarkers in 144 women at the Hospital of Gynecology and Obstetrics, Unidad Medica de Alta Especialidad, Centro Medico Nacional de Occidente in Guadalajara, Jalisco, Mexico from April 2024-January 2026.
Results: Among 144 women included in the final analysis, 57 (39.5%) developed preeclampsia; 39 (68.4%) had early-onset and 18 (31.6%) late-onset preeclampsia. The median sFlt-1/PlGF ratio was higher in women who developed the disease than in those in the control group (98.65 vs 11.86; $p<0.001$). The diagnostic performance of the sFlt-1/PlGF ratio using a cut-off value≥38 to predict preeclampsia yielded a sensitivity of 73.68% (95% CI: 61.02-83.35) and specificity of 81.61% (95% CI: 72.19-88.35). Higher sFlt-1/PlGF ratios were also observed among pregnancies with adverse maternal or perinatal outcomes.
Conclusions: The sFlt-1/PlGF ratio demonstrated good discriminatory performance for preeclampsia and association with adverse maternal and perinatal outcomes in our cohort. The sFlt-1/PlGF ratio correlates accurately with prognostic value.

Keywords: Preeclampsia, Soluble fms-like tyrosine kinase-1, Placental growth factor, sFlt-1/PlGF ratio, Maternal outcomes, Neonatal outcomes

INTRODUCTION

Preeclampsia is a multisystem hypertensive disorder of the pregnancy involving maternal and placental pathways and one of the most common obstetric complications affecting approximately 3-8% of pregnancies worldwide, representing a major cause of pregnancy-related maternal morbidity and mortality, particularly in low- and middle-income countries.[1]

The clinical presentation of preeclampsia is heterogeneous. New-onset hypertension and proteinuria are its hallmark features; however, preeclampsia may also occur in the absence of proteinuria when renal, hepatic,

hematologic, pulmonary, or neurologic organ injury is present.[2]

According to the timing of onset, preeclampsia may be classified as early-onset when it occurs before 34 weeks of gestation and late-onset if it occurs at or after 34 weeks. These subtypes differ in their pathophysiology, clinical course, and maternal and fetal outcomes.[3]

The pathophysiology is complex and multifactorial, closely linked to placental dysfunction and angiogenic imbalance. Optimal placental perfusion is essential for normal pregnancy progression and depends on the development of a regulated and coordinated vascular network involving multiple cell types and endothelial growth factors, followed by gradual invasion of the basal decidua and remodeling of the endothelium and tunica media of the uterine arteries, transforming them into highly compliant, low-resistance vessels, thereby increasing uteroplacental perfusion.[4-8]

The etiology of preeclampsia remains unknown.[9] Angiogenic factors play key roles in vascular cell proliferation, migration, stabilization, and permeability. In contrast, antiangiogenic factors regulate angiogenic homeostasis during pregnancy through antagonistic effects, their placental expression is increased in preeclampsia. The resulting elevation of the sFlt-1/PlGF ratio has emerged as a biomarker associated with the disease.[10-11]

PlGF is a 45-50-kDa dimeric glycoprotein encoded by the PlGF gene located on chromosome 14q24. PlGF shares 53% homology with vascular endothelial growth factor (VEGF). VEGF binds with high affinity to sFlt-1 receptors, promoting branching angiogenesis, whereas PlGF binds predominantly to sFlt-1 and contributes to non-branching angiogenesis.[12]

sFlt-1 is a splice variant of VEGF receptor-1 (VEGFR-1) located on chromosome 13q12 that encodes a 186-kDa glycoprotein. It lacks transmembrane and intracellular domains and therefore functions as a soluble protein that antagonizes proangiogenic factors by regulating their activity.[13]

The imbalance between proangiogenic and antiangiogenic factors results in a net antiangiogenic state that favors the development of preeclampsia. Multiple studies have demonstrated the clinical utility of the sFlt-1/PlGF ratio for the diagnosis of preeclampsia and the identification of adverse maternal and fetal outcomes.

Regardless of gestational age, a sFlt-1/PlGF ratio <38 has demonstrated high sensitivity and specificity for ruling out preeclampsia in the subsequent week, whereas a ratio ≥38 is associated with an increased probability of developing preeclampsia or another manifestation of placental dysfunction within the following four weeks. Accordingly, the ratio may serve as an adjunct to clinical assessment and as a predictor of short-term disease development.[14]

Therefore, the aim of this study was to evaluate the diagnostic and prognostic performance of the sFlt-1/PlGF ratio in pregnant women with suspected preeclampsia, with particular emphasis on its ability to discriminate preeclampsia and its association with maternal and fetal adverse outcomes.

METHODS

This was a retrospective observational cohort study conducted among pregnant women with singleton pregnancy at 24+0 to 36+6 weeks at the Hospital of Gynecology and Obstetrics, Unidad Medica de Alta Especialidad, Centro Medico Nacional de Occidente, a tertiary care obstetric center in Mexico, from April 2024 to January 2026. Medical records were reviewed to obtain available clinical, obstetric, laboratory and angiogenic biomarkers measurements data.

A total of 144 patients were included in the final analysis. Preeclampsia was diagnosed according to the established clinical criteria of the American College of Obstetricians and Gynecologists (ACOG), based on new-onset hypertension after 20 weeks of gestation and/or features of severe disease, as documented on medical records.

Participants were eligible if they met all the following criteria: maternal age ≥18 years, singleton pregnancy between 24+0 and 36+6 weeks of gestation, clinical or biochemical suspicion of preeclampsia and serum measurements of both sFlt-1 and placental growth factor (PlGF).

Patients were excluded if the sFlt-1/PlGF ratio could not be calculated or if clinical or laboratory kay data were incomplete. After exclusion of incomplete cases, the 144 pregnant women were stratified into a preeclampsia and non-preeclampsia group.

Maternal demographic and clinical characteristics collected included maternal age, height, weight, educational attainment and medical conditions. Obstetrics variables included parity, gestational age at delivery and pregnancy-related comorbidities distinct from preeclampsia.

Maternal and perinatal complications included clinically complications documented in medical records.

Measurement of angiogenic biomarkers

Blood sampling of the maternal serum levels of sFlt-1 and PlGF was performed at the time of clinical evaluation for suspected hypertensive disorder of pregnancy based on clinical or biochemical criteria, including the women in whom preeclampsia subsequently was ruled out. All biomarkers' measurements were obtained before delivery.

Measurements were performed using standardized, validated immunoassays in external laboratories, in accordance with the manufacturer's standards.

Statistical analyses

Data were collected into a Microsoft Excel database and subsequently statistical analyzed using IBM SPSS Statistics (version 26; IBM Corp., Armonk, NY, USA).

Continuous variables are presented as mean±standard deviation or median (interquartile range), as appropriate. Categorical variables are presented as counts and percentages. Between groups comparisons of continuous variables were conducted using the Mann-Whitney U test. The magnitude of between-group differences in the sFlt-1/PlGF ratio was quantified using Cohen's d effect size.

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance of the sFlt-1/PlGF ratio, with calculation of the area under the curve (AUC) and corresponding 95% confidence intervals. The optimal population-derived cut-off was determined using the Youden index. Statistical significance was defined as a two-sided $p<0.05$.

RESULTS

A total of 144 patients meeting the selection criteria were included in the study conducted between April 2024 and January 2026 at the Hospital of Gynecology and Obstetrics, Unidad Medica de Alta Especialidad, Centro Medico Nacional de Occidente in Guadalajara, Jalisco, Mexico.

Patients with suspected preeclampsia were classified into two groups according to clinical outcome: those who developed preeclampsia and those who did not. Baseline clinical characteristics are displayed in Table 1.

The mean maternal age was 31.22±5.27 years. In the preeclampsia group, the median age was 30 years (IQR, 28-33), whereas in the non-preeclampsia group was 32 years (IQR, 27-36). Maternal age did not differ significantly between groups (U=2181; p=0.266).

Regarding obstetric history, 98 patients were nulliparous and 46 were multiparous.

The mean weight was 74.8±13.6 kg, while the mean height was 1.59±0.07 m, with a mean body mass index of 31.47±6.07 kg/m². Obesity was present in 59.44% pregnant women (n=85).

The most frequent educational level was high school education in 56 participants (38.9%), followed by middle school in 38 (26.4%), a bachelor's degree in 34 (23.6%), elementary education in 12 (8.3%), and postgraduate education in 4 (2.8%) participants.

Development of preeclampsia

Of the 144 patients included in the study, 57 participants (39.5%) developed preeclampsia. Regarding timing of diagnosis, 39 cases (68.4%) were classified as early-onset preeclampsia and 18 (31.6%) as late-onset preeclampsia.

Gestational age at the time of angiogenic (PlGF) and antiangiogenic (sFlt-1) biomarker measurement differed significantly between groups and was lower among patients subsequently diagnosed with preeclampsia. In the preeclampsia group, mean gestational age at biomarkers measurement was 32±3.1 weeks, with a median of 32.6 weeks of gestation (IQR, 30-34.2), whereas in the non-preeclampsia control group mean gestational age was 34±2.2 weeks, with a median of 34.2 weeks (IQR, 33.1-35.6) (U=1484.5; $p<0.001$).

Risk factors for preeclampsia

Risk factors identified in the study population included chronic hypertension in 62 patients (43.4%), diabetes mellitus in 47 patients (32.6%), nulliparity in 45 patients (31.2%), hypothyroidism in 24 patients (16.6%), smoking in 19 patients (13.1%), chronic kidney disease in 15 patients (10.4%), autoimmune disease in 6 patients (4.1%), and thrombophilia in 3 patients (2%).

sFlt-1/PlGF ratio in the study group

The most widely accepted clinical cut-off of the sFlt-1/PlGF ratio is ≥38, based on previous clinical randomized studies and meta-analyses.

Among the 57 patients who developed preeclampsia, 73.7% (n=42) had a sFlt-1/PlGF ratio ≥38, whereas 26.3% (n=15) had a ratio below that cut-off despite developing the disease. Conversely, of the 87 patients in the control group in whom preeclampsia was ruled out, 18.3% (n=16) had a ratio ≥38, while 81.7% (n=71) demonstrated values below the cut-off (Figure 1).

The sFlt-1/PlGF ratio was significantly higher in patients who developed preeclampsia than in those in whom the diagnosis was excluded. In the preeclampsia group (n=57), the median ratio was 98.65 (IQR, 37.54-183.45), compared with 11.86 (IQR, 7.27-30.33) in the non-preeclampsia group (n=87) (U=4109; $p<0.001$).

Using a cutoff of ≥38, the diagnostic performance of the sFlt-1/PlGF ratio for predicting the disease showed a sensitivity of 73.68% (95% CI: 61.02-83.35) and specificity of 81.61% (95% CI: 72.19-88.35), with a positive predictive value of 72.41% (95% CI: 59.79-82.25) and a negative predictive value of 82.56% (95% CI: 73.20-89.14) (Figure 2).

A significant association was also observed between a sFlt-1/PlGF ratio ≥38 and the development of preeclampsia ($\chi^2=31.87$; $p<0.001$). An elevated ratio was

associated with an odds ratio of 12.43 (95% CI: 5.62-27.48).

Diagnostic performance of the sFlt-1/PlGF ratio in the study population

The prespecified clinical cut-off of 38 was evaluated alongside an exploratory population-derived cut-off identified by ROC curve analysis.

In our population, the optimal sFlt-1/PlGF cut-off for predicting preeclampsia was 55.67, with an AUC of 0.839 (95% CI: 0.762-0.910) (Figure 3).

At this cut-off, sensitivity was 70.7% (95% CI: 57.3-81.9%) and specificity was 90.7% (95% CI: 82.5-95.9%) (Figure 4).

The positive predictive value was 83.7% (95% CI: 70.3-92.7%), and the negative predictive value was 82.6%. Overall accuracy was 82.6% (95% CI: 75.5-88.4%). The Youden index was 0.614 (95% CI: 0.520-0.759).

According to gestational age, the optimal cut-off for early-onset preeclampsia was 55.73 (95% CI: 30.15-92.72), with 75% sensitivity, 87% specificity, and an AUC of 0.83 (95% CI: 0.725-0.922).

For late-onset preeclampsia, the cut-off was 32.44 (95% CI: 25.47-98.65), with sensitivity of 77.8%, specificity of 83.3%, and an AUC of 0.82 (95% CI: 0.673-0.942) (Figure 5).

The population-derived sFlt-1/PlGF threshold should not replace established clinical specific cut-offs, nor should be interpreted as direct clinical decision thresholds.

Gestational age

The association between gestational age and sFlt-1/PlGF ratio yielded a Pearson correlation coefficient of -0.175 (95% CI: -0.329 to -0.012; p=0.036) (Figure 6). As a complementary analysis, the Spearman correlation yielded a coefficient of -0.038 (p=0.649).

Simple linear regression showed that for each one-unit increase in the sFlt-1/PlGF ratio, gestational age decreased by an average of 0.0034 weeks (β=-0.00339; 95% CI: -0.00656 to -0.00022; p=0.036), with an R^2 of 0.031, corresponding to 3.1% of the observed variability.

Although a statistically significant inverse association was observed in the Pearson analysis, the low coefficient of determination indicates limited explanatory capacity of the ratio for gestational age at delivery.

Maternal complications

Amid patients with sFlt-1/PlGF ratio ≥38, maternal complications included 2 cases of placental abruption (3.4%), 9 cases of impaired renal function (15.5%), and 12 cases of abnormal placentation within the placenta accreta spectrum (20.7%).

In addition, 33 patients (56.89%) developed preeclampsia with severe features.

Preeclampsia with severe features

Of the 57 patients who developed preeclampsia, 33 developed preeclampsia with severe features; 27 (81.8%) diagnosed before 34 weeks of gestation and 6 (18.2%) after 34 weeks of gestation.

The principal diagnostic criterion for preeclampsia with severe features was severe-range blood pressure in 24 patients (72.7%), followed by laboratory abnormalities in 7 patients (21.3%) and findings suggestive of vasospasm in 2 patients (6%).

Among biochemical abnormalities, elevated transaminases were the most frequent finding (48.5%, n=16), followed by thrombocytopenia (33.3%, n=11) and elevated serum creatinine levels (30.3%, n=10).

Adverse fetal outcomes

On the other hand, in patients with sFlt-1/PlGF ratio ≥38, the most frequent fetal complication was fetal growth restriction (FGR), observed in 18 cases (31%). Stage I/IV FGR predominated (50%, n=9), followed by stage II/IV (38.5%, n=7) and stage III/IV (11.1%, n=2). No cases of stage IV/IV FGR were observed.

Oligohydramnios was documented in 6 cases (10.3%; 95% CI: 3.9-21.2%).

Table 1: Clinical characteristics of the participants.

Characteristics	Preeclampsia, (n=57)	No preeclampsia, (n=87)	P value
Maternal age (in years)	30.61±4.85	31.61±5.49	0.256
Body mass index (kg/m²)	31.61±6.40	31.48±5.93	0.905
Systolic blood pressure (mmHg)	164.77±24.88	127.82±14.84	<0.001
Diastolic blood pressure (mmHg)	100.39±17.73	78.92±10.69	<0.001

*Results are presented as mean±SD. P<0.05 is considered significant.

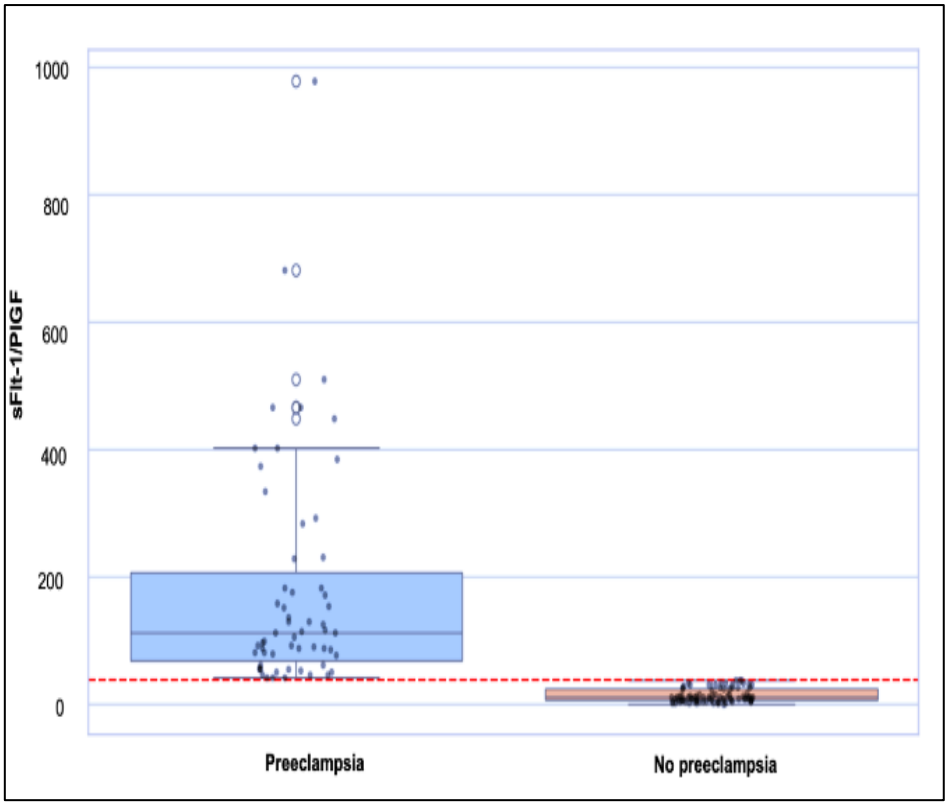


Figure 1: Distribution of the sFlt-1/PlGF ratio using a 38-cut-off value and clinical outcome.

*Most patients without preeclampsia had values below the cut-off, whereas values above this threshold predominated among patients who developed preeclampsia.

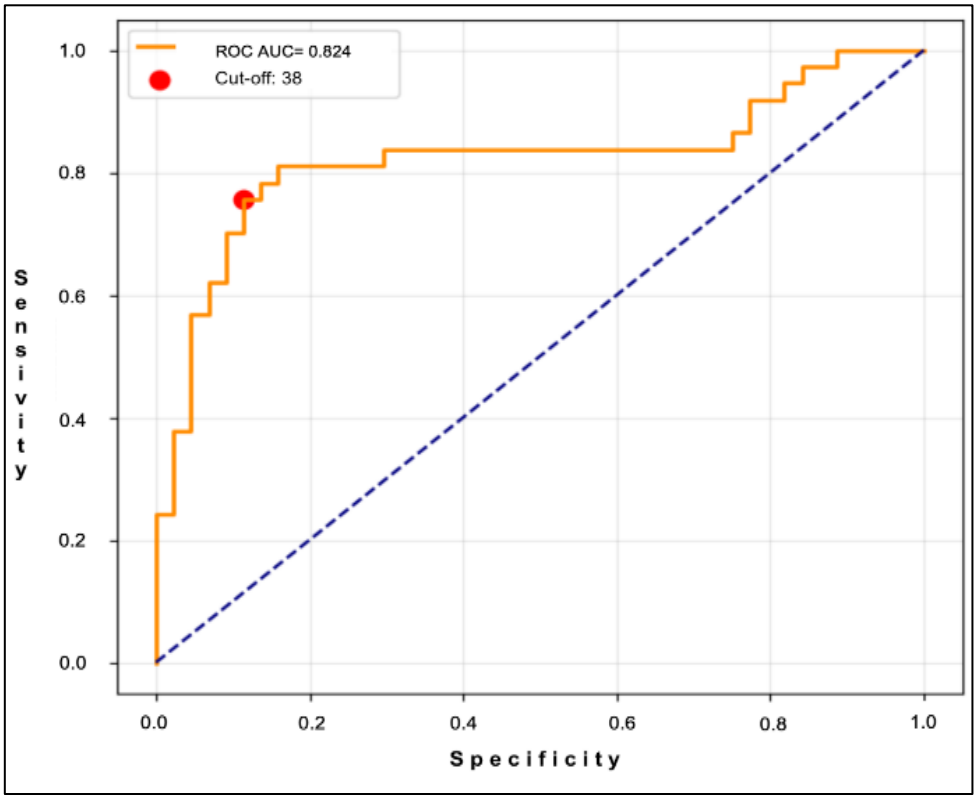


Figure 2: ROC curve for the sFlt-1/PlGF ratio using a cut-off value of 38.

*The biomarker demonstrated good diagnostic performance (AUC=0.824).

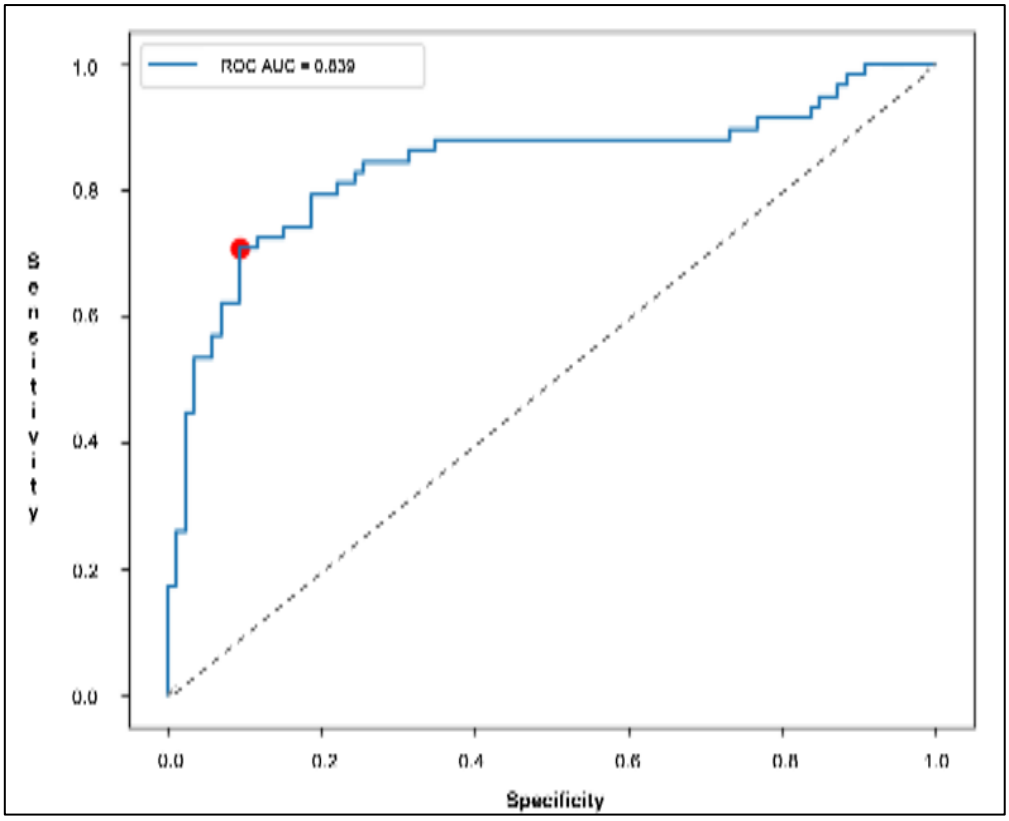


Figure 3: ROC curve for the sFlt-1/PlGF ratio using a cutoff value of 55.67.

*The area under the curve (AUC=0.839) demonstrates good discriminatory performance of the biomarker.

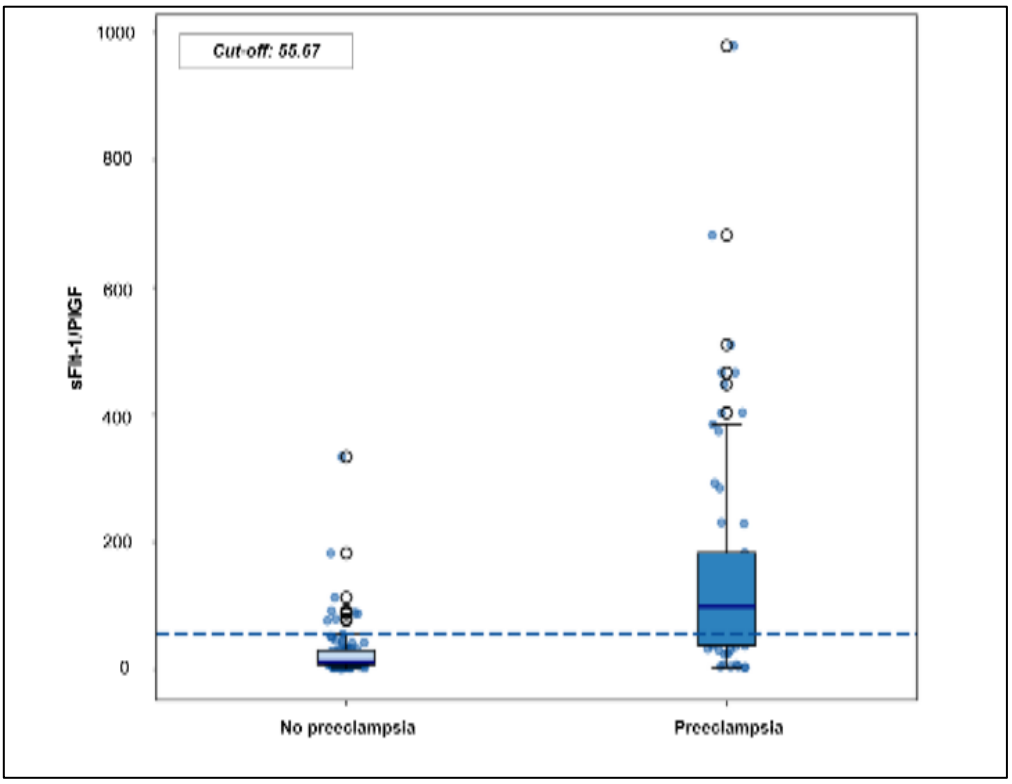


Figure 4: Distribution of the sFlt-1/PlGF ratio according to the cut-off value of 55.67.

*The dashed line represents the cut-off value obtained in the study (55.67), showing that most patients with preeclampsia had values above this threshold.

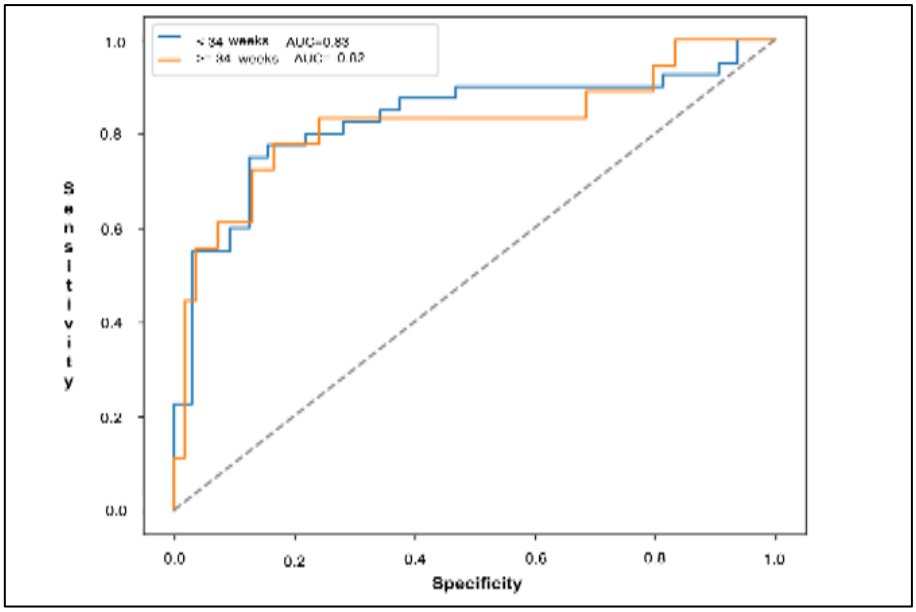


Figure 5: ROC curves for the sFlt-1/PlGF ratio in the study population according to early and late-onset preeclampsia.

*The minimal difference between the two curves indicates that the ratio maintains good discriminatory performance with similar diagnostic performance in pregnancies <34 and ≥ 34 weeks of gestation.

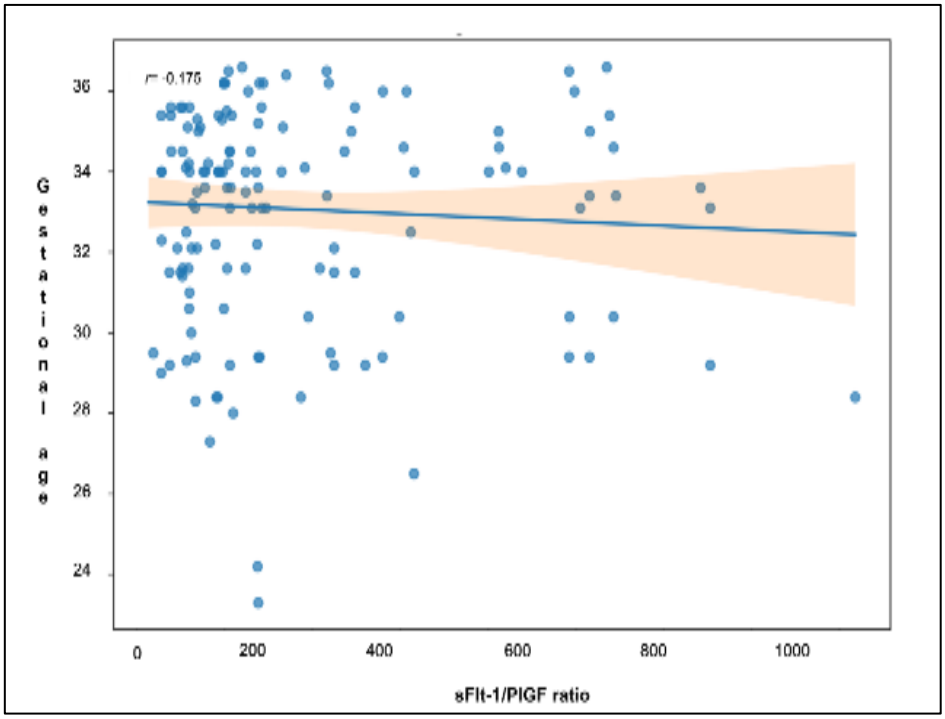


Figure 6: Correlation between gestational age and sFlt-1/PlGF ratio.

*A weak inverse correlation was observed (r=-0.175), indicating that higher sFlt-1/PlGF ratio values tend to be associated with lower gestational age at the time of delivery.

DISCUSSION

Abnormal circulating concentrations of sFlt-1/PlGF are present in preeclampsia even before the clinical onset and remain altered throughout the course of the disease; consequently, both biomarkers have emerged as potential clinical tool for risk assessment.

Given the pathophysiology of preeclampsia, in which circulating sFlt-1 levels increase while PlGF levels decrease, the sFlt-1/PlGF ratio may provide better risk

discrimination than either biomarker alone. The sFlt-1/PlGF ratio may reflect maternal endothelial dysfunction but also act as an indirect marker of placental insufficiency and fetal compromise.[15]

The prediction of short-term outcome in pregnant women with suspected preeclampsia (PROGNOSIS) study was an observational, prospective, multicenter study conducted in 14 countries that evaluated the use of sFlt-1/PlGF ratio to predict short-term development of preeclampsia. A cutoff <38 ruled out the disease within one week, with a negative predictive value of 99.3% (95% CI: 97.9-99.9), whereas a value ≥38 predicted development of preeclampsia in the following four weeks, with a positive predictive value of 36.7% and sensitivity of 66.2%.[16]

In our study, diagnostic performance of the sFlt-1/PlGF ratio using a cut-off value ≥38 to predict preeclampsia yielded a sensitivity of 73.68% (95% CI: 61.02-83.35) and specificity of 81.61% (95% CI: 72.19-88.35). Also, the results showed that higher sFlt-1/PlGF ratios were associated with lower gestational age at obstetric delivery and demonstrated good discriminatory performance for identifying patients at increased risk of developing preeclampsia.

Similar findings were reported in a study of 1,149 patients in which sFlt-1/PlGF cut-offs were defined according to gestational age. A sFlt-1/PlGF ratio >85 at 20+0 to 33+6 weeks of gestation and >100 from 34 weeks onward was strongly associated with the development of preeclampsia.[17]

Reddy et al reported that women who developed maternal complications had higher median sFlt-1 concentrations (7471 pg/ml vs. 5131 pg/ml), lower PlGF concentrations (27.2 pg/ml vs. 76.3 pg/ml), and a higher sFlt-1/PlGF ratio (445.4 vs. 74.4).[18] Similarly, Tătaru-Copos et al reported that higher sFlt-1/PlGF ratios were associated with lower gestational age at delivery, a higher incidence of low birth weight, lower Apgar scores, and a greater likelihood of neonatal intensive care unit admission.[19] In our population, fetal growth restriction was the most frequent fetal complication.

Non relevant differences were identified between the risk factors, maternal clinical characteristics and biochemical findings observed in the study population and those reported in the international literature.[20]

Integration of the sFlt-1/PlGF ratio may add value to the clinical dilemma surrounding preeclampsia. Based on obstetric history and individual risk factors, targeted follow-up complemented by measurement of the sFlt-1/PlGF ratio may be particularly useful in patients at increased risk of developing preeclampsia, supporting more precise clinical decision making, optimize resource utilization, and guide management strategies with potential prognostic implications, especially in low- and middle-income settings. Different risk profiles, biomarkers associated with adverse events, and ratio cut-offs have been established across clinical studies.[21]

Limitations of this study should be acknowledged. First, its retrospective design may limit the availability and standardization of data collection. Second, the biomarker measurements were not being standardized according to gestational age at disease suspicion or presentation.

An additional methodological limitation was the lack of on-site facilities for biomarker processing at our institution**.** Consequently, samples had to be referred to external laboratories contracted through a public procurement process, leading to substantial variability in laboratory turnaround times, making it impossible to establish temporal associations between biomarker levels and disease progression or establishing time-dependent predictive values.

Considering both maternal and fetal outcomes, the sFlt-1/PlGF ratio may have a dual application: as a biomarker for preeclampsia identification, and as a marker for maternal and fetal risk stratification. The findings in this study should be interpreted considering the retrospective, single-center design and the sample size. The population-derived cut-off identified in this cohort requires external validation before it can be considered for broader clinical application.

ACKNOWLEDGEMENTS

The authors would like to acknowledge the institutional support provided by the Gynecology and Obstetrics Hospital, Unidad Medica de Alta Especialidad, Centro Médico Nacional de Occidente of the Instituto Mexicano del Seguro Social.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee Health Research Committee 1310 of the Hospital of Ginecologia y Obstetricia, Unidad Medica de Alta Especialidad, Centro Médico Nacional de Occidente, under authorization SIRELCIS R-2024-1310-051.

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Cite this article as: Preciado ES, Lerma AL, Mendez DLO. Predictive and prognostic performance of sFlt-1/PlGF ratio for preeclampsia in women with singleton pregnancy in a tertiary care institution in Mexico. Int J Reprod Contracept Obstet Gynecol 2026;15:xxx-xx.