Application of the sFlt-1/PlGF Ratio in the Prediction and Management of Preeclampsia: Case Report in a High-Risk Pregnant Woman

Authors: German Andres Guevara Lizarazo, Laura Juliana Posso Perea, Joshuart David Pascuas Varela, Daniela Lopez Londoño, Marbin Yulieth Alvarez Ramirez, Valentina Dehaquiz Arango

Submitted: 27. March 2026
Published: 30. March 2026
Volume: 13
Issue: 2
Affiliation: Universidad Autonoma de Bucaramanga
Languages: English
Keywords: Angiogenic biomarkers; sFlt-1, PlGF, sFlt-1/PlGF ratio; Preeclampsia, Spiral arteries; VEGF; Fetal Doppler ultrasonography
Categories: Medicine
DOI: 10.17160/josha.13.2.1135

Abstract:

Preeclampsia is one of the leading causes of maternal and perinatal morbidity and mortality. Its diagnosis is based on clinical and paraclinical criteria, although in high-risk patients it can be difficult to identify at early stages. Angiogenic biomarkers, such as the sFlt-1/PlGF ratio, have emerged as highly useful complementary tools. We present the case of a 26-year-old pregnant woman with type 1 diabetes mellitus, who during the course of her pregnancy developed elevated blood pressure values and an increased sFlt-1/PlGF ratio, which guided the diagnosis of preeclampsia without severe features. Serial monitoring of biomarkers allowed close follow-up of disease progression and anticipation of its transition to severe preeclampsia, which prompted termination of pregnancy by cesarean section with favorable neonatal

JOSHA Journal of Science, Humanities and Arts

March 2026 Volume 13, Issue 2

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German Andres Guevara Lizarazo, Laura Juliana Posso Perea[1], Joshuart David Pascuas Varela[2], Daniela Lopez Londoño[3], Marbin Yulieth Alvarez Ramirez[4], Valentina Dehaquiz Arango[5]

andres1guevara@hotmail.com

Autonomous University of Bucaramanga, Bucaramanga, Colombia.

Abstract

Preeclampsia is one of the leading causes of maternal and perinatal morbidity and mortality. Its diagnosis is based on clinical and paraclinical criteria, although in high-risk patients it can be difficult to identify at early stages. Angiogenic biomarkers, such as the sFlt-1/PlGF ratio, have emerged as highly useful complementary tools. We present the case of a 26-year-old pregnant woman with type 1 diabetes mellitus, who during the course of her pregnancy developed elevated blood pressure values and an increased sFlt-1/PlGF ratio, which guided the diagnosis of preeclampsia without severe features. Serial monitoring of biomarkers allowed close follow-up of disease progression and anticipation of its transition to severe preeclampsia, which prompted termination of pregnancy by cesarean section with favorable neonatal outcomes. The sFlt-1/PlGF ratio is a valuable diagnostic and prognostic tool in preeclampsia, especially in high-risk pregnancies. Its rational use, complemented with clinical assessment and ultrasound, optimizes decision-making and improves maternal and fetal outcomes.

[1] Free University of Colombia, Cali, Colombia.
[2] University of Magdalena, Santa Marta, Colombia.
[3] Free University of Colombia, Cali, Colombia.
[4] University of Santander, Bucaramanga, Colombia.
[5] Free University of Colombia, Cali, Colombia.

Keywords: Angiogenic Biomarkers; sFlt-1; PlGF; sFlt-1/PlGF ratio; Preeclampsia; Spiral Arteries; VEGF; Fetal Doppler Ultrasonography.

Resumen

La preeclampsia es una de las principales causas de morbimortalidad materna y perinatal. Su diagnóstico se basa en criterios clínicos y paraclínicos, aunque en pacientes de alto riesgo puede ser difícil identificar en etapas tempranas. Los biomarcadores angiogénicos, como la relación sFlt-1/PlGF, han emergido como herramientas complementarias de gran utilidad. Se presenta el caso de una gestante de 26 años con diabetes mellitus tipo 1, quien durante la evolución de su embarazo desarrolló cifras tensionales elevadas y relación sFlt-1/PlGF incrementada, lo que orientó al diagnóstico de preeclampsia sin criterios de severidad. El seguimiento seriado de biomarcadores permitió vigilar la progresión de la enfermedad y anticipar su transición a preeclampsia severa, lo que motivó la terminación del embarazo mediante cesárea con resultados neonatales favorables. El cociente sFlt-1/PlGF constituye un apoyo diagnóstico y pronóstico en preeclampsia, especialmente en gestantes de alto riesgo. Su uso racional, complementado con la clínica y la ecografía, optimiza la toma de decisiones y mejora los desenlaces materno-fetales.

Palabras Clave: Biomarcadores Angiogénicos; sFlt-1; PlGF; sFlt-1/PlGF razón; Preeclampsia; Arterias Espirales; VEGF, Ultrasonografía Fetal Doppler.

Introduction

Hypertensive disorders of pregnancy are conditions that significantly affect the mother–fetus dyad. The pathophysiological changes resulting from elevated blood pressure have long represented a challenge for medical science, as timely management and resolution are crucial to prevent adverse outcomes. Within the spectrum of hypertensive disorders of pregnancy—chronic hypertension, gestational hypertension, preeclampsia, and chronic hypertension with superimposed preeclampsia (Table 1)—all pose a substantial risk to pregnancy. This report focuses on preeclampsia, one of the most relevant conditions, for which diagnostic tools are still underused but have gained increasing importance. Advances in medical technology and biomarker-based approaches are contributing to a more practical and timely diagnosis and management.

Preeclampsia (PE) complicates between 2% and 8% of pregnancies worldwide. In Latin America and the Caribbean, hypertensive disorders account for almost 26% of maternal deaths[1]. Globally, more than 70,000 maternal deaths and 500,000 fetal deaths occur each year due to these conditions. In the United States, preeclampsia remains one of the leading causes of maternal mortality, severe maternal morbidity, hospital admissions for mothers and infants, cesarean deliveries, and preterm births[2].

Preeclampsia is defined as systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg, on at least two occasions separated by 4 hours, after 20 weeks of gestation in a previously normotensive woman, or a systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥110 mmHg confirmed within a short interval (minutes). Additionally, the presence of proteinuria ≥300 mg/24 h, or a urine protein/creatinine ratio ≥0.3, or a dipstick reading of ≥2+ (if no other methods are available) is required. Preeclampsia may also be associated with maternal organ dysfunction: thrombocytopenia (<100,000/μL), renal insufficiency (serum creatinine >1.1 mg/dL or a doubling of baseline creatinine in the absence of other causes), hepatic impairment (transaminases elevated to twice the upper normal limit), pulmonary edema, or neurological symptoms (persistent headache, blurred vision, epigastric pain, persistent dizziness, tinnitus, photopsia). Furthermore, it can cause fetal compromise, such as intrauterine growth restriction, abnormal fetal Doppler findings, and oligohydramnios[1].

The main risk factors include a history of preeclampsia, chronic hypertension, pregestational diabetes mellitus, antiphospholipid syndrome, obesity, advanced maternal age, nulliparity, history of chronic kidney disease, and the use of assisted reproductive technologies[2] (Table 2).

Preeclampsia is a pathology of abnormal placentation during pregnancy progression, divided into two stages: (1) abnormal placentation in early first trimester, followed by (2) a maternal syndrome in late second and third trimester, characterized by an excess of antiangiogenic factors[1]. During normal placental implantation, cytotrophoblasts migrate into the maternal spiral arteries, forming vascular sinuses at the fetomaternal interface to nourish the fetus. In placentas destined to develop preeclampsia, cytotrophoblasts fail to switch from the proliferative epithelial subtype to the invasive endothelial subtype, leading to incomplete spiral artery remodeling. Inadequate spiral artery remodeling results in narrowed maternal vessels and relative placental ischemia. Narrow spiral arteries are prone to acute atherosis, characterized by lipid-laden macrophages within the lumen, fibrinoid necrosis of the arterial wall, and perivascular mononuclear infiltrates, further compromising placental perfusion[1]. In humans, placental ischemia can be identified noninvasively by uterine artery Doppler studies. In normal pregnancy, uterine artery Doppler confirms robust systolic and diastolic flow; by contrast, women with preeclampsia exhibit significant diastolic impairment with a characteristic early diastolic notch preceding clinical signs and symptoms[4].

During the pathogenesis of preeclampsia, an imbalance in circulating angiogenic factors has also been identified. Elevated levels of the antiangiogenic protein sFlt-1 have been reported in placentas collected from women with a clinical diagnosis of preeclampsia[3]. sFlt-1 is a soluble protein that exerts antiangiogenic effects by binding and inhibiting the biological activity of the proangiogenic proteins VEGF and PlGF. VEGF is essential for maintaining endothelial cell function, particularly in fenestrated endothelium located in the brain, liver, and glomeruli—the main organs affected by preeclampsia. Several findings have implicated sFlt-1 in the pathogenesis of preeclampsia: sFlt-1 protein levels are elevated in maternal plasma or serum, and sFlt-1 mRNA expression is significantly increased in preeclamptic placentas[1].

In recent decades, studies have advanced in demonstrating that plasma concentrations of proangiogenic and antiangiogenic factors released by the

placenta (syncytiotrophoblast) may reflect the risks of disease progression. Antiangiogenic proteins, such as soluble fms-like tyrosine kinase-1 (sFlt-1), and proangiogenic placental growth factors (PlGF), show direct and inverse correlations, respectively, with the onset of the disease[4].

As previously mentioned, the sFlt-1/PlGF ratio is a biomarker of great importance in the diagnostic approach, which has been progressively introduced since its discovery. Clinical trials and meta-analyses have positioned it as a valuable diagnostic and prognostic tool[10]. In the following section, its mechanism of action, cutoff points, and the statistical analyses with results obtained from clinical trials will be briefly explained.

In women with clinical suspicion of PE, but without severe manifestations, it is important to perform tests for an accurate diagnosis. Once PE has been confirmed through the recommended tests, the patient should be referred to a specialized center for appropriate follow-up, depending on gestational age and clinical findings. The sFlt-1/PlGF ratio does not replace the standard diagnostic tests for PE and, at present, should not be used for diagnostic confirmation[4]. Women with clinical suspicion of PE who have negative diagnostic results for PE (absence of proteinuria or target organ damage) between 20 and 36+6 weeks of gestation should undergo the sFlt-1/PlGF test[8]. In these cases, the sFlt-1/PlGF ratio may support closer monitoring and more appropriate care planning. At ≤34 weeks of gestation, an sFlt-1/PlGF ratio ≤38 is reassuring regarding the absence of preeclampsia, with a negative predictive value (NPV) of 99.3%. This diagnostic value remains valid for up to 4 weeks following the test (2 weeks [NPV 97.9% (96.0–99.0)], 3 weeks [NPV 95.7% (93.3–97.5)], and up to 4 weeks [NPV 94.3% (91.7–96.3)][5]), making it a reliable tool to exclude the disease for at least the following month. However, after 34 weeks of gestation, the cutoff point may increase up to 110. If a patient has an sFlt-1/PlGF ratio >38 and ≤85 (≤34 weeks) or >38 and <110 (>34 weeks), this indicates an increased risk of PE[10]. Nonetheless, due to the low positive predictive value (PPV) in this situation [PPV 36.7% (28.4–45.7)], the diagnosis of PE cannot be established solely based on the ratio. These patients require close maternal–fetal monitoring and repeated PE testing according to clinical findings. Follow-up visits should be frequent, with adequate counseling on possible signs and symptoms suggestive of severe features[6].

Other situations include: sFlt-1/PlGF >85 (≤34 weeks) and >110 (>34 weeks). These higher ratio values, although not confirming PE, may reflect, in clinical practice, conditions associated with an increased risk of adverse outcomes[7]. Management in these cases will depend on the local institutional protocol, but closer maternal and fetal surveillance is required in this group of patients. In individual cases, and according to local protocols, hospitalization may be considered for more intensive monitoring[8]. Again, in the presence of clinical suspicion of PE or severe features, the standard diagnostic tests should always be performed[6]. Although this test provides substantial support in the diagnostic evaluation of PE, due to its low positive predictive value (PPV 36%), it should not be used as the sole diagnostic tool. On the other hand, exclusion of PE with negative results (sFlt-1/PlGF <38) is highly reliable, given its high negative predictive value (NPV).

Clinic Case

A 26-year-old female patient, G3C1A1V1, with a history of menarche at 12 years and type 1 diabetes mellitus since the age of 14. In her first pregnancy, she presented a spontaneous abortion without the need for curettage. In her second pregnancy in 2021, she delivered by cesarean section at 38 weeks due to non-reassuring fetal status. The second and current pregnancies share the same paternity. The patient's height is 165 cm, weight 59 kg, BMI 21.6. She presented to the emergency department on 11/18/2024 with an 11.3-week pregnancy according to first-trimester ultrasound performed on 10/20/2024. The patient reported a fasting glucose level of 332 mg/dl during prenatal control, in addition to a known diagnosis of type 1 diabetes mellitus treated with insulin glargine 25 IU at night and insulin glulisine 10 IU preprandially. The patient has a glucose sensor, which she does not use.

She was admitted for metabolic control. An ultrasound was performed, showing a slightly enlarged uterus with a gestational sac inside, containing a single, live embryo with a crown-rump length of 48 mm, corresponding to 11.4 weeks, and a fetal heart rate of 165 bpm. First-trimester laboratory results: fasting glucose 332 mg/dl, hemoglobin 13.7 g/dl, hematocrit 41%, blood type O+, HIV negative, rubella IgG positive and IgM negative, toxoplasmosis IgG negative and IgM negative, Chagas IgG negative, HBsAg negative, HbA1c 9.1%.

During hospitalization in November, the patient was evaluated by the endocrinology service, where insulin adjustments were made since she presented a hypoglycemic episode during admission. The final insulin regimen was modified to insulin glulisine 13 IU preprandially and insulin glargine 20 IU at night, along with strict glucose monitoring, dietary management, and an ophthalmology evaluation, which showed no evidence of diabetic retinopathy or other clinical findings. She was discharged on 11/18/2024 with outpatient follow-up by endocrinology. Based on the above, the patient was classified as high obstetric risk for preeclampsia, and treatment with acetylsalicylic acid 150 mg daily was initiated.

The patient was readmitted on 04/11/2025, referred from the perinatology outpatient clinic, with a 33-week pregnancy due to inadequate metabolic control (63%), with glucose levels exceeding 290 mg/dl. During perinatology follow-ups, no morphological changes or malformations were observed in the fetus during initially weekly ultrasound monitoring. Fetal echocardiography showed no cardiac abnormalities except for a perimembranous ventricular septal defect, which was not identified in subsequent ultrasounds. Throughout ultrasound monitoring, the fetus demonstrated growth between the 15th and 37th percentiles, with a normal amniotic fluid index during surveillance.

During this hospitalization, the patient continued to exhibit poor metabolic control and was re-evaluated by the endocrinology service, who performed further insulin adjustments. Laboratory results during admission showed an HbA1c of 6.7%.

While hospitalized, elevated blood pressure values were noted during vital sign monitoring, though not within severe ranges (139/91 – 145/86 mmHg). For this reason, a preeclampsia workup was considered.

The patient's urine protein-to-creatinine ratio was 0.13 (negative). However, the sFlt-1/PlGF ratio was 218, and based on this finding, the patient was classified as having preeclampsia without severe features, with no thrombocytopenia and no renal or hepatic dysfunction. Given these results, fetal lung maturation was initiated, along with antihypertensive therapy with nifedipine 30 mg every 8 hours, continued metabolic control as indicated by endocrinology, and follow-up of biomarkers in 48 hours, together with a strict preeclampsia profile every 12 hours. A perinatology consultation was requested for Doppler assessment of the uterine arteries, which showed no hemodynamic abnormalities.

Forty-eight hours later, at 33.2 weeks of gestation, new results showed an sFlt-1/PlGF ratio of 153, a decreasing trend, with the patient remaining asymptomatic. The rest of the preeclampsia workup was normal, with no thrombocytopenia. Blood pressure remained within normal ranges under antihypertensive therapy. At the next 48-hour follow-up (33.4 weeks), repeat preeclampsia biomarker testing showed an sFlt-1/PlGF ratio of 94.8. Given the decreasing biomarker trend, along with adequate blood pressure control and stable metabolic control, the patient was discharged on 21/04/2025 by the perinatology service, with recommendations for follow-up laboratory tests and Doppler ultrasound in 10 days.

During pregnancy, the following ultrasound evaluations were performed:

08/01/2025: Pregnancy at 19.6 weeks, single live fetus, FHR 154 bpm, posterior left-lateral placenta, AFI 12 cm, cervical length 34 mm without funneling.

16/01/2025: Pregnancy at 20+5 weeks by biometry, perimembranous ventricular septal defect, posterior right fundal placenta (high position), AIL 12 cm.

20/01/2025: Pregnancy at 20 weeks by biometry, estimated fetal weight 390 g (17th percentile), AFI 15 cm, FHR 147 bpm. Situs solitus, normal abdominal vascular situs, cardiac axis 27°, cardiothoracic ratio 0.48. Four-chamber view with normal atrioventricular connections, foramen ovale with right-to-left shunting as expected, normal outflow tracts. Long- and short-axis cardiac views were normal. Fetal echocardiogram normal for gestational age.

05/03/2025: Pregnancy at 27.1 weeks by biometry, single fetus in cephalic presentation, left dorsal position, estimated fetal weight 1084 g (25.5th percentile). No major malformations detected. Posterior high fundal placenta, AFI 14.7 cm, uterine artery Doppler at the 80th percentile.

11/04/2025: Pregnancy at 33 weeks by biometry, fetus in cephalic presentation, right dorsal position, normal fetal anatomy. Posterior high fundal placenta, AFI 17 cm, uterine artery Doppler at the 45th percentile, biophysical profile 8/8.

At follow-up in the perinatology outpatient clinic on 25/04/2025, the patient presented control laboratory results with 24-hour proteinuria of 141 mg/dL

(negative), normal preeclampsia panel, and progressively decreasing sFlt-1/PlGF biomarker levels with a result of 96.

New follow-up with perinatology on 02/05/2025, in which an ultrasound was performed showing a single live fetus in cephalic presentation with the back to the right, biometry corresponding to 37.4 weeks, biophysical profile 8/8, AFI: 9 cm, posterior left fundal placenta grade II/IV, with evidence of septal hypertrophy measuring 7 mm. Postnatal echocardiography was recommended. The patient also presented elevated blood pressure values, not within the severe range, without warning symptoms, despite continuing antihypertensive treatment with nifedipine.

The patient was admitted again to the emergency department on 02/05/2025, with a gestational age of 36.1 weeks, showing blood pressure of 157/94 mmHg and reporting headache, without other additional symptoms. She was admitted for blood pressure monitoring, continuation of antihypertensive therapy, and preeclampsia workup. On 03/05/2025, the patient presented with severely elevated blood pressure values (173/110 mmHg), headache, and a normal preeclampsia profile; however, sFlt-1/PlGF ratio: 300. Based on these findings, she was classified as a case of preeclampsia with severe features, and urgent delivery was indicated. An immediate cesarean section was performed, resulting in the birth of a neonate at 36.2 weeks, weighing 3,240 grams, measuring 47 cm, with adequate neonatal adaptation.

Discussion

Preeclampsia remains one of the most important obstetric conditions both in our setting and worldwide, as it is a disease with a non-classical presentation that often poses diagnostic and prognostic challenges.

The case presented corresponds to a pregnant woman with type 1 diabetes mellitus, a condition that classifies her as high obstetric risk and constitutes a predisposing factor for the development of preeclampsia. The presence of personal history, suboptimal metabolic control, and the finding of borderline blood pressure values during hospitalization increased clinical suspicion and justified the performance of complementary studies.

Traditionally, the diagnosis of preeclampsia has been based on the combination of arterial hypertension after 20 weeks of gestation and the presence of proteinuria or

evidence of target-organ damage. However, not all patients meet these classical criteria, which complicates the early identification of incipient cases. In this context, the introduction of angiogenic biomarkers, such as the sFlt-1/PlGF ratio, has represented a significant advance for screening and risk stratification. In the reported patient, despite the absence of significant proteinuria or target-organ involvement in the early stages, the elevation of the sFlt-1/PlGF ratio allowed the diagnosis of preeclampsia without severe features, which facilitated close surveillance and the initiation of timely management.

The dynamic behavior of angiogenic biomarkers in this patient proved to be highly illustrative. Initially, an sFlt-1/PlGF ratio of 218 was documented, well above the established cutoff for ruling out the disease (<38), which supported the diagnosis. Subsequently, under antihypertensive therapy and improved metabolic control, a progressive decline in biomarker levels was observed (153 and later 94), coinciding with maternal and fetal clinical stability. This finding highlights the utility of serial monitoring of the ratio, not only for diagnostic confirmation but also as a tool to monitor disease progression. Nevertheless, as reported in the literature, the low positive predictive value of the test requires careful interpretation in the clinical context, complemented by other laboratory and maternal-fetal assessments.

The subsequent marked increase of the ratio to 300, associated with severe-range hypertension and headache, was consistent with progression to preeclampsia with severe features, prompting the urgent decision to terminate the pregnancy. This outcome underscores that, while angiogenic biomarkers can guide surveillance and anticipate complications, they do not replace clinical judgment or traditional criteria for therapeutic decision-making, particularly regarding delivery timing.

Another relevant aspect is the association between type 1 diabetes and preeclampsia. Evidence shows that women with pregestational diabetes have a two- to fourfold increased risk, mainly due to chronic vascular alterations, endothelial dysfunction, and a persistent pro-inflammatory state. In this case, suboptimal glycemic control throughout pregnancy may have contributed to an unfavorable endothelial milieu and the subsequent development of preeclampsia. Although prophylactic aspirin was initiated as recommended, it was insufficient to prevent disease onset, reflecting the complexity of risk in patients with multiple predisposing factors.

From a perinatal perspective, strict ultrasound surveillance ruled out intrauterine growth restriction and significant Doppler abnormalities until late gestation. This is consistent with reports indicating that not all women with preeclampsia develop early fetal compromise, although close monitoring remains essential given the risk of sudden adverse outcomes. Delivery at 36.2 weeks resulted in a neonate with adequate weight and adaptation, reflecting how timely diagnosis and management—guided by biomarkers and clinical parameters—can contribute to favorable perinatal outcomes.

This case reinforces the applicability of angiogenic biomarkers in clinical practice and highlights the need for institutional protocols promoting their rational use, especially in high-risk populations. It also emphasizes the importance of strict metabolic control in women with pregestational diabetes and the role of multidisciplinary care teams in optimizing maternal-fetal outcomes.

Conclusions

Preeclampsia continues to be a diagnostic and therapeutic challenge in obstetrics, particularly in pregnant women with comorbidities such as type 1 diabetes mellitus, who are at significantly higher risk of developing this complication. The case presented illustrates how the use of angiogenic biomarkers, specifically the sFlt-1/PlGF ratio, serves as a valuable tool to complement traditional clinical and paraclinical criteria. Its main strength lies in its high negative predictive value, which allows the disease to be ruled out at early stages and guides clinical decision-making with greater confidence.

In this patient, serial monitoring of the sFlt-1/PlGF ratio facilitated the identification of progression from preeclampsia without severe features to a severe condition, which justified the timely resolution of the pregnancy and contributed to a favorable perinatal outcome. However, the case also confirms the limitations of the biomarker, particularly its low positive predictive value, which prevents its use as a standalone definitive diagnostic test.

Finally, this report highlights the importance of a comprehensive and multidisciplinary approach in high-risk pregnancies, as well as the need to incorporate innovative tools such as angiogenic biomarkers into clinical protocols, always in conjunction with traditional clinical and ultrasound evaluation.

Gratefulness

Thank's are extended to the teachers who participated in our medical practice.

Conflict of Interest

The authors declare that they have no conflicts of interest.

Financing

Financed with own resources.

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Attachments

Hypertensive Disorders During Pregnancy
Chronic Hypertension
Gestational Hypertension
Preeclampsia • Without Severe Features • With Severe Features • Eclampsia • HELLP Syndrome
Chronic Hypertension with Superimposed Preeclampsia

Table 1. Hypertensive disorders during pregnancy ACOG

Risk factors for developing Preeclampsia during pregnancy	
High Risk	• History of Preeclampsia • Multiple gestation • Chronic Hypertension • Type 1 *or 2 Diabete Mellitus* • Kidney Disease • Sleep Apnea Syndrome • Autoimmune Disease

Moderate Risk	• Nulliparity
	• Obesity (8MI >30)
	• Family History of Preeclampsia (First degree)
	• Age >35
	• Sociodemographic characteristics
	  • African descent
	  • Low socioeconomic level
	• Personal history
	  • Low birth weight
	  • Previous pregnancy with FGR
	  • Adverse previous pregnancy outcome

Low Risk	• Interpregnancy interval >10 years
Low Risk	Term delivery without complications.

Table 2. Risk factors of preeclampsia ACOG

About the Authors

German Andres Guevara Lizarazo is a General practitioner from University of Pamplona and a Medical specialist in Epidemiology from the Universidad Autónoma de Bucaramanga. Currently, he works at the Gynecology and Obstetrics Service of The FOSCAL Clinic in Floridablanca.

Laura Juliana Posso Perea is a general practitioner graduated from Universidad Libre, Cali campus, with experience in comprehensive patient care in emergency departments and growth and development programs. She spent her rural year at the Eastern Health Network in Santiago de Cali, where she served at the Carlos Holmes Trujillo Hospital and the Desepaz Health Center. She currently has a specialization in Health Management from Universidad del Rosario, which has allowed her to broaden her focus to administration and process improvement in the health sector.

Joshuart David Pascuas Varela born in Santa Marta, Colombia; physician graduated from University of Magdalena in 2022 with more than 2 years of experience in surgical assistance, achieving multiple procedures alone and under guidance. He has been working in third and fourth level institutions in which the care of the critically ill patients is the priority. Among his core values we highlight assertive communication, responsibility, and broaden dedication to his patient's well-being.

Daniela Lopez Londoño is a physician who graduated from Universidad Libre in 2021. She completed her rural year in Cali as an outpatient, providing life cycle care, prenatal care, family planning, and postpartum care, finishing in 2022. She lived in New York between 2023 and 2024, where she studied English at Kaplan Language International, achieving a high-intermediate level. At the end of 2024, she worked at a telecommunications company providing international medical consultations. She is currently studying with the goal of entering postgraduate studies.

Marbin Yilieth Alvarez Ramirez, is a general practitioner graduated from the University of Santander, works in the area of patient care in outpatient clinics, emergencies, and hospitalizations in a humane manner, empathizing with each of their pathologies and also caring for and protecting their mental health, with greater

emphasis and interest in the area of gynecology, which is the area in which I wish to specialize, ensuring the care and health of the mother-child binomial.

Valentina Dehaquiz Arango graduated as a doctor from the Universidad Libre de Cali and completed her mandatory social service at the Carlos Holmes Trujillo Hospital, where she worked in the Growth and Development Department. There, she consolidated her skills in neurodevelopmental prevention, promotion, and monitoring, fundamental pillars of primary pediatric care. Subsequently, she worked in the pediatric emergency department, a setting that strengthened my clinical resolution skills, decision-making under high-demand conditions, and comprehensive management of acute patients. She is currently a member of the Club Noel Children's Clinic Foundation, where, in addition to clinical practice, she focuses on independent research, with an emphasis on clinical and epidemiological processes, motivated by the search for evidence that optimizes the quality of care.