УДК: 618.3-008.6-0.6:616-036-616

# PREDICTING PREECLAMPSIA IN EARLY PREGNANCY

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***Abstract.*** *According to the World Health Organization (WHO), hypertensive disorders during pregnancy — including preeclampsia — are among the leading causes of maternal mortality and morbidity. Each year, approximately 500,000 women worldwide die from pregnancy- and childbirth-related complications, with 10–15% of these deaths attributed to hypertensive conditions, particularly preeclampsia and eclampsia. These conditions are more prevalent in low-income countries, where maternal mortality rates remain alarmingly high. For instance, maternal deaths due to preeclampsia and eclampsia are significantly more common in regions such as Africa and South Asia compared to other parts of the world. Preeclampsia affects approximately 2–8% of all pregnancies and poses serious risks not only to maternal health but also to fetal and neonatal outcomes.*

*In this article, we highlight the potential of soluble endoglin (sEng), an antiangiogenic factor involved in the pathophysiology of preeclampsia, as an early predictive biomarker. By assessing maternal history during the first trimester and measuring serum levels of sEng from the 16th week of gestation onward, it may be possible to predict the development of preeclampsia at an early stage.*

***Keywords:*** *s-endoglin, placental growth factor (PlGF), prediction, TGF-β1*

## Introduction.

Until recently, the molecular pathogenesis of preeclampsia remained poorly understood. However, recent findings suggest that altered expression of antiangiogenic placental factors may contribute significantly to its clinical manifestation. Two such antiangiogenic factors — soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng) — have been detected at elevated levels in the maternal circulation weeks before the onset of preeclampsia and shown to decline rapidly after delivery [1,2]. The serum concentrations of these factors have been found to correlate with disease severity and adverse pregnancy outcomes, highlighting their potential utility as diagnostic and prognostic biomarkers.

Studies by Gilbert and colleagues using the Reduced Uterine Perfusion Pressure (RUPP) animal model have demonstrated increased urinary levels of both sFlt-1 and sEng. sFlt-1 is a soluble form of VEGF receptor-1, which binds vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), thereby neutralizing their angiogenic effects. sEng, on the other hand, is a soluble co-receptor of the TGF-β superfamily, also released by the placenta. Both of these molecules are placental in origin and play a key role in the pathophysiology of preeclampsia.

The placenta plays a central role in preeclampsia, as evidenced by the rapid resolution of symptoms following delivery and the persistence of disease if placental tissue remains. To understand the early pathogenesis, attention has been focused on abnormal placentation processes.

**Methods and materials**

Our study was conducted from January 2024 to February 2025 at the Tashkent Medical Academy's maternity complex and the Women's Health Improvement Center. Two groups were involved in the study: Group 1 (30 healthy pregnant women) and Group 2 (30 pregnant women with identified risk factors for preeclampsia). Among these, 20 women diagnosed with preeclampsia were categorized according to the FIGO 2019 risk assessment table.

| High Risk | Middle risk |
|---|---|
| Chronic Hypertension in Previous Pregnancy | First Pregnancy |
| Chronic Arterial Hypertension | Age≥40years |
| Chronic Kidney Disease | IMB≥ 35 kg/m2 |
| Pre-existing Diabetes Mellitus | Family History of Preeclampsia |
| Autoimmune Diseases | Interpregnancy interval > 10 years |

**Table 1.Risk Groups for Preeclampsia according to FIGO 2019 (FIGO – International Federation of Gynecology and Obstetrics)[3]**

**Results**

In the analyzed groups, age differences were observed as follows: Participants in Group 1 had an average age of 28 ± 0.8 years, ranging from 19 to 40 years.In Group 2, the mean age was 26 ± 0.6 years, with participants aged between 20 and 35 years. Within Group 2, women were distributed based on risk factors for preeclampsia: 18 individuals (60%) were categorized as high risk, and 12 (40%) as moderate risk. Among healthy pregnant women, 36.7% (11 individuals) were experiencing their first pregnancy, while 63.3% (19 individuals) had a history of previous births. In contrast, among pregnant women identified with risk factors for

preeclampsia, 7 women (23.3%) were primigravida, and 23 women (76.3%) were multigravida.

Prospective observations revealed that 10 women (33.3%) in the high-risk group developed systolic arterial pressure (SAP) ≥180 mmHg, and in 18 cases (60%) from both high and moderate risk groups, SAP remained at or above 140 mmHg despite administration of antihypertensive treatment. The average diastolic blood pressure recorded was approximately 101 mmHg, and the mean systolic pressure was around 151 mmHg. Further, we found that among women at risk for preeclampsia: 7 (23.33%) developed chronic hypertension 18 (30%) developed gestational hypertension 15 (50%) developed preeclampsia, among whom 9 (30%) had the mild form, and 6 (20%) experienced the severe form Blood samples were collected simultaneously from participants across all groups. Samples were centrifuged and stored at -80°C. The samples were collected during the diagnostic stage for preeclampsia, and clinical as well as perinatal outcomes were documented prior to final diagnosis confirmation for appropriate classification. The mean s-endoglin (sEng) levels in maternal venous blood among women at risk for preeclampsia were as follows:

At 16 weeks: ~6.08 ng/mL
At 21 weeks: ~9.42 ng/mL
At 26 weeks: ~12.96 ng/mL
At 31 weeks: ~20.74 ng/mL

**Discussion**

This study, which included a considerable number of patients with preeclampsia, demonstrated that serum s-endoglin levels were significantly elevated in women with preeclampsia compared to those with normal pregnancies.

It was also established that the disparity in sEng levels intensified with the increasing severity of the disease, highlighting its potential to reflect the extent of systemic endothelial dysfunction.

Our findings support previously published data, showing that variations in this anti-angiogenic factor not only accompany preeclampsia but also become more prominent in more severe cases, especially with early-onset forms of the disease.Additionally, we explored the relationship between sEng levels and the risk of adverse pregnancy outcomes. Women with preeclampsia who had sEng levels in the highest quartile were significantly more likely to develop severe maternal complications and a variety of specific unfavorable outcomes. Higher sEng levels were consistently linked to greater risks of complications such as kidney dysfunction, the need for intubation, use of inotropic medications, low platelet counts, increased liver enzymes, preterm delivery, and having a small-for-gestational-age infant—even when accounting for gestational age, blood pressure, and proteinuria at the time of measurement.Therefore, our results reinforce and expand previous knowledge,

confirming that elevated sEng levels are associated with later complications in women suspected of preeclampsia during early gestation.

**Conclusion**

In summary, this research shows a strong association between circulating s-endoglin concentrations and the clinical severity of preeclampsia, suggesting that sEng could serve as a valuable prognostic biomarker for adverse outcomes in affected patients.

Additional long-term, large-scale studies are necessary to further investigate sEng's role in the early diagnosis and clinical management of preeclampsia.

Measuring s-endoglin in early pregnancy may aid in identifying women at high risk, enabling timely intervention and preventive treatment strategies.

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