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ALGORITHM FOR LABORATORY EXAMINATION OF PREGNANT WOMEN WITH SUSPECTED PREECLAMPSIA

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Abstract
Preeclampsia is a multisystem hypertensive disorder of pregnancy that remains one of the leading causes of maternal and perinatal morbidity and mortality worldwide. Early identification of women at high risk is essential for improving obstetric outcomes. Recent advances in molecular medicine have introduced novel endothelial and angiogenic biomarkers, including endothelial nitric oxide synthase (NOS3), placental growth factor (PlGF), soluble fms-like tyrosine kinase-1 (sFlt-1), and soluble endoglin (sEng). The present study proposes a diagnostic algorithm integrating clinical risk assessment, Doppler ultrasonography, and laboratory biomarkers for the early prediction and stratification of preeclampsia risk. The combined evaluation of angiogenic imbalance and endothelial dysfunction markers significantly increases diagnostic accuracy compared to traditional clinical criteria alone. Implementation of such an algorithm in routine obstetric practice may enhance early detection and allow timely preventive interventions.

Keywords: Preeclampsia, diagnostic algorithm, endothelial dysfunction, angiogenic markers, NOS3, PlGF, sFlt-1, sEng, early prediction.

Introduction

Preeclampsia is defined as new-onset arterial hypertension after 20 weeks of gestation accompanied by proteinuria or signs of maternal organ dysfunction. Despite advances in obstetric care, the condition affects approximately 5–8% of pregnancies globally and contributes substantially to maternal and neonatal complications.[1,3]

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The pathophysiology of preeclampsia is characterized by abnormal placentation, uteroplacental ischemia, systemic endothelial dysfunction, and an imbalance between pro-angiogenic and anti-angiogenic factors. Traditional diagnostic criteria are based on clinical manifestations, which often appear after significant pathophysiological changes have already occurred. Therefore, the development of an early predictive diagnostic algorithm is of considerable clinical importance.[2,4,5]

The development of a predictive algorithm should be grounded in the two-stage model of preeclampsia: Stage I – Abnormal placentation - Inadequate trophoblast invasion and incomplete remodeling of spiral arteries lead to placental hypoxia. Stage II – Maternal systemic response.

The ischemic placenta releases anti-angiogenic factors into maternal circulation, triggering endothelial dysfunction, vasospasm, and hypertension. Key molecular mechanisms include: Decreased nitric oxide (NO) production due to reduced NOS3 activity, decreased PlGF levels, increased sFlt-1 concentration, increased sEng levels. [7,9,10]

These biomarkers reflect endothelial injury and angiogenic imbalance, forming the scientific basis for a structured diagnostic algorithm. Key molecular mechanisms include: Decreased nitric oxide (NO) production due to reduced NOS3 activity, decreased plgf levels, increased sflt-1 concentration, increased seng levels.[8,11]

These biomarkers reflect endothelial injury and angiogenic imbalance, forming the scientific basis for a structured diagnostic algorithm. Clinical Risk Assessment (11–13 Weeks) it includes- Maternal age, first pregnancy, obesity, chronic hypertension, diabetes mellitus, history of preeclampsia. Doppler Ultrasound Examination Uterine artery pulsatility index (PI) Elevated PI indicates impaired uteroplacental perfusion.[14,16,21]

If abnormal Doppler findings are detected, laboratory biomarker evaluation is recommended. This biological markers are PlGF – decreased levels indicate placental dysfunction. sFlt-1 – elevated levels reflect anti-angiogenic activity. sFlt-1/PlGF Ratio >85 suggests high probability of severe preeclampsia. Ratio <38 indicates low short-term risk. sEng – increased concentration correlates with disease severity. NOS3 expression or activity – reduced levels indicate endothelial dysfunction.[13,16]

The integration of molecular biomarkers into routine prenatal screening offers several advantages: Earlier detection compared to clinical criteria alone, dentification of subclinical endothelial dysfunction, objective risk stratification, improved timing of preventive interventions, reduction of maternal and perinatal complications. Studies demonstrate that combined biomarker screening improves predictive accuracy up to 80–90%, significantly higher than Doppler assessment alone.[14,16]

The proposed algorithm emphasizes a multimodal approach combining clinical, instrumental, and laboratory data. The use of angiogenic markers, particularly the sFlt-1/PlGF ratio, has already been incorporated into clinical practice in several countries.[18,19]. Evaluation of endothelial dysfunction through NOS3-related pathways provides a promising direction for personalized medicine in obstetrics. Future research should focus on integrating genetic polymorphisms and machine-learning models to further enhance predictive performance Preeclampsia remains a major challenge in modern obstetrics due to its unpredictable onset and rapid progression. A structured diagnostic algorithm based on angiogenic and endothelial biomarkers significantly improves early prediction and risk stratification. [20,22]

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The combined assessment of NOS3, PlGF, sFlt-1, and sEng alongside Doppler ultrasonography represents a scientifically grounded and clinically effective approach for early detection. Implementation of this algorithm into routine prenatal care may reduce maternal morbidity and improve perinatal outcomes. Further large-scale multicenter studies are required to validate and standardize biomarker thresholds for universal screening protocols. [21,23]

The main diagnostic criteria for preeclampsia include the following: Arterial hypertension - Systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg measured on at least two occasions at a minimum interval of 4–6 hours after 20 weeks of gestation in a previously normotensive woman. Proteinuria - A diagnostic threshold of ≥300 mg of protein in a 24-hour urine collection. Quantitative assessment of protein excretion in a 24-hour urine sample remains the gold standard for diagnosis. Currently, preeclampsia is classified as: Mild preeclampsia, moderate preeclampsia, severe preeclampsia, superimposed preeclampsia on chronic arterial hypertension. [17,22]

Additionally, preeclampsia is categorized according to the gestational age at onset, developing before 32–34 weeks of gestation (in some classifications before 27 weeks). Early-onset cases account for approximately 13–40% of all preeclampsia cases and are associated with more severe maternal and perinatal outcomes. This form is characterized by fetal growth restriction, reduced placental size, impaired uteroplacental and fetoplacental circulation, preterm delivery, increased neonatal morbidity and mortality. Early-onset preeclampsia is closely associated with impaired trophoblast invasion, incomplete remodeling of spiral uterine arteries, immune maladaptation, and elevated levels of endothelial dysfunction markers. If preeclampsia develops before 24 weeks of gestation, the risk of severe complications increases significantly, including: help syndrome (up to 61.5%), eclampsia (approximately 19%), pulmonary edema (approximately 16%), perinatal mortality (up to 81%). [22,24]

Although pre-eclampsia cannot be completely prevented, several measures may reduce its likelihood. Consistent and early antenatal care plays a crucial role in identifying and controlling potential risk factors before complications develop. Preventive strategies are primarily based on careful monitoring throughout pregnancy. These include routine measurement of blood pressure, regular screening of urine for protein, and observation for warning symptoms such as persistent headaches or visual changes. Maintaining a healthy body weight, staying physically active when medically approved, and properly managing existing medical conditions - particularly chronic hypertension - are also important components of risk reduction. [21,25]

Further preventive interventions may involve starting low-dose aspirin before 20 weeks of gestation or at the first antenatal visit, providing calcium supplementation in populations with insufficient dietary calcium intake, and appropriately treating pre-existing hypertension with antihypertensive therapy. [19,25]

The World Health Organization (WHO) has issued comprehensive recommendations to improve maternal health outcomes during pregnancy. These guidelines address both the prevention and management of pre-eclampsia and eclampsia and are regularly updated based on emerging scientific evidence to ensure high-quality care. [18,23]

The main WHO recommendations include calcium supplementation for pregnant women in areas with low calcium intake, administration of low-dose aspirin for those at increased risk of developing

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pre-eclampsia, and the use of magnesium sulfate to prevent the progression to eclampsia. The organization also emphasizes the importance of educating healthcare professionals to recognize early signs of the condition and strengthening healthcare systems to provide timely and effective maternal care. Through the implementation of these evidence-based strategies, WHO aims to reduce maternal and perinatal illness and death worldwide and contribute to achieving the targets outlined in the Sustainable Development Goals (SDGs).[16,24,25]

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