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ANGIOGENIC BIOMARKERS FOR PREECLAMPSIA PREDICTION

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Preeclampsia is a pregnancy condition that causes high blood pressure and organ damage, most often to the liver and kidneys. It usually manifests after 20 weeks of pregnancy and can result in major difficulties for the mother and the baby, including premature delivery, growth limitation, placental abruption, and stillbirth. The only known cure is delivery of the placenta. As far as epidemiology is concerned, preeclampsia is a serious global health issue that affects 5% to 8% of all pregnancies. Preeclampsia rates vary by geography, with the most significant rates recorded in low- and middle-income nations, where access to high-quality prenatal care and trained delivery attendants is scarce.

To study the angiogenic marker for the early prediction of preeclampsia.

A systematic reviewed of articles, focusing on the etiopathogenesis, risk factors, and management of preeclampsia. Relevant studies were identified through searches in PubMed, Scopus, and Web of Science. The findings were synthesized qualitatively to identify common themes.

This study highlighted the role of antiangiogenic mediators in the pathophysiology of preeclampsia. Preeclampsia originates in the placenta, starting with inadequate cytotrophoblast invasion and ending with widespread maternal endothelial dysfunction. Production of placental anti-angiogenic factors, specifically soluble fms-related tyrosine kinase 1 and soluble endoglin, have been shown to be upregulated in preeclampsia. These placental anti-angiogenic factors are released into the maternal circulation, their actions disrupt the maternal endothelium and result in hypertension, proteinuria, and the other systemic manifestations of preeclampsia. The molecular basis for placental dysregulation of these pathogenic factors remains unknown. Hypoxia is likely an important regulator. Other factors such as alterations in the renin-angiotensin-aldosterone axis, immune maladaptation, excessive shedding of trophoblast debris, oxidative stress, and genetic factors likely contribute to the pathogenesis of the abnormal placentation. Pooled information on placental perfusion (ultrasonography, mean arterial pressure), clinical characteristics, and biomarker levels (PlGF) can improve first-trimester prediction and preeclampsia diagnosis. Angiogenic factors (sFlt-1/PlGF ratio; PlGF alone) with or without clinical characteristics can facilitate second-/third-trimester prediction of early-onset and late-onset preeclampsia. A combination of increased sFlt-1/PlGF ratio and ultrasound can rule out early fetal growth restriction. The sFlt-1/PlGF ratio is also a reliable tool for discriminating between pregnancy-related hypertensive disorders, including superimposed preeclampsia and gestational hypertension.

An increase in angiogenic biomarkers (sFlt-1/PlGF ratio, PlGF) combined with clinical and ultrasonographic data enhance early prediction and diagnosis. The sFlt-1/PlGF ratio also aids in distinguishing preeclampsia from other hypertensive disorders and assessing fetal growth restriction risk.