RISK FACTORS FOR THE DEVELOPMENT OF PREECLAMPSIA IN WOMEN WITH SUBCLINICAL KIDNEY DISEASE

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Introduction. Subclinical kidney disease (SKD) is characterized by subtle renal impairment without overt clinical symptoms, often detectable only through sensitive laboratory and imaging markers.[1] Emerging evidence suggests that even mild, undiagnosed renal dysfunction significantly increases the risk of developing preeclampsia (PE). [2] Impaired renal reserve, endothelial dysfunction, and altered sodium handling contribute to abnormal placentation and hypertensive complications during pregnancy. Identifying risk factors for PE in women with underlying subclinical kidney disease is essential for early prevention and improved maternal–fetal outcomes.[3]

Aim. The aim of this study is to evaluate the key clinical, biochemical, and hemodynamic risk factors associated with the development of preeclampsia in women with previously unrecognized (subclinical) kidney disease.

Materials and Methods. A prospective study included 60 pregnant women aged 20–40 years in the first trimester. Based on initial screening, 30 women with markers of subclinical kidney disease (eGFR 60–90 mL/min/1.73 m², mildly elevated cystatin C, or microalbuminuria 15–30 mg/g) formed the study group, while 30 healthy pregnant women served as controls. Evaluations included serum creatinine, cystatin C, eGFR (CKD-EPI), and urine albumin-

creatinine ratio (ACR). Endothelial dysfunction markers (sFlt-1, PlGF, endothelin-1), inflammatory markers (CRP, IL-6), and oxidative stress indicators were measured. Renal Doppler ultrasonography assessed renal resistive index (RRI), and 24-hour ambulatory blood pressure monitoring was performed to detect early hypertensive changes.

Results. Preeclampsia developed in 12 out of 30 women (40%) in the subclinical kidney disease group compared to 4 out of 30 (13%) in the control group ($p<0.05$). Women who developed PE demonstrated significantly higher baseline levels of cystatin C and ACR. Elevated sFlt-1/PlGF ratio was observed early in pregnancy among those who later developed PE. Increased RRI (>0.68) was detected in 10 women (33%) and was strongly associated with subsequent PE development. Subclinical hypertension (mean systolic BP 125–135 mmHg) and reduced nocturnal BP dipping were also more frequent in the SKD group. Inflammatory markers (CRP, IL-6) were significantly elevated in patients who developed PE ($p<0.05$).

Discussion. The results indicate that subclinical kidney dysfunction is a significant independent risk factor for preeclampsia. Early alterations in renal hemodynamics, endothelial imbalance (angiogenic factors), and low-grade inflammation appear to play a central role in the pathogenesis. Among all parameters, cystatin C, ACR, and sFlt-1/PlGF ratio showed the highest predictive value. These findings emphasize the importance of early renal screening in pregnancy, even in women without known kidney disease.

Conclusion. Women with subclinical kidney disease are at significantly increased risk of developing preeclampsia. Early identification using sensitive renal and endothelial markers, along with blood pressure monitoring, allows timely preventive strategies. Integrating these assessments into routine antenatal care may reduce the incidence and severity of preeclampsia and improve pregnancy outcomes.

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