From ACOG to AHA: Reclassifying First-Trimester Blood Pressure Uncovers Uronic Acid as a Conditional Biomarker for Hypertensive Disorders of Pregnancy in low- and middle-income countries.

Oral

Adverse perinatal outcomes associated with maternal complications

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Aim: Hypertensive disorders of pregnancy (HDP) are leading causes of maternal mortality in low- and middle-income countries (LMICs), where late diagnosis contributes to poor outcomes. Current risk stratification relies on the ACOG diagnostic threshold of 140/90 mmHg, which identifies disease only after it is established. In contrast, the 2017 AHA guidelines classify BP ≥130/80 mmHg as stage 1 hypertension, offering potential for earlier risk identification. We aimed to evaluate a two-step prognostic model for LMICs combining early AHA-based hemodynamic assessment with a sequential urinary biomarker.

Methods: In a prospective LMIC cohort, we reclassified first-trimester blood pressure according to AHA guidelines.

Results: Women with AHA stage 1 hypertension (BP ≥130/80 mmHg) had a 18.8% probability of developing preeclampsia and 22.6% probability of gestational hypertension—a combined risk approaching 41.5%, with disease onset as early as 22 weeks. ROC analysis showed systolic BP >113 mmHg or diastolic BP >76 mmHg before 14 weeks were powerful predictors. In contrast, the traditional ACOG threshold identified only advanced disease. We then analyzed urinary glycosaminoglycans as

sequential biomarkers. While Syndecan-1 showed no predictive value, total uronic acids emerged as significant exclusively within the AHA stage 1 high-risk group. In this subgroup, a uronic acid cutoff <119 mg/mmol at 23–26 weeks provided approximately 60% sensitivity and 68% specificity for predicting HDP. This marker showed no association with outcomes under traditional ACOG classification, demonstrating how BP threshold determines biomarker utility.

Conclusion: We propose a two-step model for HDP prediction in LMICs. First, adopting the AHA BP classification at the first prenatal visit—a zero-cost intervention—identifies latent vascular susceptibility. Second, placentation (23-26 weeks) amplifies this susceptibility, making it detectable through changes in urinary GAG levels. This sequential approach moves beyond the reactive ACOG model toward proactive risk stratification, opening an affordable window for early intervention where resources are most limited.

Palabras Clave

(1) AHA blood pressure classification, (2) urinary glycosaminoglycans, (3) early risk stratification, (4) preeclampsia.

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