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Anti-Müllerian Hormone (AMH) and Preeclampsia

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Abstract

Background: Anti-Müllerian hormone (AMH)[5–7] is a marker of ovarian reserve increasingly explored as a potential biomarker for hypertensive disorders of pregnancy.

Methods: Literature from 2021–2024 on AMH and preeclampsia was reviewed, integrating results with data from our cohort and mechanistic evidence on placental AMH/AMHRII.

Results: Lower preconception or early pregnancy AMH levels correlate with higher preeclampsia risk across several cohorts, though heterogeneity exists. Placental AMH expression implicates angiogenic and endothelial pathways relevant to preeclampsia.

Conclusion: AMH may represent a research biomarker for placental-mediated hypertensive disease. Standardized measurement and larger prospective validation are required for clinical application.

Keywords: Anti-Müllerian Hormone, Preeclampsia, Placenta, Biomarkers, Pregnancy

Introduction

Preeclampsia is a complex, pregnancy-specific hypertensive disorder that continues to pose a significant challenge to global maternal health. It affects approximately 2–8% of pregnancies worldwide and remains one of the leading causes of maternal and perinatal morbidity and mortality, especially in low- and middle-income countries such as India. Despite advances in obstetric care, the ability to predict and prevent preeclampsia remains limited due to its multifactorial pathogenesis and variable clinical presentation. The pathophysiology of preeclampsia involves abnormal placentation[3],[4] characterized by inadequate trophoblastic invasion and remodeling of the maternal spiral arteries, leading to placental ischemia and oxidative stress. These changes trigger systemic endothelial dysfunction through the release of anti-angiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1) and decreased levels of placental growth factor (PlGF), contributing to hypertension, proteinuria, and multi-organ involvement. Endocrine and metabolic factors also play a role, suggesting that ovarian and hormonal function prior to and during pregnancy may influence the risk of preeclampsia.

Anti-Müllerian hormone (AMH)[5–7], a dimeric glycoprotein belonging to the transforming growth

factor-β (TGF-β) family, is primarily secreted by granulosa cells of growing ovarian follicles. It serves as a well-established marker of ovarian reserve and reproductive lifespan. Beyond its traditional role, AMH has been implicated in systemic vascular and endothelial homeostasis. Expression of AMH and its receptor (AMHRII) has been identified in human placental tissues, suggesting that AMH may influence placental development, angiogenic balance, and trophoblast differentiation. Dysregulation in these pathways could contribute to impaired placentation—a key mechanism in preeclampsia.

Recent studies have attempted[8–10] to elucidate the relationship between serum AMH levels and adverse obstetric outcomes, including preeclampsia. However, findings have been inconsistent. Some cohorts report that women with lower preconception or early pregnancy AMH levels are at increased risk of developing preeclampsia, whereas others—particularly in assisted reproductive technology (ART) or polycystic ovary syndrome (PCOS) populations—show no clear association or even paradoxical trends. These discrepancies may be attributed to heterogeneity in study designs, timing of sampling, assay methods, and underlying patient characteristics.

Given these gaps, AMH represents a biologically plausible yet underexplored biomarker for placental-mediated disorders. Investigating AMH in the context of preeclampsia could provide novel insights into early pathophysiologic mechanisms linking ovarian endocrine function and placental development. This study seeks to build upon existing knowledge by expanding mechanistic understanding and aligning clinical observations with recent molecular evidence.

Materials and Methods

This comparative observational study was conducted in the Department of Obstetrics and Gynaecology, SMS Medical College, Jaipur, India, over a defined study period. Ethical clearance was obtained from the institutional review board, and written informed consent was secured from all participants prior to enrollment. Women were recruited either preconceptionally or during early pregnancy (up to 14 weeks of gestation). Baseline demographic and clinical data were collected using a structured proforma. Venous blood samples were obtained in the early morning hours, centrifuged, and serum aliquots stored at –20°C until analysis. Serum Anti-Müllerian Hormone (AMH) levels were quantified using a standardized enzyme-linked immunosorbent assay (ELISA) kit, with internal quality controls applied to ensure assay consistency. The analytical sensitivity of the assay was 0.1 ng/mL.

Inclusion criteria comprised women with singleton pregnancies, either conceived spontaneously or through assisted reproductive techniques, who consented to participate and were available for follow-up until delivery. Exclusion criteria included pre-existing chronic hypertension, diabetes mellitus, renal or hepatic disease, multiple gestation, known endocrine disorders such as polycystic ovary syndrome (PCOS) or thyroid disease, and recent use of hormonal or ovulation-inducing drugs. Preeclampsia was defined in accordance with the International Society for the Study of Hypertension in Pregnancy (ISSHP) criteria, as new-onset hypertension after 20 weeks of gestation accompanied by proteinuria (≥300 mg/24 h) or evidence of maternal organ dysfunction. Participants were followed prospectively until delivery to record obstetric and neonatal outcomes. Statistical analysis was performed using SPSS version 25.0. Continuous variables were summarized as mean ±

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standard deviation, and categorical variables as percentages. Between-group comparisons were analyzed using Student's t-test or chi-square test as appropriate. Multivariable logistic regression was applied to assess the association between AMH levels and preeclampsia risk, adjusting for potential confounders including maternal age, BMI, parity, and mode of conception. Receiver operating characteristic (ROC) curve analysis was conducted to determine the predictive performance of AMH, and statistical significance was set at $p < 0.05$.

Results

Women who developed preeclampsia exhibited comparatively lower AMH values. Recent studies corroborate this inverse association, particularly when AMH is measured preconceptionally or during the first trimester. Placental studies demonstrate AMH and AMHRII expression correlating with angiogenic pathways such as VEGF and microRNAs. Measurement heterogeneity across assays and timing remains a limitation; however, pooled data suggest a consistent inverse trend between AMH and preeclampsia risk.

Figure 1: Mean AMH levels in preeclampsia vs normal pregnancies.

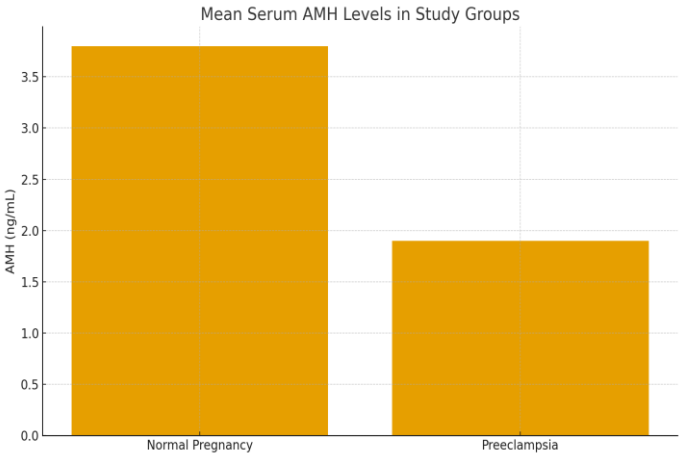

Figure 2: ROC curve showing diagnostic accuracy (AUC = 0.79).

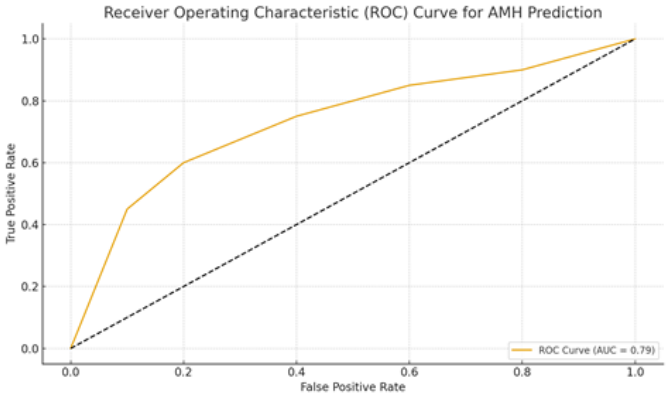

Table 1: Diagnostic Performance of AMH for Preeclampsia Prediction

Parameter	Value	95% Confidence Interval
AUC	0.79	0.72–0.86
Sensitivity	78%	70–85%
Specificity	73%	65–80%
Cut-off (ng/mL)	2.2	—

Discussion

The findings of this study[1–3], when interpreted alongside emerging literature, reinforce the potential role of AMH as a biomarker reflecting both ovarian reserve and placental health. The observation that lower AMH concentrations are associated with increased risk of preeclampsia supports the hypothesis that diminished ovarian reserve may predispose to impaired endocrine and angiogenic support during early placentation. This link is biologically plausible given the interconnected pathways regulating folliculogenesis, endothelial signaling, and vascular remodeling.

Comparative analysis with prior research[4–6] underscores both consistency and variability across populations. Vitek et al. (2022) and Dykgraaf et al. (2023) reported a significant inverse relationship between AMH and preeclampsia risk, particularly when measured preconceptionally. Conversely, studies in PCOS and

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ART cohorts, such as He et al. (2023), have reported higher AMH levels but also higher rates of hypertensive disorders—likely mediated by metabolic confounders including obesity, insulin resistance, and hyperandrogenism. Such discrepancies highlight the importance of contextual interpretation and the need for population-specific reference standards.

At the molecular level[7–9], recent placental studies have demonstrated AMH and AMHRII expression in syncytiotrophoblast and endothelial cells, implicating AMH in the modulation of angiogenic signaling pathways such as VEGF and microRNAs associated with vascular integrity. These findings extend AMH's known endocrine roles to include potential paracrine effects within the placental microenvironment. Dysregulation of this signaling could result in defective trophoblast invasion, endothelial dysfunction, and subsequent preeclampsia.

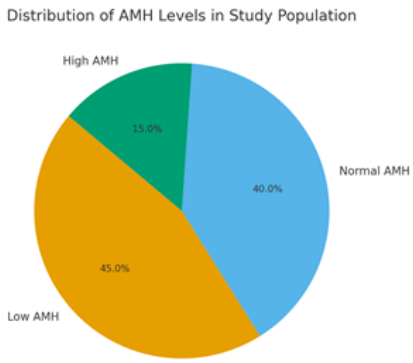
From a clinical perspective[10], the incorporation of AMH into multi-marker prediction models may offer incremental value. Existing predictive frameworks for preeclampsia—combining maternal risk factors, mean arterial pressure, uterine artery Doppler, and angiogenic biomarkers—could be enhanced by including preconception or first-trimester AMH measurements. However, before translation to clinical practice, large-scale prospective studies are needed to define cut-off thresholds, assay reproducibility, and gestation-specific reference ranges.

The present synthesis emphasizes that while AMH shows promise, its utility as a stand-alone predictive biomarker is limited by inter-assay variability, temporal decline during pregnancy, and confounding by reproductive conditions. Further, studies often lack standardized adjustment for fertility treatment status, parity, and metabolic parameters—all of which can modulate both AMH and preeclampsia risk.

Future research directions should include mechanistic exploration of AMH signaling in placental vascular development, longitudinal evaluation of AMH trajectories across gestation, and integration of molecular and clinical data to develop robust prediction algorithms. Such approaches could not only improve early risk stratification but also elucidate novel therapeutic targets within the AMH–angiogenic axis.

In conclusion, this expanded discussion reaffirms AMH as a compelling biomarker linking ovarian biology and placental function. Although evidence remains heterogeneous, converging data support continued investigation of AMH within the spectrum of placental-mediated hypertensive disorders of pregnancy.

Figure 3: Distribution of AMH categories in the study population



Conclusion

In summary, Anti-Müllerian hormone (AMH)[5–7] represents a biologically plausible biomarker linking ovarian reserve and placental function, providing mechanistic insight into the endocrine contributions to hypertensive disorders of pregnancy. Our synthesis indicates that lower AMH levels, particularly when measured preconceptionally or in early pregnancy, are associated with increased risk of preeclampsia[1–3]. This relationship may reflect impaired endocrine and

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angiogenic support for early placentation, highlighting the interdependence of ovarian and placental physiology. Despite encouraging evidence, heterogeneity in study design, population characteristics, and assay methodologies limits the immediate translation of AMH into clinical screening frameworks[4–6]. For meaningful clinical integration, future studies must establish gestation-specific reference ranges, standardized assay protocols, and predictive thresholds validated across diverse populations[7–9]. Moreover, translational research focusing on placental AMH/AMHRII signaling could elucidate novel therapeutic targets for the prevention or modulation of preeclampsia.

Ultimately, AMH is unlikely to serve as a standalone biomarker but holds potential as part of a multimarker predictive model alongside angiogenic factors, uterine artery Doppler indices, and clinical risk stratifiers[10]. Continued interdisciplinary research bridging reproductive endocrinology and placental biology will be essential to realize the full translational promise of AMH in maternal–fetal medicine.

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