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Uric acid and obstetric syndromes: preeclampsia as a starting point

Abstract

Uric acid (UA) has been proposed as a key biomarker within the spectrum of obstetric syndromes, particularly preeclampsia (PE). Acting as a proinflammatory mediator and metabolic marker, in addition to its antioxidant effect, UA is positioned as a potential indicator of placental dysfunction and adverse perinatal prognosis, determining perinatal outcomes under expectant management. This scoping review analyzes the available evidence on the relationship between UA and PE, extending its interpretation to other placental-origin syndromes such as fetal growth restriction (FGR), preterm birth, and intrauterine fetal death. Twenty relevant articles published between 2017 and 2025 were included, integrating pathophysiological, clinical, and predictive findings. Studies consistently show that hyperuricemia is associated with a higher risk of severe PE, FGR, and preterm birth, particularly when the UA/creatinine ratio adjusted for renal function is used. Although UA demonstrates moderate diagnostic utility and lacks standardization, its low cost and wide availability consolidate it as a complementary biomarker of clinical interest. Prospective research is required to establish trimester-specific cutoff values and to evaluate whether therapeutic modulation impacts maternal–fetal outcomes.

Keywords: uric acid, obstetric syndromes, preeclampsia, pregnancy

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Abbreviations: UA, uric acid; PE, particularly preeclampsia; FGR, fetal growth restriction; XDH/XO, xanthine dehydrogenase and xanthine oxidase; DAMPs, damage-associated molecular patterns; SUA/sCr, serum uric acid/creatinine ratio

Introduction

Preeclampsia is a hypertensive disorder of pregnancy that usually manifests after 20 weeks of gestation and is characterized by the onset of hypertension accompanied by proteinuria and/or organ dysfunction.[1] It is responsible for a significant proportion of maternal–fetal mortality and morbidity worldwide, with a global prevalence estimated between 2% and 8% of pregnancies, accounting for 26% of maternal deaths in Latin America.[1] Although its etiology is multifactorial, the most widely accepted mechanisms involve placental dysfunction, deficient vascular remodeling, oxidative stress, and dysregulated maternal immunoinflammatory responses.[2] These processes are not exclusive to preeclampsia; they are part of a spectrum of entities known as "obstetric syndromes," which include intrauterine growth restriction, unexplained intrauterine fetal death, and, in some models, spontaneous preterm birth.[3–5] All share a common placental origin characterized by abnormal trophoblastic invasion and consequent systemic maternal endothelial dysfunction.[2,4]

It has traditionally been observed that uric acid levels tend to be elevated in pregnancies complicated by preeclampsia; however, it is not clear whether this elevation is a cause or a consequence of renal damage or oxidative stress induced by the disease.[2] Considering that similar mechanisms occur in other obstetric syndromes, the hypothesis arises that uric acid could represent a common indicator of placental dysfunction, with potential utility beyond preeclampsia.[4–6]

The toxemia service of the Instituto Materno Infantil (1955–2006) identified several phases regarding the utility of uric acid knowledge applied to PE. An initial phase before 1976 showed elevation in 70% of patients with toxemia of pregnancy.[7] Subsequently, a British publication highlighted its importance as a predictor of perinatal mortality. Between 1976 and 1996, it was used to terminate pregnancy once fetal lung maturity was confirmed when levels exceeded 6.0 mg/dL.[7] At that time, it was believed that elevation resulted from renal dysfunction.[7] Between 1996 and 2013, this period was termed a biochemical boom for uric acid quantification in all women with hypertension during pregnancy.[7] In 1996, an AJOG article explained the prevailing clinical belief: adverse perinatal prognosis in patients with hyperuricemia was explained by placental oxidative stress.[5] This was surprising for many investigators who believed that xanthine oxidases were absent in the placenta.[5] Greater placental hypoxia led to greater placental oxidative stress and higher perinatal mortality. The correlation became dogmatic. Why a boom? Circulating uric acid concentrations were used in nephropathic patients, chronic hypertensive patients, suspected superimposed PE, and in the differential diagnosis of gestational hypertension.[5] It was even used to predict maternal mortality: values above 8 mg/dL were said to increase the risk of lethal maternal eclampsia.[5] It was even considered for follow-up of normotensive fetuses with IUGR secondary to placental vasculopathy.[5] However, from 2013 onward, national medicine misinterpreted a publication and uric acid entered a decline.[5] It was no longer requested; it disappeared from PE biochemical screening and prenatal care. This effect stemmed from the ACOG Task Force. The publication states verbatim: "Hyperuricemia is a specific biochemical marker that has been associated with increased morbidity in hypertensive complications of pregnancy.[8] It should not be used for diagnosis.".[8]

A more precise understanding of the role of uric acid in the pathogenesis or progression of preeclampsia could provide several practical benefits. First, if elevation precedes clinical manifestation,

it could serve as an early risk biomarker to identify pregnancies with high potential to develop the disease.[9] Second, if the relationship is causal or contributes actively to endothelial or renal damage, therapies aimed at reducing uric acid levels could be explored as preventive or disease-modulating interventions.[3,10] Additionally, even if not causal, integration with other biomarkers could improve multivariable models beyond the predictive capacity of individual parameters.[10]

The objective of this review is to perform a critical and updated analysis of the literature on the relationship between preeclampsia and uric acid, using this disorder as a starting point to explore its relevance across obstetric syndromes, evaluating proposed biological mechanisms, recent clinical evidence regarding prediction and risk stratification, neutral and contradictory points, and potential clinical application scenarios—in other words, to "revive" the utility of uric acid. While obstetrics abandoned its contribution, the modern world has found scenarios of understanding in metabolic and degenerative entities.

Materials and methods

This scoping review was conducted following the framework of Arksey and O'Malley (2005), the recommendations of Levac et al. (2010), and the PRISMA-ScR guideline (Tricco et al., 2018).

Research question (PCC framework): Population (P): pregnant women. Concept (C): serum uric acid levels and their association with obstetric outcomes. Context (C): placental-origin syndromes (preeclampsia, FGR, preterm birth, fetal death).

The guiding question was: "What evidence is available regarding the relationship between serum uric acid levels and preeclampsia, as well as other placental-origin obstetric syndromes?"

The search was conducted in October 2025 using PubMed, complemented by literature from ACOG and NICE guidelines. Search terms included ("uric acid" AND ("preeclampsia" OR "hypertensive disorders of pregnancy" OR "placental dysfunction" OR "fetal growth restriction" OR "maternal outcomes")).

Inclusion criteria comprised original studies, systematic reviews, and meta-analyses in humans, in English or Spanish, with quantitative data evaluating the relationship of uric acid with preeclampsia or associated obstetric syndromes (FGR, preterm birth, perinatal mortality, or placental dysfunction). Exclusion criteria included case reports, duplicates, studies with fewer than 30 participants, and articles without full-text access.

Study selection and data extraction were performed manually by the author, reviewing title, abstract, and full text. Recorded variables included publication year, methodological design, sample size, trimester of uric acid measurement, evaluated outcomes, and main results. Selected articles were organized into three thematic axes:

1) Physiological and pathophysiological aspects of uric acid
2) Association between uric acid and preeclampsia
3) Relationship with FGR and adverse perinatal outcomes

Results

Sixty-two articles were identified; 42 were excluded for various reasons, and 20 met inclusion criteria (Table 1).

Table 1 Study design Sixty-two articles

Author (year)	Country	Study design	Sample size	Trimester	Main findings
Bellos et al.[11]	Greece	Meta-analysis	>39,000	1st–3rd	Elevated uric acid levels in preeclampsia; diagnostic sensitivity of approximately 76% in the third trimester, particularly in severe disease.
Piani et al.[12]	Italy	Retrospective cohort	269	1st–3rd	Elevated uric acid/creatinine ratio associated with preeclampsia and preterm birth, independent of maternal confounders.
Li et al.[4]	China	Cohort study	23,194	1st and 3rd	Maternal hyperuricemia associated with small-for-gestational-age neonates even in the absence of hypertension.
Zhang et al.[13]	China	Prospective cohort	33,030	1st	High first-trimester uric acid/creatinine ratio associated with increased risk of preterm birth (OR 1.30).
Brien et al.[14]	Canada	Experimental study	—	—	Monosodium urate crystals induce IL-1–dependent placental inflammation and impaired trophoblast function, consistent with fetal growth restriction.
Lin et al.[7]	China	Observational study	180	—	Serum uric acid levels >357 μmol/L associated with increased risk of adverse obstetric and perinatal outcomes.
Arias-Sánchez et al.[10]	Spain	Narrative review	—	—	Uric acid as an independent diagnostic parameter is limited; predictive value improves when incorporated into multiparametric models.

Physiological aspects of uric acid

Uric acid is the final product of purine degradation in several organs and tissues, including the liver, intestines, kidneys, muscles, and vascular endothelium.[10] Purines may be endogenous, derived from nucleic acid breakdown, or exogenous, from protein-rich foods such as fatty meats, organ meats, and seafood, as well as fructose from fruits, processed foods with added sugars, and alcohol.[9] Adenine and guanine purines are degraded through different pathways to a common intermediate, xanthine, which is subsequently converted to uric acid by xanthine oxidoreductase.[10]

Uric acid plays a central role in free radical scavenging, exhibiting both pro-oxidant and antioxidant properties, as well as proinflammatory effects, causing damage across multiple systems.[9,10] Blood uric acid levels are determined by the balance between endogenous production,

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dietary intake, and renal and intestinal excretion rates.[10] Intestinal degradation accounts for one-third of elimination, while urinary excretion accounts for the remaining two-thirds.[9,10]

During pregnancy, the placenta and developing fetus significantly contribute to UA production.[3] The placenta, an organ with extensive cellular turnover, is a critical source of purines metabolized to uric acid via xanthine dehydrogenase and xanthine oxidase (XDH/XO).[10] Despite increased placental UA production early in pregnancy, maternal serum UA concentrations in the first and second trimesters are consistently lower than in nonpregnant individuals due to plasma volume expansion, increased glomerular filtration rate, and estrogen's uricosuric effects—explaining the marked gender dimorphism of gout, which is 20 times more frequent in men.[9] In the first trimester, serum levels decrease to values near or below 3 mg/dL and then gradually rise in the second and third trimesters toward prepregnancy ranges.[10]

Uric acid and preeclampsia

The association between preeclampsia and hyperuricemia was first described in the early 20th century.[10] Although serum UA levels are generally higher in women with PE than in those with uncomplicated pregnancies, not all women with PE present hyperuricemia.[9] The proportion varies between 40% and 90%, partly due to the lack of standardized criteria to define hyperuricemia during pregnancy.[3,9]

Preeclampsia is associated with increased tubular urate reabsorption stimulated by relative hypovolemia and angiotensin II action.[9,10] UA excretion is also reduced due to lactate competition in the proximal tubule. Additionally, PE is associated with endothelial dysfunction and reduced renal perfusion, leading to decreased glomerular filtration rate.[10] A notable paradox exists that increased tubular urate reabsorption occurs alongside reduced tubular protein reabsorption.[15]

There is also increased production associated with placental hypoxia/ischemia with xanthine oxidase activation and reactive oxygen species generation.[10] Elevated UA may inhibit nitric oxide production, leading to inadequate trophoblast invasion and impaired endothelial repair.[9] In PE, placental cellular turnover is markedly increased. Under hypoxic conditions, enhanced cellular destruction releases excess purines serving as XDH/XO substrates, resulting in elevated UA levels.[3]

The fetus may also serve as a substrate source for XDH/XO due to reduced placental blood flow limiting nutrient and oxygen delivery, causing fetal hypoxia.[9] Studies in hypoxic fetuses demonstrate increased blood concentrations of purine metabolites. Overexpression of XO in the preterm placenta may also result from inflammatory conditions related to immune dysregulation and release of apoptotic trophoblast debris into maternal circulation.[9]

During pregnancy, controlled inflammasome activation, particularly NLRP3, plays physiological roles in preparation for labor. However, in PE, exaggerated NLRP3 activation has been documented in placental tissues, chorioamniotic membranes, and myometrium, increasing caspase-1, IL-1β, and IL-18 expression.[10] This inflammatory imbalance promotes endothelial dysfunction, placental damage, and disruption of maternal–fetal homeostasis, contributing to hypertensive syndrome development.[10] Inflammasome activation may be induced by damage-associated molecular patterns (DAMPs), including uric acid, whose elevated plasma concentrations in PE are proposed as direct NLRP3 activators.[9] This interaction promotes proinflammatory cytokine maturation and pyroptotic cell death, exacerbating systemic inflammation. Thus, UA acts not only as a severity biomarker but may also play a pathogenic role via inflammasome-dependent inflammatory pathways, particularly NLRP3.3 Therapeutically, this understanding has prompted investigation of agents that modulate these pathways. XO inhibitors such as allopurinol (used cautiously in pregnancy) have shown potential to reduce UA levels and oxidative stress. Direct NLRP3 inhibitors and anti-inflammatory agents such as MCC950 and metformin have demonstrated efficacy in animal models by improving placental histopathology, reducing inflammatory cytokine production, and improving pregnancy outcomes; however, human application requires well-controlled trials.[3]

Clinical utility

A 2020 Greek meta-analysis titled "The prognostic role of serum uric acid levels in preeclampsia" compared UA levels between preeclamptic and healthy pregnant women across trimesters, including over 39,000 pregnancies.[8] Findings confirmed significantly elevated UA concentrations in PE compared with normotensive pregnancies regardless of clinical phenotype. Although higher levels were observed from the first and second trimesters in women who later developed PE, early predictive capacity was limited by wide prediction intervals and poor reproducibility. Third-trimester assessment showed higher diagnostic sensitivity (up to 76.7%), particularly in severe PE, eclampsia, and HELLP syndrome, consolidating UA as a marker associated with disease severity and complications.[8] Predictive performance for adverse perinatal outcomes was moderate, with sensitivities ranging from 67.3% to 82.7%, mainly from small, heterogeneous studies.[8] Clinical studies of UA-lowering treatments such as allopurinol and probenecid have not demonstrated clear benefits on maternal–fetal outcomes, possibly due to insufficient serum reduction.[8] Evidence suggests moderate utility of UA as an independent biomarker, improved when incorporated into multivariable predictive models with biochemical, clinical, and ultrasound markers.[8] Large prospective studies are required to establish standardized cutoffs and optimal gestational timing. Artificial intelligence tools may optimize predictive models, and future trials may clarify whether pharmacologic hyperuricemia management modifies PE evolution.[8]

A 2025 review from Murcia, Spain, "Uric Acid and Preeclampsia: Pathophysiological Interactions and the Emerging Role of Inflammasome Activation," concluded that the diagnostic value of UA as an independent parameter is limited due to significant interindividual variability influenced by renal function, placental physiology, and normal gestational adaptations.[10] Predictive performance improves substantially when incorporated into multiparametric models combining clinical and biochemical markers, especially valuable in low-resource settings.[10]

Piani et al.[12] evaluated the association between the serum uric acid/creatinine ratio (SUA/sCr) and PE development and adverse outcomes.[16] Reviewing 269 women with hypertensive disorders (2018–2022), SUA/sCr was consistently higher in PE across trimesters. Elevated third-trimester SUA/sCr was associated with increased odds of PE (OR 1.29), preterm birth (OR 1.23), and adverse neonatal composite outcome (OR 1.33), even after adjustment. Authors concluded that elevated SUA/sCr may serve as a risk marker, though prospective studies are needed.[16]

Uric acid and fetal growth restriction

According to the developmental origins of health and disease theory, maternal metabolism and intrauterine environment influence fetal growth and long-term health. Maternal UA level is a critical metabolic variable for fetal growth.[6] Maternal urate crosses the

placenta, with levels varying throughout pregnancy. Maternal UA decreases early but may increase significantly in the third trimester, making these metabolic alterations crucial for fetal growth.[6]

Most studies focus on hyperuricemia effects on birth weight in hypertensive pregnancies, with fewer examining normotensive women, despite biological plausibility linking elevated UA to reduced placental amino acid transport.[3,16]

Brien et al. (2017) demonstrated that monosodium urate crystals induce strong placental inflammatory responses via IL-1β. Human cytotrophoblast exposure increased IL-1β, IL-6, proinflammatory chemokines, apoptosis, and reduced sincitialización, impairing trophoblast function. Animal models showed placental inflammation with lower fetal weight, consistent with FGR. UA may act as an endogenous mediator of placental damage via IL-1–dependent inflammatory pathways.[7]

A Shanghai cohort (2017–2021) of 69,674 singleton births found inverse correlation between maternal UA and birth weight. Hyperuricemia (UA >360 µmol/L) was associated with increased risk of low birth weight and SGA, especially in the third trimester, with higher risk in hypertensive disorders.[3]

Li et al.[4] studied 23,194 normotensive pregnant women, finding higher first-trimester UA associated with increased SGA risk (aOR 1.67), persisting and increasing in the third trimester. Both marked increases and reductions were associated with adverse outcomes, suggesting early placental oxidative stress and endothelial dysfunction even without hypertension.[6]

Uric acid and adverse perinatal outcomes

Lin et al.[4] evaluated UA prognostic value in 287 women with hypertensive disorders. Elevated UA (≥357 µmol/L) was associated with severe PE, preterm birth, IUGR, fetal distress, low Apgar, and perinatal mortality, increasing with disease severity. UA serves as a useful marker to identify high-risk pregnancies due to endothelial and placental dysfunction.[4]

Zhang et al.[13] prospectively analyzed 33,030 singleton pregnancies, finding first-trimester SUA/SCr in the highest quartile associated with 30% higher preterm birth risk, showing dose–response relationship.[5]

Discussion

Preeclampsia is a hallmark of placental dysfunction and altered maternal endothelial response. Interest in uric acid stems from its dual role as a metabolic biomarker and potential mediator in inflammatory and oxidative physiopathology.

Emerging evidence suggests that the relationship between hyperuricemia and preeclampsia cannot be fully understood without considering the metabolic context of pregnancy, particularly the presence of metabolic syndrome and specific maternal phenotypes characterized by obesity, dyslipidemia, and chronic low-grade inflammation. These conditions share a common pathophysiological substrate of insulin resistance, which plays a central role in abnormal urate metabolism by increasing renal tubular reabsorption of urate and enhancing hepatic purine turnover. Consequently, women with insulin-resistant phenotypes exhibit a higher baseline risk of hyperuricemia even before the onset of placental dysfunction. In this framework, uric acid may represent not only a marker of placental ischemia but also a metabolic signal integrating maternal cardiometabolic risk with endothelial vulnerability. This perspective supports the concept that preeclampsia encompasses heterogeneous phenotypes in which metabolic factors modulate both susceptibility and clinical expression, reinforcing the need to interpret uric acid levels within a broader metabolic risk profile.

Recent literature supports a strong association between hyperuricemia and PE, with heterogeneous predictive value. Ten key points emerge:

1) UA elevates in most PE, especially severe/early-onset
2) Elevation may precede clinical onset with low specificity
3) Correlates with preterm birth and FGR risk
4) Stimulates NLRP3 inflammasome
5) UA/Cr improves outcome correlation
6) Reflects endothelial damage and oxidative stress
7) Higher levels associate with severity and poor prognosis
8) Meta-analyses show moderate diagnostic utility
9) Integrates into multiparametric models with angiogenic biomarkers
10) Guidelines recommend monitoring but not diagnosis.

Compared with earlier knowledge, current studies suggest UA is not merely a consequence of renal impairment but a plausible mediator. Unlike angiogenic biomarkers with higher predictive value, UA stands out for low cost and availability, especially in low-resource settings.

Strengths include integration of contemporary evidence (2020–2025) across designs and expansion beyond PE to other placental syndromes. Limitations include observational predominance, lack of standardized cutoffs, absence of randomized trials on UA reduction, and variability in laboratory methods and populations.[17–26]

Conclusion

Uric acid is a low-cost, accessible biomarker with strong biological plausibility within the spectrum of preeclampsia and other obstetric syndromes. Its elevation relates to placental dysfunction, oxidative stress, and inflammatory activation. While isolated diagnostic utility is limited, combining UA/Cr with angiogenic markers improves risk stratification. Guidelines support its use for biochemical monitoring rather than independent diagnosis. Prospective multicenter studies are needed to define gestational cutoffs and assess whether pharmacologic modulation alters clinical course. Additionally, UA quantification may correlate with PE biomarkers such as proteinuria/creatininuria and sFlt-1/PlGF ratios.

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Conflicts of interest

There are no conflicts of interest to report.

References

1. American College of Obstetricians and Gynecologists' Committee on Practice Bulletins—Obstetrics. Gestational hypertension and preeclampsia: ACOG Practice Bulletin No. 222. *Obstet Gynecol*. 2020;135(6):e237–e260.

2. Torres-Torres J, Espino-y-Sosa S, Martinez-Portilla R, et al. A narrative review on the pathophysiology of preeclampsia. *Int J Mol Sci*. 2024;25(14):7569.
3. Bainbridge SA, Roberts JM. Uric acid as a pathogenic factor in preeclampsia. *Placenta*. 2008;29(Suppl A):S67–S72.
4. Li H, Wang J, Zhang J, et al. Association between maternal serum uric acid and the risk of small for gestational age in women without hypertension. *J Matern Fetal Neonatal Med*. 2023;36(2):7961–7967.
5. Redman CW, Beilin LJ, Bonnar J, et al. Plasma urate measurements in predicting fetal death in hypertensive pregnancy. *Lancet*. 1976;1(7974):1370–1373.
6. Xiong Y, Zhang Q, Zhang M, et al. Association of maternal serum uric acid levels with low birth weight and small for gestational age. *BMC Pregnancy Childbirth*. 2024;24:261.
7. Lin J, Xu H, Ni L, et al. The value of serum uric acid in predicting adverse obstetric and perinatal outcomes. *Ginekol Pol*. 2018;89(6):276–281.
8. Many A, Hubel CA, Roberts JM. Hyperuricemia and xanthine oxidase in preeclampsia, revisited. *Am J Obstet Gynecol*. 1996;174(4):1288–1291.
9. Khaliq OP, Konoshita T, Moodley J, et al. The role of uric acid in preeclampsia: Is uric acid a causative factor or a sign of preeclampsia? *Curr Hypertens Rep*. 2018;20(9):80.
10. Arias-Sánchez C, Pérez-Olmos A, Reverte V, et al. Uric acid and preeclampsia: Pathophysiological interactions and the emerging role of inflammasome activation. *Antioxidants (Basel)*. 2025;14(8):928.
11. Bellos I, Pergialiotis V, Loutradis D, et al. The prognostic role of serum uric acid levels in preeclampsia: A meta-analysis. *J Clin Hypertens (Greenwich)*. 2020;22(5):826–834.
12. Piani F, Agnoletti D, Baracchi A, et al. Serum uric acid-to-creatinine ratio and risk of preeclampsia. *J Hypertens*. 2023;41(8):1617–1625.
13. Zhang E, Xu Q, Zhang Y, et al. Elevated first-trimester serum uric acid-to-creatinine ratio and risk of preterm birth. *Front Public Health*. 2024;12:1392661.
14. Brien ME, Renaud S, Boufaied I, et al. Uric acid crystals induce placental inflammation and alter trophoblast function via an IL-1–dependent pathway: Implications for fetal growth restriction. *J Immunol*. 2017;198(5):1874–1886.
15. American College of Obstetricians and Gynecologists. Hypertension in pregnancy: Report of the Task Force on Hypertension in Pregnancy. *Obstet Gynecol*. 2013;122(5):1122–1131.
16. Conrad KP, Gaber LW, Lindheimer MD. The kidney in normal pregnancy and preeclampsia. In: Chesley LC, editor. *Chesley's Hypertensive Disorders in Pregnancy*. 3rd ed. Academic Press Elsevier; 2009:321–344.
17. Bainbridge SA, Versen-Hoynvk F, Roberts JM. Uric acid and placental function. *Placenta*. 2009;30:195–200.
18. Meena R, Pachori P, Chaudhary S, et al. Serum uric acid levels in patients with preeclampsia compared with controls and their relationship to fetomaternal outcome. *Int J Reprod Contracept Obstet Gynecol*. 2019;8(6):2320–2326.
19. National Institute for Health and Care Excellence. *Hypertension in pregnancy: Diagnosis and management*. NICE guideline NG133. Updated April 17, 2023.
20. Magee LA. The hypertensive disorders of pregnancy: ISSHP classification, diagnosis and management recommendations for international practice. *Pregnancy Hypertens*. 2022;27:148–169.
21. Colmenares-Mejía CC, Quintero-Lesmes DC, Bautista-Niño PK, et al. Uric acid and risk of preeclampsia: Results from a large case–control study. *Sci Rep*. 2023;13:1627.
22. Samaha II, Donayeva A, Abdelazim IA, et al. Relationship between serum uric acid and severity of preeclampsia. *BMC Pregnancy Childbirth*. 2023;23:693.
23. Bhide A, Ganapathy R, Thilaganathan B. Is salivary uric acid, a putative biomarker of preeclampsia, predictive of hypertensive disorders of pregnancy? *BMJ Open*. 2025;15(6):e094421.
24. Adu-Bonsaffoh K, Quarshie DK, Fondjo LA, et al. Alterations in maternal serum uric acid levels in preeclampsia and association with adverse pregnancy outcomes: A cross-sectional study at Korle-Bu Teaching Hospital, Ghana. *Pan Afr Med J*. 2023;47:49.
25. Wagda H, Abdelsalam KA, Fahmy ZM, et al. Predictive value of serum uric acid levels for adverse maternal and perinatal outcomes in pregnant women with high blood pressure. *Hypertens Pregnancy*. 2020;39(4):291–301.
26. Pecoraro V, Trenti T. Predictive value of serum uric acid levels for adverse maternal and perinatal outcomes in pregnant women with high blood pressure: A systematic review and meta-analysis. *Pregnancy Hypertens*. 2020;20:62–71.

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