Pregnancy-related Acute Kidney Injury: A Narrative Review of Epidemiology, Pathophysiology, and Clinical Management

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Pregnancy-related acute kidney injury (Pr-AKI) is a severe complication of obstetric medicine that remains a significant cause of maternal and fetal morbidity and mortality. Although the burden has declined in many low- and middle-income countries (LMICs) following improvements in prenatal and obstetric care, its incidence has risen in high-income nations, due to older maternal age, the growing prevalence of diabetes and chronic hypertension, as well to more sensitive definition criteria and improved diagnostic tools. Physiological changes during pregnancy can lower the baseline serum creatinine level and may delay diagnosis.

This review summarizes current evidence on the epidemiology, pathophysiology, etiologic mechanisms, diagnostic difficulties, management, and outcomes of Pr-AKI. Globally, the condition demonstrates a paradox, i.e., decreasing incidence but persistent mortality in LMICs and increasing incidence in developed countries. Hypertensive disorders of pregnancy, particularly preeclampsia, eclampsia, and the HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome, have become the predominant causes worldwide. Distinct trimester-specific patterns, overlap with thrombotic microangiopathies and acute fatty liver of pregnancy, and the need to balance maternal and fetal health make Pr-AKI uniquely complex.

Improving Pr-AKI outcomes requires early recognition, pregnancy-specific diagnostic biomarkers, and coordinated multidisciplinary care. Strengthening antenatal surveillance, expanding access to renal replacement therapy in resource-limited settings, and providing structured postpartum follow-up are essential to reducing the global burden of Pr-AKI.

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INTRODUCTION

Pregnancy-related acute kidney injury (Pr-AKI) refers to a sudden loss of renal function during gestation or in the postpartum period. It is a sentinel event that reflects the quality of maternal health services. Despite progress in obstetric and nephrology practice, Pr-AKI remains a leading cause of preventable maternal death and a precursor to

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chronic kidney disease.[1-4]

Normal pregnancy produces extensive cardiovascular and renal adaptations. Glomerular filtration rate increases by roughly 50%, plasma volume expands, and systemic vascular resistance declines.[5-7] As a result, serum creatinine falls to about 0.4 to 0.6 mg/dL, therefore; a level of around 1 mg/dL, normal for a non-pregnant individual, may already indicate substantial impairment in a pregnant women. Awareness of these physiological changes is crucial to timely diagnosis and intervention.[8-10]

This review integrates findings from international studies to describe trends, mechanisms, clinical syndromes, management strategies, and outcomes of Pr-AKI. It highlights the differences between regions and underscores the importance of early detection and long-term follow-up.[11-23]

EPIDEMIOLOGY AND GOLBAL TRENDS

The epidemiology of Pr-AKI varies markedly across regions. In high-income countries, the incidence has risen over the past two decades due to delayed childbearing, higher rates of chronic disease, and improved case recognition.[10,24-7] In contrast, many LMICs have experienced a decline, although the overall burden remains far greater. In India, for example, septic abortion once accounted for more than half of Pr-AKI cases but now represents < 5% after the legalization of abortion and broader access to prenatal care. This is in contrast to the United States, where the incidence increased from about four to twelve per ten thousand deliveries between 2005 and 2015.[1,3,28-35]

Racial and socioeconomic disparities remain a significant factor in the incidence of Pr-AKI among different populations. Black and Native American women in the United States experience roughly 50% higher odds of Pr-AKI and 60% higher mortality than White women.[35-59] Structural barriers to timely prenatal evaluation, as well as differences in comorbidity profiles, contribute to this inequity.

Estimating the true incidence of Pr-AKI is challenging. Traditional definitions such as RIFLE, AKIN, and KDIGO are not validated in pregnancy and rely on relative rises in serum creatinine. Because physiologic creatinine levels are lower during gestation and baseline values are often unknown, many cases go unrecognized until renal dysfunction is advanced.[60-7] Routine measurement of renal indices during early pregnancy, particularly among those with higher risk facotrs or those who experience events such as hypertensive crises, bleeding, or sepsis, can improve surveillance.

PHYSIOLOGICAL ADAPTATIONS AND DIAGNOSTIC CHALLENGES

Pregnancy induces profound renal and systemic changes. Vasodilation increases cardiac output, and hormonal modulation by estrogen, progesterone, and relaxin leads to an expanded plasma volume and enhanced renal blood flow. Glomerular hyperfiltration begins early and peaks by mid-pregnancy, resulting in a physiological reduction in serum creatinine and urea concentrations.[68-71] Mild hydronephrosis or pyelocaliectasis is common in pregnancy due to progesterone-induced smooth muscle relaxation and mechanical compression of the ureters by the enlarging uterus.[68-70] Differentiating these physiologic changes from true renal pathology requires careful interpretation of laboratory parameters. Even minor elevations in serum creatinine may signify genuine kidney injury, as baseline creatinine levels are typically lower during pregnancy. Although monitoring urine output can provide useful information, its reliability is limited because of altered maternal fluid dynamics and potential therapeutic interventions. Notably, diuretics are generally avoided during pregnancy, as their use may precipitate oligohydramnios and compromise fetal well-being.[72,73]

Several biomarkers, including neutrophil gelatinase–associated lipocalin, soluble fms-like tyrosine kinase-1, and placental growth factor, have shown potential for differentiating pre–eclampsia–related dysfunction from intrinsic renal disease; however, their clinical validation and application remain limited.[73] Evaluation should include serial laboratory tests, assessment for proteinuria, and renal ultrasonography to rule out obstruction. Renal biopsy may be considered before approximately twenty-five weeks of gestation when histologic results would alter management; after this time, it is typically postponed until after delivery due to procedural risk.[68,69,74,75]

ETIOLOGY AND RISK FACTORS

The causes of Pr-AKI differ by trimester and socioeconomic contexts. In early pregnancy, dehydration from hyperemesis gravidarum and infection following unsafe abortion are predominant. Later in pregnancy, hypertensive disorders such as preeclampsia and eclampsia, the HELLP syndrome, acute fatty liver of pregnancy, and thrombotic microangiopathies become the principal etiologies.[76-84] Intra- and postpartum hemorrhages, puerperal sepsis, and amniotic-fluid embolism contribute during the peripartum period.

Advanced maternal age, chronic hypertension, diabetes mellitus, prior preeclampsia, and preexisting chronic kidney disease markedly increase susceptibility to develop Pr-AKI.[80,85-9] Socioeconomic deprivation, limited access to emergency obstetric care, and delayed referral further elevate the Pr-AKI risk. Early-pregnancy complications tend to be reversible with prompt rehydration or control of infection. In contrast, late-pregnancy and postpartum forms are often more severe and carry a greater risk of residual renal damage (Table 1).

Hypertensive disorders of pregnancy constitute the most frequent cause of Pr-AKI worldwide. Preeclampsia is defined by new-onset hypertension after twenty weeks of gestation with proteinuria or evidence of end-organ dysfunction.[81,83,90-3] When seizures develop, the condition is called eclampsia. The HELLP syndrome, which includes hemolysis, elevated liver enzymes, and low platelets, represents the most severe form of this disease spectrum. Renal involvement results from endothelial injury, vasospasm, and glomerular endotheliosis.[88,94-8]

Acute fatty liver of pregnancy typically occurs in the third trimester and is characterized by microvesicular fatty infiltration of hepatocytes due to defects in mitochondrial fatty acid oxidation. It presents with nausea, vomiting, abdominal pain, jaundice, and hypoglycemia. Because it shares clinical features with HELLP, the two disorders may coexist. Prompt delivery and supportive management can usually result in a full recovery.

Thrombotic Thrombocytopenic Purpura (TTP) and HELLP syndrome are important thrombotic microangiopathies (TMAs) leading to Pr-AKI. Although their clinical features overlap, differentiating them is crucial for management and prognosis. TTP, typically occurring in the late second or third trimester, results from severe ADAMTS13 deficiency (< 10% activity), which predisposes to microvascular platelet aggregation.[71,88,94-103]

It is characterized by thrombocytopenia, microangiopathic hemolytic anemia, and neurologic dysfunction, while renal involvement is usually mild to moderate but can cause severe AKI in up to 10% of cases. Plasma exchange is the mainstay of therapy, as delivery does not improve outcomes. In contrast, HELLP syndrome, a severe variant of preeclampsia, is marked by hemolysis, hepatic injury, and thrombocytopenia, often accompanied by hypertension and proteinuria. AKI occurs in 3 to 15% of affected women and may reach over 60% in severe cases. Prompt delivery of the fetus is the definitive treatment, typically leading to rapid maternal recovery and favorable renal outcomes. Compared with TTP or atypical HUS, HELLP-related AKI generally resolves completely after delivery, and absence of relapse on follow-up supports its distinction from TMAs persisting beyond pregnancy.[84,89,104-8]

CLINICAL MANAGEMENT AND THERAPEUTIC APPROACH

During the management of Pr-AKI, stabilization of both maternal physiology and protection of fetal health are required. A multidisciplinary team, including a nephrologist, obstetrician, intensive care specialist, pharmacist, and neonatologist, is often required to optimize care.[109-11] Initial treatment focuses on optimizing and maintaining hemodynamic stability and adequate renal perfusion. Hypovolemia should be corrected with isotonic balanced crystalloid fluids, while volume overload must be avoided in pre-eclamptic patients. Blood products are administered as necessary, and severe metabolic acidosis or hyperkalemia should be corrected promptly.[6,112-4]

In cases of severe preeclampsia, eclampsia, or HELLP syndrome, expedited delivery remains the definitive therapy. Antihypertensive medications, such as beta-blockers or hydralazine, are used to control blood pressure without compromising uteroplacental blood flow. Magnesium sulfate is given for seizure prophylaxis but requires close

Table 1. Treatments of Choice for the Syndromes Associated with Pr-AKI Risk

Condition	Specific Treatment
Preeclampsia (severe) / HELLP / AFLP	Prompt delivery of the fetus
TTP	Plasma exchange (initiated while ADAMTS13 levels are pending); Rituximab for refractory cases (with caution in the third trimester)
aHUS	Eculizumab, Plasma exchange
Septic abortion / UTI	Antibiotics; surgical removal of products of conception for septic abortion
Glomerulonephritis	Steroids and immunosuppressants (e.g., azathioprine, calcineurin inhibitors). Note that mycophenolate and cyclophosphamide are contraindicated.
Hemorrhage (Placental Abruption/PPH)	Control bleeding, volume resuscitation, and delivery
Obstructive Uropathy	Analgesics, stent, or nephrostomy

Abbreviations: TTP; thrombotic thrombocytopenic purpura, aHUS; atypical hemolytic uremic syndrome

monitoring because, in the case of declining kidney function, magnesium retention and potential toxicity risk increase.[7,115,116] When thrombotic thrombocytopenic purpura is suspected, plasma exchange should begin immediately. For cmHUS, early administration of complement inhibitors, such as antibodies targeting the membrane attack complex, could be lifesaving. Septic abortion and puerperal sepsis need broad-spectrum antibiotics and surgical evacuation of uterine contents when indicated (Table 1).[117-20]

Renal replacement therapy is initiated for the standard indications of refractory fluid overload, metabolic acidosis, hyperkalemia, or uremic symptoms. Both intermittent hemodialysis and continuous modalities are considered safe during pregnancy.[121] Evidence suggests that intensive dialysis improves fetal growth and extends gestational duration in women with advanced renal failure. Immunosuppressive therapy with corticosteroids or calcineurin inhibitors may be used when glomerulonephritis is confirmed, whereas agents such as cyclophosphamide and mycophenolate are avoided due to their teratogenicity.[6,109-11,116,120]

MANAGEMENT PRINCIPLES

The management of Pr-AKI is complex, requiring a multidisciplinary team and careful consideration of both maternal and fetal well-being. The core principles involve supportive care, management of complications, and treatment of the underlying cause (Table 2).[71,109,117,121]

MATERNAL AND FETAL OUTCOME

Pr-AKI remains closely linked to poor maternal and fetal outcomes.[76,89,98,100,122] Reported maternal mortality ranges from 4% to nearly 30%, depending on regions and their resource availability. The highest incidence of Pr-AKI-associated poor outcomes is observed in low- and middle-income countries where delayed referral and limited access to dialysis are common. In contrast, mortality in tertiary centers of high-income nations is usually < 5%. Cortical necrosis, often resulting from massive hemorrhage or sepsis, predicts irreversible renal failure and accounts for a disproportionate share of deaths.[84,88,94-8,100,104,105,123]

Renal recovery after Pr-AKI varies widely. Observational studies suggest that approximately two-thirds of survivors achieve complete recovery within six weeks of delivery, whereas up to one-quarter have partial recovery with persistent proteinuria or mild reductions in glomerular filtration rate. About 8% of patients with Pr-AKI show progress to chronic dialysis. Temporary renal-replacement therapy does not preclude recovery. Indeed, many women who require short-term dialysis regain full renal function postpartum. Even so, the episode carries long-term consequences: women with a history of Pr-AKI are nearly three times more likely to develop chronic kidney disease and twice as likely to experience cardiovascular events later in life.[84,98,99,103,124,125] Consequently, postpartum evaluation should include measurement of serum creatinine and urine protein, as well as counseling regarding hypertension, weight control, and lifestyle modification.[109,113]

Fetal outcomes mirror the severity of maternal illness. Perinatal mortality remains high, ranging from 25 to 35% globally. Prematurity, intrauterine growth restriction, and stillbirth are

Table 2. General Management Approach to Pr-AKI

Management Aspect	Key Principles and Actions
General Supportive Care	● Medication Review: Withhold potentially nephrotoxic drugs (e.g., NSAIDs, aminoglycosides), or replace with other agents. ● Dose Adjustment: Adjust maintenance doses of renally cleared medications (e.g., magnesium sulphate for preeclampsia). ● Treat Underlying Cause: Address hemorrhage with fluid/blood resuscitation, treat sepsis with antibiotics, and relieve any urinary tract obstruction.
Fluid Management	● Crucial but Challenging: The primary goal is to restore and maintain renal perfusion without causing fluid overload, a major iatrogenic risk. ● Hypovolemia: ○ Prioritize cautious isotonic fluid resuscitation to restore effective blood volume and renal perfusion. ○ Guide therapy by maternal hemodynamics, urine output trends, and resolution of clinical signs of hypoperfusion. ○ Avoid excessive fluid administration, as it increases the risk of pulmonary edema and worsens outcomes in preeclampsia and capillary leak states ● Acute Tubular Injury: ○ Restrict fluids to prevent volume overload once euvolemia is achieved. ○ Replace insensible losses and maintain even or slightly negative fluid balance based on output and weight changes. ○ Continue close monitoring for evolving electrolyte abnormalities or indications for renal replacement therapy
Pharmacological Interventions	● Diuretics (Furosemide): No proven benefit on renal outcomes and may worsen prognosis. Use should be limited to managing documented fluid overload, and it must be avoided in preeclampsia unless pulmonary edema is present. ● Low-Dose Dopamine: No evidence of benefit and carries substantial side effects. It plays no to minimal role in managing Pr-AKI.
Treatment of Underlying Obstetric Conditions	● Prompt Delivery: This is the treatment of choice for Pr-AKI caused by preeclampsia, HELLP syndrome, and acute fatty liver of pregnancy (AFLP) ● Targeted Therapies: Thrombotic microangiopathies (TMAs) do not necessarily require delivery and are treated with plasma exchange (TTP) or complement inhibition with complement inhibitors (cmHUS).
Renal Replacement Therapy (Dialysis)	■ Indications: ● Initiate RRT for standard maternal indications, as pregnancy does not alter thresholds for dialysis initiation. ● Specific indications include: ○ Refractory volume overload unresponsive to diuretics. ○ Hyperkalemia or metabolic acidosis not controlled with medical therapy. ○ Severe uremic complications such as pericarditis, bleeding diathesis, or encephalopathy. ○ Persistent severe AKI with marked azotemia (e.g., blood urea nitrogen >112 mg/dL) or prolonged oliguria/anuria despite conservative management. ■ Dialysis Strategy in Pregnancy: ● The standard of care is intensified dialysis, typically daily or near-daily sessions totaling >20 hours/week. ● Increased dialysis frequency improves maternal metabolic control and reduces fetal exposure to uremic toxins, leading to: ○ Higher live birth rates. ○ Greater birth weights and improved fetal growth outcomes. ● Dialysis prescriptions should be individualized to maintain optimal volume status, acid-base balance, and electrolyte homeostasis, while minimizing hemodynamic instability. ● Close maternal-fetal monitoring (including ultrasonography and fetal heart rate assessment) is essential throughout RRT.
Specialized Procedures	● Transcatheter Arterial Embolization (TAE): For uncontrollable postpartum hemorrhage, TAE is a safe and effective procedure. One study found that no patients (0 out of 47) developed post-contrast AKI after emergency TAE.
Renal Biopsy	● Rarely Necessary: Reserved for severe cases without a clear diagnosis where the results would change management during pregnancy, such as differentiating preeclampsia from other proteinuric diseases in the second trimester.

the most common complications. Many preterm births are iatrogenic, undertaken to preserve maternal health in severe preeclampsia or HELLP syndrome.[76,94,94,97,99,100,106] Neonates frequently

require intensive care for respiratory distress and low birth weight. Long-term follow-up of offspring from affected pregnancies suggests a higher lifetime risk of hypertension, chronic kidney diseases, and metabolic disorders, underscoring the intergenerational influence of maternal kidney injury.[76,89,98,100,103,104,126]

GLOBAL DISPARITIES IN CARE

Despite significant improvements in obstetric medicine, outcomes remain deeply unequal worldwide. In high-income countries, universal antenatal screening, rapid laboratory testing, and immediate access to dialysis have made Pr-AKI relatively rare and usually reversible. In contrast, in low-resource settings, a similar condition continues to be associated with preventable maternal death.[2,5,6,76,77,80-2,85,93,95] Many facilities lack basic laboratory capacity, dialysis equipment, or trained nephrologists. Women may travel long distances to reach tertiary centers, often arriving with advanced multi-organ failure.

Socioeconomic and cultural barriers compound the problem. In several regions, women delay seeking care because of limited autonomy, transportation costs, or fear of stigma. Unsafe abortion, still prevalent in some countries, remains a major driver of septic AKI. Broader determinants of health, including education, nutrition, and gender inequality, shape both the risk and the outcome of renal complications.[76-8,80-2,85,93,95,96,100-3,122,123,125,127,128]

Global initiatives, such as the International Society of Nephrology's 0by25 campaign, aim to eliminate preventable deaths from acute kidney injury by 2025. These initiatives emphasize that timely diagnosis and affordable dialysis must be available in every country.[76,78,96,102] Achieving this requires collaboration between governments, academic institutions, and professional societies. Investment in rural obstetric care, training of mid-level providers, and establishment of regional perinatal-nephrology networks are critical steps toward narrowing the gap.

EXPANDED EVIDENCE SUMMARY OF REPORTED CASES AND COHORTS

A synthesis of more than forty observational cohorts, representing thousands of pregnancies, provides a comprehensive picture of Pr-AKI worldwide. The incidence among hospital deliveries ranges from one in ten thousand in high-income regions to nearly 20% of obstetric admissions in some low-resource hospitals. Hypertensive disorders account for roughly half of all cases, followed by sepsis (15 to 20%) and hemorrhage (10 to 15%). Among women who require dialysis, approximately 70% recover renal function within six weeks postpartum, 10% remain dialysis-dependent, and the rest experience partial recovery.[76,78-80,85,129-32]

Regional trends highlight dramatic shifts over time. In India, septic abortion once represented the leading cause of Pr-AKI but now contributes to < 5% of cases, reflecting legal reform and improved infection control. Hypertensive disorders, particularly preeclampsia and HELLP syndrome, have become the dominant etiologies. In the United States, national datasets indicate a threefold increase in Pr-AKI incidence between 2005 and 2015, primarily attributed to older maternal age and comorbid conditions.[76-9,127] Maternal mortality in well-resourced centers is < 5%, whereas it exceeds 30% in hospitals lacking dialysis capacity. Fetal survival similarly varies, ranging from 90% in tertiary institutions to less than 60% in resource-limited environments. These differences demonstrate that Pr-AKI is not merely a biological disease but also a measure of social and systemic inequality.[76-80,83,85,94,124,127]

CONCLUSIONS

Pr-AKI remains a formidable challenge at the intersection of obstetrics, nephrology, and public health. Although incidence has declined in many developing regions, the persistence of high mortality and the rising trend in developed nations highlight its global relevance. Early recognition, prompt delivery when indicated, and coordinated multidisciplinary management are key to reducing complications. Preventive strategies should emphasize universal access to prenatal care, effective control of hypertension and diabetes before conception, and immediate management of obstetric emergencies.

In high-income countries, attention should focus on long-term follow-up after delivery to detect chronic kidney disease and cardiovascular risk.

In low- and middle-income regions, investment in health infrastructure, workforce training, and dialysis availability is essential to prevent avoidable deaths. Research priorities include the development of pregnancy-specific diagnostic criteria for acute kidney injury, validation of novel biomarkers, and investigation of long-term outcomes for both mothers and offspring.

In conclusion, Pr-AKI is a multifactorial disorder that reflects the broader landscape of maternal health. Its prevention and management demand not only clinical vigilance but also commitment to social equity and global cooperation. Through timely diagnosis, evidence-based care, and sustained investment in women's health, the burden of pregnancy-related acute kidney injury can be substantially reduced.

AUTHORS CONTRIBUTIONS

Mehdi Kashani, MD: Conceptualization, literature review, data synthesis, drafting of the manuscript, figure and table preparation, and critical revision

Juxiang Wang, MD: Literature review, regional and international data collection, and critical revision for intellectual content

Jiong Cui, MD: Methodological guidance, data verification, comparative analysis between LMIC and high-income settings, and manuscript editing

Kianoush B. Kashani, MD, MS: Senior supervision, conceptual design, expert input on nephrology and critical care aspects, and final approval of the manuscript

All authors read and approved the final version of the manuscript.

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Not applicable

Consent to Participate

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Consent to Publish

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Conflict of Interest

The authors declare no competing interests.

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