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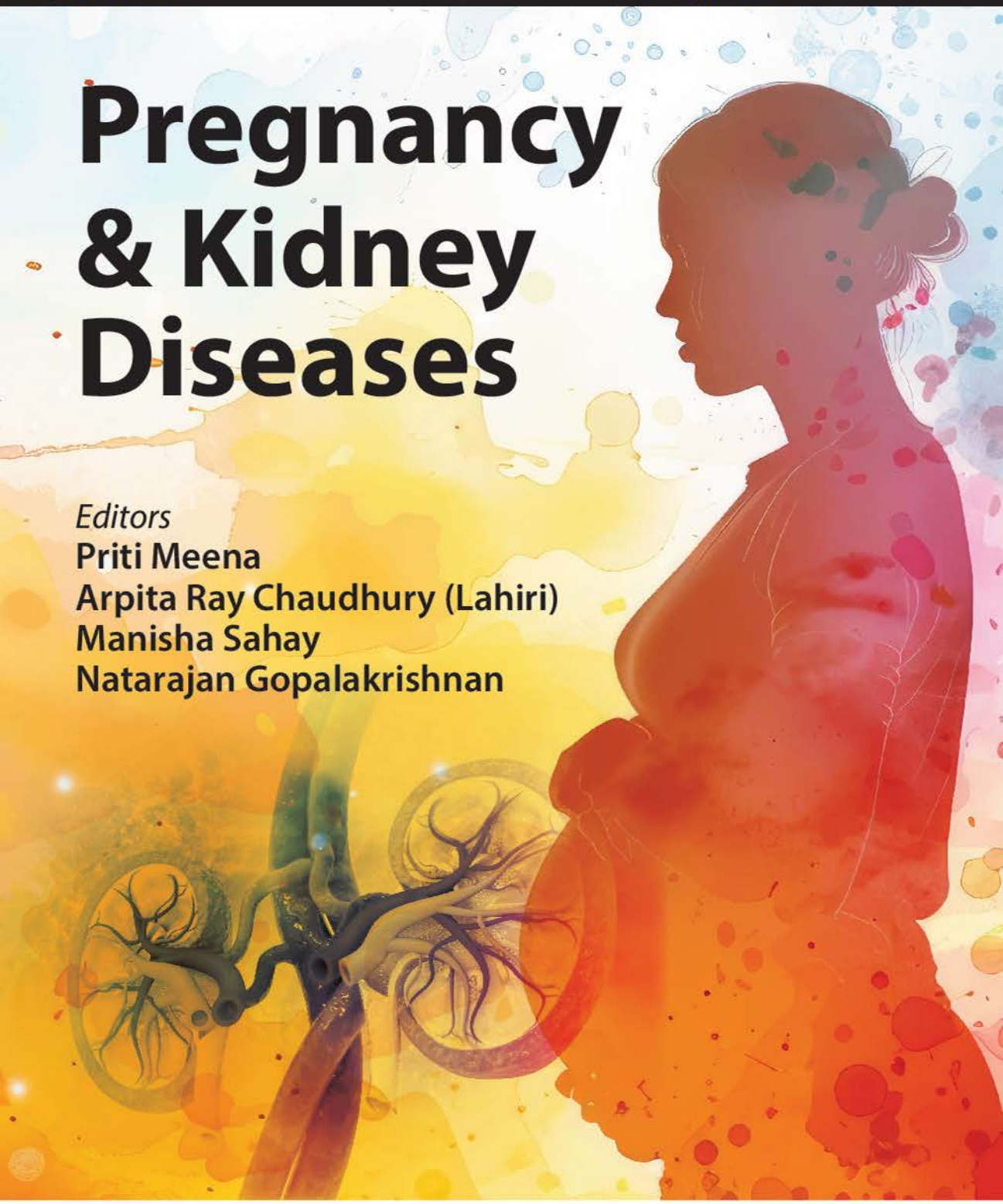

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CHAPTER 7

Acute Kidney Injury in Pregnancy

Arpita Ray Chaudhury (Lahiri)

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HIGHLIGHTS

- Acute kidney injury in pregnancy (Pr-AKI) is poorly defined as physiologic changes in pregnancy that lead to a significant increase in glomerular filtration rate (GFR) and 50% fall of creatinine, making it unsuitable to fit in standard AKI definition criteria by RIFLE (risk, injury, failure, loss, end-stage kidney disease), Acute Kidney Injury Network (AKIN), and Kidney Disease: Improving Global Outcomes (KDIGO), and a baseline creatinine is often not available.
- Pr-AKI has decreased globally because of improved obstetric care and better health care access; however, in developing countries, puerperal sepsis, hemorrhages, and severe preeclampsia remain a cause of poor maternal and fetal outcome. In developed world, pregnancy at a higher age exposes the mother to AKI due to many comorbidities like hypertension, diabetes, obesity, and chronic kidney disease.
- The thrombotic microangiopathies-associated causes of Pr-AKI, atypical hemolytic uremic syndrome (aHUS), thrombotic thrombocytopenic purpura (TTP), and antiphospholipid antibody (APLA)-related syndrome are rare but difficult to differentiate from relatively common causes like preeclampsia, HELLP (hemolysis, elevated liver enzymes, low platelet count) syndrome, or lupus nephritis flare in pregnancy. The clinical clue-based high suspicion index and differential-based battery of tests may be able to clinch the right diagnosis early. Early goal-directed therapy may improve both fetal and maternal outcome.
- Puerperal infections and hemorrhage may initiate a cascade of multifactorial AKI and may remain one of the most important preventable causes of PR-AKI. An interdisciplinary collaborative approach for sepsis prevention and early diagnosis is necessary to improve outcome.
- Management of Pr-AKI includes treatment of underlying cause, preserving kidney function, and providing supportive care for those who need dialysis and intensive care unit (ICU) support.

INTRODUCTION

Compared to acute kidney injury (AKI) in general population, obstetric AKI or AKI patients in pregnancy (Pr-AKI) are particularly prone to facing adverse outcomes, both short and long term.[1] It demands more attention as it accounts for risk of two lives and is largely preventable. Short-term outcome is a rise in maternal and fetal mortality and morbidity in resource-limited setting of the developing world. Severe AKI in pregnancy often leads to bilateral cortical necrosis and either progresses to immediate dialysis-dependent end-stage renal disease (ESRD) or gradually progresses to chronic kidney disease (CKD). The main reason lies in limited access to obstetric care and inadequate sepsis-prevention measures. Long-term

consequences include progression to CKD and increased cardiovascular risk in surviving patients and high healthcare resource utilization. In an Indian scenario, many studies have highlighted that puerperal sepsis is the main reason of increased maternal mortality, which is a highly preventable cause. Increased awareness and early diagnosis with well-adopted preventive measures may change the scenario in countries with high Pr-AKI prevalence, like India, Bangladesh, or Pakistan.

EPIDEMIOLOGY

Currently in India, majority of Pr-AKI occurs in the postpartum period or late third trimester. The postabortion sepsis of early pregnancy has decreased from 9–10% in 1980–1990 to 1.5–2% in 2014.[2] PIH remains important cause if the whole spectrum of AKI (both dialysis requiring and non requiring) is taken in account, because most of the AKI due to PIH is nondialysis requiring and resolves once the baby is delivered. However, when severe AKI or dialysis-requiring AKI is considered, puerperal sepsis and postpartum hemorrhage (PPH) remain main contributors along with currently rising diagnosis of pregnancy-related thrombotic microangiopathy (P-TMA). It is important to understand that Pr-AKI is often multifactorial, and difficult or delayed access to right health care often worsens the prognosis. In India, there are encouraging reports of declining incidence of Pr-AKI,[2,3] particularly due to decline of septic abortion and improved antenatal care (ANC) access, but postpartum sepsis still remains an important cause of mortality, and P-TMA [atypical hemolytic uremic syndrome (aHUS)/thrombotic thrombocytopenic purpura (TTP)] is increasingly being diagnosed as a cause of poor renal and maternal outcome of Pr-AKI.

Although a more sinister problem of low- to middle-income countries (LMIC), Pr-AKI is still considered as a global problem and two recent reports from Canada and US had shown a recent rise of AKI (an increase in the incidence of Pr-AKI from 2.4 per 10,000 deliveries in 1999–2001 to 6.3 per 10,000 deliveries in 2010–2011 in the US and 1.66 per 10,000 deliveries in 2003–2004 to 2.68 per 10,000 deliveries in 2009–2010) due to increasing incidence of preeclampsia pregnancy occurring in the setting of older maternal age and higher comorbidities (CKD, hypertensive disorders, diabetes, and obesity) along with higher detection of AKI. The studies also registered a higher incidence in native Americans and blacks where education is limited, thus compromising timely access to obstetric care, echoing a similar crisis in developing nations.[4,5]

DEFINITION AND DIAGNOSIS OF ACUTE KIDNEY INJURY IN PREGNANCY

Acute kidney injury in general population has well-validated diagnostic criteria evolving over time (2004–2007) by RIFLE (risk, injury, failure, loss, end-stage kidney disease) criteria,[6,7] Acute Kidney Injury Network (AKIN) criteria,[8] and Kidney Disease: Improving Global Outcomes (KDIGO) staging,[9] but these are not validated in pregnant population because of the unique physiological changes occurring during pregnancy.[10]

Pregnancy-related increase in blood volume and cardiac output with decline in systemic vascular resistance results in hyperfiltration, which gives rise to increased effective plasma renal flow; consequently, glomerular filtration rate (GFR) increases by 40–60%, with a decrease in creatinine value during pregnancy, usually 0.4–0.6 mg/dL, which confounds the detection of creatinine-based diagnosis of AKI in obstetric population. In an Indian study reporting 253 pregnant

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individuals with AKI, Pr-AKI was diagnosed based on sudden increase of >1 mg/dL in serum creatinine, oliguria/anuria >12 hours, and/or the need for dialysis.[2] A similar definition was picked up in other studies.[3,11] A creatinine prior to pregnancy is not available most of the time. In a clinically relevant condition, e.g., new-onset hypertension in pregnancy with low platelets, an increase of even smaller magnitude than that accepted in AKIN (0.3 mg/dL change accepted as stage 1) criteria, like 0.2 mg/dL, may be considered a significant pointer toward AKI [HELLP (hemolysis, elevated liver enzymes, low platelet count) or aHUS in the third trimester and postpartum] and may set up an alarm for a close follow-up with serial creatinine and relevant serologies. Renal ultrasounds that are performed to rule out postrenal causes of AKI or an underlying kidney disease may show hydronephrosis, which may be physiologic rather than pathologic. Serum complements estimation is important for diagnosing autoimmune diseases such as lupus nephritis (flare or first appearance) but may lose significance as serum complements are higher in pregnancy because of increased synthesis by the liver. Proteinuria also increases in pregnancy likely because of hyperfiltration, and higher normal has been set to 300 mg/day; therefore, the preeclampsia diagnosis includes urine protein–creatinine ratio (UPCR) >0.3 as a diagnostic criterion. It might be a reflection of tubular proteinuria too, as some studies have shown a rise in urinary retinol-binding protein (RBP) in pregnancy. Hence, albuminuria has not been the criterion for diagnosis. Increased UPCR in absence of hypertension which may be seen in 15% of normal pregnancy may mandate a close follow-up in a selective clinical scenario because pregnancy may be complicated by nephrotic syndrome as well. Kidney biopsy in pregnancy in early trimester may be of help to resolve a doubt between preeclampsia and nephrotic syndrome and may ask for a change in therapy. Late-trimester biopsy-related complications are more; people preferably may wait till the postdelivery period for biopsy.[12]

Many studies have arbitrarily considered a diagnosis of AKI when creatinine exceeds 1 mg/dL. Also, routine creatinine monitoring is not practiced as standard of care in pregnancy. All these together make early detection of AKI in pregnancy challenging and mandate a good clinical differential and blood biochemistry evaluation as soon as AKI is diagnosed in pregnancy.

ETIOLOGY OF ACUTE KIDNEY INJURY IN PREGNANCY

Causes of AKI in pregnancy may be unique to the causes related to pregnancy when an otherwise healthy woman suffers from hyperemesis gravidarum, septic abortion, puerperal sepsis, aHUS, HELLP syndrome, or preeclampsia or else it may be categorized as per causes similar in general population (prerenal, renal, and postrenal).

For *prerenal causes*, a hemodynamic compromise with volume depletion starts in the setting of hyperemesis gravidarum, septic abortion, pyelonephritis or postpartum sepsis, and antepartum or postpartum hemorrhage. There is a reversible reduction in GFR; left untreated, it leads to ischemic acute tubular injury [acute tubular necrosis (ATN)]. Usually, ATN is more common and reversible and renal function comes back in 7–21 days but in association with pregnancy, it has a predilection to progress to irreversible acute cortical necrosis (ACN) in most severe cases. Relatively rare causes of prerenal AKI include

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amniotic fluid embolism, acute fatty liver of pregnancy (AFLP), and chorioamnionitis.

Acute cortical necrosis is a pathologic diagnosis resulting from compromised vascular supply to kidney in a setting of severe hypotension with septic shock or obstetric hemorrhage or from intravascular thromboses in the interlobular and afferent arterioles diffusely involving the cortex while sparing the medulla. Often, it leads to dialysis-dependent CKD. Why ACN is fairly common in pregnancy-related AKI probably finds explanation in the fact that pregnancy is a hypercoagulable state with increased levels of coagulation factors with suppressed fibrinolysis and with any additional trigger, a series of complex and self-enhancing cascades are activated, like coagulation cascade, renin–angiotensin and complement pathways. Disseminated intravascular coagulation (DIC) may be the end result of such complex interactions, and renal recovery becomes unlikely.[13] The incidence of pregnancy-associated ACN, like overall pregnancy-related AKI, however, is decreasing. An Indian study showed a decrease in the incidence from 4.7% to 0.5% between the years 1984 and 2005.[14] While legalization of abortion has decreased the incidence of septic abortion, other causes like pregnancy-related TMA are increasingly diagnosed and both TMA and puerperal sepsis are coming up as potential causes of ACN in the postpartum period.[2,3]

Pregnancy-related *intrarenal* causes include the spectrum of diseases under hypertensive disorders in pregnancy like preeclampsia, AFLP, and HELLP syndrome. Other conditions which may be precipitated and worsened by pregnancy include pyelonephritis, sepsis, lupus nephritis, and TTP/complement-mediated aHUS. Whatever may be the initiating insult, a fullblown Pr-AKI is often multifactorial and AKI requiring dialysis often has a poor outcome leading to death or progression to dialysis-dependent CKD.[2,3]

Among *postrenal causes*, obstructive uropathy is a rare cause of Pr-AKI. The physiologic right-sided hydronephrosis occurs in up to 90% of pregnancies due to the compression of the ureter at the pelvic brim by the growing uterus, and progesterone induced smooth muscle relaxation, unless complicated by pyelonephritis this hydronephrosis causes neither decrease in urine output nor decline in GFR. Bilateral hydronephrosis, on the other hand, due to bilateral renal calculi, gravid uterus, especially with multiple gestations, polyhydramnios, and large fibroids can cause Pr-AKI. Post-delivery obstructive voiding symptoms have been reported after emergency cesarean delivery, following prolonged operation time, and postoperative analgesia that requires bladder catheterization. Iatrogenic injuries to the bladder and ureters from the cesarean section remain to be an uncommon cause for Pr-AKI. Postsurgical AKI when occurs due to breach in aseptic precaution, may result in grave consequences.

Causes of AKI in pregnancy are shown in **Figures 1 and 2**.

CLINICAL APPROACH TO PR-AKI

Medical History and Physical Examination

Whenever a Pr-AKI patient is seen in ward or emergency, both pregnancy-specific and pregnancy-unrelated AKI causes should be kept in mind. Medical history may reveal prior autoimmune diseases, like lupus nephritis, coexistent hypertension, and/or diabetes which are important, and history of nephrotoxic drugs like nonsteroidal

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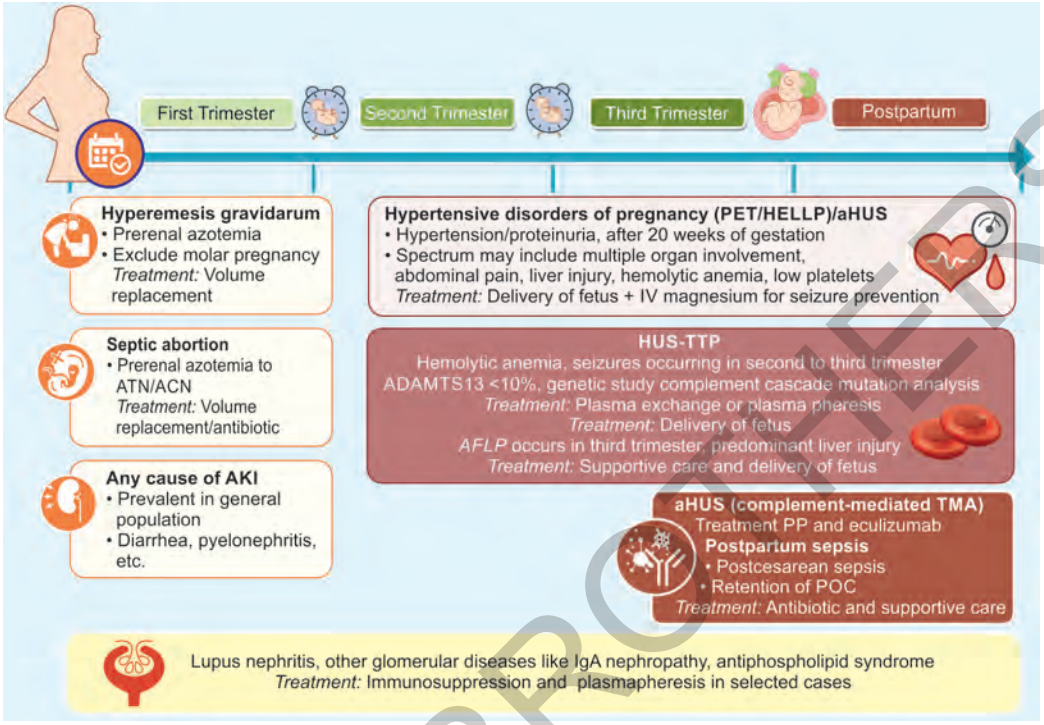

Fig. 1: Main causes of pregnancy-related AKI corresponding to timing during pregnancy with key diagnostic features. (ACN: acute cortical necrosis; AFLP: acute fatty liver of pregnancy; aHUS: atypical hemolytic uremic syndrome; AKI: acute kidney injury; APS: antiphospholipid antibody syndrome; ATN: acute tubular necrosis; HELLP: hemolysis, elevated liver enzymes, low platelets; PET: preeclamptic toxemia; POC: product of conception; TMA: thrombotic microangiopathy; TTP: thrombotic thrombocytopenic purpura)

Courtesy: Dr Priti Meena

anti-inflammatory drugs (NSAIDs) as post-lower uterine cesarean section analgesia may worsen the outcome of a resolving preeclampsia. The onset of preeclampsia is usually after 20 weeks; new-onset hypertension before 20 weeks may raise the probability of molar pregnancy, preexisting undiagnosed CKD, and inflammatory conditions like antiphospholipid antibody (APLA) syndrome (APS). Prior history of a similar episode or family history may be found in preeclampsia, congenital, or recurrent TTP.

Physical examination may detect volume depletion, dehydration, signs of other organ involvement in multisystem diseases or systemic vasculitis appearing in pregnancy, signs of enlarged tender liver in AFLP, heart failure with pregnancy and heart or kidney disease, signs of volume overload, severe anemia, and jaundice in Pr-TMA diseases.

Investigations

Urine examination may provide important information; proteinuria alone in a setting of new onset hypertension is most likely due to preeclampsia; but if associated with hematuria should bring in other glomerular diseases like lupus nephritis, systemic vasculitis and IgA nephropathy under differential diagnosis. A direct Coombs positive anemia, low C3, ANA, and anti-dsDNA positivity may

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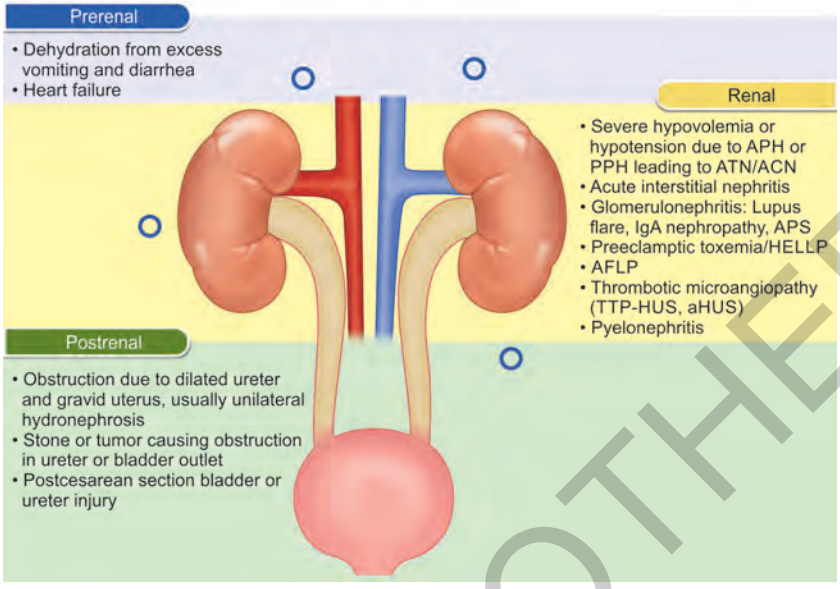

Fig. 2: Causes of AKI in pregnancy. (ACN: acute cortical necrosis; AFLP: acute fatty liver of pregnancy; aHUS: atypical hemolytic uremic syndrome; AKI: acute kidney injury; APH: antepartum hemorrhage; APS: antiphospholipid antibody syndrome; ATN: acute tubular necrosis; HELLP: hemolysis, elevated liver enzymes, low platelets; PET: preeclamptic toxemia; PPH: postpartum hemorrhage; TMA: thrombotic microangiopathy; TTP: thrombotic thrombocytopenic purpura)

Courtesy: Dr Priti Meena

confirm the diagnosis of lupus but other glomerular disease exclusion by renal biopsy. Baseline urine and blood biochemistry along with imaging is able to pick up diagnosis. The investigations done are:

- Dipstick urinalysis and microscopic analysis of sediment.
- Quantitation of protein excretion by either 24-hour urine collection or by protein-to-creatinine ratio.
- Urine culture
- Hemoglobin level and platelet count with peripheral blood smear to evaluate for microangiopathic hemolysis and thrombocytopenia.
- Total, direct, and indirect bilirubin concentration, haptoglobin, and lactate dehydrogenase (LDH) to evaluate for hemolysis.
- Serum aspartate aminotransferase (AST) and/or alanine aminotransferase (ALT) to assess liver injury in infection-associated and HELLP syndrome.
- *In specific cases:* Evaluation of the complement levels, autoantibodies of specific association like ANA, anti-ds DNA, ANA profile, perinuclear anti-neutrophil cytoplasmic antibody (p-ANCA) and cytoplasmic ANCA (c-ANCA), ADAMTS13, and antibody to ADAMTS13.
- *Imaging:* USG abdomen and thorax. The need of limited radiation exposure may be a challenge to establish diagnosis. Transvaginal ultrasonography, MRI, and low-dose CT remain the alternatives if needed.

TIMING OF ACUTE KIDNEY INJURY

The timing of AKI during pregnancy may serve as an important clue for making a differential diagnosis and plan relevant

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investigations. After significant decline in septic abortion, AKI that occurs in the first trimester in developing countries is mostly prerenal and may include any cause occurring in general population. First-onset lupus or flare of lupus nephritis may occur, and hyperemesis may be another pregnancy-specific cause. Any hypertensive disorder in early pregnancy should raise the suspicion of undiagnosed pre-existing hypertension and CKD and needs exclusion of molar or twin pregnancy. Urine microscopy and proteinuria evaluation may be helpful to identify the underlying kidney disease or identify the need for biopsy which may change the management of the patient. Most of the AKI occurring in the second and third trimesters are due to hypertensive complications such as preeclampsia/HELLP and TTP/HUS, where genetic predisposition also plays a role. Abruption placentae, severe hemorrhage, or DIC also decreased in frequency in India due to improved access to obstetric care. AFLP with AKI occurring in the third trimester may contribute to increased maternal mortality and morbidity. This relatively rare disease stands as an important differential with preeclamptic toxemia (PET)/HELLP. Atypical HUS, however, generally occurs late in the third trimester or postpartum after delivery and clinically stands as a differential of HELLP syndrome or puerperal sepsis. Pregnancy-related TTP may present in the early third trimester, earlier in timing than aHUS, and neurologic symptoms may be more prominent than renal involvement. Diagnosis of aHUS in the postpartum period often requires a high degree of suspicion, as AKI in this situation becomes multifactorial. Evaluation of intravascular hemolysis (anemia, thrombocytopenia, high LDH, low haptoglobin, and schistocytes in peripheral blood smear) may clinch the diagnosis early. Many of these diseases exhibit overlapping features, such as preeclampsia/HELLP, lupus nephritis, TTP/HUS, and AFLP.

Timing of recovery is also important. PE/HELLP/AFLP often tends to resolve following delivery, while Pr-aHUS does not.

Figure 3 shows signs and symptoms of overlapping syndromes of AKI.

ACUTE KIDNEY INJURY IN SPECIFIC SITUATIONS

Pregnancy is considered as a high-risk period for the development of different kinds of TMA. Three major syndromes including TTP, PE/HELLP, and aHUS may account for Pr-TMA

During pregnancy, immune tolerance toward the semiallogeneic fetus is necessary for the physiologic progression of gestation and the postpartum period is associated with the recovery of a pregestational immuno-competent state, where autoantibody-mediated diseases [e.g., acquired TTP, lupus nephritis, APLA syndrome, aHUS, complement mediated TMA (c-TMA)] may make their first or relapsing appearance.

The diagnostic challenge and therapeutic dilemma lie in their overlapping clinical features and at least sometimes, coexistence of more than one pathogenic mechanism. Each disorder terminates in the final event of endothelial damage and fibrin thrombi formation in the microcirculation. Both fragmented RBCs and platelet aggregates interfere with blood supply and cause ischemic injury to the target organs (kidney, brain, gastrointestinal tract, or liver). As the treatment differs, early diagnosis of the main pathologic mechanism may be crucial to intervene and improve fetal and maternal outcome.

Box 1 shows characteristics of TMA.

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	PET/HELLP	TTP-HUS	aHUS	AFLP	APS	Lupus flare
Timing in pregnancy	>20 weeks	Higher in second trimester	Higher in postpartum	Last trimester	All gestational ages	All gestational ages
Blood pressure	3+	0–3+	2+	0–2+	0–3+	0–3+
Neurological involvement	0-3+	2+ to 3+	0–1+	0	0–3+	0–3+
Fever	0	1+ to 3+	0	1+ to 3+	2+	2+
Schistocytes in PBS >2%	0–2+	3+	2+	0–1+	2+	0
Thrombocytopenia	0–3+	2+ to 3+	3+	1+ to 3+	1+ to 2+	1+ to 2+
ADAMTS13 level	0–1+	3+	1+	0	0	0
Elevated liver enzymes	0–3+	0–1+	0–1+	2+ to 3+	0–1+	0
Proteinuria	0–1+	0–1+	0–1+	1+	0–1+	1+ to 3+
Treatment	Delivery of fetus	Plasmapheresis	Plasmapheresis and eculizumab	Delivery of fetus	Aspirin + anticoagulation	Immunosuppression

Fig. 3: Signs and symptoms of overlapping syndromes of acute kidney injury. (ADAMTS13: alpha disintegrin and metalloprotease with a thrombospondin type 1 motif member 13; AFLP: acute fatty liver of pregnancy; aHUS: atypical hemolytic uremic syndrome; APS: antiphospholipid antibody syndrome; HELLP: hemolysis, elevated liver enzymes, low platelets; PBS: peripheral blood smear; PET: preeclamptic toxemia; TTP: thrombotic thrombocytopenic purpura)

Notes: Ratings: 0 = Unlikely or not present, 1+ = Mild or low likelihood, 2+ = Moderate likelihood, 3+ = High likelihood.

BOX 1: Characteristics of thrombotic microangiopathy (TMA).

TMA is defined by a pathological pattern, endothelial cell swelling, detachment from the basement membrane, thrombi in the microcirculation, and, in the kidney, "double contour" aspects of the glomerular basement membrane and a dissolution or attenuation of the mesangial matrix (mesangiolysis); it is however usually diagnosed based on a clinicopathological triad:

- Peripheral thrombocytopenia (platelet count $<100 \times 10^9$/L)
- Mechanical hemolytic anemia (hemoglobin <10 g/dL, LDH >upper limit of normal, undetectable haptoglobin, schistocytes on blood smear)
- Organ injury

(LDH: lactate dehydrogenase)

Thrombotic Thrombocytopenic Purpura (Box 2)

Thrombotic thrombocytopenic purpura is a rare hematologic condition that is caused by a severe deficiency of the von Willebrand factor (vWF)-cleaving protease ADAMTS13, resulting in excess of highly adhesive unusually large vWF multimers, which causes inappropriate platelet clumping in the microvasculature under high shear stress conditions, leading to severe thrombocytopenia and occlusive microangiopathy. The classical description of TTP consists of the pentad of microangiopathic hemolytic anemia (MAHA), thrombocytopenia, fever, and renal and neurological dysfunction, though this pentad is present in only 40% of cases. TTP is classified as

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