

PREGNANCY – AKI [PRAKI]

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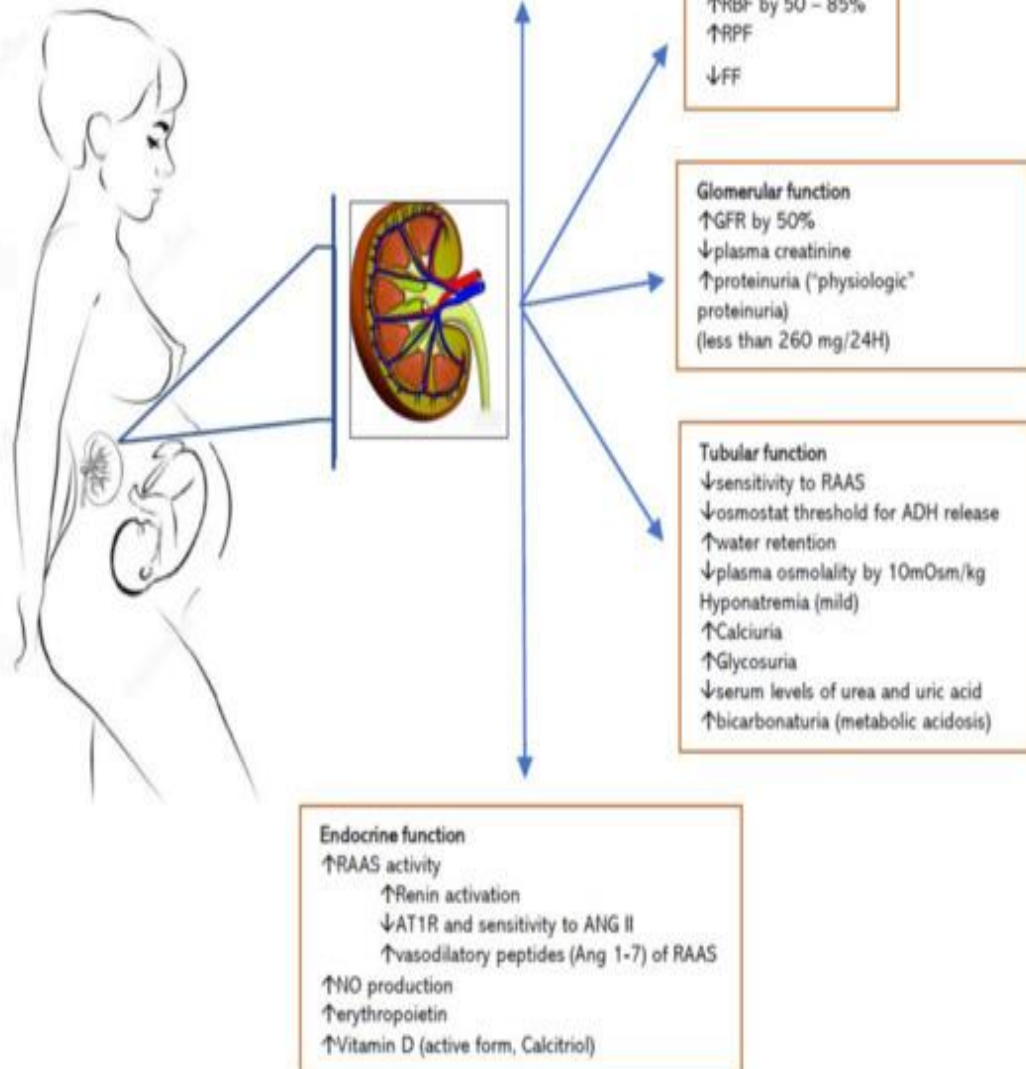
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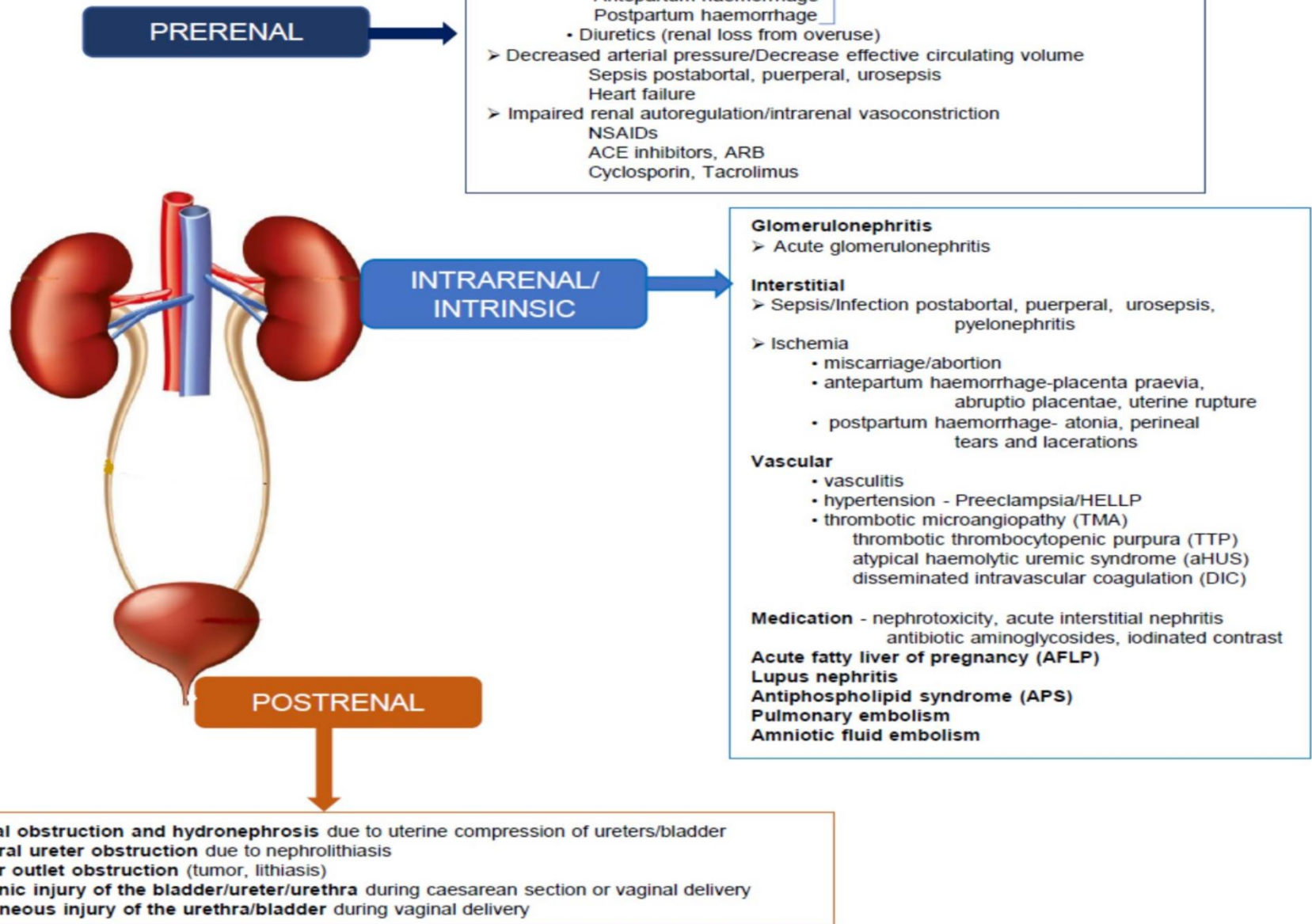
INTRODUCTION

- Pregnancy-related acute kidney injury (Pr-AKI) is a heterogeneous disease entity due to varying underlying etiologies.
- P-AKI in developing countries accounts for 5%–20% of total AKI population.
- Pr-AKI commonly occur in postpartum rather than the post- abortal period, reflecting a decline in septic abortions.
- 24 hr creatinine clearance is closest estimate of GFR in pregnancy.
- Diagnosis of AKI in pregnant women (any one of the three)
 - (1) Sudden increase in serum creatinine >1 mg/dl,
 - (2) Oliguria/anuria
 - (3) Need for dialysis.

PHYSIOLOGICAL CHANGES IN PREGNANCY



CAUSES OF P-AKI



1st trimester2nd trimester3rd trimester

D Postpartum

Septic Abortion**Clinical Features**

- Prerenal azotemia or ATN

Treatment

- Volume resuscitation, antibiotics

Hyperemesis Gravidarum**Clinical Features**

- Prerenal azotemia or ATN
- Evaluate for molar pregnancy

Treatment

- Volume resuscitation

Preeclampsia/ HELLP**Clinical Features**

- Hypertension and proteinuria* after 20 weeks of gestation
- Headache, visual disturbance, seizures, abdominal pain
- Hemolytic anemia, transaminitis, thrombocytopenia, high LDH

Treatment

- Delivery, i.v. magnesium for seizure prevention

TTP/aHUS**Clinical Features**

- TTP more common in 2nd/3rd trimester, aHUS more common in postpartum period
- Neurological involvement is more common in TTP than aHUS
- Hemolytic anemia, thrombocytopenia, elevated LDH and bilirubin

Laboratory testing

- TTP: ADAMTS-13 activity <10%
- aHUS: genetic testing for complement cascade gene mutations

Treatment

- TTP: plasma exchange
- aHUS: plasma exchange + eculizumab

Acute Fatty Liver of Pregnancy**Clinical Features**

- Nausea, vomiting, abdominal pain, jaundice, ascites
- Transaminitis, low platelets, hypoglycemia, lactic acidosis

Laboratory Testing

- Maternal and fetal testing for LCHAD gene mutation

Treatment

- Delivery, plasmapheresis and/or liver transplant in severe cases

Lupus nephritis and/or Antiphospholipid antibody syndrome**Clinical Features**

- Dysmorphic red blood cells on urine sediment, extra-renal lupus manifestations
- Low complements, positive anti-dsDNA, anti-cardiolipin antibodies, and/or anti-β₂ glycoprotein antibodies
- Kidney biopsy is only recommended if pathology will change management

Treatment

- Lupus nephritis: steroids + hydroxychloroquine + azathioprine/tacrolimus
- Antiphospholipid antibody syndrome: aspirin +/- low molecular weight heparin

PREECLAMPSIA

- **Definition** : Characterized by
 - ✓ New-onset hypertension (blood pressure $>140/90$ mmHg)
 - ✓ Proteinuria (>300 mg/dl) after 20 weeks of gestation.
- **Eclampsia** is defined as preeclampsia with the presence of seizure.

Table 5: Severe feature of preeclampsia (one or more of these findings)

Systolic blood pressure ≥ 160 mmHg or diastolic blood pressure ≥ 110 mmHg on two occasions at ≥ 4 h apart while the patient is on bed rest

Platelet count $<100,000/\text{mm}^3$

Elevated liver enzymes (twice normal concentrations)

Renal insufficiency (serum creatinine concentration >1.1 mg/dl or doubling of serum creatinine concentration) or oliguria (<500 ml in 24 h)

Pulmonary edema or cyanosis

New-onset cerebral or visual disturbances

Severe persistent right upper quadrant or epigastric pain

Clinical feature	HELLP*	AFLP**	aHUS***	TTP
Time of onset	3T	3T	Postpartum	2T/3T
Hypertension	80%-100%	25%-50%	+	0/+
AKI	Mild/moderate	Moderate	Severe	Mild/moderate
Renal prognosis	Recovery	Recovery	76% ESRD	Fair
CNS findings	+	Absent	Absent	Dominant
Hemolytic anemia	+	0/+	+	++
Thrombocytopenia	+	0/+	++	++
Coagulopathy	0/+	+	0	0
Liver transminases increase	++	++	0	0
LDH (IU/L)	+	0/+	++	++
Ammonia	Normal	High	Normal	Normal
ADAMTS-13 activity <10%	0	0	+	++
Alternative complement pathway	0/+	0/+	++	0
Management	Support measures/ delivery	Support measures/ delivery	Plasma infusion/ exchange	Plasma infusion/ exchange
Effect of delivery on diseases	Recovery	Recovery	None	None

*Urine sediment is bland in preeclampsia/HELLP syndrome, **Coagulopathy, hepatic injury, and hypoglycemia are the key feature of AFLP, ***Isolated LDH increase with normal hepatic transaminase is characteristics of HUS/TTP (P-TMA). 0: Absence, 0/+: Occasionally present, +: Sometimes present, ++: Always present, HELLP: Hemolysis, elevated liver enzymes, and low platelet count, AFLP: Acute fatty liver of pregnancy, aHUS: Atypical hemolytic-uremic syndrome, TTP: Thrombotic thrombocytopenic purpura, AKI: Acute kidney injury, CNS: Central nervous system, LDH: Lactate dehydrogenase, ESRD: End-stage renal disease, P-TMA: Thrombotic microangiopathy of pregnancy

RENAL CORTICAL NECROSIS

- Rare condition resulting from severe reduction of renal perfusion caused by vascular spasm, microvascular injury, or intravascular coagulation.
- **Abrupt onset** of oliguria, gross hematuria, flank pain and hypotension.
- Causes :Placental abruption , placenta previa , septic abortion . prolonged intrauterine fetal death and amniotic fluid embolism.
- **Hypercoagulable state** with increased level of coagulation factors with repressed fibrinolytic state.
- **Prognostic factors** : extent of necrosis, duration of oliguria, and severity of associated conditions.
- **Ultrasound: Hypoechoic Rim and Renal Cortical Rim Sign**: The hypoechoic rim seen on ultrasound, potentially with an echogenic (brighter) layer outside the rim.
- **CT Scan: Lack of Cortical Enhancement and Reverse Rim Sign**: A specific CT finding where the cortex appears hypo attenuating (darker) and non-enhancing, while the medulla enhances.
- **Dialysis as indicated and treatment of the underlying diseases.**

HD Prescription during Pregnancy

Frequency	5–6 time/week
Duration of dialysis	>36 h/week (>6 h/day)
Dry weight	Increase by 0.5 kg/week during the second and third trimesters
UF rate	6–8 mL/kg/h
Dialysate	K = 3 mmol/L, Ca = 1.5 mmol/L, HCO ₃ = 28–32 mmol/L
Anticoagulation	Low-dose unfractionated heparin

HD: hemodialysis; UF: ultrafiltration.