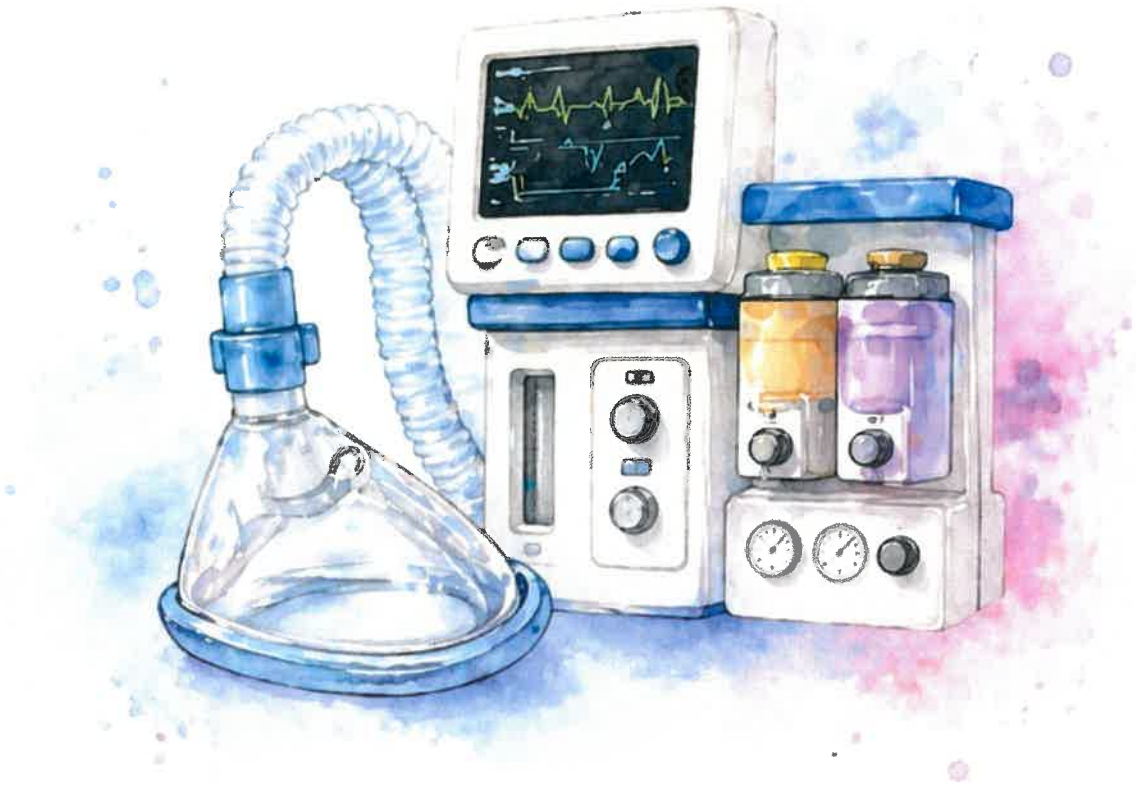
NEET SS ANESTHESIA

Updated Notes 2026



CRITICAL CARE PART-2

Instructions

- The notes must be used in conjunction with the Marrow Programme for Anaesthesia Residents videos and should not be considered standalone material.

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- This set contains notes of all main videos published in the app until March 2026. You can find notes for any additional videos published after this date within the app under the videos section.

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ACUTE KIDNEY INJURY : PART I

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Background

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- 1802 : William Heberden → Described acute kidney injury as Ischuria Renalis.
- 2004 : Bellomo et al → AKI (RIFLE criteria).
- AKIN criteria (2007).
- KDIGO criteria (2012).
- Acute kidney injury (AKI) : Syndrome characterized by a rapid deterioration of kidney function occurring within hours or days.

Classification :

- RIFLE : ADQI Group, 2004.
- AKIN : AKIN Network, 2007.
- KDIGO : KDIGO Group, 2012.

	RIFLE	AKIN	KDIGO
Diagnostic criteria			
		Increase in serum creatinine of ≥0.3 mg/dL or ≥50% within 48 hours OR Urine output of <0.5 mL/kg/hour for >6 hours	Increase in serum creatinine of ≥0.3 mg/dL within 48 hours or ≥50% within 7 days OR Urine output of <0.5 mL/kg/hour for >6 hours
Staging criteria			
Risk (RIFLE) or stage 1 (AKIN/KDIGO)	Increase in serum creatinine of 50 to 99% OR Urine output of <0.5 mL/kg/hour for 6 to 12 hours	Increase in serum creatinine of ≥0.3 mg/dL or 50 to 100% OR Urine output of <0.5 mL/kg/hour for 6 to 12 hours	Increase in serum creatinine of ≥0.3 mg/dL or 50 to 99% OR Urine output of <0.5 mL/kg/hour for 6 to 12 hours
Injury (RIFLE) or stage 2 (AKIN/KDIGO)	Increase in serum creatinine of 100 to 199% OR Urine output of <0.5 mL/kg/hour for 12 to 24 hours	Increase in serum creatinine of >100 to 200% OR Urine output of <0.5 mL/kg/hour for 12 to 24 hours	Increase in serum creatinine of 100 to 199% OR Urine output of <0.5 mL/kg/hour for 12 to 24 hours
Failure (RIFLE) or stage 3 (AKIN/KDIGO)	Increase in serum creatinine of ≥200% OR Increase in serum creatinine by >0.5 mg/dL to > 4.0 mg/dL OR Urine output of <0.3 mL/kg/hour for > 24 hours or anuria for >12 hours OR Initiation of renal replacement therapy	Increase in serum creatinine of >200% OR Increase in serum creatinine by > 0.5 mg/dL to ≥4.0 mg/dL OR Urine output of <0.3 mL/kg/hour for > 24 hours or anuria for > 12 hours OR Initiation of renal replacement therapy	Increase in serum creatinine of ≥200% OR Increase in serum of ≥0.3 mg/dL to ≥4.0 mg/dL OR Urine output of <0.3 mL/kg/hour for ≥24 hours or anuria for ≥12 hours OR Initiation of renal replacement therapy
Loss (RIFLE)	Need for renal replacement therapy for >4 weeks		
End stage (RIFLE)	Need for renal replacement therapy for >3 months		

KDIGO definition of acute kidney injury :

Stage	Creatinine criteria	Urine output criteria
1	Cr 1.5-1.9 times baseline, OR Cr increase > 0.3 mg/dL	< 0.5 ml/kg/hr x 6-12 hours
2	Cr 2-2.9x baseline	< 0.5 ml/kg/hr for > 12 hours
3	Cr > 3x baseline, OR Cr > 4 mg/dL, OR Initiation of dialysis	< 0.3 ml/kg/hr for > 24 hours, OR Anuria > 12 hours

Patients are staged based on the single most concerning feature.

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Pitfalls :

Shortcoming	Potential solution
1. Inability of S. Creat to detect changes at high GFR level	1. Sequential creatinine clearances (e.g, with urine collections of 2-8 h)
2. S. Creat is a late marker of decreased GFR	2. Biomarkers of kidney damage/cystatin C/more frequent measurement of S. Creat
3. Critical illness may confound diagnosis by creatinine criteria	3. Correction of S. Creat for fluid balance/cystatin C
4. Requirement for baseline S. Creat with different methods to handle missing baseline	4. Thorough search for baseline/reporting applied method in literature
5. S. Creat is a measure of function not of tissue damage and may miss subclinical AKI	5. Combining S. Creat with biomarkers of kidney damage
6. Urine output criteria are less frequently used, lack specificity, and may be too liberal	6. Consistent use of urine output criteria and definition of more stringent criteria
7. KDIGO definition does not provide insight into pathophysiology	7. Use of biomarkers that reflect different pathophysiological mechanisms
8. Absence of gold standard	8. No solution for the time being
9. Different criteria for similar stage may have different association with outcome	9. Consensus criteria for RRT initiation/detailed description of AKI criteria
10. KDIGO definition is unreliable for assessment of renal recovery	10. Use of cystatin C/assess recovery by S. Creat after a sufficient time from critical illness (ie, after correction of sarcopenia and fluid overload)

AKI Spectrum

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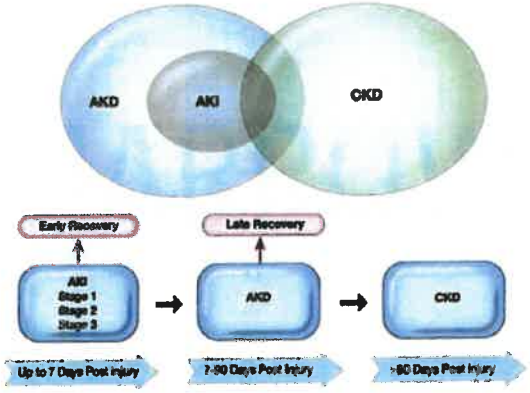
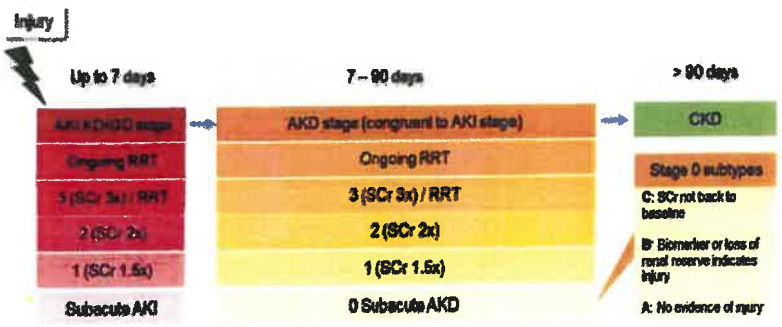


- Early recovery : S.creatinine/ urine output returning to baseline < 7 days.
- Rapid recovery : S.Cr / urine output returning to baseline < 48 hours.
- AKD :
  - Worse 1 year outcomes.
  - Recurrence of AKI.

AKI, AKD,CKD :



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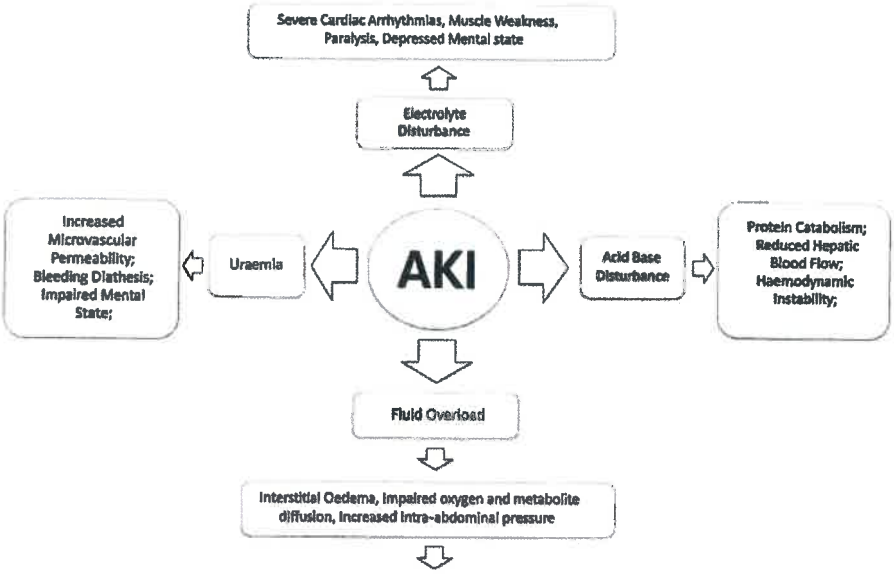
Incidence of AKI :

Incidence timing and outcome of AKI in critically ill patients.

- AKI incidence :
  - 35% : RIFLE.
  - 38% : AKIN.
  - 38% : KDIGO.
- 10-15% of AKI patients in ICU require renal replacement therapy (RRT).

Acute kidney injury → short term and long term effects :



- CKD : (14-20)%.
- Dialysis : (5-7)%.
- Coronary events : (10.3- 19.8)/1000 person year.
- Mortality : (30-40)%.

Risk factors for developing AKI in the ICU :

Non modifiable	Modifiable Risk factor

• Older age.
• Gender.
• Ethnicity.
• Comorbidity :
  • Hypertension.
  • Diabetes mellitus.
  • Chronic kidney disease.
  • Heart failure.
  • CAD & PAD.
  • COPD.
  • Obesity.
  • malignancy.
	• Hypovolaemia.
	• Sepsis.
	• Choice of fluid.
	• Nephrotoxins.
	• Major surgery.
	• Trauma, including burn patients.
	• Critical illness.
	• Anemia.
	• Emergency procedures.
	• Radiologic Contrast media.
	• COVID-19.

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Causes of AKI

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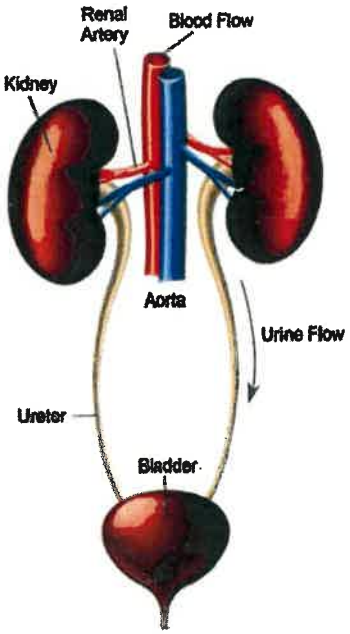
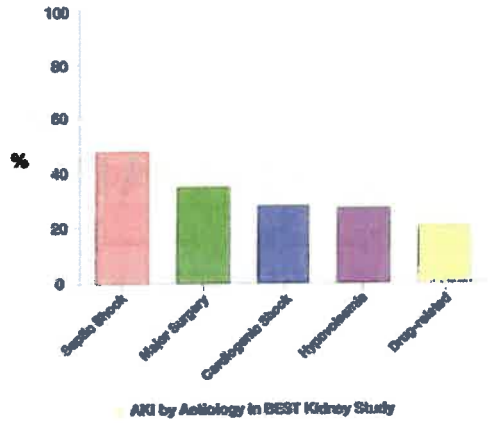
1. Prerenal : Sudden and severe reduction in blood pressure (shock) of interruption of blood flow to the kindeys from severe injury or illness.
   - Blood loss.
   - Dehydration.
   - Heart failure.
   - Sepsis.
   - Vascular occlusion.



2. Intrinsic Renal : Direct injury to the kidneys by inflammation, drugs, toxins, infection, or reduced blood supply.
   - Acute tubular necrosis :
     - Drugs.
     - Toxins.
     - Prolonged hypotension.
   - Glomerulonephritis
   - Acute tubular necrosis :
     - Drugs.
     - Toxins.
     - Autoimmune disease..
     - Infection.
   - Small-vessel vasculitis.
3. Postrenal : Sudden obstruction of urine flow due to enlarged prostate, kidney stones, bladder injury or tumor.
   - Benign prostatic hyperplasia.
   - Cervical cancer.
   - meatal stenosis/phimosis.
   - Retroperitoneal fibrosis.
   - Prostate cancer.
   - Urinary calculi.

BEST study :

- 6647 patients in ICU.
- 19.2% developed AKI.



AKI by Aetiology in BEST Kidney Study

Pathology of AKI in the ICU :

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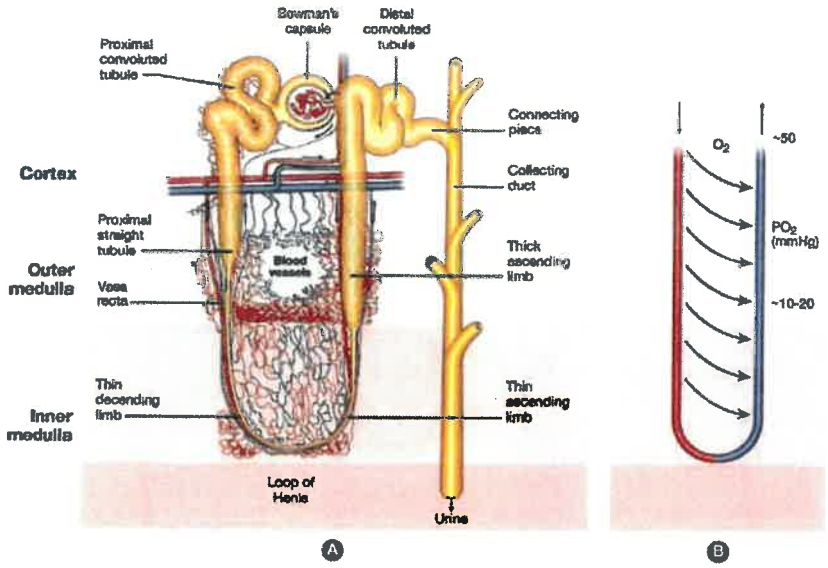

Septic AKI

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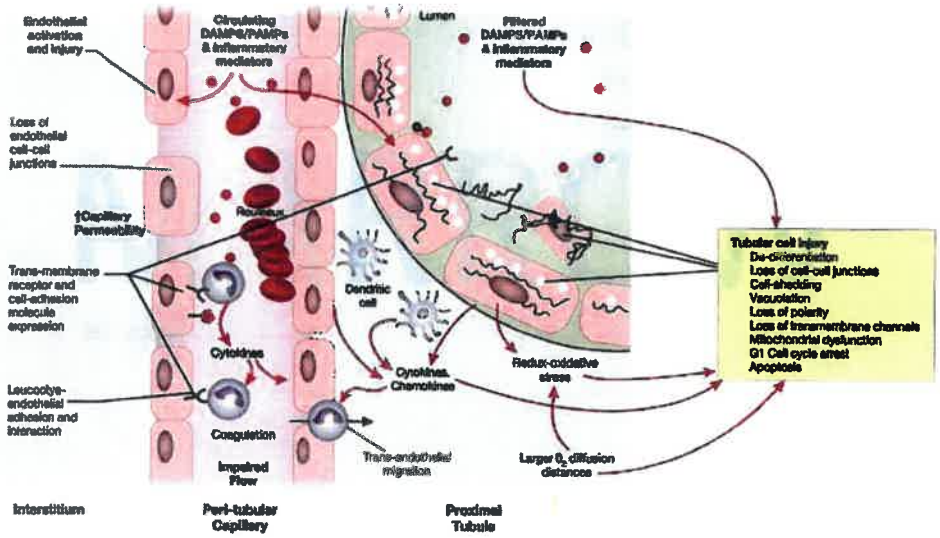
- Syndrome characterized by presence of sepsis and AKI definition.
- Incidence : >40% patient develop AKI.
- RRT: 30%.
- Mortality : 40%.
- Attributable mortality : 10%.

Pathophysiology of septic AKI :



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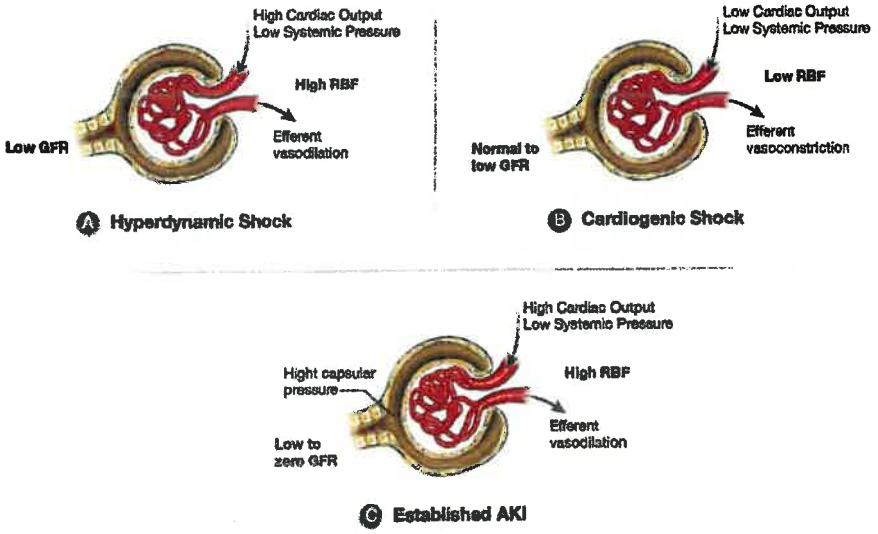
Effect on GFR :



Contrast Assoc AKI (CA-AKI) :

- Rise in SCr ≥ 0.5 mg/dl (44mmol/l) or a 25% increase from baseline value, assessed at 48 hours after a radiological procedure.
- mechanism :
  - Direct tubular toxicity.
  - Intra renal vasoconstriction.
- Incidence :
  - Normal patients : 1-2%.
  - Patients with risk factors : upto 25%.
    - Pre existing renal dysfunction.
    - Diabetes.
    - LV dysfunction.
    - Concurrent nephrotoxic medications.
  - 3.3% (if SCr> 0.5mg/dl).
  - 10.2% (if SCr> 25% baseline).
  - 10.5% (if GFR<25% baseline).

Retrospective study :

Incidence & outcome of contrast-associated acute kidney injury assessed with (RIFLE) criteria in critically ill patients of medical & surgical intensive care units :

- Incidence of CI-AKI was 15.5%.
- 55.8% had pre-existing kidney injury.
- CA-AKI patients were divided into risk (31%), injury (31%), and failure (38%).
- Dialysis : 7% patient.

- Recovery rate of AKI was 17% at the time of hospital discharge.
- Higher APACHE II scores were associated with a higher risk of CA-AKI.

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Risk factors :

Definite	Conflicting
• Pre-existing kidney disease	
• Diabetes, hypertension, CHF	• ACE-I and ARBs
• Advanced age	• Renal transplantation
• Volume depletion	• Multiple myeloma
• Hemodynamic instability	• Female
• Nephrotoxic medications	• Cirrhosis
• Large volume or high osmolality contrast agent	

Prediction of CI-AKI after PCI mehran risk model, JACC, 2004 :

Risk factors	Integer score (calculate)
Hypotension	5
IABP	5
CHF	5
Age > 75 years	4
Anemia	3
Diabetes	3
Contrast-media volume	1 per 100 ml
SCr > 1.5 mg/dl (> 132.6 μmol/l) or eGFR < 60 ml/min per 1.73 m^2	4 2 for 40-60 4 for 20-39 6 for <20

Note: Low risk: cumulative score < 5; High risk: cumulative score > 16.

7.5% for low [<5] and 57.3% for high [>16]

Preventive measures :

- Reduced dose of contrast medium.
- Isotonic saline.
- Isotonic bicarbonate solution.
- NAC.
- Diuretics.
- Statins.
- Theophylline.
- Fenoldopam.

— No role

Contrast medium :

- Dose : < 100 ml.
- Gms of iodine/EGFR (<1=3%, >1%= 25%),
- Maximum dose : (5gm/kg) / SCr,
- Route : IV preferred over arterial.

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Ionicity	Osmolality mosm/kg H_2O	Relative Osmolality	Examples
Ionic monomer	1500-1900	High	Diatrizoate, iothalamate
Ionic dimer	600	Low	Ioxaglate
Nonionic monomer	500-700	Low	Iohexol,iopamidol,iomeprol
Nonionic dimer	290-320	Iso	Iodixanol,iotrolan

Saline :

- NS preferred over 45% DNS.
- 1 hr before & upto 6 hrs after administration.
- At least 100ml/hr urine output.
- Rate : (1-2)ml/kg/hr infusion.

NaHCO3 :

- Isotonic solution of $NaHCO_3$: 850 cc of D5 + 150 cc of 8.4 % $NaHCO_3$.

NAC :

- Controversial benefit.
- Oral route preferred : Tolerable and inexpensive.
- ACC/AHA guidelines do not recommend.
- KDIGO : Suggest oral administration.
- Low dose : 600 mg bd for 2 days.

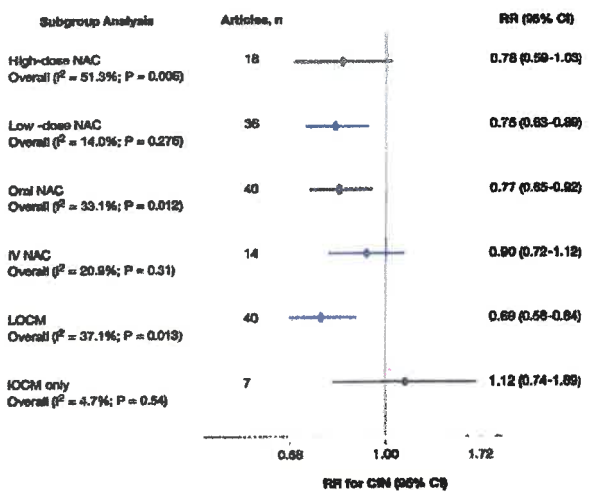
Preserve trial :

- Comparison of NS, $NaHCO_3$, NAC & Placebo.
- Result : No outcome benefits.

Effectiveness of Prevention Strategies for Contrast-Induced Nephropathy :

Pooled group	Studies, n	Pooled RR for CIN (95% CI)
High-dose NAC	18	0.78 (0.59-1.03)
IA administration	16	0.78 (0.55-1.12)
IV administration	2	0.55 (0.12-2.62)
Low-dose NAC	36	0.75 (0.63-0.89)
IA administration	30	0.77 (0.66-0.91)
IV administration	5	0.62 (0.18-2.10)
Oral NAC	40	0.77 (0.65-0.92)
IV NAC	14	0.90 (0.72-1.12)
NAC when LOCM are used	40	0.69 (0.58-0.84)
NAC when IOCM are used	7	1.12 (0.74-1.69)

CIN - Contrast-induced nephropathy
IA - Intra-arterial
IOCM - Iso-osmolar contrast media
IV - Intravenous
LOCM - Low-osmolar contrast media
NAC - N-acetylcysteine
RR - Risk ratio



ACUTE KIDNEY INJURY : PART II

Diagnosis of AKI

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History & examination :

History :

- GI loss : Vomiting, diarrhoea, haemorrhage.
- Decreased intake.
- Heart failure, hypotension.
- Procedures (Surgery, angioplasty).
- Systemic conditions (DM, HTN, IHD, PAD, jaundice).
- Infection.
- Blood in urine.
- Exposure drugs :
  i. Antibiotic exposure.
  ii. Immunosuppressive therapy (Transplant, malignancy).
  iii. Radiocontrast agent.
  iv. Herbal medicine, recreational drugs.
  v. Flank pain, anuria, hematuria.

Examination :

- Fluid status : Mucous membrane, skin turgor, sunken eyes, orthostatic hypotension.
- CNS : Stroke, neuropathy (vasculitis).
- Heart : S3, gallop, arrhythmias, murmur, raised JVP.
- Abdomen : Compartment syndrome, bruit mass, distended bladder, palpable tender kidney.
- Extremities : Peripheral pulses (PAD), rash (Vasculitis).
- Rectal and pelvic examination.

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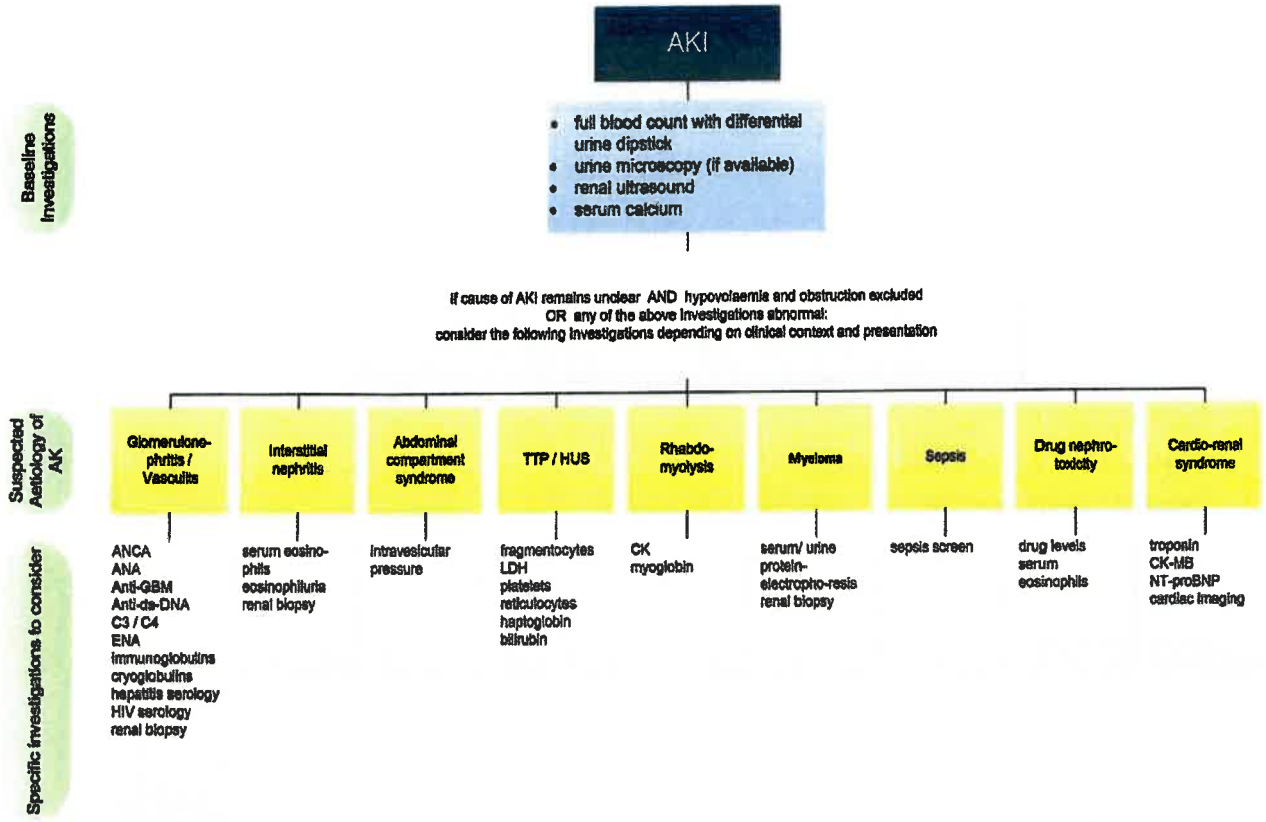
Diagnostic work-up for AKI in ICU :

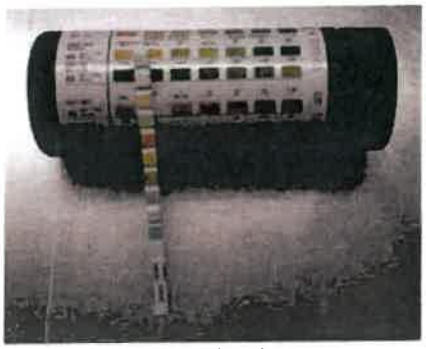


Urine analysis :

- Reagent/dipstick analysis.
- Microscopic analysis.

Dipstick analysis :

- Qualitative & easy to interpret.
- Quick bedside assessment.
- Eosinophils : Acute interstitial nephritis.
- Leukocytes/pus cells : Pyelonephritis.



Urine dipstick

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pH	• Normal range : 4.0 to 7.8 • Acidic : UTI • Alkaline : Infection with urea splitting organisms, diuretic therapy, nasogastric suction.
Hemoglobin	• RBC ± Hb : Glomerulonephritis. • Hb : Intravascular hemolysis or rhabdomyolysis.
Protein	• Detect only albumin. • Physioloical : 150 mg/d. • Microalbuminuria : 30–300 mg/d (Not detected). • Nephrotic range : >3 g/d (Glomerulonephritis). • Tubular range : 1–2 g/d (Acute tubular necrosis). • Gammopathies : Globulin (Not detected).
Osmolality	• Normal : 500–800 mOsm/kg. • Increased : SIADH, pre renal AKI. • Decreased : DI, polydipsia, renal AKI.
Specific gravity	• Normal : 1.003 to 1.030 • Increased : Pre renal AKI, glycosuria, SIADH. • Decreased : Renal AKI, DI, ATN, GN.
Glucose	• Renal threshold : 160–180 mg/dl.
Ketones	• Diabetic ± starvation Ketosis. • False positive : Sulphhydryl groups, e.g. Captopril.
Leukocyte esterase	• Dipstick is sensitive for leucocytes even after lysis. • UTI
Nitrites	• Bacteria convert nitrates to nitrites. • False negatives : Alkaline urine.

Microscopic examination :

- Red blood cells (RBCs).
- White blood cells (WBCs).
- Epithelial cells.
- Bacteria, yeast and parasites.
- Trichomonas.
- Crystals.
- Electrolytes.
- Casts ± sediments.

Electrolytes :

- Spot urine Na :
  - Urine Na <10 mmol/L = Pre-renal.
  - Urine Na >40 mmol/L = Renal.
- Fractional excretion of Na (FeNa) = $\frac{\text{Urine Na} \times \text{Plasma creatinine}}{\text{Plasma sodium} \times \text{Urine creatinine}}$
  - FeNa <0.01 (<1%) : Pre-renal.
  - FeNa >0.03 (2%) : Renal or post-renal.
  - FeNa 1–2% : Indeterminate significance.