ACUTE KIDNEY INJURY

National Standard Treatment Guideline

Ministry of Health
Republic of Maldives

ADB

JFPR
Japan Fund for Prosperous and Resilient Asia and the Pacific

From the People of Japan

World Health Organization
Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

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The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

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It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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ACUTE KIDNEY INJURY

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QUICK REFERENCE GUIDE

Acute Kidney Injury (AKI) is a sudden decline in kidney function. AKI is defined as any of the following:

- Increase in S.Cr by ≥0.3 mg/dl within 48 hours; OR
- Increase in S.Cr to ≥1.5 times baseline within the prior 7 days
- OR urine volume <0.5 mL/kg/h for 6 hours.

Presenting Clinical Features

- Often asymptomatic in early stages. Nonspecific symptoms include fatigue, nausea, anorexia, pruritus.
- Signs of underlying cause:
  - Hypovolemia: tachycardia, hypotension, dry mucous membranes.
  - Sepsis: fever, rigors, hypotension.
  - Obstruction: lower abdominal discomfort, enlarged bladder, anuria/ oliguria.
- Complications: confusion (uremia, acidosis, electrolyte imbalance), dyspnea (fluid overload, pulmonary edema).

Causes (Major Categories)

- Pre-renal (↓ perfusion): hypovolemia (bleeding, dehydration), sepsis, shock, cardiac failure, cirrhosis.
- Intrinsic renal:
  - Acute tubular necrosis (ischemia, toxins, contrast, aminoglycosides)
  - Acute interstitial nephritis (drugs, infections, autoimmune)
  - Glomerulonephritis (GN) (post-infectious, lupus, vasculitis)
  - Thrombotic microangiopathy (HUS/ TTP, malignant HTN).
- Post-renal (obstruction): prostate enlargement, stones, ureteric obstruction, bladder outlet obstruction, malignancy.

Laboratory Diagnosis & Evaluation

- Blood tests: SCr, BUN, electrolytes (esp. K, Na, bicarbonate, phosphate), CBC.
- Urine tests:
  - Dipstick + microscopy (casts: muddy brown → ATN, RBC casts → GN, WBC casts → AIN/ pyelonephritis).
  - Urine sodium/FeNa, FeUrea (helps pre-renal vs ATN in oliguric patients not on diuretics).
- Imaging: Renal ultrasound for hydronephrosis, obstruction, kidney size.
- Other: Serologies if GN suspected (ANA, ANCA, complements, anti-GBM).

- Volume assessment: daily weights, strict intake-output.

Staging

- Stage 1: SCr 1.5–1.9× baseline or ↑ ≥0.3 mg/dL; UO <0.5 mL/kg/h for 6–12 h
- Stage 2: SCr 2.0–2.9×; UO <0.5 mL/kg/h ≥12 h
- Stage 3: SCr 3.0× or ≥4.0 mg/dL, or new RRT; UO <0.3 mL/kg/h ≥24 h or anuria ≥12 h

Immediate Management goals

- Stabilize: ABCs, monitoring, IV access.
- Stop nephrotoxins (NSAIDs, aminoglycosides, ACEi/ARBs if unstable).
- Fluid resuscitation: Balanced crystalloids, guided by volume status.
- Treat cause: infection (antibiotics), obstruction (Foley/stent), hypovolemia (fluids/blood).
- Correct complications: hyperkalemia, acidosis, fluid overload.
- Renal replacement therapy (dialysis): if AEIOU criteria present.

First 30–60 minutes (stabilize)

1. ABCs, monitor, and IV access.
2. Check now: vitals, mental status, weight, strict I&O with Foley if retention suspected, POC glucose.
3. Labs: CBC, CMP, Mg/Phos, VBG/ABG... if sick, urinalysis + microscopy, urine Na (± FeUrea), cultures if sepsis.
4. Hold nephrotoxins/"SADMANS": SGLT2i, ACEi/ARB, Diuretics (if hypovolemic), Metformin, ANSAIDs, NSAIDs again for emphasis, Sulfonylureas; review aminoglycosides/iodinated contrast plans. (KDIGO: avoid nephrotoxins; change drug doses.) (KDIGO)
5. Bedside look for cause: volume status, infection, obstruction (palpable bladder). If retention → immediate bladder catheterization and arrange renal ultrasound.

Differentiate cause (often overlap)

- Pre-renal (hypovolemia/low perfusion): history of losses, low BP, dry mucosa, high BUN:Cr.
- Intrinsic (ATN, GN, AIN, etc.): muddy casts, dysmorphic RBCs/RBC casts, eosinophiluria.
- Post-renal: anuria/oliguria, fluctuating UO, enlarged bladder, hydronephrosis on US.

(Use UO + microscopy alongside SCr; meeting both SCr and UO criteria predicts worse risk.)

Fluids & hemodynamics

- If hypovolemic: give balanced crystalloids (LR/Plasma-Lyte) in small boluses (e.g., 250–500 mL; 10–20 mL/kg if shocked), reassess after each. Trials show fewer major adverse kidney events with balanced fluids vs saline (effect modest but consistent).

- If septic/hypotensive: early antibiotics, source control, fluids then vasopressors; norepinephrine is first-line to target MAP ≥65 mmHg. (For details see management of Sepsis guidelines)
- If fluid overloaded: avoid further boluses; consider loop diuretic for symptomatic overload only (does not treat AKI). KDIGO

Post-renal relief (don't delay)

- If bladder outlet obstruction is suspected → insert Foley now and monitor UO.
- If upper tract obstruction (stones, malignancy) or bilateral/solitary kidney → urgent urology for stent/nephrostomy after confirmation on ultrasound/CT (non-contrast).

Contrast exposure (if imaging needed)

- For eGFR ≥45: IV iodinated contrast is not an independent nephrotoxic risk; proceed when indicated.
- eGFR <30 or AKI: if contrast is essential, use IV isotonic fluids before/after; N-acetylcysteine not helpful; consider alternatives if feasible. Manage metformin per eGFR (hold only if eGFR <30 or AKI/arterial studies with embolic risk).

Medications

- Adjust dose as per renal impairment ... for all medicines excreted by renal route; avoid new nephrotoxins.
- Aminoglycosides: avoid if alternatives available; if necessary, single-daily dose and level monitoring. (KDIGO)

Hyperkalemia (temporize while arranging RRT if needed)

- Cardiac membrane: Calcium gluconate 10% 10 mL IV over 2–5 min; repeat if ECG changes persist.
- Shift K+: Regular insulin 10 units IV + 25 g dextrose (e.g., 50 mL D50W); consider nebulized albuterol 10–20 mg; sodium bicarbonate 50–100 mEq IV if acidotic.
- Remove K+: loop diuretics if producing urine, potassium binders, dialysis if refractory. (KDIGO)

Life-threatening complications RRT

Start RRT urgently for life-threatening fluid, electrolyte, or acid–base problems. Think AEIOU:

- Acidosis (severe, refractory)
- Electrolyte imbalance (e.g., refractory hyperkalemia)
- Ingestions (dialyzable toxins)
- Overload (pulmonary edema not responsive to diuretics)
- Uremia (encephalopathy, pericarditis, bleeding). (KDIGO)

Modality:

Use intermittent HD or CRRT; prefer CRRT if hemodynamically unstable or with acute brain injury. (KDIGO)

Vascular access (temporary) for dialysis:

- Right internal jugular → 2) Femoral → 3) Left internal jugular → 4) Subclavian last (avoid if possible, to preserve future AV access). Use ultrasound guidance; CXR to confirm IJ/subclavian tip before use. (KDIGO)

Monitoring & nursing orders

- Strict urine out (UO; aim ≥0.5 mL/kg/h), daily weights, vitals q4h (or continuous if unstable).
- Trend SCr, electrolytes, acid–base; stage AKI using both SCr and UO. (KDIGO)
- Glycemic control per local protocol; avoid hypoglycemia.
- Early dietitian input; prefer enteral nutrition. (KDIGO)

When to call/refer nephrology

- KDIGO Stage 2–3, unclear cause, rapidly rising SCr, refractory electrolyte/acid–base issues, suspected GN/AIN, need for KRT, persistent oliguria/anuria >12–24 h.

Follow-up & discharge

- Document AKI stage and etiology.
- On discharge: give medicine list with dose changes and AKI warnings (avoid NSAIDs, check labs).
- Follow-up kidney function and urine testing, typically within 3 months (earlier if severe). (AAFP)

Notes

- Do not use low-dose dopamine, fenoldopam, ANP, or routine diuretics to prevent/treat AKI. KDIGO
- Balanced crystalloids are reasonable defaults; evidence is mixed but points toward fewer kidney events than saline in many settings.

Monitoring

- Strict UO, weight, daily labs.
- Watch for complications (K+, acidosis, overload, encephalopathy).
- Adjust drug doses to kidney function.
- Document AKI and plan follow-up.

PEDIATRIC SPECIFIC CONSIDERATIONS IN AKI

Clinical Presentation

- More likely to present with **nonspecific symptoms:** irritability, poor feeding, vomiting, lethargy, seizures (due to electrolyte imbalance).
- Growth parameters and baseline creatinine are lower; even small rises in SCr may indicate significant renal dysfunction.
- Oliguria more common than in adults.
- Fluid overload manifests early as hypertension, periorbital/facial edema, respiratory distress.

Common Pediatric Causes

- **Pre-renal**: dehydration from diarrhea, vomiting, sepsis, hemorrhage.
- **Intrinsic**:
  - Hemolytic uremic syndrome (HUS) in infants/children.
  - Acute post-streptococcal glomerulonephritis.
  - Acute post-streptococcal glomerulonephritis.
  - Drug-induced (aminoglycosides, NSAIDs, chemotherapy).
- **Post-renal**: congenital anomalies of kidney/urinary tract (PUV, posterior urethral valves), stones, tumors compressing ureter.

Diagnosis in Children

- The same KDIGO criteria apply,but **age/weight-adjusted urine output** is more critical.
  - Oliguria: <0.5 mL/kg/h for >6 hours.
- Serum creatinine should be interpreted against age-specific normal values (lower in infants).
- Urine dipstick, microscopy, and renal ultrasound are key.
- Strict I&O charting is essential, including daily weight.

Management (Pediatrics)

- **Stabilization:** airway, breathing, circulation; establish IV/IO access if needed.
- **Fluids:** cautious resuscitation—10–20 mL/kg isotonic bolus if hypovolemic, then reassess. Avoid fluid overload.
- **Nephrotoxins:** avoid aminoglycosides/NSAIDs where possible; adjust all drug doses to weight + renal function.
- **Electrolyte management:**
  - Hyperkalemia: same principles (calcium gluconate, insulin+dextrose, beta-agonist, dialysis if refractory). Doses adjusted to weight.
  - Hyponatremia/hypernatremia: correct slowly, avoid rapid shifts (≤8–10 mEq/L per 24h).

- **Dialysis:**
  - Indications: refractory hyperkalemia, severe acidosis, uremia, fluid overload with pulmonary edema, encephalopathy.
  - Modalities:
    - Peritoneal dialysis is often preferred in infants/young children due to ease and hemodynamic stability.
    - Hemodialysis/CRRT in older children if available.
- **Monitoring**
  - Strict I&O, daily weights, vitals.
  - Electrolytes and creatinine at least daily.
  - Growth/nutrition support—early dietitian involvement.

Prognosis and Follow-up

- Risk of progression to chronic kidney disease is higher in children; all should have **long-term follow-up** after the AKI episode.
- Educate caregivers about avoiding nephrotoxins and ensuring early care for dehydration/sepsis.

INTRODUCTION

Acute Kidney Injury (AKI) is a sudden decline in kidney function occurring over hours to days, identified by rising serum creatinine or reduced urine output. It has replaced the term "acute renal failure" and encompasses a spectrum ranging from mild dysfunction to the need for renal replacement therapy (RRT). AKI affects approximately 5–7% of hospitalized patients and over 30% of those in intensive care units, with mortality rates varying from 15% to more than 60%, depending on severity and underlying causes. Globally, AKI impacts an estimated 13.3 million people annually and contributes to up to 1.7 million deaths, with a pooled incidence of 20–22% among hospital admissions and even higher rates in ICUs. The burden is disproportionately high in low- and lower-middle–income countries, where AKI is often community-acquired and driven by infections, dehydration, and delayed access to care.

AKI can result from pre-renal causes like hypoperfusion, intrinsic renal damage such as tubular or glomerular injury, or post-renal obstruction. It is common across medical, surgical, pediatric, oncology, and critical care settings. Older adults and individuals with comorbidities such as diabetes, cardiovascular disease, or chronic kidney disease are particularly vulnerable. Although AKI is frequently reversible, residual impairment in glomerular filtration rate (GFR) may persist, and even apparent recovery is associated with increased long-term risks including progression to chronic kidney disease, cardiovascular events, and recurrent episodes.

Early recognition and protocolized management—optimizing hemodynamics, avoiding nephrotoxins, using appropriate diagnostics, and timely escalation to RRT—are essential to reduce preventable deaths and improve outcomes. Standardized care pathways help minimize variability in recognition and treatment, especially in resource-constrained settings, and ensure that high-risk patients receive timely and effective interventions.

SCOPE OF THIS GUIDELINE

These guidelines cover identification, initial evaluation, risk stratification, stabilization, complication prevention, and escalation of care for adults with acute kidney injury. It is intended for use by health care providers, including general medical practitioners, physicians, nephrologists, nurses, and community health workers involved in the health care team.

Intended users

These AKI guidelines are designed for a multidisciplinary team across all levels of the health system, with pragmatic, context-sensitive strategies tailored to available resources. They emphasize early recognition, timely escalation, and standardized care to reduce preventable morbidity and mortality.

- At the **primary/community level**, the guidelines support frontline health workers, general practitioners, and nurses in identifying early signs of AKI—such as rising creatinine or oliguria—conducting basic risk assessments, optimizing volume status, avoiding nephrotoxins, and managing reversible causes like dehydration or medication-related insults. In settings with limited laboratory access, clinical judgment and urine output monitoring are essential. Red flags such as stage 2/3 AKI, unclear etiology, or refractory electrolyte disturbances should prompt urgent referral and early communication with higher-level facilities.
- At **secondary/district hospitals**, medical officers, general physicians, and emergency teams are guided to perform broader diagnostics (e.g., serum chemistries, acid-base status, ultrasound), manage complications (electrolyte correction, fluid therapy), and initiate source control. Where dialysis capacity is limited, clear protocols for stabilization and timely transfer to tertiary care are critical.
- At the **tertiary/critical care level**, nephrologists, intensivists, and specialist teams manage severe or complex AKI, including multiorgan involvement. They conduct advanced diagnostics (e.g., biopsy, imaging), initiate and maintain renal replacement therapy, and coordinate post-AKI follow-up to prevent progression to chronic kidney disease.
- The guidelines inform **program managers and policymakers** in developing system-wide protocols and referral pathways that ensure continuity, equity, and integration of AKI care across resource settings.

Cross-Cutting Considerations: Resource constraints—limited labs, imaging, dialysis, and specialist access—necessitate simplified decision pathways, clinical triggers for referral, and structured communication across levels. These guidelines prioritize timely recognition, essential interventions, and clear escalation thresholds to reduce preventable morbidity and mortality.

DEFINITION

AKI is defined as any of the following: increase in S.Cr by ≥0.3 mg/dl within 48 hours; or increase in S.Cr to ≥1.5 times baseline within the prior 7 days; or urine volume <0.5 mL/kg/h for 6 hours. (KDIGO 2012).

eGFR is not reliable in AKI and should not be used for staging.

For purposes of diagnosis and management,

AKI is divided into three categories:

- Pre-renal due to renal hypoperfusion (60-70%)

- Renal due to disease which involves renal parenchyma (25-35%)
- Post-renal due to diseases causing urinary obstruction (5-10%)

CAUSES, RISK FACTORS & TRIGGERS

Causes and risk factors & triggers of AKI are shown in Table

Table 1. Causes and risk factors & triggers of AKI

Category	Causes and Risk Factors	Frequency (%)
Pre-renal	Dehydration, hemorrhage, heart failure, liver cirrhosis, sepsis causing hypotension	60-70%
Renal	Acute tubular necrosis, acute glomerulonephritis, vasculitis, nephrotoxic drugs (NSAIDs (e.g., ibuprofen, naproxen), aminoglycosides (e.g., gentamicin, amikacin), amphotericin B, radiocontrast agents, ACE inhibitors, ARBs, cisplatin, cyclosporine, tacrolimus, lithium), contrast-induced nephropathy	25-35%
Post-renal	Obstruction (urinary calculi, tumors, prostate enlargement, strictures)	5-10%

EVALUATION FOR DIAGNOSIS

AKI can often be asymptomatic in its early stages. Many patients do not show obvious symptoms initially, and the condition is usually detected through laboratory findings such as a rise in serum creatinine or reduced urine output. When symptoms do occur, they are typically nonspecific, such as fatigue, nausea, anorexia, or pruritus. More severe signs like confusion, dyspnea, or edema usually appear later or when complications.

Symptoms and Signs Favoring Clinical Diagnosis

- Reduced urine output
- Fluid overload (edema, pulmonary congestion)
- Fatigue
- Nausea/vomiting
- Confusion or altered mental status

The clinical picture is usually dominated by the primary condition, which causes AKI (Table 2). Manifestations of uraemia like anorexia, nausea, vomiting, muscular cramps and signs of encephalopathy may appear later.

Staging of AKI according to kidney disease: Improving Global Outcomes (KDIGO) criteria is as follows:

Stage	Serum Creatinine (SCr)	Urine Output
Stage 1	Increase in SCr by ≥0.3 mg/dL or 1.5–1.9 × baseline	<0.5 mL/kg/h for 6–12 hours
Stage 2	2.0–2.9 × baseline	<0.5 mL/kg/h for ≥12 hours
Stage 3	3.0 × baseline or SCr ≥4.0 mg/dL or initiation of RRT	<0.3 mL/kg/h for ≥24 hours or anuria for ≥12 hours

CONFIRMATION OF DIAGNOSIS

Acute kidney injury is defined by the KDIGO criteria:

Acute Kidney Injury — KDIGO-based table

Domain	Test / Criterion	Threshold / Finding	How to use / Remarks
Diagnosis (KDIGO)	Serum creatinine	↑ ≥ 0.3 mg/dL within 48 h OR ≥ 1.5× baseline within 7 days	Either change meets AKI criteria. Compare with a reliable baseline.
	Urine output	< 0.5 mL/kg/h for ≥ 6 h	Assess alongside labs; ensure catheter accuracy when needed.
Supporting labs	eGFR	Decline; eGFR < 60 mL/min/1.73 m^2 signals reduced function	Not diagnostic of acute change unless compared with prior eGFR.
	BUN	> 20 mg/dL (nonspecific)	Often elevated in AKI; interpret with hydration, catabolism, GI bleed.
	Electrolytes / acid–base	Hyperkalemia: K^+ > 5.5 mEq/L; Metabolic acidosis: pH < 7.35 with HCO_3^- < 22 mEq/L	Life-threatening abnormalities guide urgency and therapy.
Urinalysis & microscopy	Casts / sediment	Granular "muddy brown" casts (ATI), proteinuria, active sediment	Helps distinguish intrinsic causes from pre/post-renal states.
Imaging	Renal ultrasound	—	Exclude post-renal obstruction before labeling intrinsic AKI.
Interpretation caveats	Volume status	—	Optimize volume first; both hypovolemia and fluid overload confound creatinine and UO. Reassess after correction.
Accuracy notes	eGFRcr-cys / measured GFR	—	Creatinine + cystatin C (eGFRcr-cys) improves accuracy; measure GFR when it will change management.

Note: Estimating GFR from a combination of creatinine and cystatin C (eGFRcr-cys) improves accuracy and strengthens risk relationships. GFR should be measured where more accurate ascertainment of GFR will impact treatment decisions.

Feature	Pre-renal	Intrinsic renal	Post-renal
History/exam	Volume loss, hypotension, tachycardia, dry mucosa	Toxins/ischemia, GN/AIN clues; edema, HTN	Flank/suprapubic pain, difficulty voiding, distended bladder
BUN:Cr	>20:1	<10–15:1	Variable
Urinalysis	Often bland or concentrated	**Sediment**: muddy brown casts (ATN), dysmorphic RBCs/RBC casts (GN), WBCs/eosinophils (AIN); **Proteinuria**: tubular (mild) or glomerular (heavy >2–3 g/d)	May show hematuria; can be bland
Urine Na^+ (mmol/L)	<20	>40	Variable (early low; rises if tubular injury develops)
Urine Osm (mOsm/kg)	>500	<350	Variable
FENa (%)	<1%	>2%	Variable; may be <1% early, then >2%
FEUrea (%) *(useful if on diuretics)*	<35%	>50%	Variable
Ultrasound	Normal	Normal or chronic changes	Hydronephrosis/obstruction
Response to fluids/ relief of obstruction	Creatinine improves with volume	Limited/none unless cause treated	Improves after decompression

FENa = Fractional Excretion of Sodium.

Formula: FENa (%) = (UrineNa×PlasmaCr)/(PlasmaNa×UrineCr)(Urine Na × Plasma Cr) / (Plasma Na × Urine Cr)(UrineNa×PlasmaCr)/(PlasmaNa×UrineCr) × 100

Use FEUrea (Fractional Excretion of Urea) when diuretics are on board or recently given, because diuretics raise urinary sodium and can falsely elevate FENa.

Formula: FEUrea (%) = (UrineUrea(orBUN)×PlasmaCr)/(PlasmaUrea(orBUN)×UrineCr) (Urine Urea (or BUN) × Plasma Cr) / (Plasma Urea (or BUN) × Urine Cr) (UrineUrea(orBUN)×PlasmaCr)/(PlasmaUrea(orBUN)×UrineCr) × 100

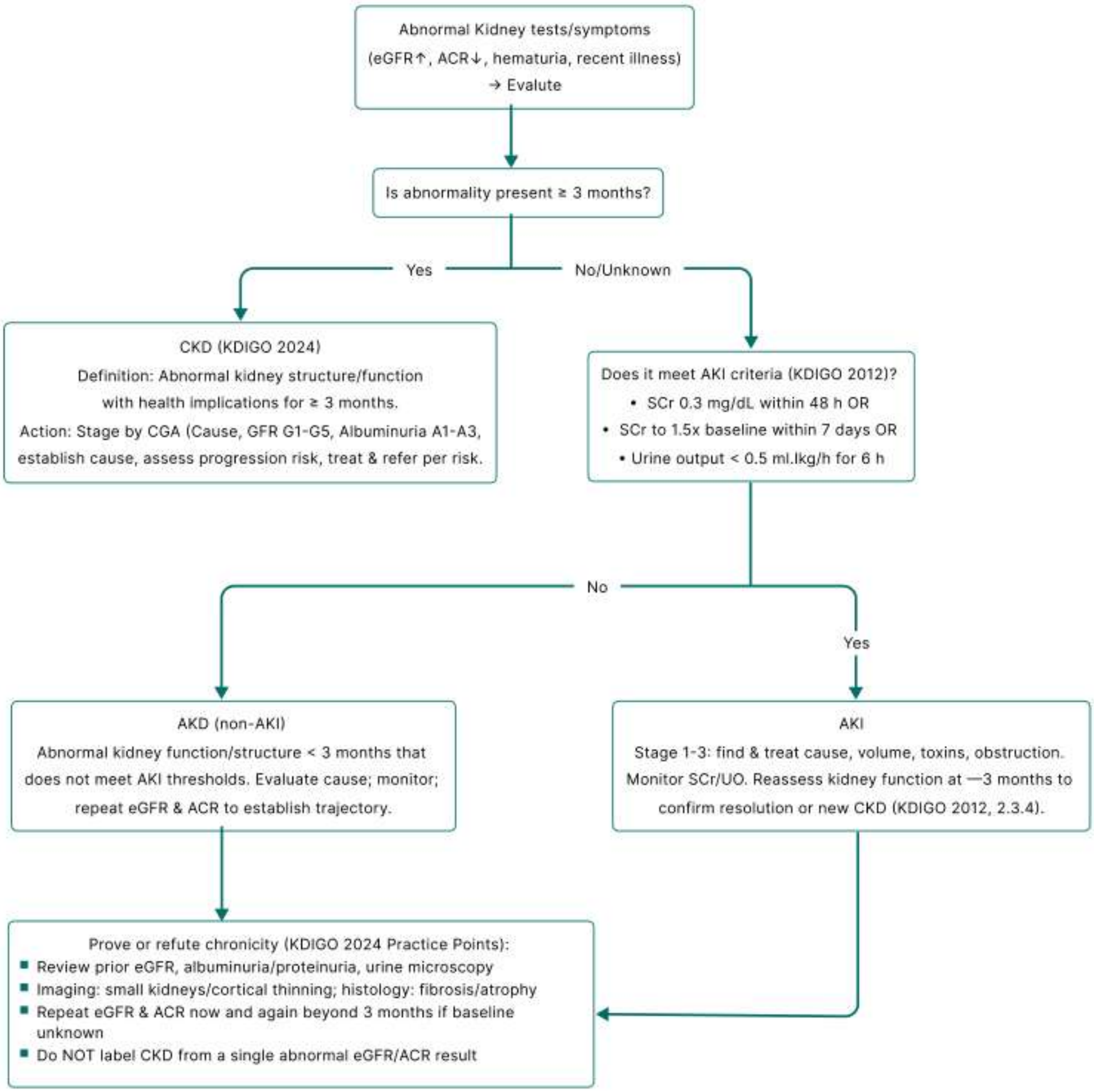
Note: CKD, sepsis, contrast nephropathy, GN, and osmotic diuresis (diuretics) can blur FENa use **FEUrea** instead. FEUrea can be altered by catabolism, GI bleed, steroids, liver disease, or very low protein intake. Always interpret with volume status, urinalysis, and ultrasound. Early post-renal patterns may mimic pre-renal indices. Always re-check after correcting volume or removing obstruction.

DIFFERENTIAL DIAGNOSIS (TABLE 2)

Mimic / alternative explanation	Clues	How to distinguish from true AKI	Next step
Chronic kidney disease (stable)	Old records of low eGFR; anemia, small echogenic kidneys, longstanding albuminuria	Abnormalities present ≥3 months; prior labs confirm	Stage as CKD; manage per CKD guideline
AKD (subacute change)	Gradual rise in creatinine over weeks; no abrupt oliguria	Does not meet KDIGO AKI time thresholds; duration <3 months	Monitor trend; repeat eGFR/ACR; treat cause
Assay interference (pseudo-creatinine rise)	DKA (ketoacids), high bilirubin, hemolysis; drugs: cefoxitin, cefazolin, flucytosine, nitromethane	Jaffe vs enzymatic assay discordance; cystatin-C normal	Re-test with enzymatic assay/cystatin-C; treat underlying issue
Inhibition of tubular creatinine secretion	Recent start of trimethoprim, cimetidine, dolutegravir/ cobicistat, dronedarone	Disproportionate creatinine rise with stable BUN, cystatin-C, urine output	Review meds; stop/ switch if possible; document expected bump
Increased creatinine production	Heavy meat meal, creatine supplements, intense exercise	Small transient SCr rise; no fall in urine output; cys-C unchanged	Repeat fasting sample after 24–48 h; counsel on intake
Under-collection / measurement error	Inaccurate intake–output charting; spilled urine; short collection window	Clinical euvolemia; labs stable; numbers don't fit exam	Educate staff; standardize timing; re-measure
Catheter or outlet issue (pseudo-oliguria)	Kinked/blocked Foley; sudden drop in recorded urine	Bladder scan shows retention; output resumes after flush/ replacement	Fix catheter; reassess before calling oliguria
SIADH / water retention states	Low urine volume with low serum Na, normal creatinine	Urine osmolality high; no creatinine rise	Treat underlying cause; fluid management
Prerenal azotemia corrected early	Brief dehydration, diuretics, GI losses; prompt fluid response	Creatinine normalizes rapidly after fluids; FeNa/urine indices prerenal	Optimize volume; avoid nephrotoxins
Pregnancy physiology	Low baseline SCr (~0.4–0.7); small absolute rises look large in % terms	Check gestational norms; correlate with BP, proteinuria	Use obstetric thresholds; evaluate for preeclampsia if indicated
Lab/sample issues	Delayed processing, sample mix-up, severe hyperlipemia	Repeat sample differs; delta check flags	Redraw; verify ID; use plasma/serum per lab protocol
eGFR equation artifacts	Extremes of muscle mass, amputation, cachexia	Creatinine-based eGFR unreliable; cys-C disagrees	Use eGFRcr-cys or measured GFR when it affects decisions

Note: Always recheck volume status, meds, and measurement accuracy before diagnosing AKI. If uncertainty persists, repeat creatinine/urine output and consider cystatin-C or a different assay to confirm.

AKI vs AKD vs CKD -- One-Page Flowchart (KDIGO)



Sources: KDIGO 2012 AKI Guideline; KDIGO 2024 CKD Guidellne (Practice Points on chronicity & AKD).
Abbreviations: SCr serum creatinine; UO urine output; eGFR estimated GFR; ACR = albumin-creatinine ratio; CGA Cause, GFR, Albuminuria.

COMPLICATIONS

Acute kidney injury can lead to several serious complications.

- Fluid overload from impaired excretion causes hypertension and pulmonary edema.
- Electrolyte disturbances, especially hyperkalemia, risk life-threatening arrhythmias.
- Metabolic acidosis develops as acid clearance falls, worsening hemodynamics and catabolism. Uremic encephalopathy can impair mentation and neurologic function.
- AKI also increases the risk of incomplete recovery and progression to chronic kidney disease, particularly after severe or repeated episodes.

MANAGEMENT

The goals of management in AKI are to

1. Prevent further kidney injury
2. Maintain adequate fluid, electrolyte, and acid-base balance
3. Avoid and treat complications such as hyperkalaemia, volume overload, and uremia
4. Optimize supportive care to promote renal recovery; and
5. identify and manage the underlying cause of AKI.

A multidisciplinary team approach involving timely nephrology consultation, appropriate monitoring, and individualized care plans is key to achieving these goals.

Management of AKI (Figure 1)

1. Focus on supportive measures to prevent progression, minimize complications, and promote renal recovery. The cornerstone is optimization of hemodynamic status: ensuring adequate fluid resuscitation in hypovolemic patients using isotonic crystalloids, while avoiding fluid overload, which can worsen outcomes.
2. In critically ill patients, functional hemodynamic monitoring (e.g., dynamic fluid responsiveness tests, central venous pressure when appropriate) may guide fluid and vasopressor therapy to maintain organ perfusion, targeting a mean arterial pressure (MAP) above 65 mmHg.
3. **Volume Responsiveness in AKIa**
   - Dynamic assessments guide safe fluid therapy in AKI, especially in critical care avoiding both under-resuscitation and fluid overload:
   - Passive Leg Raise (PLR): Transient autotransfusion; ↑ stroke volume suggests responsiveness.

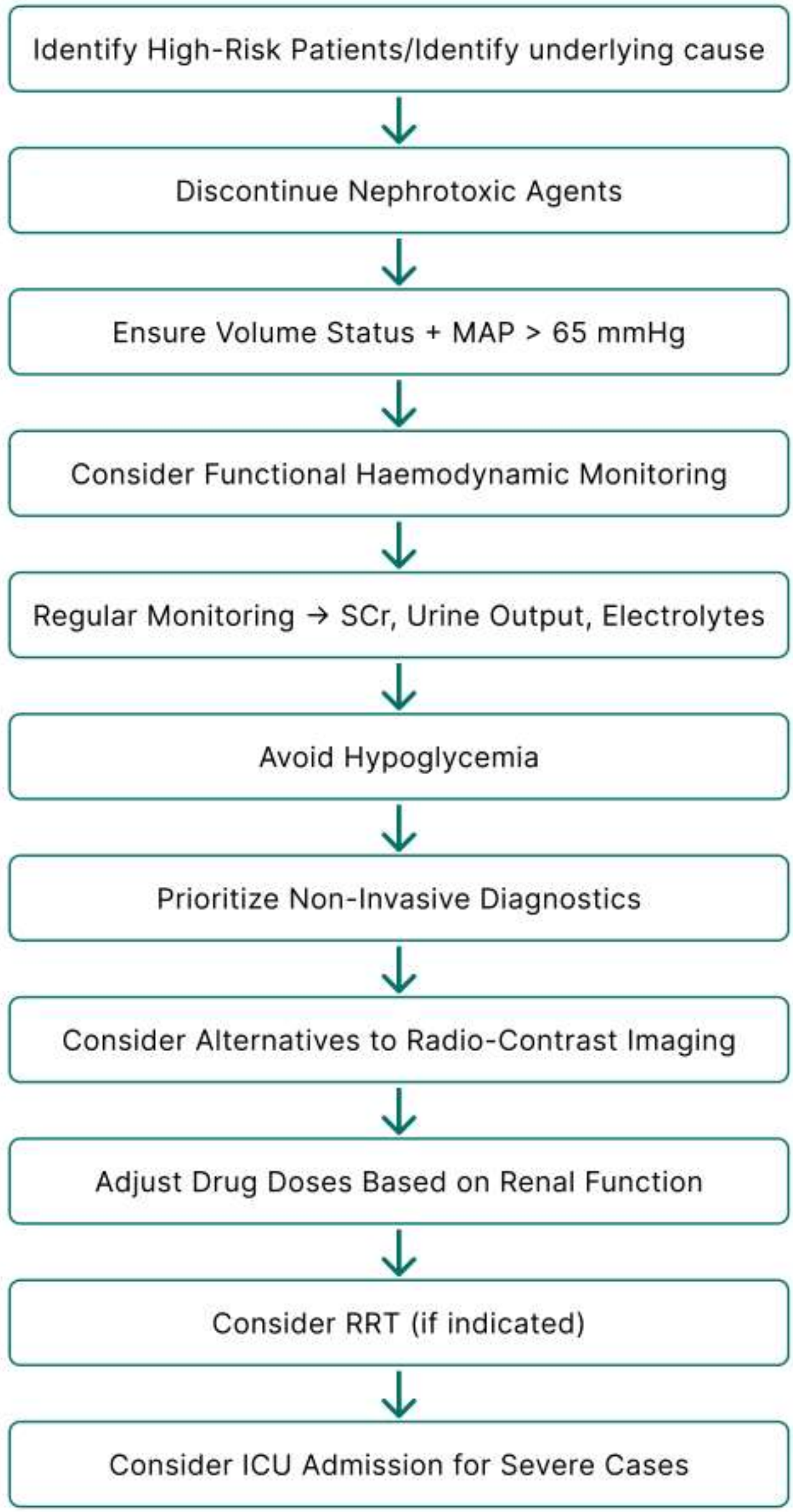
- IVC Collapsibility Index: Ultrasound-based assessment of IVC diameter variation with respiration. In spontaneously breathing patients, a collapsibility index >40% indicates volume responsiveness. >40% variation (in spontaneously breathing patients) suggests hypovolemia.

4. Avoidance of further renal insults is crucial. This includes discontinuing or minimizing nephrotoxic agents such as NSAIDs, aminoglycosides, and contrast media. When diagnostic imaging is necessary, non-contrast alternatives or low-osmolar contrast with prophylactic measures (e.g., hydration protocols) should be considered. Nutritional support should be individualized, ensuring adequate caloric intake while avoiding excessive protein loads that may increase uremic toxin burden.
5. If septic/hypotensive: early antibiotics, source control, fluids then vasopressors; norepinephrine is first-line to target MAP ≥65 mmHg. (For details see management of Sepsis guidelines)
6. Close monitoring of serum creatinine, urine output, fluid balance, electrolytes, and acid-base status is essential to guide ongoing management. Avoidance of hypoglycemia and careful glucose control are part of supportive care.
7. When complications such as refractory hyperkalemia, metabolic acidosis, or fluid overload arise, timely initiation of renal replacement therapy (RRT) may be required. Patients with severe AKI or multi-organ dysfunction may need intensive care unit (ICU) admission for advanced organ support.
8. Environmental factors should also be addressed, including avoiding unnecessary invasive procedures, minimizing risk of infections (e.g., catheter-related), and ensuring appropriate nursing care to prevent pressure injuries and other hospital-acquired complications.

Indications of referral of patients to emergency department/ hospitalization

- Refer to the emergency department any patient with stage 2 or 3 AKI.
- Also refer stage 1 AKI when the cause is unclear, when significant comorbidities are present (e.g., heart failure, cirrhosis, immunosuppression), or when there is no improvement after initial simple measures such as appropriate fluid resuscitation.
- Early evaluation in these situations helps identify serious reversible causes and prevents progression.

Figure 1. Stage-based management of AKI.



Note: In cases where contrast use is unavoidable, strategies such as pre- and post-procedural hydration and the use of low-osmolar contrast agents should be considered.

1. Intravenous fluids:

i. Normal saline or balanced crystalloids; titrated based on clinical response and fluid status. Replacement fluids to be tailored according to the composition of lost fluids, e.g. in haemorrhage; blood transfusion/packed RBCs. If blood transfusion is required, blood should be as fresh as possible, as longer-stored blood may lead to hyperkalaemia. In most other situations, hypovolaemia, buffered crystalloids like Ringer's lactate are the fluid of choice. Normal saline (0.9%) is given in case of burns, pancreatitis and diabetic ketoacidosis.

ii. (Fluid intake = Urine output + fluid loss from other sources + insensible loss of fluid about 500 mL/day.) Estimate of the amount of fluid required based on hydration status of the patient as assessed by jugular vein distension and dry tongue, etc.

iii. In patients with cirrhosis complicated by AKI, fluids should be administered slowly and titrated against jugular venous pressure (JVP).

2. Diuretics (Furosemide): 20-40 mg IV initially, repeat or adjust based on urine output and fluid status every 6-12 hours

3. Hyperkalemia management:

i. Restriction of dietary potassium (no food containing K+)

ii. Calcium gluconate 10 ml IV of 10% solution over 10 minutes or earlier if ECG changes.
If serum K+ >6.5, in addition,

iii. Insulin (5-10 units IV) with 25% Dextrose (50 ml IV) over 10 minutes. Hourly monitoring till 3 hours.

iv. Salbutamol nebulization: 10–20 mg nebulized over 10–15 min; Onset: ~30 min; duration ~2–6 hours. Caution: Monitor for tachycardia; less effective alone

v. Initiate haemodialysis (intermittent HD or CRRT based on clinical status) when other interventions are ineffective or in cases of severe, life-threatening hyperkalemia.

4. Metabolic acidosis: Sodium bicarbonate (severe metabolic acidosis): 50-100 ml IV over 10 minutes, individualized and repeated to maintain an arterial pH f >7.1.

5. Hypocalcemia: If symptomatic or ECG changes, Tab. Calcium carbonate 1 g/day OR IV Calcium gluconate 10% 10-20 ml given over 20 minutes

RENAL AKI / INTRINSIC AKI

Renal AKI / Intrinsic AKI

- Escalate early to nephrology. Treat cause + provide supportive care.
- Stabilize hemodynamics: target euvolemia; avoid both hypovolemia and overload.
- Stop nephrotoxins; adjust dose of medicines according to the level of renal impairment.
- Monitor closely: electrolytes, acid–base, creatinine, urine output, weight.

Etiology-specific

- Acute tubular necrosis: supportive care, avoid new insults; start renal replacement therapy (RRT) if indicated.
- Acute interstitial nephritis: stop the culprit drug; consider prednisone 0.5–1 mg/kg/day with taper if severe or recovery is incomplete.
- Glomerulonephritis / rapidly progressive GN: urgent workup (often biopsy); high-dose steroids ± cyclophosphamide or rituximab; plasmapheresis for selected vasculitides.
- Systemic vasculitis/inflammatory causes: treat the underlying disease with appropriate immunomodulation.

Always manage complications: hyperkalemia, acidosis, fluid overload; initiate RRT when indicated. Reassess trajectory and adjust therapy.

POSTRENAL AKI

- Decompress now:
  - Lower tract: Foley (or suprapubic) catheter.
  - Upper tract: Percutaneous nephrostomy or ureteral stent.
  - Confirm level with renal ultrasound; use non-contrast CT if needed.
- Treat the cause: stone removal, prostate management, malignancy workup. Supportive care: correct volume/electrolytes; avoid nephrotoxins.
- Track recovery: monitor creatinine and urine output; start renal replacement therapy if indications persist despite relief.
- Refer early: involve urology and nephrology.

ASSESSMENT OF RESPONSE

AKI inpatient monitoring and supportive care

Area	What to do	Frequency	Notes / Actions
Fluids & urine	Strict intake–output charting	Hourly	Use accurate measurements; verify catheter patency if output falls.
Labs	Serum creatinine, electrolytes; fluid balance	Daily	Track trends; correct K^+, bicarbonate, calcium, magnesium as needed.
Clinical review	Bedside reassessment (vitals, weight, edema, lung exam, mental status)	Daily	Watch for improvement vs deterioration; adjust plan accordingly.
Complications	Prevent and treat	Ongoing	Anticipate hyperkalemia, acidosis, volume overload, uremic symptoms; act early.
Volume status	Monitor closely; avoid unnecessary IV fluids	Ongoing	Aim euvolemia; use balanced fluids when indicated; stop excess infusions.
Diuresis for overload	Inj. Frusemide IV	As needed	Dose per overload; usual 20–40 mg/day as bolus or IV infusion; titrate to response.
Renal replacement therapy	Initiate dialysis when indicated	As indicated	Start promptly if clinically required (per local criteria/protocol).

Issues to be Assessed Before Step-up/Step-down Treatment

- Before escalating or de-escalating therapy, assess volume status (rule out ongoing hypovolemia or fluid overload), correct or account for electrolyte disturbances (especially potassium and bicarbonate), and determine whether the underlying insult is progressing or resolving.
- Evaluate the patient's clinical and laboratory response to current pharmacologic interventions to ensure the change is warranted.

Referral for Specialist Consultation

Refer promptly to nephrology in case of following conditions. Early specialist involvement improves diagnostics, timing of RRT, and outcomes.

- Severe AKI (serum creatinine >3.0 mg/dl)
- Rapidly deteriorating renal function
- Persistent hyperkalemia (>6.5 mEq/L), refractory acidosis (pH <7.15)
- Suspected intrinsic renal disease
- Need for dialysis or renal replacement therapy (RRT)

DIALYSIS

Indications (AEIOU mnemonic)

Dialysis is indicated when one or more of the following are present:

- **A** – Acidosis: Severe metabolic acidosis with pH <7.15, especially if causing hemodynamic compromise or refractory to medical correction.
- **E** – Electrolyte imbalance: Life-threatening derangements, most notably severe hyperkalemia (>6.5 mEq/L) not responsive to medical therapy.
- **I** – Intoxication: Toxic ingestion of dialyzable substances (small, non–protein-bound): toxic alcohols (ethylene glycol, methanol), lithium, salicylates, theophylline, valproate, and others where enhanced elimination alters outcome.
- **O** – Overload: Clinically significant volume overload (e.g., pulmonary edema) due to oliguria/anuria unresponsive to diuretics or conservative measures.
- **U** – Uremia: Uremic complications such as encephalopathy, pericarditis, or bleeding diathesis (uraemic hemorrhage) and rising nitrogenous waste. (BUN >100 mg/dL or rapidly rising urea/creatinine; conventionally urea >150–180 mg/dL or creatinine >6–7 mg/dL in context).

Vascular access for dialysis

- **Access type**: Temporary dual-lumen catheter for acute HD/CRRT; plan early for AV fistula or graft if dialysis is likely to continue.
- **Preferred site order**

  1. Right internal jugular (RIJV) first choice
  2. Femoral vein second (use in bedbound or short-term/CRRT)
  3. Left internal jugular (LIJV) third

  Note: Avoid subclavian except as a last resort due to stenosis risk that jeopardizes future fistulae.

- **Insertion basics:** Use ultrasound guidance for vessel identification and needle entry.
  Full barrier precautions and chlorhexidine skin prep.
  Choose appropriate length and caliber (RIJV ~15–20 cm; LIJV/femoral ~20–24 cm).
  Confirm tip position by chest X-ray for thoracic lines; document flow goals.
- **Tip position targets:** IJV catheters: lower SVC or cavo-atrial junction.
  Femoral catheters: IVC.

- **Securement and care:**
  - Suture or securement device plus transparent dressing.
  - Keep exit site dry; change dressing per protocol.
  - Use heparin or citrate lock per unit policy. Reserve the catheter for dialysis only.
- **Monitoring**
  - Check daily: site appearance, tenderness, fever, dressing integrity.
  - Check function each session: achievable blood-flow rate, negative/positive pressure alarms, recirculation.
  - Track complications: infection, dysfunction/poor flows, thrombosis, malposition, air embolism.
- **Troubleshooting**
  - Suspected infection: send blood cultures from catheter and peripheral vein; start empiric antibiotics per protocol.
  - Dysfunction: reposition patient, assess kinks, consider thrombolytic lock, guidewire exchange if needed.
  - Thrombosis or stenosis: duplex or venography and specialist input.
- **Special notes:** Coordinate site choice with vascular surgery to protect future fistula options.
  Avoid subclavian unless no other access is feasible and benefits outweigh long-term risks.

Monitoring of patients on dialysis

Immediately after each dialysis session (0–60 min)

- Vitals & fluid: BP (including symptomatic orthostasis), HR, temp, post-weight vs target dry weight, net UF achieved, cramps/dizziness/headache.
- Access check:
  - AVF/AVG: hemostasis, bruit/thrill present, no hematoma, bleeding <10–15 min.
  - Catheter: exit-site clean/dry, caps on, lock documented, no tenderness/erythema.
- Symptoms screen: chest pain, dyspnea, confusion, muscle cramps, disequilibrium.

- Labs (as indicated): post-dialysis BUN (for adequacy per schedule), K^+, HCO_3^-, Ca^{2+}, glucose (if diabetic).
- Machine/adequacy record: delivered time/ Blood Flow Rate (BFR)/Dialysate flow rate (DFR), alarms, Transmembrane Pressure (TMP)/ Ultrafiltration (UF) profile, spKt/V (Single-pool Kt/V, a measure of dialysis efficacy) or URR (Urea Reduction Ratio, an alternative matrix)entry.
- Medications given: ESA, IV iron, vitamin D analogs, anticoagulation used and issues.
- Plan tweaks: reassess dry weight, interdialytic fluid limit, BP meds timing before next run.

Between sessions (home/in-between dialysis days)

- Daily weight: morning weight; aim for interdialytic weight gain (IDWG) within unit target (e.g., ≤2–3% of dry weight).
- Home BP log: morning/evening; bring to clinic. Note dizziness or nocturnal hypotension.
- Access self-check (daily):
  - Arteriovenous Fistula (AVF)/Arteriovenous graft (AVG): feel the thrill, look for redness, warmth, swelling, prolonged bleeding.
  - Catheter: keep dressing dry/intact; report fever, chills, exit-site pain or discharge.
- Symptoms to track: dyspnea, edema, reduced exercise tolerance, pruritus, sleep disturbance, restless legs, cramps.
- Diet & fluids: adhere to sodium/fluid plan; potassium and phosphate restriction; binders with meals.
- Med adherence: antihypertensives per timing plan (some may be held pre-dialysis), ESA/iron schedule awareness; avoid NSAIDs and other nephrotoxins.
- When to call early: fever or rigors (esp. with catheter), IDWG >4–5% dry weight, no thrill (vibration felt over AVF/AVG indicating patency) / bruit (audible vascular sound over AVF/AVG), access bleeding >20 min, severe cramps/dizziness, chest pain, progressive dyspnea, confusion.

Scheduled surveillance

Domain	What	Usual timing/schedule*
Adequacy	spKt/V or URR	Monthly until stable, then at unit policy
Electrolytes/acid–base	K^+, HCO_3^- (± Ca^{2+}, Mg^{2+})	Monthly or per protocol
Bone–mineral	Ca^{2+}, PO_4^{3-} monthly; iPTH (Intact Parathyroid Hormone)	PTH every 3–6 months
Anemia	Hb	Monthly
Iron status	Ferritin, TSAT (Transferrin Saturation)	Every 1–3 months (if on ESA/iron)
Nutrition	Serum albumin (± nPCR- Normalized Protein Catabolic Rate)	Monthly–quarterly
Infection screening	HBsAg/anti-HBs, anti-HCV	Per national policy (often 3–12 monthly); after exposures
Access surveillance	Flow/pressure trends, recirculation tests; duplex (Duplex Doppler Ultrasound (vascular access imaging) if indicated	Per unit protocol (e.g., quarterly or if dysfunction)
Residual renal function	24-h urine (if present)	Every 3–6 months

Adjust frequency for instability, recent med changes, complications, or pregnancy.

Post-session/Interdialytic red flags (urgent review)

- Fever/chills (catheter patient or new AVF pain/redness).
- Refractory hypotension, chest pain, acute dyspnea, syncope.
- Severe hyperkalemia symptoms (weakness, paresthesia, arrhythmia) or ECG changes.
- Access thrombosis (no thrill/bruit) or persistent bleeding.
- IDWG well above target or rapid edema/orthopnea.

Recovery and stopping dialysis

Monitoring for recovery

- Track trends, not single values: daily SCr/BUN, electrolytes (potassium, sodium, calcium), bicarbonate, 24-h urine output, weight, and fluid balance before and periodically during sessions to guide dialysate composition and avoid rapid shifts. Acid–base status if indicated
- Reassess hemodynamics, edema, lung exam; check for ongoing nephrotoxins, sepsis, obstruction and anticoagulation effects (if used): based on the underlying risk of bleeding. Fluid removal rates and ultrafiltration targets to prevent hypotension; assess weight changes and intravascular volume tolerance.

- Review urinalysis for improving sediment (e.g., fewer casts/less hematuria).

Recovery of the underlying condition

- Pre-renal: stable BP/volume; creatinine falling after optimization.
- Post-renal: obstruction relieved (ultrasound clear); brisk diuresis then stabilization.
- Intrinsic: improving creatinine/urea; resolving proteinuria/hematuria; disease activity markers settling (e.g., GN/vasculitis).

When to consider stopping hemodialysis (trial off)

- Urine output improving and adequate for fluid/electrolyte control
  - Practical cues: >500–1000 mL/day without diuretics, or higher if on diuretics.
- Renal function improving: falling/stabilizing SCr/BUN without RRT.
- Electrolytes/acid–base maintainable medically: K^+ ≤5.0–5.5 mEq/L, HCO_3^- ≥18–20 mEq/L, no severe acidosis.
- Volume status controllable without ultrafiltration; no pulmonary edema.
- Initial indication resolved: no refractory hyperkalemia, acidosis, uremic symptoms (pericarditis/encephalopathy), or uncontrolled overload.

How to perform the trial off

- Skip the next session, monitor closely.
- Re-check labs at ~6–12 h and at 24 h (SCr/BUN, K^+, HCO_3^-), vitals, weight, symptoms.
- If stable/improving over 48–72 h, continue off dialysis and de-escalate to intermittent labs.

If failure of trial off (restart dialysis if any)

- Rising creatinine with worsening symptoms, K^+ >5.5–6.0 unresponsive to meds, worsening acidosis, or recurrent volume overload.

After discontinuation

- Plan frequent labs for 1–2 weeks, then taper.
- Remove the temporary catheter once stable off RRT (coordinate with nephrology).
- Continue kidney-protective measures: avoid nephrotoxins, adjust drug doses to eGFR, manage BP and fluids.

NUTRITION & SUPPORTIVE MANAGEMENT

- Dietary management in AKI should be individualized based on the patient's clinical status, comorbidities, and stage of kidney injury.
  - Adequate caloric intake (approximately 25–30 kcal/kg/day) should be ensured to prevent catabolism. Protein intake should be restricted moderately in non-dialyzed AKI (approximately 0.8–1 g/kg/day) to limit uremic toxin accumulation, while higher protein requirements (1.2–1.5 g/kg/day) may be appropriate for patients on renal replacement therapy (RRT).
  - Electrolyte intake, particularly of potassium, phosphorus, and sodium, should be carefully regulated according to serum levels and urinary losses.
  - Fluid intake should be tailored to match insensible losses and urine output, with adjustments as needed for ongoing fluid overload or depletion.
  - Early involvement of a renal dietitian is recommended to ensure appropriate nutritional planning and monitoring.
- Preventive measures against hospital-acquired infections, including careful catheter use and adherence to infection control protocols, are vital.
- To prevent contrast-induced nephropathy, patients at risk should receive intravenous hydration with isotonic saline (e.g., 0.9% NaCl) at a rate of 1–1.5 mL/kg/hour, starting 6–12 hours before and continuing for 6–12 hours after contrast administration.
- Environmental measures such as frequent repositioning, skin care, and prevention of pressure injuries should also be implemented, particularly in critically ill patients.

Other Methods of Management of AKI

Beyond general supportive care, non-pharmacological approaches, and dietary modifications, additional strategies are essential to mitigate complications, preserve organ function, and initiate renal replacement therapy (RRT) when clinically indicated.

RENAL REPLACEMENT THERAPY (RRT) MODALITIES

The selection of RRT modality should be individualized based on clinical context, including:

- Hemodynamic stability
- Severity and progression of AKI
- Presence of multi-organ dysfunction
- Institutional capacity and resource availability

Available modalities include:

1. **Intermittent Hemodialysis (IHD):** Preferred in hemodynamically stable patients; allows rapid solute and fluid removal over short sessions.
2. **Continuous Renal Replacement Therapy (CRRT)**: Indicated for critically ill or hemodynamically unstable patients; provides gradual solute clearance and fluid management over 24 hours.
3. **Peritoneal Dialysis (PD**): May be considered in resource-limited settings or where vascular access is challenging; suitable for selected patients including children and those with bleeding risks.

Intervention	Description / Indication
Renal Replacement Therapy (RRT)	Indicated in refractory hyperkalaemia, severe metabolic acidosis, volume overload unresponsive to diuretics, or uremic complications (e.g., pericarditis, encephalopathy). Modality choice (hemodialysis, CRRT, peritoneal dialysis) depends on the patient's condition.
Vasopressor support	Norepinephrine (or equivalent) to maintain MAP > 65 mmHg in septic/distributive shock when fluids are insufficient.
Blood transfusion (if indicated)	For clinically significant anemia causing hypoxia. Over-transfusion should be avoided to prevent fluid overload.
Targeted therapies	Plasma exchange for conditions like rapidly progressive glomerulonephritis, Hemolytic Uremic Syndrome (HUS), Thrombotic Microangiopathy (TMA); immunosuppressants (e.g., steroids) in autoimmune causes (after ruling out infection).
Early nephrology consultation	For severe, progressive, or unclear cause of AKI; guides advanced management, RRT decisions, and long-term planning.
Multidisciplinary team involvement	Monthly–quarterly

PREVENTION OF AKI

Prevention of AKI focuses on identifying at-risk individuals early and implementing measures to reduce avoidable kidney injury, since there is no specific therapy for ischaemic or nephrotoxic AKI, prevention is very important and includes:

- Identify high-risk patients early (e.g., sepsis, major surgery, critical illness, CKD, diabetes, hypovolemia, exposure to nephrotoxins).
- Maintain adequate intravascular volume and renal perfusion:
  - Aggressively restore intravascular volume in case of losses, e.g. during surgery, trauma, burns, gastroenteritis.
  - Use isotonic crystalloids for volume resuscitation in hypovolemia.

- Avoid excessive fluid administration to prevent volume overload.

- Avoid or minimize nephrotoxic exposures:
  - Limit use of NSAIDs, aminoglycosides, and radiocontrast agents.
  - Consider alternatives when possible. Hypovolaemia should be avoided in patients receiving nephrotoxic drugs.
  - If a contrast imaging is necessary:
    - Use low- or iso-osmolar contrast media.
    - Ensure pre- and post-procedure hydration.
- Monitor renal function closely in high-risk patients:
  - Regularly check serum creatinine and urine output.
  - Track fluid balance and electrolytes.
- Adjust drug dosages appropriately for renal function to avoid toxicity.
- Maintain hemodynamic stability:
  - Consider hemodynamic monitoring in critically ill patients.
  - Target mean arterial pressure (MAP) ≥ 65 mmHg.
- Ensure good glycemic control:
  - Avoid both hyperglycemia and hypoglycemia.
- Minimize unnecessary invasive procedures to reduce infection and injury risk.
- Adhere to infection prevention protocols:
  - Use catheters and invasive devices judiciously.
  - Follow strict aseptic technique.
- Promote patient education:
  - Counsel about avoiding over-the-counter nephrotoxic medications.
  - Encourage early medical consultation during acute illness.
- Engage multidisciplinary care teams where appropriate (e.g., nephrology, critical care).

FOLLOW-UP

Small changes in kidney function in hospitalized patients are associated with significant changes in short- and long-term outcomes. Patients who have recovered from AKI require structured follow-up to monitor for late complications, detect incomplete recovery, and reduce the risk of progression to chronic kidney disease (CKD). Consider these patients at an increased risk for CKD and care accordingly.

Timing	Do's	Purpose	Don'ts
Within 7–30 days post-discharge	Check serum creatinine, eGFR- Urine analysis (proteinuria/hematuria) Electrolytes- Blood pressure check	Assess recovery of renal function; detect residual dysfunction or complications early.	Don't assume normal urine output = full recovery. Don't delay testing beyond 30 days.
1–3 months post-discharge	Repeat renal function tests if prior abnormalities found Monitor blood pressure and proteinuria	Monitor for resolution or persistence of kidney injury markers; assess CKD risk.	Don't stop follow-up prematurely if proteinuria or low eGFR persist. Don't overlook BP control.
>3 months (if residual abnormalities)	Refer to nephrology Continue periodic monitoring (e.g., every 3–6 months) Cardiovascular risk assessment	Manage CKD progression risk; optimize long-term renal and cardiovascular health.	Don't delay nephrology referral in persistent dysfunction. Don't ignore cardiovascular risk factors.
At each visit / ongoing	Counsel on avoiding nephrotoxic drugs (NSAIDs, unnecessary contrast) Encourage lifestyle modification Educate on AKI warning signs	Prevent recurrent AKI; promote self-care; reduce risk of future kidney injury.	Don't reintroduce nephrotoxic medications without clear indication. Don't give excessive fluid or salt unless indicated.
If no recovery as expected	Reassess for alternative diagnoses (e.g., chronic kidney disease, obstructive uropathy, systemic illness) Initiate nephrology referral if not already done Consider imaging, serologic tests, and renal biopsy if indicated	Identify underlying cause of non-recovery; guide appropriate long-term management and prognosis	Don't continue supportive care alone without diagnostic re-evaluation Don't delay specialist input or necessary investigations

Note: Persistent abnormalities = early nephrology referral. Long-term follow-up prevents CKD and cardiovascular complications.

PROGNOSIS OF AKI

AKI prognosis varies with severity, cause, timeliness of reversal, and baseline kidney health. Mild, pre-renal insults that are promptly corrected often fully recover.

- Severe AKI (higher KDIGO stage), delayed recognition, persistent hemodynamic instability, multiorgan failure, or intrinsic injury (e.g., acute tubular necrosis, glomerulonephritis) carries higher short-term mortality and greater risk of incomplete recovery. Comorbidities such as chronic kidney disease, diabetes, heart failure, and advanced age worsen outcomes.
- Recurrent AKI episodes compound risk and accelerate progression to chronic kidney disease or end-stage renal disease.

- Even after apparent recovery, survivors have elevated long-term risks of hypertension, proteinuria, reduced GFR, and cardiovascular events.
- Early specialist involvement, mitigation of ongoing insults, and structured follow-up improve likelihood of renal recovery and reduce downstream morbidity.

PREVENTION AND HEALTH PROMOTION

- Identify and mitigate risk factors early: optimize volume status, control blood pressure and glycemia, and manage heart failure.
- Avoid or adjust nephrotoxic agents (NSAIDs, aminoglycosides, radiographic contrast, certain antivirals) especially in at-risk patients; perform medication reconciliation on every encounter.
- Ensure adequate hydration around insults (e.g., before and after contrast exposure or surgery) while avoiding fluid overload.
- Implement sepsis prevention and early treatment protocols—source control and timely antibiotics—to reduce sepsis-associated AKI.
- Use risk-based screening (elderly, CKD, liver disease, volume depletion) and apply clinical decision support for early recognition (monitor creatinine/urine output).
- Apply bundle care in hospital settings: standardized ordering, alerting for rising creatinine, and protocols for hemodynamic optimization.
- Use dose-adjusted imaging strategies: limit contrast volume, use iso-/low-osmolar agents, and employ prophylaxis in high-risk patients.
- Educate patients on warning signs (reduced urine output, dizziness, vomiting) and when to seek care.
- Promote continuity of care and follow-up after an AKI episode to detect incomplete recovery and prevent recurrence.

PATIENT EDUCATION

- Help patients recognize early warning signs (reduced urine output, swelling, confusion, shortness of breath, persistent nausea/vomiting)
- Promote avoidance of nephrotoxins (NSAIDs, unnecessary contrast, over-the-counter supplements) unless cleared by clinician
- Teach importance of maintaining appropriate volume status—when to hydrate and when to hold fluids

- Ensure understanding of prescribed medications, dosing adjustments, and adherence
- Guide patients on when and how to seek urgent care for deterioration or lack of improvement
- Encourage follow-up labs and visits to monitor recovery and detect incomplete resolution
- Explain the risk of recurrence and progression to chronic kidney disease, and actions to mitigate it (blood pressure control, glycemic control, infection prevention)
- Empower patients to communicate changes in health, medication use, or exposure to potential kidney insults to their care team promptly.
- Provide dietary guidance & emphasize low-sodium, potassium-appropriate, and phosphate-controlled diets as per renal status; discourage high-protein intake unless indicated; promote hydration strategies tailored to volume status.
- Support smoking cessation and educate on its role in worsening vascular and renal outcomes; offer referrals to cessation programs or behavioral support.

PATIENT INSTRUCTIONS

Give instructions to help recovery, prevents worsening, and reduces the chance of future kidney damage.

- Know the warning signs: Report immediately if you develop decreased urine output, swelling (especially in legs or face), shortness of breath, persistent nausea or vomiting, confusion, chest pain, or irregular heartbeat.
- Medications: Take only prescribed medicines. Stop NSAIDs (ibuprofen, naproxen), certain herbal supplements, and other over-the-counter drugs unless approved by your doctor. Inform your care team about all medications and supplements.
- Hydration: Follow advice on fluid intake—drink enough if you are volume depleted, but avoid excess if you have signs of overload (weight gain, breathlessness). Don't self-adjust fluids without guidance.
- Labs and follow-up: Attend all scheduled blood and urine tests. These monitor kidney function, electrolytes, and fluid balance. Early follow-up is critical—weekly after discharge for the first month, then as advised.
- Diet: Follow any dietary recommendations, especially regarding salt, potassium, and protein, based on your current kidney function.
- Avoid further insults: Protect your kidneys—avoid dehydration (especially with illness or heat), infections, and unnecessary contrast studies unless cleared by your doctor.

- Dialysis awareness: If dialysis was discussed or started, understand the reason, schedule, and access plan. Ask questions about what to expect.
- When to seek help: Go to the emergency department if you have worsening shortness of breath, chest pain, severe weakness, markedly reduced urine output, sudden confusion, or high potassium symptoms (palpitations, muscle weakness).
- Communication: Inform any new provider (ER, dentist, specialist) that you had AKI. Carry a summary of your recent kidney function and medications.
- Lifestyle: Quit smoking, control blood pressure and blood sugar if applicable, and rest as needed while recovering.

SUMMARY OF THE MANAGEMENT OF ACUTE KIDNEY INJURY IN ADULTS AND PEDIATRIC SPECIFIC CONSIDERATIONS.

	Adults	Pediatrics
Definition (KDIGO)	↑ SCr ≥0.3 mg/dL in 48 h or ≥1.5× baseline within 7 d or UO <0.5 mL/kg/h for ≥6 h	Same KDIGO definition; interpret SCr against age-specific normals
Urine output criterion	<0.5 mL/kg/h for ≥6 h	Same; strict weight-based tracking
Typical presentation	Often silent early; fatigue, nausea, pruritus; signs of cause (hypovolemia, sepsis, obstruction)	Irritability, poor feeding, vomiting, lethargy; early edema/HTN, respiratory distress; seizures if electrolyte deranged
Common causes – Pre-renal	Hypovolemia, sepsis, shock, HF, cirrhosis	Dehydration (diarrhea/vomiting), sepsis, hemorrhage
Common causes – Intrinsic	ATN (ischemia/toxins), AIN (drugs/infx), GN (PIGN, lupus), TMA	HUS, post-streptococcal GN, sepsis-AKI, drug-induced (aminoglycosides, NSAIDs, chemo)
Common causes – Post-renal	Prostatic enlargement, stones, malignancy, ureteric block	CAKUT (e.g., PUV), stones, extrinsic compression/tumors
Initial stabilization (first 30–60 min)	ABCs, monitors, IV access; vitals, mental status, weight, strict I&O; labs (CBC, CMP, Mg/Phos, VBG/ABG), UA+microscopy, urine Na/FeUrea; hold nephrotoxins; bedside screen for retention (Foley if needed)	Same approach; IV/IO if needed; weight-based sampling; urgent UA, chemistry; early bladder scan; involve caregiver for history (fluids/ diarrhea)
Fluids	If hypovolemic: balanced crystalloids 250–500 mL boluses, reassess; septic shock → antibiotics, fluids, then vasopressors (NE first-line)	If hypovolemic: 10–20 mL/kg isotonic bolus, reassess each bolus; avoid overload; septic shock per pediatric sepsis protocol
If fluid overloaded	Avoid further boluses; loop diuretic only for symptoms; no role for "renal-protective" diuretics	Same; careful respiratory monitoring; escalate early if pulmonary edema
Nephrotoxins	Stop NSAIDs, aminoglycosides; review contrast; renally adjust all drugs	Avoid NSAIDs/aminoglycosides where possible; weight + GFR-based dosing

Post-renal relief	Foley for suspected outlet block; urgent urology for upper tract (stent/nephrostomy)	Same; early ultrasound; urgent PUV relief in infants
Imaging	Renal US first; non-contrast CT if needed	Renal US first-line; minimize radiation; VCUG/MCU if indicated by urology
Hyperkalemia (temporize)	Calcium gluconate 10% 10 mL IV over 2–5 min; Insulin 10 U IV + Dextrose 25 g; Neb Albuterol 10–20 mg; $NaHCO_3$ 50–100 mEq IV if acidotic; remove K (diuretic if making urine, binders, dialysis)	Calcium gluconate 10% 0.5 mL/kg (max 10 mL) IV 2–5 min; Insulin 0.1 U/kg IV + Dextrose 0.5–1 g/kg (e.g., D10W 5–10 mL/kg or D25W 2 mL/kg); Neb Salbutamol 2.5–5 mg (repeat; up to 10–20 mg in larger children/adolescents); $NaHCO_3$ 1–2 mEq/kg if acidotic; remove K as above
Dysnatremias	Correct ≤8–10 mEq/L per 24 h (slower if chronic); severe symptomatic hypoNa → 3% saline bolus	Same rate; severe symptomatic hypoNa with seizures → 3% saline 2–3 mL/kg bolus; careful neuro checks
Staging (KDIGO)	Stage 1/2/3 by SCr rise and UO thresholds	Same; rely more on UO in infants (low baseline SCr)
Dialysis – when (AEIOU)	Acidosis, Electrolyte (K+), Ingestions, Overload, Uremia (encephalopathy, pericarditis, bleeding)	Same AEIOU triggers; lower threshold if refractory overload/encephalopathy
Dialysis – modality	Intermittent HD or CRRT; CRRT if hemodynamically unstable/acute brain injury	Peritoneal dialysis often first-line in infants/young children; HD/CRRT in older children where available
Vascular access for RRT	Temp: Right IJV → Femoral → Left IJV → Subclavian last; US-guided; CXR confirm	Femoral or Right IJV depending on size; avoid subclavian; US-guided; confirm tip before use
Monitoring	Strict UO (target ≥0.5 mL/kg/h), daily weight; vitals q4h (continuous if unstable); SCr/electrolytes and acid–base trending; glycemic control; document stage/etiology	Same; meticulous I&O; daily weight; frequent labs; BP and neuro checks; early dietitian input for growth/nutrition
Medications	Renal dose adjust all renally cleared drugs; avoid new nephrotoxins; therapeutic drug monitoring where relevant	Weight- and GFR-based dosing; avoid nephrotoxins; TDM for narrow-index agents (e.g., aminoglycosides if used)
Refer / Call Nephrology	KDIGO Stage 2–3, rapid SCr rise, unclear cause, refractory electrolytes/acid–base, suspected GN/AIN, need for KRT, persistent oliguria/anuria >12–24 h	Same triggers; plus suspected HUS/rapidly progressive GN, CAKUT/post-renal anomalies
Follow-up	Recheck kidney function and urinalysis (usually within 3 months; earlier if severe); counsel to avoid NSAIDs and high-risk contrasts	Higher CKD risk; schedule long-term follow-up; caregiver education on dehydration prevention, early care for sepsis/diarrhea, and nephrotoxin avoidance

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