

Summary of Recommendation Statements

Kidney International Supplements (2012) **2**, 8–12; doi:10.1038/kisup.2012.7

Section 2: AKI Definition

2.1.1: AKI is defined as any of the following (*Not Graded*):

- Increase in SCr by ≥ 0.3 mg/dl (≥ 26.5 μ mol/l) within 48 hours; or
- Increase in SCr to ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
- Urine volume < 0.5 ml/kg/h for 6 hours.

2.1.2: AKI is staged for severity according to the following criteria (Table 2). (*Not Graded*)

Table 2 | Staging of AKI

Stage	Serum creatinine	Urine output
1	1.5–1.9 times baseline OR ≥ 0.3 mg/dl (≥ 26.5 μ mol/l) increase	< 0.5 ml/kg/h for 6–12 hours
2	2.0–2.9 times baseline	< 0.5 ml/kg/h for ≥ 12 hours
3	3.0 times baseline OR Increase in serum creatinine to ≥ 4.0 mg/dl (≥ 353.6 μ mol/l) OR Initiation of renal replacement therapy OR, In patients < 18 years, decrease in eGFR to < 35 ml/min per 1.73 m ²	< 0.3 ml/kg/h for ≥ 24 hours OR Anuria for ≥ 12 hours

2.1.3: The cause of AKI should be determined whenever possible. (*Not Graded*)

2.2.1: We recommend that patients be stratified for risk of AKI according to their susceptibilities and exposures. (*1B*)

2.2.2: Manage patients according to their susceptibilities and exposures to reduce the risk of AKI (see relevant guideline sections). (*Not Graded*)

2.2.3: Test patients at increased risk for AKI with measurements of SCr and urine output to detect AKI. (*Not Graded*)
Individualize frequency and duration of monitoring based on patient risk and clinical course. (*Not Graded*)

2.3.1: Evaluate patients with AKI promptly to determine the cause, with special attention to reversible causes. (*Not Graded*)

2.3.2: Monitor patients with AKI with measurements of SCr and urine output to stage the severity, according to Recommendation 2.1.2. (*Not Graded*)

2.3.3: Manage patients with AKI according to the stage (see Figure 4) and cause. (*Not Graded*)

2.3.4: Evaluate patients 3 months after AKI for resolution, new onset, or worsening of pre-existing CKD. (*Not Graded*)

- If patients have CKD, manage these patients as detailed in the KDOQI CKD Guideline (Guidelines 7–15). (*Not Graded*)
- If patients do not have CKD, consider them to be at increased risk for CKD and care for them as detailed in the KDOQI CKD Guideline 3 for patients at increased risk for CKD. (*Not Graded*)

Section 3: Prevention and Treatment of AKI

3.1.1: In the absence of hemorrhagic shock, we suggest using isotonic crystalloids rather than colloids (albumin or starches) as initial management for expansion of intravascular volume in patients at risk for AKI or with AKI. (*2B*)

3.1.2: We recommend the use of vasopressors in conjunction with fluids in patients with vasomotor shock with, or at risk for, AKI. (*1C*)

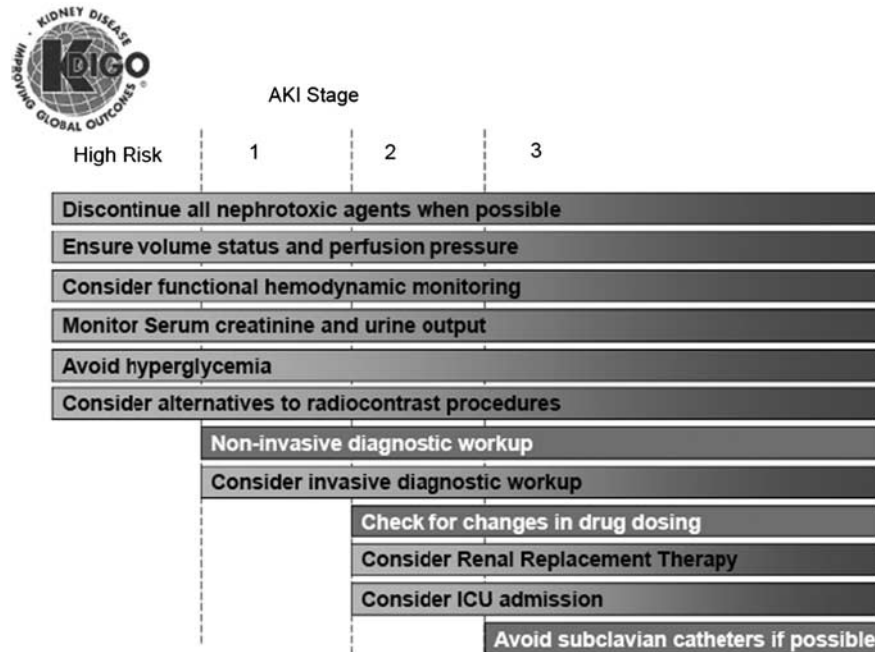


Figure 4 | Stage-based management of AKI. Shading of boxes indicates priority of action—solid shading indicates actions that are equally appropriate at all stages whereas graded shading indicates increasing priority as intensity increases. AKI, acute kidney injury; ICU, intensive-care unit.

- 3.1.3: We suggest using protocol-based management of hemodynamic and oxygenation parameters to prevent development or worsening of AKI in high-risk patients in the perioperative setting (2C) or in patients with septic shock (2C).
- 3.3.1: In critically ill patients, we suggest insulin therapy targeting plasma glucose 110–149 mg/dl (6.1–8.3 mmol/l). (2C)
- 3.3.2: We suggest achieving a total energy intake of 20–30 kcal/kg/d in patients with any stage of AKI. (2C)
- 3.3.3: We suggest to avoid restriction of protein intake with the aim of preventing or delaying initiation of RRT. (2D)
- 3.3.4: We suggest administering 0.8–1.0 g/kg/d of protein in noncatabolic AKI patients without need for dialysis (2D), 1.0–1.5 g/kg/d in patients with AKI on RRT (2D), and up to a maximum of 1.7 g/kg/d in patients on continuous renal replacement therapy (CRRT) and in hypercatabolic patients. (2D)
- 3.3.5: We suggest providing nutrition preferentially via the enteral route in patients with AKI. (2C)
- 3.4.1: We recommend not using diuretics to prevent AKI. (1B)
- 3.4.2: We suggest not using diuretics to treat AKI, except in the management of volume overload. (2C)
- 3.5.1: We recommend not using low-dose dopamine to prevent or treat AKI. (1A)
- 3.5.2: We suggest not using fenoldopam to prevent or treat AKI. (2C)
- 3.5.3: We suggest not using atrial natriuretic peptide (ANP) to prevent (2C) or treat (2B) AKI.
- 3.6.1: We recommend not using recombinant human (rh)IGF-1 to prevent or treat AKI. (1B)
- 3.7.1: We suggest that a single dose of theophylline may be given in neonates with severe perinatal asphyxia, who are at high risk of AKI. (2B)
- 3.8.1: We suggest not using aminoglycosides for the treatment of infections unless no suitable, less nephrotoxic, therapeutic alternatives are available. (2A)
- 3.8.2: We suggest that, in patients with normal kidney function in steady state, aminoglycosides are administered as a single dose daily rather than multiple-dose daily treatment regimens. (2B)
- 3.8.3: We recommend monitoring aminoglycoside drug levels when treatment with multiple daily dosing is used for more than 24 hours. (1A)
- 3.8.4: We suggest monitoring aminoglycoside drug levels when treatment with single-daily dosing is used for more than 48 hours. (2C)
- 3.8.5: We suggest using topical or local applications of aminoglycosides (e.g., respiratory aerosols, instilled antibiotic beads), rather than i.v. application, when feasible and suitable. (2B)
- 3.8.6: We suggest using lipid formulations of amphotericin B rather than conventional formulations of amphotericin B. (2A)
- 3.8.7: In the treatment of systemic mycoses or parasitic infections, we recommend using azole antifungal agents and/or the echinocandins rather than conventional amphotericin B, if equal therapeutic efficacy can be assumed. (1A)

- 3.9.1: We suggest that off-pump coronary artery bypass graft surgery not be selected solely for the purpose of reducing perioperative AKI or need for RRT. (2C)
- 3.9.2: We suggest not using NAC to prevent AKI in critically ill patients with hypotension. (2D)
- 3.9.3: We recommend not using oral or i.v. NAC for prevention of postsurgical AKI. (1A)

Section 4: Contrast-induced AKI

- 4.1: Define and stage AKI after administration of intravascular contrast media as per Recommendations 2.1.1–2.1.2. (Not Graded)
 - 4.1.1: In individuals who develop changes in kidney function after administration of intravascular contrast media, evaluate for CI-AKI as well as for other possible causes of AKI. (Not Graded)
- 4.2.1: Assess the risk for CI-AKI and, in particular, screen for pre-existing impairment of kidney function in all patients who are considered for a procedure that requires intravascular (i.v. or i.a.) administration of iodinated contrast medium. (Not Graded)
- 4.2.2: Consider alternative imaging methods in patients at increased risk for CI-AKI. (Not Graded)
- 4.3.1: Use the lowest possible dose of contrast medium in patients at risk for CI-AKI. (Not Graded)
- 4.3.2: We recommend using either iso-osmolar or low-osmolar iodinated contrast media, rather than high-osmolar iodinated contrast media in patients at increased risk of CI-AKI. (1B)
- 4.4.1: We recommend i.v. volume expansion with either isotonic sodium chloride or sodium bicarbonate solutions, rather than no i.v. volume expansion, in patients at increased risk for CI-AKI. (1A)
- 4.4.2: We recommend not using oral fluids alone in patients at increased risk of CI-AKI. (1C)
- 4.4.3: We suggest using oral NAC, together with i.v. isotonic crystalloids, in patients at increased risk of CI-AKI. (2D)
- 4.4.4: We suggest not using theophylline to prevent CI-AKI. (2C)
- 4.4.5: We recommend not using fenoldopam to prevent CI-AKI. (1B)
- 4.5.1: We suggest not using prophylactic intermittent hemodialysis (IHD) or hemofiltration (HF) for contrast-media removal in patients at increased risk for CI-AKI. (2C)

Section 5: Dialysis Interventions for Treatment of AKI

- 5.1.1: Initiate RRT emergently when life-threatening changes in fluid, electrolyte, and acid-base balance exist. (Not Graded)
- 5.1.2: Consider the broader clinical context, the presence of conditions that can be modified with RRT, and trends of laboratory tests—rather than single BUN and creatinine thresholds alone—when making the decision to start RRT. (Not Graded)
- 5.2.1: Discontinue RRT when it is no longer required, either because intrinsic kidney function has recovered to the point that it is adequate to meet patient needs, or because RRT is no longer consistent with the goals of care. (Not Graded)
- 5.2.2: We suggest not using diuretics to enhance kidney function recovery, or to reduce the duration or frequency of RRT. (2B)
- 5.3.1: In a patient with AKI requiring RRT, base the decision to use anticoagulation for RRT on assessment of the patient's potential risks and benefits from anticoagulation (see Figure 17). (Not Graded)
 - 5.3.1.1: We recommend using anticoagulation during RRT in AKI if a patient does not have an increased bleeding risk or impaired coagulation and is not already receiving systemic anticoagulation. (1B)
- 5.3.2: For patients without an increased bleeding risk or impaired coagulation and not already receiving effective systemic anticoagulation, we suggest the following:
 - 5.3.2.1: For anticoagulation in intermittent RRT, we recommend using either unfractionated or low-molecular-weight heparin, rather than other anticoagulants. (1C)
 - 5.3.2.2: For anticoagulation in CRRT, we suggest using regional citrate anticoagulation rather than heparin in patients who do not have contraindications for citrate. (2B)
 - 5.3.2.3: For anticoagulation during CRRT in patients who have contraindications for citrate, we suggest using either unfractionated or low-molecular-weight heparin, rather than other anticoagulants. (2C)

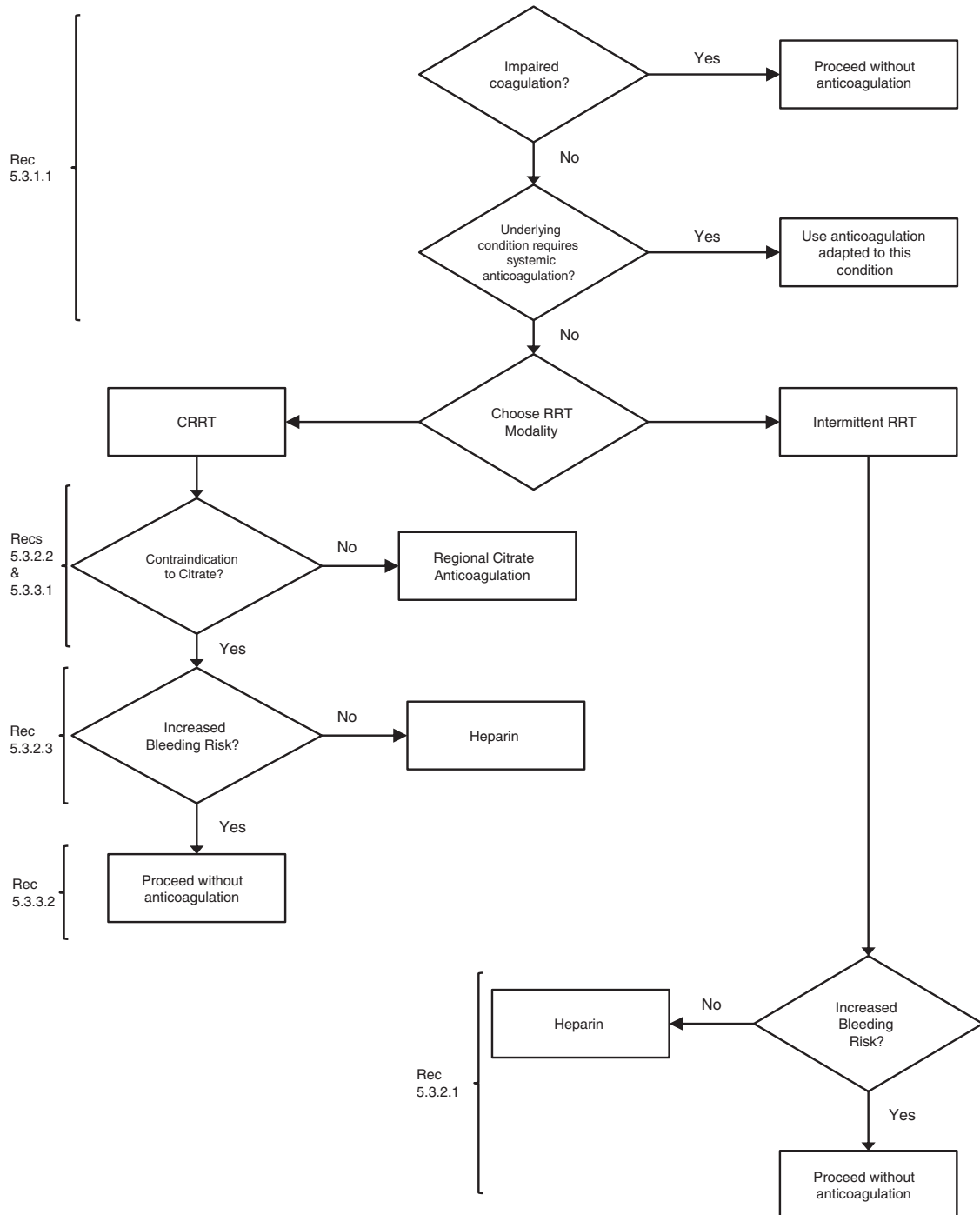


Figure 17 | Flow-chart summary of recommendations. Heparin includes low-molecular-weight or unfractionated heparin. CRRT, continuous renal replacement therapy; RRT, renal replacement therapy.

5.3.3: For patients with increased bleeding risk who are not receiving anticoagulation, we suggest the following for anticoagulation during RRT:

5.3.3.1: We suggest using regional citrate anticoagulation, rather than no anticoagulation, during CRRT in a patient without contraindications for citrate. (2C)

5.3.3.2: We suggest avoiding regional heparinization during CRRT in a patient with increased risk of bleeding. (2C)

- 5.3.4: In a patient with heparin-induced thrombocytopenia (HIT), all heparin must be stopped and we recommend using direct thrombin inhibitors (such as argatroban) or Factor Xa inhibitors (such as danaparoid or fondaparinux) rather than other or no anticoagulation during RRT. (1A)
- 5.3.4.1: In a patient with HIT who does not have severe liver failure, we suggest using argatroban rather than other thrombin or Factor Xa inhibitors during RRT. (2C)
- 5.4.1: We suggest initiating RRT in patients with AKI via an uncuffed nontunneled dialysis catheter, rather than a tunneled catheter. (2D)
- 5.4.2: When choosing a vein for insertion of a dialysis catheter in patients with AKI, consider these preferences (*Not Graded*):
- First choice: right jugular vein;
 - Second choice: femoral vein;
 - Third choice: left jugular vein;
 - Last choice: subclavian vein with preference for the dominant side.
- 5.4.3: We recommend using ultrasound guidance for dialysis catheter insertion. (1A)
- 5.4.4: We recommend obtaining a chest radiograph promptly after placement and before first use of an internal jugular or subclavian dialysis catheter. (1B)
- 5.4.5: We suggest not using topical antibiotics over the skin insertion site of a nontunneled dialysis catheter in ICU patients with AKI requiring RRT. (2C)
- 5.4.6: We suggest not using antibiotic locks for prevention of catheter-related infections of nontunneled dialysis catheters in AKI requiring RRT. (2C)
- 5.5.1: We suggest to use dialyzers with a biocompatible membrane for IHD and CRRT in patients with AKI. (2C)
- 5.6.1: Use continuous and intermittent RRT as complementary therapies in AKI patients. (*Not Graded*)
- 5.6.2: We suggest using CRRT, rather than standard intermittent RRT, for hemodynamically unstable patients. (2B)
- 5.6.3: We suggest using CRRT, rather than intermittent RRT, for AKI patients with acute brain injury or other causes of increased intracranial pressure or generalized brain edema. (2B)
- 5.7.1: We suggest using bicarbonate, rather than lactate, as a buffer in dialysate and replacement fluid for RRT in patients with AKI. (2C)
- 5.7.2: We recommend using bicarbonate, rather than lactate, as a buffer in dialysate and replacement fluid for RRT in patients with AKI and circulatory shock. (1B)
- 5.7.3: We suggest using bicarbonate, rather than lactate, as a buffer in dialysate and replacement fluid for RRT in patients with AKI and liver failure and/or lactic acidemia. (2B)
- 5.7.4: We recommend that dialysis fluids and replacement fluids in patients with AKI, at a minimum, comply with American Association of Medical Instrumentation (AAMI) standards regarding contamination with bacteria and endotoxins. (1B)
- 5.8.1: The dose of RRT to be delivered should be prescribed before starting each session of RRT. (*Not Graded*) We recommend frequent assessment of the actual delivered dose in order to adjust the prescription. (1B)
- 5.8.2: Provide RRT to achieve the goals of electrolyte, acid-base, solute, and fluid balance that will meet the patient's needs. (*Not Graded*)
- 5.8.3: We recommend delivering a Kt/V of 3.9 per week when using intermittent or extended RRT in AKI. (1A)
- 5.8.4: We recommend delivering an effluent volume of 20–25 ml/kg/h for CRRT in AKI (1A). This will usually require a higher prescription of effluent volume. (*Not Graded*)