Chapter 34
Acute Kidney Injury

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BACKGROUND

Acute kidney injury (AKI) is a common postoperative complication. The incidence of AKI after surgery may be as high as 36 % depending on the definition of kidney injury used, the length of time the patient is followed postoperatively, and the type of surgery [1]. Up to 7 % of these patients may go on to require renal replacement therapy (RRT) [2]. Some types of surgeries increase the risk of AKI [3]. The development of postoperative AKI is associated with an increased risk of 30-day readmission [4], length of stay [5, 6], and mortality [1, 5–7]. Therefore, it is important for the internal medicine consultant to consider the risk of postoperative renal failure and how it may be mitigated.

PREOPERATIVE EVALUATION

It is difficult to predict who will develop postoperative AKI. A general surgery acute kidney injury risk index has been described [8], but it is not in wide usage, perhaps because it is not evident what specific measures may reduce risk. Nevertheless, it is useful for the medicine consultant to identify high-risk patients by considering patient- and surgery-specific risk factors.

PATIENT-SPECIFIC RISKS

A large number of risk factors have been associated with the development of postoperative AKI. Patients with preexisting comorbidities tend to be at highest risk. Commonly reported risk factors derived

M.B. Jackson et al. (eds.), *The Perioperative Medicine Consult Handbook*, DOI 10.1007/978-3-319-09366-6_34,

from multivariate analyses from various patient populations and types of surgeries include:

- Lab abnormalities: anemia [9], hypoalbuminemia [6]
- Chronic medical problems: chronic kidney disease, congestive heart failure [8, 10], diabetes [8], ischemic heart disease [10], chronic obstructive pulmonary disease [6], peripheral vascular disease [11]
- Physical examination findings/patient characteristics: obesity [12], advanced age [8, 10, 13], male gender [6, 8], presence of ascites [8], hypertension [6, 8, 14]

SURGERY-SPECIFIC RISKS

In any type of surgery, intrarenal vascular tone can be disrupted due to fluid loss and/or systemic inflammatory response (SIRS) leading to renal ischemia and injury [2]. The following types of surgeries have been particularly associated with a higher risk of developing postoperative AKI:

- Cardiac surgery, especially if requiring cardiopulmonary bypass (CPB)
- Vascular surgery, especially if requiring cross-clamping or contrast
- Renal or urological surgeries
- Intraperitoneal surgery
- Emergency surgery

PERIOPERATIVE MANAGEMENT

DEFINITION OF ACUTE KIDNEY INJURY

Even small changes in serum creatinine (SCr) or transient decreases in urine output (UO) can be harbingers of renal failure and loss of function. Based on the RIFLE (risk, injury, failure, loss, end-stage disease) classification of diminished renal function as defined by the Acute Dialysis Quality Initiative (ADQI) Group [15], the Acute Kidney Injury Network (AKIN) provides a classification schema for AKI that is useful to consider when evaluating patients in the postoperative setting [16]:

- Stage 1: Increase in SCr $\geq$ 0.3 mg/dL or $\geq$1.5–2-fold increase from baseline or UO < 0.5 mL/kg/h for >6 h
- Stage 2: Increase in SCr $\geq$ 2–3-fold increase from baseline or UO < 0.5 mL/kg/h for >12 h
- Stage 3: Increase in SCr $\geq$ 3-fold increase from baseline or SCr $\geq$ 4 mg/dL with acute increase $\geq$0.5 mg/dL or UO < 0.3 mL/kg/h for >12 h or anuria for 12 h

PREVENTION OF POSTOPERATIVE KIDNEY INJURY

A recent Cochrane review did not find any pharmacologic intervention (including dopamine, diuretics, calcium channel blockers, angiotensin-converting enzyme [ACE] inhibitors, *N*-acetylcysteine, sodium bicarbonate, antioxidants, erythropoietin, or selected hydration fluids) that could reliably prevent the development of renal failure in patients with or without preexisting renal failure [17]. Statins may decrease the risk of postoperative AKI but further research is needed before widespread implementation. The majority of cases of AKI are probably largely unavoidable. Nevertheless, attention to key principles may decrease the risk of postoperative AKI:

- Maintain euvolemia based on ongoing clinical assessment—predetermined intravenous fluid rates may overshoot or undershoot actual fluid requirements.
- Maintain cardiac output—overexpansion of intravascular volume in patients with even mild cardiomyopathy or unrecognized myocardial infarction may be manifested as AKI.
- Hypotension and hypovolemia can lead to acute tubular necrosis (ATN), a common cause of postoperative AKI.
- Avoid diuretics unless needed to treat intravascular hypervolemia.
- See Chap. 4 for recommendations regarding management of ACE inhibitors and ARBs. In general, we recommend holding ACE inhibitors and ARBs the morning of surgery unless the patient is persistently hypertensive with a systolic blood pressure greater than 180. If the patient takes these medications in the evening, they may be held or decreased the evening prior to surgery.
- Anemia may be associated with the development of AKI—work up anemia (see Chap. 22) but note that blood transfusion has also been associated with AKI [6, 13].
- See Chap. 33 regarding the prevention of contrast-induced nephropathy.

EVALUATION OF POSTOPERATIVE ACUTE KIDNEY INJURY

Table 34.1 outlines some considerations of the etiology of AKI specific to the postoperative setting. The standard approach to AKI in the medical patient is applicable (e.g., considering pre-, intra-, and postrenal etiologies), but specific attention should be paid to the patient's additional perioperative risk factors. Important elements of the workup include:

- Review operative and anesthesia records: specifically assess for hypotension, blood transfusions, use of diuretics, use of contrast, use of CPB, length of cross-clamp time, urine output, and any operative complications.

TABLE 34.1 CONSIDERATIONS FOR THE ETIOLOGY OF POSTOPERATIVE AKI

Prerenal	Intrinsic renal	Postrenal
■ Hypotension after anesthesia induction ■ Insufficient intraoperative hydration ■ High nasogastric (NG) tube output ■ Increased vascular permeability ("third spacing") ■ Surgical site bleeding/drain output ■ Abdominal compartment syndrome ■ Gastrointestinal bleeding ■ Retroperitoneal bleeding	■ Acute tubular necrosis (ATN) related to intra- or postoperative hypotension ■ Rhabdomyolysis related to positioning/prolonged surgery ■ Embolism (or microemboli) with vascular surgery or CPB ■ Acute interstitial nephritis (AIN) related to antibiotics or diuretics ■ Contrast nephropathy ■ Prolonged cross-clamping in vascular surgery	■ Ureteral damage or compression ■ Bladder outlet obstruction/urinary retention

- Evaluate the patient for intravascular volume status (orthostatic vital signs, jugular venous pressure) and abdominopelvic surgical drain output (increased clear drain output that has a creatinine level greater than serum creatinine level may signify urinary leak).
- Review patient chart for recent contrast studies, ongoing and immediate postoperative urine output, surgical drain output, and nasogastric tube output.
- Urine analysis: muddy brown granular and epithelial cell casts suggest ATN; hematuria may suggest nephrolithiasis, ureteral trauma, or intrinsic renal insult; eosinophiluria may indicate interstitial nephritis; rhabdomyolysis is indicated by urine myoglobin without red blood cells in urinalysis.
- Urine labs: high urinary specific gravity, low urinary sodium, and <1 % fractional excretion of sodium (FENa) support the diagnosis of a prerenal etiology.

- Serum labs: CBC and metabolic panel.
- Studies to consider: bladder scan for post-void residual (perform in and out catheterization if bladder scan values are suspect), retroperitoneal ultrasound (US), or computed tomography (CT) scan (to assess for hydronephrosis, fluid collections).

PRINCIPLES OF MANAGEMENT

The treatment and further workup is predicated on the working diagnosis. Nephrology consultation may be required if establishing euvolemia or relieving urinary tract obstruction does not result in improvement. Of course, a nephrologist should be involved if renal replacement therapy is needed—for example, if the patient has acidosis, volume overload compromising organ function, significant hyperkalemia, or uremia. Key points in management include:

- For all patients, if urine output is difficult to quantify, place a urinary catheter – but remove this as soon as possible.
- In case of intravascular volume depletion, aggressive intravenous fluids (IVF) either with Lactated Ringers (LR) or normal saline (NS) with frequent clinical reassessment of volume status and UO.
- Volume overload and CHF: diurese in the usual fashion; work up for myocardial infarction if CHF is new for the patient.
- If there is obstruction which is not relieved by a urinary catheter, this usually merits rapid surgical or percutaneous intervention.

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