Summer 2022

Acute Kidney Injury in Perioperative Patients

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Introduction

Acute Kidney Injury in Perioperative Patients

- Acute kidney injury (AKI) is a sudden decline in kidney function with a decrease in glomerular filtration and urine output with an accumulation of nitrogenous waste products (McCance & Huether, p. 1291).
- AKI is evident by elevations in plasma creatinine and blood urea nitrogen levels (McCance & Huether, 2019, p. 1291).
- "Among different types of perioperative organ injury, acute kidney injury occurs particularly frequently and has an exceptionally detrimental effect on surgical outcomes" (Gumbert et al., 2020, p. 180).
- Three classifications to guide the diagnosis of AKI have been developed by the Kidney Disease: Improving Global Outcomes (KDIGO) Working Group (McCance & Huether, 2019, p. 1291).

Importance

- In the perioperative period, AKI is a frequent complication linked to increased morbidity and mortality (Zarbock et al., 2018, p. 1236).
- AKI development increases the hospital length of stay and costs, with an estimated $1 billion spent in industrialized countries (Gumbert et al., 2020, p. 182).
- The most common events that lead to AKI are sepsis, surgery, and infiltration of intravenous contrast dye (Billings et al., 2021, p. 409).
- Cardiac surgery associated-acute kidney injury (CSA-AKI) is a common cause of perioperative AKI, occurring in 25% to 30% of patients (Gumbert et al., 2020, p. 182).
- The mortality rate of AKI is about 23%, topping out at 36% for patients with the highest stage and diagnosis (Elghazali et al., 2021, p. 671).
- About 300,000 patients diagnosed with AKI develop chronic kidney disease annually (Gumbert et al., 2020, p. 182).
- This topic was chosen due to the occurrence of AKI among surgical patients and the relevance this disease has to anesthesia providers

Signs & Symptoms

The Mayo Clinic (2020) identifies the following as acute kidney injury symptoms:

- Decreased urine output (oliguria)
- Fluid retention (swelling in legs, ankles, and feet)
- Shortness of breath
- Fatigue
- Confusion
- Nausea
- Weakness
- Irregular heartbeat
- Chest pain or pressure
- Seizures or coma in severe cases

Risk Factors

Elghazali et al. (2021) list risk factors for AKI as the following:

- Age over 65 years
- History of previous AKI
- Chronic kidney disease (GFR<60)
- Sepsis
- Hypovolemia
- Medications with nephrotoxic potential, especially if hypovolemia is also present, e.g., gentamicin, amphotericin, tacrolimus, and NSAIDs
- Antihypertensives and diuretics in the setting of hypovolemia/hypotension, e.g., ACE inhibitors, ARBs, and diuretics
- Exposure to iodinated contrast within the last week
- Symptoms of urological obstruction or conditions that may lead to obstruction
- Diabetes
- Liver failure
- Cardiac failure
- Neurological/cognitive/functional restrictions on the ability to take fluid

KDIGO

Kidney Disease: Improving Global Outcomes AKI Staging Guidelines

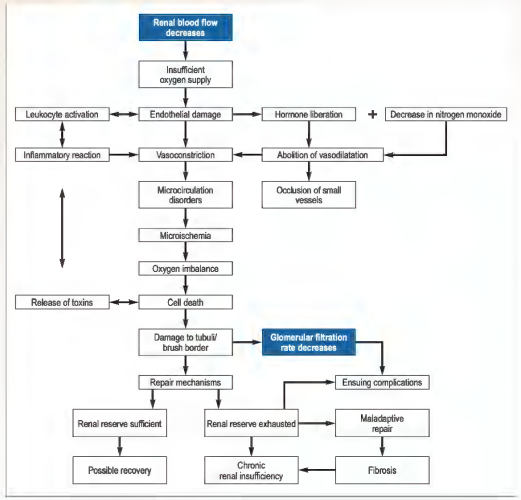
Stage	Serum Creatinine (SCr) criteria
1	1.5-1.9 times baseline or > 0.3mg/dL (> 26.5 mmol/L) increase
2	2.0-2.9 times baseline
3	3.0 times baseline *OR* Increase in serum creatinine to >4.0 mg/dL (>353.65 mmol/L) *OR* Initiation of renal replacement therapy *OR* individuals <18 years, decrease in eGFR to <35mL/min per 1.73m^2

Underlying Pathophysiology

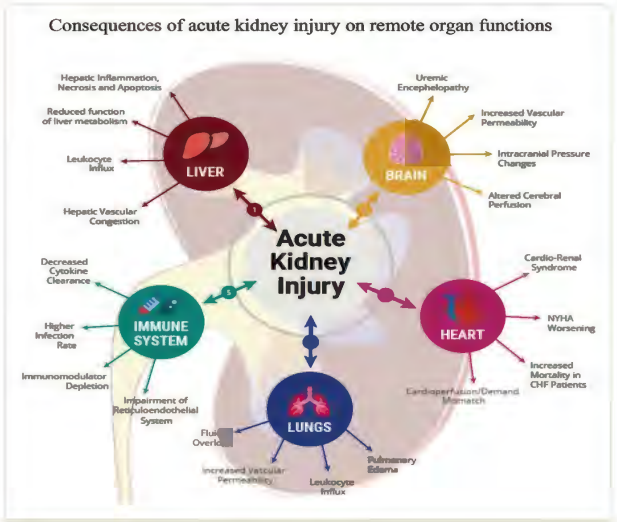
- Common causes of AKI include extracellular volume depletion, decreased renal blood flow, or toxic/inflammatory injury to kidney cells that alter renal function, which can be minimal or severe (McCance & Huether, 2019, p. 1292)
- "In the perioperative period, various factors can reduce the renal blood flow, thus leading to renal hypotension and consequently an insufficient supply of oxygen" (Weiss et al., 2019, p. 834).
- The causes of AKI can be illustrated by judging three categories of injury: (1) renal hypoperfusion (prerenal AKI); (2) disorders involving the renal parenchymal or interstitial tissue (intrarenal or intrinsic AKI); and (3) disorders associated with acute urinary tract obstruction (postrenal AKI) (McCance & Huether, 2019, p. 1291).
- Renal hypoperfusion is the most common cause of AKI, resulting from inadequate kidney perfusion (McCance & Huether, 2019, p. 1292).
- Intrarenal (intrinsic) AKI has the potential to result from ischemic acute tubular necrosis (ATN), nephrotoxic ATN (radiocontrast media), acute glomerulonephritis, diseases of the vasculature, transplant rejection, or interstitial disease (McCance & Huether, 2019, p. 1292-1293).
- "The cortico-medullary junction is relatively hypoxic (PO2 10–20 mmHg) in normal conditions, and during surgery and critical illness, systemic changes in cardiac output, systemic vascular resistance, and renal vein pressure alter cortical and medullary perfusion" (Billings et al., 2021, p. 410).
- Postrenal AKI occurs due to a urinary tract obstruction that affects both kidneys (bilateral ureteral obstruction, bladder outlet obstruction, prostate hypertrophy, tumors, neurogenic bladder, or urethral obstruction) (McCance & Huether, 2019, p. 1293).
- When renal hypoperfusion persists or drops below the autoregulatory range, endogenous vasoconstrictors released from the renal sympathetic system result in afferent arteriolar vasoconstriction (Gumbert et al., 2020, p. 183).
- "Activation of the sympathetic nervous system, stimulation of the renin-angiotensin-aldosterone system, secretion of vasopressin, and release of endothelin-1 further alter the renal microcirculation" (Billings et al., 2021, p. 410).

Significance of Pathophysiology

- Oliguria is maladaptive in the setting of acute tubular necrosis/AKI with volume overload where the kidney is not able to provide the appropriate natriuresis, possibly due to renal congestion in the instance of increased right-sided vascular pressures or activity of antidiuretic hormone (ADH) (Zarbock et al., 2018, p. 1239).
- "In patients who either did not experience AKI or developed KDIGO AKI stage 1 or 2 during the hospital stay, a trend toward increases of GFR and serum creatinine levels, respectively, was observed when comparing the preoperative values to the postoperative measurements performed on the day of surgery" (Just et al., 2021, p. 446).
- Several different processes are involved in perioperative AKI, including hemodynamic instability, microcirculatory dysfunction, endothelial dysfunction, formation of microvascular thrombi, inflammation, tubular cell injury, renal venous congestion, and intra-abdominal hypertension, and tubular obstruction (Ostermann et al., 2020, p. 123-126).
- Renal replacement therapy is required in 1–5% of patients with CSA-AKI and is associated with poor patient and renal prognosis in the short and long-term (Wang & Bellomo, 2017, p. 2).
- Within cardiac surgical settings, AKI is associated with prolonged aortic cross-clamp and ischemia time, the creation of micro- and macroemboli, low cardiac output state, prolonged hypotension, and the use of vasopressors and inotropes (Gumbert et al., 2020, p. 187).
- "In addition to low mean arterial pressure during cardiopulmonary bypass (CPB), there are reports of contact-activated systemic inflammation, triggered by blood flow across the artificial surface of the bypass circuit" (Gumbert et al., 2020, p. 187).

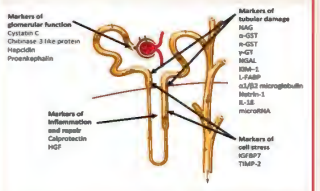


Flow chart of the pathophysiology of acute kidney injury



Labs/Biomarkers

- Serum Creatinine Level
- Serum urea and electrolytes
- Urinalysis
- Imaging of the renal tract
- Renal biopsy
- Furosemide stress test



Ostermann et al., 2020

Nursing and Perioperative Considerations

- Limiting the magnitude and duration of renal ischemia is a critical strategy in reducing the incidence of AKI (Morse, 2023, p. 753).
- Preventions that protect the kidneys include hemodynamic management, fluid management, goal-directed hemodynamic therapy, types of anesthesia/postoperative sedation (propofol, dexmedetomidine), surgical techniques, reducing nephrotoxic exposure, anemia management, and diuretic use (Ostermann et al., 2020, p. 126-128).
- Diuretic therapy and fluid administration must be offset to prevent hypotension and hypoperfusion. The best outcome is a mix of furosemide and fluid balance to avoid a fluid deficit or excess (Morse, 2023, p. 753).
- Prevention of AKI with diuretics is not practical, and utilizing diuretics in established AKI does little to alter the mortality outcome or the need for renal replacement therapy (Morse, 2023, p. 753).
- Treatment of AKI includes administering volume to achieve euvolemia, improving cardiac output by afterload reduction, and normalizing systemic vascular resistance (Morse, 2023, p. 753).

Conclusions

- Pre-surgical assessment should identify patients with risk factors for AKI
- The cause of AKI must be identified
- AKI must be recognized promptly with a rapid response to treat the underlying cause
- Complications of AKI must be identified early with the appropriate response
- Patients at risk of AKI should have an ongoing assessment of their kidney function throughout their hospital stay.
- "Optimal preoperative fluid management may help prevent cardiac surgery AKI because preoperative GFR is a strong predictor for postoperative AKI" (Just et al., 2022, p. 450).
- "A uniform classification of acute kidney injury by the Kidney Disease Improving Global Outcomes workgroup has allowed for advances in medical practice, research, and public health" (Gumbert et al., 2020, p. 196).
- Important prevention strategies include stress prevention, avoidance of nephrotoxins, a focus on the optimization of systemic hemodynamics, and the avoidance of both fluid depletion and fluid overload (Wang & Bellomo, 2017, p.13

References

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