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Acute Kidney Injury

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Topic

Acute Kidney Injury (AKI)

- AKI is defined as "a sudden decline in kidney function with a decrease in glomerular filtration and accumulation of nitrogenous waste products in the blood" (McCance & Huether, 2018, p. 1360).
- Acute kidney injury may be evidenced by elevations in serum creatinine and blood urea nitrogen levels (McCance & Huether, 2018, p. 1360).
- AKI is a common complication of surgery, with an incidence of around 15% (Shen et al., 2021, p. 1).
- Classification of the severity of AKI has been developed and follows the acronym RIFLE:
- **R**isk: increased creatinine > 1.5 or GFR decreased > 25%
- **I**njury: increased creatinine > 2 or GFR decrease > 50**%**
- **F**ailure: Increased creatinine >3 or GFR decrease > 75%
- **L**oss: Loss of kidney function lasting longer than 4 weeks
- **E**SKD: end-stage kidney disease: loss of kidney function lasting longer than 3 months

(McCance & Huether, 2018, p. 1361)

Why AKI?

- Acute kidney injury affects many people within the surgical population.
- Surgical procedures that carry a higher risk for development of AKI include cardiac, major vascular, and abdominal surgeries (Shen et al., 2022, p. 1).
- AKI increases the risk for short- and long-term morbidity and mortality postoperatively (Davison et al., 2022, p. 58).
- Complications from acute kidney injury include chronic kidney disease, myocardial infarction, stroke, heart failure, and death (Davison et al., 2022, p. 58)
- This topic was chosen because of the high prevalence and the direct association from complications during surgery.
- It is crucial to understand the pathophysiology, prevention, and treatment of AKI as an anesthesia provider.

Signs & Symptoms

- Glomerular filtration rate (GFR): a decrease in GFR (normal is 125 milliliters per minute)
- Oliguria: less than 400 milliliters of urine output in a day (McCance & Huether, 2018, p. 1362)
- Azotemia: increased serum urea levels and serum creatinine levels (McCance & Huether, 2018, p. 1360).

Presentation progresses in 3 phases

1. Initiation phase
   - Reduced perfusion or injury to kidneys, lasting 24-36 hours
   - Prevention of injury is possible during the initiation phase
2. Maintenance phase
   - Also known as the oliguric phase
   - Period after injury to the kidney has been established
   - May last weeks to months
   - Urine output is lowest during this phase
   - Serum creatinine, blood urea nitrogen, and serum potassium levels rise
   - Metabolic acidosis may occur
   - Sodium and water overload
3. Recovery phase
   - Time where renal injury is repaired and normal renal function returns
   - Glomerular filtration rate returns but the renal tubules are unable to concentrate the filtrate
   - Diuresis is common and the tubules begin to clear creatinine, with serum creatinine and blood urea nitrogen levels returning to normal
   - It is important to maintain fluid and electrolyte balance during this phase to avoid excessive loss of electrolytes.

(McCance & Huether, 2018, p. 1362)

Figure 1. Mechanisms of Oliguria in AKI. McCance and Huether, 2018, p. 1364

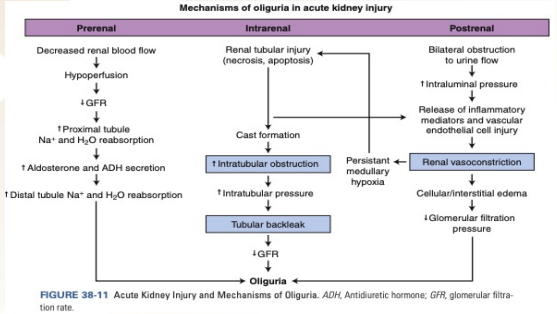


FIGURE 38-11 Acute Kidney Injury and Mechanisms of Oliguria. *ADH*, Antidiuretic hormone; *GFR*, glomerular filtration rate.

Underlying Pathophysiology

- AKI may result from renal hypoperfusion, hypotension, or direct injury to the nephrons of the kidney and may result in minor to severe alterations in kidney function (McCance & Huether, 2018, p. 1360).
- There are three etiologies of acute kidney injury:

1. Prerenal: renal hypoperfusion due to causes including shock, hemorrhage, renal thrombosis, or alterations in renal blood flow
2. Intrarenal (intrinsic): caused by disorders occurring within the kidneys such as acute tubular necrosis, nephrotoxicity, glomerulonephritis, or vascular disease
3. Postrenal: problems associated with urinary tract obstruction including tumors, prostate hypertrophy, neurogenic bladder, and bladder obstruction

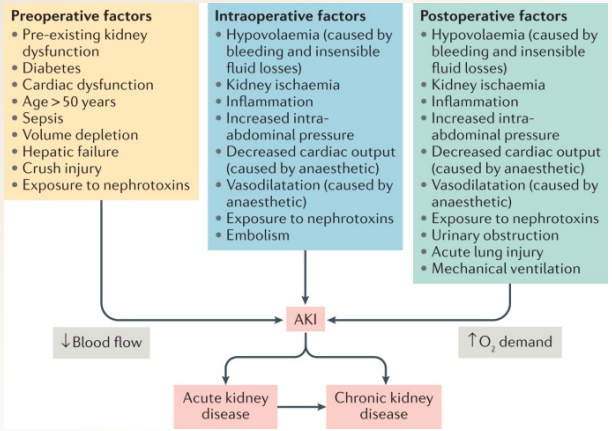
(McCance & Huether, 2018, p. 1361).

- Prerenal causes are the most common and occur because of decreased blood flow to the kidneys (McCance & Huether, 2018, p. 1360).
- During the early stages of prerenal injury, the kidneys maintain adequate perfusion via tubuloglomerular feedback by dilating the afferent arteriole and constricting the efferent arteriole (McCance and Huether, 2018, p. 1361).
- If the kidneys are unable to maintain perfusion, glomerular filtration rate ultimately decreases (McCance and Huether, 2018, p. 1361).

Significance of Pathophysiology

- "Acute kidney injury is one of the most frequent and debilitating complications of surgery and critical illness" (Billings et al., 2021, p. 409).
- Patients are at a high risk of developing acute kidney injury during surgical procedures.
- AKI incidence in surgical patients can be as high as 50% depending on the procedure and existing comorbidities (Sanaiha et al., 2020, p. 328).
- Development of a postoperative AKI can serve as a predictor for postoperative mortality (Sanaiha et al., 2020, p. 328).
- While improvements in the diagnosis of acute kidney injury have continued to advance, the mortality relating to acute kidney injury remains high (Wu et al., 2019, p. 866).
- Prompt recognition and treatment of AKI are pertinent to preventing morbidity and mortality of these patients.

Figure 2. Pathophysiology of Postoperative AKI. Prowle et al., 2021.



Implications for Nursing Care

- Nurses and advanced practice providers should be aware of factors that may place patients at risk for postoperative AKI, including preexisting renal disease, advanced age, intraoperative hypotension, and high-risk surgical procedures (Davison et al., 2022, p. 62).
- Maintenance of adequate fluid volume status before and after surgery can help in preventing renal hypoperfusion (McCance & Huether, 2018, p. 1364).
- Once an AKI has occurred, management should be directed towards the following:

1. Correction of fluid imbalances and electrolyte disturbances
2. Management of blood pressure
3. Avoidance of nephrotoxic drugs and drugs that have toxic metabolites
4. Adequate nutrition
5. Prevention of infection and prompt treatment if an infection arises

(McCance & Huether, 2018, p. 1364)

Diagnosis

- Rising serum creatinine and decreasing levels of urine output are often utilized in the diagnosis of acute kidney injury (McCance & Huether, 2018, p. 1364).
- When diagnosing AKI, one must consider the cause of the disease and whether it was prerenal, intrarenal, or postrenal (McCance & Huether, 2018, p. 1363).
- It can be difficult to differentiate between prerenal causes and acute tubular necrosis.
  - In prerenal causes, the renal tubules maintain their ability to reabsorb sodium, water, and urea.
  - In acute tubular necrosis reabsorption is compromised.
  - Blood urea nitrogen (BUN) levels will rise, while creatinine levels will remain the same in prerenal causes, making the BUN to creatinine ratio elevated (>15:1)

(McCance & Huether, 2018, p. 1363).

- While these methods are helpful in diagnosis of AKI, it may take hours to days for a diagnosis to be made, leaving acute kidney injury unrecognized (McCance & Huether, 2018, p. 1364).
- Newer biomarkers are being identified and may prove helpful in a quicker diagnosis of AKI (McCance & Huether, 2018, p. 1364).
- Included in these biomarkers are "neutrophil gelatinase—associated lipocalin, urinary cystatin C, kidney injury molecule-1, IL-18, renal vanin-1 protein, and hematopoietic growth factor—inducible neurokinin 1" (McCance & Huether, 2018, p. 1364).
- Recent studies exploring these biomarkers point to them potentially being more reliable than rises in serum creatinine, although further research should continue (McCance & Huether, 2018, p. 1364).

Treatment

- Fluid replacement: consider losses (through urine and insensible losses) and replace fluid, being careful not to overhydrate and dilute serum sodium (McCance & Huether, 2018, p. 1364).
- Manage hyperkalemia with non-potassium sparing diuretics, dietary restriction of potassium, and exchange resins that decrease potassium. If hyperkalemia is severe, dialysis may be required. (McCance & Huether, 2018, p. 1364).
- Nutrition should be aimed towards a low-protein, high-carbohydrate diet to control azotemia (McCance & Huether, 2018, p. 1364).
- If acidosis is uncontrollable, the patient is severely fluid overloaded, or has severe electrolyte imbalances, then continuous renal replacement therapy (CRRT) should be considered. CRRT is indicated in critical patients and involves the mechanical removal of water, toxins, and electrolytes from the blood at a slower rate than traditional dialysis (McCance & Huether, 2018, p. 1364).

Perioperative Considerations

- Mean arterial blood pressure should be maintained > 60 mmHg and episodes of hypotension corrected as soon as possible (Weiss et al., 2019, p. 838).
- Fluid volume status should be maintained before, during, and after surgery (McCance and Huether, 2018, p. 1364).
- For patients at high risk for AKI, avoidance of nephrotoxic drugs is crucial (Weiss et al., 2019, p. 837).
- Maintenance of normoglycemia should be maintained throughout the surgical procedure (Weiss et al., 2019, p. 837).
- Monitor for early signs of kidney injury including low urine output, rising serum creatinine levels, and rising blood urea nitrogen levels (Weiss et al., 2019, p. 837).

Conclusions

- Acute kidney injury is a common postoperative complication and can lead to increased morbidity and mortality if not promptly identified and treated.
- Risk factors for development of AKI include advanced age, male gender, obesity, and comorbidities including diabetes, heart disease, and hypertension (Shen et al., 2022, p. 2).
- Proper management of patients intraoperatively can greatly reduce the risk of developing acute kidney injury postoperatively.
- Mechanisms that cause acute kidney injury have been well researched but further research into prevention of AKI is warranted. Especially as our mean population age increases, along with the incidence of comorbidities, increasing the risk for development of acute kidney injury following surgery (Sanaiha et al., 2020, p. 332).

References

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