Acute diseases of the urogenital system, electrolyte disturbances

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- Acute Kidney Injury (AKI)
- Urinary Tract Infections (UTIs)
- Disorders of Potassium: Hypo- and Hyperkalemia

Acute Kidney Injury (AKI)

Acute kidney injury is a rapid decrease in renal function over days to weeks, causing an accumulation of nitrogenous products in the blood (azotemia) with or without reduction in amount of urine output.

- In all cases of acute kidney injury (AKI), creatinine and urea build up in the blood over several days, and fluid and electrolyte disorders develop.
- The most serious of these disorders are **hyperkalemia** and **fluid overload** (possibly causing pulmonary edema).
- **Acidosis** develops because hydrogen ions cannot be excreted.

- **Hypocalcemia** is thought to occur because the impaired kidney no longer produces calcitriol and because hyperphosphatemia causes calcium phosphate precipitation in the tissues.
- Phosphate retention leads to **hyperphosphatemia**.

Etiology

Causes of acute kidney injury can be classified as

- Prerenal
- Renal
- Postrenal

Prerenal AKI is due to inadequate renal perfusion. The main causes are:

- Extracellular fluid volumen depletion (eg, due to diarrhea, inadequate fluid intake)
- Cardiovascular disease (eg, heart failure, shock)
- Decompensated liver disease

Renal causes

Disorders may involve the blood vessels, glomeruli, tubules, or interstitium.

Glomerular Disorders (renal causes of AKI)

The hallmark of glomerular disorders is **proteinuria**, which is often in the nephrotic range (≥ 3 g/day).

Glomerular disorders are classified based on urine changes as those that manifest predominantly with

- Nephrotic range proteinuria, and nephrotic urine sediment (fatty casts, oval fat bodies, but few cells or cellular casts)
- Hematuria, usually in combination with proteinuria (which may be in the nephrotic range); the RBCs are usually dysmorphic and often there are RBC or mixed cellular casts (nephritic urine sediment)

Clinical manifestations

- **Nephritic syndrome** is nephritic urine sediment with or without hypertension, elevated serum creatinine, and oliguria

- **Nephrotic syndrome** is nephrotic urine sediment plus edema and hypoalbuminemia (typically with hypercholesterolemia and hypertriglyceridemia).

GLOMERULAR DISORDERS BY MANIFESTATIONS AND AGE

Age (yr)	Nephritic Syndrome	Nephrotic Syndrome	Mixed Nephritic and Nephrotic Syndrome
< 15	IgA nephropathy IgA-associated vasculitis Lupus nephritis Mild PIGN	Lupus nephritis Minimal change disease Focal segmental glomerulosclerosis	Membranoproliferative GN Lupus nephritis
15–40	IgA nephropathy Lupus nephritis Hereditary nephritis RPGN PIGN	Focal segmental glomerulosclerosis Lupus nephritis Diabetic nephropathy Preeclampsia IgA nephropathy Minimal change disease Late PIGN	Membranoproliferative GN IgA nephropathy Lupus nephritis RPGN
> 40	Vasculitides IgA nephropathy RPGN PIGN	Membranous nephropathy Minimal change diseses Focal segmental glomerulosclerosis Diabetic nephropathy IgA nephropathy Light chain deposition disease Late PIGN	RPGN IgA nephropathy

RPGN = rapidly progressive glomerulonephritis
PIGN = postinfectious glomerulonephritis
GN = glomerulonephritis

Postrenal AKI is due to various types of obstruction in the voiding and collecting parts of the urinary system.

Ureteric obstruction

- Stone disease
- Tumor
- Fibrosis
- Ligation during pelvic surgery

Bladder neck obstruction

- Benign prostatic hypertrophy
- Cancer of the prostate
- Drugs (tricyclic antidepressants, ganglion blocker)
- Bladder tumor
- Stone disease

Urethral obstruction

- Tumor
- Strictures

(To produce significant AKI, obstruction at the level of the ureter requires involvement of both ureters unless the patient has only a single functioning kidney.)

Symptoms and Signs

Initially, weight gain and peripheral edema may be the only findings.

Symptoms of uremia may develop later as nitrogenous products accumulate. Such symptoms include

- Anorexia
- Nausea
- Vomiting
- Weakness
- Myoclonic jerks
- Seizures
- Confusion
- Coma

Diagnosis

- Clinical evaluation, including review of prescription and over-the-counter drugs and exposure to IV contrast (iodinated)
- Serum creatinine
- Urinary sediment
- Urinary diagnostic indices
- Urinalysis and assessment of urine protein
- Postvoid residual bladder volume and/or renal ultrasonography if postrenal cause suspected

- Acute kidney injury (AKI) is suspected when urine output falls or serum blood urea nitrogen (BUN) and creatinine rise.

- Per the KDIGO (Kidney Disease: Improving Global Outcomes) Clinical Practice Guideline for Acute Kidney Injury, AKI is defined as any of the following:

  - Increase in the serum creatinine value of ≥ 0.3 mg/dL (26.52 micromol/L) in 48 hours
  - Increase in serum creatinine of ≥ 1.5 times baseline within the prior 7 days
  - Urine volume < 0.5 mL/kg/hour for 6 hours

- Other laboratory findings are

  - Progressive acidosis
  - Hyperkalemia
  - Hyponatremia
  - Anemia

Treatment

- Immediate treatment of pulmonary edema and hyperkalemia
- Dialysis as needed to control hyperkalemia, pulmonary edema, metabolic acidosis, and uremic symptoms
- Adjustment of drug regimen for degree of renal dysfunction
- Usually restriction of water, sodium, phosphate, and potassium intake, but provision of adequate protein
- Possibly phosphate binders (for hyperphosphatemia) and sodium polystyrene sulfonate (for hyperkalemia)

Urinary Tract Infections (UTIs)

Urinary tract infections (UTIs) can be divided into

- upper tract infections, which involve the kidneys (pyelonephritis), and
- lower tract infections, which involve the
  - bladder (cystitis),
  - urethra (urethritis), and
  - prostate (prostatitis).

Urinary Tract Infections (UTIs)

Most cystitis and pyelonephritis are caused by bacteria.

The most common nonbacterial pathogens are fungi (usually candidal species), and, less commonly, mycobacteria, viruses, and parasites.

Nonbacterial pathogens usually affect patients who are immunocompromised (diabetes, obstruction, or structural urinary tract abnormalities).

Urinary Tract Infections (UTIs)

Uncomplicated UTI occurs in premenopausal adult women with

- no structural or functional abnormality of the urinary tract and who are
- not pregnant and
- have no significant comorbidity that could lead to more serious outcomes.

Complicated UTI can involve either sex at any age. A UTI is considered complicated if the patient is a child, is male, is pregnant, or has any of the following:

- A structural or functional urinary tract abnormality and obstruction of urine flow
- A comorbidity that increases risk of acquiring infection or resistance to treatment, such as poorly controlled diabetes, chronic kidney disease, or immunocompromise
- Recent instrumentation or surgery of the urinary tract

Etiology (UTIs)

The bacteria that most often cause cystitis and pyelonephritis are the following:

- Enteric, usually gram-negative aerobic bacteria
- Gram-positive bacteria

In normal GU tracts:

- *Escherichia coli* (75 to 95%)

  Klebsiella or *Proteus mirabilis*, *Pseudomonas aeruginosa*
- Gram-positive bacteria: *Staphylococcus saprophyticus* (5 to 10%)

In hospitalized patients:

- *E. coli* (~50% of cases)

  Gram-negative species: *Klebsiella*, *Proteus*, *Enterobacter*, *Pseudomonas*, and *Serratia* account
- Gram-positive bacterial cocci *E. faecalis*, *S. saprophyticus*, and *Staphylococcus aureus*

Symptoms and Signs

Part of urinary tract affected	Signs and symptoms
Urethra (urethritis)	•Burning with urination •Discharge
Bladder (cystitis)	•Pelvic pressure •Lower abdomen discomfort •Frequent, painful urination •Blood in urine
Kidneys (acute pyelonephritis)	•Upper back and side (flank) pain •High fever •Shaking and chills •Nausea •Vomiting

Diagnosis

- Urinalysis
- Urine culture

Microscopic examination

Pyuria is defined as ≥ 8 WBCs/µL
Most truly infected patients have > 10 WBCs/µL.

Microscopic hematuria occurs in up to 50% of patients.

Presence of **bacteria** on UA.

Dipstick tests also are commonly used. A positive **nitrite** test on a freshly voided specimen is highly specific for UTI.

Treatment

- Antibiotics
- Occasionally surgery (eg, to drain abscesses, correct underlying structural abnormalities, or relieve obstruction)

Drugs commonly recommended for **simple** UTIs include:

- Trimethoprim/sulfamethoxazole
- Fosfomycin
- Nitrofurantoin
- Cephalexin
- Ceftriaxone

Severe infection

- For a severe UTI, you may need treatment with intravenous antibiotics in a hospital (see Society guidelines).

Disorders of Potassium: Hypokalemia

Hypokalemia is serum potassium concentration < 3.5 mEq/L caused by a deficit in total body potassium stores or abnormal movement of potassium into cells.

Etiology

Hypokalemia can be caused by decreased intake of potassium but is usually caused by excessive losses of potassium in the urine or from the GI tract.

GI tract losses

- Chronic diarrhea, including chronic laxative abuse and bowel diversion
- Clay ingestion, which binds potassium and greatly decreases absorption
- Rarely, villous adenoma of the colon, which causes massive potassium secretion

Intracellular shift

- Glycogenesis during total parenteral nutrition or enteral hyperalimentation (stimulating insulin release)
- After administration of insulin
- Stimulation of the sympathetic nervous system, particularly with beta 2-agonists
- Thyrotoxicosis due to excessive beta-sympathetic stimulation
- Familial periodic paralysis

Etiology

Renal potassium losses

Various disorders can increase renal potassium excretion.

Excess mineralocorticoid effect can directly increase potassium secretion by the distal nephrons and occurs in any of the following:

- Adrenal steroid excess that is due to Cushing syndrome, primary hyperaldosteronism, rare renin-secreting tumors, and congenital adrenal hyperplasia.
- Bartter syndrome
- Gitelmann syndrome

Liddle syndrome

Etiology

Drugs

Diuretics

- Loop diuretics
- Thiazide diuretics
- Osmotic diuretics

Laxatives

Other drugs

- Amphotericin B
- Antipseudomonal penicillins (eg, carbenicillin)
- Penicillin in high doses
- Theophylline (both acute and chronic intoxication)

Symptoms and Signs

- Mild hypokalemia (serum potassium 3 to 3.5 mEq/L) rarely causes symptoms.
- Serum potassium < 3 mEq/L generally causes muscle weakness and may lead to paralysis and respiratory failure.
- Other muscular dysfunction includes cramping, fasciculations, paralytic ileus, hypoventilation, hypotension, tetany, and rhabdomyolysis.
- Persistent hypokalemia can impair renal concentrating ability, causing polyuria with secondary polydipsia.

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ECG

ECG should be done on patients with hypokalemia!

Hypokalemia causes

- sagging of the ST segment,
- depression of the T wave, and
- elevation of the U wave.

Hypokalemia may cause premature ventricular and atrial contractions, ventricular and atrial tachyarrhythmias, and 2nd- or 3rd-degree atrioventricular block.

Treatment

- Oral potassium supplements
- IV potassium supplements for severe hypokalemia or ongoing potassium losses

Disorders of Potassium: Hyperkalemia

Hyperkalemia is a serum potassium concentration > 5.5 mEq/L, usually resulting from decreased renal potassium excretion or abnormal movement of potassium out of cells.

Factors Contributing to Hyperkalemia

Increased potassium intake (usually iatrogenic)

Oral	Dietary Oral potassium supplements
IV	Blood transfusions, IV fluids with supplemental potassium Potassium citrate solutions, Potassium-containing drugs (eg, penicillin G)

Decreased potassium excretion

Drugs	Potassium-sparing diuretics ACE inhibitors, Angiotensin II receptor blockers Direct renin inhibitor Heparin, Lithium NSAIDs
Hypoaldosteronism	Adrenal insufficiency
Kidney disorders	Acute kidney injury Chronic kidney disease Obstruction
Other	Decreased effective circulating volume

Factors Contributing to Hyperkalemia

Increased potassium movement out of cells

Drugs	Beta-blockers, Digoxin toxicity
Increased tissue catabolism	Acute tumor lysis, Acute intravascular hemolysis Rhabdomyolysis, Burns
Insulin deficiency	Diabetes mellitus Fasting
Disorders	Hyperkalemic familial periodic paralysis (rare)
Other	Metabolic acidosis

Symptoms and Signs

- asymptomatic (usually)

- cardiac arrhythmias
- flaccid paralysis

ECG

ECG should be done on patients with hypokalemia!

Slowing of conduction is characterized by an

- increased PR interval and shortening of the QT interval.
- Tall, symmetric, peaked T waves are visible initially.

Potassium > 6.5 mEq/L causes further slowing of conduction with widening of the QRS interval, disappearance of the P wave, and nodal and escape ventricular arrhythmias.

Finally, the QRS complex degenerates into a sine wave pattern, and ventricular fibrillation or asystole ensues.

Treatment

- Treatment of the cause

- For mild hyperkalemia: sodium polystyrene sulfonate
- For moderate or severe hyperkalemia:
  - IV insulin and glucose,
  - IV calcium solution,
  - possibly an inhaled beta 2-agonist, and
  - hemodialysis