

L17: Acute Kidney Injury



objectives

1. Define Acute Kidney Injury
2. Know the epidemiology of Acute Kidney Injury
3. Know the etiology of Acute Kidney Injury
4. Manage Acute Kidney Injury
5. Diagnose Acute Kidney Injury
6. Treat Acute Kidney Injury

Definition of AKI

- Definition: A rapid decline in renal function, with an increase in serum creatinine level
- ARF (acute renal failure) in one study was defined as:
 - a 0.5 mg/dL increase in serum creatinine if the baseline serum creatinine was ≤ 1.9 mg/dL,
 - an 1.0 mg/dL increase in serum creatinine if the baseline serum creatinine was 2.0 to 4.9 mg/dL, and
 - a 1.5 mg/dL increase in serum creatinine if the baseline serum creatinine was ≥ 5.0 mg/dl
- An abrupt “sudden” (within 48 hours) absolute increase in creatinine by 0.3 mg/dl (26.4 $\mu\text{mol/l}$) or percentage increase of $>50\%$ from base line or urine output <0.5 ml/hour for 6 hours
- The creatinine may be normal despite a markedly reduced glomerular filtration rate
- (GFR) in the early stages of AKI due to the time it takes for creatinine to accumulate
- in the body. This condition is also called acute renal failure (ARF).

Epidemiology

- It occurs in
 - 5% of all hospitalized patients and
 - 35% of those in intensive care units
- Mortality is high:
 - up to 75–90% in patients with sepsis
 - 35–45% in those without

The RIFLE criteria for AKI :

- 1.5-fold increase in the serum creatinine or GFR decrease by 25% or urine output <0.5 mL/kg/hour for 6 hours \longrightarrow **(RISK)**
- Twofold increase in the serum creatinine or GFR decrease by 50% or urine output <0.5 mL/kg/hour for 12 hours \longrightarrow **(INJURY)**
- Threefold increase in the serum creatinine or GFR decrease by 75% or urine output of <0.5 mL/kg/hour for 24 hours, or anuria for 12 hours \longrightarrow **(FAILURE)**
- Complete loss of kidney function (i.e., requiring dialysis) for more than 4 weeks \longrightarrow **(LOSS)**
- Complete loss of kidney function (i.e., requiring dialysis) for more than 3 months \longrightarrow **(ESRD)**

Signs & Symptoms

1. oliguric, anuric, or non-oliguric
2. Weight gain and edema are the most common clinical findings “this is due to a positive water and sodium (Na+) Balance”.
3. Characterized by azotemia “abnormally high levels of nitrogen-containing compounds (such as urea, creatinine, various body waste compounds, and other nitrogen-rich compounds) in the blood” (elevated BUN and Cr).



Nota that:

- ✓ Elevated BUN “blood urea nitrogen” is also seen with catabolic drugs (e.g., steroids), GI/soft tissue bleeding, and dietary protein intake.
- ✓ Elevated Cr is also seen with increased muscle breakdown and various drugs.
- ✓ The baseline Cr level varies proportionately with muscle mass.

Correlation between AKI classification and outcome:

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	No AKI 65.6%	AKI I 19.1%	AKI II 3.8%	AKI III 12.5%
Urine Output		<0.5 ml/kg/h for >6 h	<0.5 ml/kg/h for >12 h	<0.3 ml/kg/h for >24 h OR Anuria for >12 h
Creatinine criteria		1.5-2 times baseline OR 0.3 mg/dl increase from baseline (≥ 26.4 μmol/L)	2-3 times baseline	3 times baseline OR 0.5 mg/dl (44 μmol/L) increase if baseline > 4mg/dl (≥ 354 μmol/L) OR Any renal replacement therapy given
Mean age	60.5	62.1	60.4	61.1
ICU mortality	10.7%	20.1%	25.9%	49.6%
Hospital mortality	16.9%	29.9%	35.8%	57.9%
Length of stay in ICU (median)	2 d	5 d	8 d	9 d

Types of AKI causes

Pre renal

- 1-Volume depletion
- 2-Decreased cardiac output
- 3- hypotension
- 4- renal artery occlusion

Renal

- 1-Acute Tubular necrosis (ATN)
- 2-Acute interstitial nephritis (AIN)
- 3-Acute Glomerulonephritis (GN)
- 4-Vascular disorder

Post Renal

- 1-Ureteric obstruction
- 2-Bladder neck obstruction
- 3-Urethral obstruction

Clinical Consequences:

- A. Chronic Kidney disease
- B. End Stage Renal Disease
- C. Hospitalization
- D. Mortality

Pre-renal failure

- **Most common cause of AKI.**
- Potentially reversible.
- decrease in systemic arterial blood volume (renal perfusion) or can complicate any disease that causes:
 - **hypovolemia.**
 - Or, low cardiac output.
 - Or, systemic vasodilatation.

Etiology

Hypovolemia

1/dehydration
 2/excessive diuretic use,
 3/poor fluid intake,
 4/vomiting,
 5/diarrhea,
 6/ Cutaneous losses (burns)
 7/hemorrhage
 8/Pancreatitis

Hypotension

1/sepsis,
 2/excessive antihypertensive medications,
 3/bleeding,
 4/dehydration

Renal arterial obstruction

*Stenosis (kidney is hypoperfused despite elevated blood pressure.)

decreased renal perfusion

1/NSAIDs (constrict afferent arteriole),
 2/ACE inhibitors (cause efferent arteriole vasodilation)
 3/cyclosporin

Decreased cardiac output

- Heart failure
- Pulmonary embolus
- Acute myocardial infarction
- Severe valvular heart disease
- Abdominal compartment syndrome (tense ascites)

Cirrhosis, hepatorenal syndrome

Pathophysiology



- Because the renal parenchyma is undamaged, so tubular function is preserved (and therefore the concentrating ability).
- Therefore, the kidney responds appropriately, conserving as much (sodium + water) as possible.
- This form of AKI is reversible on restoration of blood flow; **but** if hypoperfusion persists, ischemia results and can lead to acute tubular necrosis (ATN) (as a renal causes)

Clinical features and Laboratory findings

signs of volume depletion:

- dry mucous.
- Membranes.
- Hypotension.
- Tachycardia.
- Decreased tissue turgor.
- oliguria/anuria*.

1-Decrease

-urine Na+**

2-Increase

-BUN-to-serum Cr ratio***

- urine osmolality×

-urine-plasma Cr ratio"

3-Bland urine sediment.

***always** found in prerenal failure (this is to preserve volume)

**(<20 mEq/L with fractional excretion of sodium [FENa] <1%) because Na⁺ is avidly reabsorbed

***(>**20:1** is the classic ratio)—because kidney can reabsorb urea

×(>500 mOsm/kg H₂O)—because the kidney is able to reabsorb water

" (>40:1)—because much of the filtrate is reabsorbed (but not the creatinine)

renal failure

Kidney tissue is damaged such that glomerular filtration and tubular function are significantly impaired . The kidneys are unable to concentrate urine effectively.

Causes

“Generally”



*Anti-glomerular basement membrane (GBM) disease

**Anti-neutrophil cytoplasmic antibody-associated glomerulonephritis (ANCA-associated GN)

***Immune complex GN

1- Tubular disease (ATN)

1/ischemia (**most common cause**),
2/nephrotoxins

2- Glomerular disease

1/Goodpasture's syndrome*
2/Wegener's granulomatosis**
3/poststreptococcal GN, lupus***

3- Interstitial disease

1/allergic interstitial nephritis often due to hypersensitivity reaction to medication .

1- Tubular Necrosis (ATN)

Causes of Acute Tubular Necrosis (ATN):

- **Ischemic AKI:** Secondary to severe decline in renal blood flow, as in shock, hemorrhage, sepsis, disseminated intravascular coagulation, heart failure, so Ischemia will results in the death of tubular cells.
- **Nephrotoxic AKI:** Injury secondary to substances (e.g: **contrast, gentamicins: aminoglycosides and vancomycin, NSAIDs** poisons, myoglobinuria (from muscle damage, rhabdomyolysis, strenuous exercise), hemoglobinuria (from hemolysis), chemotherapeutic drugs (cisplatin), and kappa and gamma light chains produced in multiple myeloma) that directly injure renal parenchyma and result in cell death.

Clinical features and Laboratory findings

Clinical features:

depend on the cause. **Edema** is usually present. Recovery may be possible but takes longer than in prerenal failure.

Decrease

- 1/ BUN-to-serum Cr ratio*
- 2/urine osmolality**
- 3/urine-plasma Cr ratio***

Increase

urine Na+ “

- * (<20:1, typically closer to 10:1 ratio) in comparison with prerenal failure. Both BUN and Cr levels are still elevated, but less urea is reabsorbed than in prerenal failure.
- ** (<350 mOsm/kg H₂O)—because renal water reabsorption is impaired
- *** (<20:1)—because filtrate cannot be reabsorbed)
- “ (>40 mEq/L with FENa > 2% to 3%)—because Na⁺ is poorly reabsorbed

Pre renal vs ATN

	Pre renal	Acute Tubular necrosis (ATN)
Urea/ Creatinine ration	>20:1	10-15:1
Urine	Normal	Muddy brown casts
Urine Osmolality	> 500	<350
Urine Na	<20	>20
Fractional excretion of Na	<1 %	> 1%

Tubular disease

- **Rhabdomyolysis**

1. Skeletal **muscle breakdown** caused by trauma, crush injuries, prolonged immobility, seizures, snake bites.
2. **Release** of muscle fiber contents (**myoglobin**) into bloodstream. Myoglobin is toxic to kidneys, which can lead to AKI.
3. **Lab findings** include markedly elevated **creatine phosphokinase (CPK)**, **hyperkalemia**, **hypocalcemia**, **hyperuricemia** and **Hb is positive**.
4. **Treatment** with IV fluids, mannitol (osmotic diuretic) and bicarbonate (drives K back into cells).

Course of ATN

- **Onset (insult)**

- **Oliguric phase:**

- Azotemia and uremia—average length 10 to 14 days

- Urine output <400 to 500 mL/day

- **Diuretic phase:**

- Begins when urine output is >500 mL/day

- High urine output due to the following:

- fluid overload** (excretion of retained salt, water, other solutes that were retained during oliguric phase), osmotic diuresis due to retained solutes during oliguric phase

- tubular cell damage (delayed recovery of epithelial cell function relative to GFR)**

- **Recovery phase**—recovery of tubular function

2- Acute Glomerulonephritis

- Rare in the hospitalized patient
- Diagnose by history, hematuria, RBC casts, proteinuria (usually non-nephrotic range), low serum complement in post-infectious GN), RPGN often associated with anti-GBM or ANCA
- Usually will need to perform renal biopsy

3- Interstitial disease

Box 1: Common causes* of acute interstitial nephritis^{2,4,6}

Drugs

- Antimicrobials (ampicillin, ciprofloxacin, methicillin, penicillin, rifampicin, sulfonamides)
- Nonsteroidal anti-inflammatory drugs (acetylsalicylic acid, fenoprofen, ibuprofen, indomethacin, naproxen, phenylbutazone, piroxicam, tolmetin, zomepirac)
- Acid suppressors (omeprazole, pantoprazole, rabeprazole, cimetidine)
- Others (phenytoin, furosemide, allopurinol, phenindione)

Infections

- Direct infiltration (leptospirosis, cytomegalovirus, candidiasis)
- Reactive to systemic infections (streptococcal infection, diphtheria, *Hantavirus*)

Systemic diseases

- Metabolic diseases (urate nephropathy, hypercalcemic nephropathy, oxalate nephropathy)
- Immunologic reactions (transplant rejection, systemic lupus erythematosus, sarcoidosis, cryoglobulinemia)
- Neoplastic diseases (lymphoproliferative diseases)

Idiopathic causes

*These are the most common causes of acute interstitial nephritis, but this list is not exhaustive.

Box 2: Characteristics of acute interstitial nephritis

- Acute elevation in creatinine levels (not caused by pre- or post-renal etiologies)
- General malaise, nausea (caused by buildup of metabolites)
- Normal blood pressure, no edema (distinguishes acute interstitial nephritis from acute tubular necrosis)
- Polyuria and polydypsia (kidneys unable to concentrate urine)
- Maculopapular rash (may be an early indication of drug-induced acute interstitial nephritis)
- Proteinuria (caused by tubular damage)
- Pyuria (occurs in almost all cases)
- Hematuria (occurs in about 90% of cases)
- Eosinophiluria (occurs in 80% of cases of drug-induced acute interstitial nephritis)

Postrenal failure

- a. Least common cause of AKI
- b. Obstruction of any segment of the urinary tract (**with intact kidney, Blood supply and renal parenchyma are intact as well**) causes increased tubular pressure (urine produced cannot be excreted), which leads to decreased GFR, and **note that both kidneys must be obstructed for creatinine to rise.**
- c. Renal function is restored if obstruction is relieved before the kidneys are damaged.
- d. Postrenal obstruction, if untreated, can lead to ATN.

Causes

Ureteric obstruction

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

Bladder neck obstruction

- Benign prostatic hypertrophy [BPH]
- Cancer of the prostate
- Neurogenic bladder
- Drugs (Tricyclic antidepressants, ganglion blockers,
- Bladder tumor,
- Stone disease, hemorrhage/clot)
- Retroperitoneal fibrosis

Urethral obstruction (strictures, tumor), (BPH) is the most common cause.

Urethral obstruction is an uncommon cause because obstruction must be bilateral to cause renal failure.

Retroperitoneal fibrosis

Urine Osmolality

- 1.** Urine osmolality is a measure of urine concentration. The higher the osmolality, the more concentrated the urine.
- 2.** Dehydration in a healthy person leads to increase in urine concentration (osmolality) as follows: Dehydration causes low intravascular volume, which triggers ADH release, which stimulates reabsorption of water from kidney to fill the vasculature. Increased water reabsorption leads to more concentrated urine.
- 3.** In ATN, the tubule cells are damaged and cannot reabsorb water (or sodium); so the urine cannot be concentrated, which leads to low urine osmolality.

Diagnosis

Blood tests

- Elevation in BUN and Cr levels
- Electrolytes (K⁺, Ca²⁺, PO₄), albumin levels, CBC with differential

Urine culture

if infection is suspected

CT scan

(abdomen and pelvis)—may be helpful in some cases; usually done if renal ultrasound shows an abnormality such as hydronephrosis

Renal biopsy

biopsy—useful occasionally if there is suspicion of acute GN or acute allergic interstitial nephritis

Renal arteriography

evaluate for possible renal artery occlusion; should be performed only if specific therapy will make a difference

Renal ultrasound

- Useful for evaluating kidney size and **for excluding urinary tract obstruction (i.e., postrenal failure)**—presence of bilateral hydronephrosis or hydroureter
- Order for most patients with AKI**—unless the cause of the AKI is obvious and is not postrenal

Diagnosis

Urinalysis

- A dipstick test positive for protein (3+, 4+) suggests intrinsic renal failure due to glomerular insult.
- Microscopic examination of the urine sediment is very helpful.
 - **Hyaline casts** are devoid of contents (seen in prerenal failure).
 - **Muddy brown casts** leading to muddy brown urine indicates ATN.
 - **RBC casts** indicate AGN.
 - **WBC casts** indicate AIN.
 - **Fatty casts** indicate nephrotic syndrome.

According to Doctor's slides:
urinalysis is unremarkable in pre and post renal causes

Urine chemistry

- distinguish between different forms of AKI
- Urine Na⁺, Cr, and osmolality: **Urine Na⁺ depends on dietary intake.**
 - FENa: collect urine and plasma electrolytes simultaneously = $[(\text{UNa})/(\text{PNa})/(\text{UCr})/(\text{PCr}) \times 100]$, where U = urine and P = plasma.
 - Values below 1% suggest prerenal failure.
 - Values above 2% to 3% suggest ATN.
 - **FENa is most useful if oliguria is present.**
 - Renal failure index 5 $(\text{uNa}/[\text{uCr}/\text{pCr}]) \times 100$
 - Values below 1% suggest prerenal failure.
 - Values above 1% suggest ATN.

Diagnosis

Prerenal failure versus ATN

	Prerenal Failure	ATN
Urine osmolarity	>500	>350
Urine Na ⁺	<20	>40
FENa	<1%	>1%
Urine sediment	Scant	Full brownish pigment, granular casts with epithelial casts

TABLE 7-2 Urinalysis Findings in Renal Failure

Cause	Urine Sediment	Protein	Blood
Prerenal	Benign sediment—few hyaline casts	Negative	Negative
Intrarenal			
Acute tubular necrosis	“Muddy brown” casts, renal tubular cells/casts, granular casts	Trace	Negative
Acute glomerulonephritis	Dysmorphic RBCs, RBCs with casts, WBCs with casts, fatty casts	4+	3+
Acute interstitial nephritis	RBCs, WBCs, WBCs with casts, eosinophils	1+	2+
Postrenal	Benign; may or may not see RBCs, WBCs	Negative	Negative

Complications

1/ECF volume expansion and resulting pulmonary edema— treat with a diuretic (furosemide)

2/Metabolic :

a. Hyperkalemia— due to decreased excretion of K^+ and the movement of potassium from ICF to ECF due to tissue destruction and acidosis

b. Metabolic acidosis (with increased anion gap)—due to decreased excretion of hydrogen ions; if severe (below 16 mEq/L), correct with sodium bicarbonate

c. Hypocalcemia— loss of ability to form active vitamin D and rapid development of PTH resistance

d. Hyponatremia may occur if water intake is greater than body losses, or if a volume-depleted patient consumes excessive hypotonic solutions. (Hypernatremia may also be seen in hypovolemic states.)

e. Hyperphosphatemia

f. Hyperuricemia

Complications

3/Uremia—toxic end products of metabolism accumulate (especially from protein metabolism).

4/Infection:

- ✓ A common and serious complication of AKI (occurs in 50% to 60% of cases).
- ✓ The cause is probably multifactorial, but uremia itself is thought to impair immune functions, Examples include pneumonia, UTI, wound infection, and sepsis.

Prognosis factor

<p><u>Severity of renal failure</u></p>	<p>Magnitude of increase in Cr Presence of oliguria Fractional excretion of sodium Requirement for dialysis Duration of severe renal failure Marked abnormalities on urinalysis</p>
<p><u>Underlying health of patient</u></p>	<p>Age Presence, severity, and reversibility of underlying disease</p>
<p><u>Clinical circumstances</u></p>	<p>Cause of renal failure Severity and reversibility of acute process(es) Number and type of other failed organ systems Development of sepsis and other complications</p>

Management

Obtain the following in any patient with AKI:

- Urinalysis
- Urine chemistry
- Serum electrolytes (Na⁺, K⁺, BUN, Cr), CBC
- Bladder catheterization to rule out obstruction (diagnostic and therapeutic)
- Renal ultrasound to look for obstruction

Treatment

1. General measures:

- a. Avoid medications that decrease renal blood flow (NSAIDs) and/or that are nephrotoxic (e.g., aminoglycosides, radiocontrast agents).
- b. Adjust medication dosages for level of renal function.
- c. Correct fluid imbalance.
 - If the patient is volume depleted, give IV fluids. However, many patients with AKI are volume overloaded (especially if they are oliguric or anuric), so diuresis may be necessary.
 - The goal is to strike a balance between correcting volume deficits and avoiding volume overload (while maintaining adequate urine output).
 - Monitor fluid balance by daily weight measurements (most accurate estimate) and intake–output records.
 - Be sure to take into account the patient’s cardiac history when considering treatment options for fluid imbalances (i.e., do not give excessive fluid to a patient with CHF)
 - Balanced salt solution, such as Hartmann’s or Ringer’s lactate, may be preferable to isotonic (0.9%) saline when large volume of fluid resuscitation are required.

Treatment

- d. Correct electrolyte disturbances if present.
- e. Optimize cardiac output. BP should be approximately 120 to 140/80 to 90.
- f. Order dialysis if symptomatic uremia, intractable acidemia, hyperkalemia, or volume overload develop.
- g. Pulmonary edema may result from the administration of excess amounts of fluids. If pulmonary edema is present and urine output cannot be rapidly restored, treatment with dialysis may be required to remove excess fluid.
- h. Critically ill patients may require inotropic drugs to restore an effective blood pressure but clinical trials do not support a specific role for low-dose dopamine.

Treatment

2. Prerenal:

- a. Treat the underlying disorder.
- b. Give NS to maintain euvolemia and restore blood pressure—do not give to patients with edema or ascites.
Stopping antihypertensive medications may be necessary.
- c. Eliminate any offending agents (ACE inhibitors, NSAIDs).
- d. If the patient is unstable, Swan–Ganz monitoring is indicated for accurate assessment of intravascular volume.

3. Intrinsic:

- a. Once ATN develops, therapy is supportive. Eliminate the cause/offending agent.
- b. If the patient is oliguric, a trial of furosemide may help to increase urine flow.
This improves fluid balance.

4. Postrenal

a bladder catheter may be inserted to decompress the urinary tract.
Consider urology consultation

Renal replacement therapy

- Renal replacement therapy (RRT) may be required in patients who are not showing signs of recovery. Typically, the decision to start RRT is driven by hyperkalemia, fluid over load or acidosis. The decision to institute RRT should be made on an individual basis, taking account of the potential risks and benefits, comorbidity and other aspects of the patient care. The two main options for RRT in AKI are hemodialysis and high-volume hemofiltration.

Contrast nephropathy

- 12-24 hours post exposure, peaks in 3-5 days
- Non-oliguric, FE Na <1% !!
- RX/Prevention:
 - ✓ 1/2 NS 1 cc/kg/hr 12 hours pre/post
 - ✓ N-acetylcysteine 600 BID pre/post (4 doses)
- Risk Factors:
 - ✓ CKD,
 - ✓ Older age
 - ✓ Hypovolemia, DM, CHF

Atheroembolic ARF

- Associated with emboli of fragments of atherosclerotic plaque from aorta and other large arteries
- Diagnose by history, physical findings (evidence of other embolic phenomena--CVA, ischemic digits, “blue toe” syndrome, etc), low serum C3 and C4, peripheral eosinophilia, eosinophiluria, rarely WBC casts
- Commonly occur after intravascular procedures or cannulation (cardiac cath, CABG, AAA repair, etc.)

Quick Hit

Obtain the following in any patient with AKI

- Urinalysis
- Urine chemistry
- Serum electrolytes (Na⁺, K⁺, BUN, Cr), CBC
- Bladder catheterization to rule out obstruction (diagnostic and therapeutic)
- Renal ultrasound to look for Obstruction

Diagnosis of AKI is usually made by finding elevated BUN and Cr levels. The patient is usually asymptomatic.

Three basic tests for postrenal failure

- Physical examination—palpate the bladder
- Ultrasound—look for obstruction, hydronephrosis
- Catheter—look for large volume of urine

Note that prerenal azotemia and ischemic AKI are part of a spectrum of manifestations of renal hypoperfusion. The latter differs in that injury to renal tubular cells occurs.

Quick Hit

Monitoring a patient with AKI

- Daily weights, intake, and output
- BP
- Serum electrolytes
- Watch Hb and Hct for anemia
- Watch for infection

- In evaluating a patient with
- AKI, first exclude prerenal
- and postrenal causes, and
- then, if necessary, investigate
- intrinsic renal causes

Radiographic contrast media can cause ATN (typically very rapidly) by causing spasm of the afferent arteriole. It can be prevented with saline hydration.

In the early phase of AKI, the most common mortal complications are **hyperkalemic cardiac arrest** and **pulmonary edema**.

MCQs

1/ A 73-year-old man undergoes abdominal aortic aneurysm repair. The patient develops hypotension to 80/50 for approximately 20 minutes during the procedure according to the anesthesia record. He received 4 units of packed red blood cells. Postoperatively his blood pressure is 110/70, heart rate is 110, surgical wound is clean, and a Foley catheter is in place. Over the next 2 days his urine output slowly decreases. His creatinine on post-operative day 3 is 3.5 mg/dL (baseline 1.2). His sodium is 140 mEq/L, K 4.6 mEq/L and BUN 50 mg/dL. Hemoglobin and hematocrit are stable. Urinalysis shows occasional granular casts but otherwise is normal. Urine sodium is 50 mEq/L, urine osmolality is 290 mosmol/L, and urine creatinine is 35 mg/dL. The FeNa (fractional excretion of sodium) based on these data is 35. What is the most likely cause of this acute renal failure?

- A) Acute interstitial nephritis
- B) Acute glomerulonephritis
- C) Acute tubular necrosis
- D) Pre-renal azotemia
- E) Contrast induced nephropathy

2/ A 47 year-old HIV-positive man is brought to the emergency room because of weakness. The patient has HIV nephropathy and adrenal insufficiency because. He takes trimethoprim-sulfamethoxazole for PCP prophylaxis and is on triple agent antiretroviral treatment. He was recently started on spironolactone for ascites due to alcoholic liver disease. Physical examination reveals normal vital signs, but his muscles are diffusely weak. Frequent extrasystoles are noted. He has mild ascites and 1+ peripheral edema. Laboratory studies show a serum creatinine of 2.5 with a potassium value of 7.3 mEq/L. An EKG shows peaking of the T waves and QRS duration of 0.14. What is the most important immediate treatment?

- A) Sodium polystyrene sulfonate (Kayexalate)
- B) Acute hemodialysis
- C) IV normal saline
- D) IV calcium gluconate
- E) IV furosemide 80 mg stat

MCQs

3/ An 85 year-old man who resides in a nursing home 3 days history of lower abdominal pain and unceasing fatigue and lethargy. He is afebrile, his BP is 160/92, and RR 16. His lungs are clear and his heart examination normal. There is diffuse abdominal tenderness on palpitation and a large area of fullness and dullness to percussion starting just below the umbilicus and extending to the suprapubic area. His serum sodium is 130 mEq/L, potassium 4.9 mEq/L, BUN 75 mg/dL, and creatinine is 3.5 mg/dL. His baseline BUN and creatinine were 25 and 1.3 respectively as recently as 1 month ago. A Foley catheter is placed and 1200 cc of urine is obtained.

What will be the likely clinical course for this patient with regard to his renal function?

- A) His creatinine will continue to rise slowly for 2 to 3 more days
- B) His creatinine will return to 1.3 over the next week.
- C) He will require dialysis within 24 hours
- D) He will produce minimal urinary output for at least 3 days.
- E) His renal function is unlikely to show any improvement in the futur and 3.5 will be his new baseline.

4/ A 63 year old man alcoholic with a 50-pack-year history of smoking presents to the emergency room with fatigue and confusion. Physical examination reveals a blood pressure of 110/70 with no orthostatic change. Heart, lungs and abdominal examinations are normal and there is no pedal edema. Laboratory data are as follows :

Na : 110 mEq/L, K :37 mEq/L

Cl: 82 mEq/L, HCO₃ : 20 mEq/L ,Glucose: 100 mg/dL, BUN : 5 mg/dL

Creatinine: 0.7 mg/dL

Urinalysis: normal ,Specific gravity: 1.016

Which of the following is the most likely diagnosis?

- A) Volume depletion
- B) Inappropriate secretion of antidiuretic hormone
- C) Psychogenic polydipsia
- D) Cirrhosis
- E) Congestive heart failure

MCQs

5/ A 50 year old diabetic woman presents for follow-up of her hypertension. Her blood pressure is 152/96 in the office today and she brings in that consistently in the same range over the past month. Her current medications are amlodipine 5 mg daily and hydrochlorothiazide 25 mg daily. The diuretic was added when she developed peripheral edema on the amlodipine ; now she has only trace peripheral edema. A spot urine specimen shows 280 mg of albumin per mg creatinine (microalbuminuria is present if this value is between 30 and 300 mg/mg)
What would be the best next therapeutic step in this patient ?

- A) Add clonidine.
- B) Add a beta-blocker.
- C) Increase the thiazide diuretic dose.
- D) Add an alpha-blocker.
- E) Add angiotensin-converting enzyme inhibitor or angiotensin receptor blocker.

6/ which of the following renal disease will cause severe decrease in urine output "anuria" ?

- A) BPH
- B) Unilateral kidney stone
- C) Tumor in one ureter
- D) Rhabdomyolysis

7/What is the most common diagnostic test used for hydronephrosis ?

- A) CT
- B) X-Ray
- C) MRI
- D) Ultrasound

! Answers : 1-C 2-D 3-B 4-B 5-E 6-A 7-D



Medicine433



Medicine433

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*Medicine is a science of uncertainty
and an art of probability*



MEDICINE 433