Q.1. A 57-year-old man with insulin-requiring diabetes presents with a three-day history of polyuria and polydipsia after having run out of insulin. His examination is most notable for tachypnea and orthostasis. Data are as follows:

ABG (pH/pCO₂/pO₂) 7.20/18/88

Electrolytes
- Na⁺ 125
- K⁺ 6.8
- Cl⁻ 97
- HCO₃⁻ 6
- BUN 30
- Cr 1.9
- Glucose 788

Which one of the following statements is true?

A. The patient has a single acid-base disorder: An anion gap metabolic acidosis caused by diabetic ketoacidosis
B. The patient has a single acid-base disorder: A respiratory alkalosis caused by hyperventilation
C. The patient has two disorders: An anion gap metabolic acidosis and a normal anion gap metabolic acidosis
D. The patient has two disorders: An anion gap metabolic acidosis and a respiratory alkalosis
E. The patient has a pure metabolic alkalosis from dehydration

**Answer: C.** This patient’s arterial pH is low; this defines an acidosis. The low pCO₂ does not suggest a respiratory acidosis, but the low bicarbonate of 6 does. Therefore the patient has a metabolic acidosis. The differential is then narrowed by calculating the anion gap. The anion gap (125 [97 6] = 22) is elevated. The mistake would be to stop there and summarize the patient as having an anion gap acidosis without
thinking further about the acid-base disorder. Using the delta-delta equation (see Chapter 32), we note the bicarbonate has changed from 24 to 6, or by 18. The anion gap has changed from a normal of 12 to 22, or by 10. The bicarbonate has therefore changed significantly more than the anion gap, so a normal anion gap process must be accompanying the anion gap acidosis. It is useful to make a habit of calculating the delta-delta equation every time you calculate the anion gap. This patient is a classic example of someone presenting with diabetic ketoacidosis (creating an anion gap acidosis) and a renal tubular acidosis (type 4), creating a normal anion gap acidosis.

Q.2. A 57-year-old woman with a history of hypertension comes in to the emergency room complaining of lightheadedness. She notes this comes on when she stands up, but she has not passed out. Past history is notable only for hypertension, and her physician increased her dose of medication two weeks ago. She does not recall the name of the medication. Data are as follows:

\[\text{ABG (pH/pCO}_2/\text{pO}_2) \quad 7.48/46/92\]

**Electrolytes**

<table>
<thead>
<tr>
<th>Ion</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na(^+)</td>
<td>129</td>
</tr>
<tr>
<td>K(^+)</td>
<td>3.1</td>
</tr>
<tr>
<td>Cl(^-)</td>
<td>93</td>
</tr>
<tr>
<td>HCO(_3)(^-)</td>
<td>32</td>
</tr>
<tr>
<td>BUN</td>
<td>29</td>
</tr>
<tr>
<td>Cr</td>
<td>1.9</td>
</tr>
</tbody>
</table>

Which one of the following statements is true?

A. The patient has a metabolic acidosis with a normal anion gap

B. The patient likely has a type 4 renal tubular acidosis, explaining the acid-base disorder

C. The patient has a respiratory acidosis, likely from altered mental status

D. The ideal treatment for this patient would be to discontinue the unknown medication and administer saline

E. The patient likely has a metabolic alkalosis from adrenal insufficiency
**Answer: D.** The patient’s pH is elevated, so she has an alkalosis. Her high pCO₂ would not explain this but would rather suggest a respiratory acidosis. However, her high serum bicarbonate is consistent with the elevated serum pH, thus suggesting a metabolic alkalosis. Her history is suggestive of dehydration, with orthostatic dizziness, hyponatremia, hypokalemia, and hypochloremia. To confirm, the urine chloride can be checked, and should be quite low (i.e., <10). It is difficult to precisely assess the respiratory compensation for a metabolic alkalosis, but in general, for every unit increase in serum bicarbonate above 24, the pCO₂ should increase 0.5 to 1. Here, the bicarbonate has increased by 8, so pCO₂ should increase 4 to 8 above 40, or 44 to 48. Therefore, the measured pCO₂ of 46 represents appropriate respiratory compensation. Treatment should be to discontinue the diuretic and administer normal saline to resolve dehydration. Her metabolic alkalosis should resolve as well.

**Q.3.** Which one of the following statements about acid-base disorders is true?

A. A patient with a metabolic acidosis will normally respond by increasing lung ventilation, raising the serum pCO₂ level

B. A patient with a metabolic alkalosis and low urine chloride may have hyperaldosteronism as an explanation

C. Patients who have a pneumothorax or other process (e.g., fluid in the pleural space) that prevents expansion of the lung typically present with a respiratory acidosis

D. Sepsis is associated with both a metabolic acidosis and a respiratory acidosis

**Answer: C.** When a patient develops a metabolic acidosis he or she compensates by increasing ventilation, resulting in a compensatory respiratory alkalosis and a decrease in the serum pCO₂ level (not an increase, as in Answer A). Nasogastric suction, vomiting, and over-diuresis commonly cause a metabolic alkalosis in the setting of a low urine chloride. Conn’s syndrome (hyperaldosteronism) does result in a metabolic alkalosis, but urine chloride is normal. Disorders that cause a respiratory acidosis include neuromuscular disorders, pneumothorax, and pleural effusions, which prevent proper ventilation of the affected lung (making Answer C correct).
Q.4. A 56-year-old woman presents for a new patient evaluation. Past medical history is notable for hypertension, diagnosed one year ago, which is treated with atenolol. She brings outside blood work, which shows the following:

ABG (pH/pCO2/pO2) 7.46/43/99

Electrolytes
- Na\(^+\) 144
- K\(^+\) 3.0
- Cl 108
- HCO\(_3\) 30
- BUN 18
- Cr 1.2
- Glucose 108

The best description of her metabolic findings and differential diagnosis is:

A. Respiratory acidosis with metabolic compensation; rule out CNS tumor
B. Respiratory alkalosis with metabolic compensation; rule out cirrhosis
C. Metabolic acidosis with respiratory compensation; rule out renal tubular acidosis
D. Metabolic alkalosis with respiratory compensation; rule out hyperaldosteronism
E. Metabolic acidosis and respiratory acidosis; rule out Cushing’s disease

**Answer: D.** The serum pH of 7.46 is consistent with alkalosis; this is explained by an elevated serum bicarbonate of 30. Therefore, the patient has a metabolic alkalosis. Compensation is via hypoventilation; for every 1 unit that the serum bicarbonate increases, the pCO\(_2\) increases by 0.5 to 1. Thus, the increase of serum bicarbonate of 6 (from baseline of 24 to 30) should be accompanied by an increase in the pCO\(_2\) of 3 to 6, from a baseline of 40. Her pCO\(_2\) of 43 is therefore consistent with appropriate respiratory compensation of her initial metabolic alkalosis. Her diagnosis of new onset hypertension after the age of 50, along with unexplained hypokalemia are suggestive of Conn’s syndrome, or hyperaldosteronism, making choice D correct.

Q.5. A 47-year-old woman with lupus presents for routine follow up. She has no complaints; her last lupus flare was over six months ago. Current medications
include hydroxychloroquine and ibuprofen. Physical exam is normal. Urinalysis shows pH 5.8 and calcium oxalate crystals. A basic metabolic panel shows the following:

ABG (pH/pCO2/pO2) 7.33/33/96

Electrolytes

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>138</td>
</tr>
<tr>
<td>K⁺</td>
<td>3.0</td>
</tr>
<tr>
<td>Cl</td>
<td>110</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>16</td>
</tr>
<tr>
<td>BUN</td>
<td>12</td>
</tr>
<tr>
<td>Cr</td>
<td>0.8</td>
</tr>
<tr>
<td>Glucose</td>
<td>normal</td>
</tr>
</tbody>
</table>

The best explanation for this patient’s metabolic disorder is

A. Type 1 renal tubular acidosis
B. Type 2 renal tubular acidosis
C. Type 3 renal tubular acidosis
D. Type 4 renal tubular acidosis

**Answer: A.** This patient’s serum pH is 7.33, consistent with acidosis. The serum bicarbonate of 16 confirms a metabolic acidosis. The anion gap is 12 (138 – [110 + 16]), which is normal. The patient therefore has a normal anion gap metabolic acidosis (often referred to as a non–anion gap metabolic acidosis). Renal tubular acidosis is a cause of normal anion gap metabolic acidosis. Lupus is a cause of type 1 RTA, which is typically associated with hypokalemia and oxalate crystals in the urine, confirming answer A as the correct choice.

**Q.6.** A 53-year-old male presents to the emergency department with coffee ground emesis. He awoke this morning feeling nauseated, and vomited dark material that resembled coffee grounds. He had noted melena the day before. Past history is notable for chronic alcoholism. He takes no prescription or over-the-counter medications. Physical exam is notable for no orthostasis. Parotid and lacrimal gland enlargement is noted. There is no lymphadenopathy. Lungs are clear, and cardiac exam is unremarkable. Abdomen is distended, with a positive fluid wave. Multiple
telangiectasias are noted on skin exam. The metabolic disturbance most likely to be noted on laboratory examination is

A. Metabolic acidosis
B. Metabolic alkalosis
C. Respiratory acidosis
D. Respiratory alkalosis
E. Metabolic acidosis and respiratory acidosis

**Answer: D.** This patient has the clinical sequelae of chronic alcohol use and cirrhosis. He may have esophageal varices, resulting in the gastrointestinal bleeding described. He also has ascites on physical exam. Cirrhosis is a cause of respiratory alkalosis.

**Q.7.** Which of the following clinical or laboratory findings is most consistent with type 4 renal tubular acidosis (RTA)?

A. Hypokalemia
B. Proximal renal tubular defect in bicarbonate resorption
C. May result from treatment with lisinopril
D. Urine pH remains above 5.5 with acid challenge
E. Renal glucose and amino acid resorption is impaired

**Answer: C.** The pathogenic mechanism that results in type 4 RTA is hyporeninemic hypoaldosteronism. A diffuse renal tubular defect in NH4 excretion results, along with impaired potassium excretion. A normal anion gap metabolic acidosis, with hyperkalemia, results. Type 4 RTA may result from certain medications, including ACE inhibitors (as noted in the correct answer, lisinopril), NSAIDs, or heparin. The most common cause of type 4 RTA, however, is diabetes. There is no impairment of renal glucose or amino acid resorption (resorption of these are impaired in type 2 RTA), and urine pH will acidify below 5.5 with acid challenge (which is not seen with type 1 RTA).
Q.1. Which of the following statements is/are true about antidiuretic hormone (ADH)?
A. It is the principal hormone regulating water balance
B. Its release is stimulated by increases in serum osmolality
C. Moderate to severe volume loss will override osmolar changes to stimulate ADH release
D. ADH acts on the collecting ducts of the kidney to make them permeable to water
E. All of the above are correct

Answer: E. Antidiuretic hormone's principal action is to make the collecting ducts of the kidney permeable to water, leading to water resorption. It makes intuitive sense that ADH release is stimulated by hypovolemia. ADH release is also stimulated by increases in osmolality, which prevents wide swings in serum osmolality. If osmolality is normal but volume is low, ADH will be released because hypovolemia overrides osmolality in the stimulation of ADH release.

Q.2. After seeing your patients in the clinic you get a stat call from the lab that one of the patient’s potassium was elevated at 7.2 mEq/L, and that no hemolysis in the tube was noted. If an EKG were to be done on this patient, which abnormality would be most likely?
A. PR segment depression
B. U waves
C. Peaked T waves
D. Asymmetric T wave inversions
E. Complete heart block

Answer: C. Peaked T waves are seen in hyperkalemia, although EKG changes may be absent in hyperkalemia. PR segment depression may indicate pericarditis. U waves are described in hypokalemia. Asymmetric T wave inversions may represent digitalis toxicity of left ventricular strain. Complete heart block may be seen in hypokalemia, but is not commonly seen with hyperkalemia.

Q.3. A 23-year-old woman with type 1 diabetes presents to the emergency room with abdominal pain, lethargy, polydipsia, and polyuria. She tells you that she ran out of insulin three days ago. Labs reveal a glucose of 755, creatinine of 4.0, and potassium of 7.1. In addition, her hematocrit is 43.5 and WBC count is 11,000. Which of the following statements is most accurate?
A. A reasonable first-line treatment for her hyperkalemia would be a sodium polystyrene sulfonate resin
B. No ECG is needed because her potassium will improve once insulin is administered
C. Total body potassium is probably low despite high serum levels
D. She probably has a pseudohyperkalemia because of her high WBC count

**Answer: C.** The lack of insulin in this patient (as well as the acidosis) results in shifts of potassium from the intracellular to extracellular space, where it is then excreted by the kidney. Therefore, this patient probably is total-body-potassium depleted despite her high serum levels. A sodium polystyrene resin is a reasonable treatment for chronic (not acute) hyperkalemia. Although her hyperkalemia should improve rapidly with insulin administration and correction of her acidosis, one should always perform an ECG. The high serum potassium may lead to changes that predict a lethal arrhythmia. Finally, her WBC count is not high enough (i.e., greater than 70,000) to produce pseudohyperkalemia.

**Q.4.** A patient presents with a five-day history of nausea and vomiting after eating crabs. He appears ill and is orthostatic. Serum sodium is 129 mg/dL. Which of the following statements is true?
A. Urine Na will be less than 20 mEq/L
B. Treatment should be with D5W
C. Antidiuretic hormone (ADH) levels will be low
D. Serum osmolality is likely to be high

**Answer: A.** This patient has developed hypovolemia from fluid losses from vomiting and diarrhea. There is no evidence that other osmotically active compounds have been ingested, thus serum osmolality is likely to be low. Because of hypovolemia, ADH (and aldosterone) will be stimulated, and serum ADH levels will be high. The kidney will be resorbing water and sodium, and urine sodium concentrations will be low (less than 20 mEq/L). Intravascular volume repletion with normal saline is advisable.

**Q.5.** A 57-year-old man with diabetes and previously normal renal function comes to the clinic complaining of weakness and malaise. Three weeks ago he was started on lisinopril. Examination is unremarkable. BUN is 55 and creatinine is 2.8. Potassium is 6.9. Which one of the following is true?
A. Diabetics are particularly prone to the development of hyperkalemia
B. ECG will show prominent U waves
C. A high-potassium diet is a common cause of hyperkalemia
D. All of the above are correct

**Answer: A.** This susceptibility is in part caused by the high prevalence of type 4 renal tubular acidosis (which results in hyperkalemia) in people with diabetes. ECG findings include peaked T waves, followed by QRS widening until it fuses with the T wave (resembling a sine wave). U waves are usually seen with hypokalemia. The kidney and gastrointestinal tract will increase potassium excretion in an individual with high potassium intake, and it is difficult to induce hyperkalemia by diet in an individual with normal renal function.

**Q.6.** A 78-year-old woman comes in for evaluation. She was started on hydrochlorothiazide two weeks ago for hypertension. She now complains of malaise and appears confused. Blood pressure is 128/76. She is not orthostatic. Serum sodium is 124 mg/dL. Which of the following statements is true?
A. Urine Na will be less than 20 mEq/L
B. ADH levels will be high
C. Total body water will be decreased
D. Normal saline will correct the problem

**Answer: B.** This patient has a common presentation for an elderly person placed on a thiazide diuretic. Initial response to the diuretic results in hypovolemia, and ADH secretion is activated. Water resorption occurs, but ADH release is not shut off (for unclear reasons) despite the correction in volume. Sodium is diluted by water resorption and hyponatremia develops. In the absence of hyperkalemia, hypovolema, or ACTH secretion, aldosterone release will not be stimulated, and urine sodium will not be low (i.e., <20 mEq/L). Because of the increased total body water, plasma volume is high and normal saline should not be used in treatment. The thiazide diuretic should be discontinued.

**Q.7.** A 43-year-old man with familial hypertriglyceridermia treated with gemfibrozil presents for routine follow-up. Past medical history is also notable for hypertension, treated with HCTZ. Physical examination, including blood pressure, is normal. He brings outside labs, done last week for health insurance. Labs were all normal except for a sodium level of 129 mg/dL and serum triglycerides of 1336 mg/dL. The most likely explanation of his sodium level is
A. Administration of HCTZ
B. Administration of gemfibrozil
C. Elevated triglycerides
D. Hypertension
E. None of the above

**Answer: C.** This patient most likely has pseudohyponatremia, which is artifactual decrease in reported sodium due to the presence of elevated triglycerides. Hyperglycemia and elevated protein may also result in pseudohyponatremia. HCTZ may cause hyponatremia by many mechanisms, including volume depletion (unlikely in this patient who was not orthostatic) and nonosmotic ADH release, which is commonly seen in more elderly individuals, especially women. Gemfibrozil does not commonly cause electrolyte disturbances. Hypertension itself does not cause hyponatremia.

**Q.8.** Following are four causes of hypokalemia. Which one of the five items listed does not cause hypokalemia?

A. Glucocorticoid excess
B. Acidosis
C. Laxative abuse
D. Beta-adrenergic stimulation
E. Osmotic diuresis

**Answer: B.** Hypokalemia may result from renal losses, gastrointestinal losses, or intracellular redistribution of potassium. Renal losses that result in hypokalemia include potassium-wasting diuretics, osmotic diuresis, glucocorticoid excess, mineralocorticoid excess, and leukemia. Gastrointestinal losses that result in hypokalemia include diarrhea, vomiting, nasogastric suction, and laxative abuse. Redistribution of potassium may occur, resulting in hypokalemia, in patients who have been administered insulin or glucose, in patients treated with beta-adrenergic stimulation, and in patients with alkalosis. Acidosis, on the other hand, causes hyperkalemia, making answer B the choice that does not result in hypokalemia.

**Q.9.** You are evaluating a 59-year-old man with essential thrombocytosis. He comes in today feeling well, and has no complaints. Past medical history is otherwise notable for hypertension. Medications include lisinopril, hydrochlorothiazide, and aspirin. Physical exam is normal; blood pressure is well controlled. You obtain a complete blood count and basic metabolic panel, which show a normal hematocrit and white blood count; platelet count is unchanged from three months ago at 1,150,000. Basic metabolic panel shows Na 144, K 7.2, Cl 106, CO₂ 21, BUN 17, Cr 1.1, and glucose 109. Which one of the following statements is true?
A. The EKG will show peaked T waves
B. Lisinopril should be discontinued
C. Hyperreflexia will be found on neurologic testing
D. Insulin and glucose should be administered immediately
E. More information is needed

Answer: E. When evaluating a patient with hyperkalemia the initial step is to exclude the possibility of artifactually elevated potassium ("pseudohyperkalemia"). Pseudohyperkalemia may occur when hemolysis occurs while obtaining or storing the blood specimen, and is also seen when either the WBC or platelet count is high (as seen in our patient). Therefore, more information is needed to exclude pseudohyperkalemia with this patient, by repeating potassium determination in different collection tubes. If hyperkalemia were verified, the EKG would be likely to show peaked T waves. Lisinopril could be the culprit cause of hyperkalemia. Hyporeflexia (not hyperreflexia) would be seen on physical exam. One treatment option for the acute management of hyperkalemia is the administration of insulin and glucose (dextrose), which will drive potassium intracellularly.

Q.10. When considering antidiuretic hormone and aldosterone, which one of the following statements is true?
A. Antidiuretic hormone release is stimulated by hyperkalemia
B. Aldosterone and antidiuretic hormone release are stimulated by hyponatremia
C. Aldosterone stimulates renal resorption of potassium and excretion of sodium
D. Release of antidiuretic hormone results in increases in serum osmolality
E. Release of aldosterone results in increases in serum osmolality

Answer: E. The two major hormones of sodium and water balance are antidiuretic hormone and aldosterone. Antidiuretic hormone release is stimulated by increases in serum osmolality or decreases in volume, and will result in water resorption and decreases in serum osmolality. Hyperkalemia, ACTH, and activation of the renin-angiotensin system stimulate aldosterone release. Neither aldosterone nor antidiuretic hormone release is stimulated by hyponatremia. Aldosterone stimulates renal potassium excretion and sodium resorption, which increases serum osmolality.

CHAPTER 34: ACUTE RENAL FAILURE
Q.1. Which of the following statements about acute tubular necrosis (ATN) is true?
A. ATN most commonly occurs in outpatients, usually when started on an NSAID
B. RBC casts are present if injury is severe
C. Sodium resorption is reduced, leading to a high urine fractional excretion of sodium
D. Urine is concentrated, usually with osmolality greater than 500 mOsm/kg
E. All of the above are true

Answer: C. Acute tubular necrosis, which is the most common cause of acute renal failure in the hospitalized patient (not in outpatients, as suggested in A), is a result of tubular injury. Tubular injury may result from ischemia (e.g., in volume depletion) or from a nephrotoxin (e.g., contrast dye). Sloughing of tubules result in the classic “muddy brown” granular casts (not RBC casts), and sodium absorption is reduced (as correctly noted in C). The kidneys are not able to concentrate urine, and urine osmolality is typically low (<450 mOsm/kg).

Q.2. Which of the following causes of acute renal failure is/are commonly associated with a fractional excretion of sodium (FENa) of less than 1%?
1. Obstructive uropathy from nephrolithiasis
2. Hepatorenal syndrome
3. Acute renal failure (ARF) from congestive heart failure
4. Interstitial nephritis
5. Pigment nephropathy
A. All of the above
B. None of the above
C. 1, 2, and 3
D. 2 and 3
E. 4 and 5

Answer: D. Hepatorenal syndrome and all prerenal causes of ARF are associated with a FENa of less than 1%. Postrenal causes, such as obstructive uropathy, are associated with a FENa greater than 2%, as are most intrarenal causes of ARF (including interstitial nephritis and pigment nephropathy). Hepatorenal syndrome, also a prerenal cause of ARF, is associated with a FENa of less than 1%.

Q.3. An 80-year-old woman with type I DM and severe cardiomyopathy (EF 15%) is admitted with CHF and leg edema. Her serum creatinine on admission is 1.3 mg/dL. She is started on furosemide and an ACE inhibitor. She responds poorly to initial diuretic regimen. Despite increasing furosemide and adding metolazone, urine output is poor and serum creatinine begins to rise steadily, up to 2.0 mg/dL.
Urinalysis shows 1+ proteinuria. Renal ultrasound shows normal sized kidneys without hydronephrosis or masses. Spot urine Na⁺ is 10 mEq/L. The most likely diagnosis of the worsening creatinine is
A. Congestive heart failure
B. Diabetic nephropathy
C. Interstitial nephritis
D. ATN
E. Bilateral renal artery stenosis

Answer: A. The most likely diagnosis is congestive heart failure. Because of low EF and poor cardiac output, renal function continues to decline. Aggressive use of diuretics and ACE inhibitors in this clinical situation may exacerbate renal failure. Even though the patient may have early stage diabetic nephropathy, it is not the cause of acute renal failure seen in the hospital setting. Normal-sized kidneys seen on ultrasound make hemodynamically significant renal artery stenosis unlikely.

Q.4. A 48-year-old man with a history of cirrhosis due to hepatitis C is admitted due to worsening abdominal girth, bilateral leg edema, and dyspnea. Initial serum creatinine is 1.5 mg/dL. Despite starting furosemide, urine output begins to decline over the next several days and creatinine increases to 6.0 mg/dL. Urinalysis shows no evidence of proteinuria or hematuria. Urine Na⁺ is less than 5 mEq/L. Furosemide is discontinued on day 3, and the patient is given 3 liters of normal saline and intravenous albumin over 24 hours without much improvement. The most likely diagnosis is
A. Hepatitis C induced membranoproliferative glomerulonephritis
B. Diuretic induced prerenal azotemia
C. Acute tubular necrosis from sepsis
D. Hepatorenal syndrome

Answer: D. The most likely diagnosis is hepatorenal syndrome. Even though prerenal azotemia is possible, a lack of response to volume expansion makes prerenal azotemia less likely. He has no evidence of ongoing glomerulonephritis, as shown by the absence of proteinuria, hematuria, or casts in the urinalysis. Were the patient to have acute tubular necrosis, urine sodium should be higher than seen in this patient.

Q.5. In differentiating acute renal failure due to acute tubular necrosis (ATN) from acute renal failure due to prerenal causes, which of the following statements is true?
A. In ATN, urine sodium resorption is normal, while in prerenal causes urine sodium resorption is absent.
B. In both ATN and prerenal causes of acute renal failure, the BUN/Cr ratio is usually greater than 20 to 1.
C. Cirrhosis may result in prerenal acute renal failure, while it is not a cause of ATN.
D. All of the above are true.

**Answer: C.** In acute tubular necrosis, renal tubules are damaged by either ischemia or nephrotoxins. ATN is the most common cause of acute renal failure. The damaged tubules are unable to resorb sodium, leading to high urine sodium and a fractional excretion of sodium (FENa) of greater than 2%. The BUN/Cr ratio stays close to normal; in ATN, the BUN/Cr ratio typically does not exceed 20 to 1. In prerenal causes of acute renal failure, sodium resorption is preserved, and is in fact maximized (leading to low urine sodium and FENa less than 1%). The BUN/Cr ratio is typically high, and classically is greater than 20 to 1. Prerenal causes of acute renal failure include intravascular volume depletion (e.g., blood loss, dehydration) or decreased effective circulating volume (e.g., congestive heart failure, cirrhosis). Only choice C provides an accurate description of both ATN and prerenal causes of acute renal failure.

**Q.6.** A 49-year-old man presents to the emergency room with generalized malaise and extreme fatigue. He states symptoms began several weeks ago, and that they have progressed to the point where he is fatigued just from dressing. He also states that he can’t get a good night’s sleep, as he has urinary frequency, which has improved recently. Past medical history is unremarkable. He takes ibuprofen daily for low back pain, and has been doing this for years. On examination: VS: T 37.3 P 100 R 14 BP 168/98. Oxygen saturation is 94%. HEENT is unremarkable. There is jugular venous distension to the angle of the jaw. Bibasilar rales are noted, extending up 1/3. A friction rub is noted on cardiac exam. 1+ pedal edema is present. The remainder of the examination is unremarkable. A basic metabolic panel is obtained, showing: Na 133; K 7.1; Cl 109; HCO3: 14 BUN: 98 Cr: 7.2. Glucose is 107. Appropriate management for this patient would be

A. Diuretics
B. Potassium-binding resin
C. Calcium gluconate
D. Hemodialysis

**Answer: D.** This patient is presenting with renal failure, complicated by hyperkalemia, volume overload, and pericarditis, and should be treated with hemodialysis. Indications for dialysis in the patient with renal failure include volume overload, severe metabolic acidosis, hyperkalemia, uremic complications (pericarditis, stupor, seizures, asterixis, and platelet dysfunction), or certain drug toxicities (e.g., lithium). While preparing this patient for hemodialysis, it may be appropriate to treat his hyperkalemia with potassium binders, calcium gluconate.
(and possibly potassium-wasting diuretics), but with his cardiac friction rub, hemodialysis is indicated. As patients with progressive renal failure often lose their ability to concentrate urine, polyuria often results, as seen in this patient.

Q.7. A 57-year-old woman is undergoing evaluation of angina and a positive stress test. Cardiology plans to perform coronary angiography next week, and the patient comes to you to discuss the plans. Past medical history is notable for multiple myeloma and hypertension. Current medications include lisinopril and hydrochlorothiazide, along with daily aspirin. Physical examination is notable only for good blood pressure control. Laboratory values, drawn yesterday, were unchanged from her baseline. BUN/Cr were 44/2.1. Appropriate options used to prevent deterioration of renal function while undergoing coronary angiography include

A. N-acetyl-cysteine, orally
B. Polyethylene glycol, orally
C. Corticosteroids, intravenously
D. Activated charcoal, orally

Answer: A. Contrast nephropathy is a common result from renal exposure to high osmolality contrast agents. These agents cause renal vasoconstriction and directly injure renal tubules. Risk factors for developing contrast nephropathy include renal insufficiency, diabetic nephropathy, multiple myeloma, and poor renal perfusion (as seen with heart failure, dehydration, or cirrhosis). Reversible renal failure results, with creatinine peaking one week after injury. Prevention is the best treatment, and includes minimizing dye load, hydration (1/2 NS at 1 cc/kg/hr 12 hours before and after IV contrast), and N-acetyl-cysteine, 600 mg orally twice a day for two days, starting one day prior to contrast exposure. There is no role for the other agents mentioned above in the prevention of contrast nephropathy.

CHAPTER 35: GLOMERULAR DISEASE

Q.1. An 18-year-old Caucasian male presents with rapid onset of lower extremity and periorbital edema. He admits to low-grade nocturnal fevers and sweats but otherwise feels well. He is taking no prescription or over-the-counter medications. Physical examination is notable for blood pressure of 90/50, pulse of 95, respiratory rate of 18, and no fever. HEENT examination shows no alopecia, and oral and nasal pharynxes are unremarkable. Examination of the neck reveals no palpable nodes and a normal thyroid. Lungs are clear. Cardiac examination is unremarkable. The abdomen is notable only for a questionably palpable spleen.
Extremities reveal 2 pitting edema to mid-shin. Lab screening reveals a BUN of 12 mg/dL, creatinine of 0.8 mg/dL, WBC of 10.5, Hct of 48%, and platelets of 225,000. Urinalysis shows specific gravity of 1.015, 4 protein, no RBCs, and no WBCs. Renal biopsy will most likely reveal which of the following?

A. Intense staining with IgG, IgA, and C3 on immunofluorescence
B. Immune complex deposition by electron microscopy
C. Normal light microscopy
D. Thickened capillary loops by light microscopy

**Answer: C.** This patient presents with symptoms classic for minimal change disease, which is associated with normal light microscopy. Patients will often state that they went to bed with no edema and woke up with 2 to 3 lower extremity pitting edema and/or periorbital edema. From the perspective of age, this patient is at the upper limit for first peak of minimal change disease, which runs from 4 months to 18 years, with most of those patients presenting before the age of 13 years. The patient has a normal BUN and creatinine and a urinalysis that confirms heavy proteinuria without cells—all consistent with minimal change disease.

**Q.2.** Before treatment of the individual in the preceding question, which of the following tests would be most appropriate to evaluate potential causes of minimal change disease?

A. Urine and blood for toxicology screen
B. Stool for occult blood
C. CT scan of chest and abdomen
D. ANA and dsDNA antibody titers
E. SPEP and UPEP

**Answer: C.** Secondary causes of minimal change disease include NSAID use (common), bee stings (uncommon), and lymphoma. Reviewing his history, this individual did not mention a bee sting, is taking no medications (including over-the-counter medications), but has fevers, night sweats, and a possibly enlarged spleen on physical examination. These should raise concern for lymphoma, and imaging of the chest and abdomen would be prudent.

**Q.3.** Which of the following is the most appropriate treatment for primary minimal change disease of the kidney?

A. Prednisone 60 mg orally each day for four to six weeks
B. Prednisone 40 mg and 100 mg oral cyclophosphamide for six months
C. Cyclophosphamide 750 mg IV monthly for six months
D. An ACE-inhibitor alone
**Answer:** A. Initial treatment of minimal change disease is oral prednisone. If the patient relapses, a steroid-sparing agent such as mycophenolate mofetil (Cellcept) or cyclosporine may be needed.

**Q.4.** Which of the following patients with diabetes and proteinuria should be evaluated for a cause of renal disease not related to diabetes?

1. An 18-year-old with type 1 diabetes since age 16 years who presents with 2 proteinuria found incidentally during a precollege physical
2. A 56-year-old asymptomatic woman with type 2 diabetes diagnosed at age 44 years who presents with 2 proteinuria and 10 to 15 RBCs but no WBCs on urinalysis
3. A 22-year-old with type 1 diabetes since age 10 years who presents with 3 proteinuria; ophthalmologic examination 4 months ago was normal
4. A 64-year-old with type 2 diabetes since age 50 years, now treated with insulin who presents with 1 proteinuria; she recently was treated with photocoagulation for vascular aneurysms of the retina

A. 1, 2, and 3
B. 1 and 3
C. 2 and 4
D. 4 only
E. All of the above should be evaluated for a non–diabetes-related cause of renal disease

**Answer:** A. Diabetes is a common secondary cause of renal disease, including nephrotic proteinuria. It is rare, however, for diabetic nephropathy to develop before having had diabetes for at least 10 years. The first patient above has had diabetes for only two years (asymptomatic, preclinical diabetes in type 1 diabetes mellitus is of short duration) and should be considered to have another disorder causing renal disease. The second patient, who has had diabetes for over 10 years, has a renal abnormality not associated with diabetes (i.e., hematuria); hematuria does not result from diabetic nephropathy. The third patient illustrates the point that retinopathy and nephropathy develop at the same time in almost all patients with type 1 diabetes mellitus; if nephropathy develops in the absence of retinopathy, the cause of the renal disease is unlikely to be diabetes. The fourth patient most likely has diabetes-related nephropathy. Although this patient also has diabetic retinopathy, nephropathy caused by diabetes in the absence of retinopathy is seen more commonly in type 2 diabetes as compared with type 1 diabetes.

**Q.5.** A 56-year-old African American man has a history of hypertension, but is otherwise healthy. Because of new-onset peripheral edema over the last six
months, you check labs and find a normal creatinine. His dipstick, however, is 4+ for protein and a subsequent 24-hour urine collection documents 3.5 g of protein per 24 hours. All other labs are normal and ultrasound of the kidneys is unremarkable. Which of the following would not be considered a usual manifestation of disease?

A. Albuminuria  
B. Hematuria  
C. Hypercholesterolemia  
D. Hypoalbuminemia

**Answer:** B. This patient has nephrotic-range proteinuria. Given his normal creatinine and renal ultrasound, the most likely diagnosis is focal segmental glomerulosclerosis (common in African-American patients). His presentation would be most consistent with that of any nephrotic syndrome. Specifically, low albumin, elevated cholesterol, and significant albuminuria are classic findings. Although hematuria may rarely present with one of the nephrotic syndromes, this is the exception as opposed to the rule. Hematuria is a feature of nephritic disorders.

**Q.6.** A 27-year-old African-American male with HIV, a low CD4 cell count, and a high viral load presents with edema, low total protein, and a creatinine of 3.4 mg/dL. He has no known history of kidney disease. A 24-hour urine collection reveals 4.5 grams of proteinuria in 24 hours. He has been taking ibuprofen daily for two months for knee pain. You decide to perform a kidney biopsy to aid with diagnosis. Which of the following is the least likely glomerular lesion found on histopathology?

A. Minimal change nephropathy  
B. Membranous nephropathy  
C. Focal segmental glomerulosclerosis  
D. HIV-associated nephropathy

**Answer:** B. Minimal change nephropathy may be seen with chronic NSAID use. Usually these patients also have interstitial nephritis and hence associated acute renal failure. Focal segmental glomerulosclerosis is the most common cause of nephrotic syndrome in African-Americans, and HIV-associated nephropathy occurs almost exclusively in African-Americans and in those with untreated HIV infection (with associated high viral loads and low CD4 counts in general). Membranous is the least likely in this scenario, though may be associated with hepatitis B when present.

**Q.7.** A 65-year-old man with a history of Wegener’s granulomatous successfully treated with oral cyclophosphamide presents for evaluation of hematuria (several
repeat urinalyses with >5 RBC/hpf). He has a urine protein of 200 mg in 24 hours and a stable creatinine at 1.4 mg/dL. ANCA titers are not changed from baseline. His underlying hypertension is well controlled. He has no sinus, pulmonary, or systemic symptoms. An abdominal CT shows no kidney masses, cysts, or stones. What do you do next?
A. Refer to nephrology for a kidney biopsy
B. Follow up in six months with a repeat urinalysis and a 24-hour protein
C. Start steroids and reassess in two weeks
D. Refer to a urologist for cystoscopy
E. Obtain a CT of the chest and sinuses

**Answer: D.** Because of the significantly increased risk of bladder cancer associated with the use of oral (more than IV) cyclophosphamide, any patient with this past exposure and hematuria should be referred to a urologist for cystoscopy. In the absence of proteinuria and other symptoms, it is less likely that the patient has a disease flare.

**Q.8.** Which of the following patients, all with a rapidly progressive rise in creatinine, is likely to have normal complement levels?
A. A 35-year-old man with a history of recurrent sinusitis and new pulmonary cavitary lesions
B. A 28-year-old woman with a malar rash, arthralgias, and an ANA titer of 1:640
C. A 42-year-old man with chronic hepatitis C, new-onset headaches, and palpable purpura on the lower extremities
D. A 25-year-old woman treated for “strep throat” 10 days ago with amoxicillin, with a blood pressure of 140/90 (formally normal)

**Answer: A.** It can be helpful to think of the causes of rapidly progressive glomerulonephritis (GN) as those with low complement (C3 and C4) levels and those with normal levels. In this question, Answer A describes a patient with Wegener’s granulomatosis, which causes a GN characteristically associated with normal complement levels. Answer B is a woman with systemic lupus erythematosus, Answer C is a man with cryoglobulinemia, and Answer D is a woman with poststreptococcal GN. All of these are usually associated with low complement levels.

**Q.9.** A 34-year-old woman with systemic lupus erythematosus presents for evaluation. Four months ago, she developed nephrotic syndrome. A renal biopsy showed membranous glomerulonephritis, and corticosteroids were begun. Proteinuria improved, and creatinine stabilized at 1.7. Today she awoke with bilateral flank pain. Urinalysis shows no crystals, but 4+ proteinuria was noted.
Creatinine from today’s bloodwork was 3.0. Appropriate treatment of the most likely cause of her presentation is

A. Cyclophosphamide
B. Heparin
C. Corticosteroids
D. Hydroxychloroquine

**Answer: B.** The most likely diagnosis in this patient is renal vein thrombosis. Renal vein thrombosis can occur with any cause of nephrotic syndrome, but is most common with membranous glomerulonephritis (as seen in the patient here). Concern for renal vein thrombosis should be raised in any patient with nephrotic syndrome who has sudden deterioration of renal function or worsening of proteinuria (as seen here). Patients may develop the acute onset of flank pain. Gold-standard diagnosis is obtained by venogram, but CT or MRI may demonstrate clot. Renal ultrasound may also demonstrate renal vein thrombosis, but if suspected and ultrasound is negative, further testing is warranted. Treatment is with anticoagulation.

**Q.10.** The following are pairings of causes of nephrotic syndrome and clinical features. Which pair contains a cause of nephrotic syndrome that is correctly paired with its clinical features?

A. Minimal change disease: Associated with colon cancer
B. Focal segmental glomerulosclerosis: The most common cause of nephrotic syndrome in adults
C. Membranous glomerulonephritis: Electron microscopy shows foot process effacement
D. Membranoproliferative glomerulonephritis: Associated with lupus and chronic hepatitis B

**Answer: D.** While renal biopsy is necessary to confirm a cause of nephrotic syndrome, there are clinical features that may suggest one diagnosis over another. Minimal change disease, which is seen in young children or older adults (mid-60s), may be precipitated by NSAID use, lymphoma, or bee sting (not colon cancer, as suggested in choice A above). Light microscopy is normal in minimal change disease, but electron microscopy will show foot process effacement. Focal segmental glomerulosclerosis (FSGS) affects people in their early teens through mid-30s, and may be precipitated by HIV infection, heroin use, or hyperfiltration. Focal and segmental glomerulosclerosis is seen on light microscopy, and foot process effacement is seen on electron microscopy. Membranous glomerulonephritis is the most common cause of nephrotic syndrome in adults (not FSGS, as suggested in choice B above), and may be associated with underlying adenocarcinoma (e.g., breast, lung, bowel). Membranous
glomerulonephritis may also be seen in individuals with lupus or hepatitis B infection. Light microscopy shows thickened capillary loops, and electron microscopy shows subepithelial immune complex deposition. Like membranous glomerulonephritis, membranoproliferative glomerulonephritis may be associated with lupus or hepatitis B infection (making answer D correct), and may also be precipitated by transplant rejection and shunt nephritis. Light microscopy shows capillary wall thickening, and electron microscopy shows subendothelial deposits or an electron-dense glomerular basement membrane.

Q.12. A 21-year-old male with type 1 diabetes mellitus (DM) comes for follow-up. He has noted that his urine is “foamy,” but otherwise has no complaints. He has had diabetes for four years. His last eye exam showed no retinopathy. Urinalysis shows 4+ proteinuria; basic metabolic panel is normal. The most likely cause of proteinuria in this individual is

A. Diabetes  
B. Minimal change disease  
C. Membranous glomerulonephritis  
D. Focal and segmental glomerulosclerosis  

Answer: C. Diabetic nephropathy occurs in patients with type 1 DM as well as type 2 DM. Diabetic nephropathy rarely develops before 10 years duration of disease (recall our patient was diagnosed four years prior). In patients with type 1 DM, nephropathy almost always occurs in the presence of retinopathy. The presence of nephropathy in a type 1 diabetic in the absence of eye disease (as seen in our patient) should prompt consideration of other causes of nephropathy. (The correlation between retinal disease and renal disease is less predictable in patients with type 2 DM). As membranous glomerulonephritis is the most common cause of nephrotic syndrome in adults, answer C is the correct answer.

Q.13. A patient presents with proteinuria, hematuria, hypertension, and peripheral edema. Urinalysis shows RBCs and RBC casts. Blood work shows normal electrolytes; BUN/Cr are elevated at 66/4.1 mg/dL. Serum complement is low. A possible etiology for this patient’s presentation includes

A. Goodpasture’s syndrome  
B. Wegener’s granulomatosis  
C. Thrombotic thrombocytopenic purpura  
D. Poststreptococcal glomerulonephritis  

Answer: D. Nephritic syndrome is diagnosed by the development of proteinuria, hematuria (often with RBC casts), hypertension and fluid retention, all described in this patient. Nephritic syndrome is often secondary to another systemic illness, which should be sought in any patient presenting with nephritis.
Serum complement may be used to narrow the differential diagnosis of patients presenting with nephritis. Patients with glomerulonephritis and low serum complement may have systemic diseases such as subacute bacterial endocarditis or lupus, or renal diseases such as poststreptococcal glomerulonephritis (the correct answer D), or membranoproliferative glomerulonephritis. Patients with glomerulonephritis and normal serum complement may have systemic diseases such as Wegener’s granulomatosis, Goodpasture’s syndrome, or TTP, along with renal diseases such as IgA nephropathy. (See Chapter 35 for a full listing).

Q.14. A 52-year-old Caucasian female is diagnosed by renal biopsy with membranous glomerulonephritis. Prior to discussions regarding therapy what additional testing would you recommend?
A. Mammography
B. Colorectal cancer screening
C. Chest x-ray
D. All of the above

Answer: D. The most common and frequently missed secondary cause of membranous glomerulonephritis in an adult population is adenocarcinoma of bowel, breast, or lung. The patient should be kept up to date on screening for breast cancer and colorectal cancer, and consideration should be given to imaging of her chest in this clinical presentation (there are no existing guidelines for routine screening for lung cancer).

Q.15. A 55-year-old Caucasian male presents for evaluation of dyspnea, hemoptysis, weight loss, and fatigue. The patient has a history of one to two packs of cigarettes a day for 10 years but quit five years ago. The patient denies fever or chills but does complain of muscle weakness and unspecified weight loss. He has noticed decreased urine output of dark-colored urine. Past history is negative for hypertension or renal disease. Physical exam reveals blood pressure of 175/105, pulse of 78, respiratory rate of 20, oxygen saturation on room air of 91%. His weight is 150 pounds and his temperature is 37.1° C. Physical exam is only notable for rhonchi in both lung fields and lower extremity edema. Urinalysis performed in the office reveals a specific gravity of 1.025, and dipstick reveals 4+ heme and 3+ protein. There are numerous RBC casts noted, including a BUN of 125 mg/dL and a creatinine of 3.1 mg/dL. Which of the following is (are) the most likely diagnosis in this patient?
A. FSGS
B. ANCA-associated GN
C. Membranous GN
D. Anti-GBM
E. A and C
F. B and D

**Answer: F.** This patient presents with a pulmonary renal syndrome, characterized by hemoptysis and acute renal failure. Renal involvement is indicative of glomerulonephritis. FSGS and membranous GN typically present with nephrotic syndrome and would be unlikely in this individual. With evidence of both pulmonary and renal involvement, consideration should be given to vasculitis (such as Wegener’s granulomatosis or micropolyangiitis; SLE would be less likely) or Goodpasture’s syndrome. Evaluation of this patient would typically include complement levels, ANCA testing, and testing for anti-GBM antibodies. Low complement would suggest SLE, but should be normal with micropolyangiitis, Wegener’s granulomatosis and Goodpasture’s syndrome. ANCA will be positive in Wegener’s granulomatosis and micropolyangiitis, but negative in Goodpasture’s syndrome. Evaluation should also include renal biopsy.

**CHAPTER 36: CHRONIC KIDNEY DISEASE**

**Q.1.** A 46-year-old white man with hypertension and chronic kidney disease secondary to diabetes mellitus (serum creatinine averaging 5.6 mg/dL over the past year) presents for a routine follow-up appointment. He reports no new symptoms and has not yet been placed on dialysis. His only medications are erythropoietin 5000 units twice weekly for the past six months and nifedipine. His hematocrit has been stable at 34% to 36%. Lab values obtained at this visit include BUN of 65 mg/dL, creatinine of 6.1 mg/dL, Hct of 28 %, and WBC of 8700. What is the most likely etiology for his falling hematocrit?

A. Noncompliance
B. Iron deficiency
C. Worsening renal function
D. Malnutrition
E. Folate deficiency

**Answer: B.** Patients maintained on erythropoietin will become iron deficient unless given exogenous iron. Oral absorption of iron in this population is poor. Iron stores should be checked on a quarterly basis, and intravenous iron given when the percent saturation of transferrin is less than 20% or the ferritin is less than 100.

**Q.2.** A 62-year-old woman with hypertension and diabetes mellitus has a slowly rising serum creatinine over the last 10 years—presumed due to poorly controlled
glucose levels. Her last serum creatinine was 5.1 mg/dL. Measurement of an intact PTH reveals a value of 125 ng/mL. Which of the following statements regarding her condition is false?

A. Decreased vitamin D production by the failing kidney is contributing to the increased parathyroid hormone (PTH) level
B. Decreased absorption of calcium from the gastrointestinal tract is contributing to the increased PTH level
C. Restriction of her dietary calcium will decrease the PTH level
D. Restriction of her dietary phosphorus will diminish the rise in her PTH level

Answer: C. This patient has secondary hyperparathyroidism due to chronic kidney disease. Many factors are responsible for this condition. Two basic mechanisms are most important. First, a decreased production of 1,25 OH vitamin D by the kidney, leading to decreased absorption of calcium from GI tract, results in hypocalcemia followed by an increase in PTH. Second, a decreased excretion of phosphorus by the kidney leads to hyperphosphatemia, which then combines with serum calcium to form calcium-phosphate complexes. This causes a further fall in serum calcium and a further increase in PTH. Restriction of dietary phosphorus will ameliorate hyperphosphatemia and diminish the rise in PTH. A restriction of dietary calcium will only worsen the situation and increase PTH levels.

Q.3. A 50-year-old, diabetic, hypertensive woman has significant proteinuria and mild chronic kidney disease with a creatinine of 2.0. She wishes to avoid dialysis in the future if at all possible. What advice is reasonable to give her?

A. She should try to achieve tight glucose control because this will delay progression
B. She should avoid ACE inhibitors, especially with a creatinine of 2.0, because this could worsen renal function
C. Normalization of her blood pressure will help delay progression
D. A and C are correct
E. A, B, and C are correct

Answer: D. Tight glycemic control in diabetic patients and normalization of blood pressure in hypertensive patients have both been shown to delay progression of chronic kidney disease. In addition, the use of ACE inhibitors has also been proven to slow progression, especially in diabetic patients. Although a patient may experience a slight rise in serum creatinine with ACE inhibition, the long-term benefits outweigh this possibility.

Q.4. A 62-year-old man has end-stage renal disease secondary to hypertension and has been on dialysis for the past four years. He is fairly healthy except for a history of lung cancer that was resected at age 45 years. He has no children, and
his parents are deceased. Which of the following statements is true regarding his candidacy for renal transplantation?

A. He is a reasonable candidate given his overall good health and lack of contraindications
B. He is not a good candidate because his age is over 60 years
C. He is not a good candidate because he has a history of malignancy
D. He is not a good candidate because he has few close relatives and the likelihood of an acceptable donor match is poor

**Answer: A.** This patient is fairly healthy and is a reasonable candidate for renal transplantation. There is no specific age cutoff for transplantation, and large numbers of patients in their 60s (and even 70s) have been transplanted. Any malignancy that is not considered cured is a contraindication to solid organ transplantation, but this patient’s history of lung cancer more than 15 years ago suggests that cure has likely been achieved. Finally, the lack of close, living relatives may preclude a live donor transplant but would not preclude a cadaveric transplant.

Q.5. A 65-year-old white woman with type 2 diabetes mellitus for the past 20 years presents for an initial office visit. She has a history of microalbuminuria for the past three years. Physical examination is notable only for a blood pressure of 140/70 with the remainder being normal. Urine dipstick reveals 2+ proteinuria, and a serum creatinine is 1.0 mg/dL. Which of the following statements is false?

A. This patient has chronic kidney disease
B. The patient should be treated with an ACE-inhibitor (ACE-I) or an angiotensin receptor blocker (ARB)
C. This patient is likely to also have anemia
D. The blood pressure goal for this patient would be less than 130/80
E. All of the above

**Answer: C.** The patient has chronic kidney disease (CKD) based on definition of National Kidney Foundation. Although the patient has a normal GFR, she does show evidence of structural disease by having chronic proteinuria of greater than three months’ duration. Her CKD is stage I. ACE-I or ARB therapy is recommended as first-line agents for any patient with diabetes and proteinuria or CKD and proteinuria. The desired blood pressure goal for this group is less than 130/80. Anemia is a later manifestation of CKD and would not routinely be expected until stage III or later.

Q.6. A 70-year-old African-American male is admitted to orthopedic surgery for management of a right trochanteric hip fracture sustained with minimal trauma. You are asked to provide medical consultation. His past medical history is notable
for long-standing hypertension, coronary artery disease status post endovascular stenting of the right coronary artery lesion one year ago, and chronic kidney disease (CKD) stage IV. His current medications include captopril 50 mg orally every eight hours, atenolol 50 orally every a.m., meperidine 50 mg intramuscularly every four to six hours as needed for pain, and ibuprofen 600 mg orally every eight hours for pain. Which of the following statements is true?

A. Meperidine should be discontinued because it is not effective in patients with kidney disease
B. Atenolol should be changed to metoprolol because atenolol is renally excreted and may lead to severe bradycardia in patients with advanced renal disease
C. Captopril should be discontinued because it is contraindicated in patients with stage IV kidney disease
D. Ibuprofen should be discontinued because it will worsen pre-existing renal disease
E. A and D
F. B and D

**Answer: F.** Meperidine is contraindicated in patients with advanced CKD. Normeperidine is an active metabolite of meperidine that is renally excreted. In patients with CKD, this metabolite accumulates and can lead to respiratory failure, seizures, and death. The drug is effective, but can clearly be toxic. Atenolol is also renally excreted and can accumulate in patients with CKD stages IV and V. The associated toxicity is bradycardia and heart block. If a β-blocker is indicated, as is the case for this patient (history of CAD about to undergo anesthesia), one should choose a beta-blocker that is not excreted by the kidney. Patients with CKD and HTN, especially in setting of proteinuria, should be on an ACE-I or ARB, rendering answer B false. All NSAIDS should be avoided in patients with CKD, as these patients require prostaglandins to maintain afferent arteriolar blood flow. The administration of a prostaglandin inhibitor will lead to a decrease in blood flow and a decrease in GFR.

**Q.7.** In the preceding case, laboratory values on admission were noted to be as follows: sodium 138 mEq/L, potassium 5.6 mEq/L, bicarbonate 16 mEq/L, chloride 105 mEq/L, BUN 56 mg/dL, creatinine 3.8 mg/dL, calcium 9.5 mg/dL, phosphorus 4.3 mg/dL, and Hgb 10 g/dL. Which of the following would be an appropriate recommendation?

A. Check an intact PTH
B. Check a 25-OH vitamin D level
C. Check transferrin % saturation and ferritin
D. Begin potassium citrate orally 30 mL twice daily
E. A and C
Answer: E. The patient has stage IV CKD (GFR 15–29 mL/min). Patients at this stage generally have decreased production of 1,25 OH vitamin D with subsequent increase in PTH secretion. If left untreated, this leads to a decrease in bone mineral density. In this case, the patient sustained a hip fracture with “minimal trauma.” Underlying metabolic bone disease should be suspected, and it is appropriate to check an intact PTH. Regarding the patient’s anemia, the kidney has decreased erythropoietin production at this stage. This patient will likely need erythropoietin or darbepoietin to correct the anemia. It is prudent to make sure the patient’s iron stores are replete before starting therapy, as patients respond poorly to the drug when iron deficient. Measuring a transferrin % saturation and a ferritin is appropriate. Although patient has a low bicarbonate, potassium citrate is contraindicated secondary to the elevated potassium. In this situation, sodium citrate or sodium bicarbonate would be more appropriate.

Q.8. A 42-year-old woman has severe, progressive lupus that has led to chronic kidney disease. Her last serum creatinine measurement was 7.3 mg/dL. She was formerly asymptomatic, but is now experiencing progressive, intractable volume overload. Which of the following statements regarding modality selection for renal replacement therapy is true?
A. All patients are candidates for renal transplantation
B. Patients who choose hemodialysis should have an AV fistula created at least two months prior to initiation of dialysis
C. Peritoneal dialysis is less desirable because of higher infection rates when compared to hemodialysis
D. There are no contraindications to peritoneal dialysis
E. All of the above

Answer: B. Although all patients should be considered for renal transplantation, not all are appropriate candidates. Patients who are likely to die within the next four years due to comorbid conditions, patients with active infections, or patients with recent malignancies are not transplant candidates. Peritoneal dialysis patients have fewer infections than those on hemodialysis. There are contraindications to peritoneal dialysis, including unrepaired hernias, active intraperitoneal infections (e.g., diverticulitis), and communication between the diaphragm and pleural space (common after open heart surgery), among others.

Q.9. A 56-year-old African-American woman has chronic renal failure from diabetes and hypertension that you have been following for years. Her creatinine has been stable at 4.5 mg/dL. She is on metoprolol for blood pressure control and rosiglitazone for her diabetes. Her most recent bloodwork, from earlier this morning, shows sodium of 139 mEq/L, potassium of 5.6 mEq/L, bicarbonate of 17
mEq/L, creatinine of 4.7 mg/dL, and glucose of 145 mg/dL. She is now in your office and states she feels fine. You perform an ECG, which shows normal sinus rhythm and no new changes. Which of the following is most appropriate to pursue?

A. Dietary restriction of high-potassium foods, add furosemide and sodium bicarbonate, and discontinue β-blocker
B. Dietary restriction of protein and addition of furosemide
C. Administer insulin and glucose, discontinue β-blocker, and add furosemide
D. Dietary restriction of both high-potassium foods and protein, and add spironolactone
E. Addition of sodium bicarbonate and furosemide

**Answer: A.** This patient has hyperkalemia secondary to her chronic renal insufficiency. There is nothing to suggest that this is an acute process, such as symptoms or ECG changes. Rapid administration of insulin and glucose is not necessary. There are several measures that could be taken to correct the hyperkalemia on a chronic basis. Dietary restriction of high-potassium foods is helpful, as is addition of a loop diuretic (e.g., furosemide) that will result in urinary excretion of potassium. Correcting the acidosis by adding sodium bicarbonate will improve the hyperkalemia (acidosis shifts potassium extracellularly, which increases serum values). Discontinuing the β-blocker (which may be associated with hyperkalemia) may also be helpful. Restriction of dietary protein may delay progression of the renal disease, but will do nothing to improve the hyperkalemia. Likewise, addition of a potassium-sparing diuretic (e.g., spironolactone) may only worsen matters.

**Q.10.** You have been following a 58-year-old man with hypertensive renal disease for years. Because he frequently misses appointments, his blood pressure control has been suboptimal. His creatinine has slowly risen and is now 7.5 mg/dL. His urinalysis shows only 1+ proteinuria and no hemoglobin or RBCs. A 24-hour urine collection reveals 980 mg of protein excretion per 24 hours. His BUN is 68 mg/dL, potassium is 4.5 mEq/L, and hematocrit is 34%. He has mild pedal edema, but denies nausea, significant fatigue, pruritis, or chest pain. He has no living relatives, as he was adopted. His only other past medical history includes gout and an umbilical hernia repair three year ago. He is interested in dialysis when you feel he needs it. Which of the following statements about this patient is true?

A. Because he is relatively asymptomatic, it is too early to initiate dialysis planning
B. He should be referred to a vascular surgeon for arteriovenous fistula placement if he chooses hemodialysis
C. A cadaveric transplant will have a better five-year survival in this patient than a living unrelated donor organ
D. His history of umbilical hernia repair is a contraindication to peritoneal dialysis
E. He probably has underlying hypertensive vascular disease, and this will preclude evaluation for a renal transplant

Answer: B. This patient has chronic renal failure secondary to hypertension, as supported by his mild proteinuria and small kidneys by ultrasound. Although he is relatively asymptomatic and has no urgent need for dialysis (e.g., hyperkalemia, uremia), dialysis planning should start immediately. Most experts recommend institution of dialysis when the creatinine is 8.0 mg/dL in nondiabetics (for diabetics, institute for creatinine of 6 mg/dL or higher). Referring this patient to a vascular surgeon for arteriovenous fistula placement is appropriate (if he chooses hemodialysis), as these fistulae require 6 to 12 months to mature. They are the preferred access route for hemodialysis, as they are less susceptible to infection and have better longevity when compared with catheters and prosthetic grafts. If the patient chooses peritoneal dialysis, his history of umbilical hernia repair is not a contraindication. Regarding possible renal transplant, his hypertensive disease does not preclude an evaluation. Despite his lack of blood relatives and inability to secure a living, related donor, he will do better with a living unrelated donor than a cadaveric donor (lower graft survival with cadaveric graft due to cold ischemic times during transport).

Q.11. A 65-year-old male, retired banker has well-controlled diabetes but persistently elevated blood pressure despite therapy with metoprolol and hydrochlorothiazide. His urinalysis now shows microabuminuria, but his creatinine is stable at 1.1 mg/dL. You start low-dose ramipril. Three weeks later, he returns to your office for follow-up. His blood pressure is better at 140/90, but his creatinine is now 1.8 mg/dL and his potassium is 5.2 mEq/L. What should you do next?
A. Add a loop diuretic and order renal doppler studies
B. Start kayexalate immediately and repeat the potassium in one to two days
C. Discontinue ramipril and order an intravenous pyelogram
D. Change ramipril to irbesartan and add a loop diuretic
E. Discontinue ramipril and order a magnetic resonance angiogram of the abdomen

Answer: E. In this patient with diabetes and new microabuminuria, it is appropriate to add an ACE inhibitor (such as ramipril) to attempt to delay progression of renal disease. He then, however, developed a sudden rise in his creatinine and potassium levels. It is highly likely that he may have underlying renal atherosclerotic vascular disease, especially given his poorly controlled hypertension. The ramipril should be stopped immediately. A magnetic resonance...
angiogram should be ordered, as it is the best initial step in pursuing the diagnosis, and performs better than renal Doppler studies. Thus choice E is most appropriate. The patient is stable and his potassium is only minimally elevated; there is no reason to start kayexalate. Changing ramipril to an irbesartan is not useful, as angiotensin receptor blockers are equally likely to cause a rise in creatinine in a patient with underlying renal vascular disease. Ordering an intravenous pyelogram will not aid in the diagnosis of renal vascular disease, and, in fact, the dye load may worsen the renal insufficiency.

CHAPTER 37: SELECTED TOPICS IN NEPHROLOGY

Q.1. A 70-year-old African-American man presents for evaluation of painless gross hematuria. He has a past medical history of recent deep venous thrombosis and has been on warfarin. He has a family history of sickle cell trait. He has worked in a dye factory and continues to smoke two packs of cigarettes a day. On physical examination he is normotensive, has no edema, and has an enlarged prostate without tenderness or discrete nodules. His INR is in the correct therapeutic range. Urinalysis reveals too numerous to count red cells per high power field. There is no mention of dysmorphic red cells or red cell casts. What should the evaluation include?

A. No further workup is needed because of the presence of sickle cell trait
B. No further workup because of the presence of warfarin
C. Urologic referral for cystoscopy
D. No further workup is needed because of his benign prostatic hypertrophy
E. Serologic testing for ANCA, anti-GBM, ANA, and complements, and urgent referral to nephrology for kidney biopsy

Answer: C. One third of African Americans may have sickle cell trait or sickle cell disease as the etiology of hematuria because of papillary necrosis; however, this patient has a worrisome history that would make further workup advisable. With currently accepted levels of anticoagulation, anticoagulation is not an accepted cause of hematuria. Benign prostatic hypertrophy is listed as a cause of microscopic hematuria in some reviews, although this is somewhat controversial. Serologic workup for glomerulonephritis will most likely be negative. The patient has no symptoms or signs of a systemic vasculitis. Urinalysis did not reveal the presence of dysmorphic red cells or red cell casts that can indicate a cause of hematuria. His smoking and dye exposure are risk factors for bladder cancer, making it a possibility. The patient should be referred to urology for cystoscopy.
Q.2. Which of the following is a characteristic feature that points to a glomerular cause of hematuria?

A. Isomorphic (same size and shape) red blood cells on phase contrast microscopic examination of the urine
B. Blood clot formation
C. Hematuria noted at the onset of micturition (initial hematuria)
D. Red blood cell casts on microscopic examination of the urine

**Answer: D.** Red blood cell casts are indicative of glomerular disease. Tamm-Horsfall protein is made in the tubule and can act as a matrix for red blood cell attachment. This forms a cylindrical cast shape of the tubule. Red cell casts are found in about 25% of glomerular bleeding. Isomorphic red blood cells are felt to be indicative of nonglomerular hematuria. The presence of more than 80% of dysmorphic (varying in size and shape) red blood cells in the urine is characteristic of glomerular hematuria. The variation in size and shape may be caused by deformation as red blood cells pass through the glomerular basement membrane, as well as by damage caused by changes in pH and osmolality as red cells travel through the nephron. Blood clots are not characteristic of glomerular hematuria because of urokinase and tissue plasminogen activator made in the glomeruli and tubules. Initial hematuria is indicative of urethral source of bleeding, whereas terminal hematuria is indicative of a source in the trigone of the bladder.

Q.3. A 27-year-old man has an initial evaluation for hematuria. He is found to have multiple cysts on each enlarged kidney by ultrasound. His serum creatinine is 1.0 mg/dL. There is no family history of polycystic kidney disease. Which of the following statements is incorrect?

A. The patient may have autosomal dominant polycystic kidney disease even with a negative family history
B. The patient will be on hemodialysis in two years
C. The patient may develop infections in these kidney cysts in the future
D. If the patient develops severe headaches, one should be suspicious of intracranial aneurysms

**Answer: B.** This patient has polycystic kidney disease. A positive family history is detected in 60% to 75% of patients. One common problem is infection of these kidney cysts. Antibiotics that penetrate cysts well are ciprofloxacin and trimethoprim/sulfamethoxazole. Urine cultures may be negative because fluid in cysts may not drain into the collecting system. Intracranial aneurysms of the circle of Willis are the most serious extrarenal complications of autosomal dominant polycystic kidney disease. The incidence of these aneurysms is estimated to be approximately 10%. Patients who have severe headaches or a family history of intracerebral aneurysms are screened for intracranial aneurysms. Presently, MRA
is used as the radiographic procedure of choice. The patient may develop end-stage renal disease; however, his serum creatinine at this time is normal. This is a slowly progressive disease, with most patients requiring dialysis by the fifth to sixth decade of life. It is extremely unlikely that the patient would have rapid progression of renal failure from his polycystic kidney disease within two years.

Q.4. A 24-year-old male with recurrent nephrolithiasis and a strong family history of kidney stones comes to you for evaluation. Plain radiographs show radiopaque stones; however, they are less dense than calcium stones. Urinalysis reveals hexagonal crystals. Which of the following is not a reasonable treatment to prevent recurrent nephrolithiasis?
A. Increased fluid intake
B. Alkalinized urine
C. Penicillamine
D. Allopurinol

Answer: D. The patient’s hexagonal crystals are pathognomonic for cystine stones. This is a young patient with a positive family history for kidney stones. Some patients have an autosomal recessive defect in the proximal tubule that causes decreased reabsorption of cystine from the urine. The goals of therapy are to increase fluid intake to try to decrease urine cystine concentration to less than 300 mg/liter of urine. Alkalinizing the urine is a goal of therapy; however, this is often difficult to achieve in practice because of the large amounts of citrate needed to alkalinize the urine. Medications such as tiopronin, penicillamine, and captopril have sulfhydryl groups that can form disulfide bonds with the cystine molecule to prevent stones. Allopurinol would be used if the patient had uric acid stones and a high urine uric acid excretion. These stones would be radiolucent unless mixed with calcium stones. Uric acid crystals are often rhomboid in nature and not hexagonal.

Q.5. A 45-year-old man presents to the emergency department with severe flank pain. He has a history of obesity for which he had previously undergone small bowel bypass surgery. On physical exam, he is afebrile and his abdomen is soft. His urinalysis showed too numerous to count red cells and 0 to 1 white blood cells per high power field and no bacteria. Urine dipstick was negative for proteinuria. What diagnostic test should be performed first?
A. Urine culture
B. Abdominal CT with contrast
C. Abdominal CT without contrast
D. 24-hour urine collection
**Answer: C.** The patient’s previous small bowel bypass surgery predisposes him to renal stone formation. His flank pain and isolated hematuria are most consistent with acute nephrolithiasis. The stone is most likely calcium in nature; therefore, a noncontrast abdominal CT would be the most useful initial test to detect this radiopaque renal stone. Although pyelonephritis may also present with flank pain, the patient is afebrile and has no pyuria or bacteriuria to suggest infection. An abdominal CT with contrast is indicated for patients with hematuria in which a noncontrast CT was unrevealing or in whom concern for an intra-abdominal, pelvic, or vascular pathology is warranted. A 24-hour urine collection is unnecessary in a patient with isolated hematuria and without symptoms suggestive of a systemic disease. A 24-hour urine collection for stone risk profile may be considered in patients with recurrent nephrolithiasis.

**Q.6.** A 65-year-old woman with a chronic indwelling urinary catheter complains of a four-week history of back pain. On physical exam she is afebrile and has mild bilateral costovertebral tenderness. Her urinalysis showed 15 to 20 red cells and 30 to 35 white blood cells and many bacteria per high power field. Her abdominal x-ray shows large bilateral staghorn calculi. Which of the following statements about this condition is incorrect?

A. Urine pH is generally greater than 7.2
B. Her back pain arises from infection with urease-producing bacteria
C. Urine ammonium levels are elevated
D. Long-term antibiotic course alone is adequate for management

**Answer: D.** The patient’s abdominal x-ray demonstrates large bilateral staghorn calculi. Struvite calculi account for the formation of most staghorn calculi. Patients with voiding dysfunctions (e.g., neurogenic bladder) or with indwelling Foley catheters are at particular risk for struvite stones. They form when urinary infection with urease-producing bacteria (e.g., Proteus, Klebsiella) leads to alkaline urine and high urine ammonium levels. These two conditions promote formation of magnesium-ammonium-phosphate stones. Antibiotic therapy alone is inadequate as the bacteria persist within the stones even though urine cultures may be negative. Adequate treatment requires complete removal of the calculi with a urological procedure. Without treatment, staghorn calculi may lead to recurrent urinary tract infections and renal dysfunction.

**Q.7.** A 27-year-old man presents for a pre-employment physical examination. He is without complaints, and has no significant past medical history. He takes no medications. He works as a physical fitness trainer. Physical examination is normal. The employer requests a CBC, basic metabolic panel, and urinalysis. Testing is normal, except urinalysis shows 10 to 15 RBCs per high-powered field.
No RBC casts are seen, and no WBCs are seen. Which one of the following statements is correct?

A. Exercise alone may cause transient hematuria
B. The most common cause of hematuria in adult males is gonorrhea
C. A history of analgesic use suggests nephrolithiasis as the cause of hematuria
D. A history of tobacco use suggests renal cell carcinoma as the cause of hematuria

**Answer: A.** Hematuria is defined as the presence of more than 3 RBCs per high-powered field. The most common causes of hematuria are infection (e.g., urinary tract infection) and nephrolithiasis. Gonorrhea and other causes of urethritis more commonly present with pyuria, not hematuria. Hematuria may result from systemic processes, including fever, CHF, and exercise, and is transient. Our patient, a fitness trainer, may have transient hematuria as a result of recent exercise; repeat testing should be performed after three days without exercise. Analgesic use predisposes to papillary necrosis, not nephrolithiasis. Tobacco use increases the risk of bladder cancer, not renal cell carcinoma.

**Q.8.** The following tests are used to evaluate the patient with hematuria. Which test is appropriately paired with the clinical scenario that it best evaluates?

A. Intravenous pyelography: suspected glomerulonephritis
B. Renal ultrasound: suspected papillary necrosis
C. CT scan: suspected nephrolithiasis
D. Cystoscopy: suspected medullary sponge kidney

**Answer: C.** When evaluating the patient with hematuria, the appropriate diagnostic test must be chosen. Urinalysis should be performed on all patients with suspected hematuria; the presence of clots, proteinuria, RBC casts, or WBCs will narrow the differential diagnosis and define further testing. Intravenous pyelography (IVP) should be used to evaluate the patient who has hematuria from a nonglomerular source (i.e., IVP is not the best test for evaluation of suspected glomerulonephritis). IVP is the test of choice for evaluation of suspected papillary necrosis and medullary sponge kidney, and is good for stones or ureteral tumors. Ultrasound is used for glomerular causes of hematuria, or when dye allergy or renal insufficiency is present. Ultrasound is the best test for polycystic kidney disease and renal cell CA. CT scanning has become the best test for identification of nephrolithiasis, and is often used to define masses initially diagnosed by ultrasound.

**Q.9.** A 49-year-old woman is diagnosed with hyperparathyroidism. Two weeks later, she presents to you with left-sided flank pain radiating to her groin. Urinalysis shows 25 to 35 RBCs, 3 to 5 WBCs, and too numerous to count envelope-shaped
crystals. Once the acute event passes, which one of the following will prevent recurrence (before her hyperparathyroidism is treated)?

A. Allopurinol daily
B. High-salt diet
C. Hydrochlorothiazide daily
D. Potassium citrate daily
E. Sodium bicarbonate daily

**Answer: C.** This patient is presenting with nephrolithiasis in the setting of hyperparathyroidism. Hyperparathyroidism leads to hypercalciuria and calcium oxalate stones (which appear as envelope-shaped crystals in the urine). Hypercalciuria is treated with either a low-sodium diet (calcium excretion parallels sodium excretion) or thiazide diuretics (which decrease hypercalciuria). Definitive treatment would be correction of hyperparathyroidism. Allopurinol is used for prevention of recurrent uric acid stones. Potassium citrate can be used to correct acidic urine in patients with uric acid stones. Sodium bicarbonate is not used in the management of nephrolithiasis.

**Q.10.** Which of the following is not a risk factor for papillary necrosis?

A. Sickle cell disease
B. Hemolytic-uremic syndrome
C. Diabetes
D. Urinary tract obstruction
E. Tuberculosis

**Answer: B.** Papillary necrosis results from ischemic damage to the renal papilla. Risk factors include sickle cell disease, diabetes, NSAID use, urinary tract obstruction, and tuberculosis. Hemolytic-uremic syndrome is not a risk factor for papillary necrosis.