POTASSIUM WORKSHOP

Potassium Learning Objectives

At the completion of this module the student should be able to:

- Recognize that multiple metabolic disturbances can occur simultaneously.
- Define and calculate osmolar gap.
- List causes for an elevated osmolar gap.
- Relate the acid-base disturbances seen with toxic ingestions.
- Describe the role of potassium intake, potassium losses and potassium redistribution in determining serum potassium level.
- Use results of 24 hour urine collection for potassium to distinguish between renal and extra-renal potassium losses.
- List the factors that can influence the distribution of potassium between intracellular and extracellular compartments.
- Describe the association and interaction of potassium balance with acid-base abnormalities.
- Define osmotic diuresis.
- List causes of osmotic diuresis.
- List fluid and electrolyte abnormalities caused by osmotic diuresis.
- List factors necessary for adequate potassium excretion by the cortical collecting duct.
- Describe the role of the renin-angiotensin-aldosterone system in potassium and acid-base balance.

Case 1:

Bob becomes an alcoholic and loses his job at a temporary accounting firm. Since being out of work he has had difficulty affording his alcohol. One day after drinking a very cheap pint that he bought on the street, he presents to the emergency room with weakness, nausea, headache and decreased vision.

PHYSICAL EXAMINATION: BP 100/60, pulse 100, respiration 24, temperature 97. Fundoscopic examination: retinal sheen.

LABORATORY DATA: sodium 140 mEq/L, potassium 4.0 mEq/L, chloride 104 mEq/L bicarbonate 12 mEq/L, BUN 14 mg/dl, creatinine 1.0 mg/dl, glucose 90 mg/dl, serum osmolarity 320 mosm/L.
ABG: pH 7.28, $PC0_2$ 26 mm/Hg, bicarbonate 12 mEq/L.

1) What is Bob's acid base disturbance?

2) Calculate his serum osmolarity?

3) What is meant by an osmolar gap and does Bob have one?

4) What are the causes of an osmolar gap and how does it help in making the diagnosis?

5) What did Bob drink and how does it account for his acid-base disturbance and elevated serum osmolarity?

6) Would the finding of calcium oxalate crystals on urinalysis alter the diagnosis?

7) What treatment should Bob receive?

Case 2:

John returns to Burger World and tries their "Fish Delight". The next day he develops severe abdominal cramps, nausea and watery diarrhea. Because of his nausea, he is unable to take in any liquids. He comes to the emergency room.

PHYSICAL EXAMINATION: Supine: BP 90/60, pulse 120, standing: BP 60/40, pulse 140, lungs are clear. Heart: rapid heart sounds. Abdomen: hyperactive bowel sounds, mild diffuse tenderness. Extremities: no edema.

LABORATORY DATA: Sodium 148 mEq/L, potassium 2.8 mEq/L, chloride 126 mEq/L, bicarbonate 10 mEq/L, BUN 60 mg/dl, creatinine 1.8 mg/dl, glucose 90 mg/dl.
ABG: pH 7.26, PC02 23 mm/Hg, bicarbonate 10 mEq/L.
Urine: sodium 10 mEq/L, potassium 20 mEq/L, creatinine 90 mg/dl.
24 hour urine potassium 20 mEq/day. Urinalysis: pH 5, specific gravity 1.025, no sediment.

1) Does John have a free water excess or deficit? How did he develop it?

2) What is John's volume status?

3) What is John's fractional excretion of sodium?

4) What is the etiology of John's acute renal failure?

5) What is John's acid-base status?

6) Hypokalemia can be caused by decreased potassium intake, increased potassium losses or redistribution of potassium with a shift of potassium into the intracellular compartment.

   a) What factors can induce a shift of potassium intracellularly?
   b) What are potential sources of potassium loss and how can urine electrolytes distinguish between them?

7) What are causes of hypokalemic metabolic acidosis?

8) What is the cause of John's metabolic abnormalities?

9) What should be the composition of the replacement solution that is ordered for John?

10) Which should be corrected first, the acid-base disorder or the potassium disorder, and why?

11) Why does John keep going back to Burger World?

Case 3:

Tiny Tim has taken baking soda since he was an infant. Unfortunately, his father was fired from Scrooge Industries after their earnings fell because of heavy exposure to Asian markets. There was no money to buy baking soda which is not a Medicaid approved drug. Tiny Tim was brought to the emergency room extremely weak.

PHYSICAL EXAMINATION: small for his age, 14 year old male looking pale and weak. BP 100/60, pulse 100, respiration 12, afebrile.

LABORATORY DATA: sodium 140 mEq/L, potassium 2.2 mEq/L, chloride 120 mEq/L, bicarb 8 mEq/L, BUN 20 mg/dl, creatinine 1.0 mg/dl, glucose 90 mg/dl.
ABG: pH 7.04, $PC0_2$ 30 mm/Hg, bicarb 8 mEq/L.
24 hour urine potassium: 40 mEq/day. Urine pH 6.5, specific gravity 1.006, no sediment.

1) What is Tiny Tim's acid-base disturbance?

2) Considering the severity of his metabolic acid-base disturbance, why isn't his $pC0_2$ lower?

3) Why is Tiny Tim's urine pH 6.5 (why isn't it more acidic)?

4) What is Tiny Tim's diagnosis?

5) Why is Tiny Tim at risk for nephrocalcinosis and renal stones?

6) What does baking soda contain, and why is it therapeutic for Tiny Tim?

7) How much baking soda does Tiny Tim need to restore his health?

Case 4:

Tiny Tim's brother Thin Jim is a type I diabetic. He runs out of insulin and is admitted to the hospital three days later.

PHYSICAL EXAMINATION: supine: BP 90/60, pulse 120, sitting: BP 70/50, pulse 140, respiration 24, afebrile, weight 60kg.

LABORATORY DATA: sodium 144 mEq/L, potassium 2.8 mEq/L, chloride 108 mEq/L, bicarbonate 5 mEq/L, BUN 60 mg/dl, creatinine 2.0 mg/dl, glucose 500 mg/dl.
ABG: pH 7.14, $PC0_2$ 15 mm/Hg, bicarbonate 5 mEq/L.

1) Does Thin Jim have a free water excess or deficit?

2) What will happen to Thin Jim's serum sodium concentration when he is treated with insulin?

3) What is Thin Jim's volume status?

4) What is Thin Jim's acid-base disturbance?

5) What is an osmotic diuresis and how did it contribute to Thin Jim's electrolyte disturbances?

6) What are other causes of an osmotic diuresis?

7) What will happen to Thin Jim's potassium when he is given insulin? How should this affect therapy?

Case 5:

Tiny Tim's grandfather Max is a 59 year old male and has a past history of tuberculosis. He has been having generalized malaise, weight loss and anorexia for three months. While visiting Tiny Tim and Thin Jim in the hospital, he decides to have himself checked out in the emergency room.

PHYSICAL EXAMINATION: sitting BP 90/60, pulse 110, standing BP 75/50, pulse 120, respiration 16, afebrile, brownish-grey spots noted on mucus membranes.

LABORATORY DATA: sodium 120 mEq/L, potassium 6.0 mEq/L, chloride 92 mEq/L, bicarb 16 mEq/L, BUN 30 mg/dl, creatinine 1.2 mg/dl, glucose 50 mg/dl.
ABG: pH 7.35, $PC0_2$ 30 mm/Hg, bicarbonate 16. Urine sodium 40 mEq/L, urine osmolarity 350 mosm/L.

1) Does Max have a free water excess or deficit?

2) What is Max's effective circulating volume; is it increased, decreased or normal?

3) What is Max's acid-base disturbance?

4) What factors are necessary for adequate potassium excretion by the cortical collecting duct?

5) What are Max's serum renin, aldosterone and cortisol levels?

6) What is the pathogenesis of Max's hyponatremia, hyperkalemia and acid-base disturbance?

7) Explain the urine sodium of 40 mEq/L.

Case 6:

Tiny Tim's mother Mary finds a job and is found to be hypertensive with a BP of 180/110 on an insurance physical. She had last seen a physician 14 years ago when she delivered Tiny Tim. Mary is sent to see an internist who obtains the following labs:

Sodium 144 mEq/L, potassium 3.0 mEq/L, chloride 102 mEq/L, bicarbonate 30 mEq/L, BUN 12 mg/dl, creatinine 1.0 mg/dl, glucose 90 mg/dl.
Urinalysis: pH 5, specific gravity 1.012, urine sediment negative, urine chloride 60 mEq/L, urine potassium 40 mEq/L.
24 hour urine: potassium 50 mEq/day.
ABG: pH 7.45, $pC0_2$ 44 mm/Hg, bicarb 30 mEq/L.

1) Does Mary have essential hypertension?

2) What is Mary's acid-base disturbance? Is it "chloride-dependent" or "chloride-independent"?

3) Does Mary have renal or extra-renal potassium losses?

4) What do you expect her renin and aldosterone levels to be?

5) What are other common causes of hypokalemic metabolic alkalosis and how would you exclude them?