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ELECTROLYTE GAMES FOR RAINY AFTERNOONS

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Correlate the clinical histories with the electrolytes presented. Answers are below.

A. Serum calcium 4.8 mg/dL
B. Urinary electrolytes: sodium 110 mEq/L; potassium 46 mEq/L; chloride 90 mEq/L
C. Serum potassium 6.7 mEq/L
D. Serum sodium 125 mEq/L
E. Serum calcium 5.8 mEq/L

1. A 39-year-old man with recently diagnosed nephrotic syndrome secondary to steroid-resistant focal segmental glomerular sclerosis (FSGS) was admitted to the hospital 4 weeks after cyclosporine A was added to therapy, which had caused him to become encephalopathic. His blood sugar on admission was 900 mg/dL.

2. A 65-year-old woman with a documented diagnosis of myelofibrosis was admitted to the hospital because of increasing weakness. On admission, hemoglobin was 6.9 g/dL and platelet count was 1 million.

3. A 55-year-old woman with carcinoma of the breast underwent magnetic resonance imaging of the chest with intravenous gadolinium for evaluation of suspected metastatic disease.

4. A 24-year-old woman came to the hospital because of profound weakness. Serum electrolytes upon admission showed a potassium level of 2.8 mEq/L, chloride of 88 mEq/L, bicarbonate of 35 mEq/L, and sodium of 140 mEq/L. She denied vomiting or diuretic use.

5. A 60-year-old man with end-stage renal failure for the last 15 years had severe secondary hyperparathyroidism (PTH levels > 1500) despite attempting to suppress parathyroid hormone production with nearly maximal doses of vitamin D (Hectorol 10 MIC IV after each dialysis) and calcimimetics (Sensipar 90 mg/day). The beneficial effects of both drugs were limited by evolving hypercalcemia (vitamin D analogue) and gastrointestinal symptoms (Sensipar). In view of erythropoietin resistance (early myelosclerosis due to PTH excess) and risk of calcific arteriopathy, the patient underwent total parathyroidectomy and parathyroid tissue implant in the forearm.

Correct Answers

1. **D: Pseudohyponatremia in a patient with hyperosmolar nonketotic coma induced by cyclosporine A.** To correctly assess this situation, it is important to know that there is a predictable decrement of serum sodium levels of 1.35 mEq/L for every 100 mg/dL of glucose levels above 100 mg/dL. In this case, adding 10.8 mEq/L to the reported value of 125 mEq/L results in a true sodium level of 135 mEq/L. Hypernatremia can also be present in cases of severe hyperglycemia (blood glucose > 500 mg/dL) due to profuse polyuria from the osmotic-diuretic effect of glycosuria. This results in water losses in excess of solute losses, in turn leading to hyperosmolality. Pseudonormonatremia can also be present in a similar situation of severe hyperglycemia, when "normal" serum sodium levels are corrected for the factor mentioned above (1.35 mEq/L for every 100 mg/dL of glycemia > 100).

2. **C: Pseudohyperkalemia.** This is an in vitro phenomenon due to potassium release from marked elevation of white blood cells, as in leukemia (80000-100000), or marked platelet elevation (500000-1000000) as in this case. Under those circumstances, simultaneous determination of serum potassium (clotted blood specimen) and plasma potassium (unclotted blood specimen) will show normal levels in the plasma potassium. Another clue is the absence of electrocardiographic abnormalities present in true hyperkalemia.

3. **A: Pseudohypocalcemia induced by gadolinium.** With the increasing volume of enhanced magnetic resonance imaging studies ordered in daily clinical practice, clinicians need to be aware that gadolinium interferes with the colorimetric calcium assays commonly used in hospitals. Calcium levels will be abnormally low if obtained immediately after gadolinium exposure. Knowing that pseudohypocalcemia can occur under the circumstances described above should prevent unnecessary and potentially dangerous administration of intravenous calcium.

4. **B: Surreptitious diuretic use.** The only clinical scenario of hypokalemic hypochloremic metabolic alkalosis occurring with elevated levels of sodium/chloride/potassium in the urine results from the natriuretic/chloruretic/kaliuretic effect of diuretics. When confronted with the facts, this young woman with a borderline personality admitted to using her grandmother's furosemide for "weight reduction." Bulimia can also result in a similar metabolic profile (hypokalemia, hypochloremia, and metabolic alkalosis). Under those conditions, hypochloremic metabolic alkalosis results from hydrochloric acid losses via repeated vomiting. The ensuing volume contraction elicits aldosterone release with increasing sodium for potassium exchange at the renal tubular level, hence hypokalemia develops. As a result, urinary electrolytes (in contradistinction to diuretic users) would show low sodium, low chloride, and high potassium.

5. **E: Hungry bone syndrome.** Profound and protracted hypocalcemia is the rule following total parathyroidectomy in patients who have severe secondary hyperparathyroidism for a prolonged period of time. A positive Chvostek sign and, at times, carpopedal spasms can occur days, weeks, or even months postoperatively. These patients need large amounts of calcium supplements (3-4 g of Ca carbonate TID or QID) and usually vitamin D in the form of either colecalciferol or ergocalciferol (although this is controversial) until normocalcemia is restored, thus preventing symptoms of hypocalcemia.