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Acid-Base Balance-Electrolyte Quiz – Case 35

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A 22-year-old woman was admitted to our clinic with persistent hypokalemia (serum potassium ranged between 2.2–2.8 mEq/L). Her medical and family history was unremarkable. However, she complained for polyuria and nocturia. Body weight was 62 kg and BP 100/60 mmHg. Laboratory investigation showed serum potassium 2.8 mEq/L, magnesium 1.1 mEq/L, calcium 8.6 mg/dL, urea 45 mg/dL, creatinine 0.8 mg/dL, and arterial pH 7.52 with bicarbonate 29 mEq/L. In a random urine sample: Na^+ 90 mEq/L, K^+ 80 mEq/L, Cl^- 110 mEq/L, Mg^{2+} 10 mEq/L, while the 24h urine calcium excretion was 80 mg.

Which is the most possible diagnosis?

a. Laxative abuse

b. Diuretic abuse

c. Chronic vomiting

d. Gitelman syndrome

Comment

The patient developed hypokalemia associated with inappropriate kaliuresis (serum potassium in a random urine sample >20 mEq/L, FEK^+ 18%), mild hypomagnesaemia (with inappropriate magnesiuria), metabolic alkalosis and low blood pressure.

Laxative abuse can be excluded, since it is commonly associated with metabolic acidosis and low urinary chloride levels (due to extracellular volume depletion). Chronic vomiting is also associated with low urinary chloride levels. It should be emphasized that low urinary chloride levels are a reliable index of volume depletion in patients with metabolic alkalosis.

Diuretic abuse can explain the patient's clinical and laboratory findings. However, the patient denied diuretic abuse. Thus, the patient who also exhibited hypocalciuria (24 hour urine calcium <100 mg) possibly had Gitelman syndrome. Treatment of the patient demands life-long administration of potassium and magnesium combined with spironolactone or eplerenone or even amiloride.

Even though the prognosis of the disease is excellent, there are three potential complications that should taken into account: A small risk of renal failure mediated by the volume depletion or the effects of aldosterone on renal tissue, hypokalemia and hypomagnesaemia-mediated cardiac arrhythmias and QT prolongation, as well as chronic hypomagnesaemia-mediated chondrocalcinosis.

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Diagnosis: *Gitelman syndrome*