

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Acid-Base Balance-Electrolyte Quiz – Case 41

A 59-year-old male with a past history of diabetes mellitus, coronary heart disease and hypertension presented to the emergency department with acute muscle weakness of the lower legs. He was treated with aspirin, clopidogrel, telmisartan+hydrochlorothiazide (80+12.5 mg per day), spironolactone (25 mg daily), carvedilol (12.5 mg×2 per day), metformin (1 g×2 daily) and insulin. He also reported that he had taken diclofenac (150 mg per day) the last week for increased back pain. His baseline potassium and creatinine levels were 5.2 mEq/L and 1.7 mg/dL, respectively. On admission, laboratory investigation showed glucose 200 mg/dL, creatinine 2.7 mg/dL, urea 110 mg/dL, potassium 7.6 mEq/L and bicarbonate 15 mEq/L.

The patient's hyperkalemia is mainly due to the:

- a. Acute deterioration of renal function
- b. Hypobicarbonemia
- c. Administration of diclofenac
- d. Hyperglycemia

Comment

The patient presented with acute symptomatic hyperkalemia

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ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2014, 31(2):244

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mainly due to the addition of diclofenac, which is known to inhibit prostaglandin synthesis leading to hyporeninemic hypoaldosteronism. Furthermore, these drugs (NSAIDs) can lead to reversible renal ischemia and renal insufficiency, especially in hypovolemic patients, as it was the case in our patient with uncontrolled diabetes mellitus on hydrochlorothiazide. Even though the NSAIDs-induced increase in potassium levels is mild in patients with normal renal function, it may be much higher when renal insufficiency is superimposed, as well as in patients who are also taking other drugs affecting potassium homeostasis, such as β-blockers, angiotensin-converting enzyme inhibitors or sartans and K⁺ sparing diuretics, as it was the case in our patient. The mild hyperkalemia- and renal insufficiency-induced metabolic acidosis, evidenced by the low bicarbonate levels, and the insulin deficiency and hyperglycemia may have also played a role in the development of hyperkalemia, since they can result in potassium movement out of the cells, but they cannot explain the severe life-threatening hyperkalemia of the patient.

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Answer: Administration of diclofenac