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Acid-base Balance-Electrolytes Quiz – Case 2

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A 62-year-old patient with type 2 diabetes mellitus and hypertension was treated was amlodipine (5 mg/day) and ramipril (10 mg/day). The patient was prescribed cotrimoxazole for cystitis (800 mg sulphomethoxazole + 160 mg trimethoprim × 2/day). Two days later the patient experienced muscle weakness and was transferred to the hospital.

The clinical syndrome is related to:

a) Renal failure

b) Drug-induced hyperkalemia

c) Metabolic acidosis

d) Cotrimoxazole-induced side effects.

Comment

The patient developed hyperkalemia-induced muscle weakness. The most common cause of hyperkalemia in the every day clinical practice is the administration of combination of drugs affecting potassium homeostasis. In this case both ramipril (ACE inhibitor) and trimethoprim can reduce potassium excretion. In this setting it should be emphasized that trimethoprim acts like amiloride in the collecting tubules, by directly closing the Na^+ channels in the luminal membrane of the collecting tubular cells. As a result trimethoprim can produce hyperkalemia, particularly in patients with renal failure or taking potassium supplements.

Decreased renal function may also play a role in the development of severe life-threatening hyperkalemia. It is worth mentioning that diabetic patients may exhibit the syndrome of hyporeninemic hypoaldosteronism and mild asymptomatic hyperkalemia.

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Diagnosis: *Drug-induced hyperkalemia*