CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Acid-Base Balance-Electrolyte Quiz – Case 36

A 62-year-old person treated with liposomal amphotericin B was found to have hyperphosphatemia (serum phosphate 6.1 mg/dL, baseline values 3.6 mg/dL) on the 7th day of hospitalization. Laboratory investigation including parameters of renal function, serum electrolytes, uric acid levels, as well as PTH and vitamin D levels was normal.

The patient did not develop symptoms or signs indicative of hyperphosphatemia.

Hyperphosphatemia is due to:

- a. Increased dietary phosphate intake
- b. Pseudohyperphosphatemia
- c. Decreased phosphate excretion
- d. Increased cellular destruction

Comment

Since the patient did not exhibit symptoms/signs suggestive

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of hyperphosphatemia, while the other common causes of hyperphosphatemia were ruled out by the appropriate clinical-laboratory investigation (normal renal function, as well as normal serum calcium, PTH and vitamin D levels), no increased phosphate intake (f.e. with phosphate-containing enemas or with laxatives) was reported and no clinical laboratory findings of increased cellular destruction are mentioned, the diagnosis of pseudohyperphosphatemia was suspected. In fact, it has been reported that liposomal amphotericin B is a cause of interference with the phosphate assay, resulting in falsely increased phosphate levels. Other common causes of hyperphosphatemia include hyperlipidemia, paraproteinemia, hyperbilirubinemia and hemolysis.

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