Severe Hypophosphatemia in a 79-Year-Old Man

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CASE DESCRIPTION

A 79-year-old white man with a history of progressive bone pain was admitted for evaluation 3 years ago. The patient reported that the pain began in both feet and gradually spread to the rest of the body over a 2-year period. Further assessment revealed multiple stress fractures in the feet. Bone mineral density test indicated osteopenia. Serial bone mineral density tests during the 2 years showed that his osteopenia was progressing. Results of several serum protein electrophoreses were essentially normal. His medical history, which included surgical removal of a cerebral aneurysm near the sella turcica and bacterial meningitis, suggested no pertinent etiologic factors. Physical examination revealed no clinically significant findings except unsteady gait. His serum phosphate concentrations declined from 2.5 to 1.8 mg/dL over the 2 years before admission. Laboratory test results at the time of admission are summarized in Table 1. The patient's phosphate concentration reached a nadir of 1.2 mg/dL at admission. His serum alkaline phosphatase was increased. Other notable abnormalities included low normal calcium, normal to borderline high parathyroid hormone (PTH),[2] and increased 24-h urine phosphate and calcium excretion. Other routine biochemical parameters [including ionized calcium, thyroid-stimulating hormone, and free thyroxine (T_4)] were normal. The patient underwent a whole-body scan showing multiple bone lesions. Diagnostic imaging studies including x-ray, a computer axial tomography (CAT), and MRI of the lungs, abdomen, and pelvis were reported to be normal at admission.

Based on the above findings, the patient was diagnosed with osteomalacia secondary to hypophosphatemia. He was then treated with several medications, including bisphosphonates, calcitriol, vitamin D, and calcium and phosphorus supplements. Vitamin D was given because his initial calcium and phosphate concentrations were low, and it was discontinued later following improvement of serum calcium concentrations. Despite substantial phosphorus replacement (250 mg phosphorus per tablet, 2 tablets 3 times a day), the hypophosphatemia continued, along with bone fragility and muscle weakness over the next 3 years, and his height decreased from 173 to 163 cm.

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Table 1. Patient's laboratory results at time of admission.

Test	Result	Reference interval
Phosphate, mg/dL	1.2	2.5–4.5
Calcium, mg/dL	8.5	8.4–10.2
Ionized calcium, mmol/L	1.24	1.13–1.32
Magnesium, mg/dL	2.4	1.8–2.9
Alkaline phosphatase, IU/L	560	38–126
PTH, pg/mL	75	10–65
25-Hydroxyvitamin D, ng/mL	33	9–54
1,25-Dihydroxyvitamin D, pg/mL	42	15–60
Urinary phosphate, g/24 h	1.5	0.3–1.3
Urinary calcium, mg/24 h	1026	50–150
Urea nitrogen, mg/dL	20	8–20
Creatinine, mg/dL	0.9	0.8–1.5
Uric acid, mg/dL	3.3	2.6–7.1
Glucose, mg/dL	88	70–110
Total protein, g/dL	7.6	6.0–8.2
Albumin, g/dL	4.2	3.5–4.7

Questions to Consider
• What are the common causes of chronic hypophosphatemia?
• What are the possible explanations for hypophosphatemia with hyperparathyroidism and relatively low calcium but normal vitamin D concentrations?
• Which laboratory tests are useful in the evaluation of hypophosphatemia and specifically for the diagnosis of this case?

Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the July 2014 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol60/issue7 and follow the link to the Clinical Case Study and Commentaries.

Educational Centers

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