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Case report

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Severe hyperphosphatemia & hypocalcaemia in a lately discovered end stage renal disease

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El Madhoun I,[1] Emam A[2]
[1]Consultant Nephrologist, Al Wakra Hospital, Qatar
[2]Nephrology Specialist, Al Wakra Hospital, Qatar

Correspondence: Ihab El Madhoun, Consultant Nephrologist, Renal Unit, Al Wakra Hospital, PO Box 82228, Qatar, Tel 974 40114235, Fax 974 40114100, Email ielmadhoun@hamad.qa

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Introduction

Chronic kidney disease is a major cause of hyperphosphatemia. Hyperphosphatemia usually cause mild symptoms unless accompanied by hypocalcaemia where symptoms can include muscle cramps or spasms, numbness and tingling around the mouth, bone and joint pain, weak bones, rash and itchy skin. Here we present a case with record high phosphate level and hypocalcaemia.

Case description

A 33-year-old male with no significant past medical history presented with vomiting for one week associated with generalized body ache. Family history was negative. On examination patient was a febrile, BP: 165/75mmHg, O_2 saturation: 98% on room air. Patient was pale with earthy look however; he was conscious, alert, oriented, not distressed or dyspneic with fair general condition. Blood test showed Creatinine 2041 umol/l, Urea 32 mmol/l, Bicarbonate 6 mmol/l, pH 7.09 Haemoglobin 6.7gm/dl, corrected calcium 1.2 mmol/l, phosphorus level above the upper detection limit (later confirmed> 16 mmol/l) which was repeated three times and PTH 645 pg/ml. Ultrasound showed bilateral small shrunken kidneys with lost cortico medullary differentiation consistent with chronic kidney disease (CKD).

Management

Patient was admitted to ICU mainly for severe electrolyte disturbance after receiving stat dose of IV Ca gluconate. A slow haemodialysis session was started with special custom made unusual high Ca bath of 2 mmol for 2 hours. Two units packed RBCs transfused during dialysis Corrected Ca level improved to 2.0 mmol/l, pH 7.25 and phosphorus came down to 4 mmol/l. Oral Calcium carbonate, IV Alfacalcidol & Darbepoetin alfa were started. Patient received further dialysis treatment slowly to address slow correction of uraemia. His symptoms recovered fully. He was discharged after few days with haemoglobin9.1gm/dl, corrected calcium 2.26 mmol/l, phosphorus 0.88 mmol/l.

Discussion

Hyperphosphatemia can occur when the kidney function is impaired to the extent that reduced renal phosphate excretion and other homoeostatic mechanisms fail to eliminate excess phosphate.[1] The paradigm for phosphate regulation changed dramatically with the discovery of bone-derived hormone fibroblast growth factor 23 (FGF23), which allows bone to interact with other organ systems involved in the regulation of mineral homeostasis. FGF23 inhibits production of 1, 25-vitamin D, renal phosphate reabsorption and secretion of PTH.[2] n CKD, the decline in the glomerular filtration rate (GFR) is compensated for by an early elevation of the FGF-23 concentration to decrease proximal tubule phosphate re-absorption and attempt to maintain normal phosphate concentrations.[3] FGF-23 also reduces the concentration of 1α, $25(OH)_2D_3$, lowering the effects of sodium dependent phosphate transport protein, NaPi co-transporters in the intestine and consequently reducing phosphate absorption. The reduced concentration of 1α, $25(OH)_2D_3$ leads to a reduction of calcium absorption and stimulates PTH secretion leading to secondary hyperparathyroidism (SHPT). PTH increases phosphate loss through the kidney by reducing the number of NaPi co-transporters.[4]

The above mentioned compensatory mechanisms attempt to normalise serum phosphate and calcium concentrations in CKD patients. However, as GFR continues to decline and falls below 25 ml/min, the renal phosphate excretion reaches its maximum and excess dietary phosphate accumulates leading to persistent hyperphosphatemia.[5] To our knowledge this patient presented with the highest phosphorous level ever recorded in literatures above the upper detection limit (more than 16 mmol/liter) in addition to very low Calcium level as well (corrected calcium 1.2 mmol/l) despite fair general condition & almost symptomless presentation.

Learning points

1. This is highest ever reported case of hyperphosphatemia level.
2. Cautious tailored dialysis for cases with severe hypocalcaemia & metabolic acidosis is mandatory part in the management of such cases as correction of acidosis with ignorance of hypocalcemia may aggravate hypocalcemia & leads to tetany & convulsion.

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None.

Conflicts of interest

None.

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