Severe Hyperphosphatemia After Phosphate Containing Bowel Cleansing Regimen

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Phosphate- based preparations are commonly used as bowel cleansing regimens (1). Serious metabolic complications have been described in elderly patients, in infants and patients with abnormal renal function, or gastrointestinal disorders that prolong transit time (2). Although clinically insignificant, hyperphosphatemia and hypocalcemia were reported after administration of phosphate containing preparations (2,3).

A rare case of severe hyperphosphatemia, hypocalcemia and metabolic acidosis developed following bowel cleansing with phosphate containing bowel regimen for intravenous pyelogaphy (IVP) is presented.

Case Report

A three and a half months old, male infant who had irritability five hours after taking 2 ml. of oral fleet phospho-soda was admitted to the hospital. He was given 5 ml. of fleet phospho-soda which contains 2.4 grams of monobasic and 0.9 gram of dibasic sodium phosphate. His history revealed recurrent urinary tract infections. He was under evaluation for renal pathology because of young age and male gender. His renal ultrasound (US) did not show any abnormality and family history was insignificant. He was very irritable during physical examination. He did not have bowel movement following oral phospho-soda intake. Saline enema was tried but he did not respond to it. Fluid was seen between segments of ileum on his abdominal US. His clinical status was getting worse. He did not have spontaneous eye movement but reacting to pain by openning his eye. Pupil reaction to light and corneal reflex were positive. His posture was tonic but did not have any convulsion. Blood gas analysis showed metabolic acidosis as pH and HCO_3 values are 7.2 and 16.9 mEq/L, respectively. Phosphorus and calcium values were 28.9 mg/dL and 3.8 mg/dL, respectively. Hyperphosphatemia was possibly due to intake of phosphorus containing bowel regimen. Calcium was administered (1ml/kg I.V.) immediately to correct hypocalcaemia. His clinical status improved dramatically following calcium administration. In addition, $NaHCO_3$ was given in a dose of 1 mEq/kg to correct his metabolic acidosis. His diuresis was maintained by intravenous furosemid (3 mg/kg/day) and he was hydrated intravenous fluid replacement (3000ml/m^2/day). His clinical status returned to normal in an hour whereas blood screen within 24 hours. His blood parathormon level was within the normal levels and his renal tubular function was normal. He was discharged to home on his 5th day of admission. He has been followed up for two and a half years and he is considered as a normal child.

Discussion

Phosphate homeostasis is regulated by intracellular movement of phosphorus and excretion by kidneys. In adults, 60-65% of phosphorus in diet is absorbed. In children, however, it is up to 90% of diet phosphorus that is absorbed . Increased bone phosphorus deposition in children can decrease the excretion of phosphorus, which also alters the equilibrium of phosphorus (4).

Elevation of serum 1,25 $(OH)_2D_3$ activates intestinal absorption and bone immobilization of calcium and phosphorus (4). Our patient was taking prophylactic vitamin D, which might ease the development of hyperphosphatemia. Hyperphosphatemia was reported to occur more easily in gastrointestinal tract diseases (2). Our case had transient abdominal distension and delayed bowel movement, which might cause more than usual intestinal absorption of phosphorus. Volume expansion decreases absorption of phosphorus while increasing its excretion from proximal tubule and volume depletion has an opposite effect (4). The patient presented here was not fed anything before the procedure and he might have mild dehydration which might cause an increase in the absorption of phosphorus from the gastrointestinal tract.

Genetic studies show Type 1 and 2 Na-P co-transporter localized on 5th and 6th chromosomes. Type 2 is responsible for physiological and pathological Na related phosphorus absorption (4). Genetic studies were not undertaken for our patient; however, genetic predisposition should also be taken into consideration.

Hyperphosphatemia has been shown to happen occasionally in all age groups in several studies. Deaths were reported due to hyperphosphatemia (2,5). These agents are in wide use in emergency care units for acute constipation in children (6). This process has very serious effects or can be even fatal in the existence of

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gastrointestinal or renal pathology in children (6), in elderly (1,2) and adults (5). Hyperphosphatemia has been reported to occur also in children and healthy individuals, following phosphorus containing agent intake (3,7). Gremse et al. experienced asymptomatic hyperphosphatemia in 19 children aged 3 to17, who recieved two doses (45 ml/1.7 m^2) of oral sodium phosphate (7). The case presented here developed serious metabolic acidosis with 2 ml. of oral sodium phosphate. Eventhough some studies show asymptomatic hyperphosphatemia in children (7), we experienced very serious complication of hyperphosphatemia in an infant.

Conclusion

In conclusion, widely used phosphorus containing bowel regimen can cause serious hyperphosphatemia in infants. When needed in infants , it should be used cautiously with very close follow-up for the risk of developing hyperphosphatemia and renal insufficiency.

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