

Hypophosphataemia

Pages with reference to book, From 199 To 200

Fatema Jawad (Sughrabai Millwalla Hospital, Karachi.)

Phosphorus, an important element found in the skeletal system and the soft tissues of the human body, plays an integral role in maintaining the normal functions of all the systems. The skeleton contains 85 percent of the total body phosphorus and 15 percent is distributed in the rest of the tissues. This element is the major intracellular anion found in proteins, intermediary carbohydrates and lipids. The dietary source of Phosphorus is from milk, milk products, eggs, fish, meat and nuts. Its absorption takes place by diffusion in the G. I. Tract (Juan et al., 1976), and the excretion is through the kidneys where 85 to 90 percent of all the filtered phosphorus is reabsorbed (Dennis et al., 1979). Vitamin D metabolites also affect the renal handling of Phosphorus (Popovtzer et al., 1977). A fall in the serum phosphorus levels stimulates the production of 1,25 dehydroxy vitamin D₃ in the kidney which in turn causes more absorption of Phosphorus in the small intestine.

Hypophosphataemia is caused either by increased losses in the urine, decreased absorption in the intestine or a shift of the phosphorus from the intra-cellular to the extra-cellular compartments.

Excessive loss in the urine is met with-in conditions of alkalosis, hyperparathyroidism, Vitamin D deficiency, malabsorption syndromes, diuretic therapy and lactic and diabetic acidosis. A fall in the intestinal absorption occurs in patients taking aluminium hydroxide, magnesium hydroxide or aluminium carbonate, in malabsorption syndromes, vitamin D deficiency, nasogastric-suction and vomiting and diarrhoea. The cellular shift of Phosphorus is encountered in administration of glucose, fructose, sodium lactate, epinephrine, insulin and androgens.

Often multiple factors cause hypophosphataemia. Coexisting diabetic ketoacidosis and alcoholism cause hypophosphataemia as a consequence of hyperglycaemia, ketonaemia, lactic acidosis, hyperglucagonaemia and institution of insulin therapy.

The depletion of Phosphorus in the body gives numerous signs and symptoms affecting the various systems and metabolisms. The neurological features include tremors, ataxia, hypore-flexia, parasthesia in the mouth and extremities, slurred speech and sometimes seizures and coma. The exact mechanism how hypophosphataemia leads to this is unknown. But it is postulated that a fall in diphosphoglyceric acid in the erythrocytes leads to tissue hypoxia causing a deficiency of adenosine triphosphate which in turn causes the manifestation of the neurological signs and symptoms (Lichtman et al., 1971; Travis et al., 1971).

A disturbance in the erythrocyte function and even haemolysis can be a consequence of low-phosphate levels in the blood. Diphosphoglyceric acid in the erythrocyte facilitates the release of oxygen from oxyhaemoglobin. This process is upset by a deficiency of phosphorus which in turn depletes oxygen causing tissue hypoxia. Along with this, changes in the erythrocyte morphology are also noted (Travis et al., 1971). Ech-inocytosis, Spherocytosis and microcytosis result in increased rigidity of the red cells which can eventually cause haemolysis (Jacob and Amsden, 1971).

The leucocytes activity is also influenced by the blood phosphate levels. Hypophosphataemia affects the synthesis of phosphoinositides and other phosphate compounds (Craddock et al., 1974). This depresses chemotaxis and phagocytosis and lowers the bactericidal activity of the white cells.

A deterioration of myocardial contractility (O'Connor et al., 1977) and cardiomyopathy with severe myocardial dysfunction (Darsee and Nutter, 1978) has been reported in patients with hypophosphataemia. The repletion of phosphorus in all cases normalises the disorder.

Osteopenia caused by phosphate lack leads to osteomalacia, rickets, pseudofractures, joint stiffness and arthralgia. This is due to decreased calcification of the osteoid and accelerated rate of bone resorption (Ivey et al., 1978).

Impaired glucose metabolism has been associated with hypophosphataemia (DeFronzo and Lang, 1980; Lindall et al., 1971). Diabetic angiopathies have been attributed to reduced erythrocyte diphosphoglyceric acid and impaired tissue oxygenation (Ditzel, 1979).

Acid-base disturbances are said to be caused by low phosphate blood levels. Mild acidosis with reduced serum bicarbonate has been reported (Ljunghall et al., 1979; Massry, 1977). The distal tubule in the kidney contributes to this by failing to reabsorb glucose and bicarbonate. This is caused by the direct effect of phosphorus depletion on adenosine nucleotide in the kidney cells.

Severe hypophosphataemia with serum phosphate levels of less than 1 mg per deciliter, needs phosphate replacement. The oral route is not so effective as diarrhoea ensues leading to malabsorption. Parenteral preparations are recommended in a dose of 2.5 mg per kilogram body weight in asymptomatic patients. If the individual has symptoms, the initial dose is increased by 25 to 50 percent. If hypercalcaemia co-exists, the dose is decreased by 25 to 50 percent. All commercial parenteral solutions are hypertonic and should be diluted. They are either sodium or potassium salts so should be used with care in patients with hypertension, renal insufficiency and heart disease. In conditions as diabetic ketoacidosis, hyperalimentation, haemodialysis and after cardiac surgery, prevention of hypophosphataemia by supplementary phosphates is advisable (Ditzel and Anderson, 1973; Keller and Berger, 1980; Kreisberg, 1978; Young et al., 1973).

Serum phosphorus levels should be closely monitored in patients receiving parenteral phosphate therapy. If hypocalcaemia is present, calcium salts should be administered. The dangers encountered in parenteral phosphate therapy are hypocalcaemia, metastatic calcification, hypotension, hypernatraemia and hyperkalemia. In patients with renal insufficiency, acute renal failure with hypotension and hypocalcaemia may ensue (Shackney and Hasson, 1967).

The various causes of hypophosphataemia along with the clinical and metabolic consequences must be kept in view while treating a patient with this problem. A close monitoring is absolutely necessary to prevent complications.

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