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TO DETERMINE THE INCIDENCE OF HYPOPHOSPHATEMIA IN CRITICALLY ILL PATIENTS

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Abstract:
Objective: Phosphorus plays an important role in metabolism as an intracellular anion, an enzyme component and a component of phosphorylated intermediates, and phosphorus is a component of cell membranes, nucleic acids and nuclear proteins. Hypophosphatemia is the most neglected electrolyte deficiency in our medical practice. Some clinical conditions and medications can cause hypophosphatemia. Transient hypophosphatemia rarely causes symptoms, but severe hypophosphatemia can contribute to increased morbidity and mortality in serious patients. The aim of this study was to determine the prevalence of hypophosphatemia among critically ill patients.
Place and Duration: In the Medicine Unit of ICU of Bahawal Victoria Hospital (BVH) Bahawalpur for one year duration from March 2019 to February 2020.
Material and Methods: 50 critical patients were admitted to the medical and intensive care unit within 12 months. The average age was 49.5 ± 17.4. Thirty-one (62%) patients were men and 19 (38%) were women. At admission, 16 patients (16%) had hypophosphatemia (<2.5 mg / dl) and eighty-four (84%) had normal phosphate or hyperphosphataemia. The average phosphate concentration was 3.27 ± 0.79 mg / dl (range 1.8 to 5.1 mg / dl). The occurrence of hypophosphatemia can be compared with international studies.
Results: In our study, an important clinical diagnosis of hypophosphatemia was diabetic ketoacidosis (16%), respiratory alkalosis (18%), sepsis (32%), chronic obstructive pulmonary disease (32%), respiratory failure (42%) and cirrhotic coma. (38%), renal failure (10%). The incidence of hypophosphatemia was higher in patients with breathing difficulties (p = 0.047). Patients receiving the Ss2 agonist had low serum phosphate levels, although not statistically significant, while patients with renal failure had higher serum phosphate levels. The incidence of hypophosphatemia was higher in patients with more than one disease. Mortality was higher in patients with hypophosphatemic sepsis (32%). In 60% of patients, 19 (38%) had mechanical ventilation, hypophosphatemia, hepatic coma and respiratory alkalosis. Serum calcium and albumin levels were low in patients with hypophosphatemia. No serious hypophosphatemia (<1.5 mg / dl) was observed in this study. Hemoglobin levels were not significantly changed below serum phosphate levels.
Conclusion: The incidence of hypophosphatemia was higher in patients with dyspnea. Hypophosphatemia was 15% in critically ill patients admitted to medical departments and intensive care. Hypophosphatemia is more susceptible to development in patients with sepsis, respiratory failure and chronic obstructive pulmonary disease.
Key words: hypophosphatemia, phosphorus, critical patients.

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INTRODUCTION:

Although monitoring of sodium, potassium and calcium ions is part of the routine of intensive care, little attention has been paid to phosphorus during critical observation of patients[1-2]. Phosphorus plays an important role in metabolism as an intracellular anion, an enzyme component and a component of phosphorylated intermediates, and phosphorus is a component of cell membranes, nucleic acids and nuclear proteins. In the jejunum, it is absorbed in larger proportions, especially by passive transport[3]. Excretion is mainly produced by the kidneys, so 80% of phosphorus is passively reabsorbed into the proximal tubule, sodium bound and regulated by hormonal action and food intake. About 1% of the body's total phosphorus reserve is in the blood, and phosphorus is mainly stored in mineralized tissues (bones and teeth) [4]. Phosphorus is also found in soft tissues in the form of phospholipids, phosphoglycides and phosphoproteins. As with calcium, phosphorus homeostasis involves direct involvement of the intestine, bones and kidneys protected by vitamin D (1, 25-dihydroxycholecalciferol), parathyroid hormone (PTH) and calcitonin. Vitamin D works in the intestine, increasing the absorption of calcium and phosphorus. However, hyphosphatemia is practically controlled by the kidneys[5-6]. Stimulated by lowering serum calcium, PTH secretion acts on renal reabsorption, reduces urinary calcium excretion, and prevents phosphorus reabsorption in the tubules. Conversely, when calcium levels are high, calcitonin is released, works in the opposite direction, that is, reduces phosphaturia and increases calcium excretion. Normal serum phosphate levels range from 2.8 to 4.5 mg / dl (0.4-1.8 mmol / l) [7-8]. Serum levels less than 2.5 mg / dl represent hypophosphatemia. Severe underdevelopment is a serum level equal to or lower than 1.5 mg / dL (0-48 mmol / L). Most phosphorus is absorbed in the duodenum and jejunum. The amount of net phosphate absorbed from the gastrointestinal tract is about 600-700 mg / day. Almost 80% of the filtered load is reabsorbed through the proximal tubule, 10% in the distal nephron, and the rest in the urine. Clinical features of hypophosphatemia include irritability, confusion, convulsions, coma, and neuromuscular disorders. Rhabdomyolysis, erythrocyte dysfunction and hemolysis, leukocyte and platelet dysfunction, changes in liver enzymes and myocardial muscular disorders, as well as respiratory muscle weakness and multi-organ failure[9-10]. This is associated with increased morbidity and mortality in hospitalized patients. In our case, the study was designed to investigate the incidence of hypophosphatemia in serious patients because phosphate abnormalities were also neglected.

MATERIALS AND METHODS:

This study was held in the Medicine Unit of ICU of Bahawal Victoria Hospital (BVH) Bahawalpur for one year duration from March 2019 to February 2020.

Fifty patients were enrolled in this study. All patients over the age of 16 reported to an outpatient, emergency and as well as to the medical department. In our study, normal phosphate serum levels were 2.5 to 4.5 mg / dL. Levels below 2.5 mg / dL were taken as hypophosphatemia. Severe hypophosphatemia, PO4 levels were taken as <1.5 mg / dL. To identify any decrease in serum phosphate levels, serum phosphate levels were checked on the first, third and tenth day (if any) during the patient's stay. The lowest serum phosphate level was used for the analysis. Serum phosphate was measured using a Dade Dimension automated chemical analyzer. A set of flexible reagents was used. Serum calcium was measured on the same analyzer. Serum albumin was measured manually in LAB.

Statistical analysis: All information collected from the form was entered into SPSS computer software version 16 and analyzed using a statistical program. Descriptive statistics were calculated. Quantitative age variables are presented as mean and standard deviation. Qualitative variables such as gender, phosphate status and hypophosphatemia are presented in the tables with frequency and percentage.

RESULTS:

50 patients of both sexes were included in the study. The mean age of patients was 49.5 ± 17.4 years, and the age range was 16-90 years. Sixty-two percent of men, 38% are women with a male to female ratio of 1.63: 1 (Table 1).

Table 1: Frequency distribution of demographic variables of patients (n=50)

Sex	**Frequency**	**Percentage**
Male	31	62.0
Female	19	38.0
PO4 mg/dl level (age range in years)		
	Age <40 years	Age 40-90 years
PO4 = <2.5	3 (6%)	9 (18%)
PO4 = >2.5	15 (30%)	23 (46%)

The mean serum phosphate concentration was 3.27 ± 0.79 mg / dl (range 1.8 to 5.1 mg / dl). Eight patients (16%) had diabetic ketoacidosis, 21 (42%) had respiratory failure, 16 (32%) had COAD / asthma, 16 (32%) had sepsis, 5 (10%) in 18% of the system respiratory tract malignant alkalosis, in 19 (38%) hepatic coma, in 5 (10%) renal failure. Disease groups had hypophosphatemia in 2 (25%) patients with CAD, 1 (11%) hepatic coma, 4 (25%) patients with sepsis, 1 (11%) patients with respiratory alkalosis, 2 (12%) have COPD patients, 4 (19%) with respiratory failure, 1 (20%) with renal failure, 20% had hypophosphatemia and malignancies (Table 2).

Table 2: Frequency of Prevalence of Hypophosphatemia in different Diseases

Disease Group	**=n**	**Hypophosphatemia**
DKA	8(16.0%)	2(25.0%)
Hepatic coma	19(38.0%)	1(11.0%)
Septicemia	16(32.0%)	4(25.0%)
Resp. Aklalosis	09(18.0%)	1(11.0%)
COPD	16(32.0%)	2(12.0%)
Resp. failure	21(42.0%)	4(19.0%)
Renal failure	05(10.0%)	1(20.0%)
Malignancy	05(10.0)	1(20.0%)

During hospitalization, 30 (60%) patients received antacids, 20 (40%) received ß2 receptor agonist (salbutamol / aminophylline), 16 (32%) patients received complete parenteral nutrition, 26 (52%) glucose infusion. Steroids were given to 14 (28%) patients, and diuretics were given to 10 (20%) patients (Table 3).

Table 3: Frequency Status of Phosphate in Different Treatment Modalities

Treatment Categories	**=n**	**Hypophosphatemia**
Antacids	30(60%)	5(10%)
ß2 Agonist	20(40%)	4(8%)
Ionotrips	16(32%)	3(6%)
Dextrose	26(52%)	4(8%)
Diuretics	10(20%)	1(2%)
Steroids	14(28%)	3(6%)
Total parenteral nutrition	08(16%)	2(4%)

Table 4 shows that 31 (62%) patients had difficulty in breathing and 19 (38%) patients were mechanically ventilated. Gender had no effect on serum phosphate levels.

Table 4: Analysis of Shortness of Breath and Mechanical Ventilation of Hypophosphatemia

Patients with Hypophosphatemia	**SOB**	**=n**	**%age**	**P value**
31 (62%)	Present Absent	27 4	87.0 13.0	<0.05
Mechanical ventilation 19 (38%)	<2.5 >2.5	3 16	16% 84%	<0.05

Analysis of serum phosphate levels with calcium, albumin and hemoglobin in serum is shown in Table 5. Serum calcium and albumin levels were low in patients with hypophosphatemia.

Table 5: Analysis of PO4, Hb, Albumin and Calcium Level

	PO4 (mg/dl) Level	=n	Mean
Hb	<2.5 >2.5	8 42	11.30±2.35 10.81±2.60
Albumin	<2.5 >2.5	8 42	3.51±0.42 3.51±0.58
Calcium	<2.5 >2.5	8 42	7.80±0.59 8.75±0.68

However, low phosphate had no effect on hemoglobin. In 3 (17.65%) of 17 patients who died in the study, serum phosphate levels were less than 2.5 mg / dL. These patients had more than one disease in total, and sepsis was the most common disease, and 14 other patients (82.35%) exceeded the combination of various diseases> 2.5, and the remaining 3 (17.65%) were <2.5. (Table 6).

Table 6: Frequency of Mortality and PO4 Level

Deaths (total)	PO4 Level	=n.	%age
17 (34%)	< 2.5 > 2.5	3 14	17.65 82.35

DISCUSSION:

In the study reported by Geerse, critical patients have a high incidence of hypophosphatemia due to the presence of many causative factors[10-11]. Hypophosphatemia can cause many symptoms, but often remains asymptomatic. However, hypophosphatemia is associated with increased mortality in subgroups of important patients. It is important to investigate whether hypophosphatemia alone causes higher mortality or is associated with a higher severity of the disease[12-13]. In another study reported by Betro and Pain, the incidence of hypophosphatemia increased by 2% to 5% if there was a predisposition such as alcoholism, ketoacidosis, burns or sepsis. In our study, the incidence of hypophosphatemia was 10% with predisposing conditions. Our patients did not have a history of alcoholism. 66 patients (66 women, 3 men) with anorexia nervosa were included in the retrospective analysis by Ornstein to determine the incidence of hypophosphatemia in adolescents with anorexia nervosa. The average age was 15.5 ± 2.5. Four patients (5.8%) had moderate hypophosphatemia (<2.5 mg / dl> 1.0 mg / dl) and fifteen (21.7%) had mild hypophosphatemia (<3.0 mg / dl and> 2.5 mg / dl) [14-15]. The phosphorus rarities were directly proportional to the ideal body weight (p 0.01). In our study, 8 (16%) patients developed moderate hypophosphatemia. In a study by Bollaert to see the hemodynamic and metabolic effects of rapid correction of hypophosphatemia in patients with septic shock, phosphorus was administered after a 22% increase in left ventricular operating speed (p <0.01) and without a 12% increase in systolic blood pressure in our study. Its effects has not been studied. In our study, 3 dead patients had hypophosphatemia with other diseases.

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