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Original Article

Sodium Phosphate Rectal Enema as a Source of Phosphorus in Dialysate: Use in Hypophosphatemic Critically Ill Patients Undergoing Acute Dialysis

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Abstract

Background: Phosphorus disorders, including hypophosphatemia and hyperphosphatemia, are common in critically ill patients with acute kidney injury and contribute to adverse outcomes. Standard dialysis solutions often exacerbate hypophosphatemia, necessitating innovative strategies like phosphorus-enriched dialysate for safer management.
Objective: To evaluate the use of Sodium Phosphate Rectal Enema in preparing phosphorus-enriched dialysate for acute dialysis in critically ill patients with hypophosphatemia.
Study type, settings & duration: This prospective cohort study was done at Intensive Care Unit (ICU), Liaquat National Hospital, Karachi from May 2021 to April 2023.
Methodology: A total of 100 critically ill patients with acute kidney injury, all with serum phosphorus levels below 2.0 mg/dl and undergoing hemodialysis for acute indications, were included in the study. Sodium Phosphate Rectal Enema was added to the bicarbonate component of the dialysate solution, and phosphorus levels in the finalized dialysate (prepared by mixing acetate and bicarbonate solutions) were measured. Serum phosphorus levels were then measured again following the completion of hemodialysis. Serum calcium level pre/post-dialysis and ECG monitoring for arrhythmias were performed to assess safety.
Results: Sodium Phosphate Rectal Enema was used in volumes of either 60 mL or 120 ml. The mean ± standard deviation (SD) pre-hemodialysis serum phosphorus level was 1.77±0.377 mg/dl. The mean±SD post-hemodialysis serum phosphorus level was significantly increased to 3.0±0.909 mg/dl (*p*-value <0.0001). There was an insignificant difference in pre and post hemodialysis serum calcium levels as 8.57±0.27 and 8.51±0.29 mg/dl respectively (*p*-value:0.14689).
Conclusion: Sodium Phosphate Rectal Enema can be effectively used to prepare phosphorus-enriched dialysate, aiding in the correction of hypophosphatemia during dialysis.
Key words: Sodium phosphates, renal dialysis, dialysis solutions, hypophosphatemia, critical Illness.

Introduction

Phosphorous disorders, including hypophosphatemia[1] and hyperphosphatemia,[2] are well-recognized complications of acute kidney injury (AKI) in critically ill patients. Reported incidence of hypophosphatemia in AKI requiring extensive renal replacement therapy is 17.6%.[3]

Phosphorus is a structural component of nucleic acids and cell membranes. It is also found in adenosine triphosphate (ATP), the primary energy-generating molecule essential for cellular metabolism. Alterations in phosphorus levels are a frequently encountered electrolyte imbalance among intensive care unit (ICU) patients with critical illness.[4] Hypophosphatemia holds particular importance due to its association with increased ICU morbidity and mortality.[5]

Since standard dialysate does not contain phosphorus, acute dialysis in the setting of low serum phosphorus can lead to worsening

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hypophosphatemia. Conversely, dialysis using phosphorus-enriched dialysate has been shown to help correct this imbalance. Case reports have demonstrated successful treatment of symptomatic hypophosphatemia using hemodialysis with phosphorus-enriched dialysate solutions.

To prepare phosphorus-enriched dialysate, phosphorus can be added to the bicarbonate component (Bath B) of the dialysate, which is then mixed with the acetate component (Bath A) to form the final dialysate solution. In our study, we used Sodium Phosphate Rectal Enema as a source of phosphorus to prepare phosphorus-enriched dialysate and evaluated its outcomes in the setting of acute dialysis. The primary endpoint of the study was to ensure safe dialysis in hypophosphatemic critically ill patients with AKI. The secondary endpoint was to assess improvement in serum phosphorus levels post-hemodialysis.

Methodology

This prospective cohort study was conducted in the Intensive Care Unit (ICU) of a Liaquat National Hospital, Karachi from May 2021 to April 2023. A sample size of 100 patients was statistically calculated. After taking the informed consent, study included critically ill adult patients (≥18 years) diagnosed with AKI and hypophosphatemia who were undergoing hemodialysis for acute indications. Critically ill patients were defined as those with failure of two or more organ systems. The AKI was diagnosed based on one or more of the Kidney Disease Improving Global Outcomes (KDIGO) criteria including an Increase in serum creatinine by ≥0.3 mg/dl within 48 hours, an increase to ≥1.5 times baseline within 7 days, and/or Urine output <0.5 mL/kg/h for 6 hours. Hypophosphatemia was defined as a serum phosphorus level <2.0 mg/dl, according to the reference range of the local laboratory. Patients younger than 18 years with serum phosphorus levels <1.0 mg/dl or >2.0 mg/dl, or for whom consent could not be obtained were excluded from the study. Data regarding age, sex, nature of illness, and renal replacement therapy were extracted from the electronic medical record (EMR) system by the principal investigator. Hemodialysis was performed using a polysulfone dialyzer of surface area 1.8 m^2, the dialysate flow rate of 500 mL/min, and the blood flow rate and duration of dialysis were individualized according to clinical indications, with an average session length of 3 hours' duration at a blood flow rate of 250 mL/min. Pre-/post-dialysis serum phosphorus levels were measured. To supplement phosphorus, Sodium Phosphate Rectal Enema was added to the bicarbonate component of the dialysate by trained dialysis staff using a graduated bottle for precision. Final dialysate phosphorus levels were measured**.** The volume added was based on pre-dialysis phosphorus levels (60 mL for serum phosphorus 1.6–2.0 mg/dl and 120 mL for serum phosphorus 1.0–1.5 mg/dl).

The electrocardiogram (ECG) monitoring and pre-/post-dialysis serum calcium level were monitored to assess for arrhythmias or post-dialysis hypocalcemia. The absence of either was used as a marker of the safety of Sodium Phosphate Rectal Enema use in this study.

Statistical analysis was performed using SPSS version 25. Continuous variables (age, pre- and post-dialysis phosphorus and calcium levels) were expressed as mean±standard deviation. Categorical variables (gender, co-morbidities such as diabetes, hypertension), admission diagnosis, and indication of dialysis were presented as frequencies and percentages. A paired sample t-test statistic was applied to assess statistical significance. A p-value <0.05 was considered statistically significant.

The ethical approval was obtained from the Ethical Review Committee of Liaquat National Hospital, Karachi vide letter no. 0895-2021-LNH-ERC.

Results

A total of 100 patients were included in the study. Of these, 79 (79%) were male and 21 (21%) were female. The mean age±SD of the patients was 59±1.04 years. All patients were critically ill. Regarding admission diagnosis, 44 (44%) patients were diagnosed as sepsis with multi-organ failure, 27 (27%) had acute coronary syndrome with cardio-circulatory collapse, 16 (16%) had fulminant hepatic failure, and 11 (11%) had acute pancreatitis. 61 (61%) patients had diabetes mellitus (DM) and 66 (66%) had hypertension (HTN). All patients had acute kidney injury (AKI) and underwent hemodialysis for acute indications. Among them, 76 patients received hemodialysis due to metabolic acidosis and hyperkalemia. 18 patients underwent dialysis for uremia, while 6 patients required dialysis due to methanol intoxication (Table-1).

About 120 ml of Sodium Phosphate Rectal Enema was used for 72 patients, and 60 ml for 28 patients. The phosphorus level in finalized dialysate by addition of 60 ml Sodium Phosphate Rectal Enema to the bicarbonate solution was 2.6 mg/dl. The mean±SD pre-hemodialysis serum phosphorus level was 1.77±0.377 mg/dl. The mean±SD post-hemodialysis serum phosphorus level was

significantly increased to 3.0±0.909 mg/dl (*p*-value <0.0001). There was an insignificant difference in pre and post hemodialysis serum calcium levels as 8.57±0.27 and 8.51±0.29 mg/dl respectively (p-value:0.14689) (Table-2).

Table 1: Demographic parameters of patients.

Variables		*N*	*%*
Gender	Male	79	79
	Female	21	21
Age (years)	Mean age ± SD	59±1.04	
Admission diagnosis	Sepsis with multi-organ failure	44	44
	Acute coronary syndrome	27	27
	Fulminant hepatic failure	16	16
	Acute pancreatitis	11	11
Comorbid	Hypertension (HTN)	66	66
	Diabetes Mellitus (DM)	61	61
Indication of Hemodialysis	Metabolic acidosis	76	76
	Hyperkalemia	76	76
	Uremia	18	18
	Methanol intoxication.	6	6

Table 2: Comparison of pre and post serum phosphorus and calcium levels.

Parameters		*Mean±SD*	*p-value*
Serum phosphorous level mg/dl	Pre-hemodialysis	1.77±0.377	<0.0001
	Post-hemodialysis	3.0±0.909	
Serum calcium level (mg/dl)	Pre-hemodialysis	8.57±0.27	0.14689
	Post-hemodialysis	8.51±0.29	

Discussion

Although hyperphosphatemia is more commonly observed phosphorus disorder in AKI, hypophosphatemia is frequently seen in critically ill patients[6]. As in present study, a low phosphorous level was complicating critical illness in the study population. The AKI in hospitalized patients increases mortality by 4-fold.[7] Hypophosphatemia creates a hurdle in the commencement of timely dialysis. Delay in initiation of renal replacement therapy is associated with increased mortality.[8] Uremia and its related complications including pericarditis, volume overload, acid-base disturbances, and electrolyte imbalances are significant concerns in patients with AKI.[9] In our study, acid-base disturbance in the form of metabolic acidosis and electrolyte imbalance in the form of hyperkalemia were the two commonest complications of AKI. According to a comparative analysis in the ICU, patients who had hypophosphatemia upon admission exhibited significantly higher mortality rates compared to those without hypophosphatemia.[10] Despite this, hypophosphatemia is often viewed as a marker of the severity of illness, and the increased mortality is likely attributed to the underlying critical condition itself. Consistent to literature, current study population with critical illness had hypophosphatemia. A study presented 47.3% incidence of hypophosphatemia in patients with sepsis,[11] while other reported 28.8% in surgical patients,[12] and 13% in patients with pneumonia.[13] Likewise, in present hypophosphatemic population, sepsis was the leading cause of multi-organ failure and critical illness. Phosphorus is extensively distributed throughout the body; however, it is the inorganic phosphate that plays a key role in regulating normal physiological functions of the body.[14] Critically ill ICU patients are exposed to several risk factors for hypophosphatemia, including younger age, greater illness severity, low serum albumin levels, mechanical ventilation, refeeding syndrome,[5] continuous renal replacement therapy (CRRT),[10] which leads to increased morbidity and mortality among affected patients.[15] In this study, greater severity of illness was a risk factor for low phosphorous since septicemia with multi-organ failure, acute coronary syndrome with cardiorespiratory collapse, fulminant hepatic failure with multi-system involvement, and acute pancreatitis were the reasons for hospital admission.

Hypophosphatemia is an independent risk factor for respiratory failure leading to tracheostomy formation[3]. Apart from respiratory failure, hypophosphatemia may also cause cardiac arrhythmias, rhabdomyolysis, and hemolytic anemia.[16] Therefore currently arrhythmias was kept as safety marker of this study. Fortunately, no single hypophosphatemic patient in our study developed arrhythmias with phosphorous dialysis. Furthermore, post-dialysis serum calcium measurements showed no significant changes from pre-dialysis serum calcium level, indicating the safety of phosphorus-enriched dialysis using Sodium Phosphate Rectal Enema. Conventional dialysate does not contain phosphorus,[17] making dialysis a contributing factor to hypophosphatemia in ICU patients. This often necessitates oral or intravenous phosphorus replacement before or after dialysis. However, intravenous phosphorus supplementation can be time-consuming, requires vital sign monitoring during infusion,[18] and is not readily available in all hospital pharmacies. Additionally, it contributes to the overall cost of ICU care. We utilized Sodium Phosphate Rectal Enema as a phosphorus source to prepare phosphorus-enriched dialysate. A significant increase in serum

phosphorus levels was observed after hemodialysis, rising from a mean of 1.77 mg/dL to 3.0 mg/dL.

Sodium Phosphate Rectal Enema**,** commonly used to relieve severe constipation,[19] is a cost-effective, easily accessible product. Sodium Phosphate Rectal Enema was repurposed as a phosphorus source for dialysate supplementation. It is simple to use, inexpensive, and widely available.[20] This study achieved a phosphorus concentration of 2.6 mg/dL in the final dialysate after adding 60 mL of Sodium Phosphate Rectal Enema**.**

Based on current observations, Sodium Phosphate Rectal Enema appears to be a safe and effective method for phosphorus-enriched dialysis in critically ill patients with hypophosphatemia, Since the findings have been evaluated on a substantial number of critically ill patients. However, due to a specific group of patients included currently, findings cannot be generalized, including end-stage renal disease patients undergoing chronic dialysis are limitations of this study.

Sodium Phosphate Rectal Enema was found to be a safe option for preparing phosphorus-enriched dialysate for acute hemodialysis in hypophosphatemic critically ill patients. Manual adjustment of phosphorus in the dialysate bath contributed to improvement in serum phosphorus levels post-hemodialysis and correction of hypophosphatemia. However, due to the small sample size, the findings cannot be applied as general practice, and further large-scale studies are recommended.

Conflict of interest: None declared.

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