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Original Research Article

To Assess the Correlation of Serum Phosphorus Level in Diabetic Ketoacidosis in Type-2 Diabetes Mellitus.

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Abstract:

Background: In DKA patients, phosphate and magnesium level are decreased and further reduction may occur during insulin treatment. But, usually, these electrolytes are not given, when the patient takes oral diet. If the phosphate level is low, and the patient is not taking oral diet, potassium phosphate can be given. If magnesium level found low level in DKA patient, who developed cardiac arrhythmias, magnesium sulfate can be given. Otherwise, routine supplementation is not needed.

Methods: Phosphorus levels was estimated on 1 day, 3 day and discharge/worsening of patient. It was correlated to prognosis of patient in diabetic ketoacidosis in type-2 diabetes mellitus.

Result-Phosphorous levels at Day 1, 3 and during discharge or condition worsening in patients with Type 2 diabetes mellitus with ketoacidosis (Study group) Phosphorous levels at Day 1, 3 and during discharge or condition worsening in patients with Type 2 diabetes mellitus without ketoacidosis (Control group)

Conclusion: We conclude that phosphorus the neglected aspect of diabetic ketoacidosis needs due consideration. Significant negative correlation was observed between RBS levels and serum phosphorus levels. Significant long length of hospitalization was observed in diabetic ketoacidosis patients having low phosphorus levels.

Introduction

Phosphorus (P), an essential nutrient, is the sixth most common element in the human body Phosphorus as phosphates are negatively charged ions that are highly reactive with oxygen forming phosphate ions which complex with several ions such as calcium and forms ester linkages with other molecules such as peptides and proteins. The ability of phosphorus to phosphorylate various macromolecules and form complexes with other ions involves phosphorylating kinases as well as dephosphorylating phosphatases [1] in all living systems. Phosphorus's chemical properties makes it one of the most reactive elements. The ability of phosphorus to act on its own as a signaling molecule and/or interact with various elements of the body makes it difficult to identify distinct physiological roles for phosphorus in the human body.

Homeostasis of serum Pi and Ca is a highly integrated system involving specific organ and endocrine modulators. In addition to dietary Pi, several other factors affect serum levels of Pi including specific drugs , genetic makeup, gender and the age of an individual, environmental contaminants, physiological state of the organs involved in Pi homeostasis, other nutrients, growth factors etc. Normally the ratio between Ca and Pi in the serum is 2:1. It is proposed that the changes in the level of either of these ions in the serum will change the ratio and will trigger the homeostatic responses. The ultimate goal of the homeostatic mechanism is to normalize the levels or the ratio Ca : Pi. [2]

In DKA patients, phosphate and magnesium level are decreased and further reduction may occur during insulin treatment. But, usually, these electrolytes are not given, when the patient takes oral diet. If the phosphate level is low, and the patient is not taking oral diet, potassium phosphate can be given. If magnesium level found low level in DKA patient, who developed cardiac arrhythmias, magnesium sulfate can be given. Otherwise, routine supplementation is not needed. [2]

Materials and Methods

Study Design & Period-Cross section study with One year (March 2020 to February 2021)

Place Of Study-JLN Hospital attached to JLN Medical College, AJMER.

Sample Size-Minimum of 100 patients with Type 2 diabetes mellitus with ketoacidosis and 50 patients of type 2 diabetes mellitus without ketoacidosis as control was studied.

Inclusion Criteria-

1. Patients who have given written informed consent.
2. Patients who are >18yrs age.
3. Patients with type 2 diabetes mellitus.

Exclusion Criteria-

1. Patients who have not given written informed consent.
2. Patients with malnutrition, malabsorption syndromes.
3. Patients on diuretics, steroids, phosphate binding antacids.
4. Patients with renal transplantation.
5. Patients with hyperparathyroidism, pancreatitis, burns.
6. Patients who are alcoholics, hyperventilation, panic attacks, salicylate poisoning
7. Patients with Type 1 diabetes mellitus and other causes of ketoacidosis.
8. Patient with any type of calcium abnormalities

Statistical Analysis-Depending upon the data available appropriate statistical test was applied qualitatively by chi-square and quantitatively by t-test. Statistical analysis were carried using standard formulae. Microsoft Excel 2013 and GraphPad Prism 9.0.2 software were used for data entry and analysis.

Methodology- Phosphorus levels was estimated on 1 day, 3 day and discharge/worsening of patient. It was correlated to prognosis of patient in diabetic ketoacidosis in type-2 diabetes mellitus.

Phosphorous levels at Day 1, 3 and during discharge or condition worsening in patients with Type 2 diabetes mellitus with ketoacidosis (Study group)

Phosphorous levels (Study)			
Phosphorous levels	**Number of patients**		
	Day 1	**Day 3**	**Discharge/Worsening**
Normal (2.5-4.5mg/dL)	44	40	56
Mild hypophosphatemia (2-2.5mg/dL)	40	40	30
Moderate hypophosphatemia (1-2mg/dL)	9	17	14
Severe hypophosphatemia (<1mg/dL)	7	3	0

Phosphorous levels at Day 1, 3 and during discharge or condition worsening in patients with Type 2 diabetes mellitus without ketoacidosis (Control group)

Phosphorous levels (Control)			
Phosphorous levels	**Number of patients**		
	Day 1	**Day 3**	**Discharge/Worsening**
Normal (2.5-4.5mg/dL)	26	42	47
Mild hypophosphatemia (2-2.5mg/dL)	17	8	3
Moderate hypophosphatemia (1-2mg/dL)	7	0	0
Severe hypophosphatemia (<1mg/dL)	0	0	0

Discussion

Increasing attention has been focused on hypophosphatemia in diabetic ketoacidosis in recent years. However, the clinical significance of hypophosphatemia and the role of phosphorus therapy in diabetic ketoacidosis remain controversial. **Kitabchi et al** [3] stated that the annual incidence rate for DKA from population-based studies ranges from 4.6 to 8 episodes per 1,000 patients with diabetes. **Becker et al** [4] showed that potassium and phosphorus concentrations decline in parallel after administration of insulin; thus, the use of potassium phosphate solutions in the therapy of diabetic coma has been recommended.

Insulin enhances the reduction in serum phosphorus in both diabetic and non-diabetic person. In severe uncontrolled hyperglycemia, high level of glucose results in low phosphorus levels due to intracellular phosphorylation of glucose and excessive loss in urine, resulting in diabetic osteopenia. [5,6] Therefore normalization of blood glucose leads to an improved capacity of the kidney tubules to reabsorb inorganic phosphorus. Phosphate levels should be monitored in diabetics treated with insulin. When

the level reaches <1mg/dL, it needs replacement as potassium phosphate. [7]

Phosphorus levels were measured in all patients on First day, on day 3, and at the time of clinical deterioration or complication in patients with various diseases in our research. Numerous studies have stressed the need of tracking serum phosphorus levels in DKA. David W Miller et al [8] showed that hypophosphatemia may be seen in anywhere from 20% to 80% of patients who present to emergency department with diabetic ketoacidosis, hyperosmolar hyperglycemic state and sepsis.

Conclusion

We conclude that phosphorus the neglected aspect of diabetic ketoacidosis needs due consideration. Significant negative correlation was observed between RBS levels and serum phosphorus levels. Significant long length of hospitalization was observed in diabetic ketoacidosis patients having low phosphorus levels.

Associated complications were found high in hypophosphatemic group however this observation was found to be statistically significant. So low serum phosphorus level at admission is also a feature of poor outcome. The assessment of serum phosphorous in these patients could be a useful predictor of the clinical outcome in DKA.

References

1. Krebs, E.G. and Beavo, J.A. (1979) Phosphorylation-Dephosphorylation of Enzymes. Annual Review of Biochemistry, Annual Reviews. **48**, 923–59. https://doi.org/10.1146/annurev.bi.48.070179.004423
2. Nelson, S.M.L., Sarabia, S.R.S., Christilaw, E., Ward, E.C., Lynch, S.K., Adams, M.A. et al. (2017) Phosphate-Containing Prescription Medications Contribute to the Daily Phosphate Intake in a Third of Hemodialysis Patients. Journal of Renal Nutrition, Elsevier BV. **27**, 91–6. https://doi.org/10.1053/j.jrn.2016.09.007
3. Kitabchi, A.E. and Nyenwe, E.A. (2006) Hyperglycemic Crises in Diabetes Mellitus: Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State. Endocrinol. Metab. Clin. North Am. https://doi.org/10.1016/j.ecl.2006.09.006
4. Becker, D.J., Brown, D.R., Steranka, B.H. and Drash, A.L. (1983) Phosphate replacement during treatment of diabetic ketosis. Effects on calcium and phosphorus homeostasis. American Journal of Diseases of Children (1960), United States. **137**, 241–6. https://doi.org/10.1001/archpedi.1983.02140290033009
5. Huang, D., Refaat, M., Mohammedi, K., Jayyousi, A., Al Suwaidi, J. and Abi Khalil, C. (2017) Macrovascular Complications in Patients with Diabetes and Prediabetes. Biomed Res. Int. https://doi.org/10.1155/2 017/7839101
6. Tahrani, A.A., Piya, M.K., Kennedy, A. and Barnett, A.H. (2010) Glycaemic control in type 2 diabetes: Targets and new therapies. Pharmacology & Therapeutics, **125**, 328–61. https://doi.org/https://doi.org/10.1016/j.pharmthera.2009.11.001
7. Desfougères, Y., Saiardi, A. and Azevedo, C. (2020) Inorganic polyphosphate in mammals: where's Wally? Biochemical Society Transactions, Portland Press Ltd. **48**, 95–101. https://doi.org/10.1042/BST20190328
8. Miller, D. and Slovis, C. (2000) Hypophosphatemia in the emergency department therapeutics. The American Journal of Emergency Medicine, **18**, 457–61. https://doi.org/10.1053/ajem.2000.7347