



ORIGINAL RESEARCH PAPER

Biochemistry

SERUM PHOSPHOROUS LEVELS: A PROGNOSTIC INDICATOR IN DIABETIC KETOACIDOSIS

KEY WORDS: ketoacidosis, diabetes mellitus, phosphorous.

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ABSTRACT

Diabetic ketoacidosis is one of the most common endocrinal emergency in the world. phosphorous depletion occurs in DKA owing trans-cellular shift and hyperphosphaturia. This study was undertaken to find out serum phosphorous in DKA as a prognostic indicator to assess the mortality and morbidity and determine the outcome.60 consecutive diabetic patients presenting with clinical and biochemical features of DKA admitted in emergency of GMC Jammu from August to November 2018were studied. in the study phosphorous levels at the time of admission can be used as a prognostic indicator in diabetic ketoacidosis

Conclusion: high blood sugar is associated with low phosphate value. Low phosphate value was associated with prolonged hospital stay

INTRODUCTION

Nationally, DKA contributes to approximately 100,000 hospital admissions per year¹ and accounts for 2% to 9% of hospital admissions in persons with diabetes².electrolyte disturbances and especially changes in potassium levels in diabetic ketoacidosis have been an area of interest for many decades now. there is a paucity of studies regarding changes in serum phosphorous levels during diabetic ketoacidosis and its management. Diabetic ketoacidosis is an acute complication of diabetes .diabetic ketoacidosis is characterized by hyperglycemia, ketonemia (ketonuria), metabolic acidosis (high anion gap) along with a number of metabolic dearrangements .It is more common in women from low socio-economic groups. It mainly affects type 1 diabetes patients. Among 4% of all patients with diabetes. 20% of those with type1 diabetes were admitted in hospital with manifestation of diabetes. diabetes ketoacidosis is often precipitated by intercurrent illness such as infection, inadequate insulin administration by the patient common presenting complaints were nausea ,vomiting, polydipsia, polyuria, abdominal pain and altered sensation. parameters related to mortality include duration of prior diabetes, severity of acidosis, severity of peripheral vascular insufficiency and comorbid conditions.³ Phosphate is needed for bone mineralization and cellular structural components (phospholipids, nucleotides,phosphoproteins),for energy storage as ATP , for oxygen transport as 2,3-DPG and for acid base balance(as cellular and urinary buffer).⁴ Other phosphates, such as creatinine phosphate are involved in many energy intensive physiological functions, such as muscular contractility,neurological functions and electrolyte transport dynamic changes in serum phosphorus levels take place during occurrence and management of diabetic acidosis.⁵but data regarding clinical manifestation resulting from these dynamic changes in serum phosphorous levels is very rare.

Phosphate excretion is increased in diabetics especially those with uncontrolled one.⁶ Phosphate depletion is common in diabetic ketoacidosis. Initially intracellular phosphate moves to extracellular compartment due to acidosis, dehydration.so patients with diabetic ketoacidosis can present with hyperphosphatemia i.e increased levels of serum phosphorous. Hyperphosphatemia which is usually asymptomatic can be associated with symptoms which are secondary to hypocalcemia.⁷ during treatment of diabetic ketoacidosis with insulin and intravenous fluids, phosphorous is taken up intracellular with resultant hypophosphatemia. So the study was undertaken to find out mainly the use of serum phosphorous in diabetic ketoacidosis as a prognostic

indicator to assess the mortality and determine the outcome. phosphate depletion is common in DKA. but the initial value may be high ,normal or low. Hypophosphatemia especially moderate and severe is associated with a number of clinical sequelae including neurological , muscular, cardiac respiratory and hemotological problems.⁸

MATERIAL AND METHODS

60 consecutive diabetic patients presenting with clinical and biochemical features of DKA admitted in emergency of medicine department..... from august 2018-nov 2018 were studied. All DKA patients of both type1 and type2 were included in the study and patient treated with phosphate binding drugs and patients receiving phosphate supplements were excluded from the study.5ml of venous blood was collected and serum was separated after centrifugation at 3000rpm for 10 mts. Samples were analyzed for blood glucose by glucokinase⁹ method and phosphorous by Fiske subba row method.¹⁰ patients were grouped into 2 categories according to their admission in serum phosphorous levels. Group 1 including serum phosphorous levels≤ 2.5 mg% and Group 2 including serum phosphorous ≥2.5mg% and the primary end point of this study is duration of stay in the hospital.

Table 1: comparison of admission RBS value with serum phosphorous

Admission RBS	Phosphorous≤ 5mEq/L	Phosphorous >2.5mEq/L	Statistical significant
200 to 300	0	2(6.3%)	Not significant
301 to 400	13(46.5%)	30(60%)	significant
401 to 500	15(72.2%)	18(36%)	significant

There is a statistically significant association between serum phosphorous level at the time of admission and initial blood sugar value of DKA patients.

Table 2: Comparison of hospital stay with phosphorous levels.

Duration of stay in hospital	Phosphorous ≤2.5mEq/L	Phosphorous >2.5mEq/L	Statistical significant
2	2(11.1%)	8(22%)	significant
3	2(5.6%)	16(40%)	significant
4	6(11.1%)	3(10%)	Not significant
5	15(50%)	4(20%)	significant
6	3(22.2%)	1(8%)	Not significant

There is a statistically significant association between duration of the hospital stay and serum phosphorous levels at the time of admissions in DKA patients.

DISCUSSION

Phosphorous is a widely distributed element in the human body. The normal value for phosphorus is 2.5 to 4.5mg/dL¹¹. It is present in both organic and inorganic forms but only serum inorganic phosphate is measured. Inorganic phosphorous in the form of hydroxyapatite in bone plays an important role in structural support of body and also provides phosphate for intracellular and extracellular fluid.¹² Insulin enhances the reduction in serum phosphorous in both diabetic and non-diabetic person in severe uncontrolled hyperglycemia, high levels of glucose results in low phosphorous levels due to intracellular phosphorylation of glucose and excessive loss in urine, resulting in diabetic osteopenia. Therefore normalization of blood glucose leads to an improved capacity of kidney tubules to reabsorb inorganic phosphorous. Phosphate levels should be monitored in diabetics with insulin.¹³ When the level reaches <1mg/dL, it needs replacement as potassium phosphate.¹⁴ In our study, the admission blood sugar values were inversely proportional to the serum phosphorous levels and the duration of hospital stay was prolonged in patients with low serum phosphorous levels at admission due to more frequent occurrence of complications and poor glycemic control.

CONCLUSION

In this study, higher the blood sugar values lower the serum phosphorous values, and also associated with prolonged hospital stay. So low serum phosphorous levels at the time of admission is also a feature of poor outcome

REFERENCES

1. Seshiah V, Siddharth N, Shah, AP. Textbook of Medicine, 7th edition: 1116.
2. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crises in adult patient with diabetes. *Diabetes care* 2009;32:1335.
3. Derek Le Roith, Yehiel Zick. Recent advances in our understanding of insulin action and insulin resistance. *Clinical diabetes*, 2005;23:17-24.
4. S B Baker L G Worthy. The essentials of calcium, magnesium and phosphate metabolism. *Critical Care and resuscitation*. 2002;4:301-6.
5. Carl A. Burtis, Edward R. Ashwood, David E. Bruns. Teitz textbook of clinical chemistry and molecular diagnostics. 4th edition. 2006. 1905.
6. Wyckoff J, Abrahamson MJ. Diabetic ketoacidosis and hyperosmolar hyperglycemic state. C Roland Kahn, Gordan C. Alene Moses, Robert J Smith. *JOSLINS DIABETES MELLITUS*. 14th edition Lippincott Williams 2008; 53:892-896.
7. Anderson JJ, Mahan LK. Krause's food, nutrition and diet therapy .11th ed. Philadelphia, PA: WB Saunders; 2004:128-130.
8. Daniel Geerse et al. Treatment of hypophosphatemia in the intensive care units: a review. *Int J Critical Care* 2010;11:148-165.
9. Burtis CA, Ashwood ER. Teitz textbook of clinical chemistry, third edition. W.B. Saunders company, Philadelphia, PA 1999:1815.
10. Tracy I, Setji, Ann J. Brown. Gestational diabetes Mellitus. *Clin Diab*. 2005; 23:17-24.
11. Henning TP, Cunningham TP. Biosensors for personal diabetes management. In Ramsay ed Commercial biosensors. New York: John Wiley and sons, 1998;3-46.
12. Williams, M.H 1999 Nutrition for health, fitness and sport, 6th edition. Boston: McGraw-hill.
13. The expert committee on the diagnosis and classification of diabetes mellitus: report on the expert committee on the diagnosis and classification of diabetes mellitus, *diabetes care* 1997;20:1183-97.
14. David B Snacks, David E Goldstein, Noel K Maclaren. Guidelines and recommendation for laboratory analysis in the diagnosis and management of DM. *Clin chemistry* 2002;48:436-72