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Original Research Article

Study of Serum Electrolytes Levels in Patients with Diabetic Ketoacidosis

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ABSTRACT

Background: Hyperglycemia causes fluid and electrolytes abnormalities in patients with diabetic ketoacidosis. Since electrolytes plays an important role in many body processes, its imbalance may act as a contributing factor towards complications observed in diabetes.
Aim: The present study was conducted to investigate electrolytes in patients of diabetic ketoacidosis that may be helpful in the diagnosis and description of diseases.
Material and Methods: The study population consisted of 30 patients (age:- 25-75) of diabetic ketoacidosis as cases and 30 individuals with controlled diabetes as control. Fasting and Post Meal Blood Glucose Levels were done by Two- Dimentional RXL. Concentration of serum sodium and serum potassium and HbA1c were measured by AVL 9180 (electrolyte analyzer) using the kits supplied by Roche. Urine ketone bodies was done by uristix of Siemens.
Results: The lower sodium and higher potassium levels in serum were observed in cases as compared to control group, where normal levels of electrolytes were seen.
Conclusion: This study shows that distribution of serum Na and K levels is dependent on plasma glucose levels in patients with DKA and also suggests that the electrolyte imbalance is closely related to one another.

Key words: Diabetic Ketoacidosis, Hyponatremia, Hyperkalemia, blood glucose, insulin

INTRODUCTION

Diabetic ketoacidosis (DKA) is an acute complication of diabetes mellitus which requires prompt assessment and treatment to avoid devastating consequences. DKA is characterized by hyperglycemia, ketonemia and acidosis due to increased glucose and ketone body formation with decreased peripheral utilization of glucose and ketone bodies. [1] This occurs due to a bi-hormonal disorder of insulin deficiency and glucagon excess.

Diabetic ketoacidosis (DKA) remains a major problem of uncontrolled diabetes with significantly morbidity and mortality. Although in the last 3 decades, the average worldwide immediate mortality, related to DKA, has decreased from 10% to 5%, survival has not improved strikingly. [2] Hyperglycaemia, ketosis (ketonaemia or ketonuria) and acidosis are the cardinal features of DKA and other features that indicate the severity of DKA include volume depletion, acidosis and concurrent

electrolyte disturbances, especially abnormalities of potassium homeostasis. [3] Hyperglycemia causes fluid and electrolytes to shift from the intracellular to the extracellular space, which leads to subsequent cellular dehydration and electrolyte abnormalities. [4]

In the pathogenesis of ketoacidosis, relative insulin deficiency and counter regulatory hormones excess lead to overproduction and underutilization of glucose resulting in hyperglycemia. Increased lipolysis leads to excessive formation of ketone bodies, which accumulate to produce a metabolic acidosis. [5,6]

Electrolytes play an important role in many body processes, such as controlling fluid levels, acid-base balance (pH), nerve conduction, blood clotting and muscle contraction. Electrolyte imbalance resulting from kidney failure, dehydration, fever and vomiting has been suggested as one of the contributing factors towards complications observed in diabetes and other endocrine disorders. [7,8]

Electrolytes have always played a significant role in number of diseases and change in the concentration of these parameters usually gives good indications in disease progression in number of non communicable diseases. Accordingly this study was aimed to investigate electrolytes in patients of diabetic ketoacidosis that may be helpful in the diagnosis and description of diseases.

MATERIALS AND METHODS

The study was carried out in Department of Biochemistry and central Investigation Laboratory in MGM Hospital, Aurangabad.

The Protocol of the study was submitted to the institutional Ethical Committee and permission was granted for the study.

Study groups:

- 30 patients (age:- 25-75) of diabetic ketoacidosis, referred by various hospitals to the MGM Medical College, Aurangabad.
- 30 individuals with controlled diabetes (age and sex matched) belonging to the same socio-economic status were selected as control.

Blood and urine sampling:

The blood samples (3-5ml fresh blood) will be drawn and collected in a clean, disposable plastic tube from anterior cubital vein under aseptic condition for estimation of serum sodium and serum potassium.

Fasting and Post Meal Blood Glucose Levels were done by Two-Dimentional RXL. Concentration of serum sodium and serum potassium were measured by AVL 9180 (electrolyte analyzer) using the kits supplied by Roche. HbA1c was estimated by using Ion exchange chromatography.

Urine ketone bodies were done by uristix of Siemens.

Statistical analysis:

The data were evaluated by SPSS statistical package version 16.0. The results obtained were statistically analyzed by using student t-test. Value of HbA1c was given as percentage of total hemoglobin and values of electrolytes were given in mEq/L and blood glucose levels in mg/dl. All Values were expressed as mean ± standard. The results were considered significant when $P < 0.05$.

RESULT

The results in this study showed significant increase in fasting blood glucose, post meal blood glucose and HbA1c levels in diabetic ketoacidosis ceases as compared to controlled diabetic patients ($p<0.05$) as shown in table 1. Urine shows presence of

ketone bodies in patients with diabetic ketoacidosis.

Table 1: Mean of fasting blood glucose, post meal glucose and HbA1c in cases and Controls.

Parameter	Cases of DKA (n=30)	Controls (n=30)	p-value
Fasting blood glucose (mg/dl)	216 ± 22.172	128 ± 11.154	0.003
Postmeal blood glucose (mg/dl)	338 ± 50.663	184± 42.354	0.001
HbA1c (%)	8.037 ± 1.458	6.005 ± 0.349	0.000
Urine ketone bodies	+++	-	

Values are given as mean ± SD.
p-value <0.05 considered as statistically significant.

Table 2: Mean of serum sodium and serum potassium in cases and Controls.

Parameter	Cases of DKA (n=30)	Controls (n=30)	p-value
Serum sodium (mEq/L)	126.2 ± 2.7	139.3±0.4	<0.001
Serum potassium (mEq/L)	5.73 ±0.07	4.22 ±0.03	0.003

Values are given as mean ± SD.
p-value <0.05 considered as statistically significant.

There was a significant decrease in the levels of serum sodium in cases as compared to controls (<0.001) and increased levels of serum potassium (<0.05) as shown in table 2 and figure 1.

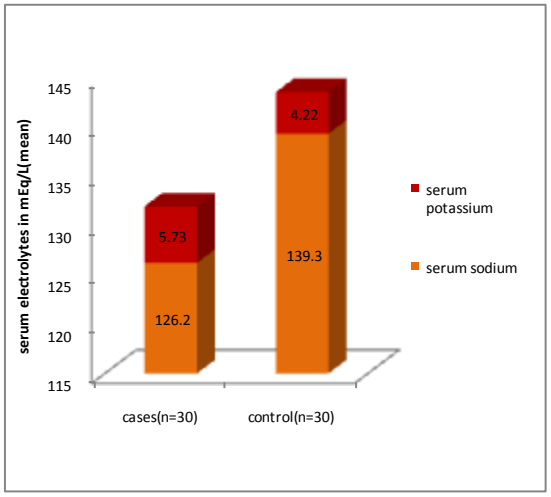

Figure 1: Mean of serum sodium and serum potassium in cases and Controls.

DISCUSSION

In this study, the mean plasma level of Na+ in patients with DKA tends to be low, despite an increase in osmolar concentration. This is because glucose draws water into the extracellular compartment thereby decreasing Na ion concentration. A very low serum Na ion level is usually due to hypertriglyceridemia but may be due to vomiting and water intake. [9,10] Patients with DKA tend to have elevated serum potassium concentration despite decreased body K+ content. [11] This is due to decreased K^+ ion excretion by the kidney once volume depletion reduces GFR, also due to the corresponding acidosis and insulin deficiency resulting in shift of k ion from intracellular to extracellular compartment. [9] Studies have shown serum K^+ to correlate independently with both blood pH and renal function. [6] Serum sodium was significantly higher in the new cases of DM than the previously diagnosed cases.

San-E-Ishikawa et al, 1994, observed that hyponatremia with hyperkalemia was found in the patients in diabetic ketoacidosis and nonketotic hyperosmolar coma, and hypernatremia with hypokalemia in non-ketotic hyperosmolar coma. [9] They concluded that the disorder may be based on the altered distribution of electrolytes between intra- and extracellular spaces, but the exact mechanism for the disorder has not been determined. [9]

When insulin is deficient, hyperglycemia develops because of an increased gluconeogenesis and accelerated conversion of glycogen to glucose (glycogenolysis), and by inadequate use of glucose by peripheral tissues, primarily muscle. From the quantitative standpoint, increased hepatic glucose production represents the major pathogenic disturbance responsible for hyperglycemia in DKA. As the glucose concentration and osmolality of extracellular fluid increase (5.6 mOsm/kg

for every 100 mg/dL increase in plasma glucose), an osmolar gradient is created that draws water out of the cells. [6,7] Glomerular filtration is initially increased, which leads to glucosuria and osmotic diuresis. The initial glucosuria prevents the development of severe hyperglycemia as long as the glomerular filtration rate is normal. However, with continued osmotic diuresis, hypovolemia eventually occurs, which leads to a progressive decline in glomerular filtration rate, and hyperglycemia become more severe. [6]

CONCLUSION

It is concluded from the results of this study that hyperglycemia produces osmotic diuresis causing changes in the distribution of electrolytes between intra- and extra cellular spaces. In addition this effect of hyperosmolality, it may also depend on the effect of insulin on the electrolytes.

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