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A Prospective Cross-Sectional Study of Serum Electrolyte Changes in Diabetic Ketoacidosis

Dr. CH. Rahul[1], Dr. R. Vikram Vardhan[2], Dr. V. Satya Prasad[3]

[1] Post Graduate Student, Department of General Medicine, Andhra Medical College, Visakhapatnam.
[2] Associate Professor, Department of General Medicine, Andhra Medical College, Visakhapatnam.
[3] Professor, Department of General Medicine, Andhra Medical College, Visakhapatnam.

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*Corresponding Author:

Dr. R. Vikram Vardhan
Associate Professor, Department of General Medicine, Andhra Medical College, Visakhapatnam.

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A B S T R A C T

Background: Diabetic ketoacidosis (DKA) remains a critical endocrine emergency with substantial morbidity and mortality. Electrolyte disturbances play a central role in its clinical course and management, yet remain underexplored in Indian populations.
Methods: This prospective observational study was conducted in 100 patients with DKA admitted to King George Hospital, Andhra Medical College, Visakhapatnam, from May 2023 to April 2024. Serum electrolytes (Na, K, Cl, Mg, Ca) were assessed at admission, 24h, and 48h post-treatment. Associations with glycemic control (HbA1c), insulin requirement, and outcomes were analyzed.
Results: Of 100 patients, 64% were male and mean age was 41.7±15.6 years. Type 2 diabetes constituted 72% of cases. At admission, hyponatremia (34%), hypokalemia (12%), hypomagnesemia (34%), hyperkalemia (30%), and hypocalcemia (8%) were frequent. Serial monitoring revealed progressive correction of dyselectrolytemia by 48h. HbA1c >8.6% was strongly associated with higher electrolyte derangements, increased insulin requirement (p=0.005), and worse outcomes. Four patients (4%) succumbed to DKA complications. Infections (52%) and treatment non-compliance (26%) were leading precipitants.
Conclusion: Electrolyte imbalances, particularly sodium, potassium, and magnesium abnormalities, are highly prevalent in DKA and strongly linked to glycemic status. Routine monitoring and tailored correction are crucial for improving clinical outcomes.

Keywords: Diabetic ketoacidosis; Serum electrolytes; Hyponatremia; Hypokalemia; Insulin therapy; India.

INTRODUCTION

Diabetic ketoacidosis (DKA) is a life-threatening acute metabolic complication of diabetes mellitus (DM). It is defined as uncontrolled hyperglycemia, metabolic acidosis and ketonemia. Irrespective of progress in insulin therapy and standard treatment protocol, DKA is become a significant cause of hospitalization worldwide and remains an important cause of morbidity and mortality, mainly in low and meddle-income countries where diabetes care is often limited[1]. The incidence of DKA is lowest in Nigeria (2.9 per 100,000) and highest in Scandinavian nations (41 per 100,000) [2]. In India, where the diabetes burden is rapidly increasing, DKA is observed in both newly diagnosed cases as well as known case of DM with default therapy. Although global mortality has fallen from 10% to <5% over the last three decades, Outcomes remain poor in elderly patients and those with comorbidities [3].

Electrolyte abnormalities are play an important role in the pathophysiology and prognosis of DKA. The pathophysiology of DKA involves in insulin deficiency together with counerregulatory hormone excess which cause hyperglycemia, dehydration, lipolysis, and ketoacidosis [4]. These metabolic changes are complicated by electrolyte imbalances, total body potassium is profoundly depleted despite normal or elevated serum levels at presentation, predisposing patients to hypokalemia and arrhythmias, once insulin therapy begins, sodium imbalance is common, with dilutional hyponatremia

from hyperglycemia or hypernatremia in prolonged dehydration, while hypomagnesemia and hypocalcemia, though often overlooked, further complicated management [5,6,7].

In previous studies have highlighted the burden and dynamic nature of electrolyte disturbances. Su et al. demonstrated that both hyperkalemia and hypokalemia were significantly associated with diabetes duration, glycemic status, and renal dysfunction in DKA patients [8]. Holkar et al. found that low sodium and high potassium level in DKA cases compared with controlled diabetics [9]. Similarly, Julius et al. observed that uncontrolled hyperglycemia with elevated HbA1c strongly correlated with electrolyte imbalance, suggesting that metabolic control influences electrolyte balance [10]. These finding emphasize the need for close observation and correction of electrolytes during the treatment period, as inappropriate replacement of electrolyte may worsen complication.

In India, studies on electrolyte change in DKA remain limited where most of the studies are retrospective or restricted to single electrolyte parameter. Therefore, this study was undertaken to bridge that gap by prospectively evaluating the serum electrolyte profile of DKA patients and correlating finding with glycemic control, insulin requirement, and clinical outcome.

MATERIALS AND METHOD

Study design and setting:

This was a prospective cross-sectional study conducted at King George Hospital (KGH), Andhra Medical College, Visakhapatnam, Andhra Pradesh, India, in the department of General Medicine. The duration of the study was one year (May 2023- April 2024). The ethical clearance was obtained from IEC, Andhra medical college and written informed consent was obtained from all participants.

Study population:

A total of 100 adult patients presenting with DKA were included in this study. The diagnosis was confirm using the American Diabetes Association (ADA) criteria like plasma glucose >250 mg/dL, arterial pH <7.3, Serum bicarbonate ≤18 mEq/L, and presence of ketonemia or ketonuria [11].

Inclusion criteria:

- Adult (> 18 years), either gender
- Biochemistry confirmed DKA
- Written informed consent obtained.

Exclusion criteria:

- Patient with kidney diseases
- Cases without detectable urine ketones
- Patient not willing to give consent.

Laboratory investigations**:**

To confirm the diagnosis, biochemical tests were performed, including RBS & serum electrolytes. The nitroprusside test was used to identify urine ketones. Other investigations such as Hb%, Total WBC count & differential count, HbA1C, LFT, urine for sugars, albumin, & microscopy were done. Chest X-ray done for identification of any lung disease. ECG was done to look for signs of ischemic heart disease & potassium changes.

Serum osmolality was calculated using the following formula:

$$2 \times Na^{+}\ (mEq/L) + \frac{Glucose\ (mg/dL)}{18} + \frac{BUN\ (mg/dL)}{2.8}$$

Management protocol:

Treatment followed standard DKA Management guidelines (11, 12). All patients received intravenous fluids, continuous insulin infusion, and potassium supplementation as per baseline levels. Bicarbonate was reserved for patients with arterial pH <6.9. Concurrent infections and comorbidities were investigated and treated appropriately.

Monitoring:

Serum electrolytes were monitored at three time points:

1) At admission (Baseline)
2) 24 hours after initiation of therapy
3) 48 hours after initiation of therapy

This allowed assessment of the dynamic changes in electrolytes during treatment.

Statistical analysis:

Data were collected & recorded in the proforma, then loaded into an Excel spreadsheet & analyzed to determine the relationship between glycemic status, electrolyte imbalance, & complications developed. The statistical analysis is carried out using the SPSS version 26.0 (SPSS Inc., Chicago, IL, USA) windows application. The mean was compared using the t-test and ANOVA. Chi-square & Kruskal-Wallis tests were employed to compare the relationships among the groups.

RESULT

Patient demographics and clinical characteristics:

A total 100 patients were included in this study were mean age of the participants was 41.72 ± 15.55 years, ranging from 19 to 83 years. In this study 64 males (64%) and 36 female (36%). Regarding diabetes type, 72 patients (72%) had type 2 diabetes mellitus, while 28 patients (28%) had type 1 diabetes. The duration of diabetes was different but most of them reporting 1-5 years (40%) after diagnosis. Table 1 and 2 represent the demographic characteristics of participant.

Table 1: Patient demographics characteristics

variable	Frequency/ Mean ±SD	Percentage/Range
Age (years)	41.72±15.55	19-83
Male	64	64%
Female	36	36%
Type 1 Diabetes	28	28%
Type 2 Diabetes	72	72%
Duration of diabetes: < 1 year	20	20%
Duration of diabetes: 1-5 year	40	40%
Duration of diabetes: 6-10 year	24	24%
Duration of diabetes: >10 year	16	16%

In this study most common symptoms were shortness of breath (46%), followed by Polyuria/thirst (22%), nausea/vomiting (14%), confusion (12%), and abdominal pain (8%). Precipitating factors for DKA included infections in 52% of cases (acute gastroenteritis 14%, urinary tract infection 12%, cellulitis 12%, and viral pneumonia 6%), non-compliance with treatment in 26%, cerebrovascular accident in 6%, myocardial infarction in 4%, and head injury in 2%. Table 2 was represent the clinical characteristic of the participants:

Table 2: Patient clinical characteristics:

Precipitating Factor	Frequency	Percentage
Infection (Total)	52	52%
Acute gastroenteritis	14	14%
Urinary tract infection	12	12%
cellulitis	12	12%
Viral pneumonia	6	6%
Other infection	8	8%
Non- compliance to treatment	16	26%
Cerebrovascular accident	6	6%
Myocardial infraction	4	4%
Head injury	2	2%
Other/Unknown	10	10%

A the time of admission of patient, the mean random blood sugar (RBS) was 412.28 ± 80.27 mg/dL, with mean pH of 7.17 ± 0.10 and HCO3 of 12.76 ± 2.71 mEq/L. Urine ketone bodies were positive in all cases (2+ in 48%, 3+ in 40%, 4+ in 12%). Glycemic control, assessed by HbA1c, showed 28 patients (28%) with controlled diabetes (HbA1c ≤8.5%) and 72 (72%) with uncontrolled (HbA1c >8.5%). The mean HbA1c was 9.18 ± 1.28%. Serum osmolality averaged 310.16 ± 18.44 mOsm/kg, with 84.6% of patients having values >320 mOsm/kg presenting with confusion or coma.
Complications occurred in 14% of patients: renal failure in 6%, heart failure in 4%, and both in 4%. These were transient, resolving with treatment. Overall mortality was 4%, all in the uncontrolled HbA1c group.

Serum Electrolyte Levels at Admission and Changes over Time

In present study the electrolyte level at the time of admission, the mean sodium level was 141.52 ± 6.94 mEq/L, potassium 4.38 ± 0.66 mEq/L, chloride 102.10 ± 5.02 mEq/L, magnesium 1.63 ± 0.73 mg/dL, and calcium 9.2 ± 1.4 mg/dL. Serial monitoring showed progressive normalization. At 24 hours, means were sodium 142.10 ± 6.72 mEq/L, potassium 4.14 ± 0.62 mEq/L, chloride 102.42 ± 4.42 mEq/L, magnesium 1.88 ± 0.73 mg/dL, and calcium 9.4 ± 1.14 mg/dL. By 48 hours, further improvements occurred: sodium 143.06 ± 6.35 mEq/L ($p<0.05$ vs. admission), potassium 4.20 ± 0.54 mEq/L ($p<0.001$), chloride 102.70 ± 4.48 mEq/L ($p<0.001$), magnesium 1.87 ± 0.55 mg/dL ($p<0.001$), and calcium 9.6 ± 1.07 mg/dL ($p<0.001$), which were shown in fig 1.

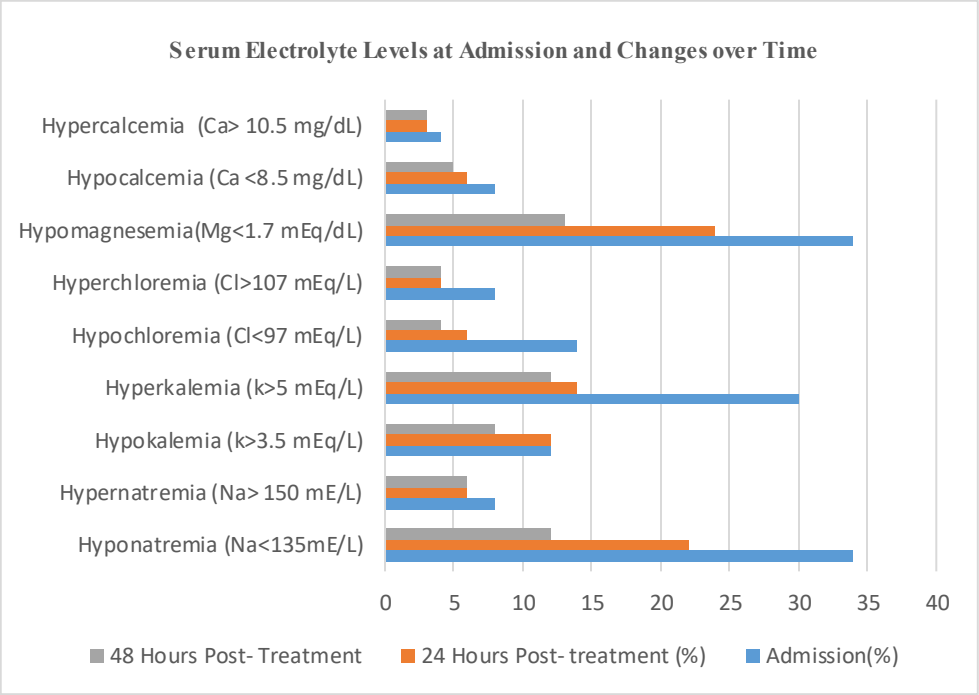

Fig.1: Serum Electrolyte Levels at Admission and Changes over Time

These changes reflect effective fluid and insulin therapy, with most imbalances peaking at admission and declining thereafter. Significantly, hypomagnesemia persisted in 13–24% of cases, suggesting slower resolution.

Associations with Glycemic Control (HbA1c)

In Present study Electrolyte changes were strongly linked to glycemic status. Patients with uncontrolled diabetes (HbA1c >8.5%) had higher rates of abnormalities across all electrolytes compared to those with controlled diabetes ($p<0.001$ for most). For instance, all cases of hyponatremia, hyperkalemia, hypomagnesemia, and other imbalances at admission and follow-up occurred in the uncontrolled group.

Independent t-tests confirmed significant differences in mean electrolyte levels between groups (Table 3). Regression analysis showed positive correlations between HbA1c and insulin requirements ($R=0.281$, $p=0.005$), with higher HbA1c predicting greater insulin needs for DKA resolution.

Table: Associations with Glycemic Control (HbA1c)

Variable	Controlled DM (Mean ± SD)	Uncontrolled DM (Mean ± SD)	p-value
Sodium (Admission)	144.93 ± 2.48	140.19 ± 7.64	0.002
Potassium (Admission)	4.25 ± 0.27	4.43 ± 0.76	0.224
Chloride (Admission)	103.93 ± 2.51	101.39 ± 5.56	0.002
Magnesium (Admission)	2.09 ± 0.34	1.46 ± 0.77	<0.001
Calcium (Admission)	9.91 ± 1.63	8.82 ± 1.05	0.003

Chi-square tests revealed associations between symptoms, precipitating factors, and HbA1c ($p<0.001$), with uncontrolled patients more likely to have severe presentations. Fluid replacement and potassium supplementation were also significantly associated with higher HbA1c ($p<0.001$ and $p=0.021$, respectively). Table 4 highlights key associations between clinical factors and HbA1c categories.

Table 4: associations between clinical factors and HbA1c categories.

Factor	Controlled DM (n=28)	Uncontrolled DM (n=72)	p-value (Chi-square)
Symptoms: Shortness of breath	8 (28.6%)	38 (52.8%)	<0.001
Symptoms: Polyuria/Thirst	10 (35.7%)	12 (16.7%)	<0.001
Precipitating: Infection	6 (21.4%)	46 (63.9%)	<0.001
Precipitating: Non-compliance	12 (42.9%)	14 (19.4%)	<0.001
Fluid Replacement: Yes	18 (64.3%)	68 (94.4%)	<0.001
Potassium Supplementation: Yes	10 (35.7%)	42 (58.3%)	0.021
Complications: Yes	0 (0%)	14 (19.4%)	0.015

Correlations and Predictors of Electrolyte Changes

In Present study electrolytes were intercorrelated negatively at admission: Na and K ($r=-0.75$, $p<0.001$), Na and Cl ($r=-0.67$, $p<0.001$), K and Cl ($r=-0.75$, $p<0.001$). Potassium negatively correlated with creatinine ($r=-0.36$, $p=0.025$) and RBS ($r=-0.37$, $p=0.035$), indicating renal involvement in imbalances. Table 6 presents correlation coefficients among key variables.

Table: Correlations and Predictors of Electrolyte Changes

Variable Pair	Correlation Coefficient (r)	p-value
Na (Admission) vs. K (Admission)	-0.75	<0.001
Na (Admission) vs. Cl (Admission)	-0.67	<0.001
K (Admission) vs. Cl (Admission)	-0.75	<0.001
K (Admission) vs. Creatinine	-0.36	0.025
K (Admission) vs. RBS	-0.37	0.035
HbA1c vs. Insulin Requirement	0.28	0.005
HbA1c vs. Serum Osmolality	0.42	<0.001

Logistic regression for uncontrolled DM identified duration of diabetes ($OR=1.37$, $p=0.039$), RBS ($OR=1.06$, $p=0.002$), and potassium ($OR=9.67$, $p=0.010$) as predictors, highlighting their role in electrolyte shifts.

Complications like renal and heart failure were more common in patients with hyponatremia (30.8% complication rate) and hyperkalemia (26.1%) at admission ($p=0.045$).

DISCUSSION

In this prospective cross-sectional study of 100 patients with diabetic ketoacidosis (DKA), electrolyte disturbances were found to be both highly prevalent at presentation and dynamic during the course of treatment. The mean age of the study population was 41.7 years, with a male predominance, and the majority had type 2 diabetes mellitus. Infections were the most common precipitating factor, followed by irregular treatment or non-adherence, which reflects both the vulnerability of patients to intercurrent illnesses and the persisting gaps in long-term diabetes care. This pattern is consistent with

previous Indian studies where infections accounted for nearly half of DKA cases and treatment non-compliance remained an important contributor (13–16).

At admission, hyponatremia (34%) was the most frequent sodium disturbance, most likely due to hyperglycemia-induced osmotic shifts. Hypernatremia, though less common, was clinically important as it indicated more severe dehydration and delayed presentation. Potassium abnormalities were striking: nearly one-third of patients had hyperkalemia at baseline despite total body potassium depletion, reflecting the impact of insulin deficiency and acidosis on potassium shifts. Similar findings have been documented in prior studies emphasizing the paradox of baseline hyperkalemia in DKA (17,18). Hypomagnesemia was also highly prevalent, affecting one-third of patients, highlighting an under-recognized abnormality that may contribute to cardiac instability and complicate potassium homeostasis (19).

During therapy, dynamic shifts in electrolytes were evident. Sodium levels increased significantly as hyperglycemia was corrected, while potassium declined as insulin therapy facilitated cellular uptake. Although this is an expected physiological response, it reinforces the need for vigilant potassium monitoring and supplementation, since hypokalemia developed in 8% of patients by 48 hours. Magnesium levels also showed significant improvement with treatment, though persistent hypomagnesemia was observed in a subset. These trends support international guidelines that recommend close electrolyte surveillance during the first 48 hours of management, as treatment-related shifts can be as hazardous as baseline imbalances if left unrecognized (12,13).

The clinical outcomes in our cohort included renal failure, cardiac failure, and combined renal-cardiac dysfunction in a minority of patients, with an overall mortality of 4%. All patients who died had HbA1c levels above 8.6%, suggesting that poor long-term glycemic control is an important determinant of prognosis. Mortality rates in our study are comparable to recent Indian series (14,16) but remain higher than those in developed countries, where rates have fallen below 1% due to better awareness, early presentation, and intensive care facilities (20).

The findings from this study highlight the central role of electrolyte monitoring and correction in the management of DKA. While fluid resuscitation and insulin infusion remain the cornerstones of therapy, inadequate attention to electrolyte dynamics may lead to avoidable complications and poor outcomes. The high prevalence of infection and non-adherence as precipitating factors also underscores the need for preventive strategies, patient education, and robust diabetes follow-up to reduce recurrence.

CONCLUSION

Electrolyte disturbances are common and clinically significant in diabetic ketoacidosis, with hyponatremia, hyperkalemia, and hypomagnesemia being the most frequent abnormalities at presentation. Serial monitoring demonstrated significant changes in sodium, potassium, magnesium, and calcium during therapy, underlining the importance of dynamic monitoring and timely correction. Infections and treatment non-compliance were the predominant triggers, while mortality was linked to poor glycemic control. Ensuring strict metabolic follow-up and early recognition of electrolyte shifts is essential to reduce morbidity and mortality in DKA.

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