Elucidating the interplay between clinical characteristics, electrolytes, and arterial blood gas abnormalities in diabetic ketoacidosis

Boorapu Rupavathi, Vijay Kumar S S
Department of Emergency Medicine, Kanachur Institute of Medical Sciences, Mangalore, Karnataka, India.

Abstract

Background: Diabetic ketoacidosis (DKA) is a life-threatening complication of diabetes, characterized by hyperglycemia, acidosis, and ketosis. Despite advances in care, the morbidity and mortality associated with DKA remain high, particularly in resource-limited settings. Early identification of factors influencing patient outcomes is critical for optimizing management.

Objective: This study aimed to elucidate the interplay between clinical characteristics, serum electrolyte levels, and arterial blood gas abnormalities in patients with diabetic ketoacidosis (DKA).

Methods: Conducted in the Emergency Medicine Department of PESIMSR Hospital, India, the study included adults presenting with DKA from September 2020 to October 2023. Clinical evaluations, laboratory tests (blood glucose, electrolytes, blood urea nitrogen, arterial blood gas analysis), and a comprehensive history were obtained.

Results: A total of 61% of patients were male, with a mean blood glucose level of 420.66 mg/dL, and an average arterial blood gas pH of 7.18. Hyponatremia (72%) and hyperkalemia (17%) were prevalent among participants. The primary precipitating factors included intercurrent illnesses and drug non-adherence.

Conclusions: Enhanced awareness and proactive management of DKA, particularly regarding electrolyte imbalances and underlying precipitating factors, are essential for improving patient outcomes and reducing complication rates.

Key words: Diabetic Ketoacidosis, Dyselectrolytemia, Emergency care.

Introduction

Diabetes mellitus is a chronic condition characterized by a spectrum of hyperglycemic and hypoglycemic complications, among which diabetic ketoacidosis (DKA) stands out as a critical emergency. DKA is an acute and life-threatening condition primarily associated with type 1 diabetes but can also occur in individuals with type 2 diabetes, particularly during periods of stress or infection. This metabolic derangement is defined by a triad of hyperglycemia, ketoacidosis, and ketonemia, resulting from a profound deficiency of insulin. In the absence of sufficient insulin, glucose cannot effectively enter cells for energy production, prompting the liver to mobilize fat stores and convert them into ketone bodies as an alternative fuel source. The resultant overproduction of ketones leads to their accumulation in the bloodstream, culminating in metabolic acidosis[1].

The presentation of DKA often brings patients to emergency departments, where timely recognition and intervention are critical to prevent severe morbidity and mortality. Laboratory evaluations play a pivotal role in diagnosing DKA, including blood glucose measurements, serum electrolyte levels, blood urea nitrogen (BUN) assessments, and arterial blood gas (ABG) analyses. The hyperglycemic state induces glucosuria, which triggers osmotic diuresis, resulting in significant fluid loss and dehydration. This fluid depletion exacerbates metabolic acidosis and can lead to pronounced electrolyte imbalances[2].

Electrolytes, such as sodium, potassium, and bicarbonate, are essential for maintaining fluid balance, nerve conduction, and muscle function. In the context of DKA, electrolyte disturbances are

Address for Correspondence:

Dr. Vijay Kumar S S
Department of Emergency Medicine,
Kanachur Institute of Medical Sciences, Deralakatte, Karnataka, India
Email: vijay8792069357@gmail.com

particularly concerning. For instance, patients typically exhibit elevated serum potassium levels despite an overall deficit due to decreased renal excretion caused by reduced glomerular filtration rate (GFR). The accompanying acidosis and insulin deficiency further shift potassium from intracellular to extracellular spaces, complicating the clinical picture[3].

Management of DKA requires a multifaceted approach that includes fluid resuscitation, insulin therapy to lower blood glucose levels, correction of electrolyte imbalances particularly potassium and restoration of acid-base homeostasis. Additionally, identifying and treating any underlying infections is crucial for effective management[4].

While numerous studies have explored the clinical profile of DKA patients, limited research has simultaneously examined the dynamic interplay between clinical characteristics, serum electrolyte levels, and arterial blood gas abnormalities in a resource-limited setting emergency department (ED). This study uniquely integrates these three critical dimensions to provide a comprehensive overview of the pathophysiological landscape of DKA at presentation. By highlighting the higher prevalence of hyponatremia and hyperkalemia, and correlating these with arterial pH and clinical severity, the study offers new insights that can refine risk stratification and guide targeted interventions. To the best of our knowledge, this is one of the few studies in India that provides an in-depth analysis of these interrelationships over an extended three-year period, contributing novel data that can enhance protocols for early detection, management, and prevention of complications in DKA especially in ED settings.

Materials and Methods

Study Setting:

This study was conducted in the department of Emergency Medicine at PESIMSR Hospital, India, a tertiary care teaching hospital, providing comprehensive emergency services. The data collection spanned a period of three year from 09.09.2020 to 10.10.2023. The study population consisted of patients presenting to the emergency department with a confirmed diagnosis of diabetic ketoacidosis (DKA), based on clinical and laboratory criteria. Age more than 18 years, Blood glucose levels >250 mg/dL, Venous blood pH <7.3, Plasma bicarbonate levels <15 mEq/L, Anion gap >14 mEq/dL and Presence of ketones in either blood or urine were included in the study while pediatric patients (age <18 years), patients who had received partial treatment for DKA at an outside facility before arriving at PESIMSR Hospital and patients who left against medical advice (LAMA) during their treatment were excluded from the study.

Upon arrival at the ED, patients presenting with symptoms suggestive of DKA such as excessive tiredness, shortness of breath, fruity-scented breath, or confusion were promptly assessed and stabilized using the standard primary survey, which included evaluation of airway, breathing, circulation, disability, and exposure (ABCDE). Adjunct assessments, including arterial blood gas (ABG) analysis, electrocardiography (ECG), and capillary blood glucose measurement, were immediately performed to confirm the diagnosis of DKA and assess its severity. For patients with a compromised airway, rapid sequence intubation (RSI) was initiated, and supplemental oxygen was administered to maintain partial pressure of oxygen (PaO2) above 80 mmHg. Patients with hemodynamic instability were initially treated with intravenous (IV) normal saline (NS) at 20 ml/kg to achieve a mean arterial pressure (MAP) above 60 mmHg. If hypotension persisted, norepinephrine (0.05–0.1 mcg/kg/min) was administered as the first-line vasopressor to restore adequate perfusion.

Other blood samples were collected for essential laboratory investigations, including random venous glucose, serum electrolytes (sodium, potassium, chloride), renal function tests (creatinine, urea), and complete blood count

Fluid resuscitation was continued with isotonic saline, with adjustments based on the patient's electrolyte status and hemodynamic response. Continuous intravenous insulin infusion at rate of 0.1 units/hour of regular insulin was initiated to control hyperglycemia and suppress ketogenesis, with hourly monitoring of blood glucose levels.

Following initial stabilization, a comprehensive history was taken from the patients or their caregivers to identify precipitating factors such as non-compliance with medications, recent changes in therapy, newly diagnosed diabetes, or underlying infections. Detailed demographic data, including age, gender, medical history, and treatment adherence, were recorded

Electrolyte imbalances, particularly potassium, were closely monitored and corrected as needed. Patients were shifted to Intensive Care Unit (ICU) for ongoing management, where further investigation into potential DKA precipitating causes, such as infections, continued.

Ethical Considerations:

The study was conducted following ethical principles outlined in the Declaration of Helsinki. Approval for the study was obtained from the Institutional Ethics Committee of PESIMSR Hospital prior to

commencement. Informed consent was obtained from all participants or their legal guardians.

Statistical Analysis:

All collected data were entered into SPSS software for statistical analysis. Descriptive statistics, including mean and standard deviation, were used to summarize continuous variables such as blood glucose levels, serum electrolyte concentrations, and arterial pH values. No comparative statistical tests were performed, as the study was observational in nature. Data were presented in tabular and graphical formats to provide a clear overview of findings.

Results:

Table 1: Demographic Profile of patients with Diabetic Ketoacidosis arriving to emergency department

Variable	Frequency (N)	Percentage (N%)
Gender		
Female	39	39
Male	61	61
Age Group (Years)		
<25years	7	7
25-45 years	36	36
>45 years	57	57
Co morbidity		
Smoking	14	14
Obesity	12	12
Hypertension	22	22
Diabetes	74	74
Compliance		
Compliant	58	78%
Non-Compliant	16	22%
Newly Diagnosed	26	26%

The demographic profile of patients with diabetic ketoacidosis showed that 61% were male, and 57% were aged over 45 years. Diabetes was the most common co-morbidity, present in 74% of patients, followed by hypertension (22%), smoking (14%), and obesity (12%) as shown in (Table 1).

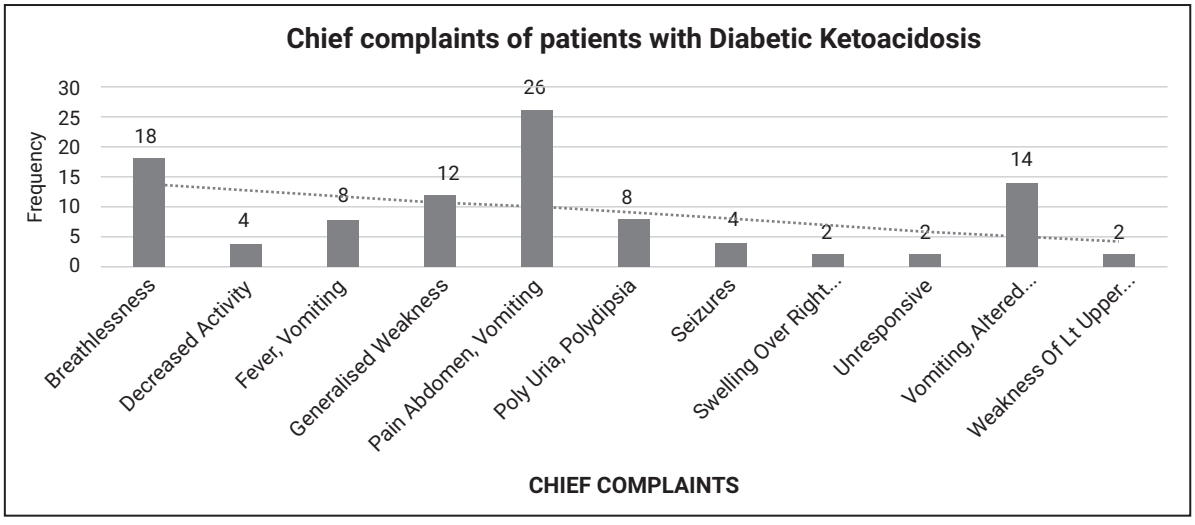

Figure 1: Chief complaints of patients with DKA on arrival to emergency department

The most common chief complaint among patients was abdominal pain with vomiting (26%), followed by breathlessness (18%) and vomiting with altered sensorium (14%). Generalized weakness (12%), fever with vomiting (8%), and polyuria with polydipsia (8%) were also noted. Less frequent presentations included seizures (4%), decreased activity (4%), and isolated cases of swelling, unresponsiveness, and limb weakness (2% each) as shown in (Figure 1)

Table 2: Precipitating factors for Diabetic Ketoacidosis

Sl.	Precipitating Factor	Frequency (N)	Percentage (N%)
1	Drug non compliance	16	16
2	Intercurrent illness	36	36
	Urinary tract infection	8	2.88
	Obstructive uropathy	2	0.72
	Cystitis	4	1.44
	Malignancy	2	0.72
	Acute gastroenteritis	2	0.72
	Cellulitis	2	0.72
	Pancreatitis	2	0.72
	Pyelonephritis	2	0.72
	Dengue fever	2	0.72
	Gluteal abscess	2	0.72
	Septic encephalopathy	2	0.72
	Acute on chronic kidney disease	2	0.72
	Others	4	1.44
3	Starvation	11	11
4	First presentation as DKA (newly diagnosed)	26	26
5	Unknown causes	11	11

The most common precipitating factor for diabetic ketoacidosis (DKA) is an intercurrent illness (36%), with urinary tract infection (UTI) being the most frequent cause. Other conditions that can trigger DKA include obstructive uropathy, cystitis, malignant gastric outlet obstruction (GOO), cellulitis, pancreatitis, pyelonephritis, dengue fever, and chronic kidney disease (CKD). After infections, other precipitating factors for DKA, in order of frequency, include first presentation of diabetes as DKA, non-adherence to diabetes treatment, prolonged starvation (11%), and other unknown causes (11%) as shown in (Table 2).

Table 3: Electrolytes in patients with Diabetic Ketoacidosis arriving to Emergency Department

Sl.	Variable	Low (n)	Normal (n)	High (n)	Mean	Std. Dev.
1	Potassium	2	81	17	**3.2**	**1.1**
2	Sodium	72	28	0	130.75	6.59
3	Chloride	36	50	16	97.96	8.15
4	Calcium	70	30	0	8.58	0.79
5	Magnesium	57	41	2	2.00	0.66
6	Phosphorus	66	18	16	3.80	1.76

The electrolyte parameters in patients with DKA reveal significant imbalances. Potassium levels averaged 3.20 mEq/L, with 2% of patients exhibiting low levels (normal: 3.5–5.0 mEq/L) and 17% presenting with high levels. Sodium levels averaged 130.75 mEq/L, with 72% of patients showing hyponatremia (normal: 135–145 mEq/L). Chloride levels averaged 97.96 mEq/L, with 36% of patients classified as low (normal range: 98–106 mEq/L). Calcium levels averaged 8.58 mg/dL, with 70% falling below the normal range (8.5–10.2 mg/dL). Magnesium averaged 2.00 mg/dL, indicating a deficiency in 57% of patients (normal range: 1.7–2.2 mg/dL). Finally, phosphorus levels averaged 3.80 mg/dL, with 66% of patients presenting with low levels (normal range: 2.5–4.5 mg/dL) as shown in (Table 3).

Table 4: Lab Parameters in patients with Diabetic Ketoacidosis arriving to Emergency Department

Sl.	Parameters	Mean	SD	MIN	MAX
1	Blood Sugar Level	420.66	77.79	236	552
2	Blood ketone bodies	3.902	1.65	0.8	7.8
3	Blood urea	45.842	27.87	12.8	137
4	Serum creatinine	1.306	1.20	0.3	5.3
5	pH	7.1768	0.10	6.85	7.3
6	PCO2	21.47	9.53	6	49
7	Bicarbonate	9.824	4.60	3	21
8	Anion Gap	27.451	6.40	13	45
9	HbA1C	12.94783	6.40	13	45
10	Osmolarity	285.271	13.57	235.7	315

PCO2 - Partial pressure of carbon dioxide, HbA1C - glycated hemoglobin

Laboratory parameters revealed the following averages: random blood sugar (RBS) was 420.66 mg/dL, blood ketones were 3.90 mmol/L, blood urea nitrogen was 45.84 mg/dL, and serum creatinine was 1.31 mg/dL. Arterial blood gas analysis indicated a pH of 7.18, bicarbonate levels of 9.82 mEq/L, and an anion gap of 27.45 mEq/L. The mean HbA1c was 12.95%, indicating poor long-term glycemic control among study participants as shown in (Table 4).

Discussion:

Diabetic ketoacidosis (DKA) represents a significant and life-threatening complication of diabetes, characterized by a triad of uncontrolled hyperglycemia (random blood sugar exceeding 250 mg/dL), metabolic acidosis (pH < 7.3, bicarbonate < 15 mEq/L), and the presence of ketones in the blood or urine. As an acute complication primarily seen in type 2 DM, DKA is often preventable.

A significant peak incidence of diabetic ketoacidosis (DKA) is noted in patients over 45 years, with 57% of

our cohort in this age group. This aligns with findings from Seth P et al. and Eugenie A. A. Anago et al., who reported similar age distributions in their studies [3,5]. The consistency may stem from shared risk factors, including the rising prevalence of type 2 diabetes and inadequate healthcare access, particularly in older adults.

In contrast, some studies report younger demographics, potentially due to variations in healthcare access and diabetes management. Populations with better education and resources may experience lower rates of DKA in younger individuals, highlighting the importance of targeted interventions for effective diabetes management. Addressing these factors is essential to mitigate DKA incidence across all age groups.

The male predominance in our study aligns with findings from other research, likely due to factors such as higher stress levels, lifestyle-related risks, and less consistent healthcare engagement among males [6]. Conversely, the female predominance noted by Elmehdawi RR et al. may be attributed to differing healthcare access or management patterns among women, as well as hormonal influences [7].

Abdominal pain and vomiting (26%) were the most common presenting complaints in this study, followed by breathlessness (18%) and altered sensorium (14%). Similar findings were reported by Seth et al. (2015) and Mahesh MG et al. (2017), who identified gastrointestinal symptoms, particularly nausea and vomiting, as frequent initial complaints in DKA. These symptoms are likely due to ongoing catabolism, acidosis, and dehydration caused by osmotic diuresis[3,8].

In contrast, Elmehdawi et al. found generalized weakness and fatigue to be the primary presentations in their cohort. This discrepancy may reflect regional differences in symptomatology and the varied clinical presentations of DKA among patients [7].

HbA1C levels were notably elevated (mean value >12) across all patients, indicating poor glycemic control. This aligns with findings from George JT et al. (2018), who reported a mean HbA1C of 12.1 ± 2.7, emphasizing the correlation between high HbA1C and adverse outcomes. In the broader population, a significant proportion of individuals with diabetes were found to be non-compliant with their treatment [9]. For instance, Mahesh MG et al. (2017) noted that 11% of participants were undiagnosed, while Kakusa et al. (2016) found that 32.8% of previously diagnosed diabetics were non-adherent to treatment [10].

Seth P et al. (2015) reported that among 60 patients, a small number were newly diagnosed, and Qari FA et al. (2002) observed that DKA was the first manifestation of type 2 diabetes in some cases [3,11]. These studies collectively highlight the challenges of diabetes management and the critical role of adherence to treatment to prevent severe complications like DKA.

The primary precipitating factors for diabetic ketoacidosis (DKA) identified in the current study were intercurrent illnesses, drug non-adherence, and starvation. These findings are consistent with Shahid W et al. (2020), who reported infection and treatment non-compliance as the leading causes [12]. Similarly, Mahesh MG et al. (2017) emphasized the significance of infections and skipped anti-diabetic treatment [8]. Among infections, urinary tract infections (UTIs) were the most prevalent, corroborated by studies from Rehman W et al. and Naveed D et al. (2009), both highlighting infections and non-compliance as major contributors to DKA[13,14].

In contrast, R.R. Elmehdawi et al. noted that the cessation of insulin treatment was the primary precipitating factor, followed by infections [7]. Seyoum B et al. reported a similar trend, with stoppage of treatment being predominant[15]. These differences may stem from varying patient populations and healthcare access.

Intercurrent illnesses, particularly UTIs, were most frequently observed, followed by chronic kidney disease and cerebrovascular accidents. This aligns with findings from Kakusa et al. (2016) and Elmehdawi RR et al. (2010), who also identified UTIs as a common infection leading to DKA[7,10]. However, Seth P et al. (2015) and Shahid W et al. (2020) highlighted pneumonia as a significant infection, indicating variability in precipitating conditions across studies[3,12]. Such disparities may be attributed to regional healthcare practices and patient demographics.

Patients with moderate to severe hyponatremia (Na^+ <130 mEq/L) often presents with significantly elevated blood glucose levels, typically in the 400-600 mg/dL range. Clinically, these patients commonly exhibit symptoms such as vomiting, abdominal pain, and altered mental status and seizuresemts reflecting both fluid-electrolyte imbalance and worsening acidosis. Most of the patients can have moderate to severe metabolic acidosis (pH < 7.30), highlighting the critical need for prompt fluid resuscitation, insulin therapy, and correction of electrolyte and acid-base disturbances. In the current study, hyponatremia emerged as the predominant electrolyte abnormality, with 72% of patients affected. This is attributed to hyperglycemia, which acts as an osmotically active substance,

leading to water movement from intracellular to extracellular compartments and resulting in dilutional hyponatremia. This aligns with findings from Shrirang Holkar et al. (2014), who also reported hyponatremia as the most common electrolyte disturbance due to osmotic diuresis and acute kidney injury [16].

Hyperkalemia was noted in 17% of participants, while 81% maintained normal potassium levels. Hyperkalemia is linked to extracellular acidosis and insulin deficiency, causing potassium to shift out of cells. Patients with hyperkalaemia (serum potassium > 5.5 mEq/L) often shows signs of moderate to severe metabolic acidosis. This electrolyte disturbance can be frequently linked to clinical symptoms such as generalized weakness, fatigue, and recurrent vomiting, and often presents with hypotension and shock, reflecting the impact of acid-base imbalance and elevated potassium on neuromuscular and gastrointestinal function.This phenomenon was also corroborated by Liamis G et al. and Manappallil RG et al., who noted that acute renal dysfunction exacerbates potassium retention despite osmotic diuresis[17,18]. Gupta A et al. (2019) similarly observed that initial potassium levels were often normal or mildly low due to insulin deficiency[19].

Hypochloremia can most commonly present with symptoms of severe vomiting and dehydration with high anion gap metabolic acidosis instead of metabolic alkalosis due to presence of ketone bodies in DKA. Hyperchloremia indicates a typical response to hyperglycemia-induced osmotic diuresis and subsequent fluid resuscitation, as also noted by Hafifa Rahmah Arrazy et al. (2019)[20].

Hypophosphatemia was observed in 66% of participants, diverging from Amarens van der Vaart et al.'s findings, which reported a higher prevalence of hyperphosphatemia[21]. This discrepancy may result from insulin deficiency leading to an extracellular shift of phosphate, while osmotic diuresis and metabolic acidosis contribute to phosphate loss in the current study. Severe hypophosphatemia less than 1.0mg/dl is often associated with high anion gap metabolic acidosis, presenting with symptoms such as breathlessness and vomiting due to diaphragm muscle weakness along with initially high RBS levels that is exceeding 400mg/dl[21].

Hypomagnesemia was found in 57% of patients, which contrasts with El-Naggar AK et al. (2022), where the majority had normal magnesium levels[22]. Osmotic diuresis, insulin therapy, and correction of acidosis likely contribute to the observed hypomagnesemia. Severe hypomagnesemia patients often presented with generalized weakness[22].

Low plasma calcium levels were noted in 70% of participants, attributed to factors such as osmotic diuresis and acidosis, which impairs calcium reabsorption and promotes the mobilization of calcium from bones. This finding contrasts with Martin et al., who reported a lower prevalence of hypocalcemia[23]. These variations underscore the complexity of electrolyte disturbances in DKA and their dependence on patient-specific factors and underlying conditions.

In the current study, most patients presented with high anion gap metabolic acidosis, consistent with typical DKA presentations. This contrasts with findings by Elisaf MS (1996), who identified mixed acid-base disorders in DKA patients, including severe high anion gap metabolic acidosis, hyperchloremic metabolic acidosis, and instances of metabolic and respiratory alkalosis, influenced by hydration status and underlying conditions such as vomiting and sepsis[24].

All patients in the current study required ICU admission, with a zero mortality rate attributed to timely intervention and appropriate treatment. The mean duration of ICU stay was 13 hours. In contrast, R.R. Elmehdawi et al. (2010) reported a mean hospital stay of 7.7 days with a mortality rate of 10%, linked to older age, late presentation, and complications such as severe metabolic acidosis[7].

Maskey et al. observed an average hospital stay of 60 days, highlighting significant variability in patient outcomes[25]. Alourfi Z et al. (2015) reported an 11.5% mortality rate, primarily due to severe infections and renal failure, particularly in older patients[26]. Similarly, Mahesh MG et al. (2017) found a 9.09% mortality rate related to late presentation, severe acidosis, and shock[8].

George JT et al. (2018) reported an overall mortality rate of 18.9%, attributed to comorbidities such as myocardial infarction and advanced age [9]. Seth P et al. (2015) noted a 10% mortality rate, which was linked to delayed presentation and extended mean insulin requirement for ketone clearance[3]. Qari FA et al. (2002) found an average hospital stay of 8.7 days with a mortality rate of 2%, associated with complications like sepsis and ARDS[11]. These contrasting findings underscore the variability in patient outcomes based on demographic and clinical factors.

Addressing these issues through targeted interventions may help reduce the occurrence of DKA and improve patient outcomes, ultimately contributing to better management of diabetes in diverse populations. Further studies are warranted to explore these dynamics and identify effective strategies for prevention and treatment.

Conclusion

This study underscores the critical need for enhanced awareness and proactive management of diabetic ketoacidosis, particularly in patients with intercurrent illnesses and those experiencing drug non-adherence. The significant prevalence of hyponatremia and hyperkalemia among DKA patients highlights the necessity for vigilant electrolyte monitoring and timely intervention. By addressing, the multifaceted factors contributing to DKA, including infection prevention and adherence to diabetes treatment, healthcare providers can significantly improve patient outcomes and reduce the incidence of this life-threatening complication.

References:

1. *Holkar S, Vaishnav D, Hivre M. Study of serum electrolytes levels in patients with diabetic ketoacidosis. International Journal of Health Sciences & Research. 2014; 4:154-7.*
2. *Gosmanov AR, Gosmanova EO, Kitabchi AE. Hyperglycemic crises: diabetic ketoacidosis and hyperglycemic hyperosmolar state. Endotext [Internet]. 2021;9:1.*
3. *Seth P, Kaur H, Kaur M. Clinical profile of diabetic ketoacidosis: a prospective study in a tertiary care hospital. Journal of clinical and diagnostic research: JCDR. 2015;9(6): OC01.*
4. *Rao VD, Pradhan B, Mallikarjuna Y, Reddy R. Clinical profile of diabetic ketoacidosis in adults. Health Renaissance. 2012;10(2):80-6.*
5. *Anago E, Medehouenou T, Akpovi CD, Tchehouenou H. Electrolyte disturbances in diabetic patients in Cotonou, Benin. Int J Res Med Sci. 2016;4(12):5430-5.*
6. *Singh H, Thangaraju P, Kumar S, Aravindan U, Balasubramanian H, Selvan T. Knowledge and awareness of diabetes and diabetic ketoacidosis (DKA) among medical students in a tertiary teaching hospital: An observational study. Journal of clinical and diagnostic research: JCDR. 2014;8(4): HC04.*
7. *Elmehdawi RR, Ehmida M, Elmagrehi H, Alaysh A. Incidence and mortality of diabetic ketoacidosis in Benghazi-Libya in 2007. Oman medical journal. 2013;28(3):178.*
8. *Mahesh MG, ShIvaSwaMy RP, ChandRa BS, Syed S. The study of different clinical pattern of diabetic ketoacidosis and common precipitating events and independent mortality factors. Journal of clinical and diagnostic research: JCDR. 2017;11(4): OC42.*
9. *George JT, Mishra AK, Iyadurai R. Correlation between the outcomes and severity of diabetic ketoacidosis: A retrospective pilot study. Journal of family medicine and primary care. 2018;7(4):787-90.*
10. *Kakusa M, Kamanga B, Ngalamika O, Nyirenda S. Comatose and noncomatose adult diabetic ketoacidosis patients at the University Teaching Hospital, Zambia: Clinical profiles, risk factors, and mortality outcomes. Indian journal of endocrinology and metabolism. 2016;20(2):199-205.*
11. *Qari FA. Precipitating factors for diabetic ketoacidosis. Saudi Med J. 2002; 23(2):173–6.*
12. *Shahid W, Khan F, Makda A, Kumar V, Memon S, Rizwan A. Diabetic ketoacidosis: Clinical characteristics and precipitating factors. Cureus. 2020;12(10):1*
13. *Rehman W, Hussain S, Azhar MA. Risk factors of diabetic ketoacidosis: Case-control study. J Sheikh Zayed Med Coll. 2013;4(2):436–439.*
14. *Naveed D, Bilal N, Nasir B, Lodhi BR. Precipitating factors for diabetic ketoacidosis. Khyber medical university journal. 2009;(1):1.*
15. *Seyoum B, Berhanu P. Profile of diabetic ketoacidosis in a predominantly African American urban patient population. Ethnicity & Disease. 2007; 17(2):234-7.*
16. *Holkar S, Vaishnav D, Hivre M. Study of serum electrolytes levels in patients with diabetic ketoacidosis. International Journal of Health Sciences & Research. 2014; 4:154-7.*
17. *Liamis G, Liberopoulos E, Barkas F, Elisaf M. Diabetes mellitus and electrolyte disorders. World Journal of Clinical Cases: WJCC. 2014; 2(10):488.*
18. *Manappallil RG, Nambiar J. Hyperkalemic cardiac arrest in a patient with diabetic ketoacidosis. Indian Journal of Critical Care Medicine: Peer-reviewed, Official Publication of Indian Society of Critical Care Medicine. 2020; 24(8):737.*
19. *Gupta A, El-Wiher N. Therapeutic challenges in management of severe acidosis and profound hypokalemia in pediatric diabetic ketoacidosis. Global pediatric health. 2019; 6:2333794X19840364.*
20. *Arrazy HR, Freddy A, Setijowati N. Correlation between the serum chloride levels and the anion gap values in initial fluid resuscitation with normal saline in patients with diabetic ketoacidosis at RSUD Dr. Saiful Anwar Malang. J Health Med Nurs. 2019; 66:88–92.*
21. *van der Vaart A, Waanders F, van Beek AP, Vriesendorp TM, Wolffenbuttel BH, van Dijk PR. Incidence and determinants of hypophosphatemia in diabetic ketoacidosis: An observational study. BMJ Open Diabetes Res Care. 2021; 9(1): e002018.*
22. *El-Naggar AK, Sameer G, Sadek AM. Impact of Hypophosphatemia and Hypomagnesaemia on Diabetic Ketoacidosis patient's Outcome in Medical Intensive Care Unit. Zagazig University Medical Journal. 2022; 28(1):45-53.*
23. *Martin HE, Wertman M. Serum potassium, magnesium, and calcium levels in diabetic acidosis. The Journal of clinical investigation. 1947; 26(2):217-28.*
24. *Elisaf MS, Tsatsoulis AA, Katopodis KP, Siamopoulos KC. Acid-base and electrolyte disturbances in patients with diabetic ketoacidosis. Diabetes research and clinical practice. 1996; 34(1):23-7.*
25. *Maskey R, Shakya DR, Nikesh B, Krishna KA, Lavaju P, Kattel V, Arjyal L, Karki P. Clinical profile of diabetic ketoacidosis in tertiary care hospital of Eastern Nepal. Indian journal of endocrinology and metabolism. 2015; 19(5):673-5.*
26. *Alourfi Z, Homsi H. Precipitating factors, outcomes, and recurrence of diabetic ketoacidosis at a university hospital in Damascus. Avicenna Journal of Medicine. 2015; 5(01):11-5.*

Conflict of interest: Nil
Source of funding: Nil

Date received: Dec 18, 2024
Date accepted: May 21, 2025