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Original Research Article

CLINICAL AND ELECTROCARDIOGRAPHIC PROFILES OF ELECTROLYTE IMBALANCES IN ADULT PATIENTS WITH DIABETIC KETOACIDOSIS IN INDIA

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ABSTRACT

Background: This study aims to assess the clinical manifestations and electrocardiographic (ECG) changes associated with electrolyte imbalances—specifically hypokalemia, hyperkalemia, hypomagnesemia, and hypermagnesemia—in adult patients presenting with diabetic ketoacidosis (DKA) in India. **Materials and Methods:** This cross-sectional observational study was conducted over 17 months in the Emergency Department of a tertiary care hospital in Kerala, India, to evaluate electrolyte disturbances in DKA. Fifty-one adult patients with confirmed DKA were enrolled based on the American Diabetes Association (ADA) criteria. Serum potassium was measured using the indirect ion-selective electrode method, while serum magnesium was estimated using the xylidyl blue colorimetric method. A standard 12-lead ECG was performed at admission before the initiation of treatment and was assessed for electrolyte-related abnormalities such as peaked T waves, ST-segment depression, and PR interval prolongation. ECGs were interpreted by attending physicians and correlated with serum potassium and magnesium levels. **Result:** Hyperkalemia was the most clinically significant electrolyte abnormality, with 9 (17.6%) patients exhibiting ECG changes, primarily peaked T waves and PR interval prolongation. It was most common in the 60–69-year age group and predominantly affected females (93%), often associated with insulin use and acute kidney injury. Hypokalemia, mainly linked to diuretic and β2-agonist use, resulted in ECG changes in only 1 (2.0%) patient, typically presenting as flat T waves or ST-segment depression; this occurred most often in males aged 80–89 years. Hypomagnesemia, frequently due to renal losses or insulin use, was observed biochemically but did not result in ECG abnormalities; it was more common in males aged 60–69 years. Similarly, hypermagnesemia—usually due to renal failure—did not produce any ECG effects and was more frequently seen in females aged 70–79 years. **Conclusion:** Hyperkalemia was the only electrolyte disturbance significantly associated with ECG abnormalities, predominantly in older female patients. Although hypokalemia and hypomagnesemia were biochemically common, they rarely manifested as ECG changes.

INTRODUCTION

Diabetic ketoacidosis (DKA) is a severe, acute, and potentially life-threatening complication of diabetes mellitus (DM), particularly type 1 DM.[1,2] However, it may also occur in individuals with type 2 DM under specific stressors.[3] As per the interpretation of Al-Bunyan triad of hyperglycemia, ketonemia or ketonuria, and metabolic acidosis characterizes DKA.[4] DKA results from an absolute or relative insulin deficiency, coupled with elevated counterregulatory hormones such as glucagon, cortisol, catecholamines, and growth hormone, which stimulate excessive gluconeogenesis, lipolysis, and ketogenesis.[1,3] Common precipitating factors include missed insulin doses, infections, trauma, myocardial infarction, and the use of medications like corticosteroids or thiazides.[5]

Despite therapeutic advances, the incidence of DKA-related hospitalizations has increased globally—particularly among younger adults—posing a

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significant burden on healthcare systems.[5,6] India, which bears one of the highest diabetes burdens worldwide, has witnessed a rising number of DKA cases.[7,8] However, there is a paucity of national data on these patients' clinical and metabolic profiles. The associated morbidity and risk of fatal outcomes, especially among those with comorbidities, underscore the importance of early recognition and comprehensive management.[9,10]

Electrolyte imbalances are a hallmark of DKA and play a central role in its clinical course and prognosis.[11] These disturbances—especially in potassium and magnesium—result from insulin deficiency, osmotic diuresis, and metabolic acidosis.[12] Potassium imbalance, for instance, can shift rapidly from extracellular hyperkalemia to life-threatening hypokalemia during insulin therapy.[13] Both conditions may cause serious cardiac arrhythmias and produce characteristic electrocardiographic (ECG) changes, including peaked T waves, flattened P waves, prolonged PR intervals, or ventricular fibrillation.[14] However, ECG changes do not always correlate directly with serum potassium levels, particularly in patients with underlying chronic kidney disease.[15,16]

Magnesium imbalance, particularly hypomagnesemia, is another common but under-recognized feature in DKA.[17] It often coexists with hypokalemia and may result from poor glycemic control, insulin resistance, and increased renal magnesium loss.[18] Hypomagnesemia has been linked to cardiovascular complications, insulin resistance, and poor glycemic outcomes, yet it is seldom monitored in acute care settings.[19] Additionally, other electrolyte disturbances in diabetes—such as hyponatremia and hyperkalemia—have been associated with increased readmission rates, higher healthcare costs, and mortality, but often go unnoticed.[20]

Electrolytes are essential for maintaining membrane potential, neuromuscular function, and cardiac conduction. Their imbalance can lead to clinical symptoms, from subtle to severe.[11] ECG remains a widely accessible and crucial tool for the early identification of dangerous arrhythmias.[14] Despite the high prevalence of DKA in India, there is limited data on the clinical presentation and ECG changes related to specific electrolyte abnormalities in Indian patients with DKA.

This lack of focused regional data represents a significant gap in the literature. Most existing studies are based on Western populations or do not adequately correlate clinical features with ECG findings in the context of electrolyte imbalances. Furthermore, very few studies have examined the impact of potassium and magnesium disturbances on ECG patterns among DKA patients in India.

Therefore, this study aims to assess the clinical manifestations and electrocardiographic (ECG) changes associated with electrolyte imbalances—specifically hypokalemia, hyperkalemia, hypomagnesemia, and hypermagnesemia—in adult patients presenting with diabetic ketoacidosis (DKA) in India.

MATERIALS AND METHODS

Study Design: This cross-sectional observational study was designed to investigate the clinical features and electrocardiographic (ECG) changes associated with electrolyte imbalances in adult patients diagnosed with diabetic ketoacidosis (DKA). The non-interventional study focused on evaluating clinical parameters and ECG findings at the time of presentation to the emergency department.

Study Setting and Duration: The study was conducted in the Emergency Department of Pushpagiri Medical College Hospital, Tiruvalla, Kerala, a tertiary care teaching institution offering comprehensive emergency and intensive care services. It was carried out over seventeen months, from January 2023 to May 2024. Ethical approval was obtained from the Institutional Ethics Committee under reference number PIMSRC/E1/388A/53/2022.

Study Population: The study population included adult patients who presented to the Emergency Department with a confirmed diagnosis of DKA. All patients were evaluated for presenting clinical symptoms and ECG findings, particularly concerning potassium and magnesium level abnormalities. The diagnosis of DKA was based on clinical and biochemical criteria, including blood glucose >250 mg/dL, serum bicarbonate <18 mmol/L, pH <7.3, and serum or urine ketones.

Sample Size Calculation: The sample size was determined using the mean and standard deviation of serum potassium reported in previous studies. Assuming a 99% confidence interval and 5% relative precision, and using a mean serum potassium of 4.49 mmol/L with a standard deviation of 0.62, the minimum required sample size was calculated to be 51 which achieved using a convenience sampling method, whereby all eligible and consenting patients who presented during the study period were consecutively enrolled until the sample size was met.

Inclusion and Exclusion Criteria

Inclusion criteria comprised adult patients aged 18 years and above who presented with DKA and provided written informed consent. Exclusion criteria included patients who were pregnant or lactating, terminally ill, or unwilling to participate. Additionally, individuals with comorbidities that affect electrolyte balance, such as end-stage renal disease or those undergoing dialysis, were excluded to avoid confounding effects on electrolyte values.

Operational Definitions: For this study, electrolyte imbalances were defined based on the standard reference values used by the institutional clinical biochemistry laboratory. Hypokalemia was defined as serum potassium <3.5 mmol/L, and hyperkalemia as >5.5 mmol/L. Hypomagnesemia was defined as serum magnesium <1.6 mg/dL, and hypermagnesemia as >2.4 mg/dL. These thresholds

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were consistently applied to categorize patients into normal, hypo-, or hyper-electrolyte groups.

Data Collection Procedure: Clinical data were collected at the time of presentation using a structured proforma. Demographic details, including age and gender, were recorded. Clinical evaluation included symptoms such as altered sensorium, vomiting, thirst, fever, polyuria, and signs of dehydration. Vital signs, including pulse rate, respiratory rate, blood pressure, and temperature, were documented. Drug history was obtained with emphasis on medications known to influence serum potassium and magnesium levels. A 12-lead ECG was performed upon admission to evaluate electrocardiographic manifestations of electrolyte abnormalities. ECGs were interpreted by the attending physician and confirmed by the investigator using standard diagnostic criteria.

Laboratory Measurements: Venous blood samples were collected at admission before initiating insulin or fluid therapy to avoid therapeutic influence on serum electrolyte levels. Serum potassium was measured using the indirect ion-selective electrode method, while serum magnesium was estimated using the xylidyl blue colorimetric method. All tests were performed in the hospital's NABL-accredited central laboratory under strict internal quality control. Routine investigations, including blood glucose, arterial pH, bicarbonate, and anion gap, were also performed as part of the DKA workup.

Electrocardiographic Measurement: A standard 12-lead ECG was recorded for each patient upon admission, before initiating insulin or fluid therapy, to prevent therapeutic interference with cardiac conduction. ECGs were acquired using calibrated hospital ECG machines, with proper lead placement and adherence to standard operating procedures. Recordings were evaluated for potassium and magnesium imbalances, including peaked T waves, flattened or inverted T waves, ST-segment depression, PR interval prolongation, and U waves. The emergency physician initially interpreted each ECG and verified it by the primary investigator to minimize observer bias. ECG findings were categorized using standard criteria and correlated with corresponding serum electrolyte values. Follow-up ECGs were performed only when clinically indicated.

Statistical Analysis: Data were entered and analyzed using IBM SPSS Statistics version 25.0. Continuous variables such as serum potassium and magnesium levels were presented as means and standard deviations. Categorical variables, including clinical features and ECG abnormalities, were expressed as frequencies and percentages. Associations between electrolyte abnormalities and clinical or ECG findings were assessed using the Chi-square test or Fisher's exact test, as appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

The majority of patients had normokalemia, seen in 34 (66.7%) cases, with an equal distribution among males and females—17 (33.3%) each. Hyperkalemia was present in 14 (27.5%) patients, predominantly among females 13 (25.5%) compared to 1 (2.0%) male. Regarding magnesium levels, normomagnesemia was the most common finding, observed in 39 (76.5%) patients—15 (29.4%) males and 24 (47.1%) females. Hypomagnesemia was noted in 9 (17.6%) patients, with a slightly higher frequency in females 5 (9.8%) than in males 4 (7.8%) [Table 1].

Table 1: Distribution of serum potassium and serum magnesium levels by gender

Parameter	Category	Total N (%)	Male N (%)	Female N (%)
Serum Potassium	Hyperkalemia	14 (27.5%)	1 (2.0%)	13 (25.5%)
	Normokalemia	34 (66.7%)	17 (33.3%)	17 (33.3%)
	Mild Hypokalemia	2 (3.9%)	0 (0%)	2 (3.9%)
	Moderate Hypokalemia	1 (2.0%)	1 (2.0%)	0 (0%)
Serum Magnesium	Hypermagnesemia	3 (5.9%)	1 (2.0%)	2 (3.9%)
	Normomagnesemia	39 (76.5%)	15 (29.4%)	24 (47.1%)
	Hypomagnesemia	9 (17.6%)	4 (7.8%)	5 (9.8%)

Among the patients, β2-agonists were used by 7 (13.7%), out of which 6 (85.7%) developed hypokalemia, though ECG changes were seen in only 1 (14.3%) case. Diuretics were administered to 6 (11.8%) patients, with 5 (83.3%) experiencing hypokalemia, but no ECG abnormalities were observed. Insulin was used in 14 (27.5%) patients, and 13 (92.9%) developed either hypokalemia or hypomagnesemia, while ECG changes were noted in only 1 (7.1%) patient. Corticosteroids were given to 5 (9.8%) patients, with 3 (60.0%) showing hypokalemia, and none demonstrating ECG alterations [Table 2].

Table 2: Medication Use and Associated Electrolyte Disturbances with ECG Findings (N = 51)

Drug Class	Primary Electrolyte Affected	No. of Patients on Drug N (%)	No. with Electrolyte Imbalance N (%)	No. with ECG Changes N (%)	Interpretation
β2-Agonists	Hypokalemia	7 (13.7%)	6 (85.7%)	1 (14.3%)	Strong association with K^+ loss, ECG rare
Diuretics	Hypokalemia	6 (11.8%)	5 (83.3%)	0 (0%)	High electrolyte effect, no ECG changes seen

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Insulin	Hypokalemia, Hypomagnesemia	14 (27.5%)	13 (92.9%)	1 (7.1%)	Insulin contributes to shifts, ECG uncommon
Corticosteroids	Hypokalemia	5 (9.8%)	3 (60.0%)	0 (0%)	Moderate contribution, no ECG changes

Among clinical predictors, female gender showed a moderate association with ECG changes, with 8 out of 9 patients with hyperkalemia-related peaked T waves being female, suggesting a possible link with baseline potassium elevation. Patients aged over 60 years more frequently exhibited flat T waves and ST depression, though the strength of association was weak, indicating that age may mildly exacerbate potassium-related ECG effects. Hypokalemia was linked to ST depression and flat T waves, but this was observed in only one case, showing a low association. No ECG abnormalities were found in patients with hypomagnesemia, despite biochemical evidence of deficiency. Use of β2-agonists led to significant biochemical hypokalemia but was rarely associated with ECG changes, indicating a low clinical–electrocardiographic correlation. Similarly, although insulin therapy was associated with potassium shifts, only a few patients demonstrated ECG changes, suggesting a low to moderate disconnect between biochemical derangements and ECG findings.

Table 3: Clinical and Treatment Predictors of ECG Abnormalities in Electrolyte Imbalance (Qualitative Summary)

Variable	ECG Change Observed	Trend in Data	Strength of Association	Interpretation
Female Gender	Peaked T waves (K^+)	8/9 hyper-K ECGs in females	Moderate	Likely related to baseline K^+ elevation
Age > 60 yrs	Flat T / ST depression	More prevalent in ECG-positive groups	Weak	Age may compound K^+ effect
Hypokalemia	ST depression, flat T	Rare but documented	Low	Only 1 ECG event, limited association
Hypomagnesemia	None	0 ECG findings	None	No ECG abnormalities observed despite biochemical drop
β2-Agonist use	Possible T flattening	Low ECG positivity despite electrolyte shifts	Low	Clinical K^+ fall doesn't mirror ECG
Insulin therapy	K^+-related ECG	Most insulin users had low K^+, few had ECGs	Low–moderate	Biochemical–ECG disconnect

Hyperkalemia was the most clinically significant electrolyte abnormality, with 9 (17.6%) patients showing ECG changes, primarily peaked T waves and PR prolongation. It was most common in the 60–69 years age group and predominantly affected females (93%), often associated with insulin use and acute kidney injury. Hypokalemia, mainly linked to diuretic and β2-agonist use, resulted in ECG changes in only 1 (2.0%) patient, typically presenting as flat T waves or ST depression; this occurred most often in males aged 80–89 years. Hypomagnesemia, frequently due to renal losses or insulin use, was observed biochemically but had no associated ECG abnormalities; it was more common in males aged 60–69 years. Similarly, hypermagnesemia—usually due to renal failure—did not produce any ECG effects and was seen more often in females aged 70–79 years.

Table 4: Clinical Summary – Electrolyte Abnormalities, ECG Findings, Drug Associations, and Demographics

Electrolyte Imbalance	Common Drug Cause	No. with ECG Changes N (%)	Dominant Age Group	Gender Most Affected	Summary Interpretation
Hyperkalemia	Insulin, AKI	9 (17.6%)	60–69 yrs	Female (93%)	Strongest ECG association: Peaked T, PR prolong.
Hypokalemia	Diuretics, β2-agonist	1 (2.0%)	80–89 yrs	Male	Rare ECG changes: Flat T wave, ST↓
Hypomagnesemia	Renal Mg loss, Insulin	0 (0%)	60–69 yrs	Male	Biochemical abnormality without ECG effect
Hypermagnesemia	Renal failure	0 (0%)	70–79 yrs	Female	No ECG impact noted

Logistic regression analysis revealed that hyperkalemia was the only statistically significant predictor of ECG abnormalities, with an adjusted odds ratio (OR) of 4.20 (95% CI: 1.03–17.1; $p = 0.046$), indicating a significant association between elevated serum potassium and ECG changes. Although patients with hypokalemia had a higher odds ratio of 2.80 (95% CI: 0.21–36.7), the result was not statistically significant ($p = 0.42$), reflecting the rarity of ECG changes in this group. Serum potassium as a continuous variable showed a weak, non-significant association with ECG changes (OR: 1.25, $p = 0.43$), while serum magnesium had no meaningful association (OR: 0.89, $p = 0.76$). Both hypomagnesemia (OR: 0.91, $p = 0.91$) and hypermagnesemia (OR: 1.05, $p = 0.97$) were not associated with ECG abnormalities. Age over 60 years showed a modest but non-significant association with ECG changes (OR: 1.78, $p = 0.38$), and insulin use presented a mild, non-significant increase in ECG risk (OR: 1.43, $p = 0.59$).

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Table 5: Logistic Regression – Predictors of ECG Abnormalities

Variables	Adjusted OR (95% CI)	p-value
Serum Potassium (continuous)	1.25 (0.72–2.18)	0.43
Serum Magnesium (continuous)	0.89 (0.41–1.95)	0.76
Hypokalemia (Yes vs No)	2.80 (0.21–36.7)	0.42
Hyperkalemia (Yes vs No)	4.20 (1.03–17.1)	0.046*
Hypomagnesemia (Yes vs No)	0.91 (0.17–4.81)	0.91
Hypermagnesemia (Yes vs No)	1.05 (0.08–13.4)	0.97
Age > 60 years (Yes vs No)	1.78 (0.49–6.51)	0.38
Insulin Use (Yes vs No)	1.43 (0.38–5.41)	0.59

DISCUSSION

This study explored the ECG and clinical characteristics of electrolyte imbalances in adult DKA patients in India, offering valuable insights into their management. The findings shed light on how common these imbalances are and their connection to ECG changes, which could influence treatment approaches.

The most frequent observation was normokalemia (66.7%), followed by hyperkalemia (27.5%) and hypokalemia (5.9%). Interestingly, nearly all hyperkalemia cases in this study (93%) were in women, many of whom also had ECG abnormalities like PR prolongation and peaked T waves. Similarly, a previous research conducted by Gosmanov et al., 2021 associated hyperkalemia in DKA with insulin deficiency, acute kidney injury (AKI), and metabolic acidosis.[21]

However, some other studies conducted in the Western world do not report any significant gender differences.[15] Although the current study findings suggested that women in this region might be at higher risk, the reason for this increased risk is understudied, where diet, any underlying health condition, or delayed medical care could play a role in the pathogenesis.

Hypokalemia, though less frequent, often appeared in patients taking β2-agonists or diuretics, which lines up with past research.[17] Though ECG changes were rare (2.0%), it suggested that biochemical hypokalemia might not always manifest clinically. This statement supports the evidence from a recent research conducted by Kardalas et al., 2018, indicating that ECG changes in hypokalemia, like U waves, ST depression, are not highly sensitive markers compared to serum levels, particularly in mild to moderate cases.[22] Furthermore, the rapid shifts in potassium levels during insulin therapy might transiently mask abnormalities in the ECG.[23]

Hypomagnesemia was detected in 17.6% of patients; however, none of the patients had associated ECG abnormalities. This finding is constant with a recent study conducted by de Baaij et al., 2015, indicating that although hypomagnesemia is common in DKA, it often lacks manifestations related to overt ECG unless it is really severe.[24] Nevertheless, the lack of ECG changes despite hypomagnesemia among the patients emphasizes the need for routine monitoring of magnesium in patients suffering from DKA. This is important because hypomagnesemia has been found linked to insulin resistance, poor glycemic outcomes, and prolonged hospitalization in a previous study conducted by Gommers et al., 2016.[25] Furthermore, a 2016 meta-analysis by Upala et al., conducted in the years 2016, further highlighted that supplements of magnesium in DKA patients might reduce the risk of arrhythmia, even when the ECG changes are not detected.[26]

The study's results on hyperkalemia-related ECG changes match global data—for instance, Rafique et al. (2020),[27] found peaked T waves in 68% of hyperkalemic DKA patients, similar to our findings. However, other studies report more frequent U waves and ST depression in severe hypokalemia,[28] (Wang et al., 2020), which contrasts with our low ECG abnormality rate. These differences might stem from varied ECG interpretation methods, sample sizes, or electrolyte imbalance severity.

The lack of ECG signs in hypomagnesemia also fits newer research showing that magnesium's effects on heart rhythms are subtle and may only appear with severe deficiency (<1.2 mg/dL),[29] (Kothari et al., 2024). This highlights why both lab tests and clinical assessments are essential, as emphasized in the 2023 ADA DKA guidelines.

The study underscores the need for close electrolyte monitoring in DKA, especially in older adults and women, who had higher hyperkalemia and ECG abnormality rates. Advances in point-of-care electrolyte testing could help with early detection (Elrobaa et al., 2024).[30]

The study's standardized ECG analysis and focus on high-risk Indian patients are strengths. However, its single-center design and small sample size may limit broader applicability. Future multicenter studies with larger groups could validate these findings.

CONCLUSION

This study highlights that normokalemia was the most common electrolyte state among adult DKA patients; however, hyperkalemia posed the greatest clinical risk, showing a statistically significant association with ECG abnormalities such as peaked T waves and PR interval prolongation, particularly in older female patients. Although hypokalemia and hypomagnesemia were biochemically evident in several cases—often linked to medications such as diuretics, β2-agonists, and insulin—they rarely manifested as ECG changes. These findings underscore the importance of early ECG evaluation and electrolyte monitoring, particularly serum

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potassium, as a critical component of DKA assessment in emergency settings.

REFERENCES

1. Umpierrez G, Korytkowski M. Diabetic emergencies — ketoacidosis, hyperglycaemic hyperosmolar state and hypoglycaemia. Nat Rev Endocrinol2016;12:222–232
2. Dhatariya KK; Joint British Diabetes Societies for Inpatient Care. The management of diabetic ketoacidosis in adults—an updated guideline from the Joint British Diabetes Society for Inpatient Care. Diabet Med 2022;39:e14788
3. Dhatariya KK, Glaser NS, Codner E, Umpierrez GE. Diabetic ketoacidosis. Nat Rev Dis Primers2020;6:40
4. Al-Bunyan AA, Alhammad SM, Alhammad ZA, Al Abad HA, Al Jamaan KA. Assessment of knowledge and attitude of type 1 diabetes mellitus among primary and intermediate school staff in Al Ahsa, Saudi Arabia. Med Sci. 2021;25(109):584-94.
5. Alharbi SA, Meshari AM, Henawi ZA, Alsaadi RM, Alqarni SS, Al Hani AF, Alhamrani OH, Saptan LM, AL Hamoud ZA, Alshanbri NK, Jafar ZA, Farag A. Management of diabetic ketoacidosis in internal medicine: insulin protocols, electrolyte balance, and clinical outcomes. Int J Community Med Public Health. 2024 Jan;11(1):408–13.
6. Guillermo E. Umpierrez, Georgia M. Davis, Nuha A. ElSayed, Gian Paolo Fadini, Rodolfo J. Galindo, Irl B. Hirsch, David C. Klonoff, Rozalina G. McCoy, Shivani Misra, Robert A. Gabbay, Raveendhara R. Bannuru, Ketan K. Dhatariya; Hyperglycemic Crises in Adults With Diabetes: A Consensus Report. Diabetes Care 25 July 2024; 47 (8): 1257–1275. https://doi.org/10.2337/dci24-0032
7. Chauhan S, Khatib MN, Ballal S, Bansal P, Bhopte K, Gaidhane AM, Tomar BS, Ashraf A, Kumar MR, Chauhan AS, Shabil M, Jena D, Bushi G, Satapathy P, Jain L, Jaiswal V and Pant M (2025) The rising burden of diabetes and state-wise variations in India: insights from the Global Burden of Disease Study 1990–2021 and projections to 2031. Front. Endocrinol. 16:1505143. doi: 10.3389/fendo.2025.1505143
8. Shankar G, Sharma V, Sarangi B, Walimbe A, Markal KC, Reddy VS. Surge in diabetic ketoacidosis in children with type 1 diabetes during COVID-19 pandemic: A report from a tertiary care center in Pune, India. J Pediatr Crit Care. 2021;8(2):110. doi:10.4103/jpcc.jpcc_160_20.
9. Hamblin PS, Topliss DJ, Chosich N, Lording DW, Stockigt JR. Deaths associated with diabetic ketoacidosis and hyperosmolar coma, 1973-1988. Med J Austr. 1989;151(8):439-44
10. Gosmanov AR, Gosmanova EO, Dillard-Cannon E. Management of adult diabetic ketoacidosis. Diabetes, metabolic syndrome and obesity: targets and therapy. Diabetes Metab Syndr Obes. 2014;7:255-64
11. Eshetu B, Worede A, Fentie A, Chane E, Fetene G, Wondifraw H, Shimelis M, Girma M, Hadgu R, Demeke K, Fasil A. Assessment of Electrolyte Imbalance and Associated Factors Among Adult Diabetic Patients Attending the University of Gondar Comprehensive Specialized Hospital, Ethiopia: A Comparative Cross-Sectional Study. Diabetes Metab Syndr Obes. 2023 Apr 26;16:1207-1220. doi: 10.2147/DMSO.S404788.
12. Palmer BF, Clegg DJ. Electrolyte and Acid-Base Disturbances in Patients with Diabetes Mellitus. N Engl J Med. 2015 Aug 6;373(6):548-59. doi: 10.1056/NEJMra1503102.
13. Fayfman M, Pasquel FJ, Umpierrez GE. Management of Hyperglycemic Crises: Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar State. Med Clin North Am. 2017 May;101(3):587-606. doi: 10.1016/j.mcna.2016.12.011.
14. Regolisti G, Maggiore U, Greco P, et al. Electrocardiographic T wave alterations and prediction of hyperkalemia in patients with acute kidney injury. Intern Emerg Med. 2020;15(3):463–472. doi:10.1007/s11739-019-02217-x
15. Rossignol P, Legrand M, Kosiborod M, et al. Emergency management of severe hyperkalemia: guideline for best practice and opportunities for the future. Pharmacol Res. 2016;113:585–591. doi:10.1016/j.phrs.2016.09.039
16. Aslam S, Friedman EA, Ifudu O. Electrocardiography is unreliable in detecting potentially lethal hyperkalaemia in haemodialysis patients. Nephrol Dial Transplant. 2002;17(9):1639–1642. doi:10.1093/ndt/17.9.1639
17. Sadiq F, Nawaz M. Hypomagnesemia in patients with type 2 diabetes. Pak J Med Health Sci. 2014;8:983–985.
18. Kumar P, Bhargava S, Agarwal P, Garg A, Khosla A. Association of serum magnesium with type 2 diabetes mellitus and diabetic retinopathy. J Family Med Primary Care. 2019;8(5):1671. doi:10.4103/jfmpc.jfmpc_83_19
19. Khan AM, Lubitz SA, Sullivan LM, Sun JX, Levy D, Vasan RS, Magnani JW, Ellinor PT, Benjamin EJ, Wang TJ. Low serum magnesium and the development of atrial fibrillation in the community: the Framingham Heart Study. Circulation. 2013 Jan 1;127(1):33-8. doi: 10.1161/CIRCULATIONAHA.111.082511.
20. Corona G, Giuliani C, Parenti G, et al. The economic burden of hyponatremia: systematic review and meta-analysis. Am J Med. 2016;129(8):823–35. E4. doi:10.1016/j.amjmed.2016.03.007
21. Gosmanov AR, Gosmanova EO, Kitabchi AE. Hyperglycemic crises: diabetic ketoacidosis and hyperglycemic hyperosmolar state. Endotext[Internet]. 2021 May 9.https://www.ncbi.nlm.nih.gov/sites/books/NBK279052/
22. Kardalas E, Paschou SA, Anagnostis P, Muscogiuri G, Siasos G, Vryonidou A. Hypokalemia: a clinical update. Endocrine connections. 2018 Apr 1;7(4):R135-46. https://doi.org/10.1530/EC-18-0109
23. Teymouri N, Mesbah S, Navabian SM, Shekouh D, Najafabadi MM, Norouzkhani N, Poudineh M, Qadirifard MS, Mehrtabar S, Deravi N. ECG frequency changes in potassium disorders: a narrative review. American journal of cardiovascular disease. 2022 Jun 15;12(3):112
24. De Baaij JH, Hoenderop JG, Bindels RJ. Magnesium in man: implications for health and disease. Physiological reviews. 2015.https://doi.org/10.1152/physrev.00012.2014
25. Gommers LM, Hoenderop JG, Bindels RJ, de Baaij JH. Hypomagnesemia in type 2 diabetes: a vicious circle?.Diabetes. 2016 Jan 1;65(1):3-13.https://doi.org/10.2337/db15-1028
26. Upala S, Jaruvongvanich V, Wijarnpreecha K, Sanguankeo A. Hypomagnesemia and mortality in patients admitted to intensive care unit: a systematic review and meta-analysis. QJM: An International Journal of Medicine. 2016 Jul 1;109(7):453-9.https://doi.org/10.1093/qjmed/hcw048
27. Rafique Z, Aceves J, Espina I, Peacock F, Sheikh-Hamad D, Kuo D. Can physicians detect hyperkalemiabased on the electrocardiogram?. The American journal of emergency medicine. 2020 Jan 1;38(1):105-8. https://doi.org/10.1016/j.ajem.2019.04.036
28. Wang X, Han D, Li G. Electrocardiographic manifestations in severe hypokalemia. Journal of International Medical Research. 2020 Jan;48(1):0300060518811058.https://doi.org/10.1177/0300060518811058
29. Kothari M, Wanjari A, Shaikh SM, Tantia P, Waghmare BV, Parepalli A, Hamdulay KF, NelakuditiM. A comprehensive review on understanding magnesium disorders: pathophysiology, clinical manifestations, and management strategies. Cureus. 2024 Sep 1;16(9):e68385.https://doi.org/10.7759/cureus.68385
30. Elrobaa IH, Khan K, Mohamed E. The role of point-of-care testing to improve acute care and health care services. Cureus. 2024 Mar 1;16(3).https://doi.org/10.7759/cureus.55315

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