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Correlation Between Glycated Hemoglobin (HbA1c) Levels and Serum Electrolyte Imbalance in Patients with Type 2 Diabetes Mellitus: A Hospital-Based Analytical Cross-Sectional Study.

Dr. B. Divya Dharshini[1*], Dr. Syeda Noorulain[2], Dr. Amancha Prathyusha[3]
[1]Assistant Professor, Department of Biochemistry, Government Medical College, Khammam, Telangana, India.
[2]Assistant Professor, Department of Biochemistry, Government Medical College, Rajanna Sircilla, Telangana, India.
[3]Assistant Professor, Department of Biochemistry, Government Medical College, Medak, Telangana, India.

Abstract

Background: Type 2 diabetes mellitus is frequently complicated by alterations in body water and renal tubular handling of ions, leading to clinically relevant electrolyte disturbances. These abnormalities can aggravate neuromuscular symptoms, cardiac excitability, and outcomes, yet they are often overlooked in routine glycemic follow-up.

Objectives: To evaluate the pattern of serum electrolyte imbalance in adults with type 2 diabetes mellitus and to assess the relationship between HbA1c and key serum electrolytes.

Methods: A hospital-based cross-sectional study was conducted among 100 adults with type 2 diabetes mellitus. Venous blood samples were analyzed for HbA1c and serum sodium, potassium, chloride, calcium, and magnesium. Electrolyte imbalance frequencies were summarized, and Pearson's correlation was used to examine associations between HbA1c and electrolyte concentrations.

Results: Participants had a mean age of 55.8 ± 10.2 years, and 60% were men. Mean HbA1c was 8.6 ± 1.7%. Overall, electrolyte imbalance was detected in 46% of patients. Hyponatremia was the most frequent abnormality (29%), followed by hypomagnesemia (21%) and hypocalcemia (17%). HbA1c showed a significant inverse correlation with sodium (r = -0.44) and magnesium (r = -0.39), whereas correlations with potassium, chloride, and calcium were not statistically significant.

Conclusion: Poorer glycemic control was associated with lower serum sodium and magnesium levels. A substantial proportion of adults with type 2 diabetes exhibited at least one electrolyte abnormality, underscoring the need for concurrent biochemical monitoring during routine diabetes care.

Recommendations: Periodic electrolyte profiling, especially sodium and magnesium, should be integrated into the follow-up of patients with elevated HbA1c, alongside targeted counseling on hydration, diet, and adherence to therapy.

Keywords: *Type 2 diabetes mellitus; Glycated hemoglobin, sodium; magnesium; electrolyte imbalance; hyponatremia.*
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Corresponding author: *Dr. B. Divya Dharshini*
Email: *divyadharshinib@gmail.com*
Assistant Professor, Department of Biochemistry, Government Medical College, Khammam, Telangana, India.

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by sustained hyperglycemia and progressive beta-cell dysfunction. Beyond microvascular and macrovascular complications, diabetes perturbs fluid balance and renal tubular transport, creating a biochemical milieu that favors electrolyte disturbances[1]. In clinical practice, these abnormalities often remain subclinical until they precipitate symptoms such as muscle weakness, cramps, altered sensorium, or arrhythmias. Recognizing electrolyte imbalance early is therefore relevant not only for acute safety, but also for overall metabolic stability.

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Glycated hemoglobin (HbA1c) reflects average glycemia over approximately 2-3 months and is widely used to monitor long-term control and guide treatment intensification [1]. Current standards emphasize individualized glycemic goals, with HbA1c values below 7% commonly targeted for many non-pregnant adults when safely achievable [2]. However, HbA1c is more than a tracking tool; it can also serve as a surrogate for the chronic osmotic and hormonal stresses that influence renal handling of sodium, potassium, chloride, calcium, and magnesium. Several pathophysiological pathways link hyperglycemia to electrolyte derangements. Glucosuria-driven osmotic diuresis promotes urinary loss of water and cations, while dehydration and counter-regulatory hormone activation can further alter tubular reabsorption. Hyperglycemia also lowers measured serum sodium concentration by shifting water from the intracellular to the extracellular compartment; correction factors have been described to interpret sodium values in this setting [3]. Potassium balance is influenced by insulin activity and acid-base status, whereas calcium and magnesium homeostasis are affected by renal excretion, dietary intake, and chronic inflammation. Magnesium deserves particular attention because it acts as a cofactor in hundreds of enzymatic reactions and participates in insulin receptor phosphorylation, glucose transport, and intracellular signaling. Experimental and clinical work indicates that magnesium deficiency can contribute to insulin resistance and impaired insulin action [4,5]. Observational studies also report associations between magnesium status, glycemic control, and diabetes complications such as retinopathy and nephropathy [6]. Intervention studies and meta-analyses suggest that magnesium replacement or supplementation can improve certain metabolic indices, although effects on HbA1c vary across trials. Recent cross-sectional evidence has explored how electrolyte profiles relate to HbA1c and treatment patterns in people with diabetes [7,8]. Even so, local data from Indian tertiary care settings remain limited, and regional dietary patterns, comorbidities, and medication use can modify electrolyte risk. Establishing the frequency of electrolyte imbalance and its relationship with HbA1c within routine outpatient and inpatient services can support pragmatic screening strategies and targeted counseling. The objectives of this study were to describe serum sodium, potassium, chloride, calcium, and magnesium levels in adults with T2DM and to determine the correlation between HbA1c and these electrolyte parameters.

Materials and Methods

Study design and setting

This hospital-based analytical cross-sectional study was conducted in the Department of General Medicine, Government Medical College and General Hospital, Khammam, Telangana, India, in collaboration with the Department of Biochemistry. The study was carried out over a six-month period from May 2025 to October 2025. The objective was to assess the pattern of serum electrolyte abnormalities among adults with type 2 diabetes mellitus and to determine their correlation with glycated hemoglobin (HbA1c) levels.

Participants

Adults aged 18 years and above with a confirmed clinical diagnosis of type 2 diabetes mellitus were screened consecutively from outpatient clinics and inpatient services under the Department of General Medicine. Participants were included if they were willing to provide written informed consent and had complete laboratory data for HbA1c and serum electrolytes, including sodium, potassium, chloride, calcium, and magnesium.

Patients were excluded if they had acute gastrointestinal fluid loss, sepsis, diabetic ketoacidosis, hyperosmolar hyperglycemic state, recent intravenous fluid therapy, known advanced chronic kidney disease, pregnancy, or current use of medications or supplements that could substantially alter serum electrolyte levels, such as loop or thiazide diuretics and magnesium supplementation. These criteria were applied to minimize the influence of acute physiological changes and major confounding factors on electrolyte measurements.

Study size

The sample size was calculated for detecting a correlation between HbA1c and serum electrolyte levels. Assuming a minimum clinically meaningful correlation coefficient of 0.30, 95% confidence level, and 80% power, the sample size was estimated using the formula for correlation studies:

$n = [(Z\alpha + Z\beta) / C]^2 + 3$

where $C = 0.5 \times \ln [(1 + r) / (1 - r)]$

For $r = 0.30$,

$C = 0.5 \times \ln [(1 + 0.30) / (1 - 0.30)]$

$C = 0.5 \times \ln (1.857)$

$C = 0.309$

Using $Z\alpha = 1.96$ and $Z\beta = 0.84$,

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$n = [(1.96 + 0.84) / 0.309]^2 + 3$
$n = [2.80 / 0.309]^2 + 3$
$n = 82.1 + 3$
$n \approx 85$

After allowing for approximately 15% non-response or incomplete laboratory records, the adjusted sample size was calculated as:

Adjusted sample size = 85 / (1 − 0.15)
Adjusted sample size = 100

Therefore, 100 participants were enrolled in the final study.

Data collection and laboratory methods

Data were collected using a structured proforma that included age, sex, duration of diabetes, current antidiabetic treatment, and relevant comorbidities. Venous blood samples were collected under aseptic precautions and transported promptly to the central laboratory for analysis. HbA1c was measured using a standardized laboratory method suitable for clinical monitoring [1]. Serum sodium, potassium, and chloride were measured using an ion-selective electrode platform. Total calcium and magnesium were estimated using automated photometric methods. Routine calibration and internal quality-control procedures were followed throughout the study period.

Operational definitions

Electrolyte imbalance was defined using standard adult reference cut-off values. Hyponatremia was defined as serum sodium <135 mmol/L, hypokalemia as serum potassium <3.5 mmol/L, hypochloremia as serum chloride <98 mmol/L, hypocalcemia as serum calcium <8.5 mg/dL, and hypomagnesemia as serum magnesium <1.7 mg/dL. Overall, electrolyte imbalance was defined as the presence of at least one abnormal electrolyte value. Glycemic control was categorized as good control when HbA1c was <7%, moderate control when HbA1c was 7–8.9%, and poor control when HbA1c was ≥9%, in accordance with commonly used clinical targets [2].

Bias

Efforts were made to reduce potential sources of bias at different stages of the study. Consecutive enrolment of eligible patients was followed to limit selection bias. Uniform inclusion and exclusion criteria were applied to all participants. Patients with acute metabolic emergencies, advanced renal disease, recent intravenous fluid therapy, and drug or supplement use known to alter electrolyte levels were excluded to reduce confounding. Laboratory investigations were performed in the same institutional laboratory using standardized methods, calibrated equipment, and internal quality-control procedures to minimize measurement bias. A structured data collection form was used for all participants, and laboratory reports were checked for completeness before analysis to reduce information bias.

Statistical analysis

Data were analyzed using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Pearson's correlation coefficient was used to assess the relationship between HbA1c and individual serum electrolyte levels. The direction and strength of correlation were interpreted in a clinical context. A two-tailed p-value of <0.05 was considered statistically significant.

Ethical considerations

Institutional Ethics Committee approval was obtained from the Government Medical College and General Hospital, Khammam, Telangana, India, before initiation of the study. All eligible participants were informed about the purpose of the study, the nature of blood sample collection, confidentiality of data, and their right to withdraw from the study at any stage without affecting their routine medical care. Written informed consent was obtained from each participant before enrolment. Participant confidentiality was maintained by using coded identifiers, and all collected data were stored securely with access limited to the investigators.

Results

Participant flow

During the study period, 128 adults with clinically diagnosed type 2 diabetes mellitus who attended the outpatient clinics or were admitted under the Department of General Medicine were considered potentially eligible. All 128 patients were examined for eligibility according to the predefined inclusion and exclusion criteria. Of these, 28 patients were not included in the final study. The reasons for non-inclusion were acute gastrointestinal fluid loss in 4 patients, sepsis in 3 patients, diabetic ketoacidosis or hyperosmolar hyperglycemic state in 4 patients, recent intravenous fluid therapy in 3 patients, advanced chronic kidney disease in 5 patients, pregnancy in 2 patients, use of

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diuretics or magnesium supplementation in 4 patients, and unwillingness to provide written informed consent in 3 patients.

After eligibility assessment, 100 participants fulfilled the study criteria and provided written informed consent. All 100 participants underwent clinical data collection and laboratory evaluation for glycated hemoglobin and serum electrolytes. There were no missing laboratory values for the primary variables, and no participant was excluded after enrolment. Therefore, all 100 enrolled participants were included in the final statistical analysis.

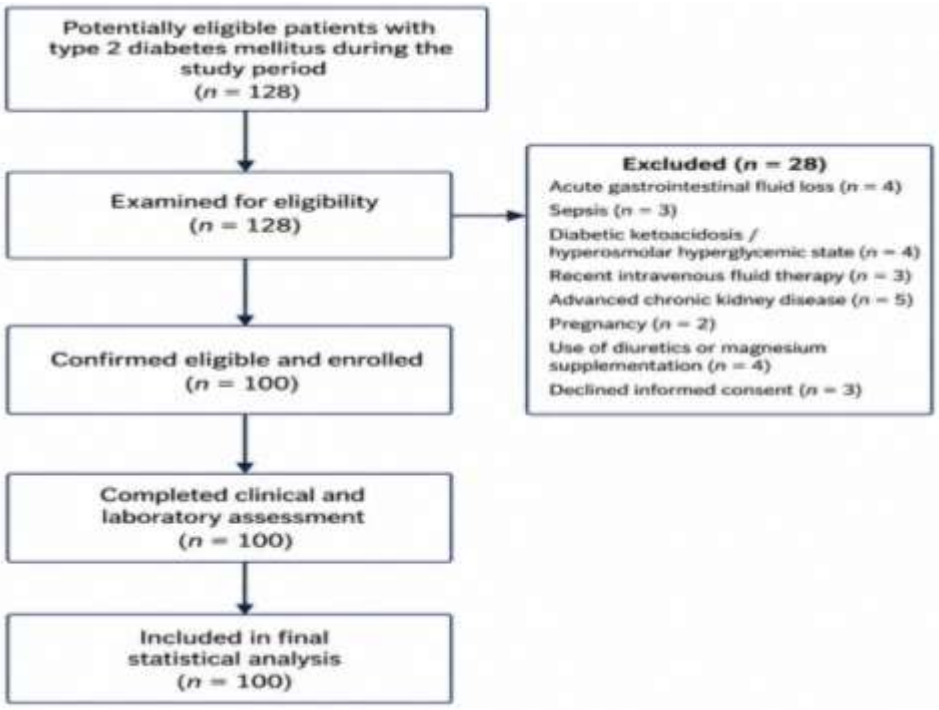

Figure 1: Participant Flow Diagram

A total of 100 adults with T2DM were included. The mean age was 55.8 ± 10.2 years, and 60% were men. Mean duration of diabetes was 7.9 ± 4.3 years, with an overall mean HbA1c of 8.6 ± 1.7%. Baseline characteristics are summarized in Table 1.

Table 1. Baseline Characteristics of Study Participants (n = 100)

Variable	Value
Age (years), mean ± SD	55.8 ± 10.2
Male, n (%)	60 (60%)
Female, n (%)	40 (40%)
Duration of diabetes (years), mean ± SD	7.9 ± 4.3
HbA1c (%), mean ± SD	8.6 ± 1.7

When categorized by glycemic control, 22% had HbA1c <7% (good control), 48% had HbA1c 7-8.9% (moderate control), and 30% had HbA1c >=9% (poor control), as shown in Table 2.

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Table 2. Distribution of Participants According to HbA1c Categories (n = 100)

HbA1c category	Number (n)	Percentage (%)
< 7% (Good control)	22	22%
7-8.9% (Moderate control)	48	48%
>= 9% (Poor control)	30	30%

Mean serum sodium was 134.5 ± 5.2 mmol/L, with hyponatremia observed in 29% of participants. Hypomagnesemia (21%) and hypocalcemia (17%) were also frequent. Overall, at least one electrolyte imbalance was present in 46% of patients (Table 3).

Table 3. Serum Electrolyte Levels and Frequency of Imbalance (n = 100)

Parameter	Mean ± SD	Electrolyte imbalance n (%)
Sodium (mmol/L)	134.5 ± 5.2	Hyponatremia - 29 (29%)
Potassium (mmol/L)	4.2 ± 0.7	Hypokalemia - 11 (11%)
Chloride (mmol/L)	100.8 ± 4.9	Hypochloremia - 9 (9%)
Calcium (mg/dL)	8.6 ± 0.7	Hypocalcemia - 17 (17%)
Magnesium (mg/dL)	1.8 ± 0.3	Hypomagnesemia - 21 (21%)

Overall, electrolyte imbalance was observed in 46 (46%) participants.
Correlation analysis demonstrated a significant negative association between HbA1c and serum sodium ($r = -0.44$, $p < 0.001$) and between HbA1c and serum magnesium ($r = -0.39$, $p = 0.001$). Correlations with potassium, chloride, and calcium were not statistically significant (Table 4).

Table 4. Correlation Between HbA1c and Serum Electrolytes (n = 100)

Electrolyte	Correlation coefficient (r)	p-value	Interpretation
Sodium	-0.44	<0.001	Significant negative correlation
Potassium	-0.19	0.06	Not statistically significant
Chloride	-0.16	0.11	Not statistically significant
Calcium	-0.22	0.07	Not statistically significant
Magnesium	-0.39	0.001	Significant negative correlation

Discussion

The present hospital-based analytical cross-sectional study demonstrates that electrolyte abnormalities are common among adults with type 2 diabetes mellitus. In this cohort of 100 participants, the mean age was 55.8 ± 10.2 years, 60% were men, and the mean duration of diabetes was 7.9 ± 4.3 years. Glycemic control was suboptimal in a large proportion of the study population, with a mean HbA1c of 8.6 ± 1.7%. Only 22% of participants had good glycemic control with HbA1c <7%, whereas 48% had moderate control with HbA1c between 7% and 8.9%, and 30% had poor control with HbA1c ≥9%. These findings indicate that the study population represented a clinically relevant group of patients with persistent hyperglycemia, in whom biochemical monitoring is important [1,2].

A major finding of the study was the high burden of electrolyte imbalance. Overall, at least one electrolyte abnormality was observed in 46 of 100 participants, indicating that nearly half of the adults with type 2 diabetes mellitus had biochemical evidence of electrolyte disturbance. This finding aligns with contemporary cross-sectional reports that describe a substantial burden of electrolyte disturbances in routine diabetes populations [7,8]. The clinical implication is practical because

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electrolyte disturbances can contribute to neuromuscular symptoms, altered sensorium, cardiac excitability, and poor metabolic stability. Therefore, electrolyte screening should not be limited to acute inpatient care but should also be considered during routine diabetes follow-up, particularly in patients with elevated HbA1c.

Hyponatremia was the most frequent electrolyte abnormality in this cohort. The mean serum sodium level was 134.5 ± 5.2 mmol/L, and hyponatremia was detected in 29% of participants. A plausible explanation is the combined effect of hyperglycemia-related transcellular water shifts and osmotic diuresis. When extracellular glucose rises, water shifts from the intracellular to the extracellular compartment, leading to a dilutional reduction in measured serum sodium. Correction factors have been proposed to interpret serum sodium values in hyperglycemic states [9]. In addition, glucosuria-induced osmotic diuresis can increase urinary sodium and water losses, particularly when hydration and dietary intake are inconsistent. The significant inverse correlation between HbA1c and serum sodium observed in this study ($r = -0.44$, $p < 0.001$) supports the concept that chronic poor glycemic control is associated with lower sodium levels.

Magnesium showed the second strongest relationship with HbA1c. The mean serum magnesium level was 1.8 ± 0.3 mg/dL, and hypomagnesemia was observed in 21% of participants. HbA1c had a significant negative correlation with serum magnesium ($r = -0.39$, $p = 0.001$), indicating that poorer glycemic control was associated with lower magnesium concentrations. This association is biologically coherent because magnesium participates in insulin receptor activity, glucose transport, intracellular signaling, and several enzymatic reactions involved in carbohydrate metabolism. Experimental evidence indicates that magnesium deficiency can worsen insulin resistance and impair insulin action [10]. Observational studies have also associated lower magnesium levels with poorer glycemic indices and diabetes-related complications, including retinopathy and nephropathy [6,9,14].

Interventional studies have suggested that magnesium replacement can improve selected metabolic parameters in patients with hypomagnesemia, although the magnitude of HbA1c reduction varies across trials and follow-up durations [11,12]. Dysmagnesemia, including both hypo- and hypermagnesemia, has also been documented in broader diabetes cohorts, particularly in patients with impaired renal function [13]. In the present study, patients with advanced chronic kidney disease were excluded to reduce confounding; even after this exclusion, hypomagnesemia remained frequent, affecting more than one-fifth of the study population. This finding supports the need for magnesium monitoring in adults with type 2 diabetes mellitus, especially when HbA1c remains elevated.

Hypocalcemia was observed in 17% of participants, with a mean serum calcium level of 8.6 ± 0.7 mg/dL. However, the correlation between HbA1c and calcium was not statistically significant ($r = -0.22$, $p = 0.07$). This suggests that although hypocalcemia was present in a notable subset of patients, its relationship with chronic glycemic control was weaker than that observed for sodium and magnesium. Calcium homeostasis is influenced by vitamin D status, serum albumin, parathyroid hormone activity, renal function, and dietary intake. Since these variables were not assessed in the present study, the independent association between HbA1c and calcium could not be clearly established.

Potassium and chloride abnormalities were less frequent. The mean serum potassium level was 4.2 ± 0.7 mmol/L, and hypokalemia was observed in 11% of participants. The correlation between HbA1c and potassium was not statistically significant ($r = -0.19$, $p = 0.06$). Similarly, the mean serum chloride level was 100.8 ± 4.9 mmol/L, hypochloremia was found in 9% of participants, and its correlation with HbA1c was not statistically significant ($r = -0.16$, $p = 0.11$). Potassium balance is dynamic and is influenced by insulin therapy, renal handling, acid-base status, dietary intake, and acute cellular shifts. Chloride levels are also affected by hydration status and acid-base balance. Therefore, a single-time-point laboratory assessment can underestimate the relationship of these electrolytes with long-term glycemic control.

The present findings emphasize that sodium and magnesium deserve particular attention in patients with poorly controlled type 2 diabetes mellitus. Among all electrolytes assessed, sodium showed the strongest inverse correlation with HbA1c ($r = -0.44$, $p < 0.001$), followed by magnesium ($r = -0.39$, $p = 0.001$). These statistically significant associations suggest that worsening glycemic status is accompanied by greater biochemical vulnerability. In contrast, potassium, chloride, and calcium showed weaker and statistically non-significant associations with HbA1c, although their abnormalities remain clinically relevant in symptomatic patients and in those receiving medications that influence renal electrolyte handling.

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Generalizability

Generalizability of these findings is strongest for adults with T2DM receiving care in similar tertiary teaching hospitals in South India, where the case-mix includes both ambulatory patients and medically stable admissions. The reported correlations reflect routine biochemical testing rather than protocol-intensive metabolic phenotyping. Caution is warranted when extrapolating to community-managed cohorts, patients with advanced kidney disease, or those with acute metabolic decompensation, because electrolyte trajectories and treatment exposures in these settings differ markedly.

Conclusion

In this hospital-based cross-sectional study of 100 adults with type 2 diabetes mellitus, electrolyte imbalance was common, affecting 46% of participants. Hyponatremia (29%) and hypomagnesemia (21%) were the leading abnormalities. Mean HbA1c was elevated, and a sizeable subgroup had poor glycemic control (HbA1c >=9%). HbA1c showed a significant inverse association with serum sodium (r = -0.44) and magnesium (r = -0.39), indicating that poorer long-term glycemia coincided with lower concentrations of these ions. Potassium, chloride, and calcium correlations were not statistically significant. Integrating electrolyte assessment, particularly sodium and magnesium, into routine follow-up for patients with uncontrolled HbA1c can support safer, more comprehensive diabetes care and prompt early corrective counseling at each visit.

Limitations

This study used a cross-sectional design, so temporal direction and causality cannot be inferred. The sample size was modest, and recruitment was limited to a single tertiary-care center, which constrains external validity. Electrolytes were measured at one time point, and medication doses, dietary intake, albumin, and acid-base status were not quantified, leaving residual confounding. Advanced kidney disease and acute metabolic emergencies were excluded, limiting applicability to those groups.

Recommendations

Routine diabetes follow-up should include periodic measurement of serum sodium and magnesium, especially in patients with HbA1c >=9% or symptoms suggestive of electrolyte disturbance. Clinicians should reinforce adequate hydration, balanced dietary salt and magnesium intake, and adherence to prescribed antidiabetic therapy. When abnormalities are detected, medication review, renal function assessment, and stepwise correction using standard protocols are warranted, with ECG monitoring for significant potassium or calcium derangements. Patient education and counseling can improve recognition of symptoms. Future studies should use multicenter recruitment, capture dietary and drug exposures, and incorporate repeat measurements to model electrolyte trajectories alongside HbA1c and clinical outcomes.

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Abbreviations

HbA1c - Glycated hemoglobin
T2DM - Type 2 diabetes mellitus
SD - Standard deviation
ISE - Ion-selective electrode
ECG - Electrocardiogram

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The study had no funding.

Conflict of interest

The authors declare no conflict of interest.

Author contributions

BDD-Concept and design of the study, results interpretation, review of literature, and preparing the first draft of the manuscript. Statistical analysis and interpretation, revision of manuscript.
SN -results interpretation, review of literature, and preparing the first draft of the manuscript, and revision of the manuscript.
AP- review of literature and preparing the first draft of the manuscript, revision of the manuscript

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Data availability

Data available on request

Author Biography

Dr. B. Divya Dharshini is an Assistant Professor in the Department of Biochemistry at Government Medical College, Khammam, Telangana, India. She is actively involved in undergraduate medical education, laboratory-based teaching, and academic activities in biochemistry. Her professional interests include clinical biochemistry, medical education, and translational research relevant to patient care. She also contributes to scholarly writing and academic publication. She serves as the corresponding author for the present work. ORCID: http://orcid.org/0000-0002-8923-2398

Dr. Syeda Noorulain is an Assistant Professor in the Department of Biochemistry at Government Medical College, Rajanna Sircilla, Telangana, India. She is engaged in teaching, academic mentoring, and practical training for medical students in the field of biochemistry. Her areas of interest include clinical biochemistry, diagnostic laboratory sciences, and biochemical aspects of disease mechanisms. She has contributed to academic and research-oriented activities in medical education and biomedical sciences.

Dr. Amancha Prathyusha is an Assistant Professor in the Department of Biochemistry at Government Medical College, Medak, Telangana, India. She is involved in academic instruction, laboratory training, and curriculum-based teaching for undergraduate medical students. Her professional interests include applied biochemistry, clinical correlations in laboratory medicine, and biomedical research. She participates in academic development and research initiatives that support evidence-based medical practice.

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