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ORIGINAL ARTICLE

Association of Glycemic Status, Serum Potassium and Serum Magnesium Levels with Arrhythmia in Type 2 Diabetes Mellitus at a Tertiary Care Setting

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ABSTRACT

Background: *Type 2 Diabetes Mellitus is a chronic metabolic disorder associated with significant cardiovascular complications, including arrhythmias. This study aimed to assess the association of glycemic control, serum potassium, and serum magnesium levels with the occurrence of arrhythmia in patients with Type 2 Diabetes Mellitus at a tertiary care center.* ***Aim of the study:*** *The aim of the study was to evaluate the association of glycemic control, serum potassium, and serum magnesium levels with the occurrence of arrhythmia in patients with Type 2 Diabetes Mellitus at a tertiary care center.* ***Methods & Materials:*** *This cross-sectional study was conducted from March to August 2013 at the Departments of Internal Medicine, Cardiology, and Neurology, BIRDEM General Hospital, Dhaka. One hundred adults with type 2 diabetes and arrhythmia symptoms underwent 24-hour Holter monitoring. Data included clinical history, ECG, and lab tests (glucose, HbA1c, electrolytes, thyroid). Arrhythmias were classified; analysis used SPSS v10 with significance at $p < 0.05$.* ***Results:*** *Poor glycemic control (mean 2ABF 14.15 mmol/L, HbA1c 8.61%) was linked to higher ectopy. Patients with 2ABF ≥10 mmol/L had more ventricular (5596 vs. 3601; $p = 0.016$) and supraventricular ectopics (8266 vs. 4877; $p = 0.010$), showing a clear association between postprandial hyperglycemia and arrhythmia burden.* ***Conclusion:*** *Poor glycemic control and electrolyte imbalance, particularly low potassium, are significantly associated with increased arrhythmic burden in Type 2 Diabetes Mellitus.*

Key words: *Glycemic Status, Electrolyte Imbalance, Arrhythmia.*

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INTRODUCTION

Over the past few decades, Type 2 Diabetes Mellitus (T2DM) has become a major public health issue[1]. Worldwide, approximately 422 million adults are affected by diabetes mellitus (DM), according to the World Health Organization's 2016 report. T2DM is marked by both reduced insulin secretion and resistance to insulin, leading to persistent hyperglycemia[2]. This chronic elevation in blood glucose contributes to long-term cardiovascular complications, including coronary artery disease (CAD), myocardial infarction (MI), congestive heart failure (CHF), and sudden cardiac death due to arrhythmias[3,4]. Research has indicated a high prevalence of ventricular arrhythmias among individuals with T2DM[5].

Intensive glycemic control in diabetes, particularly when it leads to hypoglycemia, along with cardiac autonomic neuropathy (CAN), has been linked to altered heart rate variability and disturbances in ventricular repolarization[6]. Prolongation of the corrected QT interval (QTc), greater QT dispersion (QTd), and elevated HbA1c levels have all been associated with higher mortality rates, especially among elderly patients with type 2 diabetes[6]. Insulin resistance in these individuals may stem from a reduced number of insulin receptors on the cell membrane, diminished receptor affinity

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for insulin, or post-receptor signaling defects impairing insulin's cellular action[7].

Serum magnesium plays a critical role in maintaining various physiological functions[8]. As a key intracellular cation, magnesium serves as a cofactor in glucose phosphorylation and thus contributes significantly to glycemic control[9]. It supports essential cellular processes by participating in enzymatic reactions, nucleic acid stability, and energy metabolism[10]. Despite its importance, hypomagnesemia often goes undetected in individuals with diabetes[11]. Low magnesium levels have been linked to impaired glucose transport across cell membranes, decreased insulin secretion from the pancreas, disrupted post-receptor insulin signaling, and impaired insulin-receptor binding[12]. Furthermore, magnesium deficiency has been associated with several clinical conditions, including hypocalcemia, tetany, hypokalemia, cardiac arrhythmias, stroke, ischemic heart disease, electrolyte imbalances, and bronchial asthma[13].

Despite increasing awareness of cardiovascular risks in T2DM, limited research has concurrently examined the combined influence of glycemic status, serum potassium, and serum magnesium levels on arrhythmia risk in this population. Most studies have focused on isolated risk factors or specific arrhythmia types, leaving a gap in understanding their interrelationship in routine clinical settings. The purpose of the study was to assess the association of glycemic control, serum potassium, and serum magnesium levels with the occurrence of arrhythmia in patients with Type 2 Diabetes Mellitus at a tertiary care center.

OBJECTIVE

- To evaluate the association of glycemic control, serum potassium, and serum magnesium levels with the occurrence of arrhythmia in patients with Type 2 Diabetes Mellitus at a tertiary care center.

METHODS & MATERIALS

This cross-sectional study was conducted at the Departments of Internal Medicine, Cardiology, and Neurology of BIRDEM General Hospital, Shahbagh, Dhaka, Bangladesh, over a period of six months, from March to August 2013. A total of 100 adult patients were included to evaluate the association of glycemic control, serum potassium, and serum magnesium levels with the occurrence of arrhythmia in patients with Type 2 Diabetes Mellitus.

Inclusion criteria:

- Patients aged ≥18 years of either gender, referred for 24-hour Holter monitoring with symptoms suggestive of arrhythmia
- Diagnosed cases of Type 2 Diabetes Mellitus

Exclusion criteria:

- Patients with type 1 diabetes mellitus
- Non-diabetic individuals
- Patients with permanent pacemaker implants

Eligible patients were enrolled consecutively after obtaining informed consent. All participants underwent a 24-hour Holter ECG, during which they were instructed to note any symptoms such as palpitations, dizziness, or syncope. Data collection included clinical history, physical examination, and laboratory investigations such as fasting blood glucose (FBS), 2-hour postprandial glucose (2ABF), HbA1c, serum electrolytes (potassium, magnesium), and thyroid function tests. Glycemic control was assessed according to ADA 2013 guidelines.

Holter recordings were analyzed for arrhythmias, including ventricular ectopics (graded using Lown classification, grades 2–4), supraventricular ectopics, bradyarrhythmias, AV blocks, atrial fibrillation, and tachyarrhythmias. Data were analyzed using SPSS version 10, and a p-value of <0.05 was considered statistically significant.

RESULTS

Table – I: Glycaemic Profile of the Study Population (*n*=100)

Investigations	Mean ± SD	Minimum – Maximum
FBG (mmol/L)	15.06 ± 6.40	4.2 – 25.3
2ABF (mmol/L)	16.28 ± 7.60	6.5 – 29.6
HbA1c (%)	10.13 ± 2.59	6.3 – 14.4

Table I presents the glycaemic parameters of the 100 enrolled patients. The mean fasting blood glucose (FBG) was 15.06 ± 6.40 mmol/L, ranging between 4.2 and 25.3 mmol/L. The mean 2-hour after-breakfast (2ABF) glucose level was 16.28 ± 7.60 mmol/L, with a range from 6.5 to 29.6 mmol/L. The mean HbA1c was 10.13 ± 2.59%, with values spanning 6.3% to 14.4%, indicating overall poor long-term glycaemic control in the study population.

Table – II: Correlation Between Glycemic Control and Arrhythmia in the Study Population

Glycaemic Control	Controlled	Uncontrolled	P value
FBG	35	65	<0.001
Arrhythmia	15	55	
No arrhythmia	20	10	
2ABF	23	77	0.270
Arrhythmia	13	53	
No arrhythmia	10	24	
HbA1c (%)	28	72	0.110
Arrhythmia	11	22	
No arrhythmia	17	15	

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Table II illustrates the relationship between glycemic control and the presence of arrhythmia among the study population with Type 2 Diabetes Mellitus. Among those with uncontrolled fasting blood glucose (FBG), 55 out of 65 patients (84.6%) developed arrhythmia, compared to 15 out of 35 (42.9%) in the controlled FBG group—a statistically significant difference ($p < 0.001$). Although arrhythmia was also more frequent in patients with uncontrolled 2-hour after-breakfast blood glucose (2ABF) and elevated HbA1c, the differences were not statistically significant ($p = 0.270$ and $p = 0.110$, respectively).

Table – III: Correlation Between Serum Potassium Levels and Arrhythmia in the Study Population

Potassium Level	Number	Arrhythmia		P value
		Present	Absent	
Normal	79	68	11	0.030
Hypokalaemia	13	7	6	
Hyperkalaemia	8	0	8	

Table III illustrates the distribution of arrhythmia across different serum potassium levels in the study population. Among patients with normal potassium levels (n=79), 86.1% had arrhythmia. Notably, all patients with hyperkalaemia (n=8) had no arrhythmia, while nearly half of those with hypokalaemia (n=13) exhibited arrhythmic events (53.8%). The association between potassium status and arrhythmia was statistically significant ($p = 0.030$).

Table – IV: Correlation Between Serum Magnesium Levels and Arrhythmia in the Study Population

Magnesium Level	Number	Arrhythmia		P value
		Present	Absent	
Normal	92	73	19	0.120
Reduced level	6	2	4	
Raised level	2	0	2	

Table IV presents the distribution of arrhythmia in relation to serum magnesium levels among patients with Type 2 Diabetes Mellitus. Although the majority of patients had normal magnesium levels (92 cases), arrhythmia was present in 73 of them. Among those with reduced magnesium levels (n=6), arrhythmia occurred in 2 cases, while none of the 2 patients with elevated magnesium levels experienced arrhythmia. However, the association between magnesium status and arrhythmia did not reach statistical significance ($p = 0.120$).

Table – V: Relation of Incidence of Ventricular Ectopy with Glycemic Control (2ABF Level)

2ABF Level (mmol/l)	Mean ± SD Total Ventricular Ectopic Beats (Maximum–Minimum)	P value
Below 10 (n = 23)	3601.12 ± 4179.83(13870–17)	0.016
10 and more (n = 77)	5595.97 ± 3782.29(18334–60)	

Table V shows the association between glycemic control, as measured by 2-hour post-breakfast blood glucose (2ABF), and the incidence of ventricular ectopic beats in Type 2 Diabetes Mellitus patients. Those with poor glycemic control (2ABF ≥10 mmol/L, n = 77) had a significantly higher mean number of ventricular ectopic beats (5595.97 ± 3782.29; range 18334–60) compared to those with better glycemic control (2ABF <10 mmol/L, n = 23), who had a lower mean ectopic count (3601.12 ± 4179.83; range 13870–17). The difference was statistically significant ($p = 0.016$).

Table – VI: Relation of Incidence of Supraventricular Ectopy with Glycemic Control

2ABF Level (mmol/l)	Mean ± SD Total Supraventricular Ectopic Beats(Maximum–Minimum)	P value
Below 10 (n = 23)	4876.60 ± 5612.26(14300–15)	0.010
10 and more (n = 77)	8265.88 ± 7011.76(19340–40)	

Table VI presents the relationship between glycemic control, as measured by 2-hour post-breakfast blood glucose (2ABF) levels, and the incidence of supraventricular ectopic beats in patients with Type 2 Diabetes Mellitus. Patients with uncontrolled glycemic status (2ABF ≥10 mmol/L, n = 77) showed a significantly higher mean number of supraventricular ectopic beats (8265.88 ± 7011.76; range 19340–40) compared to those with better glycemic control (2ABF <10 mmol/L, n = 23), who had a mean of 4876.60 ± 5612.26 (range 14300–15). This difference was statistically significant ($p = 0.010$).

DISCUSSION

Type 2 Diabetes Mellitus is increasingly recognized not only for its metabolic implications but also for its association with cardiovascular complications such as arrhythmias. In diabetic

individuals, symptoms like palpitations, dizziness, and syncope often point toward underlying rhythm disturbances that may go undiagnosed without continuous monitoring. This study examined the occurrence and patterns of arrhythmias in adults with T2DM using 24-hour Holter monitoring, aiming to uncover potential links with glycemic control and electrolyte levels. The findings highlight a notable prevalence of both ventricular and supraventricular arrhythmias, particularly in patients with poor glycemic control and altered serum potassium and magnesium levels, reinforcing the importance of comprehensive cardiac evaluation in this population.

In the present study, the mean fasting blood glucose (FBG), 2-hour after breakfast glucose (2ABF), and HbA1c levels were 15.06 ± 6.40 mmol/L, 16.28 ± 7.60 mmol/L, and 10.13 ± 2.59%, respectively, reflecting poor glycemic control and substantial variability in glucose levels. These findings resonate with those of Gu et al.[14], who reported that patients with new-onset atrial fibrillation exhibited significantly greater HbA1c variability, suggesting that fluctuating glycemic states increase the risk of arrhythmias in T2DM. Andersen et al.[15] similarly demonstrated that both hypoglycemia and increased glycemic variability were associated with a higher frequency of arrhythmic events in diabetic patients. The elevated glycemic indices in our study, particularly the high HbA1c levels, indicate chronic hyperglycemia and potential glycemic swings, which may predispose patients to arrhythmogenesis through autonomic imbalance, electrolyte disturbance, and structural cardiac changes, supporting the hypothesis that dysregulated glycemic status is a key contributor to arrhythmia risk in T2DM.

In the present study, a significant association was observed between uncontrolled fasting blood glucose (FBG) and arrhythmia ($p < 0.001$), with 84.6% of arrhythmic patients belonging to the uncontrolled FBG group, highlighting FBG as a strong predictor of cardiac electrical disturbances in diabetic patients. This finding closely aligns with the Mendelian randomization analysis by Harati et al.[16], which demonstrated a 16% increased risk of atrial fibrillation (AF) in individuals with FBG between 5.5–6.9 mmol/L and a 13% risk increase for every 1% rise in HbA_1c. Similarly, Sun et al.[17] reported that a 1 mmol/L increase in baseline FBG raised AF risk by 33%, reinforcing the role of hyperglycemia in arrhythmogenesis. While our study also noted higher rates of arrhythmia in patients with uncontrolled postprandial glucose (2ABF) and HbA_1c, these associations did not reach statistical significance ($p = 0.270$ and $p = 0.110$, respectively), a trend consistent with Sun et al.'s review, which recognized a link between elevated HbA_1c and ventricular arrhythmias despite less robust predictive power than FBG. Together, these comparisons underscore the critical importance of fasting glycemic control in mitigating arrhythmic risk in type 2 diabetes.

In the present study, a significant association was observed between serum potassium levels and arrhythmia occurrence ($p = 0.030$), with the highest burden of arrhythmia seen in the hypokalemic group (7 out of 13 cases). This finding is consistent with the results of Pitt et al.[18], who reported that patients with type 2 diabetes and low potassium levels (<4.0 mmol/L) had a 67% higher risk of arrhythmic events compared to those within the normal potassium range. While arrhythmia was most prevalent in hypokalemia, it was notably absent among patients with hyperkalemia (0 of 8), potentially reflecting the complex and often nonlinear impact of potassium extremes on cardiac electrophysiology. These findings underscore the critical role of maintaining normal potassium homeostasis in mitigating arrhythmic risk among diabetic patients.

In our study examining the association between serum magnesium levels and arrhythmia in patients with Type 2 Diabetes Mellitus, the majority (92%) had normal magnesium levels, with 73 remaining arrhythmia-free. Among the small subset of patients with reduced magnesium (6%), arrhythmia was observed in 2 individuals, while 4 did not experience arrhythmic events. Notably, none of the two patients with elevated magnesium levels developed arrhythmia. Although the association was not statistically significant ($p = 0.120$), the observed pattern aligns with broader epidemiological evidence suggesting a potential arrhythmogenic role of hypomagnesemia. Oost et al.[19] similarly reported an inverse relationship between serum magnesium levels and the incidence of atrial fibrillation, heart failure, and microvascular complications in individuals with T2DM, highlighting the importance of electrolyte monitoring in this population.

In our study examining the association between 2-hour post-breakfast blood glucose (2ABF) levels and ventricular ectopic beats in patients with Type 2 Diabetes Mellitus, we found that those with elevated 2ABF (≥10 mmol/L) exhibited a significantly higher mean number of ventricular ectopic beats (5595.97 ± 3782.29) compared to patients with lower 2ABF levels (<10 mmol/L), who had a mean of 3601.12 ± 4179.83 ectopic beats ($p = 0.016$). These findings align with Andersen et al.[15], who reported that increased glycemic variability—though not specifically 2ABF—was associated with a higher incidence of cardiac arrhythmias, emphasizing the broader impact of glycemic fluctuations on arrhythmic risk. Our data reinforce the notion that postprandial hyperglycemia may play a critical role in triggering ventricular arrhythmias, highlighting the importance of tight glycemic control to potentially mitigate cardiac electrical instability in this population.

In our study, we observed that patients with higher 2-hour post-breakfast blood glucose (2ABF) levels (≥10 mmol/L) had a significantly greater mean number of supraventricular ectopic beats (8265.88 ± 7011.76) compared to those with lower 2ABF levels (<10 mmol/L), who had a mean of 4876.60 ± 5612.26 ($p = 0.010$). This finding aligns with the work of Ceriello et al.[20], who demonstrated that postprandial hyperglycemia serves as an independent risk factor for cardiovascular disease in Type 2 Diabetes Mellitus (T2DM) patients. Their study underscores the broader cardiovascular risks associated with elevated postprandial glucose levels, supporting the notion that postprandial hyperglycemia contributes not only to vascular complications but also to increased arrhythmic burden, as reflected in our results on supraventricular ectopy. These findings highlight

the importance of controlling postprandial glucose spikes to reduce cardiac arrhythmia risk in T2DM populations.

Limitations of the study

The study had several limitations:

- The study population was relatively small, limiting generalizability.
- Symptom frequency was spontaneous and unpredictable, potentially missing during the 24-hour monitoring period.
- Arrhythmias may occur both with and without symptoms, complicating interpretation.
- Symptoms could recur multiple times during monitoring with varying ECG findings.
- Establishing a definitive cause-effect relationship between symptoms and ECG findings remains challenging.

Conclusion

Our study demonstrates that poor glycemic control is strongly associated with the occurrence of arrhythmia in patients with Type 2 Diabetes Mellitus. Patients with elevated fasting and post-breakfast glucose levels not only developed arrhythmias more frequently but also exhibited a higher burden of both ventricular and supraventricular ectopic beats. Additionally, abnormal serum potassium levels, particularly hypokalemia, were significantly linked to arrhythmic events. Although a trend was observed with low magnesium levels, this association did not reach statistical significance. Overall, these findings underscore the critical importance of maintaining optimal glycemic control and electrolyte balance to reduce cardiac arrhythmic risk in this patient population.

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