

The incidence of Electrocardiographic (ECG) changes suggestive of myocardial infarction in patients attending Kalafong Provincial Tertiary Hospital diabetes clinic

BN Xhekwni^{1,2*}, M de Villiers^{1,2}  and DG van Zyl^{1,2} 

¹Department of Internal Medicine, Kalafong Provincial Tertiary Hospital, Atteridgeville, Pretoria, South Africa

²Diabetes Research Centre, Faculty of Health Science, University of Pretoria, Hatfield, Pretoria, South Africa

*Correspondence: xboneswa@gmail.com



Aims: This study aims to determine the incidence of new electrocardiographic (ECG) changes suggestive of myocardial infarction (MI) in diabetic patients, both with and without typical symptoms. Additionally, it seeks to identify other ECG abnormalities indicative of the effects of diabetes mellitus (DM) on the heart.

Methods: Patients aged 30 years and older were selected from the DM clinic database covering the period from 2008 to 2023. A total of 732 patients were initially identified, of whom 634 were eligible for inclusion in the study. A subgroup of 568 patients had normal baseline ECGs. ECG abnormalities were reviewed and discussed with the senior physician, with findings documented in the electronic database. Two ECGs, at least one year apart, were compared. In patients with more than two ECGs, the first one with abnormal findings was compared with the baseline ECG. Kaplan–Meier analyses were performed to assess the time to new ischaemia and MI in patients without baseline ECG abnormalities comparing patients with Type 1 and Type 2 DM (hazard ratio [HR] 2.082, 95% confidence interval [CI] 1.049–4.133).

Results: Of the patients eligible for the study, 83% ($n = 568$) had normal ECGs at baseline. Among them, 33.1% ($n = 210$) developed ECG abnormalities at follow-up. Ninety-seven patients (15.3%) showed ECG signs suggestive of myocardial infarction/ischaemia, with 66 (16.7%) females and 31 (13%) males exhibiting these changes. The most common myocardial regions affected were the inferior wall, followed by the inferolateral wall, while the anterior wall was the least commonly affected. Type 2 DM patients exhibited a higher incidence of ECG changes suggestive of MI compared with those with Type 1 DM (16.7% vs. 9.8%). Other common ECG abnormalities included atrioventricular (AV) conduction defects (right and left bundle branch blocks, first-degree AV block), P-wave abnormalities (P mitrale), increased incidence of left ventricular hypertrophy (LVH), and poor R wave progression.

Conclusions: A higher incidence of myocardial ischaemia and other ECG abnormalities were observed in diabetic patients, particularly those with Type 2 DM.

Keywords: diabetes mellitus, electrocardiographic changes, myocardial infarction

Introduction

The global prevalence of diabetes mellitus (DM) has increased significantly, rising from approximately 108 million cases (4.7%) in 1980 to an estimated 463 million cases (9.3%) in 2021. The greatest increases have been observed in low- and middle-income countries. Projections suggest that this global burden will rise to 578 million cases (10.2%) by 2030 and 700 million cases (10.9%) by 2045.¹ This trend may be attributed to enhanced screening methods, a growing number of autoimmune conditions leading to DM, higher rates of gestational DM (which often progresses to Type 2 DM), and the increasing prevalence of obesity.^{2,3}

Type 1 DM is associated with a twofold increase in all-cause mortality and a threefold increase in cardiovascular mortality compared with the general population. In individuals with poor glycaemic control, the risk is even more pronounced, with an eightfold increase in all-cause mortality and a tenfold increase in cardiovascular mortality.^{4,5} Globally, cardiovascular disease affects approximately 32.2% of individuals with Type 2 DM and remains the leading cause of death in this population, accounting for 50.3% of all fatalities.⁶ Despite advancements in screening, a substantial proportion of patients with DM remain undiagnosed, contributing to increased morbidity, mortality,

and significant economic costs.⁷ Early detection and prevention programmes are more cost-effective than managing the complications of DM and its associated chronic illnesses.

Silent myocardial infarction (MI) is one of the most significant and common cardiovascular complications in individuals with DM. This condition is also prevalent in patients with hypertension, particularly among women and older adults.⁸ Silent MI often leads to undiagnosed coronary artery disease (CAD) in diabetic patients due to the atypical and mild nature of symptoms, which are often short-lived and do not prompt medical attention.^{2,9,10}

Studies have shown that diabetic patients with asymptomatic CAD and positive ECG changes frequently have a history of DM spanning at least five years, with 70% demonstrating poor glycaemic control.¹¹ The prevalence of silent MI is estimated at 28.5% in diabetic patients compared with 21.5% in non-diabetic individuals.^{12, 13}

DM is often diagnosed during the first presentation of an acute MI. Diabetic patients presenting with acute MI face significantly higher risks of mortality and subsequent cardiovascular events.⁹ Silent MI accounts for 8.2% of ischaemic events and is

associated with a more than threefold increase in mortality and major adverse cardiovascular outcomes.^{14–16} DM is a significant factor contributing to poorer prognoses following MI.

Routine resting ECG screening in diabetic patients is recommended by the American Diabetes Association, the European Society of Cardiology, and the European Association for the Study of Diabetes.^{17,18} These guidelines have also been adopted by the Society for Endocrinology, Metabolism, and Diabetes of South Africa (SEMDSA), making ECG assessments a standard practice during consultations.¹⁹ DM is recognised as an independent risk factor for cardiovascular diseases, yet many abnormalities remain undetected due to insufficient diagnostic skills among healthcare providers.^{20,21}

Common ECG abnormalities observed in diabetic patients include ST-T changes, left atrial enlargement, left ventricular hypertrophy, and bundle branch blocks (left or right). In a study of African Americans, common ECG findings included prolonged corrected QT interval (QTc), T-wave changes, left ventricular hypertrophy, sinus tachycardia, ischaemic heart disease, conduction defects, and premature ventricular or atrial contractions.^{16, 22–24}

This study aimed to determine the frequency of ECG changes in a South African population with DM and to assess the incidence of new ECG changes indicative of CAD over a 10-year period, irrespective of the presence of acute coronary event symptoms.

Table 1: ECG abnormalities definitions

ECG abnormalities	Definitions
Pathological Q waves	A Q wave > 40 ms (1 mm) wide, > 2 mm deep, > 25% of the depth of the QRS complex or if seen in leads V _{1–3}
ST segment elevation (STEMI)	J point elevation in ≥ 2 contiguous leads with the cut-off point as greater than 0.1 mV in all leads except V ₂ or V ₃ In leads V _{2–V3} the cut-off point is > 0.2 mV in men older than 40 and greater than 0.25 mV in those < 40 years old, or > 0.15 mV in women
ST segment depression (STD)	J point that is displaced below baseline. It can either be horizontal or down-sloping ≥ 0.5 mm at the J point in ≥ 2 contiguous leads
T wave inversion (TWI)	When present in leads other than aVR and V ₁ , have a negative T wave deflection, with an amplitude of > 5 mm in limb leads and > 10 mm in precordial leads
Left bundle branch block (LBBB)	Wide (≥ 120 ms), predominantly negative (QS) complexes in V ₁ and entirely positive (R) complexes in V ₆
Right bundle branch block (RBBB)	Wide QRS complexes, with the terminal QRS vector that is orientated to the right and anteriorly (rSR' in V ₁ and qRS in V ₆)
Left ventricular hypertrophy (LVH)	Voltage criteria = SV ₁ + [RV ₅ or RV ₆] > 35 mm
Right ventricular hypertrophy (RVH)	Relatively tall R wave in lead V ₁ (R ≥ S wave), alternatively qR pattern in V ₁ or V ₃ R
P-mitrale	A biphasic P wave in V ₁ with a broad negative component or a broad (≥ 120 ms), often notched P wave in one or more limb leads. This is indicative of left atrial overload or left atrial conduction delays without the actual atrial enlargement
P-pulmonale	An increase in P wave amplitude (≥ 2.5 mm) indicative of right atrial overload

Methods

The study was conducted at Kalafong Provincial Tertiary Hospital (KPTH), a tertiary government hospital in Pretoria, Gauteng. The hospital predominantly serves patients of African descent from lower to middle socioeconomic backgrounds. The DM clinic at KPTH, established in 2008, has effectively managed patients with difficult-to-control DM, those on insulin, and individuals with complicated DM. The clinic adheres to a structured management approach aligned with SEMDSA guidelines, and all clinical data are systematically recorded in an electronic database.

Study design

This retrospective cohort study utilised data from the KPTH DM clinic database, focusing on patients aged 30 years or older who had been followed up for at least two consecutive years.

Inclusion criteria

Eligible patients were required to have undergone at least two electrocardiograms (ECGs) at least one year apart. ECGs were interpreted by the most senior clinician present in the clinic, who had access to the patients' clinical information. To ensure accuracy, all ECGs were subsequently reviewed by the principal investigator.

Exclusion criteria

Patients were excluded if they had insufficient clinical data captured in the database, had faded or poorly calibrated ECG tracings, used medications or substances that could mimic ECG signs of ischaemia (e.g. digoxin or illicit drugs), had known ischaemic heart disease, with or without prior cardiological intervention, and had congenital heart conditions, whether surgically corrected or not.

Data collection

Data for statistical analysis were extracted from the electronic database. Predictor variables, including age, duration of DM, and glycated haemoglobin A1c (HbA1c) levels from the same year as the first ECG, were recorded. When multiple values were available (e.g. HbA1c, systolic, and diastolic blood pressure), the mean values were calculated and used for analysis.

A minimum sample size of 301 patients was calculated based on a power of 90% and an alpha threshold of 0.05, assuming a 5% incidence of new ECG changes consistent with ischaemic heart disease.

Study tool

Standard 10-second resting 12-lead ECGs were performed on all patients as part of the clinic's annual cardiovascular assessment protocol. Two types of ECG machines were used: Nihon Kohden CardiofaxQ (Model ECG-9132k; Nihon Kohden, Tokyo, Japan) before 2019, and Mindray BeneHeart R12 (Shenzhen Mindray Bio-Medical Electronics Co., Ltd, Nanshan, Shenzhen, P.R. China) from 2019 onwards.

All ECGs were performed by a single technician following standardised lead placement protocols. Machine-generated ECG interpretations were not used for analysis.

Definitions

Type 1 DM (T1DM) patients: Those with confirmed anti-glutamic acid decarboxylase (anti-GAD) or insulinoma-associated antigen

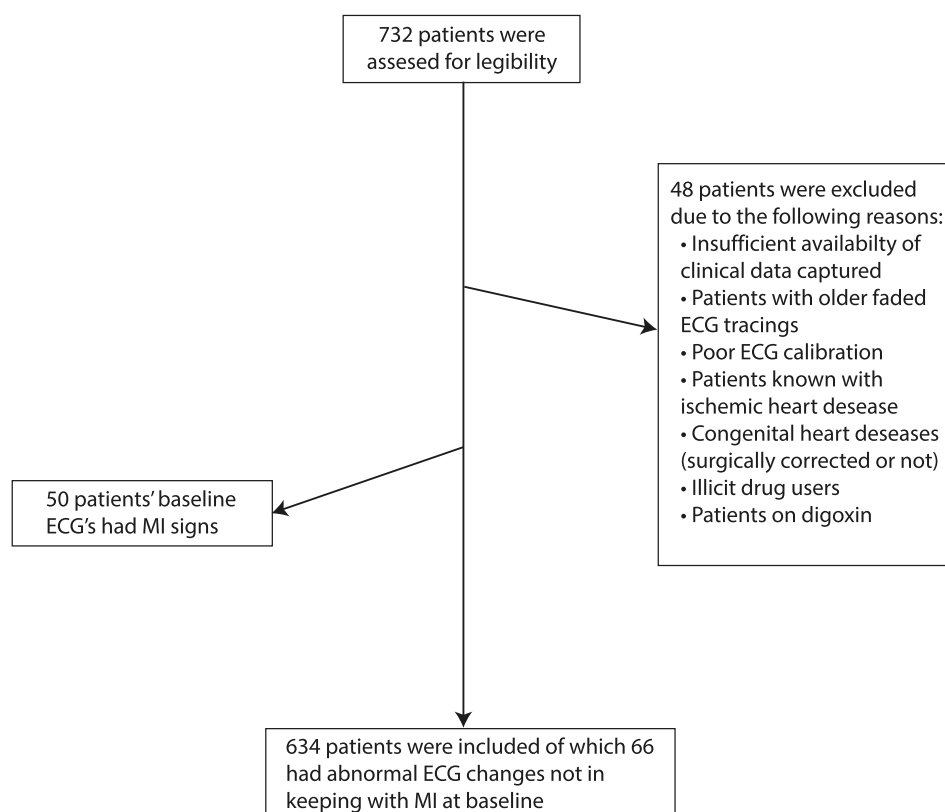


Figure 1: Diabetic patients selected for the study.

2 (IA2) antibodies, or antibody-negative patients diagnosed with DM during adolescence or requiring insulin from diagnosis.

Type 2 DM (T2DM) patients: Those with clinical features of insulin resistance (e.g. obesity, acanthosis nigricans, skin tags) and on treatment with oral antidiabetic agents alone or in combination with insulin for glycaemic control.

ECG abnormalities are defined in [Table 1](#).^{25, 26}

Other abnormalities, such as ventricular conduction defects and dysrhythmias, were also recorded. These findings were used to identify pathologies related to myocardial infarction, ischaemia, or other complications associated with DM.

Statistical analysis

Data were analysed using IBM SPSS (version 29.0.0.0; IBM Corp, Armonk, NY, USA). Continuous variables were described according to type and distribution, while categorical variables were reported as proportions. Comparisons were made using non-parametric methods for continuous data and chi-square tests for categorical data. Crude incidence rates were calculated for outcomes.

Missing data analysis showed that predictor variables had 6.3%–22.4% missing values, while outcome variables had no missing data. Missing predictor data were imputed using mean values, and paired t-tests were performed to confirm consistency between original and imputed data. Imputation preserved the sample size and minimised bias in survival analysis.

Survival analysis was performed to assess time-to-event outcomes, such as the development of ECG changes indicative of MI. Survival

curves were adjusted for confounders, including age and other predictive variables, using Cox proportional hazards modelling.

Ethical considerations

Ethical approval for the study was obtained from the Research Ethics Committee of the Faculty of Health Sciences, University of Pretoria (Ethics Reference No. 392/2022). The study was registered in the National Health Research Database of South Africa (NHRD), and permission was granted by the hospital authorities.

Results

A total of 732 patients attending the DM clinic at KPTH were initially selected. Of these, 634 met the eligibility criteria for inclusion in the study. The majority were female ($n = 395$, 62.3%). The selection process for inclusion and exclusion is detailed in [Figure 1](#).

A disproportionate number of patients with Type 1 DM ($n = 195$, 30.8%) were included, reflecting the tertiary care nature of the hospital. Additionally, 26 patients (4.1%) with DM of uncertain type were included ([Table 2](#)). These patients lacked auto-immune antibodies but presented with clinical features consistent with both Type 1 and Type 2 DM and were on insulin therapy from the time of diagnosis.

The majority of patients were on insulin therapy at baseline, with only a few using a combination of insulin and oral hypoglycaemic agents. During the study period, some patients initially receiving both oral and insulin therapy were transitioned to insulin-only treatment following the development of nephropathy.

The mean follow-up time for the study was 8.7 years (95% CI: 8.47–8.96), with a median of 10 years (95% CI: 8.6–11.4).

Table 2: Baseline characteristics

Factor	n (%)	Mean (SD) or median (IQR)
Age	634	Mean: 52.9 (SD: 14.4)
Female	395 (62.3)	
Male	239 (37.7)	
Race		
• Black	572 (90.2)	
• Coloured	3 (0.5)	
• Indian	31 (4.9)	
• White	28 (4.4)	
Type of DM		
• Type 1	195 (30.8)	
• Type 2	406 (64.0)	
• Secondary	7 (1.1)	
• Uncertain	26 (4.1)	
Duration of DM (Med) Years	569 (99.3)	Median: 10, IQR (5–16)
BMI (kg/m ²)	492 (77.8)	Mean: 31.7, SD: 6.88
HbA1C (%)	618 (89.7)	Mean: 8.6, SD: 1.98
Urine albumin to creatinine ratio (mg/mmol)	520 (82.2)	Median: 1.9, IQR (0.75–9.89)
Total cholesterol (mmol/L)	550 (86)	Mean: 4.3 SD: 1.31
LDL (low density lipoprotein) (mmol/L)	535 (84.3)	Mean: 2.4 SD: 1.08
Serum creatinine (μmol/L)	545 (86)	Median: 78.3 (55–90)
Hypertension	515 (81.2)	
Systolic blood pressure (mmHg)	594 (93.9)	Mean: 138.5 (SD: 16.96)
Diastolic blood Pressure (mmHg)	594 (93.9)	Mean: 80.4 (SD: 10.3)

Follow-up duration was calculated from the first clinic visit and baseline electrocardiogram (ECG) to the occurrence of the first ischaemic event on ECG or censoring. Patients were found to have several risk factors for coronary artery disease (CAD). Hypertension was present in 515 patients (81.2%), with uncontrolled systolic blood pressure in 248 out of 515 patients (48.2%) and uncontrolled diastolic blood pressure in 77 patients (14.9%) at baseline. Dyslipidaemia was another prominent risk factor, with the mean low-density lipoprotein (LDL) cholesterol level recorded at 2.4 mmol/L for the study population (Table 2). Statins were prescribed to 504 out of 634 patients (79.5%) at baseline. However, there was inconsistency in the prescription of aspirin, either alone or in combination with statins. The reasons for these omissions were not documented, making the accuracy of these statistics uncertain.

Among the 634 patients, 210 (33.1%) who had normal baseline ECGs later developed ischaemic changes on follow-up ECGs. However, only 97 (15.3%) of these patients exhibited ECG changes consistent with definite myocardial infarction, such as ST-segment elevation MI (STEMI) or possible STEMI (Table 3). The frequency of MI was slightly higher among females (66 of 395; 16.7%) compared with males (31 of 239; 13%), although this difference was not statistically significant ($p = 0.245$). Female patients contributed 1 712 patient-years to the study, while male patients contributed 988 patient-years.

Of the 634 patients enrolled in the study, 195 (30.8%) had T1DM and 406 (64%) had T2DM, contributing 895 and 1 661 patient-years to the study, respectively. DM due to secondary causes and those with an uncertain type of DM comprised 33 patients in total, contributing 144 patient-years.

ECG changes suggestive of MI were observed in 97 patients (15.3%), with 74 (18.2%) of these patients having T2DM and 19 (9.7%) having T1DM (Table 3). Among patients with uncertain or secondary diabetes, four (4.1%) showed ECG changes suggestive of MI (Table 3).

The crude incidence rate for ECG evidence of MI in patients with T2DM was 0.045 events/person year (95% CI: 0.035–0.056), which was significantly higher than the crude incidence rate in patients with T1DM (0.021 events/person year, 95% CI: 0.013–0.033), $p = 0.018$ (Table 3). However, the difference in incidence rates between T1DM and T2DM may be partly explained by the mean age difference between the two groups: patients with T1DM had a mean age of 42.1 years (SD: 14.92), while those with T2DM had a mean age of 58.6 years (SD: 10.72), with a statistically significant difference ($p < 0.001$). After adjusting for age, the hazard ratio (HR) for ECG evidence of MI in T2DM patients, with T1DM as the reference group, was 2.082 (95% CI: 1.049–3.67, $p = 0.013$), indicating a significantly higher risk of MI in T2DM patients compared with those with T1DM (Figure 2).

Survival analysis for ECG changes suggestive of myocardial infarction was conducted using a Cox proportional hazards model to adjust for potential confounding factors. The following baseline variables were included in the analysis.

The Cox model had a -2 log-likelihood p -value of 0.2, with a Harrell's C-index of 0.633 (95% CI: 0.574–0.686), indicating modest predictive ability. The log-minus-log plot for the type of DM demonstrated parallel lines over time, satisfying the proportional hazards assumption.

Among the analysed risk factors, only BMI was statistically significant ($p = 0.042$) in influencing the time to ECG changes indicative of MI (Table 4). All other variables, including Type 2 DM, were not significant predictors of ECG evidence of MI after adjusting for the included covariates. T2DM, when adjusted for these factors, did not significantly influence the hazard of ECG changes suggestive of MI.

The most commonly affected myocardial site for infarction and/or ischaemia observed on follow-up ECGs was the inferior wall (leads II, III, and aVF), with a frequency of 47.2%. The inferolateral wall was the second most commonly affected region of the left ventricle, showing ischaemic changes in up to 20.5% of cases on follow-up ECGs. The anterior, anterolateral, lateral, and high lateral walls were less frequently involved, with a combined baseline frequency of 19.5%, increasing to up to 20.5% on follow-up ECGs (Figure 3).

T-wave inversion (TWI) was a significant contributor to the observed changes, with a baseline frequency of 19.5%, increasing to 23.8% at follow-up. While TWI is commonly associated with ischaemia, other potential causes include myocardial strain due to hypertensive heart disease. The frequency of diffuse TWI also increased from baseline ($n = 3$, 0.4%) to follow-up ($n = 9$, 1.3%). This increase may be attributed to ischaemic cardiomyopathy, myocarditis, or pericarditis (Figure 3).

This study also demonstrated a progressive increase in the frequency of left bundle branch block (LBBB) morphology from 0.7% ($n = 5$) at baseline to 2.9% ($n = 20$) over time, as well as an increase in first-degree atrioventricular (AV) block from 0.15% ($n = 1$) to 1.5% ($n = 10$), both statistically significant ($p = 0.002$) (Figures 4A and B).

Table 3: STEMI plus possible STEMI at 2nd ECGs from normal baseline

Cases with no ischaemic baseline ECG changes	Cases with ischaemic ECGs later, <i>n</i> (%)	Crude incidence (%) of ischaemic changes in ECG (events/person year)	ECG changes of MI and possible MI later, <i>n</i> (%)	Crude incidence of MI or possible MI changes in ECG (events/person year)
Total: 634	210 (33.1)	0.077 (CI: 0.067–0.089)	97 (15.3) 74 (18.2) T2DM 19 (9.7) T1DM 4 (4.1) Uncertain DM	0.036 (CI: 0.03–0.044) 0.045 (CI: 0.035–0.056) 0.021 (CI: 0.013–0.033)
Females: 395	115 (29.1)	0.067 (CI: 0.055–0.08)	66 (16.7)	0.039 (CI: 0.03–0.049)
Males: 239	95 (39.7)	0.096 (CI: 0.078–0.117)	31 (13)	0.031 (CI: 0.021–0.045)
<i>p</i> -value	0.024		0.245	

The P-wave abnormalities were observed at baseline, as evidenced by an increased frequency of P mitrale findings on ECG, rising from 3.1% to 5.3% ($p = 0.042$). Additionally, there was a significant rise in the frequency of poor R-wave progression ($p = 0.001$), which may be associated with ischaemia. The frequency of left ventricular hypertrophy (LVH) increased by 50% over the study period ($p = 0.001$), potentially reflecting the high prevalence of hypertension in patients with diabetes (Figures 4A and B).

Discussion

This study found that 15.3% of diabetic patients followed up at the KPTH DM clinic developed ECG changes suggestive of MI over a median follow-up of 10 years. The incidence rate of ECG changes suggestive of MI was twice as high in patients with T2DM compared with those with T1DM, even after adjusting for age (HR: 2.055). Cox proportional hazards analysis indicated that none of the conventional cardiovascular risk factors, except for obesity, significantly contributed more than others in predicting ECG changes indicative of MI.

The incidence of ECG changes consistent with MI and possible MI was not significantly different between male and female patients, with a slightly higher incidence observed in females. This lack of difference is likely due to DM acting as an equaliser in MI risk between sexes.²⁷ The majority of patients who developed ECG changes suggestive of myocardial ischaemia had T2DM. The observed increase in the frequency of left bundle branch block (LBBB) morphology and first-degree atrioventricular (AV) block may be attributed to autonomic nervous system dysfunction associated with DM.²⁸ Notably, none of the patients enrolled in the study had third-degree AV block or pacemakers in place. It remains unclear whether first-degree AV block was a result of medication side effects, cardiac autonomic neuropathy due to DM, or ischaemia.

Diabetic patients tend to develop structural, functional, and mechanical changes in the left atrium, which contribute to the development of diabetic cardiomyopathy. DM is known to cause left atrial remodelling, which may later lead to atrial fibrillation and flutter.^{29,30} The presence of left atrial remodelling in

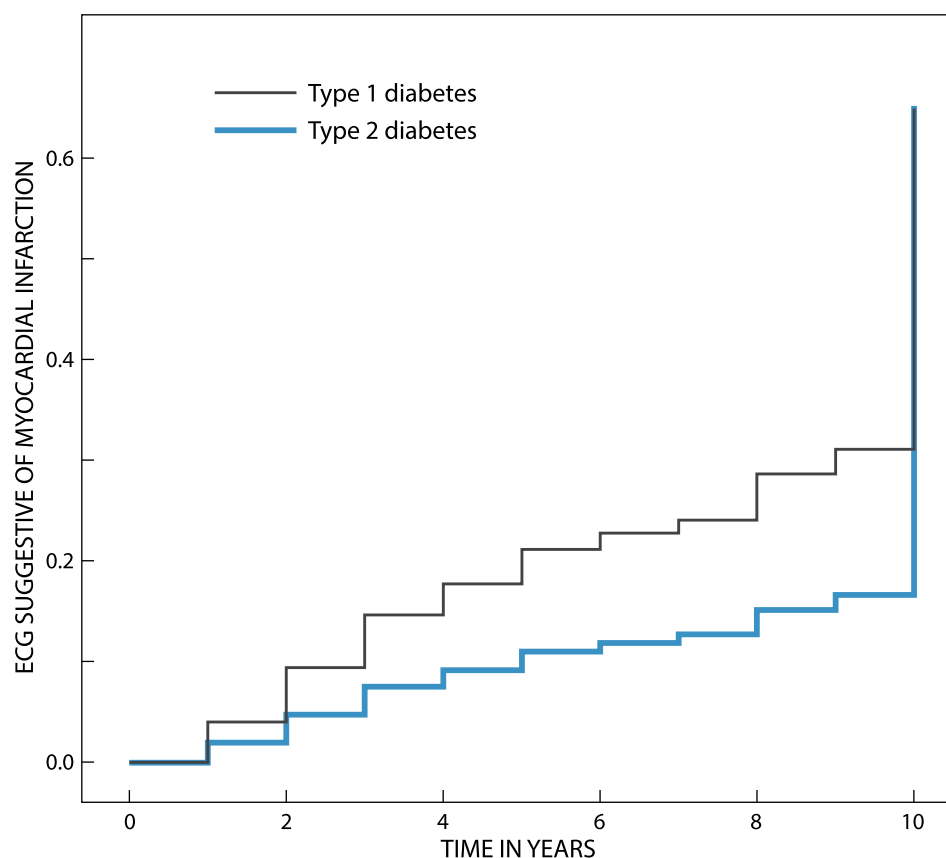
**Figure 2:** Survival analysis Type 1 vs. Type 2 DM adjusted for age.

Table 4: Survival analysis for ECG changes suggestive of myocardial infarctions using a Cox proportional hazards model

Baseline variable	Hazard ratio	95% confidence interval	p-values
Age	1.006	0.986–1.028	0.553
Sex	1.037	0.638–1.687	0.883
T2DM vs. T1DM	1.72	0.955–3.09	0.071
Duration of DM	0.986	0.234–1.746	0.377
HbA1c	1.094	0.985–1.216	0.094
BMI	1.036	1.002–1.072	0.042
Urine ACR	1.001	0.995–1.006	0.824
Systolic BP	0.994	0.977–1.011	0.473
Diastolic BP	1.007	0.979–1.036	0.619
Serum creatinine	1.003	0.998–1.008	0.192
Serum triglycerides	0.887	0.617–1.274	0.515
Total cholesterol	1.561	0.710–3.433	0.268
LDL cholesterol	0.614	0.272–1.386	0.241
HDL cholesterol	0.639	0.234–1.008	0.383
Statin use	0.975	0.573–1.658	0.925
Smoking	0.516	0.158–1.679	0.272

diabetic patients tends to occur earlier and serves as a prognostic marker for diabetic cardiomyopathy.^{31–34} The frequency of left ventricular hypertrophy (LVH) increased by 50% ($p = 0.001$) from baseline, which may be linked to the high prevalence of hypertension in diabetes. LVH is commonly observed in diabetic patients, with an increased frequency of LVH on follow-up ECGs (from 4.1% to 8.2%). This finding is consistent with a study done in Pietermaritzburg (KwaZulu-Natal) that identified a significant association between LVH and DM, with 36% of patients having LVH at baseline.³² Diabetic patients frequently present with hypertension at their first visit to the DM clinic and later develop hypertensive heart disease, with some cases of hypertension arising secondary to vascular remodelling caused by DM.

Most of the patients enrolled in this study had additional risk factors for coronary artery disease, as well as evidence of target organ damage from DM. Studies on percutaneous interventions in diabetic patients with MI have shown that

the calibre of coronary arteries is smaller in females and is inversely proportional to body mass index.^{35,36} Furthermore, diabetic patients tend to have a higher number of diseased vessels, and the development of collateral vessels is often impaired, which may explain the higher incidence of recurrent MI and the need for multiple coronary artery bypass grafting. These factors contribute to the increased risk of morbidity and mortality due to DM-related MI.^{37,38}

Recommendations

In managing diabetic patients who present with ECG changes suggestive of MI, several key clinical recommendations should be followed. ECG results should be interpreted within the clinical context and corroborated with cardiac biomarkers as necessary. It is also important to assess and manage cardiometabolic risk factors while ensuring optimal glycaemic control. The involvement of a cardiometabolic unit and a multidisciplinary team is essential for comprehensive patient care, facilitating coordinated management that addresses both cardiovascular and diabetes-related concerns effectively.

Limitations of the study

This study predominantly included patients of African descent, all from middle to lower socioeconomic backgrounds, which may limit the generalisability of the findings to other population groups and socioeconomic settings. The study also enrolled patients with more advanced disease who either had difficult-to-control blood pressure or blood glucose, had micro- or macrovascular complications, or had T1DM, which may introduce referral bias due to the level of the hospital. The presence of LBBB morphology on ECGs presented challenges, as this could be caused by factors other than previous MI.

The authors acknowledge that ECG changes are not the reference standard for diagnosing MI, and as most patients did not present with an acute MI or reported chest pain, the resting ECG was the best available diagnostic tool in the hospital. KPTH lacks a cardiology unit, and patients requiring further workup for ischaemic heart disease are referred to a cardiology unit with limited capacity and long waiting lists. The benefit of conducting annual ECGs is that it detects changes from the previous ECG; however, this strategy does not prevent ischaemic events but only identifies whether an event occurred in the preceding year.

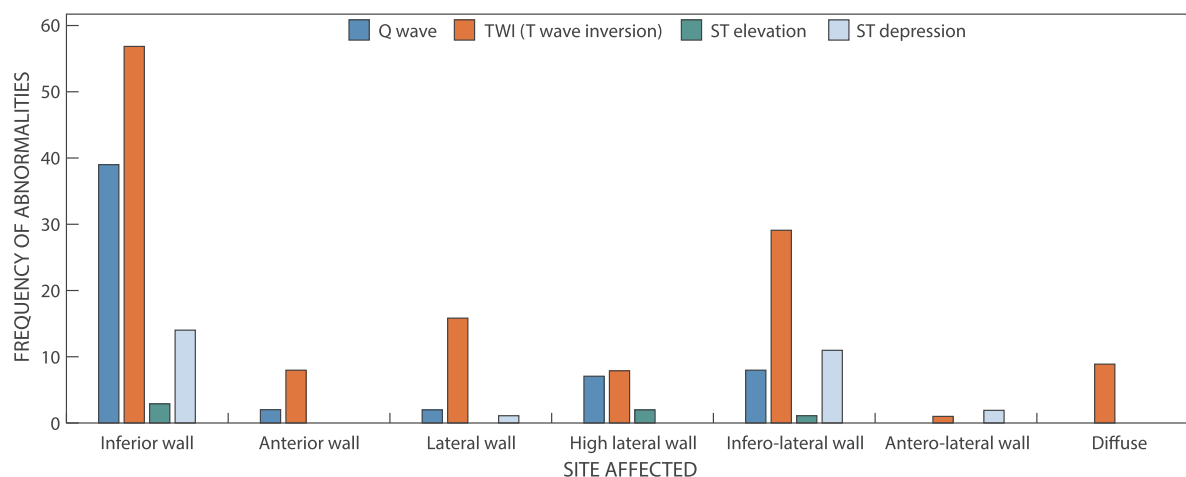


Figure 3: Sites of ECG abnormalities suggestive of myocardial ischaemia on follow-up.

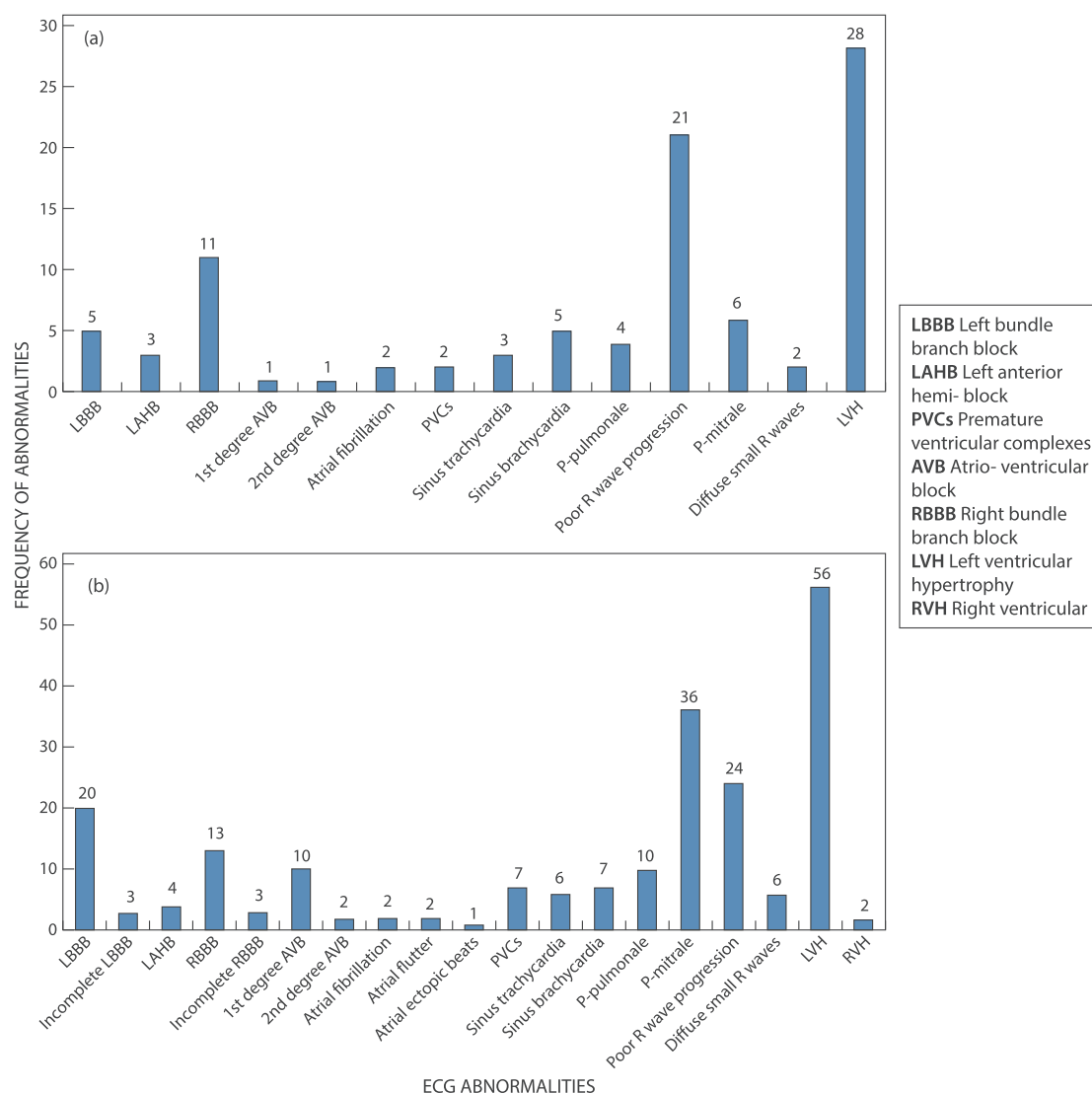


Figure 4: (A) ECG abnormalities not suggestive of MI at baseline; (B) ECG abnormalities not suggestive of MI that developed on follow-up.

Identifying ischaemic or previous MI changes on ECG allows for the implementation or intensification of secondary prevention measures if not already in place due to the presence of DM.

Baseline patient characteristics were used in the data analysis, which is a limitation, as these characteristics may change over time. The study did not account for changes in exposure, such as smoking cessation during the follow-up period, or for blood pressure, glycaemic, and lipid control, or changes in therapy. This is a limitation of the statistical technique employed (survival analysis). The majority of diabetic patients in South Africa are managed in resource-limited settings, and referrals for specialised care, particularly for non-acute MI, are often unavailable.

The study utilised serial ECG changes over time to assess the incidence of myocardial infarction (MI), rather than directly documenting actual MI events. This approach introduces the potential for survivor bias, as only patients who survived until the follow-up ECG were included in the analysis. Patients who experienced a fatal event or died before the next scheduled ECG were not captured in the study, which may contribute to the observed low frequency of anterior and septal ECG changes associated with MI.

Conclusion

The incidence of ECG changes suggestive of MI is high in patients with DM, with a higher frequency observed in T2DM. Obesity appears to play a significant role in the development of ECG changes suggestive of MI. The rate of ECG changes in keeping with MI was not significantly different between female and male diabetic patients, suggesting a loss of the protective effect in females. The absence of a sex difference in the incidence of MI, as well as the incidence of MI in relatively young T1DM patients, warrants further investigation in future studies. This study did not specifically focus on silent or minimally symptomatic MI in diabetic patients, particularly in African populations, and this should be explored in future research.

Acknowledgements – Prof. DG van Zyl is thanked for statistical analysis, and the staff at KPTH diabetes clinic are appreciated for their assistance with patient files.

Disclosure statement – No potential conflict of interest was reported by the authors.

Ethical considerations – Ethical approval for the study was obtained from the Research Ethics Committee of the Faculty

of Health Sciences, University of Pretoria (Ethics Reference No. 392/2022). The study was registered on the National Health Research Database of South Africa (NHRD), and permission was granted by the hospital authorities.

ORCID

M de Villiers  <http://orcid.org/0000-0001-8766-4296>

DG van Zyl  <http://orcid.org/0000-0001-7908-4689>

References

1. Saeedi P, Petersohn I, Salpea P, et al. Global and regional diabetes prevalence estimates for 2019 and projections for 2030 and 2045: results from the international diabetes federation diabetes atlas, 9th edition. *Diabetes Res Clin Pract.* 2019;157:107843. <https://doi.org/10.1016/j.diabres.2019.107843>.
2. Organization WH. Screening for type 2 diabetes: report of a World Health Organization and International Diabetes Federation meeting. World Health Organization. 2003.
3. Alp H, Ragbetli MC, Koksoy H, editors. Current Perspective on Diabetes Mellitus in Clinical Sciences: Karamanoglu Mehmetbey University (Karaman). 2023 Sep 14; Available from: <http://dx.doi.org/10.69860/nobel.9786053359111>.
4. Saeed M, Stene LC, Ariansen I, et al. Nine-fold higher risk of acute myocardial infarction in subjects with type 1 diabetes compared to controls in Norway 1973-2017. *Cardiovasc Diabetol.* 2022;21(1):59. <https://doi.org/10.1186/s12933-022-01498-5>.
5. Levy D. Type 1 Diabetes [Internet]. Oxford Medicine Online. Oxford University Press (Oxford); 2016. Available from: <http://dx.doi.org/10.1093/med/9780198766452.001.0001>.
6. Einarsen TR, Acs A, Ludwig C, et al. Prevalence of cardiovascular disease in type 2 diabetes: a systematic literature review of scientific evidence from across the world in 2007–2017. *Cardiovasc Diabetol.* 2018;17:83. <https://doi.org/10.1186/s12933-018-0728-6>.
7. Fan W. Epidemiology in diabetes mellitus and cardiovascular disease. *Cardiovasc Endocrinol Metab.* 2017;6(1):8–16. <https://doi.org/10.1097/XCE.0000000000000116>.
8. Omerovic E, Brohall G, Müller M, et al. Silent myocardial infarction in women with type II diabetes mellitus and microalbuminuria. *Ther Clin Risk Manag.* 2008;4(4):705–12. <https://doi.org/10.2147/TCRM.S2826>.
9. Svane J, Pedersen-Bjergaard U, Tfelt-Hansen J. Diabetes and the risk of sudden cardiac death. *Curr Cardiol Rep.* 2020;22:1–10. <https://doi.org/10.1007/s11886-020-01366-2>.
10. Kumar A, Sanghera A, Sanghera B, et al. Chest pain symptoms during myocardial infarction in patients with and without diabetes: a systematic review and meta-analysis. *Heart.* 2023;109(20):1516–24. <https://doi.org/10.1136/heartjnl-2022-322289>.
11. Daphale A, Acharya S, Shukla S. Detection of asymptomatic coronary artery disease (CAD) in newly detected type 2 diabetes mellitus (DM) by exercise treadmill test. *Int J Contemp Med Res.* 2017;4:2269–75.
12. Giraldez RR, Clare RM, Lopes RD, et al. Prevalence and clinical outcomes of undiagnosed diabetes mellitus and prediabetes among patients with high-risk non-ST-segment elevation acute coronary syndrome. *Am Heart J.* 2013;165(6):918–25.e2. <https://doi.org/10.1016/j.ahj.2013.01.005>.
13. Gazzaruso C, Coppola A, Montalcini T, et al. Screening for asymptomatic coronary artery disease can reduce cardiovascular mortality and morbidity in type 2 diabetic patients. *Intern Emerg Med.* 2012;7:257–66. <https://doi.org/10.1007/s11739-011-0527-5>.
14. Bhattacharyya MR. Psychological and biological factors in acute coronary heart disease: University of London, University College London (United Kingdom). 2008.
15. Kufazvinei TT, Chai J, Boden KA, et al. Emerging opportunities to target inflammation: myocardial infarction and type 2 diabetes. *Cardiovasc Res.* 2024;120(1):1241–52. <https://doi.org/10.1093/cvr/cvae142>.
16. Gupta N, Elnour AA, Sadeq A, et al. Diabetes and the heart: coronary artery disease. *E-J Cardiol Pract.* 2022;22(10):112–20.
17. Pop-Busui R, Boulton AJ, Feldman EL, et al. Diabetic neuropathy: a position statement by the American diabetes association. *Diabetes Care.* 2017;40(1):136–54. <https://doi.org/10.2337/dc16-2042>.
18. Cosentino F, Grant PJ, Aboyans V, et al. 2019 ESC guidelines on diabetes, pre-diabetes, and cardiovascular diseases developed in collaboration with the EASD: the task force for diabetes, pre-diabetes, and cardiovascular diseases of the European Society of Cardiology (ESC) and the European association for the study of diabetes (EASD). *Eur Heart J.* 2020;41(2):255–323. <https://doi.org/10.1093/eurheartj/ehz486>.
19. Webb D. 2017 SEMDSA diabetes management guidelines. *South African J Diab Vasc Dis.* 2018;15(1):37–40.
20. Masoudi FA, Magid DJ, Vinson DR, et al. Implications of the failure to identify high-risk electrocardiogram findings for the quality of care of patients with acute myocardial infarction: results of the emergency department quality in myocardial infarction (EDQMI) study. *Circulation.* 2006;114(15):1565–71. <https://doi.org/10.1161/CIRCULATIONAHA.106.623652>.
21. The Emerging Risk Factors Collaboration. Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: a collaborative meta-analysis of 102 prospective studies. *Lancet.* 2010;375(9733):2215–22. [https://doi.org/10.1016/S0140-6736\(10\)60484-9](https://doi.org/10.1016/S0140-6736(10)60484-9).
22. Gupta S, Gupta RK, Kulshrestha M, et al. Evaluation of ECG abnormalities in patients with asymptomatic type 2 diabetes mellitus. *J Clin Diagn Res.* 2017;11(4):OC39. <https://doi.org/10.7860/JCDR/2017/24882.9740>.
23. Harms PP, van der Heijden AA, Rutters F, et al. Prevalence of ECG abnormalities in people with type 2 diabetes: the hoorn diabetes care system cohort. *J Diabetes Complicat.* 2021;35(2):107810. <https://doi.org/10.1016/j.jdiacomp.2020.107810>.
24. Simova I, Christov I, Bortolan G. A review on electrocardiographic changes in diabetic patients. *Curr Diabetes Rev.* 2015;11(2):102–6. <https://doi.org/10.2174/1573399811666150113161417>.
25. Miwa S, Mieszczańska HZ. Cardiac Electrocardiography. *Cardiology Consult Manual* [Internet]. New York. 2018;33–62. Available from: http://dx.doi.org/10.1007/978-3-319-89725-7_3.
26. Jameson JL, Fauci AS, Kasper DL, et al. Harrison's principles of internal medicine. (No Title). 2018.
27. Kalyani RR, Lazo M, Ouyang P, et al. Sex differences in diabetes and risk of incident coronary artery disease in healthy young and middle-aged adults. *Diabetes Care.* 2014;37(3):830–38. <https://doi.org/10.2337/dc13-1755>.
28. Kanakala VV. Study of conduction blocks in acute myocardial infarction: Rajiv Gandhi University of Health Sciences (India). 2005.
29. Asghar O, Alam U, A Hayat S, et al. Obesity, diabetes and atrial fibrillation: epidemiology, mechanisms and interventions. *Curr Cardiol Rev.* 2012;8(4):253–64. <https://doi.org/10.2174/157340312803760749>.
30. Li T, Li G, Guo X, et al. The influence of diabetes and prediabetes on left heart remodeling: a population-based study. *J Diabetes Complicat.* 2021;35(2):107771. <https://doi.org/10.1016/j.jdiacomp.2020.107771>.
31. Karayiannides S. Diabetes and glucose abnormalities in cardiovascular disease: studies on prevalence and prognosis in myocardial infarction and atrial fibrillation: Karolinska Institutet (Sweden). 2022.
32. Zhao X, Liu S, Wang X, et al. Diabetic cardiomyopathy: clinical phenotype and practice. *Front Endocrinol (Lausanne).* 2022;13:1032268. <https://doi.org/10.3389/fendo.2022.1032268>.
33. Ernande L, Bergerot C, Girerd N, et al. Longitudinal myocardial strain alteration is associated with left ventricular remodeling in asymptomatic patients with type 2 diabetes mellitus. *J Am Soc Echocardiogr.* 2014;27(5):479–88. <https://doi.org/10.1016/j.echo.2014.01.001>.
34. Ritchie RH, Abel ED. Basic mechanisms of diabetic heart disease. *Circ Res.* 2020;126(11):1501–25. <https://doi.org/10.1161/CIRCRESAHA.120.315913>.
35. Lee S-Y, Shin D-H, Kim J-S, et al. The effect of sex and anthropometry on clinical outcomes in patients undergoing percutaneous coronary intervention for complex coronary lesions. *Yonsei Med J.* 2017;58(2):296. <https://doi.org/10.3349/ymj.2017.58.2.296>.
36. Mohadjer A, Brown G, Shah SR, et al. Sex-based differences in coronary and structural percutaneous interventions. *Cardiol Ther.* 2020;9:257–73. <https://doi.org/10.1007/s40119-020-00176-5>.
37. Li Y, Liu Y, Liu S, et al. Diabetic vascular diseases: molecular mechanisms and therapeutic strategies. *Signal Transduct Target Ther.* 2023;8(1):152. <https://doi.org/10.1038/s41392-023-01400-z>.
38. Bradshaw PJ, Jamrozik K, Le M, et al. Mortality and recurrent cardiac events after coronary artery bypass graft: long term outcomes in a population study. *Heart.* 2002;88(5):488–94. <https://doi.org/10.1136/heart.88.5.488>.