Available online on 15.11.2023 at http://jddtonline.info

Journal of Drug Delivery and Therapeutics

Open Access to Pharmaceutical and Medical Research

Open Access Full Text Article

Research Article

Associations of Electrocardiographic Abnormalities in Stable Type 2 Diabetes Subjects: Experience from a Tertiary Health Facility in South Eastern Nigeria

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Article Info:

Article History:

Received 22 Aug 2023
Reviewed 04 Oct 2023
Accepted 27 Oct 2023
Published 15 Nov 2023

Cite this article as:

Ezeude CM, Nkpozi MO, Abonyi MC, Onwuegbuna AA, Okechukwu UC, Anyanwu AC, Ikeabbah HE, Ezeude AM, Associations of Electrocardiographic Abnormalities in Stable Type 2 Diabetes Subjects: Experience from a Tertiary Health Facility in South Eastern Nigeria, Journal of Drug Delivery and Therapeutics. 2023; 13(11):62-72

DOI: http://dx.doi.org/10.22270/jddt.v13i11.6006

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Abstract

Introduction: Cardiovascular abnormalities are prevalent in the setting of diabetes mellitus, even among stable subjects, necessitating the need for a regular cardiovascular disease screening for this group of patients. Electrocardiogram is a simple and reliable screening test for cardiovascular abnormalities that could be easily accessible even in resource poor and rural settings. This study was carried out to determine the association between cardiovascular risk factors and electrocardiographic abnormalities in stable type 2 diabetes subjects in South Eastern Nigeria.

Materials and Methods: One hundred and thirty-six stable adults with type 2 diabetes mellitus were recruited consecutively from the out-patient diabetes clinic of Nnamdi Azikiwe University Teaching Hospital in South Eastern Nigeria. They were assessed for the risk factors for cardiovascular diseases that included smoking, obesity, dyslipidaemia, poor glycaemic control, hypertension, lack of exercise, presence of chronic kidney disease and metabolic syndrome. They also had a 12 lead electrocardiogram done. Results were analyzed using SPSS version 25. P value of < 0.05 was considered significant.

Result: A total of 128 subjects had complete results and were analyzed. There were 63 males and 65 females with a mean age of 58.43 ± 12.85 years and mean diabetes duration of 9.03 ± 7.36 years. A total of 45.3% of the subjects had electrocardiographic abnormalities. Hypertension was present in 54.9%, dyslipidaemia in 91.4%, central obesity in 74.3%, metabolic syndrome in 76.6% and chronic kidney disease in 57.9% of the subjects. Significant association was found between smoking and occurrence of AV block (p = 0.008), central obesity and QRS axis abnormality (p = 0.002), dyslipidaemia and ST segment abnormality (p = 0.001) and lack of exercise and ST segment abnormality (p = 0.000). No significant association was found between age, sex of the subjects, duration of DM, treatment modality for DM, level of glycaemic control, hypertension, presence of CKD and metabolic syndrome and any of the electrocardiographic abnormalities.

Conclusion: Electrocardiographic abnormalities are common in stable type 2 diabetic subjects. There was significant association between smoking habit, central obesity, dyslipidaemia, lack of exercise and ECG abnormalities that included AV block, QRS axis and ST segment abnormalities.

Keywords: Electrocardiographic abnormalities, stable type 2 diabetes subjects, cardiovascular disease, Nigeria.

INTRODUCTION

Cardiovascular complications are prevalent in the setting of diabetes and the risk of developing Cardiovascular diseases (CVDs) has been shown to be about two-fold higher in people with type 2 diabetes mellitus (T2DM) compared to the general population[1]. Cardiovascular abnormalities have been shown to be common even among stable T2DM subjects, necessitating the need for a regular cardiovascular disease screening for this group of patients[2]. Micro-vascular and macro-vascular abnormalities are implicated in the aetio-pathogenesis of CVDs in the background of type 2 diabetes mellitus[3]. Cardiac micro-vascular circulation abnormalities increase the risk of developing arrhythmias and sudden cardiac death[4]. High blood pressure and increased fasting blood glucose are major risk factors for micro-vascular complications while a high blood pressure is the major risk factor for macro-vascular complications among T2DM subjects[3].

Electrocardiogram is a simple noninvasive test that has been proven over time to be an easily accessible, cost effective and reliable test for evaluating cardiac abnormalities and could be very handy in resource poor and rural settings.

Chest pain most of the times could be masked in T2DM subjects with myocardial ischaemia. This medical condition termed "silent myocardial ischaemia" is common among diabetic subjects with autonomic dysfunction and resting ECG abnormalities have been found to be markers of silent ischaemia among asymptomatic diabetic subjects[5]. Although resting ECG may not be sufficient to diagnose some cardiovascular abnormalities, including silent cardiovascular disease, however, the use of coding system such as the Minnesota coding system may improve its utility[6].

In a scenario where a T2DM subject has a normal ECG finding despite a suspected myocardial ischaemia, exercise/stress ECG becomes the next choice screening test for the possible silent myocardial ischaemia[7]. Routine CVD screening in diabetic patients using ECG not only aims at identifying unrecognized CVDs, but also determining the risk for a future CVD event and thus equally serve as a vital preventive strategy for CVDs in diabetics[8,9].

Common electrocardiographic abnormalities in T2DM subjects include sinus tachycardia, heart rate variability, ST-T changes, left ventricular hypertrophy (LVH) and others[10.11]. Globally, about 30% of asymptomatic T2DM patients showed ECG abnormalities[11]

The risk factors for CVDs in the setting of T2DM include central obesity, smoking, dyslipidaemia, hypertension, lack of exercise among others. A clustering of these risk factors is described as metabolic syndrome and improvement in these factors appear to reduce the risk of diabetes and its complications[12].

The risk factors for ECG abnormalities evaluated by this study included smoking, obesity, dyslipidaemia, lack of exercise, poor glycaemic control, hypertension, presence of chronic kidney disease (CKD) and metabolic syndrome.

There is a paucity of studies on the associations of ECG abnormalities in T2DM subjects, especially the stable outpatients in the sub-Saharan Africa generally and South Eastern Nigeria in particular. This study sets out to bridge this gap in knowledge and stimulate further studies on this very important topic.

MATERIALS AND METHODS

This was a cross-sectional observational study conducted among clinically stable T2DM subjects who were evaluated for the risk factors of ECG abnormalities at the outpatient diabetes clinic at Nnamdi Azikiwe University Teaching Hospital, (NAUTH) Nnewi in South Eastern Nigeria. This study was conducted between July, 2022 and April, 2023. A total of 136 T2DM subjects were recruited for the study, 128 subjects had complete results and were analyzed. A convenient sampling method was used for recruiting the study subjects as all consenting patients with T2DM that presented to the diabetes clinic and who met the inclusion criteria were recruited for the study as they were seen consecutively. Subjects were excluded if they were less than 30 years of age, had T1DM, were pregnant, had clinical signs suggestive CVD(s) or were very ill respectively. The subjects were met on two separate occasions. At the first meeting a focused medical history of each of the subjects was taken, a focused examination, blood pressure and anthropometric measurements were respectively done. Other relevant data were extracted using a researcher structured and administered study protocol. Next, a resting electrocardiogram was done using Schiller AT-102 plus 12 lead ECG Machine. Ten electrodes were placed in the specific anatomic positions to obtain quality tracings. The four limb leads were applied to the four limbs: the right and the left legs and the right and left arms. The six chest leads were applied at the precordial locations (V1-V6). The recording was done over a period of about 10 seconds after the connections were made.

The ECG recordings were interpreted by a cardiologist using the University of Minnesota Codes for Resting Electrocardiograms[6]. The second contact with the participants was on another clinic appointment between 8 a.m and 9 a.m, after they had fasted for about 10-12 hours as instructed. Biochemical tests that included fasting blood glucose (FBG), glycated haemoglobin (HbA1c), fasting lipid profile (FLP) and serum creatinine were done. A total of 7ml of blood was collected from each subject following a venipuncture of the cubital vein, while observing full aseptic procedures, 2ml for FBS, 1ml for HbA1c, 4ml for both FLP and serum creatinine.

The samples for HbA1c were collected in EDTA bottles and measured with automated CLOVER A1c Analyzer (Infopia, Korea) and CLOVER A1c Self-Test Cartridge using the boronate affinity method[13]. The samples for FPG were collected in fluoride oxalate bottles and measured by the Trinder glucose oxidase method[14]. The blood samples for FLP and serum creatinine were collected in plain bottles. High density lipoprotein (HDL) level was measured by precipitation technique[15]. Total cholesterol level was determined using the kit employing the enzymatic and the 4-hydroxybenzoate/4-aminophenazone system (BioSystems)[16]. Triglyceride level was determined using a kit employing enzymatic hydrolysis of triglyceride with lipases (Randox) and low density lipoprotein cholesterol (LDL-C) was measured using a kit employing a precipitation technique[17, 18]. Serum creatinine was measured using Jaffe's reaction[19]. Estimated glomerular filteration rate (eGFR) was estimated from calibrated serum creatinine values using the four-variable Modification of Diet in Renal Disease (MDRD) study equation[20]. Weight and height were measured using Stadiometer (RGZ -120), waist circumference measured with a measuring tape and blood pressure measured using Accoson mercury Sphygmomanometer.

Statistical Analysis

Data were analyzed using SPSS version 25 (Chicago, IL, USA). Categorical data were analyzed and compared using Chi-square test: results presented in frequencies and percentages. The mean values of continuous variables were calculated and compared among groups using student's t-test and analysis of variance (ANOVA). The level of significance was set at $p < 0.05$.

DEFINITION OF TERMS AND CRITERIA

1. Hypertension was defined as systolic BP ≥ 140mmHg and or diastolic BP ≥ 90 mmHg measured on at least 2 separate occasions or if a patient is already on anti-hypertensive medications[21].
2. Poor glycaemic control was taken as HbA_{1C} ≥ 7.0%[22].
3. Global obesity was defined by body mass index (BMI) >30 (kg/M^2)[22].
4. Central obesity (abdominal obesity) was defined as waist circumference (WC) > 102 cm in men and 88 cm in women[23].
5. Dyslipidaemia was taken as HDL-C < 40 mg/dl or TG ≥ 150 mg/dl or LDL-C ≥100 mg/dl or total cholesterol (TC) ≥ 200 mg/dl or if the patient is on lipid lowering agents[23].
6. Diabetes mellitus was defined by fasting plasma glucose of ≥ 7.0 mmol/l (126 mg/dl) measured on at least 2 separate

occasions or the patient is already on glucose lowering agents[22].

7. Type 1 DM was defined as subjects with DM who are dependent on insulin for survival and are at risk for ketoacidosis[23].
8. Type 2 DM was defined as patients with DM on diet therapy either alone or in combination with oral glucose lowering agent(s) for glycaemic control[23].
9. Major ECG abnormality was defined as pathological Q waves (codes 1-1 and 1-2), marked ST depression ST depression (codes 4-1 and 4-2) and/or T wave inversion (codes 5-1 and 5-2), bundle branch block (codes 7-1 and 7-2) or some significant arrhythmias. Minor ECG abnormalities were defined as high voltage, axis deviation and lesser degrees of ST-T wave abnormality (4-3,5-3). "Ischaemic ECG" was defined as pathological Q waves (any code 1), ST and or T wave inversion of any degree (any code 4 or 5) or left bundle branch block (code 7, 1-1). Left ventricular hypertrophy was defined as a combination of high voltage and either ST depression or T wave inversion on the basis of appropriate Minnesota code[6].
10. Metabolic syndrome was defined by the presence of three (3) or more of the following five (5) criteria: WC > 101.6 cm in men and 88.9 cm in women, BP > 130/80 mmHg, fasting TG > 150 mg/dl (1.7 mmol/l), fasting HDL < 40 mg/dl (1.0 mmol/l) for men and 50 mg/dl (1.3 mmol/l) for women and FBS > 100 mg/dl (5.5 mmol/l) or the patient is a known diabetic[23].
11. Young age was taken as 18-44 years, middle age as 45-64 years and old age as 65 years and above[24].
12. Chronic kidney disease (CKD) was determined by eGFR < 90 ml/min/24 hours or patient had proteinuria[20].

RESULTS

A total of 128 T2DM subjects who had complete results and were analyzed. They were made up of 63 (49.2%) males and 65 (50.8%) females.

Clinical characteristics of the study subjects

The mean age of the subjects was 58.43 ± 12.85 years, mean duration of DM was 9.03 ± 7.36 years, mean BMI was 27.96 ± 5.61 kg/m^2, mean HbA1c was 8.28 ± 2.11%, mean SBP was 131.28 ± 21.26 mmHg and mean DBP was 77.05 ± 12.41mmHg. Also the mean value of serum creatinine was 88.77 ± 24.88 µmol/L and the mean eGFR was 86.10 ± 27.7 mL/min. The mean TC was 4.57 ±1.32 mmol/L, Tg was 1.37 ± 0.84 mmol/L, HDL-C was 1.06 ± 0.27 mmol/L and LDL-C was 2.90 ± 1.19 mmol/L (details in Table 1).

For the co-morbid conditions, hypertension was present in 76 (59.4%) of the subjects, dyslipidaemia in 117 (91.4%), metabolic syndrome in 98 (76.6%), global obesity in 45 (35.2%), central obesity in 95 (74.3%) and CKD in 74 (57.9) of the study participants (details as in Table 4).

For diabetes treatment, 3 (2.3%) subjects were on diet alone, 91 (71.1%) on oral anti-diabetic drugs (OADs), 11 (8.6%) on insulin and 23 (18.0%) were on a combination of OADs and insulin (details in Table 3).

Socio demographic characteristics of the study subjects

Among the participants 106 (82.8%) subjects were married, 4 (3.2%) were single and 18 (14.1%) were widowed. 2 (1.6%) had no formal education, 45 (35.1%) had primary, 28 (21.9%) secondary and 54 (41.1%) had tertiary education. 18 (14.1%) subjects smoked cigarette: 16 (12.5%) smoked previously and 4 (3.1%) were still smoking. 22 (17.2%) subjects engaged in regular exercise (details in Table 2).

A total of 45.3% subjects had abnormal ECG findings that included Q-wave abnormality (4.7%), QRS abnormality (21.9%), LVH (10.2%), T-wave abnormality (21.9%), ST segment abnormality (3.9%), AV block (0.8%), BBB (4.7%), sinus rhythm abnormality (22.7%), atrial enlargement (21.1%) and CAD (23.4%).

Significant association was found between smoking habit and occurrence of AV block (p = 0.008), central obesity and QRS axis abnormality (p = 0.002), dyslipidaemia and ST segment abnormality (p = 0.001) and lack of exercise and ST segment abnormality (p = 0.000) (details in Tables 7, 11, 14 & 16).

No significant association was found between age, sex of the subjects, treatment modality for DM, duration of DM, level of glycaemic control, hypertension, presence of CKD and metabolic syndrome and any of the ECG abnormalities, although all the ECG abnormalities were more among the T2DM subjects with poor glycaemic control, hypertension, CKD and metabolic syndrome (details in Tables 5, 6, 8, 9, 10, 12, 13 & 15).

Table 1: Baseline characteristics of the study population

Variable	Minimum	Maximum	Mean ± SD
Age (years)	32.00	93.00	58.43 ± 12.85
Serum creatinine (µmo;/L)	53.00	188.00	89.18 ± 24.61
eGFR (ml/min/24 hours)	33.70	168.5.0	85.83 ± 24.95
TC (mmol/L)	2.38	6.89	4.52 ± 1.12
Tg (mmol/L)	0.70	3.48	1.35 ± 0.63
HDL (mmol/L)	0.45	2.07	1.06 ± 0.27
LDL(mmol/L)	1.82	4.90	3.06 ± 0.84

eGFR = estimated glomerular filtration rate, TC = total cholesterol; Tg = Triglyceride; HDL = high density lipoprotein; LDL = low density lipoprotein

Table 2: Socio-demographic characteristics of the study population

Variable	Options	Frequency n (%)
Age (years)	<45	19 (14.8)
	45-64	62 (48.4)
	>64	47 (36.7)
Sex	Male	63 (49.2)
	Female	65 (50.8)
Marital status	Single	4 (3.2)
	Married	106 (82.8)
	Widowed	18 (14.1)
Previous smoking	Yes	16 (12.5)
	No	112 (87.5)
Current smoking	Yes	4 (3.1)
	No	124 (96.9)
Cigarette smoking	Yes	18(14.1)
	No	110(85.9)
Exercise	Yes	22 (17.2)
	No	106 (82.8)

Table 3: Clinical characteristics of the study subjects

Variable	Options	Frequency n (%)
DM treatment	Diet alone	3 (2.3)
	OADs	91 (71.1)
	Insulin	11 (8.6)
	Both OADs & Insulin	23 (18.0)
DM duration	Short duration	46 (35.9)
	Long duration	82 (64.1)
Glycaemic control	Good	29(22.7)
	Poor	99 (77.3)
Central obesity	Yes	95 (74.2)
	No	33 (25.8)
Body mass index	Normal	42 (32.8)
	Overweight	41 (32.0)
	Global obesity	45 (35.2)
Global obesity class	Class I	32 (71.1)
	Class II	10 (22.2)
	Class III	3 (6.7)
Stage of CKD	Stage I	6 (8.1)
	Stage II	47 (63.5)
	Stage III	21 (28.4)
Hypertensive	Yes	76 (59.4)
	No	52 (40.6)

DM = diabetes mellitus; OADs = oral anti diabetic drugs; CKD = chronic kidney disease; ACEIs = angiotensin converting enzyme inhibitors; ARBs = angiotensin receptor blockers

Table 4: Distribution of co-morbidities among the subjects

Variable	Frequency n (%)	
	Present	Absent
CKD	74 (57.8)	54 (42.2)
Hypertension	76 (59.4)	52 (40.6)
Dyslipidaemia	117 (91.4)	11 (8.6)
Metabolic syndrome	98 (76.6)	30 (23.4)

CKD = chronic kidney disease

Table 5: Association of ECG abnormalities with the age of the subjects

ECG abnormalities/Age	Frequency n (%)			p-value
	<45yrs	45-64yrs	>64yrs	
Q-wave pathology	0	2 (1.6)	4 (3.1)	0.251
QRS axis abnormality	3 (2.3)	8 (6.3)	17 (13.3)	0.011
Left ventricular hypertrophy	0	5 (3.9)	8 (6.3)	0.087
T-wave abnormality	3 (2.3)	13 (10.2)	12 (9.4)	0.667
ST segment abnormality	1 (0.8)	1 (0.8)	3 (2.3)	0.421
AV block	0	1 (0.8)	0	0.585
Bundle branch block	1 (0.8)	1 (0.8)	4 (3.1)	0.239
Sinus rhythm abnormality	4 (3.1)	16 (12.5)	9 (7.0)	0.893
Atrial enlargement	4 (3.1)	13 (10.2)	10 (7.8)	0.999
CAD	5 (3.9)	14 (10.9)	11 (8.6)	0.945

Table 6: Association of ECG abnormalities with the sex of the subjects

ECG abnormalities/Sex	Frequency n (%)		p-value
	Male	Female	
Q-wave pathology	2 (1.6)	4 (3.1)	0.425
QRS axis abnormality	11 (8.6)	17 (13.3)	0.234
Left ventricular hypertrophy	7 (5.5)	6 (4.7)	0.725
T-wave abnormality	10 (7.8)	18 (14.1)	0.106
ST segment abnormality	3 (2.3)	2 (1.6)	0.623
AV block	1 (0.8)	0	0.308
Bundle branch block	3 (2.3)	3 (2.3)	0.969
Sinus rhythm abnormality	9 (7.00	20 (15.6)	0.083
Atrial enlargement	13 (10.2)	14 (10.9)	0.900
CAD	16 (12.5)	14 (10.9)	0.606

Table 7: Association of ECG abnormalities with smoking

ECG abnormalities/Smoking	Frequency n (%)		p-value
	Smoked	Never smoked	
Q-wave pathology	1 (0.8)	5 (3.9)	0.752
QRS axis abnormality	6 (4.7)	22 (17.2)	0.106
Left ventricular hypertrophy	3 (2.3)	10 (7.8)	0.224
T-wave abnormality	5 (3.9)	23 (18.0)	0.332
ST segment abnormality	0	5 (3.9)	0.389
AV block	1 (0.8)	0	0.008
Bundle branch block	0	6 (4.7)	0.343
Sinus rhythm abnormality	4 (3.1)	25 (19.5)	0.847
Atrial enlargement	6 (4.7)	21 (16.4)	0.086
CAD	8 (6.3)	22 (17.2)	0.007

Table 8: Association of ECG abnormalities with the treatment for Diabetes Mellitus

ECG abnormalities/DM treatment	Frequency n (%)			p-value
	OADs	Insulin	Both	
Q-wave pathology	6 (4.7)	0	0	0.465
QRS axis abnormality	20 (15.6)	0	8 (6.3)	0.104
Left ventricular hypertrophy	7 (5.5)	2 (1.6)	4 (3.1)	0.385
T-wave abnormality	18 (14.1)	0	8 (6.3)	0.028
ST segment abnormality	3 (2.3)	0	2 (1.6)	0.559
AV block	0	1 (0.8)	0	0.013
Bundle branch block	3 (2.3)	0	3 (2.3)	0.197
Sinus rhythm abnormality	19 (14.9)	3 (2.3)	7 (5.5)	0.897
Atrial enlargement	16 (12.5)	2 (1.6)	9 (7.0)	0.112
CAD	20 (15.6)	2 (1.6)	8 (6.3)	0.510

Table 9: Association of ECG abnormalities with the duration of Diabetes Mellitus

ECG abnormalities/DM duration	Frequency n (%)		p-value
	Long duration	Short duration	
Q-wave pathology	6 (4.7)	0	0.060
QRS axis abnormality	20 (15.6)	8 (6.3)	0.358
Left ventricular hypertrophy	9 (7.0)	4 (3.1)	0.682
T-wave abnormality	17 (13.3)	11 (8.6)	0.676
ST segment abnormality	4 (3.1)	1 (0.8)	0.449
AV block	0	1 (0.8)	0.180
Bundle branch block	3 (2.3)	3 (2.3)	0.462
Sinus rhythm abnormality	19 (14.8)	10 (7.8)	0.905
Atrial enlargement	17 (13.3)	10 (7.8)	0.893
CAD	17 (13.3)	13 (10.2)	0.335

Table 10: Association of ECG abnormalities with glycaemic control

ECG abnormalities / HbA1c control	Frequency n (%)		p-value
	Good control	Poor control	
Q-wave pathology	2 (1.6)	4 (3.1)	0.593
QRS axis abnormality	10 (7.8)	18 (14.1)	0.108
Left ventricular hypertrophy	5 (3.9)	8 (6.3)	0.206
T-wave abnormality	7 (5.5)	21 (16.4)	0.913
ST segment abnormality	2 (1.6)	3 (2.3)	0.401
AV block	0	1 (0.8)	0.570
Bundle branch block	2 (1.6)	4 (3.1)	0.593
Sinus rhythm abnormality	7 (5.5)	22 (17.2)	0.691
Atrial enlargement	8 (6.3)	19 (14.8)	0.460
CAD	7 (5.5)	23 (18.0)	0.897

Table 11: Association of ECG abnormalities with central obesity

ECG abnormalities/Abdominal obesity	Frequency n (%)		p-value
	Present	Absent	
Q-wave pathology	5 (3.9)	1 (0.8)	0.601
QRS axis abnormality	27 (21.2)	1 (0.8)	0.002
Left ventricular hypertrophy	10 (7.8)	3 (2.3)	0.814
T-wave abnormality	24 (18.8)	4 (3.1)	0.116
ST segment abnormality	3 (2.3)	2 (1.6)	0.458
AV block	0	1 (0.8)	0.088
Bundle branch block	6 (4.7)	0	0.139
Sinus rhythm abnormality	21 (16.4)	8 (6.3)	0.699
Atrial enlargement	20 (15.6)	7 (5.5)	0.985
CAD	23 (18.0)	7 (5.5)	0.726

Table 12: Association of ECG abnormalities with hypertension

ECG abnormalities/Hypertension	Frequency n (%)		p-value
	Present	Absent	
Q-wave pathology	5 (3.9)	1 (0.8)	0.221
QRS axis abnormality	18 (14.1)	10 (7.8)	0.549
Left ventricular hypertrophy	10 (7.8)	3 (2.3)	0.174
T-wave abnormality	16 (12.5)	12 (9.4)	0.786
ST segment abnormality	3 (2.3)	2 (1.6)	0.977
AV block	0	1 (0.8)	0.225
Bundle branch block	4 (3.1)	2 (1.6)	0.710
Sinus rhythm abnormality	17 (13.3)	12 (9.4)	0.499
Atrial enlargement	17 (13.3)	10 (7.8)	0.669
CAD	18 (14.1)	12 (9.4)	0.937

Table 13: Association of ECG abnormalities with CKD

ECG abnormalities / CKD	Frequency n (%)		p-value
	Present	Absent	
Q-wave pathology	5 (3.9)	1 (0.8)	0.195
QRS axis abnormality	17 (13.3)	11 (8.6)	0.725
Left ventricular hypertrophy	10 (7.8)	3 (2.3)	0.141
T-wave abnormality	17 (13.3)	11 (8.6)	0.725
ST segment abnormality	4 (3.1)	1 (0.8)	0.305
AV block	1 (0.8)	0	0.391
Bundle branch block	4 (3.1)	2 (1.6)	0.653
Sinus rhythm abnormality	18 (14.1)	11 (8.6)	0.854
Atrial enlargement	20 (15.6)	7 (5.5)	0.054
CAD	16 (12.5)	14 (10.9)	0.570

Table 14: Association of ECG abnormalities with dyslipidaemia

ECG abnormalities / Dyslipidaemia	Frequency n (%)		p-value
	Present	Absent	
Q-wave pathology	6 (4.7)	0	0.744
QRS axis abnormality	26 (20.3)	2 (1.6)	0.752
Left ventricular hypertrophy	12 (9.4)	1 (0.8)	0.889
T-wave abnormality	27 (21.1)	1 (0.8)	0.530
ST segment abnormality	4 (3.1))	1 (0.8)	0.001
AV block	1 (0.8)	0	0.954
Bundle branch block	6 (4.7)	0	0.744
Sinus rhythm abnormality	29 (22.7)	0	0.067
Atrial enlargement	26 (20.3)	1 (0.8)	0.559
CAD	26 (20.3)	4 (3.1)	0.503

Table 15: Association of ECG abnormalities with metabolic syndrome

ECG abnormalities/Metabolic Syndrome	Frequency n (%)		p-value
	Present	Absent	
Q-wave pathology	6 (4.7)	0	0.165
QRS axis abnormality	23 (18.0)	5 (3.9)	0.430
Left ventricular hypertrophy	12 (9.4)	1 (0.8)	0.157
T-wave abnormality	21 (16.4)	7 (5.5)	0.825
ST segment abnormality	3 (2.3)	2 (1.6)	0.372
AV block	1 (0.8)	0	0.579
Bundle branch block	6 (4.7)	0	0.165
Sinus rhythm abnormality	25 (19.5)	4 (3.1)	0.275
Atrial enlargement	25 (19.5)	2 (1.6)	0.027
CAD	22 (17.2)	8 (6.3)	0.633

Table 16 Association of ECG abnormalities with exercise habit

ECG abnormalities/exercise	Frequency n (%)		p-value
	Present	Absent	
Q-wave pathology	1 (0.8)	5 (3.9)	0.155
QRS axis abnormality	3 (2.3)	25 (19.5)	0.088
Left ventricular hypertrophy	2 (1.6)	11 (8.6)	0.054
T-wave abnormality	2 (1.6)	26 (20.3)	0.487
ST segment abnormality	2 (1.6)	3 (2.3)	0.000
AV block	0	1 (0.8)	0.824
Bundle branch block	1 (0.8)	5 (4.1)	0.155
Sinus rhythm abnormality	2 (1.6)	27 (21.1)	0.785
Atrial enlargement	2 (1.6)	25 (19.5)	0.452
CAD	3 (2.3)	27 (21.1)	0.116

DISCUSSION

This study evaluated the associations of electrocardiographic abnormalities in apparently stable T2DM subjects.

Association between ECG abnormalities and the cardiovascular risk factors

The risk factors for CVDs evaluated by this study included age and sex of the subjects, duration of diabetes, smoking and exercise habits, central obesity, dyslipidaemia, glycaemic control, hypertension, presence of CKD, DM treatment and metabolic syndrome.

This study found significant association between smoking habit and QRS axis abnormality, central obesity and QRS axis abnormality, dyslipidaemia and ST segment abnormality and exercise habit and ST segment abnormality. Khanal MK et al found significant association between age, body mass index (BMI) and duration of T2DM greater than 5 years and ECG abnormalities[25]. Sinamaw D et al found that overweight, fasting blood sugar (FBS) ≥ 130 mg/dl and duration of DM over 10 years were significantly associated with ECG abnormalities, while Harms PP et al found that hypertension was significantly associated with ECG abnormalities[8,26]. Bedane DA et al also found that BMI ≥ 25 Kg/m^2 (over weight) and long duration of DM ≥ 10 years were associated with ECG abnormalities[27].

This study did not find significant association between the age, sex of subjects, duration of DM, treatment modality for DM, glycaemic control, hypertension, presence of CKD, metabolic syndrome and ECG abnormalities. However, higher prevalence rates of all the ECG abnormalities evaluated were more among the subjects with poor glycaemic control, hypertension, CKD and metabolic syndrome.

Some other studies contrastingly found significant association between age and duration of T2DM and ECG abnormalities[25,26-28]. In India most of the ECG abnormalities in asymptomtic T2DM subjects were observed among those with DM duration of 5-10 years. Unlike the finding from this study, 70% of ECG abnormalities occurred in the subjects with poor glycaemic control, increased triglycerides and decreased HDL cholesterol[29]. Nazimeek-Siewmak B et al found that an elevated blood pressure (BP) and increased fasting blood sugar were major risk factors for microvascular complications while elevated BP is the major risk factor for macrovascular complications in T2DM subjects[3].

The reason for the differences in these findings may be due to the differences in subject selection and in the study designs. Also the fact that majority (68%) of the subjects we studied were on lipid lowering agents as at the time of the study could also be a factor. In Italy, a study found significant graded association between decreasing eGFR values and the risk of cardiac conduction defects on electrocardiogram[30]. Equally Chang YK et al found a significantly higher odd ratio of micro albuminuria occurrence in patients with premature supraventricular contraction or tachycardia compared to those without ECG abnornomalities[31]. This study found that the prevalence rates of all the ECG abnormalities evaluated were higher among the subjects with CKD compared to those without CKD, although this was not statistically significant. This makes our finding similar to those of Mantovani A et al and Chang YK et al respectively.

Strength of the study

There is a dearth of published studies on the associated factors (risk factors) of electrocardiographic abnormalities in type 2 DM subjects, especially the apparently stable subjects in the sub-Saharan Africa. This group of patients could easily be missed during routine investigations for cardiovascular diseases and their risk factors. Some of existing data on this very vital topic are old compared to the geometrically rising trend in diabetes prevalence, and consequently its' complications. This study intended to bridge these gaps in literature.

Limitations

This study is hospital-based and a similar community-based study may be needed to better reflect the true association of these risk factors and ECG abnormalities in our rural communities. Also being a cross sectional study, the "snap shot" nature did not allow the researchers make inferences about cause and effect of the risk factors for the ECG abnormalities in the population studied,

CONCLUSION

The electrocardiographic abnormalities were high in the subjects with T2DM, even among the apparently stable subjects and this is expected to rise further as the burden of DM, especially T2DM continues to rise.

There was significant association between smoking habit, central obesity, dyslipidaemia, exercise habit and ECG

abnormalities that included AV block, QRS axis and ST segment abnormalities.

Ethical Approval

Ethical clearance was obtained from the Research Ethics Committee of the Nnamdi Azikiwe University Teaching Hospital, Nnewi.

Competing Interests: None.

Authors contributions:

Ezeude CM – conception, design of research and manuscript writing

Nkpozi MO – design of research and manuscript writing

Abonyi MC – Literature search

Onwuegbuna AA – data collection and interpretation

Okechukwu UC– literature search and manuscript writing/ editing

Anyanwu AC – manuscript writing and editing

Ikeabbah HE – literature search and editing of the manuscript

Ezeude AM – data collection/cleaning, data analysis and manuscript editing

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