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RESEARCH

A retrospective analysis of electrocardiographic abnormalities found in black South African patients with diabetes attending a regional hospital in KwaZulu-Natal

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Objectives: : Diabetes mellitus increases the risk of coronary heart disease and myocardial infarction (MI). Silent MI occurs with greater frequency in patients with diabetes with or without autonomic neuropathy and carries a similar prognosis to overt MI. Regular electrocardiographic (ECG) assessment is integral in the chronic management of patients with diabetes. Limited data exist on the spectrum of ECG abnormalities of black South African patients with diabetes. The primary aim of this study was a description of ECG abnormalities found and the secondary aim was the determination of factors associated with left ventricular hypertrophy (LVH) and MI.j

Setting and participants: The study was carried out at a regional hospital diabetes clinic in Pietermaritzburg, KwaZulu-Natal. The initial ECGs performed on patients from October 1, 2012 to September 30, 2014 were analysed. The first ECGs of 637 black South African patients with diabetes were studied, representing 80.1% of all eligible patients.

Results: The major ECG abnormalities detected were those suggestive of LVH (36.0%), MI (21.7%), conduction defects (17.7%), T-wave inversion (14.1%) and ventricular ectopics (6.8%). Most infarctions were silent (89.8%), and affected the inferior territory significantly more than anterior, lateral and antero-lateral territories (52.9% vs. 33.3% vs. 9.4% vs. 1. 5%, respectively). A substantial percentage of patients with MI failed to achieve target HbA1c and triglyceride levels and waist-to-height-ratios. Diet, exercise and self-monitoring of glucose were all associated with positive effects on both MI and LVH. A greater percentage of patients with LVH had evidence of MI versus those without hypertrophy (28.4% vs. 19.4%).

Conclusion: This study demonstrates a high prevalence of undiagnosed MI within this cohort of South African patients with diabetes. Patients with LVH were more prone to infarction. Poor glycaemic and triglyceride control and obesity were associated with infarction. Improving glycaemic and lipid control together with lifestyle modification may help prevent macrovascular cardiac complications.

Keywords: Diabetes mellitus, coronary artery disease, left ventricular hypertrophy, macro-vascular complications, South Africa, silent myocardial infarction

Introduction

Diabetes mellitus (DM) is a chronic disease with life-threatening micro- and macro-vascular complications. Coronary heart disease (CHD) is one such macro-vascular complication.[1] DM is recognised as an independent risk factor for CHD.[2] Poor glycaemic control escalates the risk of microvascular and possibly macro-vascular complications.[1] Silent coronary artery disease described in African patients,[3] and the incidence of CHD in Black African patients with diabetes, have been reported as lower than that in other populations.[4] We have previously noted that the majority of African patients followed at the Edendale hospital (EDH) diabetes clinic had suboptimal glycaemic control, with a prevalence of CHD of 6.3%, while 2.5% of these patients reported a history of myocardial infarction (MI).[5] Since poorly controlled patients with diabetes may suffer painless MI secondary to neuropathy, we believe these figures represent an underestimate, since silent myocardial ischaemia occurs more frequently in patients with diabetes with coronary disease and may occur with or without autonomic neuropathy.[6] Unrecognised or silent MI carries a serious prognosis, which is similar to the prognosis carried by overt MI.[7,8] In this study, we first describe the electrocardiographic abnormalities found among African South African patients with diabetes attending our regional hospital clinic in KwaZulu-Natal. We then proceed to identify factors associated with left ventricular hypertrophy and myocardial infarction.

Patients and methods

EDH is regional hospital situated in Pietermaritzburg, KwaZulu-Natal. All patients seen at this clinic are advised to have a standard 12-lead resting ECG performed annually using an Edan SE 1200® machine (Edan instruments Inc, China). Although all patients were advised to have their ECG performed, a compliance rate of 80.1% of eligible patients was obtained. We analysed the ECG retrospectively for the period October 1, 2012 to September 30, 2014. One observer interpreted ECGs and no automated ECG-generated reports were used for analysis purposes. These ECGs were collected and stored on a weekly basis. The quality of the ECGs was good. For the purposes of this study only the first ECG was considered. Future studies will concentrate on changes noted in subsequent ECGs performed on patients. The following abnormalities were documented: left ventricular hypertrophy (LVH), right ventricular hypertrophy (RVH), bundle branch block (BBB), P-wave abnormalities, T-wave abnormalities, axis, presence of pathological Q-waves, premature ventricular complexes (PVCs) and arrhythmias.

We diagnosed LVH where either of the following criteria were satisfied: the Sokolov-Lyon criteria (sum of amplitude of S wave in lead V1 or V2 and the R wave in lead V5 or V6 the amplitude of the R wave in lead aVL 11),[9] or the Cornell voltage criteria (sum of the amplitude of S wave in lead V3 and R wave in lead aVL > 24 mm in females and 28 mm in males).[10] The presence of pathologic Q-waves on ECG was used to diagnose prior MI. Pathologic Q-waves were defined as any Q-wave in leads V2–V3 ≥ 0.02s or QS complex in leads V2 and V3, or a Q-wave ≥ 0.03s and > 0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V4–V6 in any two contiguous leads in (I, aVL, V6, V4–V6, II, II, III, and aVF).[11] Antman *et al.* stated that an ECG with Q-waves meeting the abovementioned criteria is indicative of a previous MI.[12] Use of Q-waves to diagnose prior MI was also reinforced in other studies conducted by Pahlm *et al.*,[13] Horan *et al.*,[14] Savage *et al.*[15] and in the 2007 universal definition of myocardial infarction consensus statement.[11] However, using Q-waves has generally not been used as the conventional criterion to diagnose previous MI in epidemiologic studies. The territory of the infarct was classified as the presence of pathological Q-waves in ≥ two contiguous leads as follows: inferior territory: leads II, III, aVF; anterior territory: leads V1–V3; lateral territory: leads V5, V6, I, aVL; anterolateral territory: leads V1–V6 in addition to I, aVL. We recorded T-wave inversion when there was inversion of the T-wave in two or more adjacent leads in the absence of features of LVH. If LVH was present, then T-wave inversion was classified as LVH with strain pattern.

Left axis deviation (LAD) was diagnosed based on a hexaxial reference system when the R axis was between -30^0 and -90^0 and right axis deviation (RAD) diagnosed when the R axis was between $+90^0$ and $+180^0$. Left anterior fascicular block (LAFB) was diagnosed when there was LAD and T-wave inversion in lead aVL. Bifasicular block was documented when there was left anterior hemiblock present together with right bundle branch block (RBBB). Left posterior fascicular block (LPFB) was diagnosed when there was RAD together with RBBB. In this study, the term conduction defects encompassed any one or more of the following aberrations:

- RBBB;
- LBBB;
- LAFB;
- LPFB;
- bifasicular block;
- primary AV block;
- atrial fibrillation;
- atrial flutter.

The following clinical data were captured from the patient records: sex, age, smoking status, alcohol intake; medical history—type and duration of DM, HIV status, hypertension, compliance with diet and exercise as prescribed by the Society of Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA) guidelines,[16] performance of self-monitoring of blood glucose, blood pressures (both sitting and standing), height, weight, body mass index (BMI), waist circumference and waist-to-height-ratio (WTHR). We recorded the glycosylated haemoglobin (HbA1c), total cholesterol, serum triglycerides and the presence of proteinuria was determined using Makromed® (Makro Medical Pty, Denmark) urine dipsticks. A WTHR > 0.5 was used as a marker of obesity and a predictor of adverse cardiovascular outcome.[17] SEMDSA guidelines were followed outlining target HbA1c (≤ 7%) and triglyceride levels (< 1.7 mmol/l).[16]

Data handling

All data were captured onto a specially designed computerised database. This computer programme used the patient's date of birth as the primary identifier to ensure patient confidentiality.

Statistical analysis

Continuous variables were documented as mean values ± standard deviations(SD), median and interquartile range. Numbers (*n*) and percentages (%) were expressed for categorical variables. Groups were compared using either chi-square, independent samples t-test or Fisher's exact test. Statistics were gathered only on patients who met the inclusion criteria. Analysis was done per individual variable without controlling for multiple comparisons. A p-value < 0.05 was used as indicator of significance. Data were analysed using the Statistical Package for Social Science (SPSS®) version 23 for windows (IBM Corp, Armonk, NY, USA).

Ethics approval

This retrospective data review study was approved by the UMgungundlovu Health Ethics Review Board (UHERB) and the Biomedical Research Ethics Committee (BREC)—BCA 194/15.

Results

The first ECGs of 637 patients were analysed, representing 80.1% of all eligible patients seen during this period. Approximately 22% (n = 136) of these patients were HIV-infected. The majority (n = 95, 69.9%) of the HIV-infected patients were females. Table 1 provides a description of the overall study patient demographics.

Over a fifth of the total patient population (n = 138, 21.7%) demonstrated ECG evidence of a previous MI (Table 2). The inferior territory was significantly more likely to be affected (p < 0.0001 [chi-square test]).

Comparisons between the groups with and without ECG evidence of MI (Table 3) revealed that patients with MI were statistically more likely to be female, have type 2 diabetes, not be compliant with lifestyle modification (diet, exercise and performing self-monitoring of blood glucose) and failing to achieve optimal weight-to-height ratio (WTHR), triglyceride and HbA1c levels.

Statistics for determinants of MI (type of DM, gender, patients achieving WTHR < 0.5, target HbA1c achieved and HIV infection) were not found to be significant except for ECG evidence of LVH (p-value 0.027 [Fisher's exact test]) and target triglyceride achieved (p-value 0.016 [Fisher's exact test]).

Evidence of LVH was present in 229 patients (36%), 198 satisfying voltage criteria without a strain pattern, and 31 demonstrating a strain pattern. Comparisons made between the groups with and without ECG evidence of LVH (Table 4) indicated a significant association between LVH and MI. There was a significant inverse association noted between LVH and following lifestyle modification (diet, exercise and performing SMBG).

Isolated T-wave inversion in the absence of LVH was noted in 59 (9.3%) patients, and was approximately equally distributed across the inferior, anterior and lateral territory. Conduction defects were found in 113 (17.7%) and ventricular ectopics in 43(6.8%) of the patients.

Table 1: Demographics of patient population

Item	Number of patients	Percentage of patients (%)
Male	168	26.4
Female	469	73.6
Type 1 DM	123	19.3
Type 2 DM	514	80.7
Mean (±SD) (median, interquartile range [IQR]) age in years	53.64 ± 15.01 (55.0, 20.0)	
Mean (±SD) (median, IQR) duration of DM in years	8.54 ± 8.04 (6.0, 11.0)	
Number of patients:		
Following diet	447	70.2
Following exercise	307	48.2
Performing self-monitoring of blood glucose	198	31.1
Smoking	25	3.9
Consuming alcohol	26	4.1
With hypertension	256	40.2
With HIV infection	136	21.4
Number of patients with BMI ≥ 25	488	84.7
Number of patients with WTHR > 0.5	494	93.7
Mean (±SD) (median, IQR) HbA1c %	10.96 ± 2.99 (10.8, 4.6)	
Mean (±SD) (median, IQR) BMI (kg/m^2)	33.59 ± 11.86 (32.46, 9.90)	
Mean (±SD) (median, IQR) waist circumference (cm)	106.33 ± 16.69 (106.0, 23.88)	
Mean (±SD) (median, IQR) total cholesterol (mmol/l)	5.88 ± 1.00 (5.65, 1.23)	
Mean (±SD) (median, IQR) triglyceride (mmol/l)	3.38 ± 1.65 (3.00, 1.63)	
Mean (±SD) (median, IQR) WTHR	0.67 ± 0.11 (0.67, 0.13)	
Number of patients achieving target:		
HbA1c%	118 (18.5%)	
Triglyceride	408 (64.1%)	

Table 2: Incidence and territory of MI

Territory of MI	*n*	Percentage
Total of which:	138	21.7%(138/637)
Inferior	73	52.9%
Anterior	46	33.3%
Lateral	13	9.4%
Antero-lateral	2	1.5%
Anterior plus inferior	3	2.2%
Inferior plus lateral	1	0.7%

Discussion

Studies performed in Cameroon[18] and Nigeria[19] found T-wave abnormalities to be the most common ECG aberration (20.9% and 22.0%) while the major ECG abnormalities detected in our study in decreasing order of prevalence were features suggestive of LVH (36.0%), MI (21.7%), conduction defects (17.7%), T-wave inversions (14.1%) and PVCs (6.8%). These are elaborated on below.

Myocardial infarctions (MIs)

The prevalence of MIs in this cohort of patients was much higher than in other studies conducted in Cameroon[18] and Nigeria[19] (21.7% vs. 13.6% vs. 9.0%, respectively). The increased prevalence of MIs in South Africa (a country with one of the most competitive economies in sub-Saharan Africa) compared with other countries in Africa is possibly related to high prevalence of HIV infection, rapid urbanisation, increased Western diets and obesity associated with higher earning potential. These then translate into poorer glycaemic control and increased diabetes-related complications. The prevalence of HIV infection in South Africa is approximately 10%.[20] Global studies have found that HIV-infected patients are more prone to myocardial infarction due to either HIV itself, antiretroviral therapy (ART) or the increased life expectancy.[21,22] A mechanism postulated for this increase of MIs in HIV-infected patients is that both HIV infection and ART increase the risk of atherosclerosis.[23]

Both poor glycaemic and lipid control are associated with increased risk of cardiovascular disease (CVD).[2] Within our group of patients we showed that there was an association between MI and increased triglyceride and total cholesterol levels, WTHR, LVH and lifestyle modification. Anagnostis *et al.* showed in their meta-analysis that women with diabetes have a higher relative cardiovascular risk than men with diabetes.[24] Our study demonstrated that female type 2 diabetes patients were more likely to have evidence of MI. Obesity remains a global challenge and is regarded as an independent risk factor for CVD.[25] Obesity when defined using WTHR > 0.5 was shown to be associated with the presence of prior MI on ECG.

Only a very small percentage (3.8%) of the patients had reported a history of ischaemic heart disease (IHD). From this group of IHD patients, only 2.2% reported having suffered an MI compared with 21.7% of patients who demonstrated ECG evidence of MIs. This significant difference in reported versus unreported MIs is probably related to silent MIs. Touze *et al.* in their prospective study did find that coronary heart disease does exist in a silent state in Black African patients.[3] The increased prevalence of silent MIs within our population is a strong indicator of sub-optimal glycaemic control and the resultant long-term diabetic complication of autonomic neuropathy. Overall the incidence of silent MIs is higher in patients with diabetic autonomic neuropathy.[26] Added to this cauldron of diabetic neuropathy is the high prevalence of HIV infection in South Africa. The combination of HIV and DM further compounds the risk of neuropathy.[27] Studies conducted in developed countries by Sigurdsson *et al.* found that approximately one-third (30%) of their patients had silent MIs[28] while other developed world studies including the UKPDS showed between 17.5% and 60% of all MIs were unrecognised.[29–34] Our study demonstrated a much higher incidence of silent MIs (89.84%) compared with these studies performed in developed countries. A possible reason for this difference may be explained by the high prevalence of HIV infection in South Africa. In our study, we found approximately

Table 3: Characteristics of patients with and without myocardial infarction

Item	Patients with ECG evidence of MI (*n* = 138)	Patients with no ECG evidence of MI (*n* = 499)	*p*-values calculated either via chi-square [1], independent samples t-test [2], Fisher's exact test [3], Mann–Whitney [4]
Type 1 DM	19 (13.8%)	104 (20.8%)	< 0.001 [1]
Type 2 DM	119 (86.2%)	395 (79.2%)	< 0.001 [1]
Gender: Male	31 (22.5%)	137 (27.5%)	< 0.001 [1]
Female	107 (77.5%)	362 (72.6%)	< 0.001 [1]
Mean (±SD) (median, IQR) age (years)			
Males	51.2 ± 15.2 (55.0, 30.0)	49.0 ± 15.7 (45.5, 28.0)	0.465 [2]
Females	56.7 ± 11.2 (58.5, 8.8)	54.7 ± 15.4 (56.0, 18.3)	0.226 [2]
Mean (±SD) (median, IQR) duration of DM (years)	9.5 ± 8.0 (6.0, 7.5)	8.3 ± 8.0 (8.0, 10.8)	0.126 [2]
Number of patients:			
Following diet	94 (21%)	353 (79%)	< 0.001 [3]
Following exercise	63 (20.5%)	244 (79.5%)	< 0.001 [3]
Performing self-monitoring of blood glucose	37 (18.7%)	161 (81.3%)	< 0.001 [3]
Smoking	5 (3.6%)	20 (4.0%)	0.837 [3]
Consuming alcohol	5 (3.6%)	21 (4.2%)	0.759 [3]
With hypertension	83 (60.1%)	326 (65.3%)	< 0.001 [1]
With HIV infection	29 (21%)	101 (21.4%)	0.913 [3]
Mean (±SD) (median, IQR) BMI (kg/m^2)			
Males	35.2 ± 9.3 (37.2, 18.2)	28.8 ± 6.8 (30.3, 7.5)	0.271 [2]
Females	35.2 ± 9.1 (34.7, 15.5)	34.5 ± 9.0 (32.6, 9.2)	0.528 [2]
Mean (±SD) (median, IQR) waist circumference (cm)			
Males	102.0 ± 25.7 (105.0, 27.5)	98.6 ± 18.0 (102.0, 27.8)	0.430 [2]
Females	109.3 ± 17.5 (113.0, 76.0)	107.4 ± 16.6 (104.8, 22.3)	0.342 [2]
Mean (±SD) (median, IQR) WTHR			
Males	0.70 ± 0.14 (0.64, 0.26)	0.61 ± 0.08 (0.60, 0.14)	0.119 [4]
Females	0.70 ± 0.12 (0.71, 0.16)	0.68 ± 0.12 (0.68, 0.09)	0.413 [4]
Patients achieving WTHR < 0.5	6 (4.4%)	32 (6.4%)	< 0.001 [1]
Mean sitting blood pressure (mmHg)			
Systolic BP	136	132	
Diastolic BP	81	77	
Mean (±SD) (median, IQR) HbA1c%	11.4 ± 3.2 (11.3, 3.5)	10.8 ± 2.9 (11.2, 4.9)	0.045 [2]
Mean (±SD) (median, IQR) total cholesterol (mmol/l)	6.0 ± 1.4 (5.2, 1.3)	5.6 ± 0.9 (5.5, 1.2)	0.027 [2]
Mean (±SD) (median, IQR) triglyceride level (mmol/l)	2.9 ± 1.3 (2.9, 1.9)	3.1 ± 1.4 (3.0, 1.7)	0.462 [2]
Number of patients achieving target:			
HbA1c%	16 (11.6%)	117 (23.5%)	0.018 [3]
Triglyceride	76 (55.1%)	332 (66.6%)	0.016 [3]

22% of the total patient population to be HIV-infected. The combination of HIV and DM further increases the risk of developing neuropathy.[27] Pillay *et al.* recently demonstrated that HIV-infected patients with diabetes had a higher incidence of neuropathy than their HIV-uninfected counterparts.[27] The high prevalence of HIV infection coupled with suboptimal diabetes control within our population could possibly explain the higher incidence of silent MIs. Other possibilities for this high prevalence of silent MI in developing countries could include failing and aged healthcare infrastructures, and poor access to healthcare facilities and appropriate drugs due to financial constraints. One must also take into consideration that using Q-waves to diagnose previous MI does not reflect a perfect scenario. The UKPDS[31] found a high proportion of patients who had Q-waves on ECG at baseline that disappeared after three years of the study. These Q-waves were probably false positives. However, the UKPDS also showed that patients with Q-waves on ECG were at significantly increased risk for future MI and all-cause mortality.[31] Kim *et al.* also demonstrated that a significant proportion of myocardial infarcts never evolve into Q-waves and this was either related to the position of the infarct or the degree of myocardial damage.[35]

Table 4: Characteristics of patients with and without ECG evidence of LVH

Item	Patients with ECG evidence of LVH (*n* = 229)	Patients with no ECG evidence of LVH (*n* = 408)	Tests used for *p*-values, chi-square [1], independent samples t-test [2], Fisher's exact [3], Mann–Whitney [4]
Males	71 (31.0%)	97 (23.8%)	0.045 [1]
Females	158 (69.0%)	311 (76.2%)	< 0.001 [1]
Type 1 DM	37 (16.2%)	86 (21.1%)	< 0.001 [1]
Type 2 DM	192 (83.8%)	322 (78.9%)	< 0.001 [1]
Mean (±SD) (median, IQR) age (years)	53.9 ± 15.7 (54.0, 17.0)	53.5 ± 14.7 (55.0, 18.5)	0.725 [2]
Mean (±SD) (median, IQR) duration of DM (years)	8.2 ± 7.9 (4.0, 8.0)	8.7 ± 8.1 (8.5, 9.5)	0.403 [2]
Number of patients:			
Following diet	158 (35.3%)	289 (64.7%)	< 0.001 [3]
Following exercise	113 (36.8%)	194 (63.2%)	< 0.001 [3]
Performing self-monitoring of blood glucose	70 (35.4%)	128 (64.6%)	< 0.001 [3]
	9 (3.9%)	16 (3.9%)	1.000 [3]
Smoking	9 (3.9%)	17 (4.2%)	1.000 [3]
Consuming alcohol with HIV infection	52 (22.7%)	84 (20.6%)	0.006 [1]
Mean (±SD) (median, IQR) HbA1c %	10.8 ± 3.0 (11.1, 3.8)	11.1 ± 3.0 (11.3, 4.8)	0.201 [2]
Mean (±SD) (median, IQR) total cholesterol (mmol/l)	5.7 ± 1.0 (5.7, 1.1)	5.6 ± 1.0 (5.4, 1.2)	0.583 [2]
Mean (±SD) (median, IQR) triglyceride (mmol/l)	3.0 ± 1.5 (2.9, 1.7)	3.0 ± 1.3 (3.1, 1.7)	0.691 [2]
Mean sitting BP (mmHg)	139/80	129/77	
Total number of MIs	65 (28.4%)	79 (19.4%)	0.027 [3]
Mean (±SD) (median, IQR) BMI			
Males	28.5 ± 6.33 (27.6, 8.9)	29.8 ± 7.5 (29.7, 8.9)	0.238 [4]
Females	33.68 ± 7.8 (33.1, 8.9)	35.2 ± 9.5 (34.7, 10.4)	0.159 [4]
Mean (±SD) (median, IQR) Waist circumference (cm)			
Males	95.6 ± 18.3 (96.0, 22.0)	101.2 ± 19.2 (102.0, 23.0)	0.185 [4]
Females	106.5 ± 16.2 (108.0, 19.8)	108.2 ± 17.2 (108.0, 23.0)	0.646 [4]
Mean (±SD) (median, IQR) WTHR			
Males	0.58 ± 0.11 (0.58, 0.12)	0.60 ± 0.11 (0.59, 0.13)	0.225 [4]
Females	0.68 ± 0.11 (0.69, 0.11)	0.70 ± 0.11 (0.69, 0.14)	0.511 [4]
Number of patients achieving WTHR < 0.5	14 (7.5%)	19 (5.6%)	0.452 [3]

The best approach to detecting silent MI would probably be to do serial ECGs.

Between 40% and 50% of all MIs worldwide are inferior infarctions and they are generally regarded as carrying a better prognosis than anterior MIs.[36] Inferior territory MI was the most common territory involved in our cohort of patients when compared with anterior, lateral and antero-lateral territories. This may possibly be reflecting that patients with infarcts in other territories may actually not be surviving long enough to present to healthcare facilities after suffering an MI at home.

LVH

LVH was the most common ECG finding (36%) in our cohort of patients. This result is similar to the prevalence of LVH found in Gambia by Jobe *et al.* and in Nepal by Prakash *et al.* (36% vs. 35.2% vs. 34%, respectively)[37,38] and is approximately double that found in the studies done by Dzudie *et al.* in Cameroon,[18] Olamoyegun *et al.* in Nigeria[19] and Lutale *et al.* in Tanzania[39] (36% vs. 16.4% vs. 18.5% vs. 16%, respectively). Muddu *et al.* also showed that the prevalence in Uganda of echocardiographic-diagnosed LVH in newly diagnosed patients with diabetes is 19.3%.[40] A recent study carried out by Lohrmann *et al.* in disease-free black South Africans found a prevalence of LVH diagnosed by ECG voltage criteria to be 13.1%.[41]

LVH is strongly associated with hypertension and diabetes and carries an increased risk for cardiovascular mortality,[42,43] CHD and cerebrovascular disease. Obesity and glucose intolerance may account for other causes of LVH.[44] South Africa has the highest incidence of obesity in females in sub-Saharan Africa.[45] Our study demonstrated that there was a significant correlation between BMI and LVH. The increased incidence of poor glycaemic control[5,16] and high levels of obesity in South Africa could possibly explain the higher prevalence of LVH and LVH without

associated hypertension noted in our patients compared with other African countries.

A significant percentage of our patients with LVH had underlying hypertension (45%) while 40.1% of these patients with LVH had overt proteinuria. This is an expected finding as hypertension and proteinuria are commonly associated with LVH.[40,46] Electrocardiographic evidence of LVH with repolarisation abnormality (LVH with strain pattern) is associated with an increased risk of heart failure.[47] Our study found that 31 (4.9%) of the total number of patients had evidence of LVH with strain pattern. A significant percentage of patients with, versus those without, LVH had T-wave inversions in the anterior and lateral territories.

Patients with LVH are at increased risk for CHD and MIs.[2] Our study found that a significant percentage of patients with LVH versus those without LVH had ECG evidence of MIs (28.4% vs. 19.4%, respectively). The incidence of MIs in patients with LVH was much higher than the overall incidence of MIs observed in the entire cohort (28.4% vs. 21.7%, respectively). This emphasises the impact that LVH has on the risk of CHD and MIs.

In our study LVH was significantly more common within the female type 2 patients with diabetes. This finding was similar to what Valarezo-Sevilla *et al.* found in their study of the prevalence of LVH in patients with diabetes.[48]

Having found a high prevalence of LVH within our cohort of patients and understanding the potential risks of increased cardiovascular mortality, sudden cardiac death and possible undiagnosed underlying diabetic cardiomyopathy in patients with LVH, it is imperative that methods to control glycaemia, blood pressure and obesity be escalated as a matter of urgency.

Conduction defects

Conduction defects were found in approximately one in every six patients (17.7%). Our observed prevalence of conduction defects was higher than found in studies in Nigeria[19] and Cameroon[18] (17.7% vs. 7% vs. 11.9%, respectively). This higher prevalence of conduction defects found can possibly be explained by the higher prevalence of MIs and LVH within our cohort of patients. Engel *et al.* showed that the PVCs are an independent risk factor for cardiovascular mortality. They also demonstrated an association between elevated heart rate and cardiovascular mortality even in the absence of PVCs.[49] Our study demonstrated that a much higher percentage of our patients had evidence of PVCs compared with other countries in Africa like Nigeria and Cameroon (6.8% vs. 4% vs. 4.8%, respectively).

Conclusion

This study described the ECG aberrations within a cohort of Black South African patients attending a regional hospital diabetic clinic. South Africa is already burdened by obesity, HIV infection and DM. Against this backdrop of communicable and non-communicable diseases we found that LVH was the most common ECG abnormality. Patients with LVH had an expected higher prevalence of MI versus those without LVH.

A substantial percentage of patients had ECG evidence of MI and more importantly the majority of these MIs were actually silent in nature. Obesity increases the risk not only of LVH but also of poor glycaemic control, which in turn increases the risk of developing complications like MI. Measures to improve glycaemic and lipid control and body weight will help in improving long-term complications like MIs.

Strengths and limitations of this study

- Provides an in-depth understanding of ECG abnormalities found in this cohort of black South African patients with diabetes.
- Since there are limited data on such a study in South Africa this study provides an excellent opportunity to compare these findings with findings from other countries in Africa.
- This study helps us understand the high prevalence of silent myocardial infarctions in this cohort of patients.
- The study only analysed 80.1% of eligible patients. Although all patients were advised to have their ECG performed annually not all patients followed this advice.
- The ECGs were interpreted by only one observer and therefore only one description or version of the ECG interpretation is provided.j1
- The use of Q-waves to diagnose previous myocardial infarction is not a perfect scenario as there is a possibility of both false positives and negative findings. However, this study was done in a resource-limited developing country where more invasive investigations are both costly and often unavailable to these patients. Performing an ECG provides a relatively easy, non-invasive and cheap method that can be done in these settings.

Authors' contributions – The principal author (Dr Somasundram Pillay) made substantial contributions to conception and design of the work, collection of all data, interpretation and performing statistical analyses of the data and writing of the paper and final approval and agrees to be accountable for all aspects of the research. Professor Colleen Aldous made substantial contributions to critically reviewing and editing the draft paper and final approval and agrees to be accountable for all aspects of the research. Professor Richard Hift made substantial contributions to reviewing and editing the draft paper and final approval and agrees to be accountable for all aspects of the research.

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References

1. American Diabetes Association. Implications of United Kingdom Prospective Diabetes Study. Diabetes Care 2002;(supp 1):S28–32.
2. O'Donnell CJ, Elosua R. Cardiovascular risk factors.Insight from Framhingham heart study. Rev Esp Cardiol. 2008;61(3):299–310. doi:10.1016/S1885-5857(08)60118-8.
3. Touze JE, Sess D, Darracq R, et al. Silent coronary artery disease in black African diabetic patients.A prospective study of 50 patients. Trop Geogr Med. 1987;39(2):144–7. PMID:3629707.
4. Yusuf S, Reddy S, Ounpuu S, et al. Global burden of cardiovascular diseases. Circulation. 2001;104:2855–64. https://doi.org/10.1161/hc4701.099488
5. Pillay S, Aldous C, Mahomed F. Diabetic patients served at a regional hospital-what is their clinical picture? JEMDSA. 2015;20(1):60–6.

6. Ahluwalia G, Jain P, Chugh SK, et al. Silent myocardial ischemia in diabetics with normal autonomic function. Int J Cardio. 1995;48(2):147–53. PMID 7774993. https://doi.org/10.1016/0167-5273(94)02233-9
7. Kannel WB, Abbott R. Incidence and prognosis of unrecognised myocardial infarction-an update on the Framingham study. NEJM. 1984;311:1144–7. https://doi.org/10.1056/NEJM198411013111802
8. Zellweger MJ. Prognostic significance of silent coronary artery disease in type 2 diabetes. Herz. 2006;31(3):240–5. https://doi.org/10.1007/s00059-006-2790-1
9. Sokolow M, Freidlander RD. The normal unipolar precordial and limb lead echocardiogram. Am Heart J. 1949;38:665–87. https://doi.org/10.1016/0002-8703(49)90525-6
10. Norman JE, Levy D, Campbell G, et al. Improved detection of echocardiographic left ventricular hypertrophy using a new electrocardiographic algorithm. J Am Coll Cardio. 1993;21:1680–6. https://doi.org/10.1016/0735-1097(93)90387-G
11. Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. Circulation. 2012;126. doi:10.1161/cir.0b013e31826e1058.
12. Antman E, Bassand JP, Klein W, et al. Myocardial infarction redefined-a consensus document of the Joint European Society of Cardiology/American College of Cardiology committee for the redefinition of myocardial infarction: The Joint European Society of Cardiology/American College of Cardiology Committee. Journal of the American College of Cardiology. 2000;36(3):959–69. https://doi.org/10.1016/S0735-1097(00)00804-4
13. Pahlm US, Chaitman BR, Rautaharju PM, et al. Comparison of the various electrocardiographic scoring codes for estimating anatomically documented sizes of single and multiple infarcts of the left ventricle. Am J Cardio. 1998;81:809–15. https://doi.org/10.1016/S0002-9149(98)00016-2
14. Horan LG, Flowers NC, Johnson JC. Significance of the diagnostic q wave of myocardial infarction. Circulation. 1971;43:428–36. https://doi.org/10.1161/01.CIR.43.3.428
15. Savage RM, Wagner GS, Ideker RE, et al. Correlation of postmortem anatomic findings with electrocardiographic changes in patients with myocardial infarction: retrospective study of patients with typical anterior and posterior infarcts. Circulation. 1977;55:279–85. https://doi.org/10.1161/01.CIR.55.2.279
16. Amod A, Ascott-Evans BH, Berg GI, et al. The 2012 SEMDSA guideline for the management of type 2 diabetes (revised). JEMDSA. 2012;17(2):S13–S19.
17. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes:0.5 could be a suitable global boundary value. Nutr Res Rev. 2010;23(2):247–69. https://doi.org/10.1017/S0954422410000144
18. Dzudie A, Choukem SP, Adam AK, et al. Prevalence and determinants of electrocardiographic abnormalities in sub-saharan African individuals with type 2 diabetes. Cardiovascular Journal Of Africa 2012;23(10):533–7. https://doi.org/10.5830/CVJA-2012-054
19. Olamoyegun AM, Ogunmola OO, Oladosu YT, et al. Prevalence, variants and determinants of electrocardiographic abnormalities amongst elderly Nigerians with type 2 diabetes. Journal of Medicine and Medical Sciences. 2013;4(8):324–8. doi: 10.14303/jmms.2013.107.
20. Statistics South Africa. Mid-year population estimates 2013. May 2013. [cited 16 January 2015]. Available from: http://www.statssa.gov.za/publications/P0302/P03022013.pdf.
21. Althoff KN, McGinnis KA, Wyatt C, et al. Comparison of Risk and age at diagnosis of myocardial infarction, end-stage renal disease, and non-AIDS-defining cancer in HIV-infected versus uninfected adults. Clin Infect Dis. 2015;60(4):627–38. https://doi.org/10.1093/cid/ciu869
22. Freiberg MS, Chang CH, Kuller LH, et al. HIV infection and the risk of acute myocardial infarction. JAMA Intern Med. 2013;173(8):614–22. https://doi.org/10.1001/jamainternmed.2013.3728
23. Murphy R, Costagliola D. Increased cardiovascular risk in HIV infection:drugs, virus and immunity. AIDS. 2008;22:1625–7. https://doi.org/10.1097/QAD.0b013e328306a6db
24. Anagnostis P, Majeed A, Johnston DG, et al. Cardiovascular risk in women with type 2 diabetes mellitus and prediabetesis it indeed higher than men? Eur J Endocrinology. 2014;171:R245–5.
25. Hubert HB, Feinleib M, McNamara PM, et al. Obesity as an independent risk factor for cardiovascular disease:a 26-year follow-up on particiapnts in the Framingham heart study. Circulation. 1983;67:968–77. https://doi.org/10.1161/01.CIR.67.5.968
26. Niakan E, Harati Y, Rolak LA, et al. Silent myocardial infarction and diabetic cardiovascular autonomic neuropathy. Arch Intern Med. 1986;146(11):2229–30. doi:10.1001/archinte.1986.003602300169023.
27. Pillay S, Aldous C, Mahomed F. A deadly combination - HIV and diabetes mellitus: where are we now? SAMJ. 2016;106(4):378–383. doi:10.7196/SAMJ.2016.v106i4.9950.
28. Sigurdsson E, Thorgeirsson G, Sigvaldson H. Sigfusson Unrecognised myocardial infarction: epidemiology, clinical characteristics, and the prognostic role of angina pectoris: the Reykjavik study. Annals of Internal Medicine. 1995;122(2):96–102. https://doi.org/10.7326/0003-4819-122-2-199501150-00003
29. Bertolet BD, Hill JA. Unrecognised myocardial infarction. Cardiovasc Clin. 1989;20:173–82.
30. Roseman MD. Painless myocardial infarction: a review of the literature and analysis of 22 cases. Ann Intern Med. 1954;41:1–8.
31. Davis TME, Coleman R, Holman RR. Prognostic significance of silent myocardial infarction in newly diagnosed type 2 diabetes: UKPDS79. Circulation. 2013. doi:10.1161/CIRCULATION.112.000908.
32. Davis TME, Fortun P, Mulder J, et al. Silent Myocardial infarction and its prognosis in a community-based cohort of type 2 diabetic patients: the fremantle diabetes study. Diabetogica. 2004;47:395–9. doi:10.1007/s00125-004-1344-4.
33. Wackers FJ, Young LH, Inzuchi SE, et al. Detection of ischaemia in asymptomatic diabetic investigators. Diabetes Care. 2004;27(8):1954–61. https://doi.org/10.2337/diacare.27.8.1954
34. Milan study on atherosclerosis and diabetes(MISAD) group. Prevalence of unrecognised silent myocardial iscahemia and its association with atherosclerotic risk factors in non-insulin dependent diabetes mellitus. AMJ Cardio. 1997;79(2):134–9.
35. Kim HW, Klem I, Shah DJ, et al. Unrecognised non-Q wave myocardial infarction: prevalence and prognostic significance in patients with suspected coronary disease. PLoS Med. 2009;6:e1000057. https://doi.org/10.1371/journal.pmed.1000057
36. Berger PB, Ryan TJ. Inferior myocardial infarction. Circulation. 1990;81:401–411. doi:10.1161/01.CIR.81.2.401.
37. Jobe M, Kane A, Jones JC, et al. Electrocardiographic left ventricular hypertrophy among gambian diabetes mellitus patients. Ghana Med J. 2015;49(1):19–24. https://doi.org/10.4314/gmj.v49i1.4
38. Prakash O, Karki P, Sharma SK. LVH in hypertension: correlation between electrocardiography and echocardiography. Katmandu Univ Med J 2009;7(26):97–103.
39. Lutale JJK, Thordarson H, Gulam-Abbas Z, Vetvik K, Gerdts E. Prevalence and covariates of electrocardiographic left ventricular hypertrophy in diabetic patients in Tanzania. Cardiovascular J Afr. 2008;19(1): 8–14.
40. Muddu M, Mutebi E, Mondo C. Prevalence, types and factors associated with echocardiographic abnormalities among newly diagnosed diabetic patients at Mulago Hospital. African Health Sciences. 2016;16(1): 183–93. https://doi.org/10.4314/ahs.v16i1.25
41. Lohrmann GM, Peters F, Srivathsan K, Essop MR, Mookadam F. Electrocardiographic abnormalities in disease-free Black South Africans and correlations with echocardiographic indexes and early repolarisation. The American Journal of Ccardiology. 2016;118(5):765–70. https://doi.org/10.1016/j.amjcard.2016.06.006
42. Levy D. Left ventricular hypertrophy.Epidemiological insights from the Framhingham Heart Study. Drugs. 1988;35(5):1–5. PMID 2975214. https://doi.org/10.2165/00003495-198800355-00002
43. Bella JN, Devereux RB, Roman MJ, et al. Separate and joint effects of systemic hypertension and diabetes mellitus on left ventricular structure and function in American Indians (the Strong Heart Study). Am J Cardio. 2001;87:1260–5. https://doi.org/10.1016/S0002-9149(01)01516-8

44. Kannel WB. Left Ventricular hypertrophy as a risk factor:the Framhingham experience. J Hypertens Suppl. 1991;9(2):S8–9.
45. Ng M, Fleming T, Robinson M, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: a systematic analysis for the Global Burden of Disease Study 2013. The Lancet. 2014;384(9945):766–81. doi:10.1016/S0140-6736(14)60460-8.
46. Nan W, Weiwei Z, Kuanping Y, et al. Albuminuria is associated with left ventricular hypertrophy in patients with early diabetic kidney disease. International Journal of Endocrinology. 2014;351945:8 pp.
47. Kannel WB, Levy D, Cupples LA. Left ventricular hypertrophy and risk of cardiac failure:insights from the Framingham study. J cardiovasc Pharmacol. 1987;10(6):S135–40. PMID 2485019. https://doi.org/10.1097/00005344-198700106-00018
48. Valarezo-Sevilla D, Pazmiño-Martínez A, Morales-Mora N. Prevalence of left ventricular hypertropy in diabetic patients. Rev Peru Med Exp Salud Publica. 2013;30(1):69–72. https://doi.org/10.1590/S1726-46342013000100014
49. Engel G, Cho S, Ghayoumi A, et al. Prognostic significance of PVCS and resting heart rate. Ann non-invasive electrocardiol. 2007;12(2):1–9.

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