Original Article

Electrocardiogram (ECG) Diagnosis of Left Ventricular Hypertrophy and its Associations in Patients Living with Diabetes

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Abstract

Background: Macrovascular complications of diabetes mellitus (DM) include cardiac manifestations such as left ventricular hypertrophy (LVH), which can increase the risk of heart failure and death. **Objectives:** To determine associations between LVH and other variables in patients living with DM (PLWD). **Methods:** A retrospective study over 1 year was conducted on patients who attended the DM clinic at Edendale Hospital in South Africa. Electrocardiographs (ECGs) and standardised data sheets were analysed. The Sokolov–Lyon, Cornell and Romhilt–Estes methods were utilised for diagnosing LVH on ECGs. **Results:** There were 609 PLWD included in the study, with 80 PLWD (13.1%) having LVH (LVH+), whereas 529 PLWD (86.9%) had no LVH (LVH−). The Sokolov–Lyon method proved to be the best method of diagnosing LVH based on ECG (100% of patients diagnosed), with an 'R wave in Augmented Vector Left (AVL) ≥11 mm' being the best approach for determining LVH. LVH + patients were significantly younger than LVH − patients (27.22 years vs. 58.98 years, $P < 0.001$) and had lower systolic blood pressure (SBP) (118.62 mmHg vs. 139.77 mmHg, $P < 0.001$). Type 1 DM (DM1+) comprised the majority (77.5%) of LVH + patients. LVH + patients had significantly better high-density lipoproteins (1.36 mmol/L vs. 1.25 mmol/L, $P = 0.024$) and triglycerides (1.40 mmol/L vs. 1.85 mmol/L, $P = 0.010$) than LVH − patients. **Conclusion:** LVH was seen more frequently in younger patients who often had DM1+, lower SBP, higher HDL, and lower triglycerides. Most cases of LVH were diagnosed using the Sokolov–Lyon method. LVH should be screened for frequently in PLWD, irrespective of whether patients are hypertensive or not, and this should be done at all ages.

Keywords: Cornell criteria, diabetes mellitus, ECGs, left ventricular hypertrophy, Sokolov–Lyon

Introduction

In 2019, approximately 463 million adult patients were living with diabetes (PLWD) and this is expected to rise to approximately 700 million by 2045.[1] Macrovascular complications of diabetes mellitus (DM) relating to the heart include coronary artery disease (CAD), cardiomyopathy (including left ventricular hypertrophy [LVH]), arrhythmias, and sudden cardiac death.[2] Cardiovascular disease (CVD) is the leading cause of death in PLWD.[2] Additionally, clinical trials suggest that the prevalence of heart failure in PLWD ranges from 19–26%[3] as PLWD are approximately 74% more likely to develop heart failure and are 400% more likely to die from heart failure than those who do not have DM.[4] LVH can result in diastolic dysfunction, decreased blood flow to the coronary arteries with subsequent angina and/or myocardial infarction and increased risk of atrial fibrillation, heart failure and sudden death.[5] The electrocardiogram (ECG) is the most widely used non-invasive tool for cardiac investigation[6] and therefore plays an important part in the overall management of DM.

The Hoorn Diabetes Care System cohort, published in 2021, highlighted that 'little is known about the prevalence of ECG abnormalities in people with type 2 diabetes (DM2+)'.[7] In the United States of America, Sellers *et al*.[8] conducted a study

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among African Americans and found that approximately 60% of PLWD had ECG abnormalities, with females having fewer abnormalities than males, whereas patients with a longer duration of DM had more ECG abnormalities.

It has been found that even asymptomatic PLWD have more ECG abnormalities than those without DM.[9] Estimates from Somaratne *et al*.[10] highlighted that LVH has a high prevalence (approximately 56%) in asymptomatic PLWD. This has been attributed to DM independently increasing the risk of LVH by approximately 150%.[11] Palmieri *et al*.[12] re-enforced this statement where he found that patients with DM2+ with no other cardiovascular risk factors had a high prevalence of LVH of approximately 29.60%. In DM2+ patients it has been found that LVH has been associated with potentially modifiable risk factors including increased body mass index (BMI) and poor glycaemic control.[13] In patients living with Type 1 DM (DM1+), it has been found that women have a 200% increased risk of developing LVH as compared to men.[14] In Nigeria, Sani *et al*.[15] found that the most frequent ECG abnormalities in DM2+ patients were ST-segment depression and LVH. Most other studies described the association of LVH in cohort groups (e.g. hypertensive patients rather than PLWD) as LVH is the primary cardiac manifestation of hypertension.[16]

In South Africa, limited information is present on the associations between clinical and biochemical variables and ECGs in PLWD. A study conducted by Pillay *et al*.[17] addressed ECG abnormalities in Black South African PLWD and found that there was a high prevalence of undiagnosed myocardial infarctions in the cohort and that left ventricular hypertrophy increased the likelihood of developing infarctions. The Society of Endocrinology Metabolism South Africa (SEMDSA) 2017 diabetes guidelines currently used in South Africa recommends that ECG screening occurs at the initial visit and annually in PLWD.[18] Considering the burden of DM and the knowledge gaps of the associations between ECG abnormalities in PLWD, this study aims to address this by determining additional associations between LVH in the ECGs of PLWD by providing clinicians with more information from this non-invasive investigation, which is already being performed regularly in resource-limited settings.

Methods

A retrospective, analytical cohort study was performed using data collected from patients who attend a specialised diabetes clinic at Edendale Hospital (EDH), Pietermaritzburg, KwaZulu-Natal. Clinicians used a standardised, comprehensive clinic sheet for all patients consulted in this clinic, which has been approved by the University of KwaZulu-Natal Biomedical Research and Ethics Committee (BREC) – BCA 194/15. The data for this study included all patients who attended the diabetes clinic at EDH between 1 January 2019 and 31 December 2019. Patients from the EDH diabetes clinic had annual ECGs conducted on them unless clinical indication warranted additional ECG analysis.

Patient demographics, glycated haemoglobin (HbA1c%), random blood glucose (mmol/L), HIV status and type of DM were recorded in addition to other variables from the standard clinic datasheet. Missing or incomplete or incorrectly completed data were not considered.

ECGs were performed using the Edan SE® 1200R (Edan® instruments Inc, China) ECG machine at the EDH diabetes clinic and were conducted by nursing staff who were trained on correct electrode placement and sampling of ECGs. ECGs were conducted on patients at rest while lying supine.

Three ECG methods were used for LVH diagnosis: Sokolov–Lyon, Cornell's criteria and Romhilt–Estes criteria. Any ECG that met the diagnosis for LVH based on at least one criterion was considered positive for LVH. In the Sokolov–Lyon criteria, LVH was diagnosed when the sum of the S wave in V1 or V2 and the R wave in V5 or V6 was ≥35 mm OR when the R wave in AVL was ≥11 mm.[19] In the Cornell voltage criteria, LVH was defined as the sum of the S wave in V3 and the R wave in AVL ≥28 mm in males and ≥20 mm in females[20]OR using the Cornell product criteria where the Cornell voltage criteria multiplied by the duration of the QRS complex was ≥2440 ms.[21] In the Romhilt–Estes criteria, we diagnosed LVH when a score of ≥5 was present.[20] Three points were allocated for an amplitude ≥20 mm in limb leads OR amplitude of ≥30 mm (S wave in V1 or V2 or R wave in V5 or V6) in precordial leads OR ST-T wave changes in patients NOT on digoxin OR left atrial enlargement. Two points were allocated for left axis deviation (LAD), whereas 1 point was allocated for QRS ≥90 ms OR ST-T wave changes in patients on digoxin OR intrinsicoid deflection in V5 or V6 >50 ms.[20]

Good glycaemic control was defined as an HbA1c value <7%.[18] The Bio-Rad D-10® machine (Bio-Rad, USA) was used for analysing the HbA1c values in the laboratory. Both the laboratory and the machines are National Glycohaemoglobin Standardization Program (NGSP) accredited to maintain standardisation of HbA1c results, whereas the random glucose measurement (mmol/L) was determined using an Accu-Chek® glucometer (Roche, Switzerland).

Statistical analysis

Statistical analysis was conducted with numerical data using ANOVA, whereas categorical data relationships were determined using either the Chi-square or Fisher's exact tests. A P value <0.05 was used as an indicator of significance. Data were analysed by Statistical Package for Social Science (SPSS) version 25 for Windows (SPSS Inc., Chicago, IL, USA).

Results

(A) Epidemiology

Data from 609 patients who had ECGs done during the study period were included in the study. Out of these included PLWD, 80 (13.1%) had LVH (LVH+), whereas 529 (86.9%) had no LVH (LVH−). Approximately

one-sixth (97 [15.9%]) of these PLWD were HIV-infected. See Table 1 for further demographics.

(B) Diagnosis of LVH

Out of the 80 patients who were diagnosed with LVH, ECG evidence of LVH was found in all patients (100%) using the Sokolov–Lyon method, whereas the Cornell and Romhilt–Estes methods of diagnosed LVH in 49 (61.25%) and 8 (10%) cases, respectively [see Table 2]. In the Sokolov-Lyon method, the criteria of 'R wave ≥11 mm in AVL' diagnosed patients most frequently with LVH (100%). There was a good overlap between the methods of diagnosis. All patients (eight) diagnosed with LVH based on the Romhilt–Estes method were also diagnosed with Sokolov–Lyon and Cornell methods. In addition to this, all patients who were diagnosed with the Cornell method were also diagnosed with the Sokolov–Lyon method. The Sokolov-Lyon criteria diagnosed the most patients with LVH (80). This was 31 (63.27%) more patients than the Cornell method [see Table 2].

(C) Age

LVH + patients were significantly younger than LVH − patients (27.22 years vs. 58.98 years, $P < 0.001$) with LVH being significantly more prevalent in patients with type 1 DM compared to type 2 DM (5.68% vs. 3.29%, $P = 0.001$) [see Table 3].

(D) Blood pressure

In LVH + patients, there was a lower systolic blood pressure (SBP) than in LVH − patients (118.62 mmHg vs. 139.77 mmHg, $P < 0.001$). There were 17 patients with LVH + who were previously diagnosed with hypertension, with only 3 of these patients having SBP ≥140 mmHg at their clinic visit. Out of the 80 patients with LVH, only 9 patients had an SBP ≥140 mmHg at their clinic visit (3 diagnosed with hypertension and 6 non-hypertensive patients). There were no significant differences between diastolic blood pressure (DBP) and LVH [see Table 4].

(E) Type of Diabetes and Glycaemic Control

LVH + patients had a significantly higher mean HbA1c in DM1 + as compared to DM2+ patients (10.08% vs 8.92%, $P = 0.04$) [See Table 5]. There were significantly more patients with DM1 + than DM2+ with LVH (62 vs. 18, $P < 0.001$). In addition to this, LVH + patients were significantly younger in DM1+ compared to DM2+ patients (26.08 years vs. 44.57 years, $P < 0.001$). DM1+ comprised the majority (77.5%) of LVH + patients. Overall, there was no significance with mean HbA1c and LVH. This occurred in the entire cohort as well as in HIV-infected and HIV-uninfected cohorts. Significance was obtained when factoring in the type of DM (above) [see Table 5].

(F) Duration of DM

In LVH + patients, the mean duration of DM was 4.63 years as compared to 10.91 years in patients who did not develop LVH ($P < 0.001$).

(G) HIV

No statistically significant differences occurred between HIV status and the presence and absence of LVH.

(H) Lipids

There was a significantly better lipid profile (triglyceride and HDL-cholesterol levels) in LVH + patients as compared to LVH − patients [see Table 4].

Table 1: Demographics of patients in the study

Variable: number of patients (*n*)	LVH+ (*n*=80)	LVH− (*n*=529)	Total
Males	37	158	195
Females	43	371	414
DM1+	62	37	99
DM2+	18	492	510
HIV-infection	9	88	97
Hypertension	17	88	105
*HbA1c <7%	8	71	79
*HbA1c ≥7%	63	425	488
Positive family history of DM	45	289	334
Negative family history of DM	35	240	275

*Not all patients had HbA1c values

Table 2: Summary of methods used for diagnosis of LVH

Method used	Number of patients diagnosed with method (percentage)
Sokolov-Lyon	80 (100%)
S wave in V1 or V2 plus the R wave in V5 or V6 was ≥35 mm	41
R wave in AVL was ≥11 mm	80
Cornell criteria	49 (61.25%)
Cornell voltage criteria: S wave in V3 and the R wave in AVL ≥28 mm in males and ≥20 mm in females	49
Cornell voltage criteria multiplied by the duration of the QRS complex was ≥2440 ms	25
Romhilt-Estes	8 (10%)

Table 3: Relationship between LVH, age, and type of DM

	All patients		DM1+		DM2+		*P* (between Type 1 and Type 2)
	Count	Mean age (years) (±SD)	Count	Mean age (years) (±SD)	Count	Mean age (years) (±SD)	
LVH−	529	58.98 (11.84)	37	44.57 (8.74)	492	60.06 (11.32)	<0.001
LVH+	80	27.22 (5.64)	62	26.08 (5.68)	18	31.17 (3.29)	0.001
P		<0.001		<0.001		<0.001	

Table 4: Relationship between blood pressure/lipids and LVH

	LVH + Mean (mmol/L) (±SD)	LVH−Mean (mmol/L) (±SD)	*P*
Total cholesterol (mmol/L)	4.43 (1.18)	4.55 (1.25)	0.421
HDL (mmol/L)	1.36 (0.44)	1.25 (0.40)	0.024
LDL (mmol/L)	2.59 (1.05)	2.53 (1.11)	0.650
Triglycerides (mmol/L)	1.40 (1.79)	1.85 (1.40)	0.010
Systolic blood pressure Mean SBP (mmHg)(±SD)	118.62 (16.97)	139.77 (26.53)	<0.001
Diastolic blood pressure Mean SBP (mmHg)(±SD)	76.15 (11.72)	79.09 (14.55)	0.085

Table 5: Relationship between LVH, type of diabetes and HbA1c

	DM1+		DM2+		*P*
	Count	Mean HbA1c (%)(±SD)	Count	Mean HbA1c (%)(±SD)	
LVH-	37	9.24 (2.11)	492	9.48 (2.14)	0.510
LVH+	62	10.08 (1.94)	18	8.92 (2.49)	0.040
P		0.046		0.279	

(I) Body mass index (BMI)
Patients with LVH + had a significantly lower BMI than LVH − patients (28.14 kg/m^2 vs. 32.79 kg/m^2, $P < 0.001$).

(J) Gender
There were no statistically significant differences between gender and LVH.

(K) Family History of DM
There were no statistically significant differences between family history of diabetes and LVH.

Discussion

Electrocardiographic diagnosis of LVH varies depending on the method used and the population on which the ECGs were conducted. A Korean study conducted by Park *et al*.[22] determined that the Cornell criteria were the best at diagnosing LVH on ECG; however, a revision of the cut-off values was suggested in the conclusion of their study. A retrospective Danish study performed by Haxha *et al*.[23] also found that the Cornell criteria were better at diagnosing LVH than the Sokolov–Lyon method. Jaggy *et al*.[24] conducted a study on African patients determined that the Sokolov–Lyon criteria were the best method of diagnosing LVH with 61% sensitivity and 97% specificity. Another African study conducted in Tanzania determined that the Sokolov–Lyon criteria diagnose LVH almost twice as frequently as the Cornell criteria (12.2% vs. 5.1%, respectively).[25] Our study showed similar results to this Tanzanian study with 13.14% of patients being diagnosed with LVH according to the Sokolov–Lyon method, whereas only 8.05% were diagnosed with the Cornell method. There was a perfect overlap when Cornell diagnosed LVH with Sokolov–Lyon diagnosing it; however, Sokolov–Lyon diagnosed more patients with LVH than the Cornell method. There is varying data on which method is best for diagnosing LVH on the ECG; however, demographics appear to have an influencing factor on the preferred method of diagnosis. Our study was similar to the findings of other African studies and found that the Sokolov–Lyon method performed better than the Cornell criteria in diagnosing LVH on ECG. We postulate whether using the Sokolov–Lyon criteria should be the preferred method to diagnose patients of African descent with LVH as compared to the Cornell criteria.

Patients with LVH + in our study were significantly younger, thinner, and more often had DM1 + rather than DM2+. This contrasted with other studies, which suggested that LVH was more prevalent in older patients.[26] The 'Evaluation of Target Organ Damage in Hypertension' (ETODH) study found that there was a higher prevalence of LVH as patients aged,[27] with 29.4% prevalence in 18 to 40-year-olds, whereas 63.6% in patients 65 years or older. This is expected in a cohort of hypertensive patients; however, our study focussed on a cohort of PLWD with and without hypertension and hence did not follow the norms of an increasing prevalence of LVH with age.

Poornima *et al*.[28] mentioned that the most frequent cardiac abnormality in asymptomatic PLWD is LVH. A study conducted by Hosseini *et al*.[29] determined that LVH exists at early ages with factors such as high blood pressure, male, and being overweight conferring more risk of developing LVH. The only similarity between our study and their study was that LVH was found at an early age. Another study conducted by Spirito *et al*.[30] determined that LVH was more severe in younger than in older patients with an inverse relationship between age and the left ventricular wall in patients with hypertrophic cardiomyopathy. A possible reason may be associated with a phenotype and age where there is left ventricular remodelling with dysfunction of the myocardium, especially when present at a younger age.[31]

SBP plays an important role in the development and regression of LVH.[32] Our study found SBP was significantly lower in LVH + than LVH − patients. In our study, 87.50% (71 out of 80) of LVH + patients had their blood pressures controlled (SBP ≤140 mmHg at their clinic visit) with just over one-fifth (17/80) of patients with LVH + being diagnosed with hypertension. This suggests that the majority of patients who developed LVH did so independently of elevated blood pressures. This phenomenon has been recognised in patients without hypertension or a recognised pathology and is called 'LVH in normotensive individuals'.[33]A Trinidadian study estimated that the prevalence of LVH in normotensive individuals is around 3%.[33] A possible reason for this phenomenon was described by Struthers *et al*.[34] where it was suggested that 'LVH is particularly common in normotensive diabetes, possibly because insulin resistance itself stimulates left ventricular growth.' A Tanzanian study (very similar to our study) was conducted in PLWD, which focused on the ECG diagnosis of LVH. This study found that 16% of patients had LVH based on ECG and

determined that DM2+ conferred a more significant risk that DM1+ with SBP being an important risk factor for LVH.[25] It has also been found that in DM1+ patients, LVH + patients had significantly higher SBP (129.3 mmHg vs. 121.0 mmHg, $P = 0.001$) and higher DBP (78.3 mmHg vs. 75.4 mmHg, $P = 0.03$) compared to LVH − patients.[14] In addition to this, Rana *et al*.[35] determined that LVH has a prevalence of up to 31% in PLWD, with SBP not playing a role in LVH. Varying data exist on the association between SBP and LVH; however, our study promotes the idea of LVH occurring frequently in PLWD irrespective of SBP values. This is of importance as LVH should be screened for more frequently in PLWD, irrespective of whether they are hypertensive or not, and should be done at all ages, especially in DM1+ patients.

In our study, the type of DM was found to have an association with LVH. Patients with DM1+ had a greater prevalence of LVH as well as higher mean HbA1c values. It has been found that DM1+ patients who are normotensive have an increase in the left ventricular mass independent of blood pressures—this study was conducted in patients with established nephropathy.[36] This promotes the above idea that glycaemia may play a role in LVH. The proposed mechanisms causing diabetic cardiomyopathy are increased non-esterified fatty acids, altered insulin action, and hyperglycaemia, which are thought to trigger the cardiac phenotypes in PLWD.[28] A key difference between DM1+ and DM2+ is that DM1+ has no period of unaccompanied hyperinsulinemia, which leads to early-onset hyperglycaemia.[28] This explains the hyperglycaemia seen in DM1+ patients and the cardiac phenotypes being expressed at a younger age in terms of LVH. This also serves as an explanation as to why LVH was associated with a shorter duration of DM in our study.

Several studies suggest that dyslipidaemia is important in the origin of LVH.[37,38] Significant improvements in lipid profiles (higher HDL and lower triglycerides) were noted in LVH + patients in our study. There was some similarity to the above-mentioned Tanzanian study with triglycerides in DM1+ being higher in LVH + patients; however, there was no difference in HDL values in DM1+ patients LVH + vs. LVH − patients.[25] The mechanisms of improved lipids in LVH + in the study could be attributed to the younger age of the patient cohort.

Limitations of the study

- As this was a retrospective study, no causal relationships could be determined; rather, associations were defined.
- No echocardiogram confirmation of LVH was available due to the limited-resource setting – some diagnoses may be false positives or false negatives.
- The duration of patients being diagnosed with hypertension was not included.

Conclusion

LVH was seen more frequently in younger patients who often had DM1+, lower SBP, higher HDL, and lower triglycerides. Most cases of LVH were diagnosed using the Sokolov–Lyon method. LVH should be screened frequently in PLWD, irrespective of whether patients are hypertensive or not, and this should be done at all ages.

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Conflicts of interest

There are no conflicts of interest.

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