CLINICAL AND ECHOCARDIOGRAPHIC PROFILE OF HYPERTROPHIC CARDIOMYOPATHY AT THE UNIVERSITY COLLEGE HOSPITAL IBADAN, NIGERIA : A 20-YEAR RETROSPECTIVE STUDY

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ABSTRACT

Background: **Hypertrophic cardiomyopathy (HCM) is a primary heart muscle disease that occurs globally. It is a common cause of sudden death in young individuals, especially athletes. It is characterised by abnormal myocardial hypertrophy primarily affecting the ventricular chambers, with a particular predisposition for the interventricular septum. There is limited information on the clinical presentation of HCM in Nigeria. Therefore, we set out to determine the prevalence and clinical features of HCM in our hospital population.**

Methods: **The study was carried out at the University College Hospital, Ibadan. The echocardiographic records of patients conducted over twenty years (2003-2022) were reviewed. Standard criteria were used to make the diagnosis of HCM. Clinical features of those with HCM were also extracted. Descriptive and analytical statistics were performed.**

Results: **Out of 33,000 patients' echocardiographic reports reviewed, HCM was diagnosed in 28 patients (0.08%), with a male preponderance (M/F =1.3:1) and a mean age of 50.1±17.1 years. The common symptoms were palpitation (71.4%), chest pain (64.3%), fatigue (60.7%), and dyspnoea (60.7%). The mean interventricular septal wall and left ventricular posterior wall thickness were 1.87± 0.56cm and 1.63 ± 0.51cm respectively. Eighteen (64.3%) had systolic anterior motion, and left ventricular outflow tract obstruction was observed in 12(42.9%). The catenoid form of septal hypertrophy was more common.**

Conclusions: **Hypertrophic cardiomyopathy accounted for approximately 0.08% of echocardiograms in our facility, with peak presentation between 40–60 years. Clinical and echocardiographic features were similar to global reports, while apical hypertrophic cardiomyopathy appeared relatively uncommon.**

Keywords: Hypertrophic cardiomyopathy, Cardiomyopathy, Heart disease, Nigeria

INTRODUCTION

Hypertrophic Cardiomyopathy (HCM) is the most common genetic cardiac disorder and the prevalence in the general population is about 0.2% (1 in 500).[1] It is an autosomal dominant familial disease caused by a sarcomere protein gene mutation and phenotypically characterized by ventricular hypertrophy.[2] HCM occurs worldwide and in all races.[1,2] Studies have reported multiple causative mutations at different sarcomeric proteins, including β-myosin heavy chain, cardiac myosin binding protein, cardiac troponin T, α-tropomyosin, essential and regulatory light chains, and cardiac actin.[3,4] The expression of the disease can occur at any age, beginning in utero, but is more commonly present in early adolescence to adulthood[5]. The clinical presentations of the disorder are variable, ranging from asymptomatic to angina, palpitations, heart failure (HF), or sudden cardiac death (SCD).[1] Some HCM patients will have significant symptoms, including dyspnea, chest pain, and syncope, leading to significant morbidity and mortality, which may be as high as 3–6% for the former. The most dreaded complication of the disorder is SCD due to ventricular arrhythmias, and this has been reported in 0.1% of patients.[6]

The pattern of left ventricular hypertrophy (LVH) is variable in HCM and may be asymmetric hypertrophy

of the basal anterior septum, reverse curve (catenoid), sigmoid septum, apical HCM among others. The most common pattern of LVH is asymmetric septal hypertrophy involving the basal anterior septum, as well as the anterior free wall, and this is associated with the observed differences in morbidity and mortality.[7] For example, the apical form of the disease in the Japanese and North American populations is associated with a benign outcome.[8,7] It has been reported that there is an association of septal morphology and sarcomeric mutation that may assist in risk assessment and management of patients with HCM.[9,10] In addition, resting LV outflow tract obstruction has been shown by some studies to be a strong predictor of subsequent HF and death.

The clinical diagnosis of HCM is primarily based on echocardiography. The conventional diagnostic criterion is the presence of a maximal left ventricular (LV) end-diastolic wall thickness of ≥15 mm in one or more myocardial segments, in the absence of abnormal loading conditions capable of fully accounting for the degree of hypertrophy, such as severe systemic hypertension or valvular aortic stenosis. Most of the published data on HCM have come from high-income countries of North America and Western Europe. In Africa, most of the descriptions of the disease have emanated from South Africa and some cases from North Africa.[11,12,13,14] There is only one clinical study of the disorder in Nigeria[15]; others were case reports.[16-19] The aim of this study was therefore to describe the clinical and echocardiographic profile of HCM at the University College Hospital, Ibadan, Nigeria.

MATERIAL AND METHODS

Study location

The study was done in the Cardiology Unit, Department of Medicine, University College Hospital, Ibadan, Nigeria.

Study design

The study was a retrospective cross-sectional study.

Study population

Data were extracted from our institution's echocardiography database performed on adults aged eighteen years and above between January 1, 2003 and August 31, 2022.

Although images were not stored at the beginning of the registry (as the facility was not available then), all the echocardiographic dimensions, such as septal and posterior wall thickness, were meticulously stored in the database. We extracted all cases with septal or posterior wall thickness ≥12mm and reviewed them apart from the final diagnoses reported by the sonographer. In addition, we reviewed the case files of the selected cases. We excluded individuals with secondary causes of LVH, such as aortic stenosis, hypertensive heart disease, chronic kidney disease, and infiltrative cardiomyopathies, such as cardiac amyloidosis. We also excluded studies carried out on children and repeated procedures.

Criteria for diagnosing HCM

HCM was diagnosed based on echocardiographic findings of left ventricular septal wall dimension ≥ 1.5cm or between 1.2-1.5 cm if there is a family history of HCM.[20]

One major confounder in our setting is the high burden of long-standing, untreated severe hypertension with remarkable LVH, especially in younger patients (with associated renal dysfunction) and in the elderly. In these cases, we reviewed the clinical documentation in the case notes as well as the presence or absence of other features of HCM, such as systolic anterior motion (SAM) and intracavitary gradient.

The study was designed in keeping with the principles of the Helsinki Declaration on research on human subjects and was approved by the Joint University of Ibadan/University College Hospital Ethics Review Committee as part of the Ibadan Heart Disease Registry.

Clinical evaluation

We used a uniform case report form to obtain clinical details from their case notes. These details included sociodemographic information, biophysical variables, and 12-lead electrocardiogram data. Data were also obtained were age, sex, family history of HCM or sudden death, and New York Heart Association (NYHA) functional class. Furthermore, we collected information on symptoms such as fatigue, dyspnea, palpitations, chest pain/angina, pre-syncope/syncope, history of use of alcohol, cigarette smoking and history of cardiometabolic conditions (such as hypertension, diabetes mellitus, coronary artery disease, and dyslipidemia).

12-lead electrocardiography

A resting 12-lead ECG was performed on the subjects using a commercially available machine at a paper speed of 25 mm/s and standardized at 0.1mv/mm. ECG tracings were reported by two of the authors (OSO and OAO). Electrocardiographic LVH by voltage was based on Sokolow-Lyon (SV1RV5 or V6)[21], Cornell (SV3RaVL with 0.6mV added in women)[22]. ECG LVH with strain was adjudged present when there is a down-sloping convex ST segment depression (≥0.1mV) with

an inverted asymmetrical T-wave opposite to the QRS axis.[23,24] Voltages in individual leads were calculated as the mean of three complexes wherever possible. Other 12-lead ECG abnormalities were based on standard criteria.[25]

Transthoracic echocardiography

Echocardiography was carried out using the available Echocardiography machine. Two-dimensional, M-mode echocardiography and Doppler studies were performed according to the American Society of Echocardiography (ASE) recommendations in the left lateral decubitus position.[26] Measurement of left ventricular (LV) dimension was made from two-dimensional views. Parameters measured included LV internal diameter, interventricular septum, and LV posterior wall thickness measured in end-diastole and end-systole in the parasternal long-axis view. The LV end-diastolic and end-systolic dimensions were measured at the level of the mitral leaflet tips according to the ASE leading edge to leading edge convention.[26] Left ventricular (LV) fractional shortening was calculated from LV internal dimensions in diastole and systole.[23] The left ventricular ejection was assessed by modified Simpson's method.[26]

The LA diameter was measured by M-mode in the long axis of the parasternal view as the maximal distance between the posterior aortic root wall and the posterior left atrial wall at the end-systole.[26]

Doppler/Colour echocardiography was used to assess for the presence of valvular regurgitation. Left ventricular diastolic function was assessed through the Doppler filling dynamics of the left ventricle by evaluating the transmitral 'E' wave velocity (peak early mitral inflow velocity) and the 'A' wave velocity (peak atrial inflow velocity), the E/A ratio, and the deceleration time (DT): time interval of peak E wave velocity to its extrapolation to the baseline. The E/A ratio was not evaluated in individuals with atrial fibrillation or marked tachycardia.

Data management and statistical analysis

All data were entered and analysed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize the data. Continuous variables were expressed as means ± SD, while categorical variables were summarised as numbers and percentages. Pearson's chi-squared or Fisher's exact test was used to compare the frequency of categorical variables between males and females, and the Student t-test for continuous variables. All *p*-values were two-sided, and $p \leq 0.05$ was considered statistically significant.

RESULTS

Twenty-eight subjects fulfilled the criteria for the diagnosis of HCM following the full review of 33,000 echocardiograms, giving a prevalence of 0.08% or approx. 1/1000 echocardiograms. The indications for echocardiography were suspected HCM in 28.6%, hypertensive heart disease in 25%, heart failure in 17.6%, and easy fatiguability and stroke in 7.1% each. (Table 1).

Table 1:. The indication for echocardiography for the 28 HCM patients

S/N	Indication	Number (%)
1	Suspected Hypertrophic Cardiomyopathy	8 (28.6)
2	Hypertensive heart disease	7 (25.0)
3	Heart failure	5 (17.9)
4	Easy fatiguability	2 (7.1)
5	Stroke/Transient ischaemic attack	2 (7.1)
6	Electrocardiographic left ventricular hypertrophy	1 (3.6)
7	Abnormal 12 lead Electrocardiography	1(3.6)
8	Chest pain	1 (3.6)
9	Routine Echocardiography	1 (3.6)
	Total	**28 (100)**

There were 16 (57.1%) males and 12 (42.9%) females. (Table 2) The mean age of the patients was 50.1±17.1 years (range-21-83 years). The mean age for the males was 50.4 ±18.7 years (range 21-83 years) while that for females it was 49.7±15.3 years (range, 26-75 years respectively. (Tables 2 and 3).

Table 2: Clinical profile of- and co-morbidities in the 28 HCM patients

Parameter	n/%
Males	16 (57.1)
Females	12(42.90
Family History of HCM(n/%)	3(10.7)
Family history of SCD(n/%)	3(10.7)
NYHA functional class I and II(n/%)	23(82.1)
NYHA functional class III and IV(n/%)	5(17.9)
Symptoms	
Fatigue(n/%)	17(60.7)
Dyspnoea(n/%)	17(60.7)
Palpitations(n/%)	20(71.4)
Angina(n/%)	18(64.3)
Presyncope/syncope(n/%)	4(14.3)
Pedal oedema(n/%)	4 (14.3)
Ejection systolic murmur(n/%)	10 (35.7)
CV risk factors and co-morbidities	
Smoking(n/%)	2(7.1)
Alcohol consumption (n/%)	8(28.6)
Hypertension(n/%)	4(14.3)
Diabetes mellitus(n/%)	1(3.6)
Dyslipidaemia (n/%)	2(7.1)

Table 3: The biophysical profile of the 28 HCM patients

Variable	All (n=28)	Male (n=16)	Female (n=12)	p-value
Mean age (year)	50.1 ± 17.1	50.4±18.7	49.7±15.3	0.927
Median age	60.5	62	52	-
Age range	26-80	26-76	29-80	-
Body weight (kg)	71.5 ± 12.7	73.1 ± 12.4	71.2 ± 12.3	0.689
Height (cm)	165.3 ± 9.7	169.6 ± 9.9	158.3 ± 5.9	0.002
BMI (kg/m^2)	26.4±5.0	25.2±5.1	28.2±4.5	0.122
Heart rate (beats/min)	61.7 ± 13.2	78.3 ± 12.9	74.6 ± 11.5	0.520
SBP(mmHg)	135.5 ± 35.4	133.2 ± 33.2	136.3 ± 26.3	0.478
DBP(mmHg)	82.8 ± 23.2	81.9 ± 23.3	83.2 ± 16.0	0.419

The peak age at presentation was between 40-60 years at presentation.

Clinical characteristics

Three (10.7%) had a family history of sudden death/ HCM. The majority, 23(82.1%), were in NYHA classes I and II. There was no difference in the body mass index, heart rate, systolic and diastolic blood pressure. (Tables 2 and 3).

12-lead ECG findings

The following 12-lead ECG abnormalities were noted in patients with echocardiographic findings of HCM: left ventricular hypertrophy – 22(78.6%), QRS complex abnormalities- 5(17.9%), Wolf Parkinson White syndrome -3(10.7%), bundle branch block-3(10.7%), ventricular tachycardia-3(10.7%), atrial fibrillation-1(3.6%), and atrioventricular block-1(3.6%).

Echocardiographic findings

Table 4 depicts the echocardiographic parameters. The mean septal wall thickness was 1.87±0.56cm (1.86±0.51cm for males and 1.91±0.60cm for females). The mean posterior wall thickness was 1.63±0.51(1.66±0.48cm in males and 1.54±0.55cm in females). Other echocardiography parameters were also not different between males and females. Systolic arterial motion of the anterior mitral valve leaflet (SAM) was documented in 18(64.3%), while LVOT obstruction was observed in 12(42.9%).

Pattern of LVH

We could only determine the pattern of septal hypertrophy in half of the subjects. The catenoid form appears to be common (42.8%) followed by the neutral type (28.6%) and the sigmoid type (21.4%). Apical HCM was only seen in one subject (7.1%).

Procedures

Five of the HCM patients had ICD implanted, while one with atrial fibrillation had A-V node ablation as well as implantation of a pacemaker and ICD.

Table 4: The echocardiographic profile of the 28 HCM patients

Variable	All (n=28)	Male (n=16)	Female (n=12)	P-value
IVSD(cm)	1.87± 0.56	1.86 ± 0.51	1.91± 0.60	0.841
LVPWTD(cm)	1.63 ± 0.51	1.66 ± 0.48	1.54 ± 0.55	0.530
LVIDd(cm)	4.21 ± 0.90	4.15 ± 0.85	4.51 ± 0.90	0.285
LVIDs(cm)	2.66 ± 0.95	2.57 ± 0.85	2.93 ± 1.04	0.331
FS(cm)	40.43 ± 14.76	37.73 ± 17.14	43.13 ± 10.67	0.346
EF(cm)	70.28 ± 19.70	66.39 ± 23.80	75.03 ± 11.1	0.256
AO (cm)	2.97 ± 0.37	3.09 ± 0.40	2.82 ± 0.27	0.055
AVO (cm)	1.86 ± 0.54	1.96 ± 0.41	1.79 ± 0.64	0.357
LA (cm)	4.17 ± 0.95	4.09 ± 0.96	4.20 ± 0.95	0.769
ME	0.69 ± 0.23	0.59 ± 0.24	0.71 ± 0.25	0.455
MA	0.60 ± 0.25	0.60 ± 0.35	0.67 ± 0.17	0.568
DT	179.0 ± 68.7	160.8 ± 90.7	185.0 ± 51.66	0.527
IVRT	93.0 ± 76.5	75.15 ± 84.4	113.42 ± 22.58	0.234

IVSD= Interventricular septal wall thickness in diastole, LVPWTd= Left ventricular posterior wall thickness in diastole, LVIDd= Left ventricular internal dimension in diastole, LVIDs=Left ventricular internal dimension in systole, FS= fractional shortening, EF=, Ejection fraction, AO= Aortic root dimension, LA= Left atrial dimension, ME= Transmitral early filling velocity, MA= Transmitral late filling velocity, DT=Deceleration time of the E wave, IVRT= Isovolumic relaxation time

Table 5: The comparison of the present study with some previous studies in different parts of the world

Variable/Study	Current study	Ntusi et al[12]	Mbakwem et al[15]	Maron et al[4]	Lee et al[30]	Ahmed et al[28]	Sheikh et al[29]
Number of subjects	28	43	14	277	163	69	425
Mean age	50.1 ± 17.1	38.5±14.3	43.14±15.0	47±22	60.9±12.1	42±16	51.5±15.9
Males (n/%)	16 (57)	25(58)	11(78.6)	152(55)	84(52)	43(71)	272(64)
Africans/Blacks (%)	100	30	100	none	None	none	38
Family History of HCM(n/%)	3(10.7)	33(77)	NA	21(8)	NA	2(5)	107(25)
Family history of SCD(n/%)	3(10.7)	5(12)	NA	NA	NA	4(9)	77(18)
NYHA functional class I and II(n/%)	23(82.1)	33(77)	13(92.9)	NA	NA	NA	NA
NYHA functional class III and IV(n/%)	5(17.9)	10(23)	1(7.1)	NA	NA	NA	NA
Symptoms							
Fatigue(n/%)	17(60.7)	25(58)	NA	174(63)	NA	NA	262(61)
Dyspnoea(n/%)	17(60.7)	24(56)	6(42.9)	NA	121(74)	31(65)	198(47)
Palpitations(n/%)	20(71.4)	34(79)	7(50)	NA	28(17)	5(7)	160(38)
Angina(n/%)	18(64.3)	28(65)	7(50)	NA	111(68)	NA	94(46)
Presyncope/syncope(n/%)	4(14.3)	12(28)	4(28.3)	NA	20 (12)	2(4)	146(34)
Smoking(n/%)	2(7.1)	19(31)	NA	NA	NA	NA	NA
Hypertension(n/%)	4(14.3)	12(28)	NA	NA	28 (17)	NA	178(42)
Diabetes mellitus(n/%)	1(3.6)	NA	NA	NA	29 (18)	NA	NA
Alcohol consumption (n/%)	8(28.6)	9(21)	NA	NA	NA	NA	NA
Dyslipidemia(n/%)	2(7.1)	6(14)	NA	NA	NA	NA	NA
Coronary artery disease(n/%)	0 (0.0)	3(7)	NA	NA	29 (18)	NA	NA
COPD(n/%)	0(0.0)	2(5)	NA	NA	NA	NA	NA
Medical Examination							
Heart rate (beats/min)	61.7 ± 33.2	71.3±12.7		NA	NA	NA	67.6±13.7
Systolic blood pressure(mmHg)	135.5±35.4	125.8±19.2	120.45±11.28	NA	NA	NA	127±16.4
Diastolic blood pressure(mm/Hg)	82.8 ± 23.2	75.8±11.3	78.18±8.74	NA	NA	NA	75.2±11.1
Pedal oedema(n/%)	4 (14.3)	5(11.6)	NA	NA	NA	NA	NA
Ejection systolic murmur(n/%)	10 (35.7)	18(41.9)	7(50)	NA	NA	NA	NA
Electrocardiographic findings							
Sinus rhythm (n/%)	22 (78.6)	39 (90.7)	11(78.6)	NA	NA	NA	NA
Atrial fibrillation (n/%)	6 (21.4)	4 (9.3)	2(14.3)	NA	34 (21)	NA	NA
QRS abnormalities (n/%)	5(17.9)	12 (28)	1(7.1)	NA	NA	NA	NA
Voltage criteria for LVH(n/%)	22 (78.6)	22 (51)	9(64.3)	NA	137 (84)	60 (87)	183(43)
Presence of pathological Q waves(n/%)	10 (35.7)	12 (28)	1(7.1)	NA	NA	NA	78(18)
T-wave inversion	22 (78.6)	34 (79)	6(42.9)	NA	108 (66)	NA	NA
Left atrial hypertrophy	14 (50)	10 (23)	2(14.3)	NA	NA	NA	NA
LBBB	4 (14.3)	4 (9)	NA	NA	NA	NA	18(4.2%)
RBBB	2(7.2)	2 (5)	NA	NA	NA	NA	NA
PR prolongation	4(14.3)	2 (5)	NA	NA	NA	NA	NA
Echocardiographic findings							
LVEDD (cm)	4.21 ± 0.90	4.1±0.8	4.17± 5.70	NA	4.5±0.5	NA	4.52±0.7
LVESD (cm)	2.66 ± 0.95	2.7±0.6	NA	NA	2.4±0.4	NA	NA
IVSD (cm)	1.87± 0.56	1.9±0.7	23.3±7.86	2.2	1.9±0.4	-2.1 ± 0.7	NA
LVPWTd(cm)	1.37±0.79	1.2±0.4	13.66± 7.86	NA	1.1±0.3	1.3±0.4	NA
LVEF (%)	70.28 ± 19.70	71.5±8.3	NA	NA	NA	68±13	64.02±10.27
Left atrial dimension (cm)	4.17 ± 0.95	3.5±0.8	NA	NA	3.8±0.7	NA	NA
SAM	18(64.3)	9 (21)	2(14.3)	NA	80 (49)	39 (57)	50(23)
LVOT obstruction	12(42.9)	12 (28)	2(14.3)	NA	78 (48)	28 (41)	NA
E/A ratio		1.2±0.4	NA	NA	NA	1.5 (0.9-2.1)	1.2±0.59
Pattern of hypertrophy(n=14)							
Sigmoid	3(21.4)	13 (30)		75 (27)	NA	8 (12)	NA
Catenoid	6(42.8)	23 (53)		92 (33)	NA	29 (42)	NA
Neutral	4(28.6)	6 (14)		65 (23)	NA	25 (36)	NA
Apical	1(7.1)	1 (2)		5 (2)	NA	7 (10)	19(4.5)

DISCUSSION

Hypertrophic cardiomyopathy is associated with substantial morbidity and mortality, with an annual mortality rate of up to 6%.[27] Clinical information on the disease is sparse in Nigeria. In this descriptive study of 28 HCM patients following a review of 33,000 echocardiograms (approx. 8/1000 echo) conducted over twenty years, we report that HCM in Ibadan, Nigeria, is more common in males (M: F ratio =1.3:1). Male preponderance has been reported from many series in most parts of the world, ranging from 52% to 79%.

The majority presented between 40 and 60 years, with typical symptoms and signs, and various 12-lead ECG and echocardiographic features. The mean age at

presentation was 50 years. This is higher than the mean age of 39, 43, 47, and 42 years by other studies in South Africa[12], Saudi Arabia[28], Lagos (Nigeria)[15,] and the United States of America[4] respectively. It is, however, lower than the mean age of 52 and 61 years reported from the United Kingdom[29] and Taiwan[30], respectively. Family history of the disease is often difficult to obtain in developing countries, where health records and health-seeking behaviours are poor. In addition, autopsies are not often carried out in sudden deaths. We were able to obtain family history in only three cases (10.7%). This is lower than 77% in South Africa[12], 25% in the United Kingdom[29,] but higher than 8% in the USA[4] and 5% in Saudi Arabia.[28]

The majority of the cases were in NYHA class I and II, which is similar to what was obtained elsewhere (77-93%)[12,15]. Typical symptoms such as fatigue, dyspnoea, palpitation, angina, and presyncope obtained in this study appear to be similar to previous reports.[12,15,31] Heart murmurs in HCM may arise from LVOT obstruction or mitral regurgitation.[12,15,31] Mitral valve regurgitation has been documented in about 50% of cases.[31] In our cohort, murmur was documented in 10(35.7%). It was present in 41.9% of the South African cohort[12] and in 50% in the report by Mbakwem from Lagos, Nigeria.[15]

In terms of cardiovascular risk factors, the cigarette smoking rate in our cohort was lower than in South Africa (7.1% vs 31%)[12]. Hypertension was documented in 14% of our cohort compared to 28% in South Africa[12], although theirs were younger. A rate of 17% has been reported from Saudi Arabia.[28] The mean systolic and diastolic blood pressures in our cohort appear to be higher than most other series. This may also be related to the population mean blood pressures in these populations.[32] The population mean blood pressure has been reported to be higher in West Africa.[32] Hypertension has been reported as the most common comorbidity in HCM and may affect its phenotypic expression, especially left ventricular hypertrophy.[28] It is often a clinical dilemma to differentiate hypertensive heart disease from HCM[28], only one case in our cohort had a history of diabetes mellitus (3.6%); a higher rate of 18% was reported from Saudi Arabia[28], This may be explained by the high rate of diabetes mellitus in the Saudi population compared to Nigeria.[33]

Left atrial abnormalities and left ventricular hypertrophy are common 12-lead ECG abnormalities in HCM. Others include heart blocks, deep T-wave abnormalities, and ventricular tachycardia. Although none of these is characteristic of HCM. The presence of a giant negative T-wave has been reported as the most frequent abnormality.[34] This was observed in 79% of our cohort, and this is similar to the findings by Ntusi *et al.*[12] It was documented in 43% of the cohort in Lagos[15] and 66% in Saudi Arabia.[28] A normal 12-lead ECG has been observed in about 27% by Savage *et al.*[35] and this is associated with a less severe phenotype.[36]

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Typical echocardiographic abnormalities have been documented in HCM. These include asymmetric septal hypertrophy, SAM, and evidence of LVOT obstruction, amongst others.[37] The mean septal wall thickness of 1.87cm was similar to the finding by Ntusi *et al.*[12] and Lee *et al.*[30] but lower than the value reported by Mbakwem *et al.*[15], Maron *et al.*[4] and Ahmed *et al.*[28] A larger left atrial dimension was observed in our study compared to those of Ntusi *et al.*[12] and Lee *et al.*[30] This may be related to the higher mean blood pressure in our cohort. We also reported higher rates of SAM, but comparable rates of LVOT obstruction to those of Ahmed *et al.*[28] and Lee *et al.*[30] Table 5 shows the comparison of the present study with some previous studies in different parts of the world.

Strengths and limitations

This is the largest series of HCM reported from Nigeria from a large echocardiography database. The previous and only study from Nigeria reported on 14 cases. Other reports have been case reports.

Some of the limitations include the fact that this is a retrospective study, which may affect the reliability of the results. Since it is a hospital-based study, it may not reflect the true situation in the community. In addition, early or milder forms of the disease may have been missed. Because different operators collected the data over many years, the quality of the diagnosis may have been affected by this. However, the reliability of the echocardiography report in our laboratory is good and has been reported elsewhere[38] Finally, the burden of hypertension is high in our community; some cases of HCM may have been diagnosed as hypertensive heart disease. The issue of imaging quality variation/ diagnostic guideline changes over time may also be a limitation of the study.

Future directions

A multicentre prospective study is needed to define the prevalence and clinical characteristics of HCM in Nigeria. Information is also needed on the prognosis, management, and care of the disease in the country. There is no information on the genetics of the disease in the country; this needs to be explored.

CONCLUSION

We have provided basic clinical information on HCM in our facility. The prevalence of HCM in our echocardiography laboratory is 0.08% or approx. 1/1000 echocardiograms. The peak age of presentation is between 40-60 years. The clinical profile, 12-lead ECG abnormalities, and echocardiography patterns appear to be similar to reports from many parts of the world. Apical HCM is relatively uncommon.

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