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Research Article

Patterns and Predictors of Abnormal Left Ventricular Global Longitudinal Strain Among Black Africans with Hypertension at the Campus University Hospital of Lome: A Cross-sectional Study

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Abstract

Background: Hypertension is responsible for myocardial structural changes. The objective of this study was to evaluate longitudinal left ventricular function via 2D strain in hypertensive patients in Lome. *Methods*: A descriptive hospital-based cross-sectional study was conducted from March 1 to August 31, 2024. Patients with hypertension for a period exceeding one year and with a left ventricular ejection fraction greater than 50% as determined by the Simpson biplane method were included in the study. The longitudinal systolic function of the left ventricle was evaluated by means of global longitudinal strain (GLS). *Results:* The study population comprised 215 patients. The mean age of the subjects was 46.4 ± 12.3 years. GLS ranged from -28.75 to -5.14 (mean -17.37 ± -3.97). Low GLS values (>-17) were identified in 41.4% of patients. The factors associated with low GLS were as follows: hypertension that had lasted for more than 10 years (odds ratio (OR) = 14.28, 95% confidence interval (CI) 5.55-50.00; p < 0.001), uncontrolled hypertension (OR= CI, p), dyslipidaemia (OR = 12.15, CI 5.32-27.77; p < 0.001), diabetes (OR = 2.65, CI 1.12-6.27; p = 0.026), obesity (OR = 2.53, CI 1.11-5.77; p = 0.027), concentric remodelling of the left ventricle (OR = 4.93, CI 1.82-13.35; p = 0.002), and concentric LVH (OR = 3.89, CI 1.55-9.80; p = 0.004). *Conclusion*: The prevalence of longitudinal systolic dysfunction is high. The factors associated with HTN leading to this decrease include dyslipidaemia, diabetes, duration of HTN, remodelling and LV hypertrophy. This study serve to emphasise the importance of early detection and management of hypertension and its associated risk factors.

Keywords

Hypertension, Longitudinal Dysfunction, 2D Strain, Togo

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1. Background

Hypertension (HTN) has been identified as a significant cardiovascular risk factor. It is a prevalent pathology on a global scale, affecting both developed and developing countries [1, 2]. In sub-Saharan Africa, the prevalence of HTN has been documented as 46% [3]. In Togo, studies have demonstrated a prevalence of 36% in the general population and 71% in cardiology outpatient departments [4, 5].

HTN has been shown to induce structural changes in the myocardium, resulting in left ventricular hypertrophy (LVH) and progressive impairment of left ventricular (LV) systolic and diastolic function. It has been identified as the primary cause of heart failure in this region [6]. HTN is a condition which frequently results in heart failure being one of the most common complications. As demonstrated in the extant literature, impaired LV function, LVH, and myocardial fibrosis are recognised markers of target organ damage, compromised in patients with long-standing HTN [7-9]. Conventional echocardiography is capable of detecting changes in left ventricular (LV) diastolic dysfunction associated with LVH; however, global LV systolic function often remains preserved until late in the course of the disease, which makes subtle changes in LV contractile function difficult to interpret in the early stages [10, 11]. Recently, two-dimensional strain (2D strain) has emerged as a non-invasive and sensitive method for detecting early regional and global myocardial dysfunction. This method can detect such dysfunction in both symptomatic and asymptomatic patients with cardiovascular disease, and is thus a valuable addition to the conventional parameters with which to detect such dysfunction [12]. This technique involves the analysis of myocardial motion by tracking natural acoustic reflections and interference patterns seen in two-dimensional echocardiographic images. Several studies have concentrated on the early detection of myocardial systolic dysfunction in hypertensive patients [13, 14]. However, this approach has not been widely adopted or adequately integrated into routine clinical practice in our countries, and to the best of our knowledge, there is no study in Togo using Speckle tracking echocardiography.

The objective of this study was to ascertain the prevalence of early myocardial dysfunction in black Togolese hypertensive patients, as well as to identify the predictive factors of abnormal left ventricular global longitudinal strain.

2. Materials and Methods

2.1. Study Design and Population

A descriptive cross-sectional study was conducted from March 1 to August 31, 2024, in the outpatient cardiology departments of the Campus University Hospital in Lome, the capital of Togo. Campus University Hospital is a public hospital in Togo that is regarded as being of significant importance, being the third reference hospital in the country. The patients treated in this hospital were predominantly referred by general practitioners or self-referred. We consecutively included any patient with HTN lasting ≥1 year and having an LV ejection fraction (LVEF) ≥ 50% on the Simpson biplane. HTN was defined as a blood pressure ≥ 140/90 mmHg at two separate visits or a patient under treatment for HTN. The exclusion criteria comprised the following: patients with suspected active coronary artery disease as indicated by an electrocardiogram; significant valvular disease; ventricular preexcitation; atrial fibrillation; hypertrophic cardiomyopathy; pregnant status; symptoms; and biological and echocardiographic signs of congestive heart failure. Furthermore, patients exhibiting poor echogenicity and those who declined to participate were excluded from the study.

The minimum sample size was determined using the Cochrane formula as follows:

$$n = (z2 \times p \times (1-p)) / e^2$$

With: p = prevalence of HTN (without heart failure, cardiac arrythmia, and coronary artery disease) in outpatients' clinic of cardiology department of Campus Teaching Hospital, we used the prevalence of 53% reported by our previous study [5].

e = degree of accuracy (5%) z = standard normal variation. With a confidence interval to be 95% and by accepting an error of 5%, level of significance (α) 0.05, it corresponds to 1.96.

n = minimum sample size = 202.

2.2. Clinical Assessment

A comprehensive evaluation of clinical parameters was conducted, encompassing sociodemographic variables such as age and sex, the history of HTN including its duration and control status, comorbidities including diabetes, dyslipidaemia and obesity, and lifestyle factors such as smoking and sedentary behaviour.

For the duration of HTN, we divided the patients into 3 groups: < 5 years, 5-10 years and > 10 years. HTN was considered controlled if the blood pressure measurement in the last follow-up was < 140/90 mmHg in the office (clinical check). The term 'obesity' was defined as a body mass index (BMI) greater than 30 kg/m², with the term 'overweight' denoting a BMI between 25 kg/m² and 30 kg/m². Current smoking was defined as having smoked at least one cigarette in the 3 weeks prior to enrolment. Diabetes is characterised by a fasting blood glucose level that exceeds 126 mg/dL or the utilisation of antidiabetic medications. Dyslipidaemia is characterised by a total cholesterol concentration that exceeds 2 g/L, and/or LDL cholesterol levels that surpass 1.5 g/L, and/or HDL cholesterol concentrations that fall below 0.4 g/L for women and 0.5 g/L for men, and/or triglyceride levels that

exceed 1.5 g/L. The term "sedentary lifestyle" is defined as the absence of daily physical activity or activity lasting less than 150 minutes per week.

2.3. Echocardiographic Assessment

A comprehensive transthoracic echocardiography procedure was performed on all participants by sonographers who had attained the requisite level of qualification. The procedure was carried out using the Esaote MyLabX7 machine with a 3.5-5.0 MHz transducer probe. Standard motion-mode (M-mode), two-dimensional (2-D), pulse-wave (PW), continuous wave (CW), and colour Doppler echocardiography were conducted with the patient in the left lateral decubitus position. Echocardiographic measurements were performed in accordance with the recommendations of the American Society of Echocardiography (ASE) [15].

In the parasternal long-axis view, the following parameters were measured by M-mode echocardiography: LV end-diastolic diameter (LVEDd), interventricular septum thickness (IVS), posterior wall thickness (PWT), and LV end-systolic diameter (LVESd). The LV mass (LVM) was calculated by using the Devereux formula [16]: The LVM is equivalent to $0.8 \times \{1.04 \times [(LVEDd + IVS + PWT)^3 - LVEDd^3]\}$. An increase of 0.6 g was observed. The relative wall thickness (RWT) was calculated using the following formula: $RWT = 2 \times (PWT/LVEDd)$. The geometry of the left ventricle was determined by measuring the relative thickness of the left ventricular wall and the left ventricular mass index (LVMi). LV hypertrophy (LVH) was defined as LVMi > 110 g/m2 in men and > 95 g/m2 in women. The LVH is deemed to be concentric if the h/r is greater than 0.42, and eccentric if the h/r is less than 0.42.

The left ventricular ejection fraction was calculated using both the Teicholz formula and the Simpson biplane disk summation method. It was determined that good 2-D apical two-chamber (A2CH) and four-chamber (A4CH) views of the wall were obtained, and care was taken to avoid apical foreshortening. The manual tracing of the endocardial border, with a view to avoiding the papillary muscles, was performed in both end-diastole and end-systole, from the lateral to septal borders of the annulus on the 4-chamber view and the anterior and inferior annular borders on the 2-chamber view. The onset of the QRS complex was defined as end-diastole, and the termination of the T-wave as end-systole [15].

The study also incorporated various other measurements, including left atrial diameter and left atrial volume index, as well as tricuspid annular plane systolic excursion (TAPSE). Pulsed wave imaging was utilised for the purpose of evaluating the LV mitral filling pattern and diastolic function. The LV diastolic function was assessed in accordance with the algorithm recommended by ASE [17].

The left ventricular global longitudinal strain (LV GLS) of the ventricles was assessed by two-dimensional speckle tracking echocardiography (2D-STE) with the automated function imaging software of the Esaote MyLabX7, in accordance with the ASE guidelines for speckle tracking echocardiography. It is evident that the capture of all images was conducted at a frame rate that ranged from 70 to 100 cycles per second. The speckles were tracked on the standard gray-scale 2D images. Myocardial strain was then calculated by measuring the change in the position of the speckles within the myocardial segment or region of interest during the cardiac cycle. The GLS of the LV was estimated using the apical two-, three-, and four-chamber views of the LV. The endocardial and epicardial borders were traced using automated function imaging. Three apical views were utilised to derive myocardial GLS values, with the 17-segment model serving as the underlying framework. A meticulous examination was conducted to ascertain the clarity of the cardiac borders throughout the cardiac cycle. The region of interest (ROI) was subjected to visual assessment and manual adjustment in cases where the tracking was deemed inappropriate. A peak systolic longitudinal strain of less than -17% was considered to be within the normal range.

2.4. Data Analysis

The data thus generated was entered into a Microsoft Excel spreadsheet, whereupon it was checked for completeness and cleaned. The analysis of the data was conducted utilising IBM Statistical Product and Service Solutions (SPSS) statistic software version 26.0 (IBM Corp., Armonk, NY, USA). The expression of continuous variables is in the form of mean ± standard deviation, whilst that of categorical data is in the form of frequency (percentage). The association between abnormal LV GLS and aetiology and risk factors was presented as cross-tabulation using the Chi-square (χ^2) test, Fisher exact Chi-square as appropriate. It is considered significant at the 0.05 level of probability that an association is established. A multiple linear regression model was employed in order to assess the presence of independent predictors of abnormal LV GLS.

2.5. Ethical Considerations

Ethical approval for the study was obtained from the Faculty of Health Sciences of the University of Lom é Health Ethics Review Committee, a body that is recognised as having the necessary expertise in this field. Written informed consent was obtained from each participant after the purpose of the study was clearly explained to them. The study was conducted in accordance with the International Conference on Harmonization for Good Clinical Practice (ICH-GCP) guidelines and the Declaration of Helsinki.

Table 1. *General characteristics of the patients.*

Characteristics	values
Mean age	46.7 ± 12.3 years
Sex ratio (M/F)	0.55
Body mass index	
- Mean	25.6 ± 3.8 kg/m^2
- Normal	40.9%
- Overweight	26.5%
- Obesity	32.6%
Diabetes	27.6%
Dyslipidemia	35.8%
Tobacco	3.7%
Mean hypertension duration	7.22 ± 5.88 years
Controlled hypertension	
Yes	65.1%
No	34.9%
Echocardiography	
Normal	34.4%
Remodelling	24.7%
Concentric left ventricular hypertrophy	27.9%
Eccentric left ventricular hypertrophy	13.0%
Abnormality of left ventricular relaxation	65.0%
Abnormality of left ventricular compliance	3.0%

3. Results

The present study comprised a total of 215 patients. The mean age of the patients was 46.7 ± 12.3 years, with extremes of 21 and 72 years. We recorded 139 women (64.7%) and 76 (35.3%) men, i.e., an M/F sex ratio of 0.55. As illustrated in Figure 1, the distribution of patients is depicted according to both sex and age group. The largest age group represented was 40-49 years (32.1%). The mean duration of HTN was 7.22 ± 5.87 years, with extremes ranging from 1 to 35 years. Furthermore, the analysis revealed that approximately half of the population exhibited HTN between the ages of one and five years. The presence of additional cardiovascular risk factors was identified, including dyslipidaemia (35.8%), obesity (32.6%), and diabetes (27.6%). The results of the echocardiography study indicated that 27.9% of patients exhibited concentric LVH, while 36.3% demonstrated normal LV geometry. A total of 139 patients were diagnosed with LV relaxation disorders, while seven patients were found to have diastolic dysfunctions. The patient characteristics are delineated in Table 1.

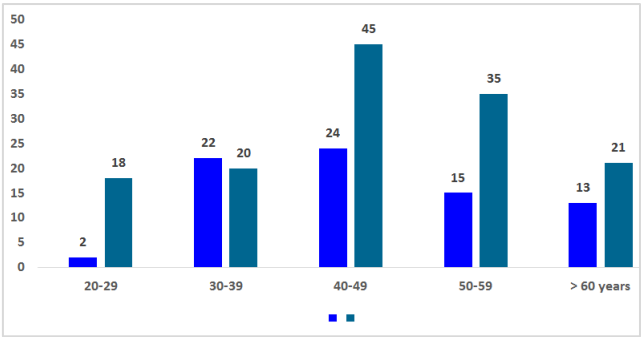

Figure 1. *Distribution of patients according to sex and age group.*

The mean LV GLS was -17.37 ± 3.87, with extremes of -28.75 and -5.14. The mean GLS was -17.67 ± 3.69 in men and -17.22 ± 3.97 in women (p = 0.44). The GLS was found

to be normal in 58.6% of patients, while 41.4% exhibited a low GLS. As demonstrated in Figures 2 and 3, there was a decline in GLS with increasing age and duration of HTN.

Univariate analysis revealed that the clinical factors associated with lower GLS in hypertensive patients were HTN lasting for > 10 years ($p < 0.001$), uncontrolled HTN ($p < 0.001$), diabetes ($p < 0.001$), obesity ($p = 0.004$) and dyslipidaemia ($p < 0.001$). The ultrasound factors associated with GLS impairment were concentric LVH ($p = 0.002$), eccentric LVH ($p = 0.026$) and remodelling ($p = 0.001$) (Table 2).

Table 2. *Factors associated with increased global longitudinal strain.*

Patients	**Normal GLS (≥ -17%) n (%)**	**Reduced GLS (< -17%) n (%)**	**p-value**
Sexe			
Male	48 (38.1)	28 (31.5)	0.31
Female	78 (61.9)	61 (68.5)	
Age groups			
< 40 years	42 (33.3)	20 (22.5)	0.35
40-60 years	65 (51.5)	54 (60.7)	
> 60 years	19 (15.1)	15 (16.9)	
HTN duration			
< 5 years	86 (68.2)	18 (20.2)	< 0.001
5-10 years	31 (24.6)	33 (37.1)	
>10 years	9 (7.2)	38 (42.7)	
Controlled HTN			
Yes	114 (81.4)	26 (18.6)	< 0.001
No	14 (18.7)	61 (81.3)	
Body mass index			
Normal	62 (49.2)	26 (29.2)	0.004
Overweight	33 (26.2)	24 (27)	
Obesity	31 (24.6)	39 (43.8)	
Diabetes			
Yes	23 (18.3)	37 (41.6)	< 0.001
No	103 (81.7)	52 (58.4)	
Dyslipidemia			
Yes	18 (14.3)	59 (66,3)	< 0.001
No	108 (85.7)	30 (33.7)	
Tobacco			
Yes	3 (2.4)	5 (5.6)	0.21
No	123 (97.6)	84 (94.4)	
Echocardiography			
Remodelling			
Yes	21 (16.7)	32 (36%)	0.001
No	105 (83.3)	57 (64%)	
Concentric LVH			

Patients	Normal GLS (≥ -17%) n (%)	Reduced GLS (< -17%) n (%)	p-value
Yes	25 (19.8)	35 (39.3)	0.002
No	101 (80.2)	54 (60.7)	
Eccentric LVH			
Yes	11 (8.7)	17 (19.1)	0.026
No	115 (91.3)	72 (80.9)	

GLS: global longitudinal strain; LVH: left ventricular hypertrophy; HTN: hypertension; n: number, %: percentage

Following a multivariate analysis, HTN that has persisted for a duration exceeding ten years, uncontrolled HTN, diabetes, dyslipidaemia, remodelling, and concentric LVH were identified as independent determinants factors of GLS decline in hypertensive patients (Table 3).

Table 3. *Independent factors associated with a decrease in GLS according to logistic regression.*

	OR	95% CI	p-value
Age	1.47	0.84-2.10	0.23
Duration of HTN > 10 years	14.28	5.55-50.00	< 0.001
Uncontrolled HTN	3.22	2.15-7.24	0.005
Diabetes	2.65	1.12-6.27	0.026
Dyslipidemia	12.15	5.32-27.77	< 0.001
Obesity	2.53	1.11-5.77	0.027
Remodelling	4.93	1.82-13.35	0.002
Concentric LVH	3.89	1.55-9.80	0.004

HTN: hypertension; LVH: left ventricular hypertrophy; OR: odds ratio; CI: confidence interval

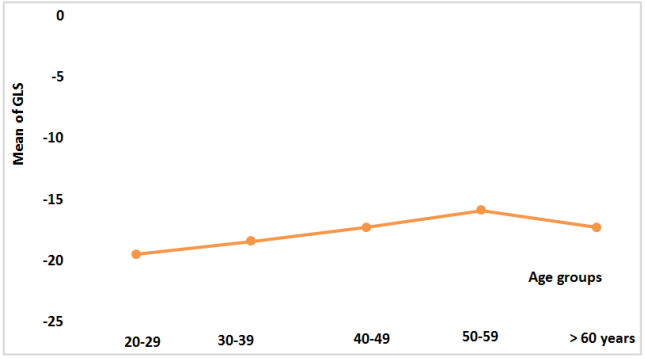

Figure 2. *Mean global longitudinal strain according to age group. There is no statistically significant relationship between the average left ventricular global longitudinal strain and age groups (p = 0.532).*

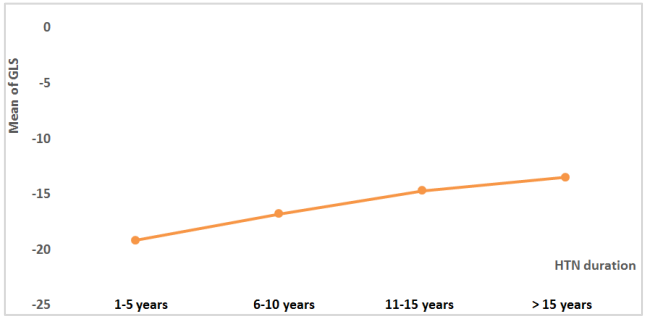

Figure 3. *Mean global longitudinal strain according to hypertension duration. The mean values of global longitudinal strain of the left ventricle decrease linearly with the duration of hypertension. The linear component is highly significant (p < 0.001) and there is no deviation from linearity (p = 0.590).*

4. Discussion

4.1. Keys Findings

A cross-sectional study was conducted on LV GLS in hypertensive patients admitted as outpatients. The primary findings of the study are as follows: myocardial dysfunction is prevalent despite a preserved LVEF. The presence of other risk factors and alterations in ventricular geometry have been observed to be associated with GLS alteration.

The present study found a prevalence of longitudinal systolic dysfunction of 41.4%. A decline in LV GLS has been documented in hypertensive patients in previous studies, with a percentage ranging from 15 to 45.5% [8, 18-20]. This discrepancy can be attributed to the utilisation of disparate thresholds for defining normal GLS, as well as to the incorporation of specific characteristics of hypertensive patients in the study. The prevalence observed in this study is comparable to that reported by Sengupta (45.5%) in their study [14]. Gonçalves et al. [18] reported a decrease in GLS in 15.3% of cases in a series of 229 hypertensive patients with preserved LVEF. However, the cut-off used in the study was lower than that employed in this study (-16%), and 53.7% of the patients in the study had grade I hypertension, which was well controlled in 61.6% of cases. Consequently, approximately half of the hypertensive patients examined during outpatient consultations exhibited longitudinal myocardial dysfunction, despite the LVEF being within the normal range. This underscores the significance of the LVEF parameter in the echocardiographic evaluation of hypertensive patients.

A significant proportion of patients with hypertension were observed to have reduced GLS, with 52% of those with HTN between 5 and 10 years and 81% of those with HTN > 10 years demonstrating reduced GLS. These results show that the duration of HTN is significantly associated with the development of longitudinal systolic dysfunction ($p < 0.002$). This finding aligns with the conclusions drawn in previous studies [20] and is consistent with the study by Narayanan et al., which demonstrated that in patients with recent HTN and a preserved systolic fraction, there was no significant change in GLS [21]. These results appear to be in accordance with data demonstrating that myocardial fibrosis in hypertensive patients progresses over time, leading to hypertensive heart disease [22].

The co-occurrence of multiple cardiovascular risk factors has been demonstrated to elevate the risk of both overt and subclinical cardiovascular complications. The present study found that major cardiovascular risk factors are associated with decreased GLS, as has been found in most studies conducted in developed countries [8, 23-25].

It has been determined through rigorous analysis and observation that obesity and HTN are independent risk factors for cardiovascular disease. However, the role of obesity in the development and progression of target organ damage in hypertensive patients remains a subject of debate [24]. The present study demonstrated a negative correlation between obesity and GLS ($p = 0.014$). A study conducted in Pakistan on a series of 134 hypertensive patients was undertaken. The patients were divided into two groups: 80 non-obese patients and 54 obese patients. The study found that the peak longitudinal systolic strain was lower in obese hypertensive patients. This suggests that obesity is a factor in LV GLS [26]. Taleb Bendiab-Soufi N. also observed a highly significant correlation between android obesity and decreased GLS on the one hand ($p = 0.014$) and obesity, characterised by a BMI >30 kg/m², and decreased GLS on the other hand [20]. This phenomenon could be attributed to the association between hypertension and obesity with heart failure of the preserved ejection fraction type.

Hypertension and diabetes have been identified as significant contributors to LV dysfunction and have been associated with an increased risk of heart failure. The combination of these factors can precipitate the onset of myocardial dysfunction. Indeed, a significant association was identified between the coexistence of HTN and diabetes, and reduced longitudinal systolic function. In an Italian study of 163 hypertensive patients, Piercarlo B. et al. [25] demonstrated a synergistic and negative impact of the combination of HTN and diabetes on LV longitudinal and radial function parameters. In a Chinese study of a series of 251 hypertensive patients, 133 of whom had impaired diastolic function, HTN and diabetes were strongly and independently associated with LV diastolic dysfunction, possibly explaining the early longitudinal systolic function impairment prior to LVEF decrease [27]. The findings of these studies indicate that improvements in glycemia and blood pressure control may contribute to the reduction of LV systolic-diastolic dysfunction progression.

As demonstrated in the study by Taleb Bendiab-Soufi et al. [23], and as is also evidenced in the present study, dyslipidaemia was identified as a substantial contributing factor to GLS impairment. In a study conducted on adolescents [28] suffering from familial hypercholesterolemia, longitudinal, circumferential and radial deformation parameters were measured via the 2D and 3D strain techniques. The results indicated that children and adolescents with hypercholesterolemia exhibited abnormalities in systolic and diastolic function parameters.

Conventional echocardiography is a reliable method that is widely used to detect altered LV systolic and diastolic function in patients with HTN. Furthermore, it is utilised in the calculation of LV mass, thereby facilitating the identification of the presence and extent of LVH. This, in turn, serves as a predictor of morbidity and mortality in hypertensive patients [29, 30]. In the present study, several parameters, including concentric or eccentric LVH and concentric LV remodelling, were found to be significantly associated with LV GLS decline in hypertensive patients. These findings are consistent with those of previous studies that identified concentric LVH, LV remodelling and LV diastolic dysfunction as factors associated with decreased GLS [31-34]. However, HTN has

been demonstrated to be associated with a reduction in the LV GLS in asymptomatic patients with a normal ejection fraction (EF), both with and without LVH, thus suggesting that LV mechanical abnormalities precede the development of LVH [35-37]. LVH is a frequent response to chronic elevation of afterload. It has been demonstrated to serve as an autonomous predictor of cardiovascular morbidity and mortality in hypertensive patients [38, 39]. The ventricular remodelling process has been linked to a number of alterations, including myocardial fibrosis and stiffness, coronary artery vasomotor disorders and an abnormal LV diastolic filling profile [40-44]. Myocardial fibrosis, which is characterised by the deposition of collagen in the myocardium, is a common complication in patients with pressure overload-related cardiac conditions. It has been observed that this fibrosis is particularly prevalent in the subendocardial layer. Moreover, there is a well-documented correlation between abnormal longitudinal function and the development of interstitial fibrosis [45].

4.2. Study Limitations

The main limitation of the study lies in the fact that we only studied longitudinal deformation via the GLS. However, this longitudinal deformation has been well validated with the LVEF in previous studies. A further limitation of this study is that it was not possible to consider parietal stress, which is an important determinant of GLS. This could influence the prevalence of longitudinal dysfunction in our study, given that approximately 40% of our patients had uncontrolled hypertension. The reduced sample size also serves to diminish statistical power and the potential for generalising findings on a broad scale across the population. Furthermore, as this study was conducted exclusively in one urban tertiary centre, it did not capture data from rural regions, which may limit the generalisability of our findings to populations with limited access to specialised cardiac care.

4.3. Implications

Notwithstanding the limitations, the findings of this study furnish critical insights pertinent to clinical practice and healthcare policy in our region. Notwithstanding the fact that the high rate GLS had reduced to a normal LVEF, this case highlights the importance of introducing a study of strain in routine echocardiography in hypertensive patients. Consequently, the assessment of longitudinal LV systolic function using strain enables the identification of subgroups with a high risk of heart failure. Nevertheless, the clinical relevance of these results must be substantiated by long-term follow-up studies. Prospective multicentre studies evaluating the medium- and long-term prognostic impact of longitudinal function and the impact of certain treatments will provide a more profound understanding of the issue in our context and enable us to reduce the incidence of heart failure in our hypertensive patients.

5. Conclusion

The prevalence of longitudinal systolic dysfunction is high in black hypertensive patients from Togo. The factors associated with HTN leading to this decrease include dyslipidaemia, diabetes, duration of HTN, remodelling and LV hypertrophy. The incorporation of 2D strain imaging into the standard echocardiographic protocol for hypertension is imperative for the identification of patients at elevated risk of heart failure, who may benefit from more assertive therapeutic interventions. The results of the present study serve to emphasise the importance of early detection and management of hypertension and its associated risk factors, with a view to preventing complications, particularly heart failure.

Abbreviations

ASE	American Society of Echocardiography
BMI	Body Mass Index
GLS	Global Longitudinal Strain
HDL	High Density Lipoprotein
HTN	Hypertension
IVS	Interventricular Septum
LDL	Low Density Lipoprotein
LV	Left Ventricular
LVEDd	Left Ventricular End-Diastolic Diameter
LVEF	Left Ventricular Ejection Fraction
LVESd	Left Ventricular End-Systolic Diameter
LVH	Left Ventricular Hypertrophy
LVM	Left Ventricular Mass
LVMi	Left Ventricular Mass index
PWT	Posterior Wall Thickness
ROT	Region Of Interest
SPSS	Statistical Package for the Social Sciences
TAPSE	Tricuspid Annular Plan Systolic Excursion
2D	Two Dimensional

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Authors Contributions

Soulemane Pessinaba: Conceptualization, Supervision, Writing – review & editing, Visualization

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Dassidi Houssouna: Writing – original draft, Validation, Writing – review & editing

Mohomed Kpelafia: Writing – review & editing

Wiyaou Kaziga: Writing – review & editing

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Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Kearney PM, Whelton M, Reynolds K, Paul M, Paul K, Jiang H et al. Global burden of hypertension: analysis of worldwide data. Lancet 2005; 365(9455): 217-23. https://doi.org/10.1016/S0140-6736(05)17741-1

[2] McEvoy JW, McCarthy CP, Bruno RM, Brouwers S, Canavan MD, Ceconi C, et al. 2024 ESC Guidelines for the management of elevated blood pressure and hypertension. Eur Heart J (2024) 45, 3912-4018. https://doi.org/10.1093/eurheartj/ehae178

[3] Cappuccio FP, Miller MA. Cardiovascular disease and hypertension in sub-Saharan Africa: burden, risk and interventions. Intern Emerg Med. 2016; 11(3): 299-305. https://doi.org/10.1007/s11739-016-1423-9

[4] Yayehd K, Damorou F, Akakpo R, Tchérou T, N'Da N, Pessinaba S et al. Prévalence de HTA et description de ses facteurs de risque à Lomé: résultats d'un dépistage réalisé dans la population générale en mai 2011. Ann Cardiol Angeiol.2013; 62(1): 43-50. https://doi.org/10.1016/j.ancard.2012.09.006

[5] Pessinaba S, Pio M, Baragou S, Yayehd K, Adamou IAR, Damorou F. Prevalence, Characteristics and Management of Hypertension in an Outpatient Clinic at the Cardiology Department in Lome (Togo). Ann Int Med Den Res 2019; 5(1): 12-16.

[6] Damasceno A, Mayosi BM, Sani M. The causes, treatment, and outcome of acute heart failure in 1006 Africans from 9 countries. Arch Intern Med. 2012; 172(18): 1386-1394. https://doi.org/10.1001/archinternmed.2012.3310

[7] Marwick TH, Gillebert TC, Aurigemma G, Chirinos J, Derumeaux G, Galderisi M, et al. Recommendations on the use of echocardiography in adult hypertension: a report from the European Association of Cardiovascular Imaging (EACVI) and the American Society of Echocardiography (ASE) dagger. Eur Heart J Cardiovasc Imaging. 2015; 16(6): 577-605. https://doi.org/10.1093/ehjci/jev076

[8] Imbalzano E, Zito C, Carerj S, Oreto G, Mandraffino G, Cusma Piccione M, et al. Left ventricular function in hypertension: new insight by speckle tracking echocardiography. Echocardiography. 2011; 28(6): 649-57. https://doi.org/10.1111/j.15408175.2011.01410.x

[9] Cikes M, Sutherland GR, Anderson LJ, Bijnens BH. The role of echocardiographic deformation imaging in hypertrophic myopa thies. Nat Rev Cardiol. 2010; 7(7): 384-96. https://doi.org/10.1038/ nrcardio.2010.56

[10] Lorell BH, Carabello BA. Left ventricular hypertrophy: pathogenesis, detection, and prognosis. Circulation. 2000; 102: 470e479. https://doi.org/10.1161/01.cir.102.4.470

[11] Edvardsen T, Rosen BD, Pan L, Jerosch-Herold M, Lai S, Hundley WG, et al. Regional diastolic dysfunction in individuals with left ventricular hypertrophy measured by tagged magnetic resonance imaging - the Multi-Ethnic Study of Atheroscleosis (MESA). Am Heart J.2006; 151: 109e114. https://doi.org/10.1016/j.ahj.2005.02.018

[12] Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantifi cation by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015; 28(1): 1 39 e14. https://doi.org/10.1016/j.echo.2014.10.003 (The paper provided updated information about deformation imaging and strain rate)

[13] Oh JK, Park JH. Role of strain echocardiography in patients with hypertension. Clinical Hypertension (2022); 28: 6: 1-11. https://doi.org/10.1186/s40885-021-00186-y

[14] Sengupta SP, Caracciolo G, Thompson C, Abe H, Sengupta PP. Early impairment of left ventricular function in patients with systemic hypertension: New insights with 2-dimensional speckle tracking echocardiography. Indian Heart J 2013; 65: 48-52. https://doi.org/10.1016/j.ihj.2012.12.009

[15] Mitchell C, Rahko PS, Blauwet LA, Canaday B, Finstuen JA, et al. Guidelines for performing a comprehensive transthoracic echocardiographic examination in adults: recommendations from the American society of echocardiography. J Am Soc Echocardiogr. 2019; 32: 1-64. https://doi.org/10.1016/j.echo.2018.06.004

[16] Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. Circulation 1977; 55: 613-8. https://doi.org/10.1161/01.cir.55.4.613

[17] Nagueh SF, Smiseth OA, Appleton CP, Byrd BF 3rd, Dokainish H, Edvardsen T, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American society of echocardiography and the European association of cardiovascular imaging. J Am Soc Echocardiogr. 2016; 29: 277-314. https://doi.org/10.1016/j.echo.2016.01.011

[18] Goncalves S, Cortez-Dias N, Nunes A, Bela A, Cabrita IZ, Sousa C, et al. Left ventricular systolic dysfunction detected by speckle tracking in hypertensive patients with preserved ejection fraction. Rev Port Cardiol 2014; 33: 27-37. https://doi.org/10.1016/j.repc.2013.07.009

[19] Kosmala W, Plaksej R, Strotmann JM, Weigel R, Herrmann S, Niemann M, et al. Progression of left ventricular functional abnormalities in hyperten sive patients with heart failure: an ultrasonic two-dimen sional speckle tracking study. J Am Soc Echocardiogr 2008; 21: 1309-1317. https://doi.org/10.1016/j.echo.2008.10.006

[20] Taleb Bendiab-Soufi N. Evaluation de la fonction longitudinale par le 2D strain dans la détection précoce de l'atteinte cardiaque chez l'hypertendu. Thèse, Université d'Alger 1 Tlemcen. 2017.
http://dspace.univtlemcen.dz/handle/112/12712

[21] Narayanan A, Aurigemma G, Chinali M, Hill JC, Meyer TE, Tighe DA. Cardiac mechanics in mild hypertensive heart disease: a speckle strain imaging study. Circ Cardiovasc Imaging 2009; 2: 382-390.
https://doi/10.1161/CIRCIMAGING.108.811620

[22] Su MYM, Lin LY, Tseng YH, Chang CC, Wu CK, Lin JL, et al. CMR-verified diffuse myocardial fibrosis is associated with diastolic dysfunction in HFpEF, JACC Cardiovasc. Imaging, 2014; 7: 991- 97. 331. https://doi.org/10.1016/j.jcmg.2014.04.022

[23] Taleb Bendiab-Soufi N, Meziane-Tani A, Ouabdesselam S, Methia N, Latreche S, Henaoui L, et al. Factors associated with global longitudinal strain decline in hypertensive patients with normal left ventricular ejection fraction. Eur J Prev Cardiol 2017; 24(14): 1463-1472.
https://doi.org/10.1177/2047487317721644

[24] Russo C, Jin Z, Homma S, Rundek T, Elkind MS, Sacco RL, et al. Effect of obesity and overweight on left ventricular diastolic function: à community-based study in an elderly cohort. J Am Coll Cardiol 2011; 57: 1368-74.
https://doi.org/10.1016/j.jacc.2010.10.042

[25] Ballo P, Cameli M, Mondillo S, Giacomin E, Lisi M, Padeletti M, et al. Impact of diabetes and hypertension on left ventricular longitudinal systolic function. Diabetes Resear and Clini Prac 2010; 90: 209- 15. 342.
https://doi.org/10.1016/j.diabres.2010.08.004

[26] Miyoshi H, Oishi Y, Mizugushi Y, Iushi A, Nagase N, Ara N, et al. Contribution of obesity to left atrial and left ventricular dysfunction in asymptomatic patients with hypertension: A two-dimensional speckle- tracking echocardiographic study. J Am Soc Hypertens 2014; 8: 54- 63.
https://doi.org/10.1016/j.jash.2013.08.005

[27] Meng XY, Zhou Y, Zhang J, Tang ZH. The association and interaction analysis of hypertension and diabetes mellitus on diastolic heart failure in a high-risk population. Int J Clin Exp Med 2015; 8: 21311-18.

[28] Vitarelli A, Martino F, Capotosto L, Martino E, Colantoni C, Ashurov R, et al. Early myocardial deformation changes in hypercholesterolemic and obese children and adolescents: a 2D and 3D speckle tracking echocardiography study. Medicine (Baltimore). 2014; 93(12): e71.
https://doi.org/10.1097/MD.0000000000000071

[29] Kehat I, Molkentin JD. Molecular pathways underlying cardiac remodelling during pathophysiological stimulation. Circulation. 2010; 122(25): 2727-35.
https://doi.org/10.1161/CIRCULATIONAHA.110.942268

[30] Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med. 1990; 322(22): 1561-6.
https://doi.org/10.1056/NEJM199005313222203

[31] Mizuguchi Y, Oishi Y, Miyoshi H, Iuchi A, Nagase N, Oki T. The functional role of longitudinal, circumferential, and radial myocar dial deformation for regulating the early impairment of left ventricular contraction and relaxation in patients with cardiovascular risk factors: a study with two-dimensional strain imaging. J Am Soc Echocardiogr 2008; 21: 1138-1144.
https://doi.org/10.1016/j.echo.2008.07.016

[32] Hare JL, Brown JK and Marwick TH. Association of myocardial strain with left ventricular geometry and the incidence of hypertensive heart disease. Am J Cardiol 2008; 102: 87-91.
https://doi.org/10.1016/j.amjcard.2008.02.101

[33] Mu Y, Qin C, Wang C, Huojiaabudula G. Two-dimensional ultra sound speckle tracking imaging in evaluation of early changes in left ventricular diastolic function in patients with essential hypertension. Echocardiography 2010; 27: 146-154.
https://doi.org/10.1111/j.1540-8175.2009.00984.x

[34] Kane GC, Karon BL, Mahoney DW, Redfield MM, Roger VL, Burnett Jr JC, et al. Progression of left ventricular diastolic dysfunction and risk of heart failure. JAMA 2011; 306: 856-863. https://doi.org/10.1001/jama.2011.1201

[35] Rosen BD, Saad MF, Shea S, Nasir K, Edvardsen T, Burke G, et al. Hypertension and smokingareassociated with reduced regional left ventricular function in asymptomatic: individuals the Multi-Ethnic Study of Atherosclerosis. J Am Coll Cardiol. 2006; 47(6): 1150-8.
https://doi.org/10.1016/j.jacc.2005.08.078

[36] Kang SJ, Lim HS, Choi BJ, Choi SY, Hwang GS, Yoon MH, et al. Longitudinal strain and torsion assessed by two-dimensional speckle tracking correlate with the serum level of tissue inhibitor of matrix metalloproteinase-1, a marker of myocardial fibrosis, in pa tients with hypertension. J Am Soc Echocardiogr. 2008; 21(8): 907 11.
https://doi.org/10.1016/j.echo.2008.01.015

[37] Kouzu H, Yuda S, Muranaka A, Doi T, Yamamoto H, Shimoshige S, et al. Left ventricular hypertrophy causes different changes in longitu dinal, radial, and circumferential mechanics in patients with hyperten sion: a two-dimensional speckle tracking study. J Am Soc Echocardiogr. 2011; 24(2): 192-9. https://doi.org/10.1016/j.echo.2010. 10.020

[38] Casale PN, Devereux RB, Milner M, Zullo G, Harshfield GA, Pickering TG, et al. Value of echocardiographic measurement of left ventricular mass in predicting cardiovascular morbid events in hypertensive men. Ann Intern Med. 1986; 105(2): 173-8. https://doi.org/10.7326/0003-4819-105-2-173

[39] Devereux RB, Wachtell K, Gerdts E, Boman K, Nieminen MS, Papademetriou V, et al. Prognostic significance of left ventricular mass change during treatment of hypertension. JAM A. 2004; 292(19): 2350-6.
https://doi.org/10.1 001/jama.292.19.2350

[40] Doering CW, Jalil JE, Janicki JS, Pick R, Aghili S, Abrahams C, et al. Collagen network remodelling and diastolic stiffness of the rat left ventricle with pressure overload hypertrophy. Cardiovasc Res. 1988; 22(10): 686-95.
https://doi.org/10.1093/cvr/22.10.686

[41] Jalil JE, Doering CW, Janicki JS, Pick R, Clark WA, Abrahams C, et al. Structural vs. contractile protein remodelling and myocardial stiffness in hypertrophied rat left ventricle. J Mol Cell Cardiol. 1988; 20(12): 1179-87. https://doi.org/10.1016/0022-2828(88)90597-4

[42] Wicker P, Tarazi RC, Kobayashi K. Coronary blood flow during the development and regression of left ventricular hypertrophy in renovascular hypertensive rats. Am J Cardiol. 1983; 51(10): 1744-9. https://doi.org/10.1016/0002-9149(83)90222-9

[43] Canby CA, Tomanek RJ. Role of lowering arterial pressure on maximal coronary flow with and without regression of cardiac hypertrophy. Am J Phys. 1989; 257(4): H1110-8. https://doi.org/10.1152/ajpheart.1989.257.4. H1110

[44] Haider AW, Larson MG, Benjamin EJ, Levy D. Increased left ventricular mass and hypertrophy are associated with increased risk for sudden death. J Am Coll Cardiol. 1998; 32(5): 1454-9. https://doi.org/10.1016/S0735-1097(98)004 07-0

[45] Lumens J, Delhaas T, Arts T, Cowan BR, Young AA. Impaired subendocardial contractile myofiber function in asymptomatic aged humans, as detected using MRI. Am J Physiol Heart Circ Physiol. 2006; 291(4): H1573-9. https://doi. org/10.1152/ajpheart.00074.2006