Original Article

Impact of antihypertensive treatment on myocardial mechanics in elderly hypertensive patients with different left ventricular geometry patterns: a two-dimensional speckle-tracking echocardiography study

Xiaoyan Kang, Jiayu Zhang, Junyu Liu, Junwang Miao, Shuai Li, Chunsong Kang^, Jiping Xue

Department of Ultrasound, Shanxi Bethune Hospital, Shanxi Academy of Medical Sciences, Tongji Shanxi Hospital, Third Hospital of Shanxi Medical University, Taiyuan, China

Contributions: (I) Conception and design: C Kang; (II) Administrative support: C Kang, J Xue; (III) Provision of study materials or patients: X Kang, J Zhang, J Liu; (IV) Collection and assembly of data: X Kang, J Miao, S Li; (V) Data analysis and interpretation: X Kang, J Zhang, J Liu; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Chunsong Kang, MD. Department of Ultrasound, Shanxi Bethune Hospital, Shanxi Academy of Medical Sciences, Tongji Shanxi Hospital, Third Hospital of Shanxi Medical University, No. 99 Longcheng Street, Taiyuan 030032, China. Email: kangcs2023@163.com.

Background: Hypertension can cause left ventricular remodeling, and the degree of myocardial mechanical damage is different in patients with different geometries. Antihypertensive treatment can improve myocardial mechanics, but the improvement in myocardial mechanics in hypertensive patients with different geometries is not clear. This study aimed to assess the impact of antihypertensive therapy on myocardial mechanics in elderly patients diagnosed with primary hypertension, considering diverse left ventricular geometry patterns.

Methods: This study conducted a retrospective cohort analysis by randomly selecting 221 elderly patients diagnosed with primary hypertension from the outpatient department of Bethune Hospital in Shanxi Province, between January and June 2017. In this study, we included 191 elderly hypertensive patients (mean age 67.71±3.26 years, 98 women) who had successfully attained the target blood pressure levels through the administration of antihypertensive medications. The participants were stratified into distinct left ventricular geometry categories, namely normal geometry (NG; 54 cases), concentric remodeling (CR; 42 cases), eccentric hypertrophy (EH; 41 cases), and concentric hypertrophy (CH; 54 cases). The four groups were compared in terms of the global longitudinal strain (GLS) and its changes at baseline and 12 and 24 months after antihypertensive therapy.

Results: The baseline GLS exhibited a gradual decrease in the following sequence: NG, CR, EH, and CH groups (all $P<0.05$). GLS demonstrated improvement in the CR, EH, and CH groups after 12 and 24 months of treatment; the GLS of CR group changed from –19.65%±1.27% to –20.72%±2.06%, then to –21.25%±1.99%; EH group changed from –18.76%±1.39% to –20.57%±1.75%, then to –21.37%±1.88%; CH group increased from –17.51%±1.42% to –20.04%±2.17%, then to –20.81%±2.51% (all $P<0.05$). After 24 months of treatment, GLS in the NG group improved from –20.84%±1.36% at baseline to –21.90%±1.99% ($P<0.05$). Within the CR, EH, and CH groups, the improvement in GLS (ΔGLS) following 12 months of treatment exceeded ΔGLS between 12 months and 24 months of treatment (all $P<0.05$). ΔGLS after 12 months of treatment progressively increased in the order of NG, CR, EH, and CH groups (all $P<0.05$), while ΔGLS between 12 and 24 months of treatment exhibited similarity across the four groups ($P>0.05$).

^ ORCID: 0009-0004-7150-1627.

Conclusions: The degree of myocardial mechanical damage is different in elderly primary hypertensive patients with different left ventricular geometries. After antihypertensive treatment, the myocardial mechanical damage continues to improve in all groups, and the concentric hypertrophy group had the most significant improvement.

Keywords: Antihypertensive therapy; hypertension; left ventricular geometry; myocardial mechanics; two-dimensional speckle-tracking echocardiography

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Introduction

Hypertension constitutes a significant public health concern, and its prevalence along with the associated cardiovascular disease risk in elderly populations, is notably escalating with the aging population (1). The principal target organ affected by hypertension is the left ventricle (LV). Prolonged elevation of blood pressure induces sustained increases in LV afterload, prompting compensatory hyperplasia and hypertrophy of cardiomyocytes, augmentation in myocardial mass, and enlargement of the ventricular cavity. Consequently, these changes alter LV geometry and compromise both systolic and diastolic functions (2). While left ventricular ejection fraction (LVEF) is conventionally employed to assess LV systolic function, it remains frequently within the normal range during the early stages of abnormal systolic function. Notably, global longitudinal strain (GLS), measured utilizing the speckle-tracking technique, can identify subclinical left LV systolic dysfunction. This parameter serves as a reliable prognostic indicator for cardiovascular diseases (3-5).

Previous studies have indicated that hypertension-induced left ventricular (LV) remodeling initially manifests as alterations in myocardial mechanics, followed by diastolic dysfunction and eventually left ventricular hypertrophy (LVH). Thus, abnormalities in myocardial mechanics are considered the earliest detectable changes in hypertension. The cardiac geometry can be categorized based on the left ventricular mass index (LVMI) and relative wall thickness (RWT) into four types: normal geometry (NG), concentric remodeling (CR), eccentric hypertrophy (EH), and concentric hypertrophy (CH). Research has demonstrated that different LV geometric patterns exhibit varying degrees of impairment in myocardial mechanics (6,7). Tadic *et al.* (8) reported a progressive decline in GLS across the sequence of LV geometries: NG, CR, EH, and CH. Regardless of the geometry, aggressive antihypertensive treatment is needed to prevent further left ventricular remodeling, especially in EH and CH patients. A prior study (9) has suggested that antihypertensive treatment can reverse myocardial fibrosis and improve myocardial mechanics. However, the extent of improvement in myocardial mechanics following antihypertensive therapy among patients with different LV geometries and the underlying mechanisms remain inconclusive.

Therefore, the aim of this study is to evaluate the changes of myocardial mechanics before and after antihypertensive treatment in elderly hypertensive patients with different left ventricular geometry by two-dimensional speckle tracking echocardiography, and to explore the potential mechanisms behind these changes, so as to provide scientific evidence for personalized treatment strategies in hypertensive patients and optimize clinical decision-making. We present this article in accordance with the STROBE reporting checklist (available at https://qims.amegroups.com/article/view/10.21037/qims-24-1419/rc).

Methods

Participants

This study randomly selected 221 elderly patients with primary hypertension who visited the outpatient department of Shanxi Bethune Hospital from January to June 2017 and conducted a retrospective cohort analysis. Antihypertensive therapy was initiated for patients who had not received prior treatment, and adjustments were made for those already undergoing antihypertensive therapy to achieve the target blood pressure values (130–150/<90 mmHg) recommended for elderly patients who are hypertensive in China within a 3-month period (10). Subsequently, the maintenance of antihypertensive therapy was implemented. Following the

exclusion of 30 patients due to reasons such as loss to follow-up, substandard blood pressure, or poor-quality images, the final group comprised of 191 elderly patients who were hypertensive, aged 65 to 79 years (67.71±3.26 years), consisting of 93 men and 98 women. The patients were categorized into four groups based on LVMI and RWT: (I) the NG group (54 cases): LVMI ≤115 g/m^2 (male) or ≤95 g/m^2 (female) and RWT ≤0.42; (II) the CR group (42 cases): LVMI ≤115 g/m^2 (male) or ≤95 g/m^2 (female) and RWT >0.42; (III) the EH group (41 cases): LVMI >115 g/m^2 (male) or >95 g/m^2 (female) and RWT ≤0.42; (IV) the CH group (54 cases): LVMI >115 g/m^2 (male) or >95 g/m^2 (female) and RWT >0.42 (11).

The inclusion criteria for patients were as follows: (I) patients aged between 65 to 79 years (10); (II) patients exhibiting systolic blood pressure (SBP) ≥140 mmHg and/or diastolic blood pressure (DBP) ≥90 mmHg, confirmed through three measurements conducted on non-consecutive days (10); (III) patients achieving the target blood pressure value through medication within a 3-month period.

Conversely, the exclusion criteria for patients were as follows: (I) patients with secondary hypertension, cardiomyopathy, rheumatic heart disease, congenital heart disease, severe heart valve disease, myocardial infarction or unstable angina, pulmonary heart disease, a history of stroke, diabetes mellitus, and severe liver and kidney diseases; (II) patients who were lost to follow-up or presented with poor-quality images (Figure S1). The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of Shanxi Bethune Hospital (No. YXLL-2020-045) and informed consent was obtained from all the patients.

Collection of general clinical data

All patient data, encompassing sex, age, weight, height, heart rate, duration of hypertension, body mass index (BMI, kg/m^2), and body surface area (BSA, m^2), were systematically collected. The BMI and BSA were computed using the formulas:

$$BMI = weight / height^2 \quad [1]$$

$$BSA = 0.0061 \times height + 0.0128 \times weight - 0.1529 \quad [2]$$

Blood pressure assessments were conducted in a clinical setting. Following a resting period of 5 to 10 minutes, blood pressure was measured on the upper arm in a seated position, using a standard sphygmomanometer. The upper arm was supported at heart level during the measurement, employing an appropriately sized arm cuff positioned 2.5 cm above the elbow bend. Measurements were taken at 3-minute intervals, and the average of three readings was calculated and recorded.

Image acquisition

Images of the study participants were captured at three time points: at the baseline, 12 months after the initiation of antihypertensive therapy, and 24 months after antihypertensive therapy. The GE Vivid E9 ultrasound machine equipped with an M5S probe (1.5–4.5 MHz, with a frame rate of 60–80 frames per second), and the Echo PAC analysis software were used in the study.

For each image acquisition, participants were directed to rest for 5 minutes while being connected to an electrocardiogram in the left-lateral decubitus position for echocardiography. The following parameters were measured: intraventricular septal thickness at end-diastole (IVSTd), left ventricular posterior wall thickness at end-diastole (LVPWTd), left ventricular end-diastolic diameter (LVEDD), and left ventricular end-systolic diameter (LVESD) using M-mode echocardiography in the parasternal long-axis view of the LV. LVEF was measured in apical 4-chamber and 2-chamber views employing the biplane Simpson's method. In the apical 4-chamber views, early diastolic mitral peak flow velocity (E) was measured using pulsed Doppler, and early diastolic mitral annular mean velocity (e') at the septal and lateral areas was obtained through tissue Doppler. Subsequently, mean value of the ratio of E to e' (E/e') was calculated. Dynamic images were acquired under conditions of maximum frame rate and optimal image quality, stored in three standard transthoracic apical views (apical 4-chamber, 2-chamber, and 3-chamber views) and subjected to offline analysis. All measurements were performed in at least three cardiac cycles, and average values were computed.

Left ventricular mass (LVM) was calculated using the following equation:

$$LVMg = 0.8 \times 1.04 \times \left[(IVSD + LVPWD + LVEDD)^3 - LVEDD^3 \right] + 0.6 \quad [3]$$

where IVSD represents interventricular septal thickness at end-diastole and LVPWD represents left ventricular posterior wall thickness at end-diastole.

LVMI was corrected with BSA:

$$LVMI = LVM / BSA \quad [4]$$

Meanwhile, RWT was calculated:

$$RWT = 2 \times (LVPWD) / LVEDD \quad [5]$$

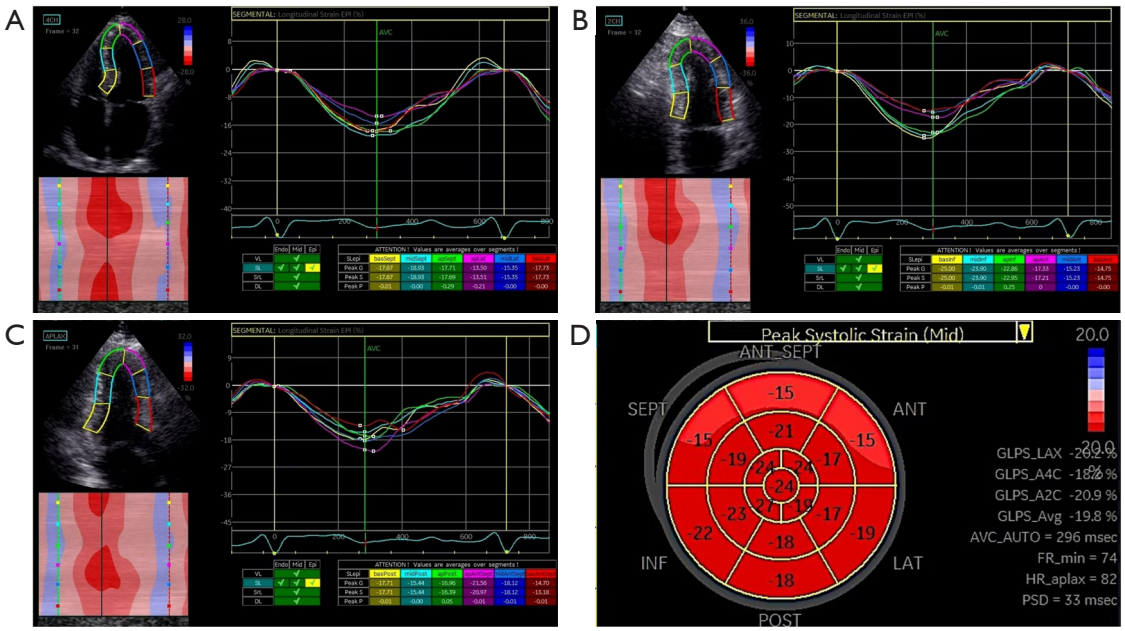

Figure 1 The analysis of GLS and acquisition of bull's eye diagrams. (A-C) The LV endocardium was appropriately labeled in apical 4-chamber, in apical 2-chamber, and 3-chamber views. (D) The software automatically computed the GLS value for the entire left ventricular myocardium and created a bull's eye diagram illustrating the longitudinal strain across 17 segments. GLS, global longitudinal strain; LV, left ventricular.

Image analysis

The acquired images were imported into the Echo PAC workstation for complete analysis. The "Q-Analysis" program was employed to access the "2D Strain" analysis mode. Dynamic images exhibiting clear LV endocardium in apical 4-chamber, 2-chamber, and 3-chamber views were selected, and the LV endocardium was appropriately labeled. Subsequently, the software automatically initiated the tracking process, generating the region of interest. Manual adjustments to the tracing line were applied if deemed necessary to ensure successful tracking of all LV segments. Upon clicking "Approve", the software automatically computed the GLS value for the entire left ventricular myocardium and created a bull's eye diagram illustrating the longitudinal strain across 17 segments (*Figure 1*).

Repeatability test

A subset of 20 elderly patients who were hypertensive were randomly chosen, and their images were subjected to analysis by two experienced physicians using the standard methodology as previously described, and the GLS of the LV was ascertained. All physicians have more than 8 years of specialized work experience. Subsequently, one of the physicians reanalyzed the same set of images after a one-week interval. This process allowed for the assessment of repeatability both between different observers and for the same observer over time.

Statistical methods

The data were subjected to statistical analysis using SPSS 26.0 software. Normal distribution of measurement data was assessed through the Kolmogorov-Smirnov test. For normally distributed measurement data, descriptive statistics were presented as mean ± standard deviation. Baseline comparisons among multiple groups were conducted using one-way analysis of variance (ANOVA), followed by post-hoc pairwise comparisons using the least significant difference (LSD)-*t* test. Count data were summarized as frequencies (percentages) and compared between groups using the Chi-squared test. Intra-group comparisons of parameters at baseline, 12 months, and 24 months of antihypertensive therapy were performed using repeated-measures ANOVA. Changes in ΔGLS from baseline to 12 months and between 12 and 24 months of antihypertensive therapy were assessed within each group using paired-samples *t*-tests. Concurrent ΔGLS comparisons among

Table 1 General clinical data of each group

Characteristics	NG (n=54)	CR (n=42)	EH (n=41)	CH (n=54)	χ^2/F value	P value
Sex, n					1.817	0.611
Male	29	19	17	28		
Female	25	23	24	26		
Age (years)	68.11±2.98	67.74±3.19	67.20±2.76	67.67±3.90	0.641	0.607
SBP (mmHg)	147.67±7.38	149.40±7.27	155.54±10.00[ab]	156.13±10.40[ab]	11.496	<0.001
DBP (mmHg)	84.91±7.35	83.29±10.08	95.51±11.68[ab]	95.69±7.55[ab]	25.261	<0.001
BMI (kg/m^2)	25.34±2.61	25.35±3.55	24.31±3.17	24.59±2.46	1.510	0.213
Heart rate (beats/min)	75.98±6.18	73.00±8.74	73.59±8.18	75.93±8.63	1.801	0.149
Duration of hypertension (years)	13.13±9.54	12.00±7.36	13.34±8.87	12.80±7.15	0.215	0.886

Data are presented as mean ± standard deviation, unless otherwise specified. [a], P<0.05 compared with the NG group; [b], P<0.05 compared with the CR group. 1 mmHg = 0.133 kPa. BMI, body mass index; CR, concentric remodeling; CH, concentric hypertrophy; DBP, diastolic blood pressure; EH, eccentric hypertrophy; NG, normal geometry; SBP, systolic blood pressure.

multiple groups during specific time intervals after antihypertensive therapy were conducted using one-way ANOVA, with subsequent pairwise comparisons facilitated by the LSD-*t* test. The repeatability test analysis was executed through Bland-Altman plots. Statistical significance was established at a threshold of P<0.05 and all tests were two-sided.

Results

Comparisons of general data

The SBP and DBP values in both the CH and EH groups exhibited statistically significant elevation compared to the NG and CR groups (all P<0.05). Conversely, no statistically significant differences were discerned in terms of sex, age, BMI, heart rate, and duration of hypertension across the four groups (all P>0.05), as detailed in *Table 1*.

Comparisons of echocardiographic parameters at baseline among the four groups

The CR group exhibited statistically significant elevations in IVSTd, LVPWTd, and LVMI compared to the NG group. Additionally, the CR group demonstrated significantly higher RWT than both the NG and EH groups (all P<0.05).

In the EH group, IVSTd and LVPWTd were statistically significantly higher than those in the NG group (all P<0.05). Moreover, LVMI and E/e' were statistically significantly elevated in the EH group compared to the NG and CR groups (all P<0.05). At the same time, the EH group exhibited statistically markedly higher LVEDD and LVESD than the other three groups, along with statistically obvious lower RWT than the CR and CH groups (all P<0.05).

The CH group displayed significantly higher IVSTd, LVPWTd, LVMI, and E/e' compared to the other three groups. Additionally, LVEDD and LVESD in the CH group were statistically markedly higher than those in the NG and CR groups, while they were statistically significantly lower than those in the EH group. RWT was statistically obviously higher in the CH group than in the NG and EH groups (all P<0.05).

Baseline GLS exhibited variation among the four groups, demonstrating a gradual decrease in the order of NG, CR, EH, and CH groups, with statistically significant differences (all P<0.05) (*Table 2*).

Comparisons of echocardiographic parameters in each group prior to and following antihypertensive therapy

Following 12 and 24 months of antihypertensive therapy, the CH group displayed gradual decreases in IVSTd, LVPWTd, LVEDD, LVESD, and LVMI, with statistically significant differences (P<0.05). Additionally, the E/e' value at 12 or 24 months of antihypertensive therapy was statistically significantly lower than the baseline value (P<0.05).

In the EH group, LVEDD, LVESD, and LVMI demonstrated a progressive decrease after both 12 and 24 months of antihypertensive therapy, with statistically

Table 2 Baseline echocardiographic parameters in each group

Characteristics	NG (n=54)	CR (n=42)	EH (n=41)	CH (n=54)	F value	P value
IVSTd (mm)	9.57±0.57	10.21±0.84[a]	10.17±0.83[a]	11.37±0.73[abc]	49.817	<0.001
LVPWTd (mm)	9.17±0.50	10.17±0.66[a]	9.90±0.58[a]	11.37±0.65[abc]	115.594	<0.001
LVEDD (mm)	45.26±2.91	44.26±2.51	50.54±2.82[ab]	48.67±2.73[abc]	35.742	<0.001
LVESD (mm)	30.85±2.10	30.02±1.81	34.44±2.24[ab]	33.19±2.05[abc]	35.996	<0.001
LVMI (g/m^2)	77.35±12.79	88.43±8.74[a]	108.98±10.03[ab]	120.96±11.39[abc]	154.691	<0.001
RWT	0.41±0.02	0.46±0.03[a]	0.39±0.03[b]	0.47±0.04[ac]	74.138	<0.001
LVEF (%)	63.54±4.36	64.60±3.64	63.80±3.39	63.85±3.84	0.465	0.761
E/e'	9.39±1.93	8.62±2.11	12.07±2.65[ab]	13.15±2.60[abc]	43.824	<0.001
GLS (%)	−20.84±1.36	−19.65±1.27[a]	−18.76±1.39[ab]	−17.51±1.42[abc]	97.141	<0.001

Data are presented as mean ± standard deviation, unless otherwise specified. [a], P<0.05 compared with the NG group; [b], P<0.05 compared with the CR group; [c], P<0.05 compared with the EH group. CR, concentric remodeling; CH, concentric hypertrophy; E/e', average value of ratio of early diastolic mitral peak flow velocity (E) to early diastolic mitral annular velocity (e') at the septal and lateral sides; EH, eccentric hypertrophy; GLS, global longitudinal strain; IVSTd, intraventricular septal thickness at end-diastole; LVPWTd, left ventricular posterior wall end-diastolic thickness; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVMI, left ventricular mass index; LVEF, left ventricular ejection fraction; NG, normal geometry; RWT, relative wall thickness.

significant differences (all P<0.05). In this group, the E/e' value at 12 or 24 months of antihypertensive therapy was statistically markedly lower than the baseline value (P<0.05).

After 24 months of antihypertensive therapy, the EH group demonstrated a statistically significant elevation in RWT, while the CH group exhibited a statistically significant reduction (both P<0.05).

GLS exhibited a gradual enhancement after 12 and 24 months of antihypertensive therapy in the CR, EH, and CH groups; the GLS of CR group (−19.65%±1.27% *vs.* −20.72%±2.06% *vs.* −21.25%±1.99%), EH group (−18.76%±1.39% *vs.* −20.57%±1.75% *vs.* −21.37%±1.88%), CH group (−17.51%±1.42% *vs.* −20.04%±2.17% *vs.* −20.81%±2.51%) (all P<0.05). In the NG group, baseline GLS revealed statistically insignificant differences compared to GLS at 12 months after antihypertensive therapy (P>0.05) but was lower than GLS at 24 months after antihypertensive therapy (−20.84%±1.36% *vs.* −21.90%±1.99%) (P<0.05) (refer to *Table 3*).

Comparisons of ΔGLS over time in each group following antihypertensive therapy

Within the NG group, the change in ΔGLS between baseline and 12 months of antihypertensive therapy exhibited no statistically significant difference compared to that between 12 and 24 months of antihypertensive therapy (P>0.05). Conversely, in the CR, EH, and CH groups, ΔGLS between baseline and 12 months of antihypertensive therapy was higher than ΔGLS between 12 and 24 months of antihypertensive therapy; the ΔGLS of CR group (−1.08%±1.38% *vs.* −0.52%±0.62%), EH group (−1.81%±1.12% *vs.* −0.79%±0.56%), CH group (−2.52%±1.67% *vs.* −0.77%±0.94%) (all P<0.05) (*Table 4*). Furthermore, ΔGLS between baseline and 12 months of antihypertensive therapy demonstrated a progressive increase in the order of NG, CR, EH, and CH groups, with the lowest value observed in the NG group and the highest in the CH group (−0.54%±0.89% *vs.* −1.08%±1.38% *vs.* −1.81%±1.12% *vs.* −2.52%±1.67%, all P<0.05) Notably, ΔGLS between 12 and 24 months of antihypertensive therapy did not exhibit statistically significant differences among the four groups (P>0.05) (*Figure 2*).

Test results' repeatability

The mean discrepancy in GLS measured by the same observer at various time points was 0.095, with a 95% confidence interval (CI) ranging from −0.463 to 0.653. When measured by different observers, the mean discrepancy in GLS was 0.245, with a 95% CI from −0.304 to 0.794. Bland-Altman analysis revealed a consistent pattern wherein the difference in repeatedly measured GLS corresponded to the average value of GLS.

Table 3 Comparisons of echocardiographic parameters in each group prior to and following antihypertensive therapy

Groups	Time	IVSTd (mm)	LVPWTd (mm)	LVEDD (mm)	LVESD (mm)	LVMI (g/m^2)	RWT	LVEF (%)	E/e'	GLS (%)
NG	Baseline	9.57±0.57	9.17±0.50	45.26±2.91	30.85±2.10	77.35±12.79	0.41±0.02	63.54±4.36	9.39±1.93	−20.84±1.36
	12 months of AT	9.65±0.71	9.30±0.54	45.52±2.30	31.17±2.42	79.88±8.67	0.41±0.03	63.22±7.73	9.56±3.25	−21.38±1.71
	24 months of AT	9.41±0.72	9.39±0.60	45.39±2.41	30.87±1.96	78.74±9.47	0.41±0.03	64.63±3.98	9.09±2.18	−21.90±1.99[a]
CR	Baseline	10.21±0.84	10.17±0.66	44.26±2.51	30.02±2.15	88.43±8.74	0.46±0.03	64.60±3.64	8.62±2.11	−19.65±1.27
	12 months of AT	9.98±0.68	9.95±0.58	44.38±2.70	30.19±2.56	86.11±8.26	0.45±0.03	63.90±6.15	9.52±2.47	−20.72±2.06[a]
	24 months of AT	10.26±0.63	9.98±0.60	44.19±2.22	29.79±1.75	87.52±9.07	0.45±0.03	65.00±3.438	9.45±1.17	−21.25±1.99[ab]
EH	Baseline	10.17±0.83	9.90±0.58	50.54±2.68	34.44±2.24	108.98±10.03	0.39±0.03	63.80±3.39	12.07±2.65	−18.76±1.39
	12 months of AT	9.98±0.76	9.88±0.56	48.85±2.04[a]	32.90±1.85[a]	101.76±9.86[a]	0.41±0.03	64.41±3.94	8.27±1.83[a]	−20.57±1.75[a]
	24 months of AT	10.05±0.95	9.80±0.72	46.73±2.02[ab]	31.76±1.92[ab]	94.59±10.01[ab]	0.42±0.04[a]	64.20±4.25	7.93±1.68[a]	−21.37±1.88[ab]
CH	Baseline	11.37±0.73	11.37±0.65	48.67±2.73	33.19±2.05	120.96±11.39	0.47±0.04	63.85±3.84	13.15±2.61	−17.51±1.42
	12 months of AT	10.67±0.87[a]	10.50±0.67[a]	45.76±2.65[a]	31.19±2.31[a]	99.36±11.76[a]	0.46±0.04	63.59±4.88	8.48±2.11[a]	−20.04±2.17[a]
	24 months of AT	9.98±0.71[ab]	9.96±0.61[ab]	44.37±2.10[ab]	30.02±1.54[ab]	87.23±10.47[ab]	0.45±0.04[a]	64.63±3.38	9.00±2.09[a]	−20.81±2.51[ab]

Data are presented as mean ± standard deviation, unless otherwise specified. [a], P<0.05 compared with baseline value; [b], P<0.05 compared with values after 12 months of antihypertensive therapy. CR, concentric remodeling; CH, concentric hypertrophy; E/e', average value of ratio of early diastolic mitral peak flow velocity (E) to early diastolic mitral annular velocity (e') at the septal and lateral sides; EH, eccentric hypertrophy; GLS, global longitudinal strain; IVSTd, intraventricular septal thickness at end-diastole; LVPWTd, left ventricular posterior wall end-diastolic thickness; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVMI, left ventricular mass index; LVEF, left ventricular ejection fraction; NG, normal geometry; RWT, relative wall thickness.

Table 4 Comparisons of the degree of GLS improvement in each group over time following antihypertensive therapy

Groups	ΔGLS (%)		*t* value	P value
	Baseline–12 months of AT	12–24 months of AT		
NG	−0.54±0.89	−0.51±0.75	0.209	0.853
CR	−1.08±1.38[a]	−0.52±0.62	2.216	0.032
EH	−1.81±1.12[ab]	−0.79±0.56	5.037	<0.001
CH	−2.52±1.67[abc]	−0.77±0.94	6.866	<0.001
F value	22.892	1.963		
P value	<0.001	0.121		

Data are presented as mean ± standard deviation, unless otherwise specified. Between baseline and 12 months of antihypertensive therapy, [a], P<0.05 compared with the NG group; [b], P<0.05 compared with the CR group; [c], P<0.05 compared with the EH group. ΔGLS, the improvement in global longitudinal strain; AT, antihypertensive therapy; CR, concentric remodeling; CH, concentric hypertrophy; EH, eccentric hypertrophy; NG, normal geometry.

Discussion

LV remodeling serves as an independent risk factor for cardiovascular events such as heart failure and myocardial infarction in hypertensive patients (12). Different left ventricular geometries are associated with varying cardiovascular risks, and myocardial mechanics serve as predictors of cardiovascular events (13). In the early stage of the course of hypertension, left ventricular myocardial mechanics may be impaired. Currently, cardiac magnetic resonance (CMR) technology is considered the gold standard for evaluating myocardial structure and function. However, due to the influence of examination time and cost, its clinical application is rather limited. Compared with CMR, echocardiography has obvious advantages. However, usually LVEF is not sensitive to early injury of left ventricular systolic function and cannot reflect subclinical left ventricular myocardial systolic function impairment. GLS measured by two-dimensional speckle tracking echocardiography (2D-STE) technology is considered to be superior to LVEF in the assessment of antihypertensive subclinical cardiac function reduction and prognosis (5), and has a good correlation with the GLS obtained by CMR (14,15).

Antihypertensive therapy plays a crucial role in delaying hypertension-induced target organ damage, substantially diminishing the risk of cardiovascular events, particularly in elderly patients who are hypertensive (16). Studies have demonstrated that myocardial mechanics in patients with hypertension can significantly improve following antihypertensive therapy (17-19). However, the impact

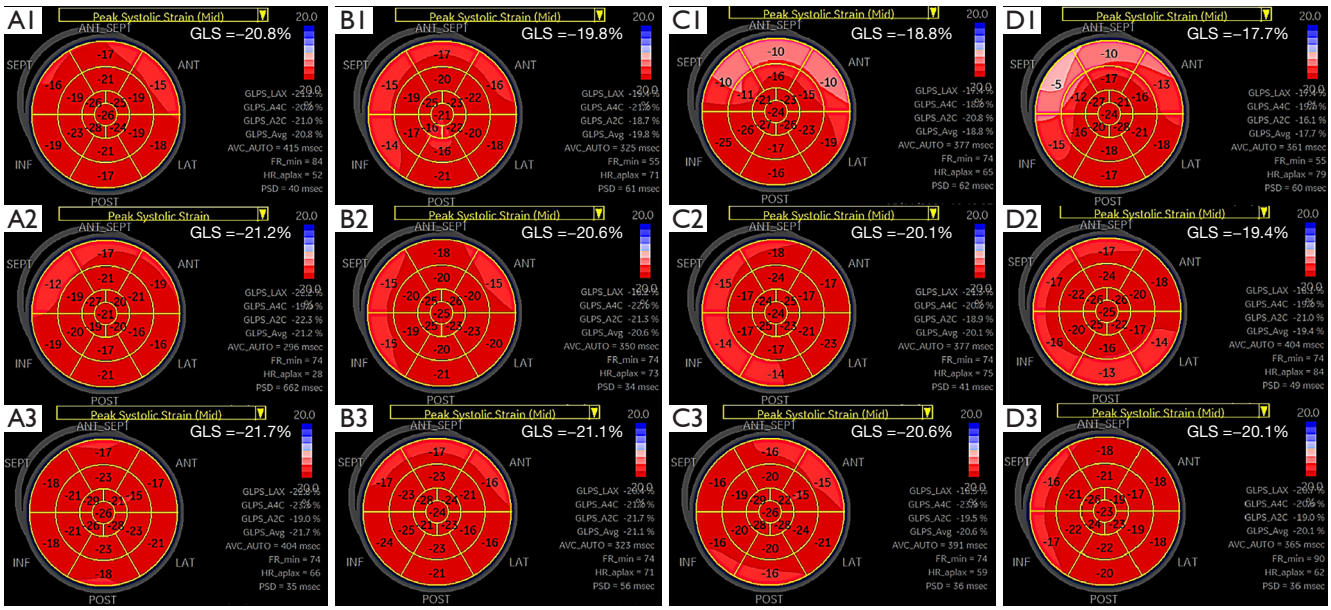

Figure 2 Bull's-eye diagrams of myocardial mechanics before and after blood pressure reduction in different geometry groups of elderly hypertensive patients. (A) Normal geometry group; (B) concentric remodeling group; (C) eccentric hypertrophy group; (D) concentric hypertrophy group. Dark red represents normal myocardial mechanics, while light color indicates impaired myocardial mechanics; the larger the light-colored area, the greater the degree of myocardial impairment. At baseline, from the normal geometry group to concentric remodeling, eccentric hypertrophy, and concentric hypertrophy groups, the light-colored area gradually increases, indicating a progressive worsening of myocardial mechanics; after blood pressure treatment, the light-colored area decreases and the dark red area increases, indicating an improvement in myocardial mechanics. 1: baseline; 2: after 12 months of blood pressure treatment; 3: after 24 months of blood pressure treatment. GLS, global longitudinal strain.

of antihypertensive therapy on myocardial mechanics in hypertensive patients with diverse LV geometry patterns remains unclear.

In this study, we delved into the effect of antihypertensive therapy on GLS in elderly patients who are hypertensive exhibiting different LV geometry patterns, using two-dimensional strain echocardiography.

The results indicate that GLS is more sensitive than LVEF in assessing left ventricular systolic function in hypertensive patients with different left ventricular geometric patterns. Elderly hypertensive patients with varying left ventricular geometry exhibit different degrees of myocardial mechanical impairment, with those in the EH and CH groups showing more significant damage. This suggests that abnormalities in the left ventricular geometry in hypertensive patients lead to a reduction in myocardial strain and overall cardiac function, and myocardial mechanical impairment is more severe in groups with left ventricular wall hypertrophy. The possible reasons are that hypertrophic myocardial cells and myocardial interstitial fibrosis jointly lead to a relative decrease in the density of myocardial arterioles and capillaries, insufficient myocardial cell perfusion, myocardial cell damage or even apoptosis, and a significant decrease in myocardial systolic function (12). The cross-sectional study by Tadic *et al.* on 211 hypertensive patients (including 74 NG patients, 53 CR patients, 46 EH patients, and 38 CH patients) showed that left ventricular GLS in patients with eccentric and concentric LVH was lower than that in patients with normal left ventricular geometry and CR, which is consistent with our research results (20). Studies (6,21,22) indicate that interstitial fibrosis is considered to be the main cause of GLS reduction. Poulsen *et al.* (23) found that GLS reduction following myocardial remodeling in hypertensive patients is also associated with exacerbated subendocardial ischemia. Additionally, interstitial fibrosis often increases left ventricular wall stiffness, decreases left ventricular compliance, and leads to elevated left ventricular filling pressures, resulting in diastolic dysfunction (6,21,22). This aligns with our study's results, which show significant

diastolic dysfunction in the CH and EH groups.

This study demonstrates that elderly hypertensive patients with abnormal LV geometry exhibit progressive improvement in GLS after 12 and 24 months of antihypertensive treatment. However, in the hypertensive NG group, there was no significant difference in GLS after 12 months of antihypertensive treatment compared to the baseline position, and GLS was greater than the baseline position after 24 months of antihypertensive treatment. This indicates that in hypertensive patients with abnormal left ventricular geometry, GLS continuously improves with the progress of antihypertensive treatment. While in those with normal left ventricular geometry, the degree of myocardial damage is relatively mild, and the change in GLS is not obvious after 12 months of antihypertensive treatment. This is mainly due to the fact that antihypertensive treatment improves myocardial cell hypertrophy and myocardial interstitial fibrosis, reverses left ventricular structural remodeling, improves myocardial microvascular circulation, reduces the degree of myocardial cell hypoxia, and improves myocardial mechanics (9). In our study, both the CH group and the EH group showed a progressive reduction in LV diameter and LVMI after 12 and 24 months of antihypertensive treatment. Wachtell *et al.* (24) observed in a cohort of 679 hypertensive patients with LVH that antihypertensive treatment over three years resulted in improved LV systolic function, which was inversely correlated with changes in LV mass. Additionally, in the current study, both the EH and CH groups exhibited significant improvements in diastolic function after 12 months of antihypertensive therapy.

Following antihypertensive treatment, myocardial interstitial fibrosis is reduced, LV wall stiffness decreases, LV compliance increases, and diastolic function improves correspondingly. Additionally, our study found that in both the NG group and the CR group, despite no significant changes in LV mass index or diastolic function parameters after 12 and 24 months of antihypertensive therapy, GLS showed improvement. This suggests that GLS recovery precedes other conventional echocardiographic parameters, indicating that GLS improvement may be one of the earliest markers of benefit from antihypertensive therapy.

Our findings demonstrate that elderly hypertensive patients with LV remodeling exhibit significant early improvement in LV myocardial mechanics following standardized antihypertensive treatment. It is hypothesized that the initial reduction in blood pressure during antihypertensive therapy leads to early improvement in myocardial mechanics. Consistent with our results, Motoki *et al.* (25) observed a significant enhancement in GLS after 3 months of antihypertensive therapy in hypertensive patients, with only minor changes in the subsequent 9 months.

Furthermore, our findings revealed that the ΔGLS between baseline and 12 months of antihypertensive therapy was highest in the CH group and lowest in the NG group, exhibiting a gradual decrease in ΔGLS in the order of CH, EH, CR, and NG groups. This observation may be linked to the baseline GLS in each group. Cheng *et al.* analyzed 182 patients who were hypertensive undergoing antihypertensive therapy for 6 months and found that those with lower baseline GLS experienced greater improvements in GLS after treatment, possibly associated with a reduction in LVMI (26). A meta-analysis by Tadic *et al.* on the effect of antihypertensive therapy on myocardial strain supported the notion that the degree of enhancement in GLS after antihypertensive therapy was independently correlated with the extent of reductions in LVMI, emphasizing the role of LV remodeling reversal in enhancing LV myocardial mechanics (9).

There are several limitations in this study. Firstly, some participants exhibited a prolonged duration of hypertension, and our study did not delve into the potential impact of the duration of hypertension on myocardial function. Exploring this aspect in future research could provide valuable insights. Secondly, different antihypertensive drugs exert their effects on the cardiovascular system through distinct mechanisms. Our study did not account for the potential influence of drug type on cardiac structure and function, highlighting the need for further investigations that consider and differentiate the effects of various antihypertensive medications. Thirdly, myocardial mechanics, in addition to being influenced by critical factors such as blood pressure and LVMI, may also be affected by other variables including smoking and arteriosclerosis. Therefore, there may be some degree of bias in the evaluation. Fourthly, the small sample size may limit the generalizability of our findings. To enhance the robustness of our results and facilitate long-term observations, future studies should consider expanding the sample size and incorporating extended follow-up periods.

Conclusions

In conclusion, the degree of myocardial mechanical impairment in elderly patients with hypertension varies depending on the left ventricular geometric pattern.

Following antihypertensive treatment, the extent of improvement also differs, with the CH group experiencing greater benefits. Recovery of GLS typically precedes improvements in other conventional echocardiographic parameters, establishing GLS as a crucial metric for assessing the efficacy of antihypertensive therapy.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Ethics Committee of Shanxi Bethune Hospital (No. YXLL-2020-045) and informed consent was obtained from all the patients.

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