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ORIGINAL ARTICLE

Blood pressure is normal, but is the heart?

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Abstract

Background There is no detailed strain analysis of cardiac functions in treated hypertensive pediatric patients. The aim of this study was to evaluate the cardio-protective effects of different drug classes in treated pediatric hypertensive patients.

Methods Sixty non-obese-treated hypertensive patients with preserved left ventricular (LV) systolic function and 45 age-, sex-, and body mass index-matched healthy subjects underwent clinical evaluation, including 24-h ambulatory blood pressure monitoring, standard echocardiographic examination, tissue Doppler imaging, and two-dimensional Speckle Tracking Echocardiography. The patients were divided into two subgroups based on the effects of the drugs on the Renin Angiotensin Aldosterone System. The subgroup hypertension (HT) 1 received angiotensin-converting enzyme inhibitor or angiotensin receptor blocker, and HT 2 subgroup received calcium channel blocker, β-blocker, or diuretics.

Results There was no difference between the two groups and subgroups with respect to clinical, demographic, ABPM, ventricular volumes, ejection fraction, and tissue Doppler imaging (TDI) parameters. For patients and controls, respectively, global longitudinal strain was -18.70 ± 3.41 versus -21.01 ± 3.82 ($P < 0.001$), and global radial strain was 40.6 ± 9.8 versus 54.8 ± 12.8 ($P = 0.004$). Peak LV twist and peak LV torsion were not significantly different. The patient subgroup analyses with each other revealed no difference in systolic and diastolic myocardial deformation properties.

Conclusions Strain parameters were reduced in all treated hypertensive children compared to normotensive children, and the various cardiac mechanic parameters were similarly abnormal no matter what type of antihypertensive agent was used.

Keywords Child · Hypertension · Antihypertensive drugs · Strain · Systolic function

Introduction

Hypertension (HT) is fast becoming a common medical condition in the pediatric population [1]. The prevalence of HT in children ranges from approximately 1 to 4.5% in the USA [2]. Longstanding HT leads to left ventricle hypertrophy (LVH) which has been proven to cause the irreversible deterioration of left ventricle (LV) function, ultimately resulting in congestive heart failure [3, 4]. However, ejection fraction (EF) and fractional shortening (FS) often remain preserved until late in the course of the disease, making subtle changes in LV contractile function difficult to interpret in the early stages [4].

Tissue Doppler imaging (TDI) and Speckle Tracking Echocardiography (STE) are relatively new echocardiographic techniques which are useful in assessing the early changes in regional and global systolic and diastolic myocardial function in children [5]. Unlike TDI, a principal advantage of STE is that it is not dependent on the ultrasound wave angle and compares closely with results obtained by MRI tagging [6].

In children, information on the effects of blood pressure (BP)-lowering therapy on cardiac end-organ damage is mostly limited to uncontrolled studies in heterogeneous populations with primary and secondary HT [7]. This study aimed to measure LV strain, twist, and torsion in treated primary HT patients to evaluate the cardiac end-organ protective effects of different antihypertensive drug classes.

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Methods

Study sample

The study group included 60 consecutive patients aged < 18 years of age with a diagnosis of primary arterial HT who were selected at the Pediatric Nephrology Clinic of University Hospital. Hypertension was defined as average systolic (SBP) or diastolic BP (DBP) ≥ 95th percentile using the latest guideline [7] and treated for primary HT for at least 6 months. We excluded patients with secondary HT, "white coat" arterial HT, or hypertrophy of the myocardium caused by HT; treated with combination antihypertensive medication; treated with steroids or cardiodepressants; and patients diagnosed with endocrinological or metabolic disorders. All patients had a normal renal function (estimated glomerular filtration rate ≥ 90 ml/min) [8]. Healthy normotensive children were recruited from the local population. They were referred to our hospital because of cardiac murmur and underwent electrocardiography with a negative medical history and no signs or symptoms of acute or chronic disease. All participants in the control group were examined by the same pediatrician, and the results of physical examination were normal.

Clinical and laboratory characteristics, including age, gender, weight, height, body mass index (BMI), SBP and DBP (office and ambulatory), mean arterial pressure (MAP), heart rate, and SBP and DBP load were recorded. We investigated the use of different antihypertensive medications on cardiac effects. The patients were divided into two subgroups based on the effects of the drugs on the Renin Angiotensin Aldosterone System. HT 1 subgroup received angiotensin-converting enzyme inhibitor (ACEI), or angiotensin receptor blocker (ARB), and HT 2 subgroup received calcium channel blocker (CCB), β-blocker, or diuretics.

Blood pressure

Office blood pressure measurements

Systolic blood pressure and DBP were measured three times at intervals of 1–2 min using an oscillometric device (Riester Ri-Champion model, Germany) with the cuff covering two thirds of the upper arm and the bladder encircling > 80 and < 100% of the upper arm circumference. Blood pressure value was the average of the last two measurements. Office hypertensive values were defined as SBP or DBP values ≥ 95th percentile for age, height, and sex, and office normotensive values were defined as SBP or DBP values < 95th percentile for age, height, and sex.

Ambulatory blood pressure monitoring

The ambulatory blood pressure monitoring (ABPM) was set to record BP and pulse rate in 20-min intervals during the day and 30-min intervals during the night. The mean values of SBP, DBP, and the MAP for 24 h, day and night, were calculated using a licensed ABPM program (Mobil O'Graph NG) oscillometric devices (Numed Healthcare, Sheffield, UK). The ABPM profile was determined on the basis of the mean daytime, nighttime, and 24-h BP values and adjusted to the sleeping patterns and activities while awake in the diaries of each child. HT was defined as SBP or DBP values equal to or exceeding the 95th percentile for sex, age, and height in 24 h, day and night [7]. Nevertheless, in order to compare the results with the normative values for ABPM, SBP, and DBP, values were converted into SDS values using the most recent normative values [9]. A nondipping profile was defined using ABPM reference data as a nocturnal BP decrease of less than 10% compared with average daytime BP values [7].

Standard two-dimensional and Doppler chocardiography

The patient and control group were evaluated with 2D and color-coded conventional transthoracic echocardiography by the same pediatric cardiologist using the same echocardiography machine (Vivid E9, GE Healthcare, Norway) in standard precordial positions [10]. The studies were stored (EchoPAC software products 12.1; GE Vingmed Ultrasound AS) and digital images analyzed offline by an author who was blinded to the medical diagnosis, BP, and medical therapy. LV volumes and left ventricular ejection fraction (LVEF) were measured using the Simpson method. LV mass (LVM) was calculated using the formula that was proposed by Devereux et al. [11]. The left ventricular mass index (LVMI) was calculated by dividing the LVM (g) by the height in meters. Left ventricular hypertrophy (LVH) was defined as LVMI above 51 g/height$^{2.7}$ [7]. LV filling patterns were evaluated on the apical four-chamber view by pulsed-wave Doppler echocardiography with the sample volume located between the tips of the mitral valve leaflets during diastole. Early diastolic flow (E-wave), late diastolic flow (A-wave) velocities, and the E/A ratio were calculated from the recordings, and the deceleration time of the E-wave was measured. Early diastolic (E'-wave), late diastolic (A'-wave), and systolic (S-wave) velocities were measured at the lateral parts of the mitral annuli on the apical four-chamber views. For TDI parameters, z scores were calculated from the data of Eidem [12].

2D speckle tracking, twist, and torsion analysis

Gray images were obtained from apical four-chamber (A4C), three-chamber (A3C), two-chamber (A2C), and parasternal short-axis (level of the papillary muscle) views based on the recommendations of the American Society of Echocardiography [13]. All the images which were obtained at the left lateral decubitus position and under electrocardiogram (ECG) monitoring were stored for offline analysis. The endo-myocardial borders of the LV were marked manually at the end of systole. Epicardial marking was performed automatically by the computer. Tracking accuracy was verified in real time and corrected by adjusting the region of interest or by manually correcting the contour to ensure optimal tracking. Longitudinal, transverse, and radial strain and strain rates were assessed from six basal and six midventricular segments of the LV including apical, mid, basal segments at the four-chamber; two-chamber and three-chamber view of the LV; and anterior, septal, and inferior segments at the short-axis view of the LV. Peak basal and apical rotation, peak LV twist, and peak LV torsion were automatically calculated.

Statistical analysis

Numerical values are expressed as mean ± standard deviation, and categorical data are given as percentages. SPSS 22.0 was used for the statistical analysis. The Kolmogorov-Smirnov test was used to determine whether the data were distributed normally. The unpaired Student's *t* test was used to compare the means of normally distributed data, while the Mann-Whitney *U* test was used for non-normal distributions. Correlations among the quantitative data were analyzed using Pearson's correlation test. The results are given with the 95% confidence interval (CI) and were considered significant at $p < 0.05$. Comparison of variables between the HT 1 and HT 2 subgroups was performed by using the unpaired *t* test. Multivariable linear regression analyses were performed to assess the relationships between independent variables, including MAP, antihypertensive agent, age, BMI, and LVM with dependent variables, including strain, twist, and torsion measures.

Results

Clinical characteristics, office BP, and ABPM

Among the initial sample of 80 patients, 4 were excluded because of technically inadequate ABPM or STE recordings; 16 patients had uncontrolled HT or LVH (Fig. 1). The study included 60 patients, of whom 33 (55%) were girls. The median duration of time since initiation of drug therapy was 3.5 ± 1.2 years. Clinical, demographic, and the mean results of office BP and ABPM measurements of treated patients and controls are listed in Table 1. There were no statistically significant differences between the two groups with respect to age, sex, height, weight, and BMI. The mean office SBP and nondipper rate levels were not significantly different in patients compared with controls ($p = 0.21$). The HT 1 and HT 2 subgroup analyses revealed no differences in clinical or demographic data, or the mean results of office BP and ABPM measurements (Table 2).

Standard two-dimensional and Doppler findings

No structural heart disease was detected. LV volumes and EF, normalized to body surface area, did not differ significantly from those of controls. Also, no difference was found in LV diastolic function evaluated by DT (ms), E' (m/s) A'(m/s), E'/

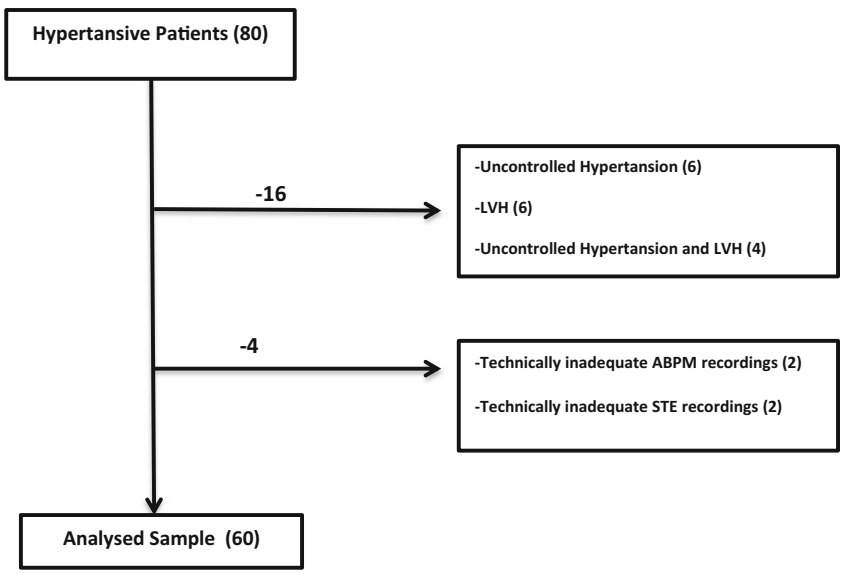

Fig. 1 Patient selection flow chart. The final study population consisted of selected patients with normal blood pressure that had been confirmed by ABPM and who were scanned by standard echocardiography. *LVH* left ventricular hypertrophy, *ABPM* ambulatory blood pressure monitoring *STE* speckle tracking echocardiography

Table 1 Demographic characteristics of the patients and control groups

	Patients ($n = 60$)	Control ($n = 45$)	P value
Age (years)	12.3 ± 3.4	12.8 ± 3.9	0.79
Gender (M: male. F: female)	33M/27F	28M/17F	0.46
Height (cm)	151.6 ± 15	153 ± 16.3	0.44
Weight (kg)	36.4 ± 5.1	37.5 ± 4.5	0.42
BMI (kg/m^2)	22.5 ± 3.1	21.3 ± 2.9	0.40
İnitiation of drug therapy (years)	3.5 ± 1.2	–	–
Office SBP (mmHg)	112 ± 11.2	110.5 ± 4.3	0.35
Office DBP (mmHg)	58.5 ± 3.9	56.9 ± 1.4	0.38
24-h SBP (mmHg)	118 ± 11.4	109.5 ± 9.8	0.32
24-h DBP (mmHg)	66 ± 5.9	64 ± 4.1	0.12
Daytime SBP (mmHg)	122 ± 7.1	113 ± 4.2	0.10
Nighttime SBP (mmHg)	110 ± 4.3	101 ± 3.9	0.06
Daytime DBP (mmHg)	70 ± 6.5	68 ± 4.8	0.25
Nighttime DBP (mmHg)	62 ± 3.4	58 ± 3.0	0.12
24-h SBP load (%) (median)	18 (10–34)	16 (9–34)	0.35
Dipper (n) (%)	38(63%)	30 (66%)	0.26
Nondipper (n) (%)	22(37%)	15(33%)	0.21
Antihypertensive medication, n (%)			
ACEI	12 (20)	–	–
ARB	18 (30)	–	–
β-blocker	16 (26)	–	–
CCB	9 (15)	–	–
Diuretic	5 (9)	–	–

BMI body mass index, *SDP* systolic blood pressure, *DBP* diastolic blood pressure, *ACEI* angiotensin-converting enzyme inhibitor, *ARB* angiotensin receptor blocker, *CCB* calcium channel blocker

A', E/E', Tei index, E/A ratio, and E/E' ratio between the groups (Table 3). There were no statistically significant differences between the subgroups according to antihypertensive therapy, standard echocardiographic and TDI parameters, and dipper and non-dipper profiles.

STE during systole

In comparison with the normal controls, treated patients with HT showed significantly lower values for global longitudinal systolic strain (GLS) and global strain rate (GSR) and for radial strain and strain rate values of the LV (Table 4). There was no statistically significant difference between the subgroups according to antihypertensive therapy (Table 5, Fig. 2). The dipper, non-dipper profile analyses revealed no differences in systolic myocardial deformation properties.

Twist and torsion

LV twist and torsion showed no statistically significant differences between the two groups and two subgroups.

Table 2 Demographic characteristics of the subgroups

	HT 1 ($n = 30$)	HT 2 ($n = 30$)	P value
Age (years)	12.4 ± 3.8	12.6 ± 2.8	0.80
Gender (M: male. F: female)	17M/13F	16M/14F	0.64
Height (cm)	152.4 ± 12	150 ± 11.1	0.40
Weight (kg)	37.4 ± 4.8	35.5 ± 2.5	0.54
BMI (kg/m^2)	22.6 ± 2.5	21.9 ± 2.2	0.45
İnitiation of drug therapy (years)	3.2 ± 1.4	3.6 ± 1.2	0.22
Office SBP (mmHg)	114 ± 11.2	106.5 ± 8.1	0.65
Office DBP (mmHg)	61.5 ± 3.9	58.6 ± 2.5	0.28
24-h SBP (mmHg)	120 ± 12.6	117.6 ± 11.2	0.38
24-h DBP (mmHg)	68 ± 7.2	62 ± 5.4	0.24
Daytime SBP (mmHg)	124 ± 9.3	123 ± 4.6	0.12
Nighttime SBP (mmHg)	112 ± 4.8	108 ± 4	0.46
Daytime DBP (mmHg)	70 ± 8.5	70 ± 2.4	0.25
Nighttime DBP (mmHg)	64 ± 3.8	60 ± 3.2	0.52
Dipper (n) (%)	10 (33%)	8 (26%)	0.16
Nondipper (n) (%)	20 (67%)	22 (74%)	0.18

HT 1 subgroup: received angiotensin-converting enzyme inhibitor, or angiotensin receptor blocker; HT 2 subgroup: received calcium channel blocker, β-blocker, or diuretics

BMI body mass index, *SBP* systolic blood pressure, *DBP*diastolic blood pressure

Discussion

In this study, we demonstrated reductions in strain parameters in treated hypertensive children compared to normotensive children, and the various cardiac mechanical parameters were similarly abnormal no matter what type of antihypertensive agent was used.

Tissue Doppler imaging has been used as a predictor for cardiovascular risk monitoring of raised LV diastolic pressure [23]. In children, Zamojska et al. [14] and Agu et al. [15] showed that in untreated and newly diagnosed hypertensive children, TDI velocities for E' and A' are significantly lower, and E/E' ratio is increased compared with controls. The present data may seem to be in contrast with the described "early diastolic impairment" in this population. This may be explained on the basis of the restrictive inclusion criteria in our study, because relatively younger patients were included with short durations of HT and with under-control HT. Clinical trials have shown that in patients with HT and echocardiographic evidence of dysfunction, BP-lowering with antihypertensive therapy leads to improved diastolic function according to TDI and conventional echocardiography [10, 16]. However, we did not detect this effect on systolic functions according to STE. The relationship between the longitudinal GLS and GSR has been demonstrated in hypertensive adult studies. Mizuguchi et al. [17] and Atilgan et al. [18] found in their study that the patients with

Table 3 M-mode echocardiographic measurements, pulsed-wave, and tissue Doppler results of study groups

	Patients ($n = 60$)	Control ($n = 45$)	P value
EF (%)	70.6 ± 7.4	72.1 ± 9.3	0.87
FS (%)	39.8 ± 6.5	41.2 ± 8.6	0.78
IVSd (mm/m^2)	6.3 ± 1.2	6.8 ± 1.8	0.13
LVDs (mm/m^2)	19.8 ± 2.3	20.2 ± 2.6	0.92
LVDd (mm/m^2)	27 ± 4.1	25 ± 4.6	0.35
LPWD(mm/m^2)	5.6 ± 1.7	5.9 ± 1.8	0.48
LVM (g)	58.20 ± 9.05	56.90 ± 7.05	0.40
LVMI, g/m$^{2.7}$	42.70 ± 8.21	40.14 ± 7.60	0.36
E (m/s)	0.97 ± 0.14	0.98 ± 0.3	0.16
A (m/s)	0.60 ± 0.13	0.59 ± 0.12	0.58
E/A	1.58 ± 0.43	1.61 ± 0.46	0.33
DT(ms)	141.5 ± 30.6	135.4 ± 34.81	0.44
E' (m/s)	0.16 ± 0.03	0.18 ± 0.03	0.26
A'(m/s)	0.81 ± 0.01	0.76 ± 0.01	0.34
E'/A'	1.97 ± 0.61	1.85 ± 0.54	0.40
E/E'	5.3 ± 1.1	5.1 ± 0.9	0.54
Tei index	0.57 ± 0.13	0.56 ± 0.67	0.40

EF ejection fraction, *FS* fractional shortening, *IVSd* end-diastolic interventricular septum, *LVIDd* end-diastolic left ventricular internal diameter, *LVPWd* end-diastolic left ventricular posterior wall, *LVM*left ventricular mass, *LVMI* left ventricular mass index, *E* early mitral inflow (E), *A* late mitral inflow, *E/A* mitral E/A ratio, *DT* mitral deceleration time, *E'* early diastolic velocity, *A'* late diastolic velocity, *E'/A'* mitral E'/A' ratio, *E/E'* mitral E/E' ratio, *MPI* myocardial performance index

arterial HT had reduced LV longitudinal deformation, which is consistent with our findings. Correspondingly, Chen et al. found that despite normal EF, both controlled and uncontrolled BP groups displayed an overall reduction in the 2D-STE parameters when compared with the control group, and the changes were more obvious in the uncontrolled BP group regardless of LVH [19]. There is only one study in pediatric HT patients using STE. Navarini et al. found decreased strain values compared with healthy subjects [20]. Unlike us, they included new and pretreatment patients together in the patient group.

In adult hypertensive patients, there is a study showing that LV twist and torsion are preserved in high-normal BP subjects and significantly reduced in hypertensive patients [21]. In our study population, twist and torsion were not different in treated patients compared with controls. The potential explanations in our patient population could be that HT had not continued to have an impact on the myocardium to impair LV twist and torsion or the disease state had not been present for sufficiently long to have impaired LV twist and torsion.

Decreased 2D-STE values might be explained by the presence of irreversible regional subendocardial myocardial ischemia and increased perivascular and interstitial fibrosis [22]. It is also possible that the level of end-systolic wall stress is different between the groups, affecting the subendocardial fiber shortening. These pathophysiologic changes are likely to lead to decreased longitudinal systolic contraction.

In the subgroup analysis, the absence of differences in cardioprotective effects among the antihypertensive drugs suggests that they are not superior to each other. In children, information about the effects of antihypertensive treatment on cardiac and end-organ damage is mostly limited to uncontrolled studies. A recent review identified a total of 3454 children with comparing antihypertensive agents as either monotherapy or combination therapy and different doses of the same medication. No studies to date have investigated the efficacy of prevention of end-organ damage [23]. A review about the effects of antihypertensive therapy in adults has

Table 4 Comparison of left ventricular global strain and strain rate values according to views of echocardiography

		Patients ($n = 60$)	Control ($n = 45$)	P value
Global longitudinal	GS (%)	− 18.70 ± 3.41	− 21.01 ± 3.82	< 0.001
	SR(s^{-1})	− 1.16 ± 0.24	− 1.35 ± 0.39	< 0.001
Global longitudinal four-chamber	GS (%)	− 17.88 ± 2.67	− 20.77 ± 1.77	< 0.001
	SR(s^{-1})	− 1.04 ± 0.18	− 1.29 ± 0.34	< 0.001
Global longitudinal three-chamber	GS (%)	− 19.07 ± 3.07	− 21.01 ± 2.64	< 0.001
	SR(s^{-1})	− 1.13 ± 0.19	− 1.30 ± 0.32	< 0.001
Global longitudinal two-chamber	GS (%)	− 20.38 ± 3.13	− 22.42 ± 3.83	< 0.001
	SR(s^{-1})	− 1.19 ± 0.22	− 1.38 ± 0.30	< 0.001
Global circumferential	GS (%)	− 17.55 ± 4.08	− 17.30 ± 3.44	0.19
	SR(s^{-1})	− 1.27 ± 0.29	− 1.14 ± 0.21	0.36
Global radial	GS (%)	40.6 ± 9.8	54.8 ± 12.8	0.004
	SR(s^{-1})	1.18 ± 0.41	1.49 ± 0.54	0.003

Values are expressed as mean ± standard deviation

GS strain, *SR* strain rate

Table 5 Comparison of left ventricle global strain values according to subgroups

		HT 1 ($n = 30$)	HT 2 ($n = 30$)	P value
Global longitudinal	GS (%)	− 18.60 ± 2.31	− 18.90 ± 3.01	0.44
	SR(s^{-1})	− 1.11 ± 0.18	− 1.19 ± 0.30	0.40
Global longitudinal four-chamber	GS (%)	− 17.70 ± 2.08	− 18.02 ± 2.88	0.34
	SR(s^{-1})	− 0.94 ± 0.11	− 1.08 ± 0.22	0.38
Global longitudinal three-chamber	GS (%)	− 18.84 ± 2.15	− 19.27 ± 3.25	0.24
	SR(s^{-1})	− 1.09 ± 0.16	− 1.16 ± 0.24	0.22
Global longitudinal two-chamber	GS (%)	− 19.92 ± 3.31	− 20.98 ± 3.43	0.25
	SR(s^{-1})	− 1.14 ± 0.18	− 1.21 ± 0.25	0.28
Global circumferential	GS (%)	− 17.94 ± 4.11	− 17.07 ± 3.88	0.69
	SR(s^{-1})	− 1.29 ± 0.32	− 1.21 ± 0.24	0.66
Global radial	GS (%)	39.1 ± 9.1	40.9 ± 9.9	0.48
	SR(s^{-1}	1.15 ± 0.37	1.20 ± 0.45	0.42

Values are expressed as mean ± standard deviation. HTN 1 subgroup: received angiotensin-converting enzyme inhibitor, or angiotensin receptor blocker; HTN 2 subgroup: received calcium channel blocker, β-blocker, or diuretics

GS strain, *SR* strain rate

showed that antihypertensive drugs have not reduced cardiovascular mortality and morbidity [24].

Ambulatory blood pressure monitoring is considered superior to standard BP measurement in the evaluation of target organ damage related to HT [3]. On the contrary, there are also studies in the literature on the inadequacy of ABPM in evaluating target organ damage. A recent study claimed that ABPM parameters had no significant correlation with LVH or diastolic functions in children and adolescents with primary HT [25]. Also, the clinical interpretation of 24-h ABPM is assessed according to reference values. These data derived from different sources may vary according to different incomes, population, country, and years. Furthermore, ABPM values may not always reflect BP because many patients cannot move freely during the day and cannot sleep comfortably at night. In conclusion, it is difficult to assess the target organ damage by ABPM parameters alone, suggesting that there are many factors that need to be investigated.

It currently remains unclear whether or not cardiac changes can be reversible by adequate BP control, but considering the extent of the observed changes in our study, we consider it rather unlikely. Nevertheless, in order to answer this question, STE analyses on a large patient cohort with isolated arterial HT are necessary.

There are several limitations to this study. The present study is a single-center observational study. It is limited by the relatively small sample size. The precision of our BP readings may have been affected by a limited choice of BP cuffs provided by the Riester Ri-Champion device. However, this device has previously been used in children by Miranda and colleagues [26]. In addition, children older than 5 years who did not require special small BP cuffs were included in the study. Blood pressure values were assessed using centile charts [7]. However, there may be differences in DBP because of the measurement method [27].

Cardiac strain analyses of patients before drug treatment were not performed. We could not provide any markers of myocardial fibrosis, such as serum carboxy-terminal propeptide of procollagen type I.

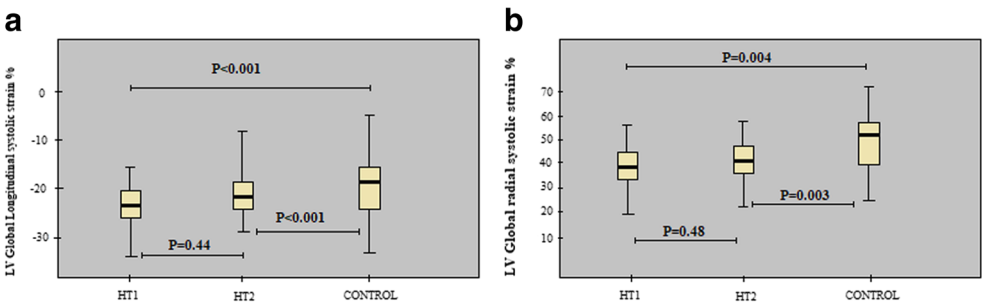

Fig. 2 **a** Comparison of left ventricle global strain values according to subgroups. **b** Comparison of left ventricle global radial strain values according to subgroups. HT 1: received angiotensin-converting enzyme inhibitor or angiotensin receptor blocker; HT 2: received calcium channel blocker, β-blocker, or diuretics

Conclusion

Left ventricular systolic functions were found to be impaired in treated HT children suggesting that BP lowering with current antihypertensive therapies did not reverse or prevent from the progression of LV remodeling. The assessment of LV contraction by 2D-STE can give new insight into myocardial function in HT that might improve pathophysiologic understanding and identify patients at risk who could need a more aggressive antihypertensive treatment program.

Compliance with ethical standards

The study was conducted in accordance with the Declaration of Helsinki and was approved by the local ethics committee. The detailed consent forms were signed by the parents of all subjects before participating in the study.

Conflict of interests The authors declare that there is no conflict of interests regarding the publication of this paper.

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