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Comparison of Left Atrial Function between Hypertensive Patients with Normal Atrial Size and Normotensive Subjects Using Strain Rate Imaging Technique

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Background: Patients with hypertension are at risk of structural and functional changes in the left atrium (LA). There are only a few studies on the impact of hypertension on LA function, especially in hypertensive patients with a normal LA size. We, therefore, designed this study to evaluate LA function in patients with a normal LA size via deformation imaging.

Objectives: We assessed regional longitudinal strain rate imaging (SRI) profiles along with tissue velocity imaging (TVI) in the LA walls to quantify LA reservoir function and explore changes in LA function in hypertensive patients with a normal value of LA size.

Patients and Methods: One hundred twenty-four subjects with normal angiography (mean age = 56.28 ± 8.91 years, 46% male), who were referred to the Echocardiography Laboratory of our institution, were enrolled in this study. These subjects were categorized into two groups: hypertensive (75 cases) and age-matched normotensive (49 cases) groups. All the cases of the patient and control groups had a normal LA size. SRI parameters included strain (ST, %) and strain rate (SR, s^{-1}), and tissue imaging parameters such as peak systolic velocity (Sm, m/s) were measured in four septal, lateral, anterior, and inferior LA walls at the mid-level.

Results: Compared with the controls, the patients with a history of hypertension showed significantly lower values of Sm, ST, and SR in each segment of the LA. There was no effect of age on these indices. Also, no differences regarding Sm, ST, and SR were found between the septal, lateral, anterior, and inferior LA walls in each group. By multivariate linear regression analysis, a history of hypertension was the only independent determinant of average LA strain rate in the all the individuals ($P < 0.001$). When this analysis was repeated in the patients with a history of hypertension, the only independent determinant of average LA strain rate was heart rate ($P = 0.026$).

Conclusions: In our subjects, with a normal value of LA size, the effect of hypertension on LA reservoir function was independent of age, sex, heart rate, left ventricular mass index, and left ventricular ejection fraction. Additionally, heart rate independently correlated with reduced TVI and SRI parameters in the patients with hypertension.

*Keywords:*Heart Atria; Echocardiography; Hypertension

1. Background

The left atrium (LA) makes a considerable contribution to cardiac performance and accomplishes its role through three phases in which it acts first as a reservoir during ventricular systole, second as a conduit during early ventricular diastole, and third as an active contractile chamber during late ventricular diastole (1). Hypertension alters atrial dynamics significantly, with resultant increased LA volume and active emptying volume consequent to altered LV diastolic function (2). Patients with hypertension are at risk of structural and functional changes in the LA. Conventional methods for the assessment of LA function included blood flow velocity during atrial contraction, peak mitral inflow A wave velocity and its velocity time integral, and atrial emptying fraction (3, 4); nevertheless, none of these measures has been established. Since the introduction of tissue Doppler imaging (TDI), echocardiography has been able to noninvasively assess regional myocardial function. Overall heart motion, tethering effect, and cardiac rotation have influenced the accuracy of TDI in the assessment of regional function in the cardiac chambers (5-7).

In recent years, strain rate imaging (SRI) has been shown to be an accurate method for quantifying regional myocardial function independent of cardiac rotation and tethering effect. SR indices have been calculated as the difference in tissue Doppler velocities between two different myocardial regions, showing the rate by which deformation occurs (6, 8, 9). By this technique, although

Implication for health policy makers/practice/research/medical education:
To detect echocardiographic factors enabling the detection of left atrial dysfunction in hypertensive patients before the manifestation of left atrial enlargement and left ventricular hypertrophy.

not as frequently as studies on the left ventricle (LV), some studies have sought to evaluate LA function in normal subjects (10) and in patients with atrial fibrillation (6, 11, 12). Meanwhile, a few studies have focused on quantifying LA function in hypertensive patients (12, 13). Some investigations have pointed out that SRI enables the detection of LA dysfunction in hypertensive patients before the manifestation of LA enlargement and LV hypertension (13, 14).

2. Objectives

In the present study, we aimed to assess regional longitudinal strain/SR profiles along with tissue velocity imaging (TVI) in the LA walls to quantify LA function and explore changes in LA function in hypertensive patients with a normal value of LA size.

3. Patients and Methods

One hundred twenty-four subjects (mean age = 56.28 ± 8.91 years, 46% male), who were referred to our echocardiography laboratory, were enrolled in this study. All the patients had a normal LA size in echocardiography based on the American Society of Echocardiography guideline (15). These subjects were categorized into two groups: group 1, patients with a history of hypertension and group two, normotensive cases. Group 1 consisted of 75 patients with essential hypertension who had normal coronary arteries proved by coronary angiography.

A history of hypertension was defined as having a blood pressure > 140/90 mmHg or taking antihypertensive medication. Group 2 included 49 age-matched subjects with no history of hypertension. The exclusion criteria for both groups were comprised of LV ejection fraction (EF) < 50%, LA enlargement based on the ASE recommendations, any history or evidence of ischemic heart disease by surface electrocardiography, exercise stress test, coronary angiography, or regional wall motion abnormality on transthoracic echocardiography, rhythm other than sinus rhythm, and higher than grade one of diastolic dysfunction. Required data for the study population was prospectively gathered through interview as well as clinical and echocardiographic evaluation. Blood pressure was measured prior to echocardiography. The heights and weights of all the subjects were recorded. The body mass index (BMI) was calculated as the weight (kg)/height $(m)^2$. Our institutional Review Board approved the protocol of this study.

3.1. Echocardiographic Measurements

Two-dimensional echocardiography was performed using a Vivid 7 (GE, Vingmed, Horten, Norway) with a 3.5-MHz probe in the subjects, lying in the left decubitus position, by a single experienced echocardiologist. All the echocardiographic evaluations were performed using previously published guidelines (15). Left ventricular end-diastolic diameter (LVID), internal dimension at the end of diastole, posterior wall thickness (PWT), and interventricular septal thickness (IVST) were measured. The LV mass was calculated according to the Devereux formula (16) and was normalized for the body surface area (BSA) (17). Left ventricular hypertrophy (LVH) was defined as an LV mass index of > 131 g/m^2 for the men and >100 g/m^2 for the women. LA volume was estimated using the formula (15): $8/3\pi$ (A1 × A2/L), where A1 and A2 represent the maximal planimetered LA area acquired from the apical four and two-chamber views, respectively, and L is length.

3.2. Tissue Velocity and Strain Rate Imaging in the Left Atrium

Event timing from conventional Doppler was performed by using installed software on the echo machine. Atrial Doppler-derived strain and strain rate signals were reconstituted by placing an appropriate size sample volume at the mid-level of the LA walls. Tissue velocity and SR images were recorded using apical four, there, and two-chamber views. The frame rate was set at > 100 frames/sec. Peak systolic velocity (Sm, m/s), strain (ST, %), and SR (s^{-1}) were measured by positioning of sample volume on each mid-segment of the LA septal, lateral, anterior, and inferior walls. Peak strain was measured from these points during ventricular end-systole as a criterion of LA reservoir compliance (Figure 1). One experienced echocardiography specialist analyzed all the echocardiography records. Intra-observer variability for TDI parameters in LA was published in our previous study (18). Intra-observer variability for TDI parameters (ST and SR) was calculated via the formula: [(first observation – second observation)/first observation] and was 6.95 ± 5.19%.

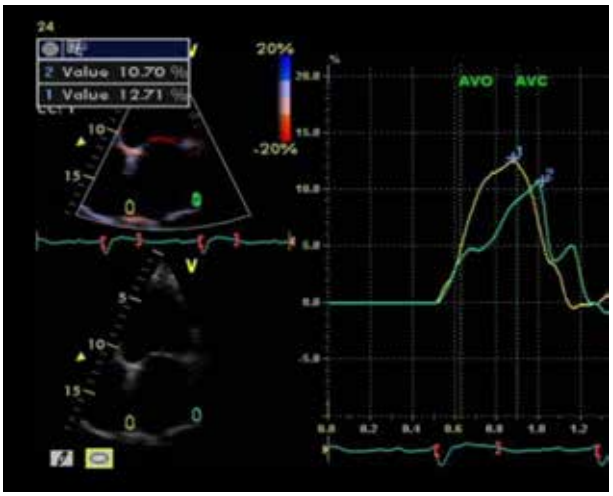

Figure 1. Measuring Points (+1 and +2 indicated by arrows) Show the Peak Strain of the Left Atrium on the Left Atrial Walls at End Systole-Early Diastole of the Left Ventricle and Are Considered as Left Atrial Reservoir

3.3. Statistical Analysis

The results are presented as mean ± standard deviation for the numerical variables or by absolute frequencies and percentages for the categorical variables. The continuous variables were compared using the Student *t*-test or the nonparametric Mann-Whitney U test whenever the data did not appear to have normal distributions. The categorical variables were compared using the chi-squared test or the Fisher exact test, as required. The mean values of the SRI parameters were compared between the different LA walls using the one-way ANOVA test, and pairwise multiple comparisons were done using the Bonferroni test for each pair of the means. All the p values were 2-tailed, with statistical significance defined by a P value ≤ 0.05.

A linear regression analysis employing patient-related characteristics was performed to examine the factors determining the SR of the LA. This analysis was applied once in all the 124 study subjects, and the second regression analysis was performed on the 75 hypertensive patients. Variables with a P value < 0.10 in univariate analysis were entered into the multivariate model.

4. Results

4.1. Clinical Characteristics and Echocardiographic Findings

The general characteristics and echocardiographic findings of the study groups are summarized in Table 1. There were no significant differences in terms of age and heart rate between the hypertensive and normal groups. The frequency of the male individuals was greater in the hypertensive group than in the normotensive group (73.5% vs. 29.3%, respectively). According to Table 1, the hypertensive group had remarkably higher systolic and diastolic blood pressure, larger LA dimension, LA volume, and surface area than did the controls. (All the study subjects had normal LA dimensions.) The hypertensive patients also had significantly lower LVEF and higher LV mass index. There were no differences between the two groups in heart rate, LV dimension, and pulmonary artery pressure.

Table 1. Clinical Characteristics and Echocardiographic Parameters of the Study Groups

	Control Group (n = 49)	Hypertensive Group (n = 75)	P Value
Demographics, Mean ± SD			
Mean age	54.88 ± 8.27	57.22 ± 9.25	0.158
Heart rate, min	72.63 ± 15.92	74.79 ± 11.47	0.448
Body surface area, m^2	1.87 ± 0.20	1.83 ± 0.19	0.232
Body mass index, Kg/ m^2	25.74 ± 3.97	28.05 ± 3.68	0.001
SBP [a], mmHg	114.59 ± 7.35	135.33 ± 19.60	< 0.001
DBP [a], mmHg	79.08 ± 2.21	85.39 ± 8.47	< 0.001
Risk Factors, No. (%)			
Diabetes mellitus	2 (4.1)	17 (22.7)	0.005
Current cigarette smoking	9 (18.4)	7 (9.3)	0.142
Hyperlipidemia	1 (2)	38 (50.7)	< 0.001
Medication, %			
β blockers	0	73.3	-
ACE [a] inhibitors	0	29.3	-
ARB [a]	0	12	-
calcium antagonists	0	10.6	-
Echocardiographic Findings, Mean ± SD			
LA [a] anterior to posterior wall diameter, mm	31.94 ± 3.46	34.36 ± 2.96	<0.001
LA septal to lateral wall diameter, mm	31.62 ± 3.12	34.03 ± 3.44	<0.001
LA Volume, cc	37.60 ± 10.11	45.80 ± 11.11	<0.001
IVS [a] thickness, mm	9.04 ± 1.24	11.62 ± 1.64	< 0.001
LVPW [a] thickness, mm	8.82 ± 1.19	11.21 ± 1.31	< 0.001
LA surface area, cm^2	15.04 ± 2.52	17.04 ± 3.52	0.001
LV[a] end-diastolic diameter, mm	45.35 ± 4.20	44.29 ± 4.49	0.201
LV end-systolic diameter, mm	33.49 ± 4.79	32.28 ± 4.71	0.192
Pulmonary artery pressure, mm	28.5 ± 0.71	29.95 ± 4.04	0.623
LV mass, g	130.28 ± 32.91	174.60 ± 39.09	< 0.001
LV mass index, g/m^2	69.47 ± 15.64	97.01 ± 20.45	< 0.001
LV hypertrophy, No. (%)	0	22 (29.3)	< 0.001
LV ejection fraction (%)	57.45 ± 2.47	56.00 ± 2.01	0.001

[a] Abbreviations: ACE, angiotensin-converting enzyme inhibitors; ARB, angiotensin II receptor blockers; DBP, diastolic blood pressure; IVS, interventricular system; LA, left atrium; LV, left ventricle; LVPW, left ventricular posterior wall; SBP, systolic blood pressure

4.2. Comparison of Tissue Velocity, Strain, and Strain Rate Parameters

The TV, strain, and SR parameters are presented in Table 2. Compared with the controls, the patients with a history of hypertension showed significantly lower values of Sm, ST, and SR in each segment of the LA (Table 2). This comparison was performed in the male and female individuals separately, and there was no effect of sex on the difference between the two groups in relation to the mentioned parameters. The possibility of the existence of any difference in the four LA walls was evaluated via a comparison between the values of the measured parameters of the different walls; no differences as regards Sm, ST, and SR were found between the septal, lateral, anterior, and inferior LA walls in each group.

Among the hypertensive patients, 31 (41%) cases were using renin-angiotensin system inhibitors (RASI) and this group, compared to those not using this medication, had lower ST (14.85 ± 6.17 in no RASI vs. 14.45 ± 5.50% in RASI) and SR (1.32 ± 0.57 in no RASI vs. 1.27 ± 0.40 s^{-1} in RASI); this difference, however, did not constitute statistical significance. The value for Sm was similar for both groups: 0.08 ± 0.01 in those receiving no RASI versus 0.08 ± 0.02 m/s in the ones receiving RASI.

According to the multivariate linear regression analysis (Table 3), hypertension was the only independent determinant of average LA strain rate in the all the 124 study individuals (P < 0.001). When this analysis was repeated in the patients with hypertension (Table 4), the only independent determinant of average LA strain rate was heart rate (P = 0.026).

Table 2. Comparison of Tissue Velocity and Strain Rate Imaging Parameters between the Two Study Groups

	Control Group (n = 49)	Hypertensive Group (n = 75)	P Value [a]
Peak Systolic Velocity, m/s			
LA [b] septal	0.09 ± 0.02	0.07 ± 0.02	< 0.001
LA lateral	0.10 ± 0.02	0.08 ± 0.21	< 0.001
LA anterior	0.11 ± 0.02	0.09 ± 0.02	< 0.001
LA inferior	0.12 ± 0.10	0.08 ± 0.09	0.047
Average Sm	0.10 ± 0.03	0.08 ± 0.03	< 0.001
Strain, %			
LA septal	24.59 ± 11.09	13.74 ± 7.32	< 0.001
LA lateral	28.09 ± 12.98	13.72 ± 8.11	< 0.001
LA anterior	30.13 ± 14.53	16.14 ± 8.56	< 0.001
LA inferior	29.18 ± 13.69	16.33 ± 8.64	< 0.001
Average ST	27.10 ± 11.94	14.98 ± 5.86	< 0.001
Strain Rate, 1/s			
LA septal	2.10 ± 0.56	1.17 ± 0.59	< 0.001
LA lateral	2.31 ± 0.68	1.37 ± 0.83	< 0.001
LA anterior	2.37 ± 0.59	1.41 ± 0.77	< 0.001
LA inferior	2.34 ± 0.63	1.29 ± 0.62	< 0.001
Average SR [b]	2.28 ± 0.49	1.31 ± 0.50	< 0.001

[a] P value, Level of significance.
[b] Abbreviations: LA, left atrium; SR, strain rate

Table 3. Results of Multivariable Linear Regression Analysis of Data From All the Study Subjects with Respect to the Factors Correlating with Changes in Left Atrial Average Strain Rate [a]

Variable	β Coefficient	95% CI [b]	P Value
Age	0.001	-0.014, 0.015	0.927
Gender	0.080	-0.182, 0.341	0.545
Heart rate	0.005	-0.003, 0.014	0.225
History of diabetes mellitus	0.232	-0.085, 0.548	0.149
History of hyperlipidemia	0.047	-0.221, 0.315	0.729
Left ventricular end-systolic diameter	-0.019	0.053, 0.015	0.262
Left ventricular end-diastolic diameter	0.005	-0.034, 0.44	0.798
Left ventricular ejection fraction	-0.006	-0.069, 0.054	0.838
Left ventricular mass index, g/m^2	0.002	-0.010, 0.005	0.584
Left atrial volume	0.004	-0.006, 0.015	0.420
Group (hypertensive vs. normotensive)	-1.080	-1.463, -0.697	< 0.001

[a] R square = 49.7%
[b] Abbreviation: CI, confidence interval

Table 4. Results of Multivariable Linear Regression Analysis of Data from the Patients with a History of Hypertension with Respect to the Factors Correlating with Changes in Left Atrial Average Strain Rate [a]

Variable	β Coefficient	95% CI [b]	P Value
Age	-0.001	-0.017 , 0.014	0.867
Gender	0.111	-0.211, 0.433	0.493
Heart rate	0.015	0.002, 0.028	0.022
Diabetes mellitus	0.218	-0.100, 0.537	0.175
Hyperlipidemia	0.067	-0.208, 0.343	0.626
Systolic blood pressure	0.001	-0.012, 0.013	0.932
Diastolic blood pressure	0.001	-0.025, 0.027	0.948
Left ventricular end-systolic diameter	-0.030	-0.068, 0.008	0.124
Left ventricular end-diastolic diameter	-0.018	-0.030, 0.066	0.460
Left ventricular ejection fraction	-0.042	-0.121 , 0.038	0.296
Left ventricular mass index, g/m^2	0.001	-0.010 , 0.010	0.956
Left atrial volume	0.004	-0.009, 0.016	0.548
RAS [b] inhibitors	-0.085	-0.371, 0.202	0.555

[a] R square = 20.4%
[b] Abbreviations: CI, confidence interval; RAS, rennin-angiotensin system

5. Discussion

The findings of this study showed that in the subjects with a normal value of LA size, the effect of hypertension on LA function (reservoir function) was independent of age, sex, heart rate, LA volume, LV mass index, and LVEF. In addition, in the patients with hypertension, after adjustment for other related factors, heart rate was independently correlated with reduced TVI and SRI parameters.

Hypertension alters atrial dynamics significantly, with resultant increased LA volume and active emptying volume consequent to altered LV diastolic function. Hypertension also accelerates the normal aging process with patients as early as decade 4 having similar LA size to that of normal controls in decade 8 (2). Previous studies have shown the usefulness of the early detection of LA dysfunction by SRI in the choice of treatment and in the prediction of the efficacy of defibrillation strategy for the treatment of atrial fibrillation (19). Moreover, LA enlargement along with aging has been correlated with reduced LA systolic SR in previous reports (6).

Decreased LA systolic SR may be an indicator for LA impaired function. Hypertension is associated with LA enlargement and impaired function. The early detection of LA dysfunction is of great importance because it is possible to preserve LA reservoir function in hypertensive patients without dilated LA (13).

Kokubu et al. (13) concluded that mean LA systolic SR was significantly lower in the hypertensive patients, either with or without dilated LA, than in the normal subjects. In their study, SR in the hypertensive patients with a normal value of LA size was 2.19 s^{-1} and the value for the normal controls was 2.53 s^{-1}. The average SR in our study was 1.31 ± 0.50 s^{-1} in the hypertensive patients with a normal value of LA size and 2.28 ± 0.49 s^{-1} for the normal controls. The possible reason for this difference between our study and the Kokubu et al. study is the fact that in our study the hypertensive patients and the normal controls were not matched in terms of LA dimension and LA dimension in the hypertensive patients was significantly larger than that of the controls, although these dimensions were within the normal range of value. Among the hypertensive patients in our study, 31 patients were using RAS inhibitors, and LA mean SR in this group was non-significantly lower than that of the patients not using RAS inhibitors. It has been reported (14) that mild hypertension results in a reduction in the early diastolic SR of the LA even in patients with preserved peak systolic SR and unchanged maximal LA volume.

5.1. Study Limitations

It is clear that there are some limitations to our study that were inevitable. In the matching of the patients, there was a statistically significant difference between the two groups in EF measurement. This difference was only about 1.7% in EF, which is obviously not clinically important. Also, it is obvious that some groups of drugs can have a potentially important impact on LA function, but ethically we had no permission to discontinue them. There are some newer imaging modalities for better evaluation of LA function and size such as two-dimensional strain imaging and three-dimensional echocardiography modalities, none of which was available in our institution during the study period.

5.2. Conclusion

According to the results of this study on subjects with a normal value of LA size, the effect of hypertension on LA reservoir function was independent of age, sex, heart rate, LV mass index, and LVEF. Additionally, heart rate was independently correlated with reduced TVI and SRI parameters in the patients with hypertension.

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Authors' Contributions

Mohammad Sahebjam: Study concept and design, Critical revision, Study supervision. Asghar Mazarei, Acquisition of data. Masoumeh Lotfi-Tokaldany: Analysis and interpretation of data, Drafting of the manuscript. Neda Ghaffari: Drafting of the manuscript. Arezoo Zoroufian: Critical revision. Mahmood Sheikhfatollahi: Statistical analysis.

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