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ORIGINAL ARTICLE

Does Blood Pressure Control Have an Impact on Right Ventricular Systolic Function Assessed by 2-D Speckle Tracking Echocardiography?

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Abstract

Background: The main cardiovascular risk factor related to higher likelihood of heart failure is hypertension [HTN]. There are plenty of researches on the pathological effects of HTN on the morphology and structure of the left ventricle [LV], but research investigating right ventricle [RV] mechanics in patients with hypertension have just recently emerged. So we aimed to assess whether blood pressure controlled or not had an impact on the systolic function of the RV.

Methods: One hundred seventy two participants were assessed by 2D echocardiography for LV function m-mode, right ventricle RV including tissue doppler imaging[TDI] to evaluate RV myocardial performance index [MPI], RV s wave, tricuspid annular plane systolic excursion[TAPSE]. Speckle tracking study was done for global longitudinal strain [GLS] of LV RV and free wall strain.

Results: Total of 172 participants were divided into, 86 were hypertensive in group I and 86 were normotensive in group II. TAPSE, MPI, RV s wave, PASP, LV-GLS, GLS of RV, and free wall strain were lower in group I. Subgroup analysis showed lower RVGLS in uncontrolled hypertensive patients.

Conclusions: Uncontrolled hypertensive patients had lower RV-GLS in comparison to controlled hypertensive patients. The predictors of impaired RV GLS in uncontrolled BP patients were BMI, RV MPI, and LVGLS

Keywords: Hypertension; Right Ventricular Systolic Function; Strain Echocardiography

INTRODUCTION

Generally, the population in many European countries appears to have a 30-45% prevalence of hypertension [1]. In Egypt, the five-year period from 2006 to 2011 observed a 44% increase in the percent change in hypertension, with a total prevalence of almost 39.7% of the population [2]. With a higher risk of heart failure [HF), acute coronary syndrome, stroke, and cardiac mortality, hypertension [HTN) is one of the main important cardiovascular risk factor [3]. It is well-established that hypertension negatively affects the left ventricle's [LV) structure and function [4]. On the other hand, the Right Ventricle [RV], a forgotten chamber that was once thought to have no major role in the heart's pump function, there is growing research demonstrating RV remodeling in arterial hypertension; the majority of these researches concentrated on RV diastolic function or hypertrophy [5–9]. One important parameter that has already been used in a variety of cardiovascular diseases is RV strain as it is readily available and simple to use, strain analysis contributes crucial data about the mechanics and function of the RV,

and the research on RV mechanics in hypertension patients has only lately emerged, and has demonstrated is a marked decline in RV longitudinal deformation [10–13]. Our study aimed to assess whether blood pressure controlled or not had an impact on right ventricle systolic function.

METHODS

Study population

Between September 2023 and May 2024, 172 participants were included in the study at cardiology department of Zagazig University Hospital and Al-Ahrar Teaching Hospital. 86 patients were classified as hypertensive as they were using antihypertensive medications or because their blood pressure consistently rose above 140/90 mm Hg on two or more times. [14], and were matched with 86 control volunteers. Patients with cor pulmonale, coronary artery disease, structural heart disease, and other causes of LVH were excluded. All participants were subjected to full history taking, blood pressure assessment, and body mass index [BMI) calculation

Conventional Echocardiographic Assessment

The assessment was done by [Vivid E 9 commercial ultrasound scanner, Horten, Norway). A standard evaluation of the left and right ventricles was performed in accordance with the guidelines supplied by the American Society of Echocardiography and the European Association of Echocardiography [15]. Two experienced and independent echo-cardiographers blinded to the patient's clinical data performed m-mode LV ejection fraction, septal thickness, and posterior wall thickness [PWT).

The RV myocardial performance index [MPI], RV s wave were assessed by Tissue Doppler imaging [TDI), and tricuspid annular plane systolic excursion [TAPSE) are all part of the right ventricular assessment [15]. When the tricuspid regurgitant jet was absent, mean pulmonary artery pressure was assessed rather than pulmonary artery systolic pressure [PASP) [15].

Assessment of left ventricle using speckle tracking echocardiography (STE)

Vivid E9 with soft were used to create two-dimensional STE pictures, which were taken from the left ventricular apical three, four, and two-chamber views. Three consecutive beats' worth of views were obtained, and they were then saved in cine-loop format. The software system automatically created epicardial tracing for each view after manually defining the endocardial border.

Assessment of right ventricle using (STE)

RV free wall strain and global longitudinal strain[RV-GLS) were assessed in accordance with guidelines of the American Society of Echocardiography and the European Association of Echocardiography [15]. Using the RV-focused view, six strain segmevvnts were created for RVGLS, matching the segmentation of the RV into six parts [basal, middle, and apical]. The free wall strain of RV was calculated by averaging the values of the three peak systolic strain segments.

Ethical standards

Every patient who was approved to be included in the study had a written consent. The General Organization for Teaching Hospitals and Institutes (GOTHI) approved the study on September 27, 2023, with an IRB number code of HAH00029.

Statistical Analysis

With SPSS, the data was analyzed [21]. Quantitative data was shown using the mean standard deviation. The student t-test was utilized to compare the means of the two groups, and the Chi-square test was employed to assess the qualitative data. The factors influencing a given variable were assessed using univariate and multivariate regression tests. The Pearson correlation was used to assess the relationship between the variables.

RESULTS

We enrolled 172 participants, 86 were hypertensive in group I and 86 were normotensive in group II. The hypertensive group I was older in age, had higher BMI, SBP, and DBP in comparison to normotensive group II with statistical significant difference [p<0.001](Table 1).

Hypertensive group I had significantly lower FAC in comparison to normotensive [33.96±3.38 vs 41.5 ± 6.97 respectively].TAPSE was also statistically significantly lower in hypertensive group I in comparison to normotensive [14.1 ± 2.42 vs 20.9 ± 2.02 respectively].In addition to RV MPI was higher in hypertensive group I than in normotensive [0.48 ± 0.09 vs 0.28 ± 0.09]. RV-GLS was significantly lower in hypertensive group I in comparison to normotensive [-15.6 ± 14.4 vs 20.2 ± 0.89], RV-free strain was significantly lower in hypertensive group I comparison to normotensive [-17.9 ± 14.98 vs22.4 ± 0.51], PASP was elevated in hypertensive group I than normotensive [37.02 ± 2.7 vs 20.5 ± 4.19, P<0.001](Table 2).

We divided our HTN patients into 2 subgroup analyses according to blood pressure [BP] control, 56 [65.1%] controlled BP group, and 30[34.9%]

uncontrolled BP group. The uncontrolled BP group was significantly older in age, had higher BMI, and had lower LVGLS. RVGLS was significantly lower in comparison to the controlled BP group [-14.2± 16.2 versus -17 ± 13.1, p 0.003]. FAC, TAPSE, RV` S, and RV MPI were statistically insignificant (Table 3, Figure 1&2 case demonstration of controlled and un-controlled BP subgroups respectively).

RV GLS had a high statistical significant negative correlation with age, BMI, IVS, PWT, RV MPI, SBP, DBP, and PASP. While there was a significant positive correlation with LVGLS, FAC, and TAPSE (Table 4, Figure 3). By regression analysis BMI, RV MPI, and LVGLS were significant predictors of poor RV GLS in individuals with uncontrolled blood pressure [0.03, 0.03, and 0.001, respectively] (Table 5).

Table (1): Basic characteristics of the studied groups

		Group I Hypertensive N=86		Group II Normotensive N=86		t-test	P
Age\ years Mean ±SD		54.8 ± 6.83 (50.6-59.6)		50.97 ± 3.15 (45-51.7)		4.66	<0.001 HS
BMI Mean ± SD		31.9 ± 1.08 (29-33)		24.8 ± 0.99 (21-25)		46.8	<0.001 HS
SBP (mm\Hg) Mean ± SD		165.7 ± 24.4 (160-170)		113.9 ± 4.62 (100-110)		10.8	<0.001 HS
DBP (mm\Hg) Mean ± SD		110.8 ±11.97 (115-120)		74.2 ± 4.95 (70-80)		9.15	<0.001 HS
Disease duration\ years Mean ± SD		7.88 ± 1.45 (6.5-9.7)		------		------	-----
		N	%	N	%	X^2	P value
Gender	Male	48	55.8	37	44.6	2.16	0.105 NS
	female	38	45.2	46	55.4		
Controlled BP(<140\90)	Yes	56	65.1	--		--	--
	No	30	34.9				

BMI:body mass index; **SBP**:systolic blood pressure; **DBP**:diastolic blood pressure;**BP**:blood pressure

Table (2): Echocardiographic data of the studied groups

	Group I Hypertensive N=86	Group II Normotensive N=86	t-test	P value
IVS(mm) Mean ±SD Range	12.2 ± 0.59 (12-13.5)	8.22 ± 0.76 (8-9.6)	38.2	<0.001
PWT(mm) Mean ±SD Range	13.23 ± 0.97 (12.5-14)	8.23 ± 0.72 (8-9.4)	36.97	<0.001
EF% M-mode Range	66.5±3.9 (62.6-68.8)	64.5±5.1 (65.4-69.7)	5.8	0.43
LVGLS% Mean ±SD Range	17.2 ± 2.47 (14 -17.6)	23.2 ± 0.59 (21.5-24.4)	21.16	<0.001 HS

FAC% Mean ±SD Range	33.96 ± 3.38 (31.5-33.6)	41.5 ± 6.9 (39-42.6)	3.06	0.001 S
TAPSE(mm) Mean ±SD Range	14.1 ± 2.42 (14-15.7)	20.9 ± 2.0 (19.4-21.5	6.37	<0.001 HS
RV S wave cm\s Mean ±SD Range	9.2 ± 1.57 (8.5-9.6)	15.2 ± 3.71 (12.5-16)	1.46	0.002 S
RV MPI Mean ±SD Range	0.48 ± 1.97 (0.44.2-0.49.4)	0.28 ± 0.09 (0.25-0.29)	1.96*	0.001
RV GLS% Mean ±SD Range	-15.6 ± 14.4 (14-17.6)	20.2 ± 0.89 (20-23.5)	10.1*	<0.001 HS
RV Free strain% Mean ±SD Range	-17.9 ± 14.98 (17.1-19.4)	22.4 ± 0.51 (21.6-23.8)	8.96*	<0.001 HS
PASP(mmHg) Mean ±SD Range	37.02 ± 2.7 (38.5- 41.3)	20.5 ± 4.19 (18.5-27.7)	24.6	<0.001 HS
Mean PAP Mean ±SD Range	25.7±5.8 (22.7-29.6)	22.4±4.9 (20-23.7)	13.9	0.98

IVS: interventricular septum, **PWT**: posterior wall thickness; **EF**: ejection fraction ;**LVGLS**:left ventricular global longitudinal strain; **FAC**: fractional area change; **TAPSE**:tricuspied annular plane systolic excursion ;**RVS**: right ventricular systolic wave ;**MPI**: myocardial performance index; **GLS**: global longitudinal strain ;**PASP**:pulmonary artery systolic pressure

Table (3): Subgroup analysis according to blood pressure control

		Controlled BP N=56 %= (65.1)		**Not controlled BP N=30 % = (34.9)**		**Test t-test\ MW***	**P**
		N	**%**	**N**	**%**	**X^2**	**P value**
Gender	**Male**	31	55.4	17	56.7	0.016	0.545
	female	25	44.6	13	43.3		
		Mean ±SD		Mean ±SD		t	p
Age\ years Range		53.8 ± 7.03		57 ± 5.89		2.36	0.02
BMI		31.7 ± 1.02		32.3 ± 1.09		2.58	0.01
SBP (mm\Hg)		128.2 ± 9.74		168 ± 22.3		11.5	<0.001
DBP (mm\Hg)		80.9 ± 9.78		98.7 ± 5.11		9.29	<0.001
Disease duration\ years		7.95 ± 1.48		7.77 ± 1.42		0.554	0.581
IVS		12.1 ± 0.63		12.3 ± 0.47		1.57	0.120
PWT		13.1 ± 1.11		13.6 ± 0.52		2.41	0. 72
LVGLS		18.6 ± 1.94		16.4 ± 2.89		3.48	0.001
FAC		38.8 ± 3.13		39.3 ± 3.92		0.56	0.593
TAPSE		20.7 ± 2.73		21.5 ± 1.66		1.35	0.003
RV` S		14.7 ± 3.1		13.5 ± 4.31		1.33	0.197
RV MPI		0.40 ± 2.11		0. 45± 1.53		1.06	0.307
RV-GLS		-17 ± 13.1		-14.2± 16.2		2.98*	0.003
RV-FWS		-11.38 ± 15.3		-10 ± 14.6		0.696*	0.491
PASP		36.5 ± 4.74		37.2 ± 4.24		1.44	0.147

BMI:body mass index; **SBP**:systolic blood pressure; **DBP**:diastolic blood pressure;**BP**:blood pressure; **IVS**: interventricular septum, **PWT**: posterior wall thickness ;**LVGLS**:left ventricular global longitudinal strain; **FAC**: fractional area change; **TAPSE**:tricuspied annular plane systolic excursion ;**RVS**: right ventricular systolic wave ;**MPI**: myocardial performance index; **GLS**: global longitudinal strain ;**PASP**: pulmonary artery systolic pressure, MW: Mann–Whitney test

Table (4): Correlation between RV strain and all data of subgroups

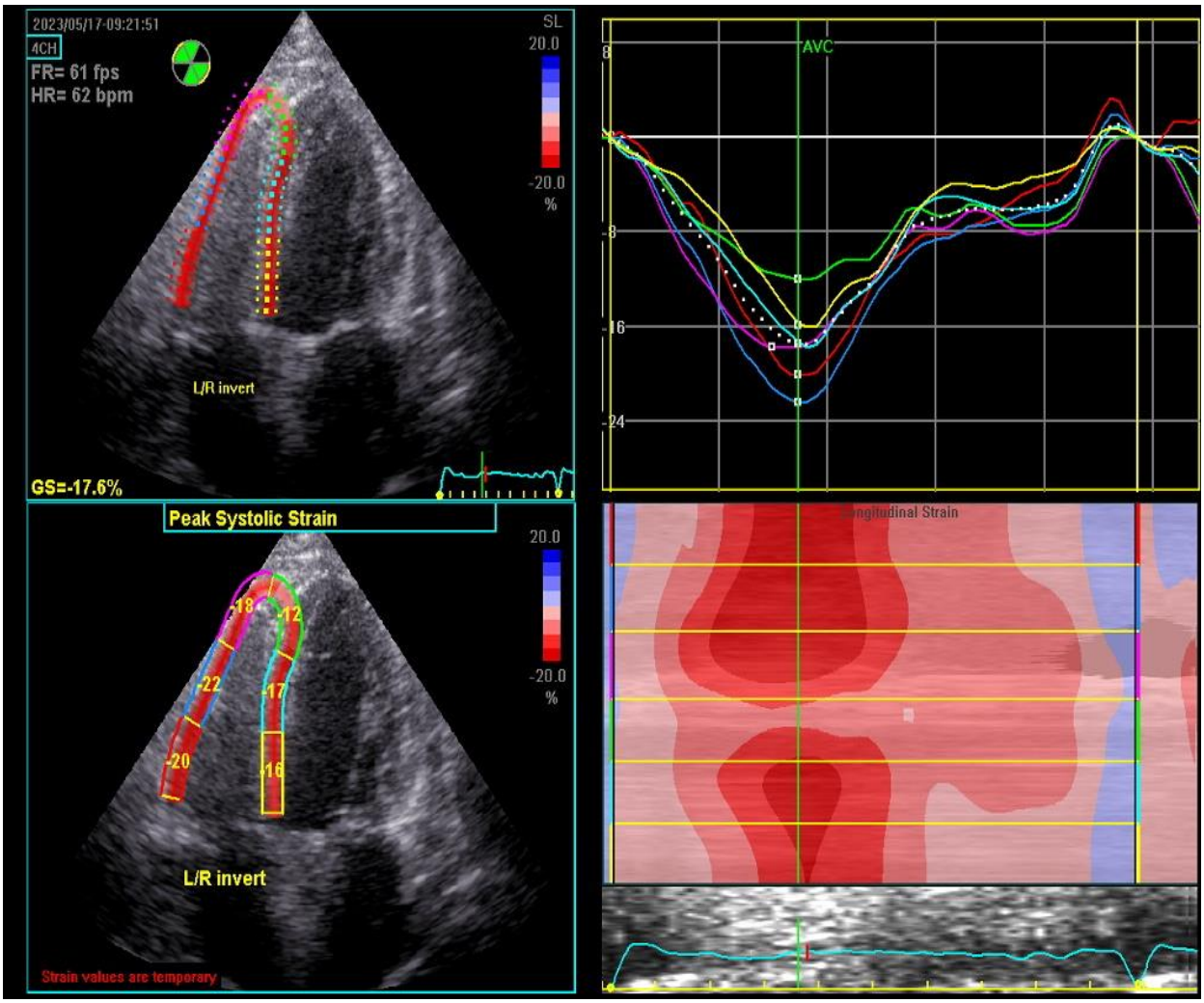
	RVGLS	
	R	P
Age	**-0.296****	**<0.001**
BMI	**-0.803****	**<0.001**
Duration	-0.067	0.541
IVS	**-0.799****	**<0.001**
PWT	**-0.789****	**<0.001**
LVGLS	**0.774****	**<0.001**
FAC	**0.209****	**0.006**
TAPSE	**0.401****	**<0.001**
RV` S	0.095	0.219
RV MPI	**-0.365****	**<0.001**
SBP	**-0.531****	**<0.001**
DBP	**-0.482****	**<0.001**
PASP	**-0.707****	**<0.001**

BMI:body mass index; **SBP**:systolic blood pressure; **DBP**:diastolic blood pressure;**BP**:blood pressure.**IVS**: interventricular septum, **PWT**: posterior wall thickness ;**LVGLS**:left ventricular global longitudinal strain; **FAC**: fractional area change; **TAPSE**:tricuspied annular plane systolic excursion ;**RVS**: right ventricular systolic wave ;**MPI**: myocardial performance index; **GLS**: global longitudinal strain ;**PASP**:pulmonary artery systolic pressure

Table (5): Multi-variate regression analysis of RV GLS predictors among studied cases

	Unstandardized Coefficients		Standardized Coefficients		
	B	**S. Error**	**Beta**	**T**	**P**
Age	0.111	0.189	0.033	0.588	0.557
BMI	-1.667	0.802	-0.336	**-2.077**	**0.03***
IVS	-1.060	1.146	-0.116	-0.925	0.356
PWT	-0.988	1.102	-0.136	-0.897	0.371
LVGLS	1.717	0.477	0.311	**3.603**	**<0.001****
FAC	0.072	0.167	0.021	0.429	0.669
TAPSE	0.208	0.469	0.027	0.443	0.658
RV MPI	-1.338	0.612	-0.101	**-2.186**	**0.03***
SBP	-0.049	0.076	-0.058	-0.637	0.525
DBP	-0.002	0.157	-0.001	-0.011	0.991
PASP	0.173	0.186	0.084	0.929	0.354
Disease duration	0.565	0.123	0.054	0.568	0.768
RV -free strain	0.370	0.071	0.365	5.18	0.351

BMI:body mass index; **SBP**:systolic blood pressure; **DBP**:diastolic blood pressure;**BP**:blood pressure.**IVS**: interventricular septum, **PWT**: posterior wall thickness;;**LVGLS**:left ventricular global longitudinal strain; **FAC**: fractional area change; **TAPSE**:tricuspied annular plane systolic excursion ;**RVS**: right ventricular systolic wave ;**MPI**: myocardial performance index; **GLS**: global longitudinal strain ;**PASP**:pulmonary artery systolic pressure



GS::global strain, L\R invert: left to right

Figure (1): RV STE case demonstration of the controlled BP subgroup: **upper left** image shows parametric color-coded display of end-systolic strain with GS of RV= -17.6 %; **lower left** image shows segmental end-systolic strain of both interventricular septum (IVS) and (free wall strain). FWS was calculated manually by averaging (the basal, mid, and apical free wall segments 20+22+19 divided by3= -20.3%; **upper right** image shows strain–time curves. The RV global strain variations during the cardiac cycle are shown by the white dotted line, while the colored curves indicate the segmental strain changes; **lower right** image shows an anatomical color-coded M-mode display of segmental strain variations during the cardiac cycle.

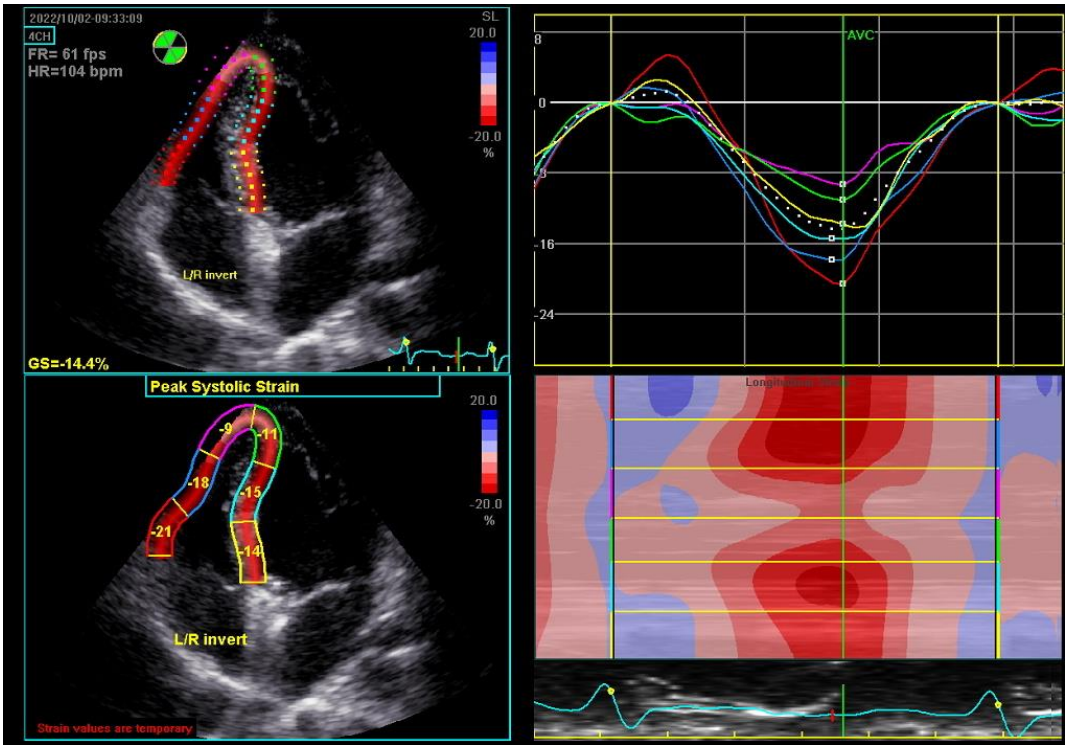


Figure (2): RV STE case demonstration of the uncontrolled BP subgroup: **upper left** image shows parametric color-coded display of end-systolic strain with GS of RV= -14.4 %; **lower left** image shows segmental end-systolic strain of both interventricular septum (IVS) and (free wall strain). FWS was calculated manually by averaging (the basal, mid, and apical free wall segments 21+18+9 divided by3= -16%; **upper right** image shows strain–time curves. The RV global strain variations during the cardiac cycle are shown by the white dotted line, while the colored curves indicate the segmental strain changes; **lower right** image shows an anatomical color-coded M-mode display of segmental strain variations during the cardiac cycle.

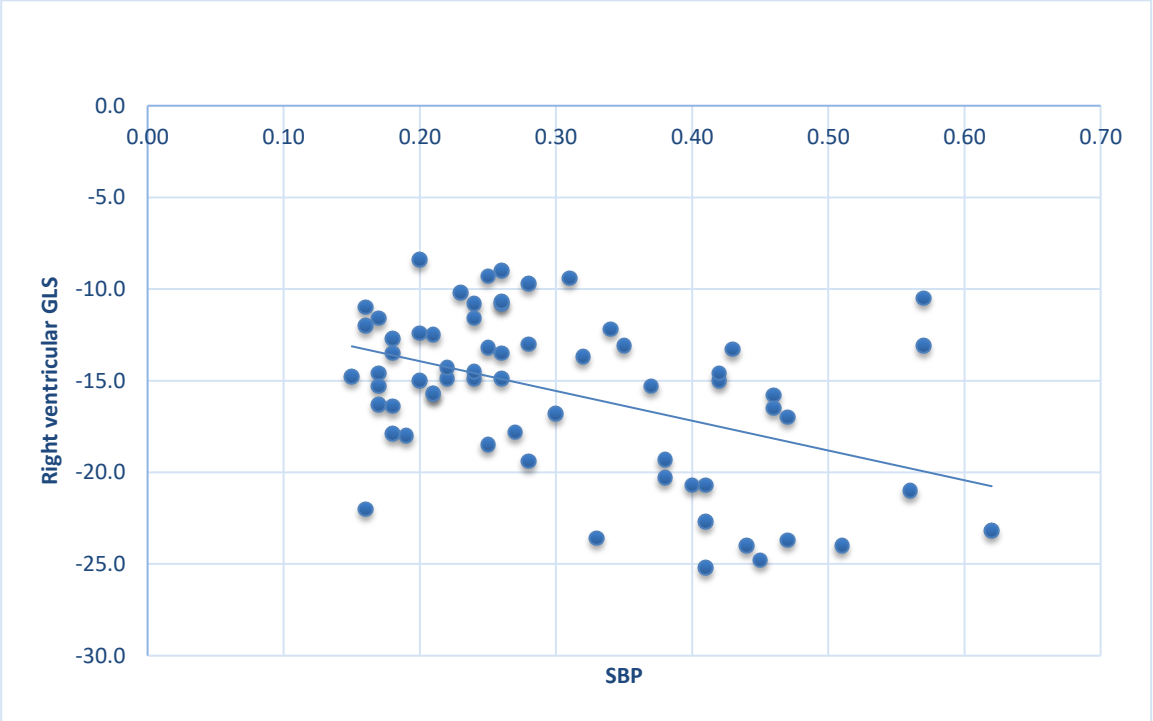


Figure (3): Showing good negative correlation between right ventricular GLS and SBP. GLS: global longitudinal strain, SBP: systolic blood pressure

DISCUSSION

Our research made a substantial contribution to the current understanding of how arterial hypertension affects RV mechanics as our results demonstrated that patient with HTN had impaired RV systolic function assessed by conventional, TDI, and STE-derived RV–GLS, free wall strain. This finding reinforce the concept that elevated RV filling pressures, ventricular interaction, transmission of elevated LV filling pressure to the pulmonary circulation and eventually to the RV, the detrimental effects of bio-humoral systems [sympathetic nervous system and renin-angiotensin-aldosterone] on RV, and ventricular interaction are all possible causes of poor RV systolic function in HTN [16]. Actually, the fact of ventricular interdependence was demonstrated in our study as there was impaired LV systolic function by STE-derived LVGLS in addition to impaired RV systolic function.

Hanboly et al [17] revealed that patients with hypertension had lower RV GLS. In hypertension individuals, segmental strain analysis revealed that the RV free wall's apical and mid segments were more deteriorated than its basal segment. Tadic et al [18] concluded that RV GLS was more reduced in untreated hypertension patients than in control subjects. Tumuklu et al [19] reported a reduced RV strain in hypertension patients both with and without LV hypertrophy = when compared to normotensive controls. Pedrinelli et al [20] concluded that in the mid-tertile of the blood pressure distribution, RV peak systolic strain decreased, but it remained unchanged in the upper one.

Actually we investigated more the hypertensive group and aimed to assess whether blood pressure controlled or not was influencing RV systolic function; so subgroup analysis in our study revealed that uncontrolled BP patients have lower RV-GLS in comparison to controlled BP, in addition, it is worth mentioning that STE- derived longitudinal strain was the only significant parameter in this subgroup analysis so we suppose that STE-RV GLS was superior to other conventional and TDI in detection subclinical RV impairment in uncontrolled BP patients. This finding was powered in our study by the good negative correlation between the RV-GLS and systolic blood pressure [SBP). Tadic et al [18] also reported significantly lower RV-GLS with inadequately regulated hypertension, in comparison to the patients with well-regulated hypertension.

Our results revealed that the predictors of impaired RV GLS in uncontrolled BP patients were BMI, RV MPI, and LVGLS. Xue et al [21] revealed that LV mass index, SBP, and relative wall thickness were the predictors of reduced RV-free strain in their study; with the difference in our study that their population study age was elderly.

Limitations: The following were limitations of our study; good image quality for STE endocardial border optimization, and the relatively small sample size of our population. We didn't follow up our patients to translate this impaired RV function into adverse clinical cardiac events.

CONCLUSIONS

Hypertensive patients had subclinical impaired RV systolic function. Uncontrolled hypertensive patients had lower RV-GLS in comparison to controlled hypertensive patients. The predictors of impaired RV GLS in uncontrolled BP patients were BMI, RV MPI, and LVGLS.

RECOMMENDATIONS

Hypertensive patients should be regularly assessed by RV -STE side by side with LV assessment. RV-GLS could be used for risk stratification in uncontrolled hypertensive patients. Future follow-up studies at long-term periods are recommended to relate impaired RV function to adverse clinical events.

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