Utility of Right Ventricular Strain Imaging in Predicting Pulmonary Vascular Resistance in Patients With Pulmonary Hypertension

Nasim Naderi, MD;[1] Zahra Ojaghi Haghighi, MD;[2] Ahmad Amin, MD;[1] Farah Naghashzadeh, MD;[3] Hooman Bakhshandeh, MD;[4] Sepide Taghavi, MD;[1] Majid Maleki, MD[1]

From the Shaheed Rajaei Cardiovascular, Medical and Research Center, Heart Failure and Transplantation Department, Tehran University of Medical Sciences, Tehran, Iran;[1] Echocardiography Research Center, Shaheed Rajaei Cardiovascular, Medical and Research Center, Tehran University of Medical Sciences, Tehran, Iran;[2] Heart Failure and Transplantation Department, Massih Daneshvari Educational Hospital, National Research Institute of Tuberculosis and Lung Disease, Shaheed Beheshti University of Medical Sciences, Niavaran, Tehran, Iran;[3] and the Shaheed Rajaei Cardiovascular, Medical and Research Center, Tehran University of Medical Sciences, Tehran, Iran[4]

Pulmonary vascular resistance (PVR) has important prognostic implications in the assessment of patients with pulmonary hypertension. Using echocardiography to measure PVR would have the advantage of being able to follow patients serially and to assess their response to treatment noninvasively. The authors sought to assess whether right ventricular strain rate imaging (SRI) can predict PVR in patients with pulmonary hypertension. The study population consisted of 46 patients referred for right heart catheterization. The inclusion criteria was mean pulmonary artery pressure $\geq$25 mm Hg in right heart catheterization in patients with pulmonary hypertension including chronic systolic heart failure. Echocardiography was performed to obtain SRI just before right heart catheterization. Mean values of peak systolic longitudinal strain and strain rate obtained from basal and mid-right ventricular free wall were calculated. The control group consisted of 35 healthy adults matched for age and sex. The most significant correlations were between basal right ventricular strain and strain rate (SR) and mean pulmonary arterial pressure (r=0.63, P=.000), transpulmonary gradient (r=0.6, P=.001), and PVR (r=0.5, P=.003). SR was independently correlated with PVR (PVR=26.9−16.9$\times$basal right ventricular SR; r=0.53, P=.003). The present study shows that basal right ventricular free wall strain and SR could be independently correlated with PVR in patients with pulmonary hypertension.

The primary abnormality in pulmonary hypertension is the increased afterload on the right ventricle due to elevated pulmonary vascular resistance (PVR) caused by the remodeling of the resistance pulmonary arteries. A large number of studies have clearly demonstrated that it is cardiac function that determines prognosis and exercise capacity.[1] In particular, it is worth noting that pulmonary arterial pressure (PAP) by itself does not correlate with prognosis.[1] Patients with known or suspected pulmonary hypertension undergo right heart catheterization for the assessment of hemodynamic parameters, including PVR. PVR and other hemodynamic variables have important prognostic implications in the assessment of patients with pulmonary hypertension.[1–7] Assessment of PVR is of great importance in the management of chronic heart failure, and it is an essential component of the evaluation of orthotopic heart transplant recipients.[8] Currently, this information is obtained only via invasive cardiac catheterization. An accurate noninvasive measurement of PVR would be helpful in eliminating the risk, cost, and discomfort associated with cardiac catheterization. While right heart catheterization would still be necessary to diagnose pulmonary hypertension, using echocardiography to measure PVR would have the advantage of being able to follow patients serially and to assess their response to treatment noninvasively.[1–7]

Recent studies have demonstrated that strain imaging provides an objective means to quantify global and regional left and right ventricular (RV) functions. In particular, RV myocardial strain has been shown to accurately quantify the RV function in patients with pulmonary hypertension.[9–11,13,14]

We aimed to assess whether RV systolic strain and strain rate (SR) can predict PVR in patients with pulmonary hypertension.

METHODS

Patient Selection

Among 110 patients referred to our center for right heart catheterization for evaluation of pulmonary hemodynamics for pulmonary hypertension between October 2009 and April 2011, 46 were enrolled according to the following inclusion/exclusion criteria. The inclusion criteria comprised pulmonary hypertension (mean PAP $\geq$25 mm Hg in right heart catheterization) of any type, including chronic systolic heart failure and New York Heart Association (NYHA) functional

Address for Correspondence: Zahra Ojaghi Haghighi, MD, Echocardiography Research Center, Shaheed Rajaei Cardiovasular, Medical & Research Center, Tehran University of Medical Sciences, Vali-Asr Avenue, Niyayesh Boulevard, Tehran 199691-1151, Iran
E-mail: z.ojaghihaghighi@gmail.com

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classes II or III. The exclusion criteria comprised atrial flutter/fibrillation or any other arrhythmias confounding the echocardiographic measurements, history of previous cardiac surgery such as coronary artery bypass grafting and any valve repair or replacement, advanced documented lung disease, complex congenital heart disease, and poor echocardiographic window. The control group was composed of 35 healthy adults matched for age and sex. The control patients were chosen among healthy volunteers by taking into account history, physical examination, electrocardiography, echocardiography, and negative results of recent noninvasive or invasive tests for cardiovascular diseases in men older than 45 and women older than 55 years. The study was approved by the ethics committee of Shaheed Rajaie Cardiovascular, Medical and Research Centre, and informed consent was obtained from all the patients.

Right Heart Catheterization

All patients were assessed in the catheterization laboratory with 7F balloon-tipped, triple lumen thermodilution catheters (Edwards Life Sciences, Irvine, CA) and Vigilance (VGSVSYS) monitors (Edwards Life Science). All measurements were acquired with the patients at rest in the supine position while breathing room air. The pressures were all averaged in 3 consecutive heart beats at end expiration. The following variables were measured for each patient: mean right atrial pressure; systolic and end-diastolic RV pressures; systolic, diastolic, and mean PAP; pulmonary capillary wedge pressure (PCWP); mixed venous oxygen saturation; and cardiac output via both thermodilution and the Fick technique. The transpulmonary gradient was calculated by subtracting the mean PAP from PCWP. PVR was calculated by dividing the transpulmonary gradient (TPG) by cardiac output.

Echocardiographic Study

A complete 2-dimensional (2D) color Doppler echocardiogram was performed in each subject using a commercial GE Vivid 7 with a three-MS variable frequency harmonic phased array transducer just before performing right heart catheterization. The left ventricular global systolic function was evaluated in terms of the ejection fraction, employing the biplane Simpson method. RV systolic function was evaluated in accordance with the American Society of Echocardiography guidelines for the echocardiographic assessment of the right heart in adults using the following parameters: tricuspid annular plane systolic excursion (TAPSE) and tissue Doppler-derived tricuspid lateral annular systolic velocity (s'). TAPSE<16 mm and S' velocity <10 cm/s indicated RV systolic dysfunction. The right atrium area was measured at the end-systole in the four-chamber view.[15]

Strain Rate Imaging in the Right Ventricle

Real-time 2-D color Doppler myocardial imaging was recorded from the RV, using standard 4-chamber apical views at a high frame rate (>150 FPS) and the narrowest sector angle possible. The region of interest was placed at the basal and mid segments of the RV free wall and kept at the center of the ultrasound sector to ensure the accuracy of the insonation angle with the long-axis motion to measure peak systolic longitudinal strain (RVSTR) and peak systolic longitudinal strain rate (RVSR). A sample volume of 7 mm to 10 mm was utilized for the calculation of longitudinal SR (Figure 1). The data obtained were stored in digital format and analyzed offline with dedicated software by an experienced person who was blinded to the clinical characteristics of the patients. For each variable, 3 representative beats were analyzed and the mean was calculated.

Statistical Analysis

All analyses were conducted using a commercially available package (SPSS version 19, SPSS, IBM, Armonk, NY) to compare the strain imaging data between the patients and the healthy controls. The unpaired Student *t* test was utilized for the continuous variables. The quantitative values are presented as mean±standard deviation (SD). Spearman's rank correlation coefficient was employed for variance analysis. The correlations between strain and SR and several echocardiographic and hemodynamic findings were assessed via linear regression. Stepwise multiple regression analysis was performed to assess the independent correlation between strain and SR and PVR. A *P* value of .05 was considered statistically significant. Interobserver and intraobserver variability were calculated as the absolute difference divided by the average of the two observations for all the parameters. Eleven cases were analyzed for the calculation of the interobserver and intraobserver variability.

RESULTS

Patient Characteristics

Among the 46 patients, 29 were diagnosed as precapillary pulmonary hypertension in accordance with updated clinical classification of pulmonary hypertension:[5] 18 cases with idiopathic pulmonary hypertension; 3 cases with pulmonary hypertension secondary to connective tissue diseases; 2 cases with pulmonary hypertension secondary to thalassemia; 2 cases with chronic thromboembolic pulmonary hypertension; and 4 cases with pulmonary hypertension secondary to congenital heart diseases (2 cases with patent ductus arteriosus and 2 cases with ventricular septal defect). The study population also included 17 patients with postcapillary pulmonary hypertension and chronic systolic heart failure referred for an evaluation of heart transplantation. They had a mean left ventricular ejection fraction of 17.65%±5.5% and a high mean PAP of 38.7±10.4 mm Hg. Six of them had a TPG >15 mm Hg (mean PVR=5.7±1.8 mm Hg).

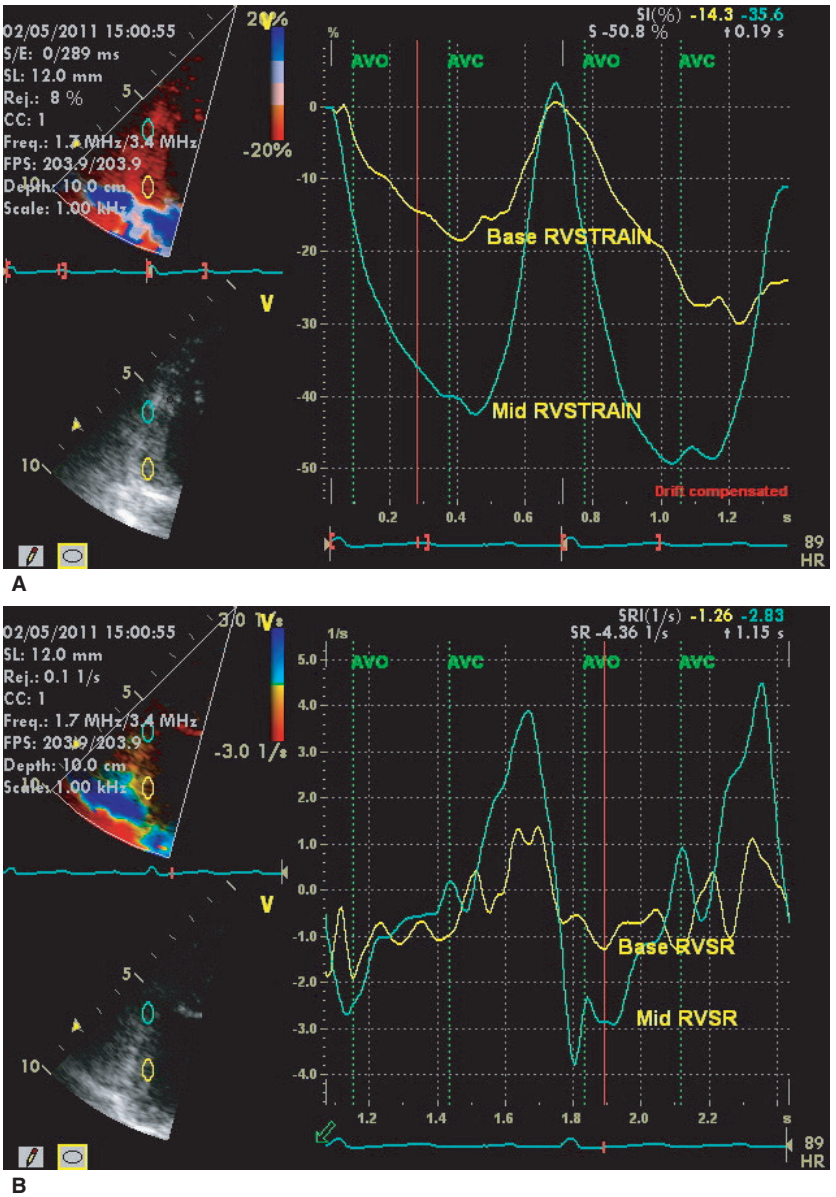

FIGURE 1. Strain rate imaging of right ventricular free wall in a patient with pulmonary hypertension peak systolic strain (A) and strain rate (SR) (B). AVO indicates aortic valve opening; AVC, aortic valve closing; RV, right ventricle.

Table I depicts the general characteristics of the patients and healthy controls and Table II demonstrates right heart catheterization data of the study population. There was a significant correlation between cardiac output/cardiac index and PVR measured by the two Fick and Thermodilution methods (r=0.7, P=.000 for cardiac output/cardiac index and r=0.9, P=.000 for PVR).

All precapillary pulmonary hypertension patients had LVEF >50% and their symptoms had developed recently (less than a year). Additionally, with the exception of one patient who was in NYHA class III, the remainder were in NYHA class II.

Strain and Strain Rate

SRI parameters could be measured in all patients and healthy persons, with a feasibility of 100%. The SRI measurements were obtained from the basal and middle segments of the RV free wall. The SRI measures were significantly reduced in the patients compared with the controls (Table III). These reduced strain parameters were more prominent in the middle segment of the right ventricle.

The intraobserver variability was 11.4%±7% and 12.4%±10% for strain and SR, respectively, and the interobserver variability for strain and SR was 12.3%±10% and 13.2%±3%, respectively. These

TABLE I. Characteristics of Patients With Precapillary, Post-Capillary, and Healthy Control

	Precapillary PH (n=29)	Postcapillary PH (n=17)	Normal (n=35)	*P* Value
Age, y	35.3±13.3	43.2±16	36.14±12.2	NS
Female/male	24/5	1/16	18/17	–
BSA, m^2	1.68±0.15	1.7±0.12	1.85±0.21	.001
Systolic BP, mm Hg	109.7±20	107.5±19	115±17	NS
Diastolic BP, mm Hg	67±10	68±10	74±5	NS
Heart rate, beats per min	83±18	84±12	74±10	NS
RVEDD, mm	45±1.1	36.4±11.5	26.5±3.9	.000[a]
				.000[b]
RVSm, cm/s	9.7±2.1	9.2±2.6	14.11±1.7	.000[a]
				.000[b]
TAPSE, mm	16.9±3	14.3±3.5	25.9±2.9	.000[a]
				.000[b]
RVFAC, %	23.5±6.9	27.9±7.5	39.1±3.7	.000[a]
				.000[b]
RAA index, cm^2/m^2	12.3±6.1	12.6±3.8	6.6±1.3	.000[a]
				.000[b]

Abbreviations: BP, blood pressure; BSA, body surface area; NS, not significant; RAA, right atrium area; RV, right ventricle; RVEDD, right ventricular end diastolic diameter; RVFAC, right ventricular fractional area change; TAPSE, tricuspid annular plane systolic excursion. Values are mean±standard deviation. [a]Precapillary pulmonary hypertension (PH) vs normal. [b]Postcapillary PH vs normal.

values were compatible with those reported in previous studies.

Postcapillary Pulmonary Hypertension Group

There was no significant difference with respect to the RV strain imaging measures between the patients with precapillary and postcapillary pulmonary hypertension (Table III). No correlation was found between the hemodynamic parameters and RV strain and SR in this group of patients even after adjusting for parameters such as RV function and tricuspid regurgitation severity (*r* value, 0.05–0.3). There were also no correlations between TAPSE and strain imaging data, PVR, and/or cardiac output/index in this group. RVSm and RVFAC were only correlated with cardiac output/index (r=0.5, P=.04).

Precapillary Pulmonary Hypertension Group

The most significant correlations were between basal RV strain and SR and mean PAP, TPG, and PVR (Table IV). A stepwise multiple regression analysis was performed to determine the independent relationship between basal RV strain and SR and PVR. SR was independently correlated with PVR (PVR=26.9−16.9 × basal RV SR; r=0.53, P=.003) (Figure 2).

The correlations between the above hemodynamic parameters and middle RV segment strain were significant but weak (r=0.4, P=.03 for all 3 variables) and there was no correlation between middle RV SR and

TABLE II. Right Heart Catheterization Findings

	Precapillary PH (n=29)	Postcapillary PH (n=17)
RA pressure, mmHg, mean±SD	9.5±4.3	10.8±5
RVS pressure, mmHg, mean±SD	86.1±34	57.9±17.2
RVEDP, mm Hg, mean±SD	9.5±4	11.6±5
SPAP, mm Hg, mean±SD	89.6±35.9	58±17
DPAP, mm Hg, mean±SD	48.4±18.2	29.1±8.1
Mean PAP, mmHg, mean±SD	61.1±21	38.7±10.4
PCWP, mmHg, mean±SD	10.6±3.4	26.7±8.3
TPG, mmHg, mean±SD	50.5±20.6	11.9±7
PVR, Wood, mean±SD	12.4±7.2	3.4±2.1
Thermodilution		
PVR, Wood, mean±SD	12.5±8.5	3.1±1.7
Fick		
CO, thermodilution, L/min	4.5±1.1	3.7±1
CI, thermodilution, L/min/m^2	2.8±0.6	2.14±0.5
CO, Fick, L/min	4.5±1.3	4±1.2
CI, Fick, L/min	2.8±0.8	2.5±0.7

Abbreviations: CI, cardiac index; CO, cardiac output; DPAP, diastolic pulmonary artery pressure; PAP, pulmonary artery pressure, PCWP, pulmonary capillary wedge pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RA, right atrium; RV, right ventricular; RVEDP, right ventricular end diastolic pressure; RVSP, right ventricular systolic pressure; SD, standard deviation; SPAP, systolic pulmonary artery pressure; TPG, transpulmonary gradient.

TABLE III. Strain Imaging Parameters in the Study Groups

	Precapillary PH	Postcapillary PH	Normal	*P* Value
Basal RV strain, %	15.1±4.4	16±4.1	29.7±4.4	NS[a]
				.000[b]
				.000[c]
Basal RV strain rate, 1/s	0.87±0.2	0.9±0.2	2±0.6	NS[a]
				.000[b]
				.000[c]
Middle RV strain, %	14.3±3.6	14.2±4	33.2±4.4	NS[a]
				.000[b]
				.000[c]
Middle RV strain rate, 1/s	0.89±0.2	0.9±0.2	2.2±0.5	NS[a]
				.000[b]
				.000[c]

Abbreviations: NS, nonsignificant; RV, right ventricular. Values are mean±standard deviation. [a]Precapillary pulmonary hypertension (PH) vs postcapillary. [b]Precapilllary PH vs normal. [c]Postcapillary PH vs normal.

hemodynamic parameters. The analysis of combined basal and middle RV strain and SR showed a significant correlation between these data and the aforementioned hemodynamic parameters, mean PAP (r=0.6, P=.001 for combined RV strain, r=0.51, P=.004 for combined RV SR), TPG (r=0.57, P=.001 for combined RV strain, r=0.53, P=.003 for combined RV SR), and PVR (r=0.5, P=0.008 for combined RV strain, r=0.5,

TABLE IV. Correlation Between SRI and Hemodynamics in Precapillary PH Group

	Mean PAP	TPG	PVR
Basal RV strain, %			
r	0.57[a]	0.54	0.5
P	.001[b]	.003	.008
Basal RV strain rate, 1/s			
r	0.57	0.54	0.62
P	.001	.002	.000
Combined RV strain			
r	0.56	0.45	0.55
P	.002	.01	.002
Combined RV SR			
r	0.52	*r*=0.5	*r*=0.5
P	.005	.009	.007

Abbreviations: PAP, pulmonary artery pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RV, right ventricular; SRI, strain rate imaging; TPG, transpulmonary gradient. [a]Spearman's rank correlation coefficient. [b]*P* value.

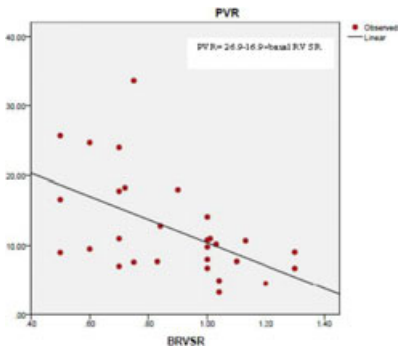

FIGURE 2. Linear correlation between pulmonary vascular resistance (PVR) and basal right ventricular (RV) strain rate (SR). PVR=26.9–16.9×basal RV SR (*r*=0.53 *P*=.003).

P=.008 for combined RV SR). None of the strain imaging measures were correlated with cardiac output and/or cardiac index and statistical adjustment for RV function and tricuspid regurgitation (TR) severity showed no improvement in the correlation in this group of patients. Regarding echocardiographic findings, RV SM was correlated with basal strain and SR (*r*=0.5, *P*=.005), PVR (*r*=0.42, *P*=.02), cardiac output (*r*=0.5, *P*=.007), cardiac index (*r*=0.42, *P*=.02); however, TAPSE and RVFAC were correlated only with cardiac output/cardiac index (*r*=0.5 and *P*=.007 and *P*=.02, respectively).

Statistical analysis was performed after removing the data of the congenital cases, and similar results were obtained.

With regard to TPG, 6 patients in the postcapillary group had inappropriate pulmonary hypertension (a precapillary reactive component). The strain imaging data of these patients were added to those of the precapillary pulmonary hypertension patients and analyzed. Mean PAP, TPG, and PVR were more significantly correlated with basal RV strain, SR, and combined strain data (*r* value 0.6, *P*=.001).

DISCUSSION

PVR is an important hemodynamic parameter used in the diagnosis and management of patients with heart failure and pulmonary hypertension. The gold standard method for the assessment of PVR remains invasive right heart catheterization. Previous studies have described noninvasive Doppler-based echocardiographic methods in determination of PVR. Abbas and colleagues[6] showed a significant correlation between the ratio of TR velocity to RV outflow tract (RVOT) VTI (TRV/$RVOT_{VTI}$) and invasively determined PVR. They proposed a clinically feasible formula for PVR estimation; however, they had included mostly pulmonary hypertension patients with left-sided heart causes, as many of them had high pulmonary capillary wedge pressures and most of them were referred for valvular or ischemic heart disease. On the other hand, Roule and colleagues[7] found that RVOT spectral Doppler tracing could not be achieved properly in PH patients with a PVR >2 Wood unit due to their specific hemodynamic over right heart structures and anatomical variations in this group of patients, suggesting that the Abbas formula might be erroneous in this context. Recently, the evaluation of regional myocardial function through strain imaging has been used in many pathological processes directly involving the myocardium and hemodynamic abnormalities. Detection of subtle changes in myocardial contractile function and better determination of it independent of loading conditions are among the advantages of this method.[9–11,13,14] The present study demonstrated that basal as well as combined RV peak systolic strain and SR had a weak (*r*=0.5) although significant correlation with invasively determined afterload measures of the pulmonary circulation in patients with precapillary pulmonary hypertension. Furthermore, our stepwise multiple regression analysis showed that basal and combined RV SR could be independent predictors for PVR. Although these findings should be interpreted with caution given the low value of the measured r^2 (coefficient of determination), they are consistent with those from other studies[10,11] that showed that RV myocardial strain correlate significantly with pulmonary hemodynamics in patients with precapillary pulmonary hypertension, an index of disease severity. In contrast with two similar studies,[11,14] there was no correlation between RV strain or cardiac index in the current study. This might be related to a higher baseline cardiac index and a lower right atrial pressure in our patients, denoting that our study population might have had less severe pulmonary hypertension. (They were mostly in NYHA functional class II with dyspnea on exertion.) Although elevated PVR results in the disturbance of myocardial contractility, as can be shown by disturbed strain and strain rate numbers, it does

not always dramatically affect cardiac output in less severe cases. In a study by Rajagopalan and colleagues,[14] mean right atrial pressure, an important factor indicating right-sided performance and prognosis in pulmonary hypertension, was slightly higher than that in the present study (10±6 vs 9.5±4.3), which implies more progressive disease in their patients. Accordingly, these differences might explain our observation of a correlation between mean PAP and PVR and RV strain and SR rather than cardiac index.

In the present study, we also analyzed the data of postcapillary PH patients. There was no correlation between strain imaging and the hemodynamic data. Interestingly, despite less severe pulmonary hypertension and lower TPG in the patients with left ventricular dysfunction, RV strain and SR were not significantly different between the heart failure and pulmonary hypertension groups. RV dysfunction in patients with left ventricular dysfunction can be secondary to various mechanisms such as pulmonary venous congestion, RV myocardial involvement by cardiomyopathic processes, and RV ischemia. Thus, as Rajagopalan and colleagues[11] also discussed in their study, the reduction in RV strain and SR could not simply be explained by hemodynamic load in heart failure patients and this might be an explanation for any correlation between hemodynamic data and strain/SR in this group of patients. The effects of left-sided heart failure on RV function need to be determined in future studies. We found no correlation between the hemodynamic data and RV strain/SR in the patients with postcapillary PH; nevertheless, adding the data of 6 heart failure patients with high TPG and PVR (a precapillary reactive component) to those of the precapillary group increased the significance of the correlations. Elevated PVR had obvious effects on RV strain and SR; moreover, the addition of the data of the postcapillary PH patients with precapillary reactive component (high PVR) augmented the significance of the correlation between PVR and strain/SR. Therefore, it could be possible to predict high PVR via SR in this group of patients. Our small sample size (only 6 postcapillary PH patients with high PVR) precluded any subgroup analysis. Future studies are required to shed sufficient light on this issue since it could be a useful index in the follow-up of cardiac transplant candidates.

LIMITATIONS

Among the shortcomings of the present study is that tissue Doppler-derived strain measurements, similar to other Doppler modalities, are dependent on the direction of the Doppler angle. Interpretation of SR should, therefore, be made with caution if tissue direction deviates more than 30 degrees from the direction of the ultrasonic beam. Along with other tissue Doppler-based studies, the present study also had high rates of interobserver and intraobserver variability. As a result, it would be better if Doppler-based study replaced the speckle tracking–based method, which is a superior method to delineate altered RV wall mechanics.[16,17] Another weak point is that an invasive hemodynamic study was not performed in the healthy controls because it would not be ethically acceptable. Finally, the sample numbers of the study are limited and there are no outcome data.

CONCLUSIONS

Our present study shows that basal and combined RV free wall strain and SR could be independently correlated with PVR in patients with precapillary pulmonary hypertension. Strain imaging study may be a very useful measure of evaluating PH patients. Perhaps RV strain imaging is a better integrator of load/function than PVR. Further studies are needed to determine whether it would be useful to evaluate PVR in heart failure patients who are candidates for heart transplantation.

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