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Original research

ASSESSMENT OF PULMONARY VASCULAR RESISTANCE VIA DOPPLER ECHOCARDIOGRAPHY IN ISCHEMIC HEART DISEASE WITH REDUCED EJECTION FRACTION

Tran Ke Toan[1], Nguyen Thi Thuy Hang[2], Ho Anh Binh[3], Cao Thi Thuy Phuong[3], Duong Thi Thuy Linh[3]

[1]Gia Lai General Hospital

[2]Hue University of Medicine and Pharmacy

[3]Cardiovascular Center - Hue Central Hospital

ABSTRACT

Objectives: *To determine pulmonary vascular resistance (PVR) by echocardiography - Doppler; and evaluate the correlation between pulmonary vascular resistance and some variables such as left ventricular EF, PASP, TAPSE, and tissue S-wave of the tricuspid valve in patients with ischemic heart disease.*

Method: *Observational study on 82 heart failure patients with reduce ejection fraction brought upon by ischemic heart disease, at the Cardiology Department of the University of Medicine and Pharmacy from 04/2016 - 05/2017.*

Results: *The average PVR is 3.91 ± 1.85 WU. There was not significant difference between age and gender groups ($p > 0.05$), but there was a statistically significant difference of PVR in patients with NYHA III and IV compared to NYHA I, II ($p < 0.05$). When EF < 30%, the rate of increased PVR is higher than the normal value (35.4 % vs 4.9%). There is a strong correlation between LVEF and PVR ($r = - 0.545$, $p < 0.001$), especially when PVR < 8 WU ($r = - 0.618$, $p < 0.001$). When the PASP increases, the rate of increased PVR is higher than normal value (54.9% vs. 9.8%, $p < 0.001$), with an enough correlation coefficient ($r = 0.361$, $p < 0.001$). In patients with RV systolic dysfunction evaluated by TAPSE and S' wave, there was a significant difference in the rate of increased PVR from the normal value (41.5% vs 1.2%; 34.1 vs 8.5%, p 0.001). PVR is closely correlated with TAPSE ($r = -0.590$; $p < 0.001$) and is inversely correlated with S' wave ($r = -0.590$; $p < 0.001$).*

Conclusions: *Increased PVR is the primary mechanism for pulmonary hypertension and right ventricular dysfunction in patients with HFrEF brought upon by IHD. The evaluation of PVR in patients with left ventricular dysfunction by echocardiography is important in clinical practice.*

Keywords: *Pulmonary vascular resistance, echocardiography, heart failure, reduced ejection fraction.*

I. INTRODUCTION

To balance the afterload of the heart, the connection of the right ventricle (RV) and pulmonary circulation plays an important role. The left ventricle (LV) partially transmits the forces to the RV through the interventricular septum. Therefore, the synchronous contraction and relaxation of LV and RV were achieved. When the LV failure occurs, the RV afterload is gradually increased due to post-capillary pulmonary artery hypertension (PAH) which translates into elevated pulmonary vascular resistance (PVR) [1].

Ischemic heart disease (IHD) is a consequence of myocardial oxygen supply and demand imbalances, caused by coronary atherosclerosis [2]. The long-term effect is heart failure. Heart failure with reduced ejection fraction (HFrEF) increases when the left ventricular ejection fraction (LVEF) is below 40%. When LV failure progresses, the RV function will be affected via multiple mechanisms in which increased

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Corresponding author: Ho Anh Binh. Email: drhoanhbinh@gmail.com. Phone: 0913489896.

PVR plays a vital role [3 - 6]. Recent studies suggest that increased PVR, pulmonary hypertension, and RV systolic dysfunction are independent factors for poor prognosis in patients with LV failure [6 - 8].

Cardiac catheterization is the gold standard of the PVR evaluation, but its use is limited by its invasive nature and certain risks. Using echocardiography helps to evaluate RV function, PASP and PVR, which is the most reliable, non-invasive technique [9].

We aim to engage our comprehension of PVR in patients with HFrEF brought upon by IHD, we conducted this study to determine PVR by using echocardiography - Doppler in HFrEF brought upon by IHD; and evaluate the correlation between PVR and some variables such as LVEF, PASP, TAPSE, and tissue S-wave of the tricuspid valve (S') in these population.

II. METHODS

2.1. Study population

We enrolled 82 patients with HFrEF (LVEF < 40%) brought upon by IHD at the Cardiology Department of the University of Medicine and Pharmacy from 04/2016 - 05/2017. IHD also called coronary heart disease (CHD) diagnosed with the significant stenosis of denovo coronary arteries or with documented (prior) MI or coronary artery revascularization (either with PCI or CABG) [2, 9]. Patients with congenital heart disease, primary pulmonary hypertension, or non-IHD diseases lead to pulmonary hypertension were excluded from the study.

2.2. Methodology

Observational Study was performed. All patients underwent investigations and evaluate the cardiovascular risk factors [10]. Doppler echocardiography (EnVisor CHD; Philips, USA) was performed after enrollment. All data of echocardiography were collected [11]:

+ Evaluating the structure and measuring routine echocardiogram parameters.

+ LVEF measured by Simpson method.

+ RV systolic function is assessed by using tissue S-wave of the tricuspid valve (S'), and TAPSE index. TAPSE is defined as the distance traveled between end-diastole and end-systole at the lateral corner of the tricuspid annulus. TAPSE has been validated to correlate strongly with RVEF measured by radionuclide angiography, with low observer variabilities [9].

+ Pulmonary artery systolic pressure (PASP) was calculated by tricuspid regurgitation peak velocity (TRV peak) and right atrial pressure.

+ PVR was calculated by tricuspid regurgitation velocity (TRV) and the time-velocity integral of the RV outflow tract (VTIRVOT) according to the Arm E. Abbas formula. A cutoff value for the Doppler equation was generated to determine PVR >2 Wood units (WU) [12].

Table 1: Important parameters of echocardiography

HFrEF: LVEF < 40%
Normal TAPSE value: ≥ 16mm
Normal S' value: ≥ 9 cm/s
PASP = 4 (TRV)2 + RA pressure
PVR = (Vmax of TR / VTIRVOT) x 10 + 0.16

2.3. Statistical analysis

SPSS 20.0 was used for data analysis. Variables described by rate, mean value ± standard deviation. Regression analysis was used to determine the correlation between PVR and LVEF, PASP, TAPSE, and S' wave. The significance level is 0.05, and the corresponding confidence level is 95%.

III. RESULT

3.1. Baseline characteristics

Most patients had at least one cardiovascular disease risk factors: HBP, smoking and diabetes mellitus (DM). The mean age was 74.1 ± 10.9, with 46 males. There was 43.9% of patients hospitalized with NYHA class III and IV.

Table 2: Clinical characteristics

Parameters	n (%)
Age	
Mean	74.1 ± 10.9
Min	42
Max	93
Age > 70 (%)	70.1
Gender (M/F)	46/36
Hypertension	47 (57.3)
Smoking	41 (50.0)
Diabetes	12 (14.6)

Parameters	n (%)
NYHA (%)	
I	8.5
II	47.6
III	28.0
IV	15.9
CCS (%)	
I	25.6
II	31.7
III	26.8
IV	13.4

The table 2 showed mean of LDL-Cholesterol was 2,77 ± 0,91 µmol/l. The mean of LVEF was 31.5 ± 5.4%. LV regional wall motion abnormalities were found in the majority of patients (70.7% of hypokinesia; 53.7% of akinesia). There was 42.7% of RV systolic dysfunction according to TAPSE, 42.7% of RV systolic dysfunction according to S' waves. The increased of PASP was 72%.

Table 3: Paraclinical characteristics

Parameters	Value	Parameters	Value
LDL-Cholesterol (µmol/l)(Mean)	2.77 ± 0.91	Echocardiography	
ECG (Rate, %)		LVEF (Simpson, %) (Mean)	31.5 ± 5.4 (Rate,%)
Arrhythmia	35.4	Hypokinesia	70.7
ST-T changes	65.9	Akinesia	53.7
Necrosis Q	75.4	Decreased TAPSE	42.7
Pathological T wave	98.8	Decreased S'	42.7
		Increased PASP	72.0

3.2. Pulmonary vascular resistance

The mean PVR was 3.91 ± 1.85 WU. There was not statistical difference between age and gender groups (p > 0.05). There was 35.4% of cases had normal PVR (35.4%). increased PVR (43.9%) prevails, compared to the group with moderate or severe PVR elevation (p < 0.001).

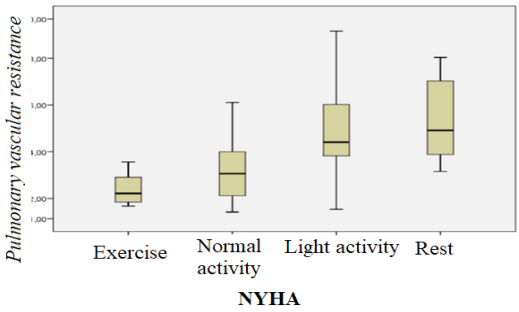

Figure 1: Comparison of mean PVR values by NYHA groups

In the NYHA III/IV group, the proportion of patients with elevated PVR was higher than the normal PVR value (p < 0.001). The mean PVR increased gradually from NYHA group I to group NYHA IV (p < 0.001) (p < 0.001). In the HFrEF group, the rate of increased PVR is 35.4% compared to 4.9% for normal PVR (p < 0.001). Patients with both elevated PVR and elevated PASP had the highest incidence (54.9%).

Table 4: The rate of increased PVR follows LVEF, TAPSE, S' wave, and PASP

	Increased PVR, n (%)	Normal PVR, n (%)	Total, n (%)	p
EF				
< 30%	29 (35.4)	4 (4.9)	33 (40.2)	
> 30%	24 (29.3)	25 (30.5)	49 (59.8)	< 0.001
Total	53 (64.6)	29 (35.4)	82 (100.0)	

	Increased PVR, n (%)	Normal PVR, n (%)	Total, n (%)	p
S’				
Decreased	28 (34.1)	7 (8.5)	35 (42.7)	
Normal	25 (30.5)	22 (26.8)	47 (57.3)	< 0.001
Total	53 (64.6)	29 (35.4)	82 (100)	
TAPSE				
Decreased	34 (41.5)	1 (1.2)	35 (42.7)	< 0,001
Normal	19 (23.2)	28 (34.1)	47 (57.3)	
Total	53 (64.6)	29 (35.4)	82 (100)	
PASP				
Increased	45 (54.9)	14 (17.1)	59 (72.0)	< 0,001
Normal	8 (9.8)	15 (18.3)	23 (28.0)	
Total	53 (64.6)	29 (35.4)	82 (100)	

There was 54.9% of cases had increased PVR and increased PASP, while only 9.8% of cases had increased PVR only ($p < 0.001$). The percentage of patients with both increased PVR and RV systolic dysfunction as determined by the TAPSE index were 41.5% and by the S’ wave was 34.1% ($p < 0.001$).

3.3. Correlation of PVR with LVEF, PASP, S’ wave, and TAPSE index

There was a strong correlation between the PVR and LVEF ($r = - 0.545$, $p < 0.001$), according to the regression equation: $Y = - 0.187X + 9.281$. When PVR < 8 WU, this correlation was stronger ($r = - 0.618$, $p < 0.001$), according to the regression equation: $Y = - 0.171X + 9.079$. There was a moderate positive correlation between PVR and PASP ($r = 0.361$; $p = 0.001$), according to the regression equation: $Y = 0.049X + 2.084$. There was a strong negative correlation between PVR and TAPSE ($r = -0.590$; $p < 0.001$), according to the regression equation: $Y = - 0.249X + 8.237$. There was a moderate negative correlation between PVR and S’ wave ($r = - 0.402$; $p <0.001$), according to the regression equation: $Y = - 0.283X + 6.659$

IV. DISSCUSSION

We enrolled 82 HFrEF patient throught by upon IHD with high risk of cardiovascular risk factor. RV systolic dysfunction might develop in association with LV dysfunction via multiple mechanisms [8]. Bursi et al showed that the percentage of PASP was 79% [13]. In the study of C. Jaarsmar et al, there was a correlation between LVEF and TAPSE measured by cardiac MRI ($r = 0.50$, $p < 0.001$) [14]. Our study results was similar to those of previous studies.

In the NYHA III/IV group, the rate of increased PVR was much higher than in the other group ($p < 0.001$). In terms of pathogenesis, increased PVR was a consequence of pulmonary vascular remodeling, which was an important link in the progression to “reactive” pulmonary hypertension in patients with left heart disease, which in turn causes dyspnea on exertion [8]. When we analyzed the degree of heart failure, we also found a high incidence of increased PVR in the EF < 30% group (54.9%). This result was similar when compared to the study of Fabregat-Andrés et al. [15].

Our study found that the rate of patients with both increased PVR and RV systolic dysfunction was 75.6% (41.5% assessed by TAPSE and 34.1% assessed by S’ wave), compared to 54.9% in the presence of elevated PASP. The increased PVR and RV systolic dysfunction are two important factors in the progression of LV systolic failure. Pham Thi Tuyet Nga (2013) [4] showed an increased PVR in majority of patients. Nguyen Thi Mai Ngoc (2012) [3] studied patients before atrial septal defect closure, and the results showed that the average PVR was 2.25 ± 1.31 WU compared to a control group of 1.31 ± 0.20 WU.

In our study, PVR was closely correlated with LVEF ($r = -0.545$; $p < 0.001$), especially when SCMP < 8 ($r = - 0.618$; $p < 0.001$). This result was similar to that of Garcia-Alvarez et al. [15]. M Assadpour Piranfar showed the mean PVR decreased, corresponding to LVEF ($p = 0.004$) [16].

Increased PVR causes pulmonary artery hypertension. It is therefore not surprising that PVR

and PASP are correlated. With the results obtained, we affirm once again this correlation with r = 0.361 and p = 0.001, lower than authors Nguyen Tan Vuong [5] with r = 0.55 and Pham Thi Tuyet Nga [4] with r = 0.65. The population selection of Nguyen Tan Vuong and Tran Thi Tuyet Nga enrolled both valvular heart disease and congenital heart disease. The prolongation period of progress pulmonary vascular remodeling in congenital heart diseases was clearly longer than in IHD, so PVR would be higher.

With the RV systolic function, we found a strong positive correlation of PVR and TAPSE (r = - 0.590, p < 0.001) and a moderate negative correlation with S' waves (r = -0.402, p < 0.001). In study of M Assadpour Piranfar, the average PVR decreased as TAPSE increased and vice versa to the 18mm TAPSE cutoff point (p = 0.026) [16].

V. CONCLUSION

PVR by using echocardiography is a crucial index that helps early recognition of RV systolic dysfunction secondary to HFrEF throught by upon IHD as well as treating patients and possibly reversing the clinical manifestations effectively and improving prognosis.

REFERENCES

1. Dini FL, Pugliese NR, Ameri P, Attanasio U, Badagliacca R, Correale M, et al. Right ventricular failure in left heart disease: from pathophysiology to clinical manifestations and prognosis. Heart Fail Rev. 2023;28(4):757-766.
2. Nguyen Quang Tuan. Stable coronary disease in 100 things to know about stable coronary disease. 2014, Medical Publishing House. 22-24, 55, 62-67, 93-101.
3. Nguyen Thi Mai Ngoc. Evaluation of pulmonary artery resistance by ultrasound Doppler before and after treatment to close the Atrial septal defect. Hanoi Medical University. 2011.
4. Nga Pham Thi Tuyet Nga. Study of pulmonary vascular resistance in patients with mitral stenosis before and after balloon angioplasty. Hanoi Medical University. 2013.
5. Vuong Pham Tan Vuong. Study of pulmonary vascular resistance by Doppler echocardiography in patients with mitral stenosis. 2015, Hue University of Medicine and Pharmacy.
6. Haddad F, Hunt SA, Rosenthal DN, Murphy DJ. Right ventricular function in cardiovascular disease, part I: Anatomy, physiology, aging, and functional assessment of the right ventricle. Circulation. 2008;117(11):1436-48.
7. Lankhaar JW, Westerhof N, Faes TJ, Gan CT, Marques KM, Boonstra A, et al. Pulmonary vascular resistance and compliance stay inversely related during treatment of pulmonary hypertension. Eur Heart J. 2008;29(13):1688-95.
8. Voelkel NF, Quaife RA, Leinwand LA, Barst RJ, McGoon MD, Meldrum DR, et al. Right ventricular function and failure: report of a National Heart, Lung, and Blood Institute working group on cellular and molecular mechanisms of right heart failure. Circulation. 2006;114(17):1883-91.
9. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1-39.e14.
10. Goff DC, Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB, Gibbons R, et al. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;129(25 Suppl 2):S49-73.
11. Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland JGF, Coats AJS, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. European Heart Journal. 2016;37(27):2129-2200.
12. Abbas AE, Fortuin FD, Schiller NB, Appleton CP, Moreno CA, Lester SJ. A simple method for noninvasive estimation of pulmonary vascular resistance. J Am Coll Cardiol. 2003;41(6):1021-7.
13. Bursi F, McNallan SM, Redfield MM, Nkomo VT, Lam CS, Weston SA, et al. Pulmonary pressures and death in heart failure: a community study. J Am Coll Cardiol, 2012;59(3):222-31.
14. Schalla S, Jaarsma C, Bekkers SC, Waltenberger J, Dennert R, Crijns HJ, et al. Right ventricular function in dilated cardiomyopathy and ischemic heart disease: assessment with non-invasive imaging. Neth Heart J. 2015;23(4):232-40.
15. Fabregat-Andrés Ó, Estornell-Erill J, Ridocci-Soriano F, Pérez-Boscá JL, García-González P, Payá-Serrano R, et al. Prognostic Value of Pulmonary Vascular Resistance by Magnetic Resonance in Systolic Heart Failure. Arq Bras Cardiol. 2016;106(3):226-35.
16. Assadpour M, Piranfar MK. Correlation between pulmonary vascular resistance and some cardiac indices. Iranian Cardiovascular Research Journal. 2011;5(30):92-96.