Relation of Left Ventricular Filling Paterns to The Ratio of Pulmonary Venous to Mitral A Velocity in Patients with Left Ventricular Systolic Dysfunction

Bilge M., Güler N., Eryonucu B.

Department of Cardiology, School of Medicine, Yüzüncü Yıl University, Van, Turkey

Objective: **In the present study, we retrospectively evaluated the ratio of pulmonary venous to mitral A velocity (PV-A/A) in patients with left ventricular (LV) systolic dysfunction with three specific types of LV filling patterns.**

Method: **Transthoracic and transesophageal echocardiography were performed in 44 patients with LV systolic dysfunction in sinus rhythm, aged 20 to 73 years (mean 54±12), and in 11 control subjects without cardiovascular disease aged 24 to 69 years (mean 53±7). Patients were divided into three groups according to LV filling pattern: Group I; those with an impaired relaxation filling pattern (E wave/A wave <1, n=16), group II; those with a pseudonormal filling pattern (1≤ E/A<2 , n=12) and group III; those with a restrictive filling pattern (E/A≥ 2, n=16).**

Results: **The control group and the three patient groups were not different with respect to age, gender and heart rate. All patient groups showed significantly lower LV ejection fraction than the control group, and was lowest in group III. The mitral peak A wave velocity was significantly lower in group III than in the first, control and second groups (p<0.001, p<0.01and p<0.05, respectively) and was highest in group I. The pulmonary A wave velocity did not differ among groups I, III and the controls, and was higher in group II than in the controls (p<0.01). The PV-A/A ratio was significantly higher in group III than in the control, groups I and II (p<0.001, p<0.001and p<0.05, respectively). The ratio did not differ between the controls and group I, but were lower compared to the group II (p<0.05).**

Conclusion: **An increased ratio of PV-A/A was noted in subjects with LV systolic dysfunction with restrictive and pseudonormal LV filling patterns. This supports the idea that the PV-A/A ratio reflects LV filling pressures.**

Key words: *Ventricular filling patern to mitral A velocity, ventricular systolic dysfunction*

Studies have shown that the mitral A wave velocity was diminished while the pulmonary venous A wave (PV-A) velocity was augmented because of decreased left ventricular (LV) compliance in patients with elevated LV filling pressure (1,2). Recently, Ito et al.(3) reported that an increased ratio of pulmonary venous to mitral A velocity (PV-A/A) was a useful marker for elevated pulmonary capillary wedge pressure among patients with LV systolic dysfunction. But, the pulmonary venous flow (PVF) velocity pattern was obtained with transthoracic echocardiography in this study. Although PVF velocity patterns can also be obtained with transthoracic approach, the qualitative and quantitative analysis of the atrial wave is problematic because the atrial velocities are very low and therefore difficult to differentiate from baseline wall motion noise (1,2). In addition, they did not evaluate normal ratio PV-A/A in subjects without LV systolic dysfunction. In the present study we retrospectively investigated the ratio of PV-A/A in subjects with LV systolic dysfunction with three specific types of LV filling patterns and in normal subjects who underwent previously transesophageal echocardiography.

Material and Method

Study population: Fifty patients with left ventricular systolic dysfunction who underwent transesophageal echocardiography previously were considered for this retrospective study. Six were excluded because of inadequate Doppler echocardiographic recordings of the PVF velocity pattern, thus yielding a remaining study population of 44 patients with left ventricular systolic dysfunction. There were 23 men and 21 women ranging in age from 20 to 73 (mean 54±12). The diagnosis of left ventricular systolic dysfunction was based on two-dimensional echocardiography (left ventricular ejection fraction<50%). Patients were excluded if they had atrial fibrillation, primary valvular disease, or more than grade 2 mitral regurgitation. The presumed etiology of left ventricular systolic dysfunction was ischemic in 24 patients. Ischemic left ventricular systolic dysfunction was considered present if the patient had a documented previous myocardial infarction or a >50% luminal diameter stenosis on any major epicardial coronary artery in coronary angiography. Patients had nonischemic left ventricular systolic dysfunction if they had angiographically normal coronary arteries. All patients were being treated with classic cardiac drugs, including digitalis, diuretics, angiotensin-converting enzyme inhibitors, nitrates or a combination of these. Because the study population was drawn from patient groups with abnormal LV systolic function in the previous prospective studies, the patients did not take any drugs for at least 12 hours before the echocardiographic studies.

Echocardiographic studies: All analysis were made

Accepted for publication: 26 September 2000

Table I. Clinical and echocardiographic data of the patient and control groups.

Parameters	Control group	Group I (E/A<1)	Group II (1≤ E/A<2)	Group III (E/A≥2)
Number	11	16	12	16
Age (yr)	53±7	57±12	56±11	50±16
Male/female	6/5	7/9	5/7	9/7
Heart rate (beats/min)	81±15	84±11	83±10	85±12
LVDD (mm)	44±3	54±10 *	57±5 **	65±9 # ¶
LAD (mm)	28±4	38±4 #	42±4 #	48±7 # ¶¶ ‡
LVEF (%)	64±3	43±4 #	39±7 #	30±7 # ¶¶ ‡‡
E (cm/s)	82±19	52±15 **	71±12	94±26 ¶¶ ‡
A (cm/s)	55±13	71±19 *	50±7 ¶	32±12 ** ¶¶ ‡
E/A	1.52±0.21 &	0.74±0.15 &	1.42±0.22 &	3.22±1.39
DT (ms)	190±11	188±50	133±27 ** ¶	111±30 # ¶¶
PV-A (cm/s)	19±3	25±7	31±11**	25±6
PV-A/A	0.36±0.12 & ‡	0.35±0.10 & ‡	0.62±0.21	0.87±0.28‡

*Values are expressed as mean±SD. *p<0.05, versus control group; **p<0.01, versus control group; #p<0.001, versus control group; ¶p<0.01, versus group I; ¶¶ p<0.001, versus group I; ‡p<0.05, versus group II; ‡‡p<0.01, versus group II; & p<0.001, versus group III. LVDD, left ventricular end-diastolic diameter; LAD, left atrial diameter; LVEF, left ventricular ejection fraction; E, mitral E velocity; A, mitral A velocity; DT, mitral E deceleration time; PV-A, pulmonary venous A velocity*

from the recordings on videotapes. All patients underwent a transthoracic examination using a commercially available Doppler echocardiography unit (Vingmed CFM 725) with a 3.25 MHz probe before the transesophageal evaluation. Left ventricular end-diastolic diameter (LVDD) and left atrial diameter (LAD) were measured from parasternal M-mode recordings accordings to standard criteria (4). Left ventricular ejection fraction (LVEF) was determined from apical views using a modified Simpson's rule (5).

Transmitral flow was recorded in the standard apical four chamber view with the sample volume positioned between the tips of the mitral leaflets (6-8). The peak velocity in early filling (E) wave and at atrial contraction (A) wave and the deceleration time of the E wave were measured. The E wave deceleration time was obtained by extrapolating the initial slope of E wave deceleration to the zero line (6-8). The ratio of the peak velocities of the E and A waves was derived.

Trans-esophagial echocardiography (TEE) was performed using a 5 MHz multiplane probe (Vingmed CFM 725). All patients were studied in the fasting state using 10% lidocaine spray for posterior pharyngeal anesthesia. No sedation or atropine was administered. The TEE probe was inserted with the subject lying in the left lateral position. The procedure was performed with continuous monitoring of heart rate and a one lead electrocardiogram. TEE was tolerated well by all patients, and there was no complication.

We measured the PVF velocity patterns by positioning the transesophageal pulsed Doppler sample volume over the left upper pulmonary vein approximately 1 to 1.5 cm proximal to its entrance to the left atrium (9-10). The pulmonary venous (PV-A) and mitral A velocities were measured by TEE. The ratio of pulmonary venous to mitral A velocity (PV-A/A) was derived. All Doppler measurements were averaged on 3 cardiac cycles. All patients were in sinus rhythm at the time of the study. Before beginning TEE, informed consent was obtained from each subject.

The patients were divided into three groups according to the Doppler transmitral flow velocity profile: group I (n=16) was characterized by an impaired relaxation pattern, with E/A<1; group II (n=12) included patients with a pseudonormal pattern, with 1≤ E/A<2 and group III (n=16) consisted of patients with restrictive pattern characterized by E/A≥ 2.

Eleven subjects (ranging in age from 24 to 69 years, mean 53) with normal cardiovascular findings and no evidence of heart failure were selected as controls. Indications for the TEE study in the control subjects included suspected cardiac source of embolism (5 subjects), suspected congenital heart disease (3 subjects) and suspected aortic disease (3 subjects).

Statistical analysis: Data were presented as mean ± SD. One-way analysis of variance was used to compare continuous variables between groups, with a subsequent Scheffe test for multiple means comparisons. P value< 0.05 was considered statistically significant.

Results

Baseline characteristics of the control and the three patient groups: The clinical and echocardiographic data for each group are listed in Table I. The control group and the three patient groups were not statistically different with respect to age, gender and heart rate.

The LVDD and LAD were significantly larger in three patient groups than in the control group (p<0.001 and p<0.01, respectively), and were largest in groups III and II. All patient groups showed significantly lower LVEF

than the control group ($p<0.001$ for each comparison), and was lowest in group III.

Because the study patients were grouped on the basis of E/A ratio, peak E velocity was significantly higher in group III than in group I and II ($p<0.001$ and $p<0.05$, respectively). The E/A ratio was higher in group III than in groups I, II and the controls ($p<0.001$ for each comparison). The mitral E deceleration time was shorter in groups II and III compared to the group I and the controls, but did not differ between groups II and III.

Pulmonary venous A, mitral A waves velocities and their ratio (PV-A/A): The mitral peak A wave velocity was significantly lower in group III than in the first, control and second groups ($p<0.001$, $p<0.01$and $p<0.05$, respectively), was highest in group I. The pulmonary A wave velocity did not differ among groups I, III and the controls, and was higher in group II than in the control group ($p<0.01$).

The PV-A/A ratio was significantly higher in group III than in the controls, groups I and II ($p<0.001$, $p<0.001$and $p<0.05$, respectively). The ratio did not differ between the controls and group I, but were lower compared to group II ($p<0.05$).

Discussion

Analysis of the results of recent echocardiographic Doppler studies suggest that the PVF velocity pattern may be more sensitive to loading conditions than the mitral flow velocity pattern(7,11-17). In patients with elevated LV filling pressure, the mitral A wave velocity is supressed, and the PV-A velocity increases because of marked elevation of afterload imposed on the left atrium(1-3,18-19). Conversely, in subjects with normal LV filling pressure, there is an increase in the mitral A wave velocity and a concomitant decrease in the PV-A velocity (1-2,7,18-19). Nishimura et al. (2) showed that the pulmonary capillary wedge pressure correlated best with the PV-A velocity in the pulmonary vein at atrial contraction. However, the acute alterations in the load they produce could not accurately simulate changes in chronic loading conditions in patients with heart disease. Recently, using transthoracic echocardiography, Ito et al. (3) found the PV-A/A ratio to best predict increases in the pulmonary capillary wedge pressure in patients with LV systolic dysfunction. They have also suggested that the PV-A/A ratio could reflect net atrioventricular compliance.

As known, patients with impaired LV relaxation often have normal or near normal LV filling pressures (7). In contrast, patients with restrictive or pseudonormal filling patterns are often associated with elevated LV filling pressures (7,20-22).

Based on these findings, we retrospectively evaluated the ratio of PV-A/A in subjects with LV systolic dysfunction with three specific types of LV filling patterns and in normal subjects.

As expected, the PV-A/A ratio was significantly higher in groups with restrictive and pseudonormal filling patterns than in group with impaired relaxation and the controls, but the PV-A/A ratio was similar in group with impaired relaxation and the controls. These results showed the close relationship between the PV-A/A ratio and the LV filling pattern in patients with LV systolic dysfunction, supporting the idea (3) that the PV-A/A ratio reflects LV filling pressures. The reason why the ratio showed a correlation with pulmonary capillary wedge pressure is unknown. However, it has been suggested that each A velocity runs in opposite directions during altered loading conditions (3).

We found that the PV-A velocity was higher in the group with pseudonormal filling pattern than in the group with restrictive filling pattern, although this difference was not significant. This result supports that, in the lack of an increase in PV-A velocity in the patients with restrictive filling pattern, the atria may have undergone fibrosis (22).

Limitations of the study: In the present study, invasive hemodynamic measurements were not performed. However, LV filling patterns have previously been correlated with simultaneous hemodynamic data (7,20-22). Because PVF velocity patterns were only obtained by transesophageal echocardiography, we could not compare the results with transthoracic echocardiographic findings.

In conclusion, an increased ratio of pulmonary venous to mitral A velocity was noted in subjects with LV systolic dysfunction with restrictive and pseudonormal LV filling patterns. This supports the idea that the PV-A/A ratio reflects LV filling pressures.

References

1. Nishimura RA, Abel MD, Hatle LK, Tajik AJ: Assessment of diastolic function of the heart: background and current applications of Doppler echocardiography: Part II. Clinical studies. Mayo Clin Proc 64: 181-204, 1989.
2. Nishimura RA, Abel MD, Hatle LK, Tajik AJ: Relation of pulmonary vein to mitral flow velocities by transesophageal Doppler echocardiography: effect of different loading conditions. Circulation 81: 1488-1497, 1990.
3. Ito T, Suwa M, Kobashi A, Hirota Y, Kawamura K: Ratio of pulmonary venous to mitral A velocity is a useful marker for predicting mean pulmonary capillary wedge pressure in patients with left ventricular systolic dysfunction. J Am Soc Echocardiogr 11: 961-965, 1998.
4. Sahn DJ, DeMaria A, Kisslo J, Weyman A: Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. Circulation 58: 1072-1083, 1978.
5. Schiller NB, Shah PM, Crawford M et al: Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography. Committee on standards, subcommittee on quantitation of two-dimensional echocardiograms. J Am Soc Echocardiogr 2: 358-367, 1989.
6. Giannuzzi P, Imparato A, Temporelli PL et al: Doppler-derived mitral deceleration time of early filling as a strong predictor of pulmonary capillary wedge pressure in

postinfarction patients with left ventricular systolic dysfunction. J Am Coll Cardiol 23: 1630-1637, 1994.

7. Appleton CP, Hatle LK, Poop RL: Relation of transmitral flow velocity patterns to left ventricular diastolic function: New insights from a combined hemodynamic and Doppler echocardiographic study. J Am Coll Cardiol 12: 426-440, 1988.
8. Appleton CP, Hatle LK, Poop RL: Demonstration of restrictive ventricular physiology by Doppler echocardiography. J Am Coll Cardiol 11: 757-768, 1988.
9. Ito T, Suwa M, Hirota Y, Otake Y, Moriguchi A, Kawamura K: Influence of left atrial function on Doppler transmitral and pulmonary venous flow patterns in dilated and hypertrophic cardiomyopathy: evaluation of left atrial appendage function by transesophageal echocardiography. Am Heart J 131: 122-130, 1996.
10. Suwa M, Otake Y, Ito T, Moriguchi A, Hirota Y, Kawamura K: Noninvasive estimation of pulmonary capillary wedge pressure from pulmonary venous flow using tranesophageal echocardiography. Am J Noninvas Cardiol 8: 207-214, 1994.
11. Phillips RA, Coplan NL, Krakoff LR et al: Doppler echocardiographic analysis of left ventricular filling in treated hypertensive patients. J Am Coll Cardiol 9: 317-322, 1987.
12. Masuyama T, St Goar FG, Alderman EL, Poop RL.:Effects of nitroprusside on transmitral flow velocity patterns in extreme heart failure: A combined hemodynamic and Doppler echocardiographic study of varying loading conditions. J Am Coll Cardiol 16: 1175-1185, 1990.
13. St Goar FG, Masuyama T, Alderman EL, Poop RL.:Left ventricular diastolic dysfunction in end-stage dilated cardiomyopathy: A simultaneous Doppler echocardiographic and hemodynamic evaluation. J Am Soc Echocardiogr 4: 349-360, 1991.
14. Shen WF, Tribouilloy C, Rey JL et al: Prognostic significance of Doppler-derived left ventricular diastolic filling variables in dilated cardiomyopathy. Am Heart J 124: 1524-1533, 1992.
15. Mulvagh S, Quinones MA, Kleiman NS, Cheirif JB, Zoghbi WA:Estimation of left ventricular end-diastolic pressure from Doppler transmitral flow velocity in cardiac patients independent of systolic performance. J Am Coll Cardiol 20: 112-119, 1992.
16. Schwengel RH, Hawke MW, Fischer ML, Gottlieb SS, Plotnick GD: Abnormal Valsalva blood pressure response in dilated cardiomyopathy: Association with 'pseudonormalization' of echocardiographic Doppler transmitral filling velocity pattern. Am Heart J 126: 1182-1186, 1993.
17. Vitarelli A, Ferro Luzzi M, Penco M, Ciciarello F, Fedele F, Dagianti A: PVF velocity pattern in patients with heart failure: transesophageal echocardiographic assessment. Cardiology 88: 585-594, 1997.
18. Rossvoll O, Hatle LK: Pulmonary venous flow velocities recorded by transthoracic Doppler ultrasound: Relation to left vetricular diastolic pressures. J Am Col Cardiol 21: 1687-1696, 1993.
19. Stoddard MF, Pearson AC, Kern MJ, Ratcliff, Mrosek DG, Labovitz AJ: Left ventricular diastolic function: comparison of pulsed Doppler echocardiographic and hemodynamic indexes in subjects with and without coronary artery disease. J Am Col Cardiol 13: 327-336, 1989.
20. Vanoverschelde JJ, Raphael DA, Robert AR, Cosyns JR: Left ventricular filling in dilated cardiomyopathy: relation to functional class and hemodynamics. J Am Coll Cardiol 15: 1288-1295, 1990.
21. Himura Y, Kumada T, Kambayashi M et al: Importance of left ventricular systolic function in the assessment of left ventricular diastolic function with Doppler transmitral flow velocity recording. J Am Coll Cardiol 18: 753-760, 1991.
22. Ito T, Suwa M, Otake Y, Moriguchi A, Hirota Y, Kawamura K.: Left ventricular Doppler filling pattern in dilated cardiomyopathy: relation to hemodynamics and left atrial function. J Am Soc Echocardiogr 10: 518-525, 1997.

Correspondence:
Mehmet Bilge, M.D
Yüzüncü Yıl Üniversitesi
Tıp Fakültesi Kardiyoloji AD
65200 Van, TURKEY