

Mitral Valve Prolapse In Atrial Septal Defect

M Kestelli

Citation

M Kestelli. *Mitral Valve Prolapse In Atrial Septal Defect*. The Internet Journal of Cardiology. 2001 Volume 1 Number 2.

Abstract

Mitral valve prolapse is encountered at a rate of 25-95 % in patients with atrial septal defect. In this study, we aimed at obtaining physiopathological patterns causing mitral valve prolapse in patients with atrial septal defect by comparing the patients with atrial septal defect but no mitral valve prolapse (Group 1) to those with atrial septal defect and mitral valve prolapse (Group 2) in accordance with the following echocardiographic parameters: diastolic interventricular septum thickness, diastolic left ventricular posterior wall thickness, left ventricular ejection fraction, left ventricle end-diastolic diameter, left ventricle end-systolic diameter, right ventricular out-flow tract diameter, right ventricular in-flow tract diameter and mitral valve area calculated with pressure half-time. There were significant differences between the two groups in terms of diastolic interventricular septum thickness and left ventricular posterior wall thickness (10 ± 2.16 to 8.7 ± 0.38 and 9.27 ± 1.56 to 8.1 ± 1.18 , $p < 0.0035$ and $p < 0.0006$ respectively). The left ventricular ejection fraction was found to be significantly high in patients with atrial septal defect and mitral valve prolapse (72.26 ± 5.48 to 69.06 ± 6.23 , $p < 0.011$).

INTRODUCTION

The Incidence of mitral valve prolapse in patients with atrial septal defect (ASD) has been reported to be high^{1,2,3,4}. This incidence is about 35 %. In their study, Lippé et al. have found the incidence to of MVP in patients with ASD to be 95 % by means of two- dimensional echocardiography⁵.

The reason for this high incidence has not been completely explained yet. The discussion on the high incidence of MVP in patients with ASD have focused on two hypotheses⁶. One of these is the anatomical co-existence of ASD and MVP as a result of a congenital common connective tissue defect. The other hypothesis is the anomaly occurring in the geometry of the left ventricle due to ASD.

In this study, it was tried to obtain physiopathological patterns causing MVP by echocardiographically comparing the patients with ASD and MVP to the ones with ASD but no MVP.

MATERIAL AND METHOD

In this study, the echocardiographic parameters of 33 patients with ASD but no MVP (Group 1) and those of 30 patients with ASD and MVP (Group 2) were compared. Group 1 consisted of 26 female and 7 male patients and the average age was 48.5 ± 12 (between 21-65 years of age). Group 2 consisted of 25 female and 5 male patients and the average age was 45.5 ± 6 (between 22-62 years of age). There were no statistical differences between the two groups

in terms of age and gender (table 1). In both groups, there was a case of mitral insufficiency and the assessment of their left ventricles was within normal limits.

Figure 1

Table 1: Characteristics of groups according to gender and age.

	n	Female	Male	Age
Group1 (mvp -)	33	26	7	48.5 ± 12
Group2 (mvp +)	30	25	5	45.5 ± 6

The groups were compared according to the following echocardiographic parameters: diastolic interventricular septum thickness (DIVST), diastolic left ventricular posterior wall thickness (DLVPWT), left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), right ventricular out-flow tract diameter (RVOTD), right ventricular in-flow tract diameter (RVITD) and mitral valve area (MVA) calculated with pressure half-time.

In the study, the parameters were obtained by utilising Ultramark 6-3.5 mHz electronic doppler echocardiograph. Measurements were carried out parasternal long axis by one and the same person. The results were statistically assessed with t-student test. Values under $p < 0.05$ were regarded as

being statistically significant. Patients exhibiting segmentary wall movement disorder were not included in the study.

RESULT

Between the two groups, there were statistically significant differences in terms of diastolic interventricular septum thickness and diastolic left ventricular posterior wall thickness. DIVST was 10 ± 2.16 mm in Group 1 while it was 8.76 ± 0.38 mm in Group 2 ($p < 0.0035$). DLVPWT was 9.27 ± 1.56 mm in Group 1, whereas it was 8.1 ± 1.8 mm in Group 2 ($p < 0.006$). Besides, LVEF was found to be significantly high in Group 2 (72.26 ± 5.48 to 69.06 ± 6.23 , $p < 0.011$). Echocardiographical parameters of both groups have been shown in Table 2.

Figure 2

Table 2: Echocardiographic results. Abbreviations: (DIVST) diastolic interventricular septum thickness, (DLVPWT) diastolic left ventricular posterior wall thickness, (LVEF) left ventricular ejection fraction, (LVEDD) left ventricular end-diastolic diameter, (LVESD) left ventricular end-systolic diameter, (RVOTD) right ventricular out-flow tract diameter, (RVITD) right ventricular in-flow tract diameter and (MVA) mitral valve area calculated with pressure half-time.

	ASD		IVST	LVPWT	LVEF	LVEDD
Group 1	Without MVP	Mean	10	9,27	69,06	43,09
		SD	2,16	1,56	6,23	5,25
Group 2	With MVP	Mean	8,76	8,1	72,26	43
		SD	1,38	1,18	5,48	6,99
		P	0,0035	0,0006	0,011	0,47

	ASD		LVESD	RVOTD	RVITD	MVA
Group 1	Without MVP	Mean	26,39	33,09	49,63	4,54
		SD	4,55	5,02	5,51	0,36
Group 2	With MVP	Mean	25,06	35,33	50,66	4,56
		SD	4,54	6,88	6,83	0,54
		P	0,12	0,07	0,25	0,43

DISCUSSION

Isolated mitral valve prolapse is observed in 2.5-5 % of general population^{7,8}. The incidence is higher in women than in men. Today, MVP is the most common cause of isolated mitral insufficiency which requires mitral valve surgery⁹.

Isolated MVP is divided into two groups: functional and

anatomical. Anatomical MVP comprises chorda elongation and leaflet elongation. In a study, as an example of functional MVP, dehydration was formed by giving furosemid to healthy women with previously normal cardiac findings, and hence, functional mitral valve prolapse was simulated. MVP which developed in these patients was discontinued with rehydration¹⁰.

The rate of observing MVP in patients with ASD varies between 35-95 % in various studies¹⁻⁵. MVP has regressed after ASD has been repaired. Right ventricular volume loading, right ventricular dilatation and paradoxical septal movement have been observed in patients with ASD^{11,12,13}. As a result, MVP is functional in patient with ASD.

The relationship between functional MVP and right ventricular dilatation resulting from pulmonary hypertension which develops secondary to toxication has been shown in a study. In this study, MVP has been observed at a rate of 9.3 % in patients with a right ventricle of normal size, 30 % in those with borderline right ventricular dilatation and 56 % in the ones with serious right ventricular dilatation¹⁴.

It has been tried to be shown by Scraiber 15 et al. that there is a relationship between ASD and MVP, and that it is related to end-diastolic movement of septum. We have not been able to relate the left ventricular eccentricity occurring due to end-diastolic intraventricular septum paradoxal movement developing secondary to right ventricular volume loading to MVP occurring in systol.

As for our result, when we take the mitral leaflet as the base of a triangle, and the chorda-papiller muscle system and the left ventricle wall as the sides of the triangle, MVP occurs as a result of the mathematical irregularity of this system. During left ventricular ejection, while the minor (transverse) axis of the heart shortens 27-37 %, the major axis (between the base and the apex of the heart) shortens 9 %. We are of the opinion that an excessive increase in the minor axis of the above mentioned triangle will cause functional MVP. In our study, LV ejection fraction was found to be significantly high in group MVP with ASD.

In the light of our results, in patients with ASD, if the left ventricular wall is thin, MVP occurs due to a better left ventricular filling and a higher left ventricular ejection fraction. However, we believe that further research on left and right ventricular volume equation and leaflet co-aptation physiology is necessary.

References

1. Pocock WA, Barlow JB: An association between the billowing posterior mitral leaflet syndrome and congenital heart disease, particularly atrial septal defect. *Am Heart J* 1971;81(5):720-2.
2. Jeresaty RM: Mitral valve prolapse-click syndrome in atrial septal defect. *Chest* 1975;67(2):132-3.
3. Leachman RD, Cokkinos DV, Cooley DA: Association of ostium secundum atrial septal defects with mitral valve prolapse. *Am J Cardiol* 1976;38(2):167-9.
4. Menachemi E, Aintablian A, Hamby RI: Ostium primum atrial septal defect. Associated with mitral valve prolapse. *N Y State J Med* 1975;75(12):2234-5.
5. Lieppe W, Scallion R, Behar VS, Kisslo JA: Two-dimensional echocardiographic findings in atrial septal defect. *Circulation* 1977;56 (3):447-56.
6. Devereux RB, Perloff JK, Reichert N, Josephson ME: Mitral valve prolapse. *Circulation* 1976;54(1):3-14.
7. Savage DD, Garrison RJ, Devereux RB, Castelli WP, Anderson SJ, Levy D et al. Mitral valve prolapse in the general population. 1. Epidemiologic features: The Framingham Study. *Am Heart J* 1983;106:571-6.
8. Hickey AJ, Wolfers J, Wilcken DE. Mitral valve prolapse, prevalence in an Australian population. *Med J Aust* 1981;1:31-3.
9. Guy FC, MacDonald RP, Fraser DB, Smith ER. Mitral valve prolapse as a cause of hemodynamically important mitral regurgitation. *Can J Surg* 1980;23:166-70.
10. Lax D, Eicher M, Goldberg SJ: Mild dehydration induced echocardiographic signs of mitral valve prolapse in healthy females with prior normal cardiac findings. *Am Heart J* 1992;124(6):1533-40.
11. Diamond MA, Dillon JC, Haine CL, Chang S, Feigenbaum H: Echocardiographic features of atrial septal defect. *Circulation* 1971;43(1):129-35.
12. Kerber RE, Dippel WF, Abboud FM: Abnormal motion of the interventricular septum in right ventricular volume overload. Experimental and clinical echocardiographic studies. *Circulation* 1973;48(1):86-96.
13. Weyman AE, Wann S, Feigenbaum H; Dillon JC: Mechanism of abnormal septal motion in patients with right ventricular volume overload: a cross-sectional echocardiographic study. *Circulation* 1976;54(2):179-86.
14. Garcia-Dorado D, Garcia EJ, Bello L, Maroto E, Almazan A, Gomez A et al: Mitral valve prolapse secondary to right ventricular enlargement in patients with pulmonary hypertension after toxic rapeseed oil ingestion. *Eur Heart J* 1985;6(1):85-90.
15. Schreiber TL, Feigenbaum H, Weyman AE: Effect of atrial septal defect repair on left ventricular geometry and degree of mitral valve prolapse. *Circulation* 1980;61 (5):888-96.
16. Rankin JS, McHale PA, Arentzen CE, Ling D, Greenfield JC Jr, Anderson RW: The three-dimensional dynamic geometry of the left ventricle in the conscious dog. *Circ Res* 1976;39(3):304-13.

Author Information

Mert Kestelli, MD

Department of Cardiovascular Surgery and Cardiology, Surgery and Cardiology, Atatürk State Hospital