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MVP: A CLINICAL AND ECHOCARDIOGRAPHIC EXPERIENCE OF MITRAL VALVE PROLAPSE IN KHYBER PAKHTOONKHWA

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All the authors contributed significantly to the research that resulted in the submitted manuscript.

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ABSTRACT

Objective: To study the clinical presentation and echocardiographic findings in patients having mitral valve prolapse.

Methodology: This observational study was conducted in cardiology department LRH Peshawar, from December 2012 to March 2013. All patients with echocardiographic evidence of mitral valve prolapse were included in the study. Their clinical presentation and echo cardiographic characteristics were studied.

Results: A total of 142 patients have echocardiographic evidence of mitral valve prolapse were included. Their mean age was 19.45±7.32 years. Males were 57(40.14%). The most frequent symptoms were palpitation in 128(90.14%), chest pain in 111(78.16%), dizziness and vertigo in 103(72.34%).Mean BP was 101.34±14.32 mmHg systolic and 60.24±15.87 mmHg diastolic. Mean pulse rate was 82.46±18.74 per minute. Midsystolic ejection click was found in 79(55.63%), late systolic murmur in 47(33.08%), pansystolic murmur was observed in 7(4.9%). AF was found in 19(13.38%) and complete RBBB in 11(7.74%). Echo findings were having thin leaf mitral valve(Non Classic MVP) in 113(79.58%) and thick leaflet(classic MVP) in 29(20.42%).Severe mitral regurgitation in 8(5.6%), severe PAH 2(1.4%), Mean LA size was 4.13±1.3cm, Mean Left ventricular end diastolic diameter (LVEDD) was 5.13±1.4cm, Mean Ejection Fraction(EF) was 60.45±6.8%. Mean age of patients having classic MVP(29(20.42%)) was 31±7.8 years, 10(34.48%) have AF, 6(20.69%) have severe MR, 2(6.9%) have severe PAH ,mean LA size was 4.52±1.7cm, mean LVEDD was 5.56±2.1cm and mean EF was 53.76±8.5%.

Conclusion: Nonclassic MVP is more frequent than classic form. Patients with classic MVP is more often have older age, severe MR and ECG findings of arrhythmias than non classic form.

Key Words: Mitral Valve Prolapse (MVP), Mitral Regurgitation (MR), Pulmonary Artery Hypertension (PAH)

INTRODUCTION

Mitral valve prolapse (MVP) has been given many names including the systolic click-murmur syndrome, Barlow syndrome and redundant cusp syndrome.[1,2] It is a variable clinical syndrome which results from diverse pathogenic mechanisms of one or more portion of the mitral valve apparatus, valve leaflets, chordae tendineae, papillary muscle and valve annulus.[1] The vast majority of patients with MVP are asymptomatic and remains so throughout their lives. Although early studies called attention to an MVP syndrome, with a characteristic systolic non ejection click and various nonspecific symptoms such as fatigability, palpitations, postural orthostasis, anxiety and other neuropsychiatric symptoms as well as symptoms of autonomic dysfunction.[1]

In its nonclassic form, MVP carries a low risk of complications. In severe cases of classic MVP, complications include mitral regurgitation, infective endocarditis, congestive heart failure, and in rare circumstances, cardiac arrest, usually resulting in sudden death. The estimated prevalence of MVP in some studies is reported to be between 0.4% and 35%.[2-5] A frequent diagnosis of MVP and emphasis on possible complications may have physical, mental and socioeconomic effects in this young teenage population.

Patients with classic mitral valve prolapse have excess connective tissue that thickens the spongiosa. This weakens the leaflets and adjacent tissue, resulting in increased leaflet area and elongation of the chordae tendineae. Advanced lesions-also commonly involving the posterior leaflet-lead to leaflet folding, inversion, and displacement toward the left atrium.[6]

MVP is a common disorder with a variable clinical course that is determined by the presence and magnitude of mitral regurgitation (MR).[7] Given the prognostic implication of MR, identification of factors associated with progression is important for risk stratification and surgical decision making. Conventional 2-dimensional tomographic imaging has shown that the progression of MR is determined primarily by the degree of deformation of valvular structure.[8-10] However the mitral valve has a complex 3-dimensional (3D) morphology that may not be completely assessed with 2-dimensional imaging techniques.

The aim of this study was to know the clinical presentation and echocardiographic findings in patients having mitral valve prolapse.

METHODOLOGY

This observational study was performed in cardiology department, Lady Reading Hospital Peshawar from December 2012 to March 2013. Using the 5% prevalence, the minimum sample size was 73 patients but we extended our sample size to 142. All the patients of both genders fulfilling the echocardiographic mitral valve prolapse criteria were included in the study. MVP was defined according to current standard guidelines by 2 D echo cardiography as single or bileaflet prolapse at least 2 mm beyond the long-ax is annular plane, with or without leaflet thickening in the long-axis parasternal view and the other views. The two main types of MVP, the classic and non-classic MVP were defined as follows:

a) Classic MVP: The parasternal long-axis view shows >2 mm superior displacement of the mitral leaflets into the left atrium during systole, with a leaflet thickness of at least 5 mm.

b) Nonclassic MVP: Displacement is >2 mm, with a maximal leaflet thickness of <5 mm

Other echocardiographic findings that should be considered as criteria are leaflet thickening, redundancy, annular dilatation, and chordal elongation.

All patients with echocardiographically confirmed MVP were examined in detail using M-Mode, 2-D and color Echocardiography for classical, non-classical MVP, severity of mitral regurgitation, Pulmonary artery hypertension, other valvular lesions, LA size, Left ventricular end diastolic diameter(LVEDD), fractional shortening(FS) and Ejection fraction(EF).

All the patients with confirmed echocardiographic findings of MVP were then subjected to detail history by the Trainee cardiologist for various symptoms including chest pain wither typical or atypical, apprehension, palpitations, lightheadedness, presyncope, syncope, fits, shortness of breath, vertigo, dizziness, past History of TIA/CVA and infective endocarditis etc. After completing history, Cardiac examination was performed in detail with special emphasis on auscultatory findings of mid systolic click, late systolic murmur and pansystolic murmur. 12 lead Electro-cardiography (ECG) of all the patients were taken and seen for any evidence of arrhythmias including atrial fibrillation (AF), premature atrial contraction (PAC), premature ventricular contraction (PVC), supra ventricular tachycardia (SVT), complete heart block (CHB), right bundle branch block (RBBB) and ventricular tachycardia (VT) etc.

All the demographic, clinical, echocardiographic variable were entered to a specially designed proforma. SPSS version 16.0 software was used for statistical analysis.

RESULTS

A total of 142 patients having echocardiographic evidence of mitral valve prolapse were included in the study. Their mean

age was 19.45±7.32 years. Males were 57(40.14%) and females were 85(59.85%). The most frequent symptoms was palpitation in 128(90.14%), chest pain in 111(78.16%), dizziness and vertigo in 103(72.34%), apprehension in 93(65.5%), presyncope in 85(59.85%), shortness of breath in 41(28.87%), syncope in 24(16.90%). Mean BP was 101.34±14.32 mmHg systolic and 60.24±15.87 mmHg diastolic. Mean pulse rate was 82.46±18.74 per minute. Cardiac auscultatory findings including Midsystolic ejection click was found in 79(55.63%), late systolic murmur in 47(33.08%) and pansystolic murmur was observed in 7(4.9%). These findings are summarized in Table 1.

ECG findings including AF was found in 19(13.38%), complete RBBB was found in 11(7.74%), Premature atrial contraction (PACs) in 8(5.6%) cases, Premature ventricular contraction (PVCs) in 7(4.9%) and CHB in 01 patients (0.7%) as shown in Table 2. Two patients (1.4%) presented with history of CVA and 01(0.7%) with infective endocarditis.

Echo findings were having thin leaf mitral valve (Non Classic MVP) in 113(79.58%), thick leaflet(classic MVP) in 29(20.42%), Mitral regurgitation(MR) in 114(80.28%) including mild MR in 94(66.19%), moderate mitral regurgitation in 12(8.45%), severe mitral regurgitation in 8(5.6%), mild PAH in 7(4.9%), moderate PAH in 4(2.8%), severe PAH in 2(1.4%) patients, mean LA size was 4.13±1.3cm, mean left ventricular end diastolic diameter was 5.13±1.4cm, mean fractional shortening(FS) was 28.72±4.6% and mean ejection fraction(EF) was 60.45±6.8% as shown in Table 3. Among the 29 patients of classic MVP which make 20.42 % of the study population, mean age was 31±7.8 years, 10(34.48%) have AF, 6(20.69%) patient have severe MR, 2(6.9%) have severe PAH , mean LA size was 4.52±1.7cm, mean left ventricular end diastolic diameter was 5.56±2.1cm and mean ejection fraction was 53.76±8.5%. These findings are summarized in Table 4.

Table 1: Baseline and Clinical Characteristics of Patients with MVP

Variable	Frequency	Percentage
Males	57	40.15%
Females	85	59.85%
Mean Age(years)	19.45±7.32	
Mean systolic Blood pressure(mm of Hg)	101.34±14.32	
Mean Diastolic Blood pressure(mm of Hg)	60.24±15.87	
Mean pulse rate (Heart beats/min)	82.46±18.74	
Palpitation	128	90.14%
Chest pain	111	78.16%
Dizziness/ vertigo	103	72.34%
Apprehension	93	65.5%
Presyncope	85	59.85%
Dysopnea	41	28.87%
Syncope	24	16.90%
Midsystolic ejection click	79	55.63%
Late systolic murmur	47	33.08%
Pansystolic murmur	7	4.9%

Table 2: Electrocardiographic Characteristics of Patients with MVP

Variable	Frequency	Percentage
AF	19	13.38%
Complete RBBB	11	7.74%
Premature atrial contraction (PACs)	8	5.6%
Premature ventricular contraction (PVCs)	7	4.9%
Complete Heart Block	01	0.7%

Table 3: Echocardiographic Characteristics of Patients with MVP

Variable	Frequency	Percentage
Thin leaf mitral valve	113	79.58%
Thick leaflet	29	20.42%
Mild MR	94	66.19%
Moderate mitral regurgitation	12	8.45%
Severe mitral regurgitation	8	5.6%
Mild PAH	7	4.9%
Mod PAH	4	2.8%
Severe PAH	2	1.4%
Mean LA diameter(cm)	4.13±1.3	
Mean LV End diastolic Diameter(cm)	5.13±1.4	
Mean Fractional shortening	28.72±4.6	
Mean EF (%)	60.45±6.8	

Table 4: Echocardiographic and Clinical Characteristics of Patients with Classic MVP (n=26)

Variable	Frequency	Percentage
Age	31±7.8 years	
AF	10	34.48
Severe MR	6	20.69
Severe PAH	2	6.9
Mean LA size	4.52±1.7cm	
Mean LVEDD	5.56±2.1cm	
Mean EF %	53.76±8.5	

DISCUSSION

This study was the first study in our local population to know the clinical and echo cardiographic variables and complications of patients with mitral valve prolapse. Our study population was the patients came for the cardiologist opinion regarding their symptoms. They all were subjected to transthoracic echocardiography. A total of 142 patients having confirmed echocardiographic mitral valve prolapse were included for detailed history regarding their symptoms, examination and various echocardiographic variables. Subjective evidence of prior to the strict criteria for the diagnosis of mitral valve prolapse, the incidence of mitral valve prolapse in the general population varied greatly.[6] Some studies estimated the incidence of mitral valve prolapse at 5 to 15 percent or even higher.[11,4] Recent elucidation of mitral valve anatomy and the development of three-dimensional echocardiography have resulted in improved diagnostic criteria, and the true prevalence of MVP based on these criteria is estimated at 2-3%.[2]

As part of the Framingham Heart Study, the prevalence of mitral valve prolapse was estimated at 2.4%. There was a near-even split between classic and nonclassic MVP, with no significant age or sex discrimination.[12] But our study shows that most of the patients have non classic Mitral valve prolapse i.e. about 80% as compare to classic form that is about 20%. That about one fifth of patients with Mitral value prolapse have classic form. Contrary to the study of framingam, most of the patients with classic form were older and more often hemodynamically significant mitral valve as compare to Nonclassic form i.e. about 34% of patient with classic MVP have AF as compare to 13% of the whole study population, 20.69% patient have severe MR as compare to 5% for whole study population, all patients with severe PAH were having classic MVP, mean LA size was 4.52±1.7cm for classic MVP as compare to 4.1 for whole study population, mean Left ventricular end diastolic diameter was 5.56 cm for classic MVP as compare to 5.1% for whole study population and mean ejection fraction was 53.76% as compare to 60% for whole study population including classic and non classic MVP. This hemodynamically significant findings in patients with classic MVP as compare to whole study population including both classic and Nonclassic MVP is because of 34% of these patients have severe MR. MVP is observed in 7% of autopsies in the United States.[13]

Historically, the term mitral valve prolapse syndrome has been applied to MVP associated with palpitations, atypical chest pain, dyspnea on exertion, low body mass index, and electrocardiogram abnormalities in the setting of anxiety, syncope, low blood pressure, and other signs suggestive of autonomic nervous system dysfunction.[14] our study is supported by these fact as about 90% of our patients have palpitation, 78% have chest pain, 72% have dizziness, 65% have apprehension, presyncope in 60% while syncope was the presenting symptom in 16.90%. In our study arrhythmia occurs in more than 10% of patients which is supported by Terechtchenko et al, they observed supraventricular arrhythmias more frequently in MVP describing its association with increased parasympathetic tone.[15] The arrhythmias were more often in patients with classic form as compare to non classic form.

In this study most patients with MVP have mild MR, which usually have a benign course. Mitral valve prolapse is frequently associated with mild mitral regurgitation, where blood aberrantly flows from the left ventricle into the left atrium during systole.[16] In the United States, MVP is the most common cause of severe, non-ischemic mitral regurgitation.[2] This is occasionally due to rupture of the chordae tendineae that support the mitral valve.[14] Severe mitral regurgitation occurs only in about 5.6% of patients with MVP, most of them were having classic form of prolapsed than non classic form. The most frequent cause of non-ischemic Mitral regurgitation is Rheumatic heart disease in developing countries like us. Generally MVP is benign. However, MVP patients with a murmur, not just an isolated click, have an increased mortality rate of 15-20%.[13] The major predictors of mortality are the severity of mitral regurgitation and the ejection fraction.[17]

In a study by Maleva et al, found that there is significant reduction in global strain in classic form of MVP as compared to non-classic, while in non-classic type the reduction in longitudinal deformation was detected only in septal segments. Transforming growth factor-ß1 and ß2 serum levels are elevated in classic as compare to nonclassic. so the left ventricular functions deterioration and existence of primary cardiomyopathy in young adults with MVP, which may be caused by increased transforming growth factor-ß signaling.[18] Similar was the case in our study as the mean ejection fraction was less in classic form of mitral valve prolapse than non classic form but we have

studied it only by 2D and M-mode Echocardiography. The incidence of mitral valve prolapse in patients with atrial septal defect (ASD) has been reported to be high.[19-22] This incidence is about 35%. In their study, Lieppe et al, have found the incidence to of MVP in patients with ASD to be 95% by means of two-dimensional echocardiography.[23] But we have not studied the ASD association with mitral valve prolapse. The reason for this high incidence has not been completely explained yet. The discussion on the high incidence of MVP in patients with ASD has focused on two hypotheses.[24] 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.

Our study shows that it occurs more frequently in females as compare to males which is supported by the findings of Sattur et al.[25] As in our study most of the study have included only the referred patients. Studies in the1970s and early 1980s based diagnosis on either auscultation or nonspecific echocardiography views. Barlow et al, studied 12,050 black children for auscultatory evidence of MVP and reported prevalence of 1.85% and 1.80% in 11 to 14 year olds and 15 to 18 year olds, respectively.[26] This wide variation of MVP prevalence noted in the literature could be secondary to the variety of study populations, ranging from healthy volunteers to hospital-based populations, and the use of several diagnostic modalities including auscultation, phonocardiography, nonspecific views of two-dimensional echocardiography and M-mode echocardiography.[27,28] Freed et al, using the current standard definition of MVP in a large community-based sample of adults between 26 and 84 years of age, concluded that the prevalence of MVP and its complications was lower than previously expected.[12] Earlier studies suggested that the prevalence of MVP increases throughout childhood and peaks in the early 20s.[2,29] This probably explains the lack of significant differences, although they found numerical differences in prevalence, with more female subjects having MVP than the male subjects.

In our study most of the MR noted was of trace severity. Our findings are in concordance with previous studies demonstrating a similar association between MVP and MR.[12,30,31] Also noted in our study, pulmonary artery hypertension was associated only in patient having severe mitral regurgitation.

LIMITATIONS

The echocardiography technique and interpretation depended on the individual cardiologist. We used only 2 D and colour Doppler transthoracic echocardiography. We have not used the three dimensional echocardiography which is more specific and reliable for mitral valve anatomy.

CONCLUSION

Nonclassic MVP is more frequent than classic form. The patient more oftenly present with anxiety, palpitations and atypical chest pain. Patients with classic MVP are more often have older age, severe MR and ECG findings of arrhythmias than non classic form.

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