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LEFT VENTRICULAR SYSTOLIC DYSFUNCTION AS A SURROGATE FOR RHEUMATIC MYOCARDITIS IN PATIENTS WITH ISOLATED RHEUMATIC MITRAL STENOSIS

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ABSTRACT

Objective: To know the frequency of LV dysfunction as a surrogate for rheumatic myocarditis in patients with isolated mitral stenosis (MS) after successful percutaneous transluminal mitral commissurotomy (PTMC).

Methodology: This Descriptive study was conducted from January to December 2011 in Cardiology Department of Lady Reading Hospital Peshawar. Patients with severe isolated MS undergoing echocardiography before and after PTMC were included.

Results: A total of 141 patients with severe isolated MS have undergone successful PTMC were studied. Females were 97(68.8%). Mean age was 26.74±7.24years.TTE was performed before PTMC showing mean MVA 0.8cm±0.2cm^2. Mean MV gradient was 22.3± 16.7 mm of Hg. Mean LA diameter was 4.8± 2.4 cm. AF was found in 63 (44.36%). Mean RVSP was 55.67±23.41mm of Hg. LV systolic dysfunction i.e. EF< 50 % was found in 24 (17.02%) of patients. The LV systolic function was mildly impaired in 13 (9.2%), moderately impaired in 7(4.9%) and severely impaired in 4 (3%) of the study group. TTE 24 hours after PTMC shows mean left atrial diameter of 4.3±2.3cm, mean MV area of 1.74±0.41cm^2, mean MV gradient of 5.21±4.9 mm of Hg, mean RVSP 29±16.34 mm of Hg, while left ventricular systolic dysfunction was present in 14.3% of patient with mild degree impairment in 10 (7.1%), moderately impaired in 6 (4.2%) and severely impaired in 4 (3%) of the study group.

Conclusion: The LV systolic dysfunction which is used as a surrogate of rheumatic myocarditis occurs in about 14% of patients with isolated severe MS after successful PTMC.

Key Words: Rheumatic Myocarditis, Mitral Stenosis, Left Ventricular Systolic Dysfunction

INTRODUCTION

Rheumatic heart disease (RHD) is one of the leading causes of cardiac morbidity and mortality in developing countries. Recent reports has documented incidence of RF as high as 206/100,000 and RHD prevalence as high as 18.6/1000 though there are variations in the different geographical areas. The mitral valve is the most frequently affected valve in rheumatic heart disease. It is solely affected in 25% and is affected with other valves in 40% of patients.[1]

In Pakistan rheumatic heart disease remains endemiclike other third world countries. The prevalence of RHD in Pakistan was found to be 22/1000 in inner Lahore and 5.7/1000 in rural Pakistan in recent studies.[2]

Mitral stenosis can lead to enlargement of the left atrium leading to increased risk of thrombus formation.[3] In the last two decades; percutaneous transluminal mitral commissurotomy (PTMC) has become the treatment of choice for patients with symptomatic rheumatic mitral stenosis (MS). Since its introduction in 1984 by Inoue et al, BMV by Inoue technique has been widely used in the treatment of mitral stenosis.[4] Several studies have reported good immediate, short-term and long-term results.[5,6]

The incidence of Mitral Stenosis (MS) has decreased in developed countries due to impressive alleviation of rheumatic fever. However, in our part of the world it is one of the major health problems and a great challenge for the health care professionals.[7-9]

Patients with severe symptomatic MS, 50% or more have chronic AF.[10] Both LA enlargement and AF may alter the course of disease by providing the potential source of mural thrombi (LA clot) and the risk of systemic embolism.[11,12] Apart from cerebral embolization these emboli may result in occlusion of arteries of the extremities, occlusion of the aortic bifurcation and visceral or myocardial infarction.[10]

Left ventricular function in patients with mitral stenosis has been investigated during the last decades. First angiographic studies found higher left ventricular end-systolic volumes and lower ejection fraction in patients with mitral stenosis than in controls.[13,14] Ventriculography showed distorted contraction of the postero-basal segment and occasionally anterior hypokinesis, that was related to the rigidity and immobilization of mitral valve complex, proposed to be due to scarring of the mitral valve complex and fibrosis of the papillary muscle.[13-16] In some angiographic studies, generalized rather that regional LV dysfunction was found in patients with mitral stenosis that was explained by rheumatic myocarditis.[17] Low ejection performance indexes could be often found in mitral stenosis and were related to altered loading conditions.[18]

Rheumatic mitral stenosis is one of the commonest valvular heart lesions in developing countries. Since rheumatic heart affection is more severe and the degree of valvular damage is greater in developing countries than in industrialized Western communities, it seems appropriate to examine the frequency and extent of left ventricular dysfunction in our patients.

METHODOLOGY

This descriptive cross-sectional study was conducted from January to December 2011 in Cardiology Department of Lady Reading Hospital Peshawar. Patients with severe isolated mitral stenosis undergone successful percutaneous transluminal mitral commissurotomy (PTMC) were included by non probability consecutive technique and were evaluated first with transthoracic echocardiography (TTE) and then with transesophageal echocardiography (TEE) at echocardio-graphy suite of Lady Reading Hospital, Peshawar before PTMC and 24 hours after PTMC. The sample size was calculated using 10 % of prevalence of rheumatic myocarditis in patients with mitral stenosis.[17,19] Transthoracic Echocardiography had been used in this study for measurements of cardiac chamber dimensions and for the assessment of left ventricular performance. Severe mitral stenosis was defined by echocardiographic criteria as associated with mean transvalular gradient of more than 10 mm of Hg, pulmonary artery pressures of more than 50 mm of Hg and a valve area of less than 1 cm^2.

Transthoracic echocardiography was performed by a standard technique using Toshiba Xario 2100 and Philips D H 11echocardiographic machines pre and 24 hour post PTMC. M-mode measurements were recorded according to American Society of Echocardiography criteria.[20] The mitral valve area was measured by continuous wave Doppler using the pressure half time method. The mean transmitral diastolic pressure was estimated from the maximal transmitral flow velocity using a modified Bernoulli equation. LA diameter was taken in the parasternal long axis view in M-mode at end systole. Measurements were taken in three beats in patients with normal sinus rhythm and in ten beats in atrial fibrillation and the mean values were taken for analysis. Assessment of left ventricular performance was performed using Simpson method since Low ejection performance indexes could be often found in mitral stenosis and may be related to altered loading conditions so TTE was repeated 24 hours after PTMC to know the true incidence of myocarditis after improving preload with successful PTMC.[18]

All the patients with Diabetes, hypertension, suboptimal PTMC, mitral regurgitation more than grade 1, ECG evidence of Coronary artery disease or any echocardiographic evidence of ischemia / segmental wall motion abnormalities were excluded from the study. Patients with suspected peripartum cardiomyopathy were also excluded from the

Table 1: Demographic and Echocardiographic Variables

Variables	Number (%)
Male	44 (3.12%)
Females	97 (68.8%)
Age(Years)	26.74±7.24
Atrial fibrillation	63 (44.36 %)
Mean Left atrial diameter(cm)	4.8± 2.4
Mean Mitral valve area(cm^2)	0.8 ±0.2
Mean Mitral valve gradient(mm of Hg)	32.3± 16.7
Right ventricular systolic pressure (mm of Hg)	55.67±23.41
Right ventricular Diameter(cm)	2.37±0.56

study. The demographic, clinical and echocardiographic variables were enter through a specially designed proforma.

Optimal PTMC was defined as post PTMC mitral valve area of $\geqslant$1.5cm^2 or at least 25 % increase in valve area with no more than one grade increase in MR and with no major complication.

All the data were analyzed by SPSS (Statistical Package for Social Sciences) Version 19.0 for Windows. Categorical variables were expressed as numbers and percentages while continuous variables were expressed as mean±SD (Standard deviations).

RESULTS

We included 141 patients with severe isolated mitral stenosis have undergone successful PTMC in this study. Males were 44(31.2%) and females were 97(68.8%). Mean age was 26.74±7.24years. Pre PTMC TTE shows mean mitral valve area of 0.8±0.2cm^2. Mean mitral valve gradient was 22.3±16.7 mm of Hg. Mean left atrial diameter was 4.8±2.4 cm. AF was found in 63(44.36%). Mean right ventricular diameter was 2.37±0.56 mm of Hg. mean right ventricular systolic pressure was 55.67±23.41 mm of Hg. Mean left ventricular end diastolic diameter was 4.32± 0.47cm.These are shown in Table 1.

Left ventricular systolic dysfunction i.e. ejection fraction (EF)< 50 % was found in 24(17.02%) of patients. The left ventricular systolic function was mildly impaired (40-50% EF) in 13(9.2%), moderately impaired (30-40%) in 7(4.9%) and severely impaired (<30%) in 4(3%) of the study group. These are shown in Table 2. After 24 hours of procedure Transthoracic echo shows mean left atrial diameter of 4.3±2.3cm. Mean mitral valve area of 1.74±0.41cm^2, mean mitral valve gradient of 5.21±4.9 mm of Hg, mean RVSP 29±16.34 mm of Hg and Mean Left ventricular end diastolic diameter was 4.43±0.48cm. While 24 hour post PTMC left ventricular systolic dysfunction was present in 14.3% of patient with mild degree impairment (40-50% EF) in 10 (7.1%), moderately impaired (30-40%) in 6 (4.2%) and severely impaired (<30%) in 4 (3%) of the study group. 24 hour post PTMC results re shown in Table 3.

DISCUSSION

The developing countries represent the true evidence and spectrum of rheumatic heart disease. There is variable data regarding the existence of true rheumatic myocarditis. We most often come across clinical scenario in which the young patients have globally impaired left ventricular function with isolated mitral stenosis having no evidence of ischemia, hypertension, peripartum cardiomyopathy or any other known cause for impaired left ventricular function. A few studies in the developing and developed countries shows that the possible mechanism for this may be Rheumatic myocarditis or myocardial factor.[17] We designed this study to know the frequency of globally impaired LV function as a surrogate of rheumatic myocarditis in patients with isolated rheumatic mitral stenosis have undergone successful

Table 2: Categories of Systolic Dysfunction (Ejection Fraction<50%)

Categories	Percentages (n) before PTMC	Percentages (n) after PTMC
Mild systolic dysfunction (EF 40%-50%)	9.2% (13)	7.1% (10)
Moderate systolic dysfunction (EF 30-40%)	4.9% (7)	4.2% (6)
Severe systolic dysfunction (EF<30%)	3% (4)	3% (4)

Table 3: Echocardiographic Parameter 24 hours After Successful PTMC

Variables	Values
Mean Left atrial diameter(cm)	4.3± 2.3
Mean Mitral valve area(cm^2)	1.74 ±0.41
Mean Mitral valve gradient (mm of Hg)	5.21± 4.9
Right ventricular systolic pressure (mm of Hg)	29±16.34
Right ventricular Diameter(cm)	2.20±0.57
Left ventricular end diastolic diameter(LVEDD)in cm	4.43±0.48

PTMC.

The impairment in left ventricular function in our study population with isolated mitral stenosis was about14%. Harvey et al, found an incidence of 13 per cent in their patients, which is supporting our findings.[19] McDonald found in his study that the globally impaired LV which is a marker of rheumatic carditis have an incidence of 30 per cent.[21] While Kennedy et al, found that 37 per cent of patients with mitral stenosis had evidence of impaired myocardial contraction.[22] Kamblock et al, question the existence of true rheumatic myocarditis who studied 95 patients in acute rheumatic fever.[23] The reason might be that they just studied during hospitalization for rheumatic fever and not studied its chronic sequale. This high incidence in our patients was not completely unexpected as increased incidence of rheumatic fever and rheumatic heart disease in this part of the world.[2] Two factors might have been responsible; first, patients in our study had severe mitral stenosis necessitating percutaneous transluminal commisurotomy. In such a selected group of patients the degree of mitral stenosis and functional disability would be greater than in a random selection of patients present in other studies. Secondly, the severe and florid nature of rheumatic fever, its early age of onset, and the significant hemodynamic burden in our patients is different from that in western countries. A similar demographic for rheumatic fever and rheumatic heart disease is described by Egyptian investigator the socioeconomic status of which is just like our country.[24]

In our study the mean right ventricular pressure was more than 50 mm of Hg which is about the same as described by Botras et al.[25] Hypokinesis of the right ventricular contraction have been reported in mitral stenosis by Heller and Carleton, which again supporting our findings in favour of rheumatic myocarditis.13 of their 25 patients, 20 showed distortion, immobility, and rigidity of the postero-basal area of the left ventricle. Similarly, Horwitz et al, described localized abnormalities in the wall of the left ventricle for which Fibrosis in or near the papillary muscles was postulated as a possible cause.[14] However, the reduction of myocardial performance in our patients are consistent with the view that a generalized abnormality of myocardial mechanical performance is responsible for the occurrence of left ventricular dysfunction in some patients with mitral stenosis. The present study suggests that abnormalities in contractility of the left ventricular myocardium can be responsible for the impaired myocardial function in patients with mitral stenosis and that such impairment is clinically significant. It may be a contributing factor in some patients who have an unsatisfactory clinical response to mitral commissurotomy. Histopathological studies in patients with mitral stenosis showed that the failure of improvement after mitral commissurotomy was related to the extent of myocardial involvement by the rheumatic process.[26]

The current interesting work by Yıldırımtürk et al, evaluated deformation parameters-longitudinal and circumferential strain and strain rate in patients with mitral stenosis and apparently normal left ventricular function with Velocity Vector Imaging.[27] Longitudinal strain and strain rate were reduced at all the levels of the left ventricle: base, mid-ventricle and apex; circumferential strain and strain rate at the mid-ventricular level also were significantly reduced. Higher myocardial performance index suggested impaired myocardial performance. This study indicates that the subclinical global left ventricular dysfunction in patients with mitral stenosis is a sequence of rheumatic myocarditis. Subclinical myocardial dysfunction due to rheumatic myocarditis can be responsible for part of the symptoms in symptomatic patients with mild-moderate mitral stenosis.

CONCLUSION

The left ventricular systolic dysfunction which is used as a surrogate of rheumatic myocarditis occurs in about 14% patients with severe mitral stenosis have undergone successful PTMC.

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