HOW SEVERE IS SEVERE MITRAL STENOSIS?

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SUMMARY

Mitral valve is the target in most of patients having rheumatic fever. Detailed information on 1136 adults patients with pure mitral stenosis included chambers dimensions (left atrium, left ventricle and right ventricle), trans-valvular gradient (MVG), mitral valve area (MVA) systolic pulmonary artery pressure (SPAP) as documented by tricuspid regurgitation on two dimensional echocardiography and calculated by doppler was obtained. Mitral valve gradient increases to 5 mmHg wen the area is 2.1 cm^2. MVG increases to 10 mmHg when MVA is 1.4 cm^2. Left atrium starts increasing as MVA decreases below 2.18 cm^2. Pulmonary artery pressure as estimated by Tricuspid regurgitation increased beyond 30 mmHg when MVG increases more than 8mmHg and increases to 45 mmHg when MVG increases to 12 mmHg. Pulmonary artery pressure increases to 60 mmHg when MVG increases to 13 mmHg. Pulmonary artery pressure starts building up as mitral valve area decreases below 1.8 cm^2. Pulmonary pressure increases above 45 mmHg as MVA decreases to 1.4 cm^2. Patients develop severe pulmonary hypertension as pressure escalates to 60 mmHg when MVA reduces below 0.8 cm^2. MVA is the major criterion for classifying the severity of MS. In patients with MVA less than 1.5 cm^2 seem have severe MS as LA is enlarged and SPAP is elevated. Early intervention may be considered in patients with MVA less than 1.5 cm^2.

INTRODUCTION

Rheumatic heart disease remains a common entity in developing countries.[1-3] It afflicts children from lower social background and follows a relentless course in certain cases. Introduction of new modalities to better visualize the valvular structure and balloon based therapeutics has renewed interest in this field. Mitral valve is the target in most of patients having rheumatic fever. Mitral stenosis is one of the commonest presentations following rheumatic fever.[2]

There are still various aspects of this ailment which are shrouded with mystery.[4,5] Simple basic questions pertaining to pathophysiology remain unanswered! We need to know as to what is the relationship between trans-valvular gradient and mitral valve area? When does left atrium enlarge during the course of disease? When does pressure start increasing in pulmonary circulation? Above all when do we classify mitral stenosis as severe mitral stenosis and on which criteria?[5] A comprehensive echocardiographic study with pulsed wave, continuous wave and colour doppler offers comprehensive evaluation of cardiac dimensions, valvular structure and mobility, trans-valvular gradient, documentation of any regurgitation and quantification of pulmonary artery pressure. Trans-thoracic echocardiography is usually sufficient for day to day decision making. This can be repeated at intervals to gauge the progression of disease with little patient discomfort.[6-10] Trans-esophageal echocardiography may be utilized for added information when required and specially for detailed anatomi-

cal and physiological evaluation of atrial appendage.[8-10]

A retrospective, computer data based study on echocardiography and doppler was performed on patients with pure mitral stenosis to elucidate the above-referred questions. The aim was to study the interactions between different parameters and understand the course of illness and to help to classify the severity of mitral stenosis based on echocardiography and doppler.

MATERIAL AND METHODS

Echocardiographic examinations were performed by two well trained echocadiographers on all patients and was recorded on video. All measurements are made on line and recorded. The recordings were reviewed by a member of the staff and a computer based report was generated.

Echocardiographic data of all the patients examined at Cardiology department, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar is entered in a custom designed software in Unix based environment in a central computer. All adult patients undergoing echocardiography in the last four years have been included in this study. Information was sought on adult patients with pure mitral stenosis with no regurgitation and other significant lesion. Detailed information on 1136 patients with pure mitral stenosis included chambers dimensions (left atrium, left ventricle and right ventricle), transvalvular gradient (MVG) peak and mean, mitral valve area (MVA) on two dimensional echocardiography and calculated by doppler systolic pulmonary artery pressure (SPAP) as documented by tricuspid regurgitation. Patients with no tricuspid regurgitation were assigned to normal pressure group (20 mmHg). All the data was entered in statistical software package SPSS for windows. Relationships between different parameters was sought. Left atrial dimension was studied against mitral valve area and gradient. Pulmonary artery pressure was analysed in relationship to mitral valve area and gradient and left atrial size. Relationship between mitral valve gradient and area were studied. Relation ship between left ventricular diameter and right ventricular diameter versus other parameters were studied.

RESULTS

Data of 1135 adult patients was analysed and following observations were made. Relationship of mitral valve area and gradient: Mitral valve gradient increases with reduction in valve area. Mitral valve gradient increases to 5 mmHg wen the area is 2.1 cm^2. MVG increases to 10 mmHg when MVA is 1.4 cm1. For a comparative MVG of 15 mmHg the corresponding area is 0.6 cm2. (Fig.–I)

Relationship of left atrium to mitral valve gradient: Left atrium appears to be a sensitive parameter dilating during the early course of disease. Left atrium starts dilating as soon as MVG increases more than 6mmHg. It increases further with further increase in gradient.

Relationship of Mitral valve area and gradient

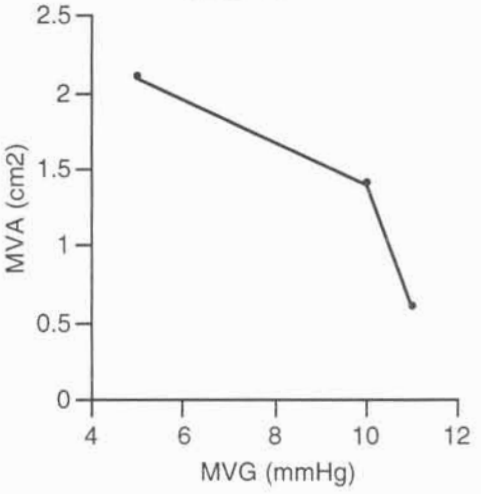

FIG. I

Relationship of left atrium to mitral valve area: Left atrial size starts increasing as MVA decreases below 2.18 cm^2. Even in mild stenosis, left atrium is enlarged. Left atrium dilates more with further reduction in valve area as estimated on planimetry and derived by doppler.

Relationship of Pulmonary artery pressure to mitral valve gradient: Pulmonary artery pressure as estimated by Tricuspid regurgitation increases beyond 30 mmHg when MVG increases more than 8mmHg and increases to 45 mmHg when MVG increases to 12 mmHg. Pulmonary artery pressure increases to 60 mmHg when MVG increases to 13 mmHg. As MVG increase SPAP elevates correspondingly, though the curve flattens as MVG increases beyond 10 mmHg. (Fig.–II)

Relationship of Pulmonary artery pressure to mitral valve area: Pulmonary artery pressure starts building up as mitral valve area decreases below 1.8 cm^2. SPAP rises above 30 mmHg as MVA decreases below 1.8 cm2. Pulmonary pressure increases above 45 mmHg as MVA decreases to 1.4 cm^2. Patients develop severe pulmonary hypertension as pressure escalates to 60 mmHg when MVA reduces below 0.8 cm^2. (Fig.–III)

Left ventricular size did not depict any significant relationship with MVA, MVG, SPAP or left atrium. Fractional shortening did not change significantly with the progression of disease. Right ventricle did not have statistically significant relationship with the severity of disease as evident by MVA and MVG or with pulmonary hypertension in this range.

DISCUSSION

Rheumatic heart disease remains unchecked in most developing countries.[1-3] In many developed countries the disease is seen in the immigrant population. Mitral valve disease is the commonest sequel to rheumatic fever. There has been a renewed interest in the natural history and haemodynamic developments in the course of disease with new modalities being available to study them.[1] Echocardiography and doppler allow cardiologists to measure chambers sizes and function, determine mitral valve area and document physiologi-

Relationship of Pul hypertension and Mitral valve gradient

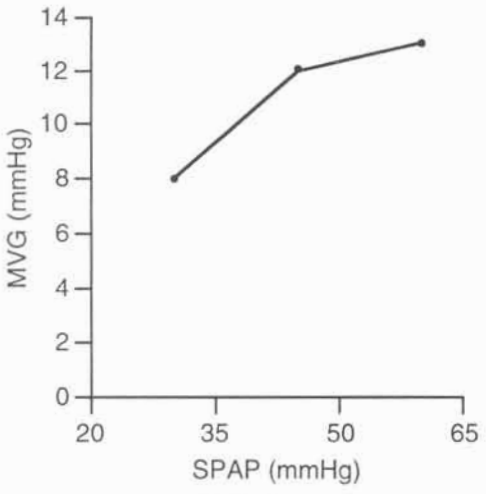

FIG. 2

Relationship of Pulmonary hypertension and Mitral valve area

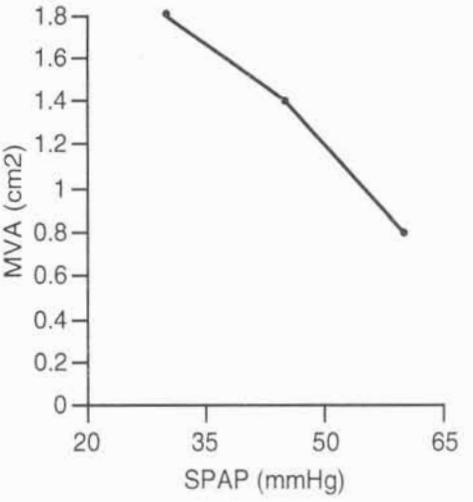

FIG. 3

cal parameters including transvalvular gradients and pressures. This non-invasive modality can comprehensively evaluate anatomy and physiological results.[7-11] With the introduction of invasive cardiology especially percutaneous mitral valvuloplasty, mitral stenosis has been the focus of intense research into the disease process and the management.[12,13]

With the technological advances new questions are being asked? What is the basis of classifying severity of any lesion in general and mitral stenosis in particular? When does left atrium enlarge in the course of disease? When does pulmonary hypertension develop? What is the relationship between mitral valve area and trans-mitral gradient?[6] What should be the optimal timing of intervention? Can early intervention deliver better results with reduced morbidity and mortality? Or should we wait till the disease has progressed too far?[12-13]

Mitral valve area estimation by planimetry by transthoracic echocardiography (TTE) remains an important parameter for the assessment of severity mitral valve stenosis.. MVA was estimated by Gorlin and Gorlin in 1951 by employing hydrodynamic laws to the data acquired by cardiac catheterization.[4] MVA is being estimated by trans-esophageal echocardiography (TEE) and various doppler techniques.[14] Magnetic resonance imaging is being employed to depict valvular abnormalities and estimate MVA.[15] MVA estimated by TTE remains the gold standard in day to day management of patients and has been shown to correlate with derivation of MVA by other techniques and modalities.[14-15] New insights are being sought by employing stress echocardiography. Exercise echocardiographic and doppler parameters can be dramatically different in patients with similar resting values. This can further assist us in selecting patients for medical or otherwise opt for interventional therapy.[15-18] Conventionally MVA lesser than 1.0 cm^2 has been regarded as severe mitral stenosis and between 1.0 and 1.5 cm^2 as moderate stenosis and more than that as mild stenosis. Perhaps time has come to reassess this criteria of this classification!

This large data offers a unique opportunity to study the interplay of various parameters and understand the evolution of disease. Left atrium seems to be a very sensitive parameter of disease severity, enlarging very early in disease. Left atrium starts dilating as soon as MVA reduces below 2.18 cm^2 and MVG rises above 6mmHg. This signifies sufficient pressure build up to cause left atrial dilatation at quite an early stage of disease course.

Pulmonary artery pressure estimated by tricuspid regurgitation seem to start building up as MVA decreases below 1.8 cm^2 and MVG increases more than 8mmHg. It seems that important and significant haemodynamic changes take place early in the course of disease usually classified as mild disease. By the time MVA has reduced to moderate severity, left atrium has dilated more and pressure in pulmonary circulation has increased further.

What really is the criteria of classifying the severity of mitral stenosis?[12,18,21] Does this significant enlargement of left atrium with attended risk of developing atrial arrhythmias and development of pulmonary hypertension not enough evidence that the disease is significantly severe? While classifying severity of mitral stenosis it should be born in mind that all these parameters are being assessed at rest and the patient is bound to alter the haemodynamics during effort. Mitral valve gradient increases during effort with varying level of increase in pulmonary artery pressure. Stress echocardiography should be considered more frequently and perhaps routinely in mild to moderate disease to select patients who merit early intervention.

In contrast to above referred classification, this new comprehensive data suggests that the classification should be revised. It is proposed that patients with mitral stenosis with MVA less than 1.5 cm^2 should be classified as critical or severe stenosis. By this time MVG has increased significantly, LA is enlarged and pulmonary hypertension has set in.

Balloon mitral valvuloplasty is being recognised as a preferred technique for patients with mitral stenosis, specially in young, pregnant and those who had undergone intervention before.[19-22] With the evidence that early intervention delivers better long term results with reduced attended morbidity, a strong case can be advocated for early intervention with few or no symptoms.[22]

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