AORTIC STENOSIS – HOW SEVERE IS SEVERE?

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SUMMARY

Computer based echocardiographic data of 544 adults patients with pure Aortic stenosis was retrieved from the last 03 years on line record. The data was analyzed employing SPSS 8.0 version. Mean age was 37.95 ± 21.6 years. Mean Left ventricular end diastolic dimension was 5.08 ± 1.1 cm and end systolic dimension was 3.59 ± 1.1 cm. Left ventricular size did not enlarge with increase in aortic valve gradient (AVG), age. Left atrial size was 3.95 ± 1.23 cm and increased with increase AVG RV and or Left Ventricular (LV) function. RV size was 2.05 ± .56 cm. It increased with increase in LV end diastolic, diameter and LV wall thickness. Right ventricular (RV) size increased beyond 2.5 cm as LA increased more than 4 cm. RV size was inversely related to fractional shortening Fractional shortening (FS) reduced with increase in LV size beyond 5.8 cm (p>.0001). FS correlated inversely with increase in LA (p<.0001). Peak aortic valve gradient was (AVG) 56.96 ± 24.67 mmHg and mean AVG was 34.38 ± 15.52. AVG had increase correlation with FS. LV wall thickness increased with increase in AVG. (p<. 001). AVG mean of 59 mmHg and peak of 74 mm Hg seemed critical as LV hypertrophies on further increase. To conclude, enlasgement of LA, RV, and LV are markers of severe As, FS reduces as AS progresses and peak AVG of 74 mmHG and mean gradient of 50 mmHg shall be the standard for documenting severe AS in adults.

INTRODUCTION

Many asymptotic patients with hemodynamically severe obstruction are now identified by echocardiography. The increasing use of echocardiography, especially over the past 15yEArs or so, has dramatically changed our understanding of the prevalence and progression of aortic valve stenosis. The advent of more effective non-invasive monitoring of ventricular function, improvement in prosthetic valves, advances in valve-reconstructing techniques, and the development of useful guidelines for choosing the proper timing of surgical intervention have all worked in concert to improve the outcome.[2-4]

Aortic stenosis is the mechanical obstruction to blood flow across the aortic valve, and is found to be present in a growing percentage of adults over the age of 65 years. The clinical outcome is related to presence or absence of symptoms and is extremely poor once features of angina, syncope and congestive heart failure become apparent.

In aortic stenosis, a hemodynamic burden is placed on the ventricles, initially tolerated, but eventually leading to hypertrophy. Left ventricular hypertrophy causes to increase in diastolic filling pre3ssue leading to diastolic dysfunction, this leads on the systolic dysfunction which culminates in left ventricular failure. Symptoms of aortic stenosis are dyspnoea, angina and syncope.

On the part of the physicians, it is critical for them to be able to diagnose the severity of aortic stenosis on the basis of various parameters such as, transvalvular gradient, aortic valve area and aortic-jet velocity, and advise intervention at the appropriate stage.[3,4]

When does left ventricular wall hypertrophy during the course of disease. Does left ventricle respond to increasing gradient by dilating. When does right ventricle dilate in the course of disease. Does left atrium dilate as a response to increasing aortic valve gradient? Echocardiography and doppler provide useful information to study this interaction of physiological stress and anatomical responses.

The aim of this study was to look at various parameters and correlate them to assess the critical point in the course of the disease, where surgical intervention might be contemplated. There are many questions regarding basic heaemodynamic which remain unanswered.

MATERIALS AND METHODS

This is a retrospective; computer data based study conducted in the Cardiology Department of PGMI, LRH Peshawar. Well supervised echocardiographic examinations were carried out on 544 patients. The data used in this study was retrieved from computer records of patients over the last 3 yrs.

The patients were all adults and presented with moderate to severe aortic stenosis. Peak aortic valve gradient AVG (P) was correlated with factors such as left ventricular end-diastolic diameter LV (ED), interventricular septal thickness (IVS), left atrial diameter (LAD) and aortic diameter. Mean aortic valve gradient AVG (M) was studied against fractional shortening (FS), left ventricle posterior wall thickness (LVPW), left ventricle end systolic diameter (LVES). Relationship between FS and LV (ED), IVS, LVD, aortic diameter and that between right ventricle diameter RVD and LV (ED), LAD, IVS, aortic diameter and FS considered were studied.

All data was entered and processed in the Statistical Software Package SPSS 8.0 for Windows.

RESULTS

The total number of patients was 544, which included 35 % of females and 65 % of males. Mean age was 37.95 ± 15.16 years. in this cohort data LV end diastolic diameter was 5.08 ± 1.12 cm and LV and systolic diameter was 3.59 ± 1.12 cm and fractional shortening was 29.91% ± 8.8. interventricular septum was 1.04 + 30 cm and left ventricular posteria was 3-9 + 1.2 cm and RV diameter was 2.05 ± 0.56 cm.

Aortic valve gradient recorded on cir doppler peak was 56.96 ±24.67 mmHg and mean was 34.38 ± 15.52 mmHg. Various parameters were taken into consideration in this study, and were correlated with each other to assess the in the course of the disease, inter play of various factors which contribute towards gauging the severity of aortic stenosis (AS).

Relationship between peak aortic valve gradient and (Left ventricular will thickness, function diamensions and left atrium. The peak AVG when correlated with factors such as LV (ED), LV(ES) LAD and aortic diameter did not show. There was a greater significance in relationship between AVG (P) and IVS and LVPW (p=0.0001). FS reduces as AVG (P) increases (p<.08).

Relationship between mean aortic valve gradient and fractional shortening. The mean AVG versus FS, showed a weakly significant relationship (p=0.080). However, the significance was higher with IVS and LVPW (p=0.0001). there was no correlation ship between mean aortic valve gradient and left ventricular dimension and left atrium.

Relationship between Left ventricular function and left ventricular wall thickness, dimensions and aortic valve gradient. The relationship between FS and LV (ED), IVS, LV(ES) LVPW, did show interplay but did not prove to be significant statistically (p=0.820). FS was 30% as LVED ws 5.5, reduced to 25% as LVED increased to 5.8 and decreased to 20% as LVED increased to 6.0. FS reduces as AVG peak and mean increases (p=0.08).

Relationship between RV Diameter vs. The relationship between RV diameter and LV (ED), LV(ES) LAD, LVPW IVS, and FS proved to be very statistically significant, as expected (p=0.0001). this taken as evidence of pulmonary artery hypertension, implies that increase in LVED, LVES, LAD, LVPW an IVS and decrease in FS mark setting in of pulmonary hypertension. RV diameter increases beyond it upper limit as left atrium enlarges more than 4.0 cm RV starts enlarging as LV and end diastolic increases more than 5.8 cm. As LV wall thickness increases more than normal RV size starts increasing.

Critical AS by international standards is associated with aortic valve area (AVA) of less than 0.7cm2 and pressure gradient of above 50mmHg. In our study, we reached somewhat similar results for documenting severity of AS. That being, peak aortic valve gradient AVG (peak) of 74mmHg and mean aortic valve gradient AVG (mean) of 50mmHg.

DISCUSSION

Reduction in aortic valve area and increasing pressure gradient across the valve produces severe obstruction to flow and a progressive pressure overload on the left ventricle. The concentric hypertrophy that develops goes on to produce both systolic and diastolic LV dysfunction, producing symptoms of CHF. In our study as peak gradient across the valve increased, there was marked increase in interventricular thickness and posterior wall thickness leading to hypertrophy. The hypertrophy involved the left ventricle and eventually, the right ventricle. Along with that, there was also progressive decrease in fractional shortening leading to failure.[2-5]

In the study by Pellika et al, aortic-jet velocity and ejection fraction were independent predictors of the risk of subsequent cardiac events.[6] In the study by Otto et al., the only predictors of outcome were aortic-jet velocity, rate of change of this velocity and the functional status. Neither of these studies allowed any conclusions to be drawn about how to select high –risk patients who might benefit from early intervention.[7] In a study, the extent of valvular calcification and the rate of progression of aortic-jet velocity were found to be strong independent predictors of outcome.[4] Comparing the results of our study to the ones described, we see that using transvalvular pressure gradient as one of the critical parameters to adjudge severity, gives much reliable results.

Peak Doppler gradients tend to over estimate the severity of AS in patients with mild to moderate AS when compared to the cath peak to peak data. As the severity of AS increases the peak Doppler gradient becomes more reliable. Peak Doppler gradient represents the instantaneous maximal difference between left ventricular and aortic pressure. Peak to peak cath gradient is an approximation that should not authorize us to say that Doppler peak instantaneous gradient "overestimate" peak to peak cath gradients. If we could superimpose cath LV and Ao pressure curves, and measure the instantaneous higher separation between both pressures curves we could obtain a "cath peak instantaneous gradient" and then be authorized to compare it with peak Doppler gradients.[7] Studies were carried out to strive for a correlation of plus/minus 0.2cm in AVA, and plus/minus 15mm in either peak compared to peak to peak, or mean compared to mean. It was found that in patients with severe AS, peak Doppler gradient tends to correlate better.[1,2]

It is assumed that the orifice is round and symmetrical. This is not really the case, and there are sometimes multiple jets flowing across an orifice with a complex shape. The highest velocity recorded does not always necessarily represent the average flow across the valve, and this is a limitation of non-invasive valve area assessments. In one study it was concluded, that calculated valve area might be severely reduced because cardiomyopathy inhibits LV from completely opening a mildly but not severely stenotic valve. The presence of low output may lead physicians to the false conclusion that the valve is severely stenotic (aortic pseudostenosis).[9,10]

American Heart Association and American College of Cardiology have issued guidelines, recommending Doppler echocardiography for diagnosis and assessment of severity of AS.[11]

Echocardiography with Doppler examination of the aortic valve now provides a more accurate assessment of the transvalvular gradient and the area of the aortic valve. It can visualize the valvular anatomy and make clear the severity of obstruction. It is also useful in assessing the extent of LVH and LV ejection performance. Typically, a gradient of more than 50mmHg or a valve area of less than 0.7cm2 indicates critical stenosis capable of causing symptoms and death. Many asymptomatic patients with hemodynamically severe AS are now identified by echocardiography, and early intervention can be considered in such cases. Especially so in our set up, where we also face the problem of patients not reporting symptoms promptly. Keeping such cases in mind, it is worthwhile to consider the optimal timing for intervention, at the optimal stage of severity of AS. It is critical for physicians to consider diagnosis of AS in evaluating adults with symptoms due to outflow obstruction.[13,14]

Many physicians are reluctant to refer patients with severe aortic stenosis for valve replacement as long as they remain asymptomatic.[15] Once symptoms of heart failure develop, patient's life span is drastically

shortened. Kelly and colleagues followed the clinical course of 51 asymptomatic patients with severe aortic stenosis for a mean of 17 months. Severe aortic stenosis was defined as a doppler-derived peak systolic pressure gradient of at least 50 mm Hg. During the study, 21 patients (41%) began experiencing symptoms. Eight patients in the asymptomatic population died, but only two of them died of cardiac causes (heart failure in one and sudden cardiac death in the other). Both patients had had symptoms for at least 3 months before death. This and the previous study group concluded that the clinical course of patients with severe aortic stenosis could be followed safely until symptoms develop. However, Otto et al., found that patients with asymptomatic AS whose peak gradient was 64mmHg had a risk of becoming symptomatic, and 70% required intervention within 2 yrs. The risk of myocardial damage and failure that might ensue in patients with severe AS and impaired LVF may preclude an optimal postoperative outcome—something, that should not be compromised upon. Thus, assessing severity of stenosis at the optimal time and the optimal stage is of utmost importance.[14,17,19]

CONCLUSIONS

Onset of left ventricular hypertrophy correlates with peak AVG of 74 mm and mean AVG of 50 mmHg. Other markers of severe AS are enlargements of left atrium and right ventricle. Fractional shortening deteriorates with increase in LV and diastolic and systolic dimension.

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