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## Original Research

# A Study of Echocardiographic Indices in subjects with Degenerative Aortic Valve Disease

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**Abstract**

**Background:** While the elderly constituted only 24 million in 1961, the 2001 census has shown that the elderly population of India accounted for 77 million. As there is no Indian study available on DAVD in Indian population, the purpose of this is to study the various echocardiographic indices in DAVD in Indian study subjects and its Association with various cardiovascular risk factors.

**Materials & methods:** A total of 78 cases of Degenerative aortic valve disease having ejection systolic murmur (ESM) at aortic area from OPD and IPD were studied. A detailed clinical history was taken of all the patients who were included in the study with special attention to risk factor identification, Patients were specifically asked about history regarding dyspnoea, angina and syncopal attacks. Clinical information concerning age, gender, systemic hypertension, coronary artery disease, cerebrovascular episode, cigarette smoking, and diabetes mellitus were noted for each patient. Two-dimensional assessment of the aortic valve was performed on the basis of the parasternal long-axis and short-axis views. Degenerative aortic valve disease was characterized by an abnormal irregular thickening or a focal or diffuse increase of the echogenicity of the leaflets with or without reduced systolic opening. All Doppler echocardiographic recordings were registered with 100 mm/s and performed in expiration.

**Results:** Total number of 78 subjects was studied out of which 56 were male and 22 were female. The mean age of the total study subjects was 71.09 yrs. Group A includes 38 (48.7%) subject and group B includes 40(51.3%) subjects. Subjects of Group A and B was further divided into two groups on the basis of presence or absence of regional wall motion abnormality (RWMA). Group A had 13(34.21%) subjects with RWMA and group B had 18(45%) subjects with RWMA. The mean value of IVS and PW in aortic stenosis group was 13.5 ±1.60 and 12.5 ± 1.53 and aortic sclerosis group was11.39±1.22 and 10.47±1.08 respectively. The difference between the group for IVS and PW was statistically significant (P<0.0001). The Mean gradient in group A was 6.46±2.23 mm/Hg and group B was 23.07 ±14.81 mm/Hg and the difference difference between two groups were statistically significant (p <0.001). The aortic valve area in group A was 3.45 ± 0.27cm$^2$ and Group B was 1.54 ±0.5 cm$^2$ and the difference difference between two groups were statistically significant (p <0.001). Statistically significant effect was demonstrated for Age, BMI, hypertension and Smoking.

**Conclusion:** Thus we may conclude that measurement of Aortic valve area by continuity equation should be a part of routine evaluation of the aortic valve before deciding the management strategies in a given case.

**Key words:** Echocardiographic, Indices, Degenerative aortic valve disease



## Introduction

Despite the dramatic decline of rheumatic heart disease in developed countries over the past 5 decades, there has not been a concordant decline in the prevalence of valvular heart disease. Degenerative aortic valve disease (DAVD) has become the most common cause of valvular heart disease in the Western world, causing significant morbidity and mortality. The first descriptions of acquired calcific AS available in literature was by Stokes in 1845. Later in 1904 Mönckeberg in 1904 described the histopathological features of DAVD in his publication "Der

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normalehistologische Bau und die Sklerose der Aortenklappen."[1]

Degenerative Aortic stenosis (DAS) is now the most common indication for valve replacement in Europe and North America, with an ever-increasing disease prevalence due to the ageing population. With increase in the average life span, DAVD incidence has increased in Indian population and is no longer considered as a benign consequence of aging. However exact incidence of degenerative Aortic Valve Disease is not known in Indian population.[2]

Degenerative aortic valve disease is a slowly progressive disorder with a disease continuum that ranges from mild valve thickening without obstruction of blood flow, termed aortic sclerosis, to severe calcification with impaired leaflet motion, aortic stenosis. Aortic sclerosis is defined echocardiographically by focal areas of valve thickening, typically located in the leaflet centre with commissural sparing and normal leaflet mobility, valvular hemodynamics are within normal limits, with an Antegrade velocity across the valve less 2.5 m/s. Although a systolic outflow murmur may be auscultated on physical examination in some cases, there are no clinical symptoms reliably associated with aortic sclerosis.[3]

In the past, this process was thought to be "degenerative" because of time-dependent wear-and-tear of the leaflets with passive calcium deposition. Until few years ago, it was considered a physiologic process related to aging without clinical relevance. However, aortic valve sclerosis is not observed in about 50% of people over 80 years old. Now, there is compelling histopathologic and clinical data suggesting that calcific valve disease is an active disease process akin to atherosclerosis with lipoprotein deposition, chronic inflammation, and active leaflet calcification. DAVD is Characterized by progressive dystrophic calcification of the valve cusps , the early stages of DAVD are similar to the active inflammatory process of atherosclerosis including basement membrane disruption, inflammatory cell infiltration, lipid deposition and calcification .Overlap in the clinical factors associated with calcific valve disease and atherosclerosis and the correlation between the severity of coronary artery and aortic valve calcification provide further support for a shared disease process.[4, 5]

The clinical risk factors associated with the genesis and progression of atherosclerosis, including age, gender, diabetes, low-density lipoprotein (LDL) cholesterol, hypertension, and smoking have been implicated in the development of DAVD. These realizations, in addition to pathological insights gained from emerging imaging modalities, have led to the exploration of a variety of therapeutic interventions to delay or prevent the progression of DAVD.[6] The other causes of aortic stenosis includes rheumatic, congenital, Bicuspid aortic valve disease, this thesis will not address this form of AS.[6]

Patients 65 years of age with aortic stenosis involving a 3-cuspid aortic valve (unassociated with mitral valve disease) usually have extensive atherosclerosis involving the major epicardial coronary arteries and usually other arterial systems. Aortic sclerosis is common in the elderly and is associated with an increase of approximately 50 percent in the risk of death from cardiovascular causes and the risk of myocardial infarction, even in the absence of hemodynamically significant obstruction of left ventricular outflow.[7, 8]

Degenerative aortic stenosis remains asymptomatic for many years and the risk of death is then 1% per year. But once spontaneous symptoms develop, mortality rises sharply. Various studies shown that aortic sclerosis progresses to aortic stenosis over the period of time and early risk factor modification may halt or slow the disease process. An effective medical strategy to slow or stop the progression of AS will have major public health benefits, in view of higher prevalence of AS with age and increasing life expectancy of the general population.[7- 10] While the elderly constituted only 24 million in 1961, the 2001 census has shown that the elderly population of India accounted for 77 million. As there is no Indian study available on DAVD in Indian population, the purpose of this is to study the various echocardiographic indices in DAVD in Indian study subjects and its Association with various cardiovascular risk factors.

**Materials & methods**

This hospital based analytic observational study was performed in the parent institute from Jan 2010 to November 2011. A total of 78 cases of Degenerative aortic valve disease having ejection systolic murmur (ESM) at aortic area from OPD and IPD were studied. All the subjects were interviewed, examined and investigated as per the predesigned proforma.A detailed clinical history was taken of all the patients who were included in the study with special attention to risk factor identification, Patients were specifically asked about history regarding dyspnoea, angina and syncopal attacks.Clinical information concerning age, gender, systemic hypertension, coronary artery disease, cerebrovascular episode, cigarette smoking, and diabetes mellitus were noted for each patient.History systemic hypertension was defined as known case of hypertension and the patient was under antihypertensive medication. Resting blood pressure was measured at the right arm after subjects had been in a sitting position for a minimum of 5 min. Blood pressure was controlled prior to echocardiographic examination.A thorough general and systemic examination was done in each

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study subjects.Complete blood count, fasting lipid profile, blood urea and serum creatinine, fasting and post meal blood sugar ,12 lead ECG was done in all study subjects.For M mode and 2 D echo, patient was placed in supine or left lateral position at an angle of 30 degree. The transducer was placed between second and fifth left intercostal space to left sternal border. Keeping transducer in this position, the beam was swept in an arc of the cardiac apex and base of heart ,by tilting the transducer thereby focussing successively on position of right ventricle, the left ventricular cavity at the level of chordae or mitral leaflet anterior mitral leaflet, root of aorta and aortic valve leaflets. On M-mode chest wall is seen as horizontal lines, cardiac wall and valves as wavy signals and blood filled cavities as relatively echo free areas. All M-mode tracings were obtained at 50 mm/s. Measurements of left ventricular end-diastolic diameter (LVEDD) and left ventricular end systolic diameter (LVESD) and interventricular septal wall thickness (IVS) and posterior wall thickness (PW) as well as left atrial (LA) diameter were performed according to the guidelines of the American Society of Echocardiography.64 Measurements were taken on M mode as follows.LVEDD -Left ventricular internal diameter at the end of diastole in mm, LVESD-Left ventricular internal diameter at the end of systole in mm, AO - Aortic root dimension at end diastole (mm), IVS- Septal wall thickness (mm), PW-Posterior wall thickness (mm), EF- Ejection Fraction in %, Left ventricular mass (LVM) was calculated according to the Troy formula, LV mass (gram) = 1.05 ([LVIDD + PWT + SWT] 3- [LVIDD] 3) LV mass was indexed to BSA to calculate LV mass index (LVMI $gm/m^2$). Two-dimensional assessment of the aortic valve was performed on the basis of the parasternal long-axis and short-axis views. The LVOT and the aortic root were visualized in the parasternal long-axis view, using the intercostal space from which the clearest image of these structures was obtained. The diameter of left ventricular outflow tract (LVOT) measured from the inner edge to inner edge of the septal endocardium, and the anterior mitral leaflet in mid-systole using the zoom mode with careful angulation of the transducer and with gain and processing adjusted to optimize the images. LVOT diameter was used to calculate cross sectional area of LVOT.Aortic valves were scanned from the Parasternal Long-axis, Parasternal short-axis and the apical five-chamber view for total number of cusps in systole, cusp mobility, valve calcification using zoom mode. Degenerative aortic valve disease was characterized by an abnormal irregular thickening or a focal or diffuse increase of the echogenicity of the leaflets with or without reduced systolic opening.All Doppler echocardiographic recordings were registered with 100 mm/s and performed in expiration.Statistical analysis was performed with the help of SPSS 13.0 software on a statistician's personal computer.

**Results**

Total number of 78 subjects was studied out of which 56 were male and 22 were female. The mean age of the total study subjects was 71.09 yrs. The most common symptom was chest pain present in 32 (41.02%), dyspnea was present in 30(38.4%) and Syncopal attacks were present in 15(19.23%) patients. History of Pedal oedema was present in 7(8.9%) and palpitation was present in 3(3.8%) of subjects. Most common risk factor was hypertension present in 49(62.8%) of subjects. 45 (57.6%) patients gave history of smoking. Coronary artery disease (CAD) was present in 43(55.1%) of patients. History of diabetes (DM) was present in 15(19.2%) and chronic kidney disease (CKD) was present in 6(7.6%) of subjects. Group A includes 38 (48.7%) subject and group B includes 40(51.3%) subjects. Subjects of Group A and B was further divided into two groups on the basis of presence or absence of regional wall motion abnormality (RWMA). Group A had 13(34.21%) subjects with RWMA and group B had 18(45%) subjects with RWMA. Hypertension was the most common risk factor associated with both groups with 50% of subject of group A and 75% of group B subjects were having hypertension and the difference between the two group were statistically significant (p=0.022). Coronary artery disease was present in 70% of group B and 39% of group A subjects and the difference between the two group were statistically significant (p= 0.005). Smoking was associated with 70% of group B subjects and 38% of group A subjects and the difference between the two group were statistically significant (p=0.024). There was no significant difference between known diabetics and chronic kidney disease subjects in two groups. Both fasting and post prandial blood sugar were higher in Group B than Group A and difference in both groups was statistically significant (p<0.0001). Mean value of Serum triglyceride was 141.8 ±39.85mg/dl in group A and 181.9 ±34.43 mg/dl in group B and the difference between two groups were statistically significant (p=0.001). The mean value of Total cholesterol in group A was 211 ±23.32mg/dl and Group B was 243.4 ± 25.21 mg/dl and difference between two groups were statistically significant (p <0.001). The mean value of LDL cholesterol in Group A was 142.9 ±21.51 mg/dl and group B was 175.5± 19.10 mg/dl and difference between two groups were statistically significant (p <0.001). The mean value of HDL in group A was 45.79± 6.57 and group B was 40.38 ± 5.77 mg/dl and difference between two groups were statistically significant (p <0.001).The mean value of Serum Creatinine in group A was 1.06±0.43 mg/dl and group B was 1.36 ±0.63 mg/dl and difference between two

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groups were statistically significant (p =0.001). There was no significant difference in blood urea level in both the groups (p=0.09). The LA dimension was 36.4 ±5.72 mm in aortic stenosis group and 33.21± 3.67mm in aortic sclerosis group and the difference between the group was statistically significant(p=0.04). LVEDD and LVESD were also increased in aortic stenosis group and difference between the group was statistically significant (p<0.05). The mean value of IVS and PW in aortic stenosis group was 13.5 ±1.60 and 12.5 ± 1.53 and aortic sclerosis group was11.39±1.22 and 10.47±1.08 respectively. The difference between the group for IVS and PW was statistically significant (P<0.0001). The EF was lower in aortic stenosis group (59.8 ±10.23) than aortic sclerosis group (64.34 ±8.54) and the difference between the group was statistically significant (p=0.037). The LV mass index was more in aortic stenosis(142.0 ±37.63g/$m^2$) group than in aortic sclerosis group (102.8 ±30.33g/$m^2$) the difference between the group was statistically significant (p=0.014). The maximum aortic velocity in aortic sclerosis was 1.32 ±0.3 m/sec and aortic stenosis was 2.96 ± 0.9m/sec and the difference difference between two groups were statistically significant (p <0.001). The Peak Gradient in group A was 11.48 ± 3.9mm/Hg and Group B was 38.49± 21.38 mm/Hg and the difference difference between two groups were statistically significant (p <0.001). The Mean gradient in group A was 6.46±2.23 mm/Hg and group B was 23.07 ±14.81 mm/Hg and the difference difference between two groups were statistically significant (p <0.001). The aortic valve area in group A was 3.45 ± 0.27$cm^2$ and Group B was 1.54 ±0.5 $cm^2$ and the difference difference between two groups were statistically significant (p <0.001). Statistically significant effect was demonstrated for Age (p=0.001, OR=1.42, 95% CI =1.14-1.76), BMI (P<0.001, OR 2.38, 95% CI =1.49-3.82), hypertension (p=0.004, OR=10.21, 95% CI =2.14-48.65) and Smoking (p=0.038. OR =5.10, 95% CI =1.09-23.83),

**Table No 1: Echocardiographic parameters in study subjects**

| Sr. No. | Parameter | | Mean value (SD) |
|---|---|---|---|
| 1 | AO(mm) | | 28.78 ± 3.20 |
| 2 | LA(mm) | | 34.85 ± 5.06 |
| 3 | RV(mm) | | 19.18 ± 2.56 |
| 4 | LVEDD(mm) | | 44.46 ± 7.68 |
| 5 | LVESD(mm) | | 28.56 ± 7.53 |
| 6 | IVS(mm) | | 12.47 ± 1.77 |
| 7 | PW(mm) | | 11.51 ± 1.67 |
| 8 | LVEF (%) | | 62.01 ± 9.66 |
| 9 | LVMI(g/m2) | | 122.9 ± 39.35 |
| 10 | Aortic Valve Indices | Vmax(m/sec) | 2.16 ± 1.007 |
| | | Peak Gradient (mm/Hg) | 25.34 ± 19.99 |
| | | Mean Gradient (mm/Hg) | 14.97 ± 12.66 |
| | | AVA($cm^2$) | 2.48 ± 1.041 |

**Table No 2: Subjects were divided into two groups group A includes subjects with sclerotic aortic valve without aortic stenosis (Aortic Valve Area ≥ 3cm2) and group B includes subject with aortic stenosis with Aortic Valve Area less than 3 $cm^2$.**

| GROUP | Group A Aortic Sclerosis AVA ≥3 $cm^2$ n=38 | Group B Aortic Stenosis AVA <3 $cm^2$ n=40 |
|---|---|---|
| Subjects With RWMA | 13(34.21%) | 18(45%) |
| Subjects Without RWMA | 25(65.79%) | 22(55%) |
| TOTAL SUBJECTS | 38(48.7%) | 40(51.3%) |

**Table no 3: ECG changes in study subject**

| Sr. No. | ECG changes | Group A Aortic sclerosis (Mean) n = 38 | Group B Aortic stenosis (Mean) n = 40 |
|---|---|---|---|
| 1 | Old ant. wall infraction | 1 | 4 |
| 2 | Old inf. Wall infraction | 0 | 1 |

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| | | | |
|---|---|---|---|
| 3 | Ant. wall ischemia | 8 | 3 |
| 4 | Inf. wall ischemia | 3 | 4 |
| 5 | Lateral wall ischemia | 1 | 6 |
| 6 | LVH | 5 | 12 |
| 7 | LBBB | 1 | 1 |
| 8 | RBBB | 0 | 3 |
| 9 | CHB | 0 | 1 |
| 10 | AF | 0 | 1 |

**Table No 4: Comparison of Echocardiographic Parameters in Group A and B**

| Sr. No. | Parameter | Group A Aortic sclerosis n = 38 (Mean) | Group B Aortic stenosis n = 40 (Mean) | P Value |
|---|---|---|---|---|
| 1 | AO(mm) | 28.36 ± 2.34 | 29.17 ± 3.83 | 0.268 |
| 2 | LA(mm) | 33.21 ± 3.67 | 36.4 ± 5.72 | 0.004 |
| 3 | RV(mm) | 18.60 ± 2.72 | 19.73 ± 2.29 | 0.053 |
| 4 | LVEDD(mm) | 42.52 ± 7.69 | 46.3 ± 7.30 | 0.029 |
| 5 | LVESD(mm) | 26.44 ± 7.39 | 30.57 ± 7.17 | 0.041 |
| 6 | IVS(mm) | 11.39 ± 1.22 | 13.5 ± 1.60 | <0.001 |
| 7 | PW(mm) | 10.47 ± 1.08 | 12.5 ± 1.53 | <0.001 |
| 8 | LVEF (%) | 64.34 ± 8.54 | 59.8 ± 10.23 | 0.0371 |
| 9 | LV MI($g/m^2$) | 102.8 ± 30.33 | 142.0 ± 37.63 | 0.014 |

**Table No 5: Comparison of AV Indices in Study Groups A and B**

| Sr. No. | Parameter | Group A Aortic sclerosis (Mean) | Group B Aortic stenosis (Mean) | P value |
|---|---|---|---|---|
| 1 | Vmax (m/sec) | 1.32 ± 0.30 | 2.96 ± 0.90 | <0.001 |
| 2 | Peak Gradient (mm/Hg) | 11.48 ± 3.90 | 38.49 ± 21.38 | <0.001 |
| 3 | Mean Gradient (mm/Hg) | 6.46 ± 2.23 | 23.07 ± 14.81 | <0.001 |
| 4 | AVA ($cm^2$) | 3.45 ± 0.27 | 1.54 ± 0.50 | <0.001 |

**Table No 6: Multiple Logistic Regression Analysis Showing Independent Risk Factors for Progressive Aortic Valve Disease**

| Sr. No. | Clinical Parameters | Adjusted Odds Ratio | 95% C. I. | 'p' value |
|---|---|---|---|---|
| 1 | AGE | 1.42 | 1.14-1.76 | 0.001 |
| 2 | BMI | 2.38 | 1.49-3.82 | < 0.001 |
| 3 | HYPERTENSION | 10.21 | 2.14-48.65 | 0.004 |
| 4 | SMOKING | 5.10 | 1.09-23.83 | 0.038 |

**Discussion**

This study was conducted from January 2010 to November 2011 in the parent institute.Study was conducted with 78 subjects of DAVD who were either attending medicine OPD or admitted in medical wards. The results were analyzed and compared with the available literature.

In our study history of diabetes mellitus was present in 19% of all study subjects and 16% in aortic sclerosis group and 28% of aortic stenosis groups but the difference between the groups was not statistically significant (p=0.4523).7.6% subjects were k/c/o CKD in our study, however there was no statistically significant difference between the two groups.

Emi I .K et al.[11] in 1991 studied 120 patients undergoing aortic valve replacement which consist of 30 patients of DAVD, 13 (43%) had systemic hypertension, 6 (20%)) had diabetes mellitus, 15 (50%) had coronary artery disease. Fourteen patients (47%) had history of smoking. Thirteen patients (43%) had total serum cholesterol level greater than 200 mg/dl and 5 (17) patients had a triglyceride level greater than 220 mg/dl.

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Chambers John et al.[12] in 2004 studied 91 patients with aortic stenosis. History of hypertension was present in 39% patients and CAD was in 46% patients of AS.

In our study we studied Echocardiographic indices in 78 DAVD. We divided subject into two groups on the basis of aortic valve area calculated by continuity equation.

Group A consist of subjects with aortic sclerosis with AV A more than or equal to 3cm$^2$(AVA ≥ 3 cm$^2$) and Group B consist patient with aortic stenosis with AVA less than 3 cm2. (AVA < 3 cm$^2$).

The LA dimension (mm) was 36.4 ±5.72 in aortic stenosis group and 33.21± 3.67 in aortic sclerosis group and the difference between the groups was statistically significant.

LVEDD (mm) was 42.52 ±7.69 in aortic sclerosis and 46.3±7.30 in aortic stenosis group. (p=0.0292). LVESD (mm) was 26.44±7.39 in aortic sclerosis and 30.57±7.17 in aortic stenosis and difference between the group was statistically significant (p<0.05).

The mean value of IVS and PW thickness in aortic stenosis group was 13.5 ±1.60 mm and 12.5 ± 1.53 mm and aortic sclerosis group was 11.39±1.22 mm and 10.47±1.08 mm respectively. The difference between the group for IVS and PW thickness was statistically significant (P<0.0001).

The EF was 59.8±10.23 % in aortic stenosis group and 64.34±8.54 % in aortic sclerosis group and the difference between the group was statistically significant (p=0.037).

The LVMI in aortic stenosis group was 142 ±37.63 gm/m2 and aortic sclerosis group was 102.8± 30.33 gm/m2 and the difference between the group was statistically significant (p<0.001).

The maximum aortic velocity (Vmax) in aortic sclerosis was 1.32 ± 0.30 m/sec and aortic stenosis was 2.96 ±0.90 m/sec and the difference difference between two groups was statistically significant (p <0.001).

The Peak Gradient in aortic sclerosis was 11.48 ± 3.90 mmHg and aortic stenosis was 38.49 ±21.38 mmHg and the difference difference between two groups was statistically significant (p <0.001).

The Mean Gradient in aortic sclerosis was 6.46 ±2.23 mm/Hg and aortic stenosis was 23.07±14.81 mm/Hg and the difference difference between two groups was statistically significant (p <0.0001).

The Aortic Valve area in aortic sclerosis was 3.45 ±0.27 cm2 and aortic stenosis was 1.54±0.5cm2 and the difference difference between two groups were statistically significant (p <0.0001).

Emi I .K et al[11] in their study, after applying multiple logistic regression analysis for various risk factors associated with the degenerative valve group, they observed that best overall fit was achieved with a four-variable model which included gender, hypertension, smoking, and triglyceride level. A statistically significant effect was demonstrated by gender (p=0.01) and smoking (p=0.03), followed by a borderline effect for systemic hypertension (0.11) and triglyceride level (p=0.15). Fendley Stewart B. et al[5] (1997), in their study they observed that independent clinical factors associated with degenerative aortic valve disease included age (twofold increased risk for each 10-year increase in age), male gender (twofold excess risk), present smoking (35%) increases in risk) and a history of hypertension (20%increase in risk).

In our study after applying multiple logistic regression analysis after adjusting for other risk factors statistically significant effect were demonstrated for Age, hypertension and Smoking, for progressive aortic valve disease.

Hence as we can say that degenerative aortic valve disease is progressive disorder with a disease continuum that ranges from mild valve thickening without obstruction of blood flow, termed aortic sclerosis, to severe calcification with impaired leaflet motion, or aortic stenosis. The mean age of subjects with increasing stenosis was found to statistically more as compared to those with just aortic sclerosis. The factors found to be associated with progressive aortic stenosis in addition to Age were Smoking, increasing Body Mass Index and presence of hypertensions.

Subjects having RWMA had lower ejection fractions as compared to those having no RWMA. These subjects with RWMA also had lower peak and mean gradients across the aortic valve against those having no RWMA though their aortic valve areas were comparable.

Similarly subjects having increased LV Mass and diastolic dysfunction had lower peak and mean gradients across the aortic valve due to restricted filling as compared to those having lower LV Mass and no diastolic dysfunction though their aortic valve areas were comparable.

John Chambers et al[12] in 2004 studied 91 patients with aortic stenosis, In their study mild AS was present in 26%, moderate AS in 30% and severe AS in 44% patients, LVEDD was 47±0.7 mm and LVESD was 30±0.6, IVS was 16±0.3 mm and LV mass was 277±98 gm and LV mass index (g/m2) was 152±48. The mean gradient was 40± 20 mmHg and peak gradient was 63± 30 mmHg .The mean aortic valve area was 0.9±0.4.cm$^2$.Luis M. Moura et al[13] in 2007 performed an open-label, prospective study evaluating 121 consecutive patients with asymptomatic moderate to severe aortic stenosis. The LVEDD was 51.77±5.1mm.and LVESD was 33.9 ± 4.4 mm. The mean Ejection fraction was 54.9 ± 3.1 %. The maximum aortic velocity was 3.63±0.63 m/sec. The peak and mean gradient across the aortic valve were 54.3±18.5 mmHg and 35.7 ±13.3 mmHg. The mean aortic valve area calculated by continuity equation was 1.21±0.38 cm$^2$.

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Subjects having Aortic regurgitation in addition to aortic stenosis had increased peak and mean gradients across the aortic valve due to increased flows as compared to those having no aortic regurgitation despite both these groups having comparable aortic valve areas. Thus the severity of aortic stenosis cannot be judged only by transvalvular velocity or gradient, in subjects having (I) RWMA & low ejection fractions, (ii) left ventricular hypertrophy leading to impaired filling of left ventricle and diastolic dysfunction, and (iii) subjects having associated aortic regurgitation leading to increased flows and increased peak and mean gradients. Hence the valve area—measured by the continuity equation is a must to evaluate the severity of aortic valve obstruction and deciding the future management strategies.

## Conclusion

Thus we may conclude that measurement of Aortic valve area by continuity equation should be a part of routine evaluation of the aortic valve before deciding the management strategies in a given case. This is taking into consideration the future burden of elderly subjects on the nation and increased incidence of degenerative aortic valve disease in the times to come whom would also have associated problems of hypertension, dyslipidaemias, CAD & CKD. Early risk factor identification and modification shall also form a part of effective medical strategy to halt or slow the progression of Aortic stenosis.

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