

E-ISSN: 2616-3470 P-ISSN: 2616-3462

© Surgery Science

www.surgeryscience.com

2020; 4(4): 265-267 Received: 04-09-2020 Accepted: 09-10-2020

Dr. Anupama Rao

Assistant Professor, Department of Cardiac Surgery, Hi-Tech Medical College, Pandara, Bhubaneshwar, Odisha, India

Dr. Anurag Vidhale

Consultant and HOD, Department of CTVS, Avanti Institute of Cardiology, Dhantoli, Nagpur, Maharashtra, India

Dr. Padma S

Assistant Professor, Department of Anaesthesia, Sapthagiri Institute of Medical sciences, Bangalore, Karnataka, India

Dr. PK Dash

Director of Cardiac Sciences, Capital Hospital, Bhubaneshwar, Odisha, India

Corresponding Author: Dr. Anupama Rao

Assistant Professor, Department of Cardiac Surgery, Hi-Tech Medical College, Pandara, Bhubaneshwar, Odisha, India

Clinical profile of undergoing aortic valve replacement for aortic stenosis

Dr. Anupama Rao, Dr. Anurag Vidhale, Dr. Padma S and Dr. PK Dash

DOI: https://doi.org/10.33545/surgery.2020.v4.i4e.567

Abstrac

The prevalence of calcific aortic stenosis increases with age, being present in 2% to 4% of adults over the age of 65 years. Population-based studies such as the Helsinki Aging Study showed a prevalence of aortic sclerosis to be 21% in adults aged 55–71 years and according to Cardiovascular Health Study (CHS) the incidence of aortic sclerosis was 26% in adults over the age 65 years or older. 75 consecutive patients who underwent aortic valve replacement with TTK-Chitra valve for aortic stenosis at the Department Of Cardio-Thoracic and Vascular Surgery, were studied prospectively. Patients were admitted after routine cardiology OPD screening and investigations as per institutional pre-admission protocol which included transthoracic echocardiography, ECG, chest roentgenogram and haematological investigations. There was no significant difference in EF of patients pre and post operatively. Significant change was observed at one year follow up. The mean EF pre-operatively was $61.00\pm7.26\%$, followed by $61.04\pm7.76\%$, $62.00\pm5.56\%$ and $62.96\pm5.14\%$ at immediate post-op, 3 months and 1 year respectively.

The functional class of patients showed significant improvement at one year post surgery.

Keywords: Clinical profile, aortic valve replacement, aortic stenosis

Introduction

Among several anatomic types of aortic valvular stenosis, calcific aortic stenosis is the commonest variety and poses greater surgical technical problems than due to aortic stenosis of congenital or rheumatic origin. Calcific 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, to aortic stenosis. In the past, this process was thought to be —degenerativel because of time-dependent wear-and-tear of the leaflets with passive calcium deposition. Now, there is compelling histopathology 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 [1].

The prevalence of calcific aortic stenosis increases with age, being present in 2% to 4% of adults over the age of 65 years. Population-based studies such as the Helsinki Aging Study showed a prevalence of aortic sclerosis to be 21% in adults aged 55–71 years and according to Cardiovascular Health Study (CHS) the incidence of aortic sclerosis was 26% in adults over the age 65 years or older [2].

Among the proposed theories for the initiating factor that leads to aortic stenosis, there is most support for a mechanical stress hypothesis. The complex three dimensional anatomy of tricuspid aortic valve allows stress sharing between the leaflets and sinus of valsalva which is altered in disease states.

End stage degenerative aortic stenosis is characterised by accumulation of irregular fibrocalcific masses on aortic side of non-coaptal portion of the leaflets. Microscopically, described changes include increased leaflet thickness, lipid accumulation, collagen fibre disarray and calcific deposits.

Patients with aortic stenosis gradually develop myocardial fibrosis, typically located sub-endocardially at the basal segments of LV. This fibrosis affects LV systolic and diastolic function thus has a profound impact on long term clinical outcome [3].

The standard diagnostic evaluation of aortic stenosis includes assessment of leaflet anatomy and the extent of valvular calcification by echocardiography. The severity of aortic stenosis can be measured accurately and reliably on the basis of antegrade velocity, mean pressure gradient, and continuity equation valve area. Beyond this information, echocardiography provides an assessment of left ventricular hypertrophy, diastolic dysfunction, and regional and global systolic function with calculation of ejection fraction. Beyond echocardiography, a variety of newer diagnostic methods have proved feasible for evaluating the presence and severity of calcific aortic valve disease, including electron beam computed tomography and MRI [4].

Methodology

75 consecutive patients who underwent aortic valve replacement with TTK-Chitra valve for aortic stenosis at the Department Of Vascular Surgery, were studied Cardio-Thoracic and prospectively. Patients were admitted after routine cardiology OPD screening and investigations as per institutional preadmission protocol which included transthoracic echocardiography, ECG. chest roentgenogram haematological investigations. Following admission a detailed clinical examination and echocardiogram was performed and findings were recorded. Patients were classified into NYHA Class I to IV based on their symptoms of angina and dyspnoea.

NYHA I – patients with cardiac disease, with symptoms present at unaccustomed work only. NYHA II- patients with cardiac disease, symptoms present during accustomed work.

NYHA III- in patients with cardiac disease, symptoms present during less than accustomed activity.

NYHA IV- in patients with cardiac disease, symptoms present at rest.

Echocardiograms for the assessment of functional and hemodynamic valve performances were always carried out by the same echocardiographer. Dimensions were measured from the standard two-dimensional and M-mode echocardiography. Flow velocity in the left ventricular outflow tract and across the valve was measured by means of pulsed and continuous wave Doppler ultrasonography, respectively.

The following parameters were collected from each patient: left ventricular ejection fraction [%], mean and peak prosthetic valve gradients, valve effective orifice

Area (EOA), LVIDd, PWTID and IVSTD. The modified Bernoulli equation was used to calculate peak and mean pressure gradients across the prosthesis. EOA is calculated with the continuity equation, similar to native aortic valve area by the velocity time integral method. EOA= CSA (LVOT- VTI / VTIAo); where, CSA — Cross sectional area, VTIAo is the velocity time integral across the prosthetic valve.

Coronary angiography was performed in 44 cases [patients > 40 years, chronic smokers, chest pain, ECG evidence of ischemia], to rule out coronary artery disease.

Patients were taken up for aortic valve replacement after detailed informed consent including consent for lifelong anticoagulation and it's management. Aortic valve replacement was done under general anaesthesia, on cardiopulmonary bypass as per existing standard operating practice detailed later. The intra-operative and postoperative data were collected in the study Performa. Echocardiography was performed postoperatively in ICU on the day of surgery, prior to discharge, once at three months and once at one year follow up and then yearly thereafter.

Inclusion Criteria

All patients with aortic stenosis undergoing aortic valve replacement for aortic stenosis with TTK Chitra valve within the study period were included. This comprised 75 patients.

Exclusion Criteria

- All patients of severe aortic stenosis undergoing valve replacement with valves other than TTK Chitra were excluded.
- Patients undergoing additional procedures including coronary artery bypass grafting (CABG), aortic root widening, mitral or tricuspid valve procedures were excluded.
- Patients who have undergone surgery prior to or after the study period were excluded.

Results

The mean age of patients was 43.69±10.16 years, with a range of 22 to 66 years.

Table 1: Age distribution of patients studied

Age in years	No. of patients	%
<30	8	10.7
31-40	21	28.0
41-50	27	36.0
51-60	18	24.0
>60	1	1.3
Total	75	100.0

Out of 75 patients in the study group, 65 were males [86.7%] and 10 were females [13.3%].

Table 2: Gender distribution of patients studied

Gender	No. of patients	%	
Female	10	13.3	
Male	65	86.7	
Total	75	100.0	

The BSA of patients ranged from 1.21 to $2.03/m^2$. The mean BSA was $1.6\pm0.18/m^2$.

Table 3: BSA in /m² of patients studied

BSA/m ²	No. of patients	%
1-1.51	27	36.0
1.51-2	47	62.7
>2	1	1.3
Total	75	100.0

The presenting symptoms were angina, dyspnoea and syncope. Angina was the most common symptom and was present in 60 patients [80%], with syncope and dyspnoea in 30 patients [40%] and 59 patients [78.7%] respectively. Most patients had more than one symptom at presentation.

Table 4: Symptoms of patients studied

Symptoms	No. of patients (n=75)	%
Angina	60	80.0
Syncope	30	40.0
Dyspnoea	59	78.7

Sixty seven patients [89.3%] were in NYHA class II preoperatively. 7 patients [9.3%] were in NYHA class II and 1 patient [1.3%] was in NYHA class I.

Table 5: NYHA class

NYHA class	No. of patients	%
I	0	0.0
II	67	89.3
III	7	9.3
IV	1	1.3
Total	75	100.0

There was no significant difference in EF of patients pre and post operatively. Significant change was observed at one year follow up. The mean EF pre-operatively was 61.00±7.26%,

followed by $61.04\pm7.76\%$, $62.00\pm5.56\%$ and $62.96\pm5.14\%$ at immediate post-op, 3 months and 1 year respectively. With respect to individual valves, mean EF of each type of valve is given in Table. The mean EF of #19 valve was $61.33\pm3.67\%$ post operatively, $62.33\pm4.33\%$ at 3months and $61.56\pm3.78\%$ at 1 year. For # 21 valve, the EF values were $60\pm5.17\%$, $61.46\pm4.7\%$ and $62.79\pm6.21\%$ at post op, 3 months and 1 year respectively. The mean EF of #23 valve was $61.52\pm3.47\%$, $62.56\pm6.12\%$ and $63.85\pm4.12\%$ at post op, 3 months and 1 year respectively. The mean EF of #25 valve was $61.46\pm4.2\%$, $62.92\pm5.07\%$ and $63.38\pm4.79\%$ at post op, 3 months and 1 year respectively.

Table 6: Comparison of pre op EF with post op, 3 months and 1 year follow up EF.

EF%	Min-Max	Mean± SD	difference	t value	P value
Pre op	35.00-70.00	61.00±7.26	1	1	-
Post op	30.00-70.00	61.04±7.76	0.027	0.039	0.969
Follow up 3 months	40.00-70.00	62.00±5.56	0.986	1.646	0.104
Follow up 1 year	40.00-70.00	62.96±5.14	1.946	2.973	0.004**

Discussion

Patients with aortic stenosis may remain asymptomatic for several years and develop symptoms gradually over time. The classic triad of symptoms are chest pain, heart failure and syncope ^[5].

Chest pain in aortic stenosis occurs on exertion and relieved with rest similar to angina of coronary artery disease.

Heart failure symptoms include Orthopnoea, paroxysmal nocturnal dyspnoea and dyspnoea on exertion. They may occur due to LV systolic or diastolic dysfunction. LV systolic dysfunction is a consequence of afterload mismatch or ischaemia. LVDD results from LV hypertrophy or is chaemia. Syncope occurs exertion induces systemic vasodilation Wheras stroke volume remains fixed due to aortic stenosis. Therefore, systole blood pressure declines. Syncope at rest may be due to atrial or ventricular arrhythmias.

In addition, patients may present with features of infective endocarditis or embolic phenomenon.

Clinical examination

Carotid pulse may show delayed and plateued upstroke (pulses prvus et tardus). Systolic hypertension may be present in some patients. Pulses alterans is another feature in aortic stenosis. On auscultation, S1 may be normal. Soft or absent A2 component of S2. Paradoxical splitting of S2 due to delayed closure of aortic valve. Ejection systolic murmur radiating to carotids is classic.

Pathophysiology

When the aortic valve becomes stenotic, resistance to systolic ejection occurs and pressure gradient between LV and aorta starts to develop. This increases LV systolic pressure. To compensate this mechanism, LV hypertrophy occurs. Parallel replication of sarcomeres cause concentric hypertrophy. LVEDP rises, pulmonary capillary pressure rises. There is decrease in cardiac output due to diastolic dysfunction.

Possible complications of AS include sudden death, heart failure, conduction defects, calcific embolisation. Sudden death is more common in symptomatic individuals than asymptomatic [6]

The natural History of aortic stenosis indicates it's progressive nature and valve replacement becomes inevitable in order to prevent adverse hemodynamic changes. Many studies have proven the advantages of valve replacement over medical therapy ^[7,8].

Conclusion

The presenting symptoms were angina, dyspnoea and syncope. Angina was the most common symptom and was present in 60 patients, with syncope and dyspnoea in 30 patients and 59 patients respectively. Most patients had more than one symptom at presentation.

References

- 1. O'Brien KD, Reichenbach DD, Marcovina SM, Kuusisto J, Alpers CE, Otto CM. Apolipoproteins B, (a) and E accumulate in the morphologically early lesion of-degenerative valvular aortic stenosis. Arterioscler Thromb Vasc Biol. 1996;16:523–532.
- 2. Wongpraparut N, Apiyasawat S, Crespo G, Yazdani K, Jacobs LE, Kotler MN. Determinants of progression of aortic stenosis in patients aged greater than or equal to 40 years. Am J Cardiol. 2002;89:350-352.
- 3. Larry R Kaiser, Iring L Kron, Thomas L Spray. Mastery of Cardiothoracic Surgery; 2nd Edition 2007, 410.
- 4. Shiono M, Sezai Y, Sezai A *et al*. Long term results of cloth covered Starr Edwards ball valve. Ann Thorac Surg 2005; 80:204.
- 5. Tintinalli JE, Kelen GD, Stapezynsky JS, eds. Vascular Emergencies. 6th ed. *Emergency medicine*. A comprehensive study guide. New York: Mc Graw Hill 2004.54.
- 6. Rafique AM, Biner S, Ray I, Forrester JS *et al.* Mera analysis of prognostic value of stress testing in patients with asymptomatic severe aortic stenosis. Am J Cardiol. 2009;104:972-7.
- 7. Pibarot P, Dumesnil JG: Prosthetic heart valves: selection of the optimal prosthesis and long term management. Circulation 2009;119(7):1034-48.
- 8. Pai RG, Kapoor N, Bansal RC, *et al.* Malignant natural history of asymptomatic severe aortic stenosis: benefit of aortic valve replacement. Ann thorac surgery 2006;82(6):2116-22.