OUTCOMES OF AORTIC VALVE REPLACEMENT IN AORTIC STENOSIS AND AORTIC REGURGITATION WITH SEVERE LEFT VENTRICULAR DYSFUNCTION-A RETROSPECTIVE STUDY

Thesis submitted for the partial fulfillment for the requirement of the Degree of MCh (Cardiothoracic and Vascular Surgery)

BY

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DECLARATION

I hereby declare that this thesis titled, **"OUTCOMES OF AORTIC VALVE REPLACEMENT IN AORTIC STENOSIS AND AORTIC REGURGITATION WITH SEVERE LEFT VENTRICULAR DYSFUNCTION-A RETROSPECTIVE STUDY"** has been prepared by me under the capable supervision and guidance of **Dr. Vivek pillai,** Associate professor of the Department, Division of Cardiothoracic and Vascular Surgery (CTVS), Sree Chitra Tirunal Institute for Medical Sciences & Technology (SCTIMST), Thiruvananthapuram, Kerala.

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CONTENTS

	Page No
INTRODUCTION	**1**
REVIEW OF LITERATURE	**3**
AIMS AND OBJECTIVES	**9**
MATERIALS AND METHODS	**11**
STATSITICAL ANALYSIS	**15**
RESULTS	**17**
DISCUSSION	**20**
CONCLUSION	**26**
BIBLIOGRAPHY	**28**
ANNEXURES	**34**
Abbreviations	
Observation Chart	
Plagiarism Report	
Master Chart	

INTRODUCTION

INTRODUCTION

Aortic valve disease is one of the most common valve disorder leading to surgical intervention world wide. Patients with severe aortic stenosis/aortic regurgitation have a grave prognosis, with a mortality rate of 8-9% per year.[1, 2] With the onset of severe symptoms the average life expectancy is three years; that for angina is five years, and for syncope it is three years. The worst prognostic indicator is congestive heart failure, for which the average life expectancy is less than two years.[3] Aortic valve replacement is a class I indication in patients with severe AS/AR who present with symptoms or who demonstrate signs of cardiac dysfunction, defined as resting left ventricular ejection fraction (LVEF) of 50%or less according to American College of Cardiology/American Heart Association and European Society of Cardiology guidelines. [4,5] Aortic valve replacement improves hemodynamics [6] and the late survival rate is good .[7,8] In the absence of complications, ventricular hypertrophy regresses and ventricular performance improves.[9] The results of valve replacement in patients with aortic stenosis/ aortic regurgitation with severe left ventricular dysfunction have not been defined, and its effects on left ventricular function are not known. [10,11] Therefore, there is uncertainty about the role of surgery in such patients. We propose to retrospectively evaluate the effects of aortic valve replacement in patients with severe aortic stenosis/ regurgitation with underlying severe LV dysfunction with respect to left ventricular function, functional class, survival rate and mortality on long term. We hypothesize that aortic valve replacement in patients with AS/AR with underlying severe LV dysfunction would have beneficial effect in terms of functional class, left ventricular function.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Heart valve disease is one of the most important cardiovascular diseases. The prevalence varies with age, gender and different societies [12]. In the developing countries like India, the burden of valvular heart disease (VHD) among adults is enormous. Among VHD, Aortic stenosis is the most common type and its prevalence is projected to double in next two decade [13]. There are various causes of aortic valve diseases including rheumatic, degenerative, traumatic, congenital, and infectious heart diseases. Both aortic stenosis and aortic regurgitation remains common in developing countries, because of the increase in prevalence of rheumatic heart diseases [14].

Aortic valve diseases involving stenosis and regurgitation has an insidious onset and progresses slowly. Symptoms of aortic stenosis usually develop gradually after an asymptomatic latent period of 10-20 years. Thus in this patients underlying valvular dysfunction may be present for years without symptoms, but functional deterioration is often rapid once congestive heart failure, angina or syncope with effort is present[15]. The severity of aortic valvular diseases may not be easy to assess clinically. Medical therapy doesn't prevent the natural progression of the disease, but aortic valve replacement improves survival and relieves symptoms. Left un-operated patients who eventually become symptomatic, face a dismal prognosis of up to 50% mortality over 2 yrs[16]. Several agencies[17] recommend AVR in case of severe, symptomatic AS, with surgical aortic valve replacement (SAVR) being the standard approach for patients with a low to intermediate surgical risk.

Pathophysiology of aortic stenosis/aortic regurgitation

Aortic stenosis

Aortic stenosis can be considered as a continuum from aortic sclerosis to severe aortic stenosis. Progression of stenosis is associated with increasing obstruction of blood flow through the left ventricular outflow tract and occurs over many years[15].Only 10% of patients with aortic sclerosis advance to hemodynamically important aortic stenosis.15 In aortic sclerosis, mild valve thickening or calcification affects normal leaflet motion[18]. With the progression of the disease, leaflets become thicker, calcific nodules develop, and new blood vessels appear[19]. In aortic stenosis, calcium nodules located within the layers of the leaflet bulge outward toward the aorta and extend to the sinuses of Valsalva, causing restricted leaflet motion and obstruction of left ventricular outflow during systole[20]. Bicuspid aortic valve account for about half of all occurrences of aortic stenosis[21]. Bicuspid aortic valve stenosis typically occurs at fifth to sixth decade than does tricuspid valve stenosis which occurs at seventh to eighth decade. Calcific aortic valve disease (CAVD) is the most common cause of aortic stenosis which was previously considered a normal consequence of aging[20]. CAVD is an active cellular biological process characterized by alterations of the cells within the layers of the aortic valve. Rheumatic heart disease another cause of aortic stenosis rarely nowadays because of aggressive treatment of penicillin-sensitive streptococcal infections[22].

In aortic valve diseases, progresses the left ventricle encounters chronic resistance to systolic ejection. The ventricle requires to generate a higher systolic pressure than the opposing pressure produced by the rigid, calcified aortic valve. An increased resistance to systolic ejection is called afterload. To compensate for a high afterload, the left ventricular myocardial wall thickens as per the Lapalace law with LV internal

dimensions remaining normal[12]. Thickening of the left ventricular wall, known as concentric hypertrophy, strengthens left ventricular systolic contraction to maintain adequate stroke volume and cardiac output[13].

The sequelae of left ventricular hypertrophy (LVH) may be detrimental although is a compensatory mechanism. High LV afterload include decreased LV myocardial elasticity and coronary blood flow and increased myocardial workload, oxygen consumption, and mortality[25]. LVH increases diastolic pressure and delays left ventricular untwisting; thus, a forceful atrial contraction is needed for optimal filling of the left ventricle to maintain stroke volume and cardiac output. Late manifestations of left ventricular hypertrophy include a smaller left ventricular chamber size, which decreases preload and worsens systolic dysfunction. The result is insufficient stroke volume, cardiac output, and ejection fraction[18]. Finally, backward transmission of increased left ventricular pressure to the lungs may cause pulmonary venous hypertension and reactive vasoconstriction of the pulmonary vasculature[21]. As a result of the detrimental effects associated with left ventricular hypertrophy, patients with aortic stenosis become increasingly dependent on atrial kick to maintain stroke volume and cardiac output. Loss or compromise of atrial kick as a result of atrial fibrillation, ventricular pacing, and/or intravascular fluid volume overload may precipitate pulmonary congestion, hypotension, and angina[18]. Atrial arrhythmias may result from an extension of calcific infiltrates from the aortic valve into the conduction system[21].

Aortic regurgitation (AR)

AR caused by malfunction of the valve leaflets or by dilatation of the aortic root and annulus, or combination of these factors. Rheumatic disease is still the most common aetiology of AR in developing countries like India .However, in developed world, the

leading cause of AR is either congenital (particularly due to bicuspid leaflets) or degenerative disease, including annuloaortic ectasia. AR causes LV volume overload. The total stroke volume ejected by the LV (=effective stroke volume + regurgitant volume) is increased; in severe AR regurgitant volume may equal or even exceed effective stroke volume. The main compensatory mechanism is increase in LV end-diastolic volume(LVEDV) which serves to maintain a normal effective stroke volume. Left ventricular ejection fraction is initially normal, however, LV end-diastolic pressure rises. Over time LVEDV continues to increase further and ejection fraction drops; these changes may actually precede the development of clinical symptoms. Considerable eccentric myocardial hypertrophy can occur with chronic AR and at autopsy heart weights of up to 1000 g have been reported[26].

As already mentioned, in both AS/AR the time interval from the initiation of disease process to the first development of symptoms is very long over years. Most of the patients who has their first presentation of symptomatology have underlying LV dysfunction. Thus, their symptoms are due to both primary valve disease and underlying LV dysfunction. Since medical therapy can't prevent the progression the disease process, surgical treatment is highly recommended in this patients. Multiple studies have confirmed the beneficial effects of surgical aortic valve replacement (SAVR) on mortality, symptom relief and increased quality of life at subsequent follow-up[27]. Schwarz[28] in his study has concluded that surgical aortic valve replacement (SAVR) has been proved to improve symptoms and prolong survival, with very low morbidity and mortality. These studies have evaluated the outcomes in patients undergoing aortic valve replacement with underlying mild to moderate LV dysfunction.

There is a scarcity of literature regarding outcomes of patients with aortic valve diseases with underlying severe LV dysfunction undergoing surgical aortic valve replacement. Few studies[29], high-risk surgical patients undergoing open AVR have respectable short and mid-term survival. Also, morbidity outcomes in this subset of patients are less often reported.

In the era of TAVI, there has been renewed interest in the outcomes of conventional AVR for high-risk patients.Although recent studies have demonstrated Transcatheter aortic valve implantation (TAVI) is a relatively safe and effective procedure to treat aortic stenosis in patients who are at extreme or high risk for conventional cardiac surgery[30]. In countries like India, economical constraints have limited the usage of TAVI in such patients. However, in modern era of medical practice, individualized patient care calls for increased knowledge of outcomes so that patients can have realistic expectations following SAVR. The prerequisite for knowledge of morbidity outcomes and future physical performance in the process of share decision-making is imminent. So, in our study, we aim to provide data on patient-relevant outcomes beyond what is known, in an effort to improve decision-making as a result. We report a set of outcomes such as functional class, and echocardiography parameters like ejection fraction , LVEDV and LVESV in patients suffering from severe AS/AR with underlying LV dysfunction after AVR.

AIMS AND OBJECTIVES

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To evaluate outcomes aortic valve replacement in patients with Severe AS/AR associated with severe LV dysfunction in terms of

1. Functional status

2. Left ventricular function as assessed by echocardiography

METHODOLOGY

MATERIALS AND METHODS

Study design: retrospective study

Settings: SCTIMST (tertiary referral centre, University level hospital operating about 100 aortic valve replacement surgeries)

Participants:

Twenty five adult patients with severe AS/AR with severe LV dysfunction who have undergone aortic valve replacement from 2008-2013

Inclusion criteria

1. Adult patients with severe AS/AR with severe LV dysfunction (Ejection fraction <35%) who have undergone AVR from jan 2008- dec 203

Exclusion criteria

1. Patient undergoing emergency /redo surgeries

2. Patients with comorbidities like CKD on dialysis, COPD, Cerebrovascular diseases

3. Patients with coronary artery disease

4. Patients on IABP and prior cardiac arrest

Patient characteristics

Twenty five patients were included. Of these, patients suffered from aortic valve disease. All patients were included only in the presence of a poor LV function (ejection fraction<35%), low transvalvular gradient (<30 mmHg) and enlarged left ventricle (end-diastolic diameter >6 cm, end-systolic diameters >5.3 cm).

20% of study population were females and rest of 80% were males. Average age at surgery was 44years (19-64years). Out of 25 patients, 13 patients were operated for aortic stenosis, in these patients, 6 had bicuspid aortic valve,2 had rheumatic heart disease and 5 had degenerated aortic valve.12 patients were operated for aortic regurgitation, in this group 3 patients had acute regurgitation due to aortic dissection and rest of 9 patients had chronic regurgitation due to rheumatic heart disease. Of the 25 patients, were New York Heart Association (NYHA) class II (18%) and NYHA class III (66%) and NYHA class IV (16%). Patients in NYHA functional class IV were taken for emergency surgery and rest of patients were taken for elective surgery.

Echocardiography

All patients underwent transthoracic echocardiography at admission to the hospital by an experienced cardiologist. After induction of anesthesia, transesophageal echo was performed in all patients prior to surgery. Measurements of left ventricular dimensions were made from 2D echocardiographic images in the parasternal long axis view an M-mode. EF were calculated by modification of Simpson's method with two apical views.

Surgical techniques

All surgical records were reviewed to determine the type and size of aortic valve prosthesis or aortic root enlargement was performed concomitantly with aortic valve replacement.4 patients undergone bentall’s procedure and 21 patients underwent aortic valve replacement only.6 patients received bio-prosthetic valves and rest of the patients received mechanical valves. The average aortic clamp time was 85.6 min. All patient who underwent aortic root replacement surgery received custodial cardioplegia. Rest of patients received St.thomas cardioplegia.

Follow-up

Follow up data was collected from the medical records at 1 month, 3 months, 6 months, 1 yr, 3yr, 5yr. These includes patients functional class (NYHA), echocardiography parameters like LV ejection fraction, LV end-diatolic dimensions and LV end-systolic dimensions.

STATISTICAL ANALYSIS

STATISTICAL ANALYSIS

Statistical analysis was performed using the statistical software package of social science (SPSS 21, Chicago, Illinois, USA). All data were expressed as mean and standard deviation. The relationship of preoperative variables to postoperative ejection fraction and LV dimensions were assessed by repeated measures of ANOVA test. Differences were considered significant at a value of $P<0.05$.

RESULTS

RESULTS

Clinical outcome

The 30-day mortality was 16% (4 of 25 patients) among patients with aortic valve disease and severe LV dysfunction (EF<35%) and aortic valve replacement. Of these, all patients suffered from aortic valve disease with severe left ventricular dysfuction . Two patients died with sepsis with multi organ dysfunction, one patient died on 2nd postoperative day due to decompenstated heart failure and one patient died after 1month with arrthymias. The survival rate of for patients with aortic valve replacement with severe left ventricular dysfunction at 5years was 84%.

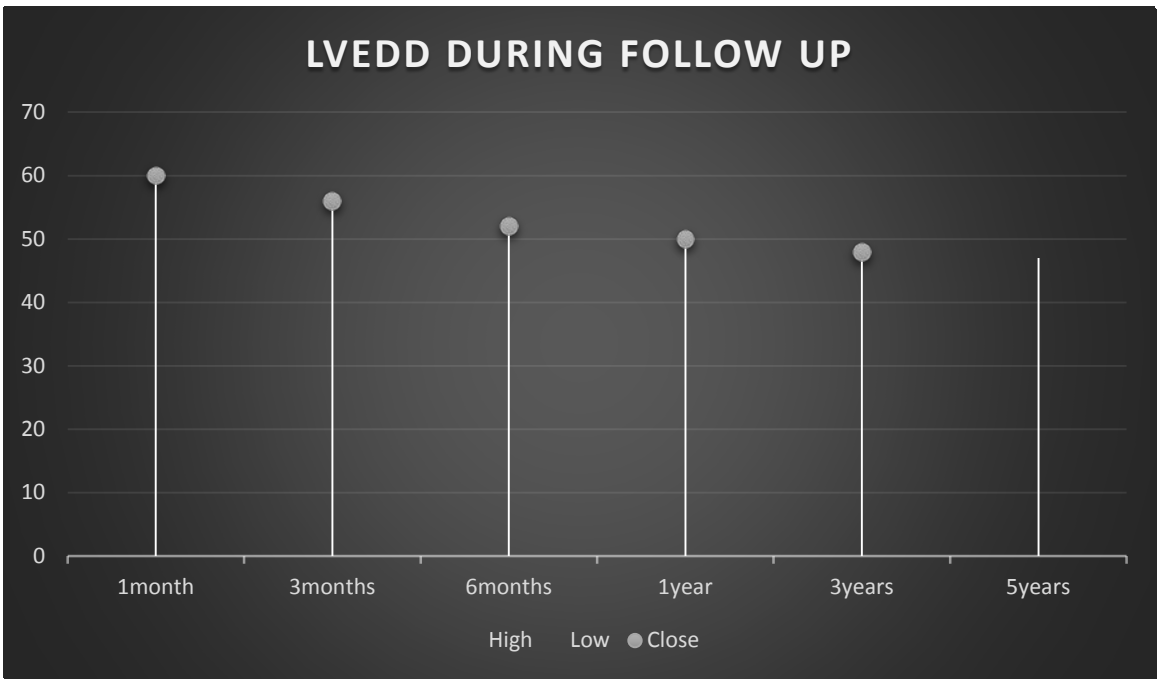
NYHA functional class

Symptomatic improvement was noted in most of the survivors. 82 percent were severely symptomatic (NYHA class III and IV) before and none after operation. The significant change in NYHA functional class III/IV preoperatively vs. follow-up.

LV- Ejection fraction

EF was assessed echocardiographically at follow-up among survivors. All survivors showed a positive change of EF at follow-up. The EF increased significantly during follow up on average at 1month- 40%,3months- 45%,6months -51%,1year- 56%,3years- 57% and 5years- 58%. Preoperative EF is 31+/-5 %, after aortic valve replacement during follow up EF was 55+/-7% at 1year and 58+/-6.8 at 5years.There was significant improvement in LVEF during follow up after aortic valve replacement at end of 1year and 5 year follow up(P<0.001).

Left ventricular diameters



Left ventricular end diastolic dimensions on average at 1month- 60mm, 3months -56mm, 6months- 52mm, 1year -50mm, 3years -48mm and 5year -47.5mm.Preoperative LVEDD was 64+/-8.46mm,after aortic valve replacement at 1year LVEDD was 49.66+/-4.68mm and at 5years LVEDD was 47.57+/-4.1mm.There was a significant improvement in LVEDD during follow up at 1year and 5years(P<0.001)

Ventricular end systolic dimensions on average at 1month- 48mm, 3months- 44mm,6months- 40mm,1year- 37mm,3years- 35mm and 5year- 34.47mm. Pre-operative LVESD was 52.5+/-5.6mm.LVESD after aortic valve replacement at 1year was 36.71+/-6mm and at 5years was 34.47+/-5.7mm. There was a significant improvement in LVESD during follow up at 1year and 5years (P<0.001). Following aortic valve replacement showed significant improvement in left ventricular dimensions during follow up (P<0.001). No patient in this study had severe PPM.

DISCUSSION

DISCUSSION

This is a single center based retrospective study of patients with severe LV dysfunction, who underwent aortic valve replacement for aortic valve disease. The aim of the study is to measure the outcomes in this group of patients following aortic valve replacement in terms of survival rates, functional status and left ventricular function.

In our study twenty five patients with severe LV dysfunction (EF<35%) underwent aortic valve replacement during 2008 to 2013.These patients were followed for 5year. All patients were aged more than 19years, average age at time of surgery was 44years.20% of study group contain female patients. All the patients had no major coronary artery disease, chronic kidney disease, COPD and not on ventilator and IABP preoperatively.

Out of 25 patients, 13 patients were operated for aortic stenosis, in these patients,6 had bicuspid aortic valve,2 had rheumatic heart disease and 5 had degenerated aortic valve.12 patients were operated for aortic regurgitation, in this group 3 patients had acute regurgitation due to aortic dissection and rest of 9 patients had chronic regurgitation due to rheumatic heart disease. The average aortic clamp time was 85.6min. All patient who underwent aortic root replacement surgery received custodial cardioplegia. Rest of patients received st.thomas cardioplegia. There was no significant patient prosthesis mismatch after surgery.

During follow up,5 year survival was 84%. Out of 25 patients 3 had mortality after surgery with in 1month and 1 had mortality after 1month.2 patients died from sepsis with MODS,1 patient died from decompensated heart failure and 1 patient died from ventricular tachy arrthymia after 1month.

During follow up, there was improvement in the functional status of patient, gradually over 6months to 1year.Most of the patients were in class1 at end of 5 years follow up.

Patients in this study during follow up, showed significant improvement in LVEF. Improvement in LVEF was rapid during 1year follow. From 1year to 5 years, there was gradual improvement. Most of LVEF was recovered with in 1year.

During 5 years follow up, LV dimensions measured in terms of LVEDD and LVESD showed a significant improvement. There was a significant improvement in mass/volume ratio during 5 years follow up. Rapid improvement was seen during 1 year follow up.

Aortic stenosis

The aortic valve stenosis is defined as an obstacle to the flow of blood through the aortic valve during left ventricular (LV)ejection. This LV outflow obstruction due to increased systolic blood pressure, prolonged ejection time, increased blood pressure and decreased diastolic aortic pressure is established as a trans-valvular gradient. These alterations are established when the valve area is reduced by at least 50%[31]. The pressure overload is initially compensated by the development of myocardial hypertrophy without dilatation of the LV chamber (concentric hypertrophy) that is able to maintain for many years normal systolic function. The increases in systolic blood pressure, ventricular mass and ejection time lead to increased consumption of oxygen by the myocardium. The increase in oxygen consumption and its contributing to decreased myocardial ischemia cause further deterioration of LV function[32]. In more advanced disease, the disappearance of effective compensatory mechanisms is associated with an imbalance between pump function and LV afterload (afterload mismatch). At this stage, the ventricular chamber dilates, the ejection fraction (EF) is reduced and both the ventricular filling pressure and pulmonary pressure increase. This stage usually coincides with the occurrence of severe

stenosis and the onset of symptoms. Usually, the symptoms in patients with AS appear around the 6th decade of life after a long latency period, characterized by progressive thickening and calcification of the aortic valve or progressive myocardial dysfunction, or both. In patients in whom symptomatic severe AS is not treated, the prognosis is poor. For this reason, the ACC/AHA guidelines[33] recommend valve replacement in patients with severe symptomatic AS (Class I: aortic valve replacement (AVR) is indicated for symptomatic patients with severe AS, with level B of evidence). When the EF begins to decrease, the preload reserve of the LV is often limited by cardiac hypertrophy and increased myocardial stiffness. As the valve narrowing progresses, LV afterload increases further, and the LVEF can become markedly reduced, primarily because of afterload mismatch without preload reserve. The reduction of after load following the aortic valve replacement in patients with aortic stenosis with severe LV dysfunction with preserved myocardium show reverse remodeling. Both cellular hypertrophy and diffuse fibrosis regress, and these changes are accompanied by structural, functional and biomarker improvement. Both cellular hypertrophy and diffuse fibrosis are plastic, whereas focal replacement fibrosis is irreversible. During follow up, the mass/volume ratio decreases as cellular hypertrophy and diffuse fibrosis regress, due to this ventricular wall stiffness decreases, oxygen consumption decreases, diastolic and systolic function improves and ejection increases following aortic valve replacement. In this study there was improvement in functional status, LV EF, LV functions and decrease in mass/volume ratio following aortic valve replacement due to reverse remodeling

Aortic regurgitation

Acute severe AR imposes a sudden excessive volume load on an unprepared LV that is normal in size, resulting in a dramatic extreme rise in LV diastolic pressure (LVDP),

which may approach or indeed equal the aortic diastolic pressure. Because LV pressure exceeds the left atrial pressure during diastole, the resulting rapid ventriculoatrial gradient causes the mitral valve to close prematurely before the onset of the next systole. The premature mitral valve closure is beneficial in the sense that the high LVDP is not transmitted to the pulmonary venous system, thus preventing pulmonary edema and clinical left heart failure. However, the protection afforded by premature mitral valve closure is lost when a further rise in the ventriculoatrial gradient opens the mitral valve in late diastole, leading to diastolic mitral regurgitation. Mitral regurgitation in acute AR may occur either in diastole or in systole (when the LVDP exceeds the left atrial pressure). It is likely that persistence of the ventriculoatrial gradient, as a result of extension of the high LVDP level to the isovolumic contraction period and the early systole, causes the mitral valve to open during this period, resulting in early systolic mitral regurgitation. Mitral regurgitation is usually effective to lower LVDP; the left atrium thus serves as a reservoir for blood regurgitated from the aorta to the LV. However, left atrial pressure may rise further, leading to pulmonary edema and circulatory failure. Chronic aortic regurgitation causes volume overload of the left ventricle (LV). The total stroke volume ejected by the LV (sum of effective stroke volume plus regurgitant volume) is increased; in severe AR regurgitant volume may equal or even exceed effective stroke volume. An increase in LV end-diastolic volume is the main compensatory mechanism needed to maintain a normal effective stroke volume. Left ventricular ejection fraction is initially normal, however, LV end-diastolic pressure rises. In time LV end-diastolic volume continues to increase further and ejection fraction drops; these changes may actually precede the development of clinical symptoms. Considerable eccentric myocardial hypertrophy can occur with chronic AR and at autopsy heart weights of up to 1000 g have been reported.In chronic AR there is not only

volume overload but also an increase in afterload and therefore of systolic wall stress. Surgical correction of AR results in a decrease in afterload and frequently an improvement of the ejection fraction.

Following aortic valve replacement in patients with aortic regurgitation shows improvement in functional status, LVEF and LV dimensions. In case of acute AR, valve replacement removes the regurgitation hence LVDP decreases and eliminates the mitral regurgitation. Thus improves LV function. In case of chronic AR, following aortic valve replacement the LVEDV decreases and reverse remodeling of eccentric myocardial hypertrophy lead to decrease in afterload and oxygen consumption. Thus LVEF, LV dimensions improves during follow up. After AVR, LV reverse remodeling occurs both in patients with acute and chronic AR[34]. Thus patients in the current study showed improvement in functional class, LVEF and LV dimensions during follow up.

CONCLUSIONS

CONCLUSIONS

Surgical AVR yields excellent short- and long-term outcomes for severe aortic valve disease patients with underlying LV dysfunction. Another important finding to is that SAVR is capable of reestablishing survival to that expected for an age- and gender-matched population sample, demonstrating the important benefit of cost effective benefits of SAVR in the treatment of this high-risk patients .

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Rapaport E: Natural history of aortic and mitral valve disease. Am J Cardiol 35: 221, 1975.

2. Frank S, Johnson A, Ross J Jr: Natural history of valvular aortic stenosis. Br Heart J 35: 41,1973

3. Ross J Jr, Braunwald E: Aortic stenosis. Circulation 38 (suppl V): V-61, 1968

4. 4.Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP III, Guyton RA, et al. 2014AHA/ACC Guideline for the Management of Patients with Valvular Heart Disease: Executive Summary: a report of the American College of Cardiology/American Heart Association TaskForce on Practice Guidelines.J Am Coll Cardiol. 2014;63:2438-88.

5. Baumgartner H, Hung J, Bermejo J, Chambers JB, Evangelista A, Griffin BP,et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendationsfor clinical practice. J Am Soc Echocardiogr. 2009;22:1-23: quiz 101-2.

6. Bristow JD, Kremkau EL: Hemodynamic changes after valve replacement with Starr-Edwards prosthesis. Am J Cardiol 35: 716

7. Starr A, Grunkemeier, Lambert LE, Thomas DR, Sugimura S, Lefrak EA: Aortic valve replacement: a ten year followup of non-cloth-covered vs cloth-covered caged ball prosthesis. Circulation 56 (suppl I1): 11-133, 1977.

8. Barnhorst DA, Oxman HA, Connolly DC, Pluth JR, Danielson GK, Wallace RB, McGoon DC: Long-term followup of isolated replacement of the aortic or mitral valve with the Starr-Edwards prosthesis. Am J Cardiol 35: 228, 1975, 1975

9. Dodge HT, Frince M, Stewart DK: Functional evaluation of the hypertrophied heart. Circ Res 35: (suppl 11): 11-122, 1974.

10. Kennedy JW, Doces J, Stewart DK: Left ventricular function before and following aortic valve replacement. Circulation 56 ,944, 1977

11. Pantely G, Morton MJ, Rahimtoola SH: Effects of successful uncomplicated valve replacement on ventricular hypertrophy, volume, and performance in aortic stenosis and aortic incompetence. J Thorac Cardiovasc Surg 75: 383, 1978

12. Rosenhek R, Iung B, Tornos P, Antunes MJ, Prendergast BD, Otto CM, et al. ESC Working Group on Valvular Heart Disease Position Paper: assessing the risk of interventions in patients with valvular heart disease. Eur Heart J. 2012;33(7):822–8.

13. Nkomo VT, Gardin JM, Skelton TN, et al. Burden of valvular heart diseases: a population-based study. Lancet 2006;368:1005–11.

14. Niloufar Samiei, Mohammad Reza Hakimi, Yalda Mirmesdagh, Mohammad Mehdi Peighambari, Alireza Alizadeh-Ghavidel, and Saeid Hosseini. Surgical outcomes of heart valves replacement: A study of tertiary specialied cardiac center. ARYA Atheroscler. 2014 Sep; 10(5): 233–237.

15. Morton BC. Natural history and management of chronic aortic valve disease. Can Med Assoc J. 1982; 126: 477-80.

16. Kodali SK, Williams MR, Smith CR, et al; PARTNER Trial Investigators. Two-year outcomes after transcatheter or surgical aortic-valve replacement. N Engl J Med 2012; 366:1686-95.

17. Management of Valvular Heart Disease of the European Society ofCardiology (ESC)European Association for Cardio-Thoracic Surgery(EACTS). Guidelines on the management of valvular heart disease. Eur Heart J 2012;33:2451–96.

18. Carabello BA, Paulus WJ. Aortic stenosis. Lancet. 2009;373 :956–966.

19. Otto CM, Bonow RO. Valvular Heart Disease: A Companion to Braunwald's Heart Disease. 3rd ed. Philadelphia, PA: Elsevier Saunders; 2009.

20. Rajamannan NM, Evans FJ, Aikawa E, et al. Calcific aortic valve disease: not simply a degenerative process: a review and agenda for research from the National Heart and Lung and Blood Institute Aortic Stenosis Working Group. Executive summary: calcific aortic valve disease-2011 update. Circulation. 2011;124(16):1783–1791.

21. Kurtz CE, Otto CM. Aortic stenosis: clinical aspects of diagnosis and management, with 10 illustrative case reports from a 25-year experience. Medicine. 2010;89(5):349–379.

22. Bonow RO, Mann DL, Zipes DP, Libby P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 9th ed. Philadelphia, PA: Elsevier Saunders; 2012.

23. Bloechlinger S, Grander W, Bryner J, Dünser MW. Left ventricular rotation: a neglected aspect of the cardiac cycle. Intensive Care Med. 2011;37(1):156–163.

24. Arabello BA, Paulus WJ. Aortic stenosis. Lancet. 2009;373(9667):956–966.

25. 25.Ozkan A, Kapadia S, Tuzcu M, Marwick TH. Assessment of left ventricular function in aortic stenosis. Nat Rev Cardiol. 2011;8(9):494–501.

26. Gerald Maurer. Aortic Regurgitation. Heart . 2006 Jul; 92(7): 994–1000.

27. Bouma BJ, van Den Brink RB, van Der Meulen JH, et al. To operate or not on elderly patients with aortic stenosis: the decision and its consequences. Heart 1999;82:143–8.

28. Schwarz, P. Baumann, J. Manthey, M. Hoffmann, G. Schuler, H.C. Mehmel, et al.The effect of aortic valve replacement on survival Circulation, 66 (1982). 1105-1110.

29. Thourani VH, Gorav Ailawadi, Szeto W, Todd M. Dewey, Robert A. Guyton, Michael J. Mack, Outcomes of Surgical Aortic Valve Replacement in High-Risk Patients: A Multi institutional Study . Ann Thorac Surg 2011;91:49 –56.

30. Cohen EA, Ko DT, Oakes GH, Koh M, Guo H, Natarajan MK, Wijeysundera HC. Outcomes following transcatheter aortic valve implantation (tavi) in ontario. Canadian journal of cardiology. 31, 2015.

31. Ross J, Braunwald E. Aortic Stenosis. *Circulation* 1968; **38**(Suppl)**:** 61 – 67.

32. Gravanis MB, Robinson PH, Hertzler GL. Hypertrophic cardiomyopathy evolving into a hypokinetic and dilated left ventricle: Coronary embolization as a probable pathogenetic mechanism. *Clin Cardiol* 1990; **13:** 500 – 505.

33. Bonow RO, Carabello BA, Chatterjee K, de Leon AC, Faxon DP Jr, Freed MD, et al. 2008 Focused Update Incorporated Into theACC/AHA 2006 Guidelines for the Management of Patients WithValvular Heart Disease: A Report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1998 Guidelines for theManagement of Patients With Valvular Heart Disease): Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *Circulation*2008; **118:** e523 – e661.

34. Regeer MV, Versteegh MI, Ajmone Marsan N, Schalij MJ, Klautz RJ, Bax JJ .Left ventricular reverse remodeling after aortic valve surgery for acute versus chronic aortic regurgitation. 2016 Oct;33(10):1458-1464. doi: 10.1111/echo.13295. Epub 2016 Jun 25

APPENDIX

ABBREVIATIONS

LV	-	Left Ventricular
AS	-	Aortic Stenosis
AR	-	Aortic Regurgitation
EF	-	Ejection Fraction
LVEDD	-	Left Ventricular End Diastolic diameter
LVESD	;	Left Ventricular end Systolic diameter
PPM	-	Patient Prosthesis Mismatch

OBSERVTAION CHART

Observations

Patient name

Age

Sex

Diagnosis

Surgery

Coronary angiography

Medications

Pre operative	1 month	3 months	6 months	1 year	3 years	5 years
Functional Class						
EF						
LVEDD						
LVESD						
Survival						

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OUTCOMES OF AORTIC VALVE REPLACEMENT IN AORTIC STENOSIS AND REGURGITATION WITH SEVERE LEFT VENTRICULAR DYSFUNCTION-RETROSEPCTIVE STUDY.

P.SIRISH

S.SNO	AGE	SEX	DIAGNOSIS	SURGERY	PREOP FUNCTIONAL STATUS	PREOP ECHO				INTRAOPERATIVE		POST OP FUNCTIONAL STATU					
						LVEF	LVEDD	LVESD	AS/AR	ACT	PPM						
												1MONTH	3MONTS	6MONTHS	1YEAR	3YEARS	5YEARS
1	29	f	ascending aortic dissection with severe AR	Bentall's procedure(avr#25chvp)	class 3	35	61	50	severe	100	no	class2	class 1	class1	class1	class1	class1
2	29	m	annuloaortic ectasia with sev.ar	Bentall's procedure(avr#25chvp)	class 3	36	76	51	severe	89	no	class2	class 1	class1	class1	class1	class1
3	48	m	ascending aortic dissection with severe AR	Bentall's procedure(avr#25chvp)	class 3	35	66	48	severe	107	no	class2	class2	class2	class2	class2	class2
4	49	m	annuloaortic ectasia with sev.ar	bentall's procedure(avr#29chvp)	class 3	36	61	50	severe	102	no	class2	class2	class2	class2	class1	class1
5	30	m	rhd sev.ar	avr#21chvp	class2	20	80	59	severe	98	no	class2	class2	class1	class1	class1	class1
6	19	f	type a aortic dissection with sev.ar	bentall's procedure#25chvp	class 4	17	68	63	severe	95	no	expired					
7	56	m	bav sev as	avr#21PM	class3	33	59	47	severe	110	no	class2	class1	class1	class1	class1	class1
8	55	m	bav sev as	avr#23 c-e perimount	class2	37	71	58	severe	97	no	class2	class1	class1	class1	class1	class1
9	53	m	rhd sev ar p/ie	avr#23mh	class3	33	68	58	severe	62	no	class2	class2	class1	class1	class1	class1
10	39	m	rhd sev.as	avr#25sjm	class3	32	59	49	severe	75	no	class2	class2	class1	class1	class1	class1
11	26	m	rhd sev ar	avr#23sjm	class2	24	48	39	severe	75	no	expired					
12	36	m	bav sev as	avr#19sjm	class2	27	50	44	severe	107	no	class2	class2	class1	class1	class1	class1
13	37	f	rhd sev ar	avr#25sjm	class3	35	71	59	severe	68	no	class2	class2	class1	class1	class1	class1
14	64	m	bav sev as	avr#23chvp	class3	33	53	44	severe	55	no	class2	class1	class1	class1	class1	class1
15	32	m	bav sev as	avr#29chvp	on ionotopes with class 4	20	62	54	severe	97	no	class2	class2	class2	class1	class1	class1
16	61	f	sev.cal.as	avr#19pm	cardiac arrest-emergency	30	67	53	severe	78	no	expired					
17	62	m	sev.cal.as	avr#23mtx	class3	35	62	46	severe	109	no	class2	class2	class2	class1	class1	class1
18	62	m	sev.cal.as	avr#19pm	class3	30	54	45	severe	106	no	class2	class2	class1	class1	class1	class1
19	20	m	rhd sev ar	avr#23chvp	class3	33	69	56	severe	78	no	class2	class2	class2	class1	class1	class1
20	44	m	sev.cal.as	avr#27chvp	class2	33	65	57	severe	63	mild ppm	class2	class2	class1	class1	class1	class1
21	42	m	rhd sev ar	avr#25chvp	class2	30	76	61	severe	58	no	class2	class2	class1	class1	class1	class1
22	62	f	bav sev as	avr#19pm	decompenstated chf	15	53	44	severe	108	no	expired					
23	52	m	sev.cal.as	avr#23pm	class2	30	69	59	severe	76	no	class2	class2	class1	class1	class1	class1
24	37	m	rhd sev ar	avr#23chvp	class3	35	73	56	severe	57	no	class2	class2	class2	class1	class1	class1
25	54	m	rhd sev as	avr#21chvp	class3	22	57	52	severe	70	no	class2	class2	class2	class2	class2	class2

POST OP ECHO																	
LVEF						LVEDD						LVESD					
1MONTH	3MONHS	6MONTHS	1YEAR	3YEARS	5YEARS	1MONTH	3MONTHS	6MONTHS	1YEAR	3YEARS	5YEARS	1MONTH	3MONTHS	6MONTHS	1YAER	3YAERS	5YEARS
42	45	50	50	50	50	61	61	55	50	50	50	48	45	45	40	40	40
45	48	52	52	52	52	65	60	58	54	54	52	50	46	44	42	42	42
50	56	60	64	64	64	60	55	50	40	40	40	45	40	35	30	30	27
50	55	60	65	65	65	60	60	55	55	50	50	50	45	40	35	35	35
46		48	52	54	54	62	58	58	54	52	50	58	55	50	46	40	40
55	58	65	65	65	65	46	46	46	46	46	46	29	28	28	28	28	28
42	48	54	56	56	56	67	60	55	54	54	54	56	50	45	40	40	40
42	46	52	54	58	60	64	64	60	58	54	50	56	50	48	45	40	34
35	40	45	50	55	60	56	53	51	51	48	48	45	40	36	36	35	35
35	44	56	58	58	60	48	42	40	40	40	40	36	28	22	22	22	22
36	40	52	56	60	60	54	50	50	50	50	50	48	44	40	35	35	35
40	44	50	56	56	60	55	52	50	48	48	48	44	42	40	38	38	38
42	46	55	59	59	60	60	58	57	53	50	50	52	48	44	40	40	38
42	48	52	58	58	60	60	55	52	48	48	48	45	42	36	32	30	30
48	50	56	66	66	66	52	48	46	44	42	42	38	33	30	27	27	26
35	38	40	44	48	52	64	58	55	51	48	48	53	48	44	42	40	40
38	44	50	55	62	62	60	55	50	48	45	45	50	44	40	36	34	28
34	38	44	50	56	56	72	66	60	54	52	52	56	50	44	42	38	38
35	39	44	50	56	56	64	60	54	50	50	50	51	47	44	40	40	40
35	40	44	53	58	58	65	60	55	50	50	50	55	50	44	40	38	38
35	40	50	55	60	60	66	56	50	47	40	40	50	45	40	35	30	30
27	30	30	35	35	35	57	55	50	48	46	46	48	44	44	40	40	40