A dissertation on

MITRAL VALVE REPLACEMENT WITH CHORDAL PRESERVATION-A RETROSPECTIVE ANALYSIS OF OUTCOME IN COMPARISON WITH CLASSICAL MITRAL VALVE REPLACEMENT

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CERTIFICATE

This is to certify that the dissertation entitled "MITRAL VALVE REPLACEMENT WITH CHORDAL PRESERVATION A RETROSPECTIVE ANALYSIS OF OUT COME IN COMPARISON WITH CLASSICAL MITRAL VALVE REPLACEMENT" presented here is the original work done by Dr. N. Girish in the department of cardio thoracic surgery, Govt General Hospital ,Madras Medical college, Chennai 600003, in partial fulfillment of the University rules and regulations for the award of Mch Cardiothoracic degree under our guidance and supervision during the academic period from 2003-2005.

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Introduction

t is well known that traditional mitral valve replacement when compared to mitral valve repair carries a higher morbidity and mortality. This has been attributed to the preservation of the mitral subvalvular apparatus in repair techniques, but not all valves can be repaired, especially those of rheumatic etiology. Mitral valve replacement has been the procedure of choice usually adopted in these conditions, but the results have not been comparable. By preserving the annular ventricular continuity in mitral valve repair good LV function both early and late post operative period has been achieved.

A surgery in which annulo-papillary continuity is preserved during replacement has shown better early and late results than traditional MVR techniques.

As the awareness of the deleterious effects of the loss of annulo -ventricular continuity has increased chordal preservation during mitral valve replacement has gained in popularity.

Anatomy of mitral valve

Anatomy of mitral valve

Mitral valve consists of 1)an annulus.2) leaflets.3) subvalvar apparatus of chordae and papillary muscles.

The mitral annulus is a pliable junctional zone of fibrous and muscular tissue joining the left atrium and ventricle that anchors the hinge portion of the anterior and posterior mitral leaflets. The annulus has two major collagenous structures: (1) the right fibrous trigone, which is part of the central fibrous body and is located at the intersection of the atrioventricular membranous septum, the mitral and tricuspid valves, and the aortic root; and (2) the left fibrous trigone at the junction of the mitral valve and left coronary cusp of the aortic valve). The anterior mitral leaflet spans the distance between the commissures (including the trigones) and is in direct fibrous continuity with most of the left and noncoronary aortic valve cusps.

The mitral valve has two major leaflets, the much larger anterior (or aortic) leaflet and the smaller posterior (or mural) leaflet; the latter usually contains three (or sometimes more) scallops separated by clefts which are developed to variable degrees in different individuals. The central portions of the leaflets on the atrial surface are termed the *rough zone*, with the remainder of the free edge leaflet surface being the *clear zone*. The ratio of the height of the rough zone to the height of the clear zone is 0.6 for the anterior leaflet and 1.4 for the posterior leaflet, as the clear zone on the posterior scallops is only about 2 mm high. The two leaflets are separated at the annulus by the posteromedial and anterolateral commissures, which are usually distinctly developed.

The histological structure of the leaflets includes three layers: (1) the fibrosa, the solid collagenous core that is continuous with the chordae tendinae; (2) the spongiosa, which covers the atrial aspect and forms the leaflet leading edge (it consists of few collagen fibers but has abundant proteoglycans, elastin, and

mixed connective tissue cells); and (3) a thin fibroelastic covering of most of the leaflets. On the atrial aspect of both leaflets, this surface (the *atrialis*) is rich in elastin. The ventricular side of the fibroelastic cover (the *ventricularis*) is much thicker; it is confined mostly to the anterior leaflet and is densely packed with elastin.

Annular Size, Shape, and Dynamics

The average mitral annulus cross-sectional area ranges from 5.0 to 11.4 cm^2 in normal human hearts (average is 7.6 cm^2). The posterior annulus circumscribes about two thirds of the mitral annulus. The magnitude of change in mitral annular area is in the 20% to 40% range. Annular area decreases to a minimum in early to mid systole. The human mitral annulus is roughly elliptical (or D- or kidney-shaped), with greater eccentricity (i.e., being less circular) in systole than in diastole, in its most elliptical configuration, the ratio of minor to major diameters is approximately 0.75 the. During the cardiac cycle, annular regions adjacent to the posterior leaflet (where the leaflet attaches directly

to the atrial and ventricular endocardium) move toward (during systole) and away from (during diastole) the relatively immobile anterior annulus

Chordae Tendinae and Papillary Muscles

Epicardial fibers in the left ventricle descend from the base of the heart and proceed inward at the apex to form the two papillary muscles, which are characterized by vertically oriented myocardial fibers. The anterolateral papillary muscle usually has one major head and is a more prominent structure; the posteromedial papillary muscle can have two or more subheads and is flatter. A loop from the papillary muscles to the mitral annulus is completed by the chordae tendinae continuing into the mitral leaflets, which are then attached to the annular ring. The distance from the tip of the human papillary muscle to its corresponding mitral annulus averages 23.5 mm from the tip of the anterolateral papillary muscle to the left trigone, and 23.2 mm to the point between the anterior and middle scallops of the posterior leaflet. The distance

from the tip of the posteromedial papillary muscle to the right trigone is 23.5 mm and to the annular point between the middle and posteromedial scallops of the posterior leaflet is 23.5 mm. The posteromedial papillary muscle is usually supplied by the right coronary artery (or a dominant left circumflex artery in 10% of cases); the anterolateral papillary muscle is supplied by both the left anterior descending and circumflex coronary arteries

The posteromedial and anterolateral papillary muscles give rise to chordae tendinae going to both leaflets. The chordae are classically and functionally divided into three groups. First-order chordae originate near the papillary muscle tips, divide progressively, and insert on the leading edge of the leaflets; these primary chordae prevent valve edge prolapse during systole. The second-order chordae (including two or larger and less branched "strut" chordae) originate from the same location and tend to be thicker and fewer in number they insert on the ventricular surface of the leaflets at the junction of the rough and clear zones, which is demarcated by a ridge corresponding to the line of leaflet

coaptation. The second-order chordae (including the strut chordae) serve to anchor the valve and are more prominent on the anterior leaflet; second-order chordae may also arborize from large chordae that go to the leaflet free edge (first-order chordae). The third-order chordae, also called tertiary or basal chordae, originate directly from the trabeculae carnae of the ventricular wall, attach to the posterior leaflet near the annulus, and can be identified by their fan-shaped patterns. Additionally, distinct commissural chordae and cleft chordae exist in the commissures. In total, about 25 major chordal trunks (range 15 to 32) arise from the papillary muscles in humans, equally divided between those going to the anterior and posterior leaflets.

During diastole, the papillary muscles form an inflow tract; during systole, they create an outflow tract, which later becomes obliterated due to systolic thickening of the papillary muscles and augments LV ejection by volume displacement. The anterior and posterior papillary muscles contract simultaneously.

Mitral Regurgitation

Four different types of structural changes of the mitral valve apparatus may produce regurgitation: leaflet retraction from fibrosis and calcification, annular dilation, chordal abnormalities (including rupture, elongation, shortening, or apical tethering or "tenting" as seen in FMR and IMR), and possibly papillary muscle dysfunction. Carpentier et al classified mitral regurgitation into three pathoanatomic types based on leaflet and chordal motion: normal leaflet motion (type I), leaflet prolapse or excessive motion (type II), and restricted leaflet motion (type III). Type III is further subdivided into "a" and "b" based on leaflet restriction during diastole (type IIIa) or during systole (type IIIb, as typically seen in patients with IMR) Mitral regurgitation with normal leaflet motion is caused by annular dilation, which is often secondary to LV dilation; as a rule, insufficient leaflet coaptation area or incomplete mitral leaflet coaptation is present. Examples include patients with dilated cardiomyopathy and some with ischemic heart disease

complicated by IMR. Normal leaflet motion is also associated with leaflet perforation secondary to endocarditis. Leaflet prolapse typically results from a floppy mitral valve with chordal elongation and/or rupture, but can be seen in patients with coronary artery disease who have papillary muscle rupture or, rarely, papillary muscle elongation. Mitral regurgitation due to restricted leaflet motion is associated with rheumatic valve disease (type IIIa and type IIIb), ischemic heart disease (IMR with type IIIb restricted systolic leaflet motion with or without annular dilation), and dilated cardiomyopathy (type IIIb plus annular dilation).

In chronic mitral regurgitation the LV function gradually declines. The regurgitant volume into the left atrium during systole is added to the forward stroke volume and tends to increase the total forward output and ejection fraction in early phase. However progressive LV dilation increasing the wall tension which leads to increased systolic wall stress

and afterload.

After MVR, with rapid rise in the afterload, adaptation of the LV to this change is based on the annulo ventricular continuity.

Fixation of the mitral annulus with a rigid prosthesis interferes with the distention and contraction of the basoconstrictor. Also after MVR the LV volume decreases because of the elimination of the regurgitant LA volume. After MVR with chordal transaction the EF is determined by contractility; preload and after load. Afterload is increased because of loss of low impedance pathway into LA; also reducing preload is the loss of regurgitant LA volume plus a residual gradient across the prosthesis impedes LV filling.

All this contributes to a low cardiac out put syndrome despite satisfactory prosthetic valve function.

All the above mentioned problems were not seen with traditional mitral valve repair technique.

Review of literature

Review of literature

In 1922 Wiggers and Kats and later Rushmer et al proposed the concept of annulo -ventricular continuity. According to this concept, the LV geometry and functional are a result of a dynamic interaction between mitral annulus and left ventricular wall. The attachment between the mitral annulus and LV wall moderates the LV distensibility during diastole and wall stress during systole.[1]

First effective open approach using cardiopulmonary by pass was not made until 1957 by Lillehei and colleagues and later independently by Merendino and Bruce.[5]

In 1960 McGoon and colleagues described an effective repair for MR due to ruptured chordae[4]

The first mitral valve prosthesis was implanted on Sept 21 1961 by Albert Starr and Edwards from University of Orgean.

Problems of post op low cardiac output, thromboembolism, anticoagulation and bleeding came to the fore.[12]

In 1964 Lillehei introduced the concept of chordal preservation during mitral valve replacement to reduce the problems of post operative low cardiac output syndrome. But his observations went unheeded at that point of time.[15, 39]

In the original technique described by Lillehei the posterior leaflet was bound to the annulus with a running stitch. He reported a reduction of peri operative, mortality from 37% to 14%.[15]

During the same period Mitral valve repair gained acceptance by removing the need for anticoagulation and better left ventricular function and hemodynamics afforded by it;

In1981 David reintroduced the concept of annulo ventricular continuity after extensive experiments in canine models with mitral valve replacement. He introduced the concept of total chordal

preservation.[5,6,7]

In the technique originally described by him the AML is incised at its base and carried to both sides and brought centrally towards the free edge of the leaflets and a triangular segment of the leaflet is excised leaving the chordae attached to the free edge which is resuspended to the mitral annulus by sutures used to secure the prosthetic valve. The PML with its chordae is left intact, in patients with myxomatous MR undergoing MVR chordae are shortened by imbricating the PML in the mitral annulus using sutures used for MV fixation. The advantage he claimed with this technique was that the chordae were maintained in their natural anatomic location with reduced risk of LVOT obstruction with the reduction in the bulk of leaflet tissues. Miki Goor and Hennin and their colleagues produced clinical results suggesting that chordal preserving during mitral valve replacement improved hospital survival and global LV function. Carabello's group again documented preservation of systolic ejection performance in mitral valve replacement with chordal preservation

In 1985 Feike et al reported a technique where the anterior leaflet was split from the centre of the free edge to the annulus. Incisions were made on either side of the split towards the commisures to detach the AML from the annulus the remaining two halves of the leaflet were left with the intact chordae leaflet was trimmed to remove thickened and calcific areas and then rotated posterior and sutured to the posterior mitral annulus. This technique was suitable in implanting tilting disc prosthesis where disc entrapment by the subvalvar apparatus was of concern.[42]

Disadvantages reported were that this disturbs the normal geometry and relationships of mitral subvalvar apparatus which could alter the distribution of the LV wall stress disturb chordal tension during papillary muscle contraction thereby reducing the global LV systolic and diastolic function.

In 1988 Khonsari et al described a technique of total chordal preservation where after detaching the annulus between the two commisures an ellipse of tissue was excised and the rim of leaflet

tissue containing chordae was attached to the annulus (Khonsari I) if leaflet was thickened and calcified it was then divided into 2-5 segments bearing chordae which were re attached to the annulus (Khonsari II). PML was retained in toto. Using this technique no LVOTO or prosthetic function impairment was claimed[33]

Myocardial rupture was prevented by maintaining the tethering effect of the intact sub valvar apparatus.

In1988 Miki's described a technique to maintain more normal chordal tension wherein AML was incised and separated from annulus and divided at its center, anterior and posterior commisures were incised and papillary muscles were slit, excessive cuspal tissue and fibrous calcific nodules were excised. The two chordal segments thus created were sutured to the respective antero lateral and posteromedial commisures. PML was incised in center and prosthetic valve seated in position plicating the PML and including the PML and chordae in the sutures for securing valve. Advantages claimed were that this

technique was simple, there is no LVOTO, allows a larger prosthesis to be seated and very well suited for low profile valves.[27]

In 1990 Rose and Oz described a technique were the AML was stretched posteriorly and a central portion was excised, the rim of the remaining leaflet tissue containing the marginal chordae, defect in the AML was closed with a running suture parallel to the annulus. Valve sutures reinforced the previous suture line. Prosthetic valve was sutured to orient the leaflets perpendicular to the native orientation.[36]

There were low chances of LVOTO with reduced bulk of leaflet tissue, reduced risk of thrombosis on the residual leaflet.[44,45]

Vander Slam described yet another technique for preserving anterior leaflet.[41]

Sarris et al demonstrated that in an open chest swine model the changes induced by chordal transection could be completely reversed by reattaching the papillary muscle to the mitral annulus.[21,23]

Experiments by Hansen and associates demonstrated that LV function was superior with intact subvalvar apparatus, intermediate with preservation of PML alone and poorest with loss of all chordae.[23]

Horskote et al showed that partial chordal preservation preserving PML alone improved event free survival[14]

Hennein et al showed that after chordal excision, exercise capacity, LV systolic dimension and cardiac index did not improve and that the LV function declined. In contrast after preservation of

the entire sub valvar apparatus the exercise capacity improved markedly, LV function improved and early ejection fraction improved [22]

David et al reported results in patients undergoing re do mitral valve replacement after initial MVR, where all chordae were transected. 4-0 PTFE sutures were used to create new chordae if they were transected previously. Incidence of low cardiac out put and operative mortality was low in the chordal preservation and reconstruction group.[8]

Hetzers study reported major advantages of chordal preservation in the form of reduction in the mortality, improved early and late left ventricular function improved long term survival and elimination of the risk of the dreaded complication of midventricular rupture.[48]

Hassan Mottcha et al in their study noted that total chordal preservation did not cause left ventricular tract obstruction after mitral valve replacement.[38]

Ghiskine Deklunder et al in their prospective study noted that anterior chordal transaction impairs not only regional left ventricular function but also regional right ventricular function in mitral regurgitation.[50]

Aims and objectives

The aim in this study was to study in retrospect the possible out come benefits of chordal sparing surgery when mitral valve replacement is done for rheumatic mitral regurgitation in terms of left ventricular function by assessing parameters of left ventricular systolic and diastolic function.

The parameters assessed were hemodynamic stability in the immediate peri and post operative period, left ventricular ejection fraction preoperative and postoperative, left ventricular end systolic and diastolic dimensions pre and postoperative .

Patients and method

343 Patients underwent mitral valve replacement between July2003 and August 2005. Of these 105 patients underwent mitral valve replacement surgery for chronic dominant mitral valve regurgitation. In 55 of these procedures the classical mitral valve replacement technique was followed. In 50 patients chordal sparing technique was followed, in 10patients both AML and PML and in 40 the posterior chordal apparatus was alone preserved

In both groups of patients preoperative NYHA class, LV end systolic, end diastolic dimensions and preoperative ejection fraction were noted. Surgery was conducted with a standard mid sternotomy incision, bicaval and aortic cannulation, core was cooled to 28*C,. Heart was arrested with hyperkalemic blood cardioplegia, with

topical ice slush being used to cool myocardial temperature further. Left atrium was opened parallel to the interatrial groove. Surgery was conducted after inspecting the valve and suitability for chordal preservation assessed.

In majority a Starr Edwards caged ball prosthesis model 6120 was used. the other valve used being St. Jude mechanical bileaflet prosthetic valve . Suturing was done with continuous 2-0 ethibond suture in the classical MVR patients or after plicating the posterior leaflet with the valve fixation suture in the PML area and dividing and reattaching the anterior leaflet chordae in the complete preservation group. Patients were electively ventilated post operatively with inotropic supports being dictated by the hemodynamics of the patient. The patients out come after surgery where the subvalvar apparatus was preserved either completely or partially were compared against the group in whom the

classical technique was followed. The variables assessed were post operative needs for and dosage of multiple inotropic supports, duration of ventilator support. Post operative LV function was assessed with a pre discharge echo cardiography. The parameters noted were the LV end systolic and end diastolic dimensions post operative LV ejection fraction, reduction in NYHA class.

Tables

Table no 1 age distribution

Patients (years)	Class MVR	Chordal spr MVR
10-20	12	5
21-30	16	16
31-40	14	21
41-50	11	7
51-60	02	01

Table no 2

Sex distribution

	Class MVR	Chdr spr MVR
females	25	20
males	30	30

Table no 3

Symptoms

	Class MVR	Chdr spr MVR
NYHA I	nil	nil
NYHA II	43	36
NYHA III	12	14
NYHA IV	nil	nil

Table no 4

Associated co morbidities

Associated conditions	Class MVR	Chdr Spr MVR
TR > mod	16	12
AR	8	12
CAD	2	nil

Table no 5

Ejection fraction

EF	Class MVR	Chdr Spr MVR
>70%	4	2
60-70%	36	34
50-59%	8	10
<50%	7	4

Table no 6

Let ventricular systolic dimensions

Dimensions (mm)	Class MVR	Chdr Spr MVR
>50	4	2

40-50	22	18
30-39	24	24
20-29	5	6

Table no7

Electrocardiogram atrial fibrillation

Rhythm	Class MVR	Chdr Spr MVR
Sinus rhythm	40	32
Atrial fibrillation	15	18

Table no 8

Surgery

Classical MVR	55
Chdr Spr : PML only	40
: AML+PML	10

Table no 9

Ventilator Support.

surgery	12 hrs	24hrs	24-36hrs	>36hrs
Clas MVR	16	20	10	9
CSPML +AML	7	2	1	
PML only	18	16	4	2

Table no 10

Morbidity AND Mortality

	Classical MVR	Chdr MVR
Re-exploration	6	4
Low out put state	5	4
Wound infection	12	16
PVE	nil	1
Mortality	6	5

Table no 11

Inotropic supports

inotrope	Classical MVR	Chrdal spar MVR
dopamine	34	37
Dopa+dobutamine	14	11
Dopa+dobut+adrnl	7	2

Table no 12

Pre Discharge echo LV dimensions and EF

surgery	EF dec	EF inc	LVeSD inc	LVeDD dec
CLASSIC	34	15	40	9
CSP PML	29	6	28	7
AML+PML	2	8	3	7

Observation

A total number of 343 patients underwent mitral valve replacement for isolated mitral valve disease between July 2003 to august 2005.Of this 105 (30.6%) patients were operated for dominant mitral valve regurgitation .The rest had combined dominant mitral stenosis with regurgitation or were more of stenotic valve.

Of the 105 patients who underwent surgery for dominant regurgitant lesion fifty had either PML or PML and AML preserved (47.6%).the rest had classical MVR (53%) The Khonsari I or the

Feikes technique was adopted in the both chordal preservation group.

The valve implanted was Starr Edwards caged ball valve model no 6120 in 80 patients and 25 St Jude mechanical bileaflet valve in 25 patients

Table no 1 Age distributions.

In the classical MVR group the majority of the patient

were in the second decade with a minimum age of 12yrs and maximum age of 53yrs .

The majority of patients in the chordal sparing group were in the third decade with a minimum age of 14yrs and maximum age of 52yrs. 12 patients were over 40 years in he the classical MVR group and 7 were over 40 years in the chordal sparing group.. In both the groups patients over 40 years under went coronary angiography. 2 patients one male and one female had significant CAD which required added CABG procedure.

Table no 2 Sex distribution

Of the 105 patients 60 (57.1%) were males and 45 were females. In the chordal sparing group the males were 30(60%) and females were 20(40%) the distribution in the classical group was 25 (46%)females and 30(54%)males.

Table no 3. Table no 3 Symptoms

Majority of the patients were in

NYHA class II in both the groups, 43(78%) in the classical group and 40(80%) in chordal sparing group. 12 patients were in class III in the classical group and 10 in the chordal sparing group were in class III.no patients were in class I of classII.

Table no 4. Associated co morbidities

Tricuspid regurgitation was the dominant co morbid condition in both the groups 16 of 55 (29%)patients in the classical group. of these 5 patients under went associated de Vegas procedure. 12 of 50 (24%) in the chordal sparing group had associated tricuspid regurgitation 3 patients had associated de Vegas tricuspid annuloplasty done. Coronary artery disease requiring concomitant coronary artery bypass grafting was noted in 2 patients both in the classical group. Evidence of healed endocarditis was noted in 2 patients

in the form of perforated cusps.

Table no 5. Ejection fraction

Ejection fraction was in the high normal range in both the groups ejection fraction of less than 50 % was noted in 7(12%) patients in classic MVR group and 4(8%) patients in the chordal sparing group

Table no 6. LV dimensions

LV end systolic dimension and LV end diastolic dimensions were measured preoperatively. LVeSD greater than 50 mm was noted in 4(7.2%) of the 55 patients in the classical MVR group. The same was noted in 2(4%) of the 50 patients in the chordal sparing group. LVeDD of greater than 70 mm was noted in 8(14%) of classical MVR group and 5(10%) no of the chordal sparing MVR group.

Table no 7. Pre operative Atrial Fibrillation Atrial

fibrillation was present in 15(27%) of the 55 patients in the classical group and 18(36%) of the 50 patients in the chordal sparing group.

Postoperative period

Table no 8 type of surgery

50 patients underwent chordal sparing surgery of this in 10(20%) patients both anterior and posterior chordae were preserved while in 40 only the posterior leaflet was preserved

In 55 patients classical MVR was done.

Table no 9.Ventilator support

9 (16%) patients in the classical mvr group needed more than 36 hrs of ventilator while 2 (4%) patients in the chordal sparing group (PML preservation) needed more than 36 hrs of ventilator support. Majority of patients in both the group were weaned from ventilator

within 24 hrs

Table no 10. Morbidity and Mortality

10 patients were rexplored for excessive drainage through the intercostals tubes. The other complications noted were postoperative fever wound infection. Jaundice was noted in 5 patients which necessitated change of anticoagulation strategies.

Mortality

A total of 11(10.4%) deaths were noted in the entire group of this 6(10.9%) deaths were in the classical MVR group and 5(10%) were in the chordal sparing group. The deaths in the chordal sparing group were in the posterior leaflet preservation group. The deaths were due to postoperative low cardiac output syndrome necessitating high doses of inotropic support, prolonged ventilation, and onset of multi organ failure.

Table no11. Inotropic support

7(12.7%) patients in the classical MVR group required 3 inotropic drugs in the form of dopamine dobutamine and adrenaline while in the chordal sparing group (4%)patients with PML preservation alone needed 3 inotropic supports.

Table no 12. Pre discharge echo

There was a reduction in the LVeSD in 7 of 10patients with total retention of chordae .the LVeDD remained same in 7 patients with PML preservation alone .There was an increase in both the LVeSD and LVeDD in 9 patients with classical MVR.

The post operative ejection fraction increased in the total chordal sparing MVR

A reduction in post operative LV ejection fraction was noted in n of patients in whom the classical MVR was done.

Discussion

The majority of the patients in this group were in their second decade of life this is in contrast to the age group usually observed in the western population where the majority of the population is in the 5th and 6th decade. In the western population the mitral valve prolapse or ischemic mitral regurgitation predominates whereas in Indian population rheumatic heart disease predominates. Kirklin describes an accelerated form of RHD MR occurs in certain geographical areas where severe MS also is noticed in a younger age group. The interval before appearance of symptomatic MR is also shortened than for stenosis with a higher chance for previous severe attack of RF.[51]

The majority of the patients in this group were males. The major etiology of regurgitation was rheumatic heart disease. Ling et al in their series of chronic MR noticed

70% of their study group to be males .The majority of cause of regurgitation in their group was a flail leaflet due to ruptured chordae.[48]

Delahaye et al in their study of MR incidence and pathology noted males to be affected more (70%) their group had more of MVP (70%) as the cause of mitral regurgitation.[46]

The majority of patients in this group were in NYHA class II. The next major group was in class III . This was noticed for both the chordal sparing and classical MVR group. No patients were in NYHA class IV or class I. This is of particular significance because symptom severity is one of the preoperative predictor of post operative left ventricular function.[18]

Grigioni et al in their study of 109 patients with mitral regurgitation noted that the majority of patients in their

series were in NYHA class III.

Rosen et al in their study of 31 patients with severe MR due to MVP were in class III state.[32]

Tricuspid regurgitation was found to occur most commonly in both the groups of patients.

Important Tricuspid valve regurgitation is a constant phase risk predictor for death after mitral valve replacement surgery.[49]

Coexisting coronary artery disease is also an important predictor of post operative out come in the long term.

The ejection fraction preoperatively in majority of the patients in this study was in the normal or high normal range. Enriques Sarano et al reported preoperative ejection fraction as a predictor of late out come after mitral valve surgery. Though ejection fraction is considered to be load dependent it is of considerable significance in post operative predictor of out come

following MVR. In their work on echo cardio graphic prediction of LV function Zile MR and Fleming et al report an ejection fraction of lesser than 50% as a cut off point in mitral regurgitation wherein left ventricular systolic dysfunction is said to set in.

The mean left ventricular end systolic dimension in this study was 32 mm and ranged from 28 to54 mm. Zile MR and Fleming et al report Larger LVeSD greater than 50mm or left ventricular end systolic volume index of greater than 50ml/sq m has greater predictor value among all the echo indices for left ventricular dysfunction post mitral valve replacement [26]. Alternative proposed measures include end systolic wall stress index, an end diastolic dimension greater than 70mm, increased left atrial size. Crawford et al in their operative report on 48 patients with chronic MR noted that a low ejection fraction or higher LVeSD was an important predictor of poor late post operative out

come.[34]

A total of 33 patients in both the group were in atrial fibrillation with higher no in the chordal sparing group. Preoperative atrial arrhythmias have a bearing on the out come in the early phase after surgery.[33]

Type of surgery for mitral regurgitation has a bearing on both the early and late out come after mitral valve surgery.

Akins et al indicate that mvr is a risk factor for late mortality in comparison with mitral valve repair in univariate analysis, Gilinov et al found by both univariate and multivariate analysis that mitral valve replacement was a risk factor, in comparison to repair.[24, 29]

Higher inotropic supports were needed in 2 of the chordal preserving mvr and 7 of the classical mvr patients they also had to be supported on ventilator for a higher period of time (>36 hrs)

Of significance in the complete chordal preservation group no one needed higher supports of prolonged ventilation. The overall mortality in this series was 10.4% with a mortality of 10.9%in the classical group and 10% in the chordal sparing group. The mortality rate was nil in the total chordal preserving group.

In the subgroup requiring these measures the mortality was predictably higher with the cause of death being low cardiac out put state with multi organ failure. Enriquez Sarano et al in their report from Mayo Clinic report a surgical mortality of 2.6% for valve repair and 10.3% for replacement with a late survival of 58% at 10 years following MVR.[31]

After valve replacement for chronic mitral regurgitation ejection fraction is lower than preoperatively in most patients

Patients undergoing repair have better postoperative ejection indices than similarly matched patients with

valve replacement,

In the complete chordal preservation group both the LV end diastolic and end systolic dimensions returned to normal or decreased further.

LV contractility remains normal or normalizes. Lv hypertrophy did not reduce appreciably in mitral valve replacement in comparison to the valve repair group. [48, 50]

Summary

Three hundred and forty three cases of mitral valve replacements were carried out between July 2003 to Aug 2005. Of these 105 patients had dominant mitral valve regurgitation. Most of the patients operated were in NYHA class II. The second peak was in class III most of these patients affected were in the second decade with males outnumbering females.

Tricuspid regurgitation was found to be the most common associated pathology in this group needing an associated tricuspid deVegas annulolplasty. Significant coronary artery disease requiring CABG was noted in two patients one male and one female both under going classical MVR because of calcific valve in one and evidence of healed endocarditis in one.

Ejection fraction preoperatively was normal in majority of patients with both classical MVR and chordal sparring MVR.

Left ventricular end systolic dimension greater than 50mm was noted in 4 of the classical MVR and of 2 in the chordal sparring group.

35 patients had preoperative atrial fibrillation.

Of the 50 patients with chordal sparring MVR10 had total preservation of the subvalvar apparatus. in the total chordal sparing mitral valve replacement group the need for prolonged postoperative ventilator support and higher inotropic supports was not seen. In the classical MVR group19 patients required prolonged ventilation support of greater than 24 hours.

There were 11 deaths in this series, 5 in the chordal sparing and 6 in the classical group. Of note there were no deaths in the total chordal sparing group. Postoperative echo showed a significant improvement in the ejection fraction and decrease in end systolic dimension in the total chordal sparing mvr group. There was no left ventricular out flow tract obstruction in the total chordal sparing MVR group.

Conclusion

Of the 50 chordal sparing MVR group either in the complete or partial form better hemodynamics were noted both in the immediate and early post operative period .

Chordal sparing MVR has been established to protect the left ventricular systolic and diastolic function in the late postoperative period.

A well designed prospective trial with a larger group of patients and a longer follow up period is needed to evaluate further this technique.

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