Surgical Intervention for Moderate Ischemic Mitral Regurge During Coronary Revascularization (Comparative Study)

Wahid Mohamed Osman, Al-Husseiny Al-Husseiny Gamil, Mohamed Abdelaziz Shaarawy, Saleh Raslan Hussein and Gamil Karam Mohamed

Cardiothoracic surgery, Faculty of Medicine, Al-Azhar University.
s_eldayem@yahoo.com

Abstract: Background: Ischemic mitral regurgitation is a common clinical problem of coronary artery disease (CAD), developing acutely after myocardial infarction in up to 19 % of patients. For patients with ischemic mitral regurgitation (IMR), it is not clear whether adjunctive mitral valve (MV) repair at the time of coronary artery bypass graft surgery (CABG) is beneficial. **Objective:** To compare the outcome of patients with moderate ischemic mitral regurgitation , those surgically corrected at time of coronary artery bypass grafting either by repair or replacement, versus those treated by coronary artery bypass grafting alone. **Patients and methods:** Between October 2009 and October 2011, a cohort of sixty patients with ischemic heart disease associated with moderate ischemic mitral regurgitation. The study was carried out in the department of cardiothoracic surgery of Al Azhar University and Nasr Institute. All patients had CAD with moderate IMR and were admitted for CABG combined with mitral repair versus CABG alone. **Results:** After our study evaluation, we found that patients who were offered the combined approach of CABG with repair, showed more improvement as regard to clinical and echocardiographic parameters, compared to those who were offered the CABG alone procedure. We also found that a worse preoperative left ventricle (LV) function is a risk factor to the persistence or progression of the IMR grades in the CABG only patients. **Conclusion:** We concluded that the MV repair procedure done in our first group patients offered some protection against the persistence or progression of the IMR grades.

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Key Words*:* Coronary artery bypass graft (CABG), ischemic mitral regurgitation (IMR).

1. Introduction

Ischemic mitral regurgitation was defined as mitral valve regurgitation caused by coronary artery disease ***(Gillinov et al., 2001).*** It is a common complication of coronary artery disease and may develop in the acute or chronic phase. In chronic IMR, mitral valve leaks but the leaflets and subvalvular apparatus appear normal. Chronic IMR is therefore not a disease of the valve per se, but rather a disease of the left ventricle ***(Agricola et al., 2008).***

Even with a preserved ejection fraction (EF), chronic moderate-severe mitral regurge (MR) results in a significant reduction in intrinsic contractile function and reserve. Functional impairment was load independent reflecting a predominant defect in calcium cycling rather than impaired peak force generating capacity due to myofibrillar attenuation defects in isolated myocyte ***(McGinley et al., 2007).***

Ischemic mitral regurgitation varies in severity and is judged by echocardiography on a four graded scale ***(Enriquez-Saranom et al., 1993).*** The management of ischemic mitral regurgitation remains controversial. Patients with significant mitral regurgitation in the setting of coronary artery disease have a dismal long-term prognosis whether treated medically or surgically ***(Hvass et al., 2003).***

There is general agreement that patients with severe ischemic mitral regurgitation should undergo mitral valve surgery at the time of coronary artery bypass grafting. However, the importance of moderate ischemic mitral regurgitation in such patients is controversial ***(Miller, 2000).*** The late survival of patients with ischemic mitral regurgitation undergoing coronary revascularization remains poor; however, intervention on the mitral valve appears to benefit those with symptomatic heart failure ***(Borger et al., 2006).***

With improvements in perioperative management and myocardial protection as well as refinement of simplified techniques for repairing the incompetent mitral valve in the setting of ischemic disease, the incremental risk is likely less today than previously ***(Acker et al., 2006)***. So the objective of our study was to compare the outcome of patients with moderate ischemic mitral regurgitation, those surgically corrected at time of CABG either by repair or replacement, versus those treated by CABG alone.

2. Patient and Methods

Sixty patients with moderate ischemic mitral regurgitation admitted to cardiothoracic surgery department in Al-Azhar University Hospitals and Nasr Institute, Between October 2009 and October 2011, were enrolled in this study. This study was done after obtaining approval from the local institutional review board and human subject's protection. Informed consent was obtained from all patients. This study was a cross sectional prospective observational study; Patients were divided into two randomized groups;

- Group (I): comprising 30 patients managed surgically at time of CABG either by MV repair or replacement.
- Group (II): comprising 30 patients managed by CABG alone.

Any patient with moderate ischemic mitral regurge that may has a history of congestive heart failure, left ventricular dysfunction (ejection fraction < 50%), evidence of left ventricular scarring tissue with no structural mitral abnormality was included in this study. While any patient with rheumatic or infective mitral regurge, degenerative mitral regurge or ruptured cordae tendinae was excluded.

Pre-, intra- and postoperative data were collected for each patient in both groups. All survivors were subjected to three and six-month follow-up assessment.

(A) Preoperative assessment: All patients were subjected to the following: Full history taking, complete systemic examination, complete cardiac examination and Investigations included: Laboratory, X-ray, ECG, echocardiography and coronary angiography.

(B) Operative procedure:

After decision of surgery had been established, and the patients were prepared for surgery, all patients were managed according to the following lines in the operating room. Premedication therapy with midazolam and meperidine was given for all patients at the night of operation and in the morning. ECG electrodes were placed for monitoring. Peripheral and central venous lines and arterial line (usually right radial) and urinary bladder catheter were inserted. Temperature was monitored by a nasopharyngeal probe. Basal activated coagulation time (ACT) and arterial blood gases (ABG) were obtained.

Anesthesia;

All patients were operated under general anesthesia. Induction of anesthesia was obtained with Fentanyl and Sodium Thiopental, maintenance was obtained either with Fentanyl or Propofol and muscle relaxation was obtained with Pancromium Bromide or Atricrium Besylate. Prophylactic antibiotics (third generation cephalosporin) were given with induction of anesthesia and during cardiopulmonary bypass (CPB).

Surgical Procedures:

In all cases, the surgical approach used was a median sternotomy and Cardiopulmonary bypass was instituted. The conduits left internal mammary artery (LIMA), radial artery and/or saphenous vein were then prepared according to the grafts needed and planned for the patient, and then the pericardium was opened and plicated. Heparin was given IV (3mg/kg, with ACT > 400). CPB was started with aortic cannula, two venous cannula in group 1 or two-stage single right atrial cannula in group 2 and cardioplegic cannula were inserted in the usual manner. Cardiopulmonary bypass with nonpulsatile perfusion flow (2.2–2.4 L/min/m^2) was established. After cooling to 28-30 ^oC, the aorta was cross clamped.

Cold crystalloid solution at 4 ^oC was injected in cardioplegic cannula at root of the aorta under pressure. The first dose consists of 15-20 ml/kg, followed by 10 ml/kg every 30 minutes. The cardioplegic solution is a hyperkalaemic buffered crystalloid solution. Topical cooling was done by using slush solutions or ice. Distal ends of conduits were anastomosed to target vessels in two groups and then mitral valve repair in group 1. Rewarming started then declamping the aorta after LIMA anastomoses. The proximal ends were anastomosed to the aorta with partial clamping the aorta.

We used the conventional inferior approach incising the left atrium posterior to the interatrial groove for exposure of the mitral valve. In 4 patients we used transseptal approach.

Examination of the valve and valve analysis

Once exposure is obtained, inspection of the valve is carried out .valve analysis takes a few minutes utilizing nerve hooks, forceps, and insufflations of the ventricle with saline to determine and corroborate the pathology already diagnosed by preoperative trance thoracic echo (TTE). An initial inspection reveals the amount of annular dilatation. Traction sutures in the annulus at each commissure to elevate the valve and facilitate exposure of the leaflets, chorda tendinae and papillary muscles. Most often the entire valve appears normal apart from dilatation of the mitral annulus mostly elongated posterior mural annulus.

Repair techniques

(1) Ring annuloplasty: Sizing of the annuloplasty ring can be performed before or after placement of the annuloplasty sutures depending on the size of the left atrium and adequate exposure. When choosing an annuloplasty ring, the intertrigonal distance and the surface area of the anterior leaflet are measured.

After the size of the ring is chosen, the annuloplasty sutures are placed in accordance with the geometry of the mitral annulus. Once all sutures are placed, the annuloplasty ring is slipped down onto the mitral valve annulus, and the sutures are tied to secure the ring.

(2) Papillary muscle plication (Sandwich plasty): Sandwich plasty consists of 2 procedures. The first procedure is the papillary muscle head approximations of the anterior and posterior mitral valve leaflets to achieve coaptation of the 2 leaflets. At the anterolateral commissural portion, a Teflon-pledgeted 3-0 Ticron suture with a double-armed needle is passed through the papillary muscle head of the posterior leaflet and through the papillary muscle head of the anterior leaflet, reinforced with another Teflon patch. The same approximation suture is made at the posteromedial commissural portion. The second procedure is mitral annuloplasty with an exactly sized Carpenter–Edwards Physio-ring as mentioned before.

(3) The edge-to-edge repair (Alfeiry technique): The basic concept of this technique is that mitral regurgitation can be corrected simply by suturing the free edge of the diseased leaflet to the corresponding edge of the opposing leaflet exactly where the regurgitant jet is located. When the jet of the regurgitation is in the central part of the mitral valve, the application of the edge-to-edge technique produces a double orifice valve configuration (double orifice repair). On the other hand, when the mitral valve lesion is localized in proximity of a commissure, its surgical correction by the edge-to-edge, results in a single orifice mitral valve with a relatively smaller area (paracommissural repair).

All our patients did paracommissural repair as the leaflets looked normal and the abnormality was dilated annulus.

A 4/0 polypropylene suture is then passed in a standardized manner along the free edge of the anterior and posterior leaflets. Pledgets are not necessary. After reconstruction, the residual mitral area can be measured, by introducing Hegar valve dilator in to the orifice, a global valve area of more than 2.5cm is usually considered acceptable. Final competence is evaluated by forceful saline filling of the left ventricle.

The left atriotomy was closed by a running 3/0 polypropylene suture. In cases where transseptal approach was used, the septum and right atriotomy were closed by running 4/0 polypropylene sutures. Deareating maneuveres were employed before removal of aortic cross clamp. Weaning from CPB was achieved with inotropic support in almost all the patients. Hemostasis was carefully performed. We used 2 mediastinal tubes for drainage. Left pleural tube was separately inserted for pleural drainage. The pericardium was routinely left opend, sternotomy was closed and muscles were closed in layers.

The following operative data were collected in all patients: grafts performed, operative times and types of mitral valve repair in group (1).

(C) Postoperative data included:

Using of inotropic support, intensive care unit (ICU) stay, duration of ventilator support, mortality and morbidity, period of hospital stay and echocardiography data.

Follow-up: All patients were subjected to clinical assessment and echocardiography at our patient clinic after 3 and 6 months.

Data analysis:

All data were analyzed using commercially available program (SPSS for Windows ver.10.0). Differences are presented with 95% CIs. Dichotomous data were compared by the chi-square χ^2 test. Means are presented with SD and were compared by a 2-sample t-test and percents were compared by t-test for percentage. Statistical significance was accepted at a $P \leq 0.05$.

3. Results

I. Preoperative data

Patients' characteristics are listed in table 1. In the preoperative data, there was no significant difference in the two groups according to age, sex ratio, risk factors, new York heart association (NYHA) class and echo data (except the left atrium (LA) dimensions).

II. Operative Data

In the operative data, there was significant difference between the two groups in total operative times and there is no statistical significance in Intraoperative details of coronary anastomosis (Table, 2).

III. Immediate Postoperative Data

In the postoperative data, there was no significant difference between the two groups (table 3).

Clinical Evaluation: Assessment of the patient's functional status (mean NYHA class- dyspnea) in the early 3 and 6 months follow-up period, showed statistically-significant results in both groups (Table, 5). The parameters that showed statistical significance versus their preoperative match were: the number of preoperative infarctions, LVEDD, LVESD, LVEF %, and no grafting of the right coronary system. Using Spearman's correlation-coefficient, a positive correlation was found between LVEDD, LVESD, the number of preoperative infarctions and the grade of MR after CABG. A negative correlation was found between the preoperative LVEF %, grafting the right coronary system and the grade of IMR after CABG Table (6).

Table (1): Preoperative characteristics of the two groups of patients.

Characteristics	Groups		P-value
	Group ICABG + MV Repair No=30	Group II CABG alone No=30	
Age (years) (Mean ± SD)	41.85 ± 5.95	44.65 ± 6.13	0.71
Sex (M/F)	18/12	19/11	0.75
Risk factors (No/%)			
Diabetes mellitus	15/50	16/53	0.806
Hypertension	18/60	20/66	0.222
Dyslipidemia	10/33	10/33	1.00
Smoking	23/76.6	25/83.3	0.631
Family history	13/43.3	9/30	0.607
Previous MI	20/66	24/80	0.345
NYHA class			
Class II (%)	13/43	16/53	0.322
Class III (%)	17/56	14/46	0.543
Echo (Mean ± SD)			
LVEDD (cm)	5.8±0.2	5.9±0.5	0.162
LVESD (cm)	4.3±0.41	4.46±1.01	0.213
LVEF (%)	45±1.7	47±0.8	0.431
LA diameter (cm)	4.9±0.6	4.2±0.11	<0.05
PASP (mmHg)	53±2.7	51±2.4	0.742
Jet Area (cm^2)	6.8±0.5	6.9±0.8	0.331
MR Echo-grade	2+	2+	0.22

MI: myocardial infarction, LVEDD: left ventricle end diastolic dimension, LVESD: left ventricle end systolic dimension, LVEF: left ventricle ejection fraction, LA: left atrium, PASP: pulmonary artery systolic pressure, MR: mitral regurge.

Table (2): Operative data in the study groups

Variable	Group I (n=30) CABG + MV Repair	Group II (n=30) CABG alone	P value
Operative times			
CPB (min)			
- Range	99 - 143	86-115	
- Mean ± SD	122 ± 1.9	90 ± 2.2	< 0.04
ACC (min)			
- Range	63 - 92	34 - 75	
- Mean ± SD	68 ± 8.5	44 ± 11.41	< 0.02
TOT (min)			
- Range	198-270	162-210	
- Mean ± SD	220 ± 21.3	180 ± 3.5	< 0.05
Surgical Procedure			
RCA or PDA graft	12 (40 %)	9 (30 %)	0.154
LIMA-LAD	30 (100%)	30 (100%)	0.324
Mean anastomotic points	3.32 ± 0.51	3.41 ± 0.62	0.452
Type(s) of Mitral repair			
Ring Annuloplasty	11 (37 %)	None	-
Alfieri Stitch Repair Only	15 (50 %)	None	-
RA + Papillary Muscle Plication	4 (13 %)	None	-
Mitral Replacement	None	None	-

CPB: cardiopulmonary bypass time, ACC: aortic cross clamp time , TOT: total operative time , RCA: right coronary artery, PDA: posterior descending artery, LIMA: left internal mammary artery, LAD: left anterior descending artery.

Table (3): Immediate Postoperative Data

Variable	Group I (n=30)	Group II (n=30)	P Value
Immediate postoperative data Mean ± SD			
Mechanical Ventilation (hrs)	13 ± 2.3	16.2 ± 3.5	0.17
Inotropic Support (hrs)	22 ± 3.8	26 ± 5.2	0.67
ICU stay (hrs)	43 ± 1.9	45 ± 2.1	0.24
Hospital Stay (days)	7.88 ± 3.3	8.43 ± 3.1	0.541
Mortality and Morbidity Complications			
Mortality	0	0	
Re-exploration for Bleeding	3 (10%)	1 (3 %)	0.443
Low Cardiac Output	2 (6 %)	3 (1 %)	0.412
Respiratory Tract Infection	4 (12 %)	3 (10 %)	0.331
Renal Insufficiency	0	2 (6 %)	0.451
Sternal wound Infection	2 (6 %)	1 (3 %)	0.565

Immediate Postoperative echocardiographic data: showed little differences with no statistical-significance but Evaluation of early 3 and 6 months postoperative echocardiographic data: showed statistical-significance differences (Table, 4).

Table (4): Postoperative Echocardiographic Data.

Echo (Mean ± SD)	Groups		P-value
	Group I CABG + MV Repair No=30	Group II CABG alone No=30	
Immediate			
LVEDD (cm)	5.5+0.1	5.8+0.14	0.754
LVESD (cm)	4.11+0.1	4.21+0.4	0.542
LVEF (%)	46+2.3	44+2.1	0.761
LA diameter (cm)	4.2+0.03	3.8+0.2	0.267
PASP (mmHg)	50+1.3	51+1.2	0.641
Jet Area (cm^2)	3.8+0.2	4+0.4	0.752
MR Echo-grade	+1	+2	0.340
Early (3 months)			
LVEDD (cm)	4.5+0.61	5.5+0.23	<0.05<0.04<0.05<0.03<0.02<0.05<0.05
LVESD (cm)	3.5+0.2	4+0.42	
LVEF (%)	52+1.2	48.7+0.35	
LA diameter (cm)	4+0.1	4+0.32	
PASP (mmHg)	41+3.5	49+1.2	
Jet Area (cm^2)	3.4+0.14	3.7+0.7	
MR Echo-grade	1+	+2	
Late (6 months)			
LVEDD (cm)	4+0.5	5+0.49	<0.05<0.04<0.05<0.04<0.05<0.05<0.04
LVESD (cm)	3+0.2	3.6+0.41	
LVEF (%)	56+2.2	51+2.6	
LA diameter (cm)	3+0.6	3.7+0.3	
PASP (mmHg)	40+2.4	47+1.3	
Jet Area (cm^2)	3.2+0.1	3.4+0.2	
MR Echo-grade	+1	+2	

LVEDD: left ventricle end diastolic dimension, LVESD: left ventricle end systolic dimension, LVEF: left ventricle ejection fraction, LA: left atrium, PASP: pulmonary artery systolic pressure, MR: mitral regurge.

Table (5): Postoperative NYHA Class Assessment

NYHA class	Group I	Group II	P value
Early 3 months			
No or I	21 (70 %)	18 (60 %)	< 0.05
II	9 (30 %)	6 (20 %)	< 0.04
III	None	6 (20 %)	< 0.02
Mean NYHA	2 ± 0.2	2.1 ± 0.7	< 0.03
Late 6 months			
No or I	25 (83 %)	21 (70 %)	< 0.04
II	4 (13 %)	5 (16 %)	< 0.05
III	1 (3 %)	4 (13 %)	< 0.03
Mean NYHA	1.5 ± 0.3	1.6 ± 0.5	< 0.05

Table (6): Correlation between IMR follow-up and different Variables

	R	P	Significance
> 1 MI	0.504	0.01	< 0.01
Low LVEF%	0.606	0.01	< 0.01
LVEDD	0.730	0.01	< 0.01
LVESD	0.419	0.01	< 0.01
No PDA grafting	0.475	0.01	< 0.01

MI: myocardial infarction, LVEF: left ventricle ejection fraction, LVEDD: left ventricle end diastolic dimension, LVESD: left ventricle end systolic dimension, PDA: posterior descending artery.

4. Discussion

Ischemic mitral regurgitation is a relatively common manifestation of coronary artery disease and occurs after acute myocardial infarction or as a chronic condition; the management of ischemic mitral regurgitation represents a therapeutic challenge ***(Kim et al., 2005).***

In addition, Ischemic MR is not a homogenous entity; patients may require different therapeutically strategies which make the interpretation of reports about ischemic MR as one entity difficult or even impossible ***(Ryden et al., 2001).***

Once IMR is initiated, end-diastolic LV volume and wall stress increase in consequence to the increased preload. Left ventricular mass also increases progressively without a concomitant increase in end-diastolic wall thickness, resulting in generalized loss of myocardial contractile function. Increased wall stress causes more LV dysfunction, which in turn results in further PM displacement and leaflet tenting. As LV dilation occurs, annular dilatation occur leading to valvular dysfunction thereby augmenting valvular incompetence. Chronic IMR usually induces more MR in a self-repeating fashion ***(Borger et al., 2006).***

In the literature, there is a general consensus among cardiac surgeons that patients with severe (echo-grade 3+ or 4+) ischemic mitral regurgitation must be submitted to mitral valve surgery concomitant with coronary artery bypass graft surgery ***(Enriquez-Sarano et al., 2005).*** On the other hand, the urgency of offering the surgical solution to patients of moderate (grade 2+) Ischemic MR is still debated ***(Gorman and Gorman, 2003).***

Because the morbidity and operative mortality associated with combined revascularization and mitral valve replacement are high and long-term survival is poor, some authors have advocated revascularization alone, ***(Arcidi et al., 1988 and Duarte et al., 1999).*** Whereas others have recommended revascularization combined with mitral valve repair ***(Czer et al., 1996 and Aklog et al., 2001).***

The debate over the prudence of adding a mitral valve procedure to coronary bypass had centered on the incremental increase in operative risk imposed by a more complex procedure. With more recent improvements in perioperative management and myocardial protection as well as refinement of simplified techniques for repairing the incompetent mitral valve in the setting of ischemic disease, this incremental risk is likely less today than previously ***(Tolis, 2002).*** The surgical options vary starting by different techniques of valve repair up to valve replacement ***(Enriquez-Sarano et al., 2005).***

In this thesis, we comparatively-studied the course of un-repaired moderate mitral regurgitation after CABG alone, versus CABG with repair, in order to assess the impact of un-repaired moderate ischemic mitral regurgitation on the immediate and early outcome of CAD patients undergoing standard CABG using CPB. The final goal was to detect and identify the factors which could predict the progress

or regression of moderate IMR after the CABG surgical procedure alone.

In our study both groups were homogenous regarding the demographic data with no statistical significant difference in age, gender, risk factors for ischemic heart disease. . Other studies which included similar patient cohorts are like ***Enriquez-Sarano et al, (2005), Harris et al, (2002), Tolis (2002), Calafiore et al., (2004), Lam et al., (2005), and Campwala et al., (2006).***

Regarding NYHA CLASS, majority of patients in our study were in NYHA class II-III with overall mean NYHA class of 2.3 in group I and mean NYHA of 2.11 in group II. The "earlier intervention" concept was agreed upon by other groups and hence similar mean values of preoperative NYHA class (between II-III) was reported in the series by other surgeons like ***Lam et al (2005),*** who reported mean NYHA of 2.4 , ***Tolis (2002) and Campwala et al (2006)***, who had mean NYHA class of 3.3.

Regarding previous MI in group (I) (66%) had previous MI and (80%) in group (II) with no statistical significant difference.

This percentage of patients who had positive history of previous MI was comparable to other studies by ***Wong et al., (2005), Harris et al., (2002), Calafiore et al., (2004) and Paparella et al., (2003)*** where the percentages were (79.3%), (80%), (61.7) and (77.8%) of patients respectively.

According to the site of MI was that, in our patient sample, 70 % of patients had previous postero-inferior MI with a right-dominant coronary circulation (p = 0.003). This finding conforms well with and even explains statements presented by other authors saying that, although anterior MI are more common to occur in CAD, IMR is much more common after a postero-inferior MI ***(Kumanohoso et al., 2003)***. In agreement to this, ***Burch et al, (1999)*** stated that Ischemic MR usually occurs with right or circumflex coronary infarction that involves the posterior ventricular wall, posterior papillary muscle, and adjacent mitral annulus.

The preoperative Echocardiographic data including LVESD, LVEDD, mitral jet area, and EF showed no statistical significant difference. The mean for the EF was 45 ± 1.7and 47 ± 0.8 in Group I and Group II respectively. The mean EF for our study groups was high in comparison to other studies by ***Goland et al., (2009)*** are (37% ± 11% and 39% ± 11%), ***Harris et al., (2002)*** are (38% ± 13.8% and 38.7% ± 12.6), ***Kang et al., (2006)*** are (36 ± 11% and 36 ±11%), and ***Wong et al., (2005)*** are (39 ± 13.6% and 42.2 ± 15.3%) in Repair groups and CABG only groups respectively.

However, LA dimension was the one item of the preoperative Echocardiographic data which showed statistical significant difference between two groups, where mean LA dimension in repair group was higher than that of CABG only group.

In our study group, we found no correlation between the preoperative LA diameter and the presence of moderate MR (p = 0.235 = NS). Although unexpected, this can be explained by the fact that the left atrium is exposed to the LV filling pressures through the open mitral orifice during diastole, the size of which should therefore be influenced by the same factors that determine the diastolic filling pressure of the left ventricle ***(Desjardins et al., 1996).***

In ***2004, Beinart et al*** studied 395 patients with acute MI and found that increased LA volume, obtained within the first 48 hours of admission, was an independent predictor of 5-years mortality. A point of concern was that by the time of 48 hours from acute MI left ventricular adaptation to diastolic dysfunction could not have happened. Moreover, using LA volume index to represent LA size might influence comparing the results as others used LA diastolic dimension instead.

As expected, operative data analysis including total pump time and ischemic time revealed a significant difference when comparing the two groups. This significant difference in favour of group II was observed. It is the mitral valve procedure which adds this time. However, under sized ring annuloplasty without additional manoeuvres to repair the valve was sufficient to control the mitral regurgitation in 11 patients (37%); Alfieri annuloplasty stitch only in 15 (50 %); and ring annuloplasty combined with papillary muscles placation (tucking bases together) in 4 patients (13%). Mitral repair was not done in group II patients.

As regard to total pump time, group I showed a mean pump time of 122 ± 1.9 min, while it was 90 ± 2.2min in group II. When compared with other studies, results from ***Goland et al., (2009)*** and ***Harris et al., (2002)*** also showed statistically significant difference between their two groups, the first study reported a total pump time of 156±42 and 90±48 min for Repair group and CABG only group respectively; while the second study reported 203±68 and 134±52 min for corresponding groups respectively.

While for cross clamp time, Group I showed ischemic time of 68 ± 8.5 Min, and group II showed 44 ± 11.41min. In the same fashion, ***Goland and colleagues (2009)*** reported 114±30 and 60±36 min, and ***Harris and colleagues (2002)*** reported 123±69 and 63±27 min for their Repair group and CABG only group respectively.

In spite of increased length of operation in group I, the use of cardiac support whether pharmacological (Inotropic drugs) or mechanical (IABP) was not statistically different between the two groups.

In group I, 28 (93.3%) patients needed therapeutic dose of inotropic support and 2 (6.7%) needed high dose of inotropic support , while in group II, 24(80%) patients received inotropic support (therapeutic dose) and in only 6(20%) patients needed high therapeutic dose of inotropic support. Neither of both groups needed IABP.

Likewise, ***Wong and colleagues (2005)*** in their study of 251 patients having IMR including 31 patients who received concomitant mitral valve annuloplasty during CABG, had no statistical difference between their two groups in the use of high support with the baseline mean EF of their patient's slightly less than ours. And they had higher rates of IABP insertion, 6.4% and 2.3% in corresponding groups respectively. This difference might be attributed statistically to the large number of (n=251) patients and less LV functions included in their study.

Similarly, ***Ogus et al., (2004)*** in their study on 31 patients who had IMR and received CABG only they reported use of Inotropic support in 55% of patients and IABP insertion in 23% of patients. However, their group study had severely impaired LV function (mean EF 25±5%).

Analysis of post operative data regarding ventilation time, duration of inotropic support need and ICU stay showed no statistical significant difference between the two groups. ***Wong et al., (2005)*** reported a higher mean time of ventilation in patients of repair group (19.4% versus 8.6%) and statistically higher median hospital length of stay in the same group again that could be due to their large number of patients and lesser selection of cases than in our case and also in their study.

Postoperative complications in the form of reopening, renal insufficiency, low cardiac output and sternal wound infection, did not reach statistical significant difference between the two groups.

Hospital mortality rate in our study was none. Mortality for CABG alone has ranged from 8.8% to 21% compared with operative mortality of 2% to 9% for patients undergoing CABG plus mitral valve repair according to ***McGee et al., (2004)*** and ***Mallidi et al., (2004).*** This may be to the good selection of cases in our patient cohort with earlier performance of the surgical intervention while NYHA class is still not confirming presence of CHF. Our mortality-morbidity results were better than that reported by other authors like ***Aklog et al., (2001)*** (mortality of 2.9 % 140 patients); ***Grossi et al., (2001)*** (2.5 % in 223 patients); and ***Beinart et al., (2004)*** (4.4 % in 395 patients); and ***Crabtree et al., (2008)*** (10.1 % in 257 patients).

Furthermore, ***Harris and colleagues (2002)*** using univariate predictors of 30-days mortality found that preoperative NYHA classification, severity of MR, surgical procedure and cross clamp time were not significant predictors of early mortality, however, they found CPB time was a significant (P=0.002) independent predictor of early mortality. Although this was unexpected finding, as aortic cross clamp time is expected to be more strongly associated with poor outcome, the authors related this finding to the prolonged perfusion on bypass after removal of the cross clamp in cases in which the heart is struggling to get off pump and therefore CBP time is a marker for poor ventricular function and, therefore survival.

Even though, the previously mentioned studies found no relation between surgical procedure and early mortality, some authors postulated the possibility of CPB time being a surrogate for intervention on the mitral valve and hence, suggested performance of off-pump CABG before conduction of CPB and attacking mitral valve.

Now, before the outcome of these two strategies dealing with IMR is evaluated, it is important to first answer the simple question of whether CABG alone corrects the IMR in the short term?!

In our study, patients of both groups showed postoperative clinical improvement without either mortality or serious morbidity, with the improvement being more apparent in group I (CABG plus mitral repair) compared to group II (CABG alone). The combined techniques of mitral repair with CABG, needed longer operative time frames in group I i.e.: Total Operative time, Cardiopulmonary Bypass time and Aortic Occlusion time. However, no statistical significance was present upon comparing values like ease of weaning off-CPB (inotropic support, need for intra aortic ballone counter pulsation (IABCP); as well as postoperative parameters (mechanical ventilation, ICU and hospital stay); and perioperative mortality-morbidity. Our results were conforming to other series ***Tolis (2002), Calafiore et al., (2004), Campwala et al, (2006), Wong et al., (2005) and Kumanohoso et al., (2003).***

The combined approach (CABG+MR) improved NYHA class III dyspnea from preoperative 56 % of IMR group I to only 7 % of patients, with no or class I symptoms in 83 % of patients. Mean NYHA class decreased from preoperative mean of 3.64 to early PO of 2.6 and late PO of 1.8. The preoperative LVEF% of 46.7 % stepped up to 53.2 % and 58.2 % in the early and late PO periods

respectively. Moreover, postoperative IMR decreased to echo-Grade 1+, and jet area decreased from mean preoperative value of 7.3 cm2 to 3.54 and 3.3 cm2 over the early and late PO periods (respectively). On the other hand, CABG alone did also improve the clinical condition soundly in group II patients: dyspnea class III decreased from preoperative 46 % of IMR group II to only 20 %, with no or class I symptoms in 70 % of patients. Mean NYHA class improved from preoperative mean of 3.61 to early PO of 2.8 and late PO of 2.1. The preoperative LVEF% of 47.8 % stepped up to 50.3 % and 56.6 % in the early and late PO periods respectively. Postoperatively, IMR remained in Grade 2+, and mean jet area decreased from preoperative value of 7.7 cm2 to 4.4 and 4.2 over the early and late PO periods (respectively). It is worth-mentioning that the differences between the two groups showed no statistical significance in the immediate PO period and significance during the early and late PO periods. This may be explained by the immediate postoperative edema of the cardiac muscle with interstitial spaces still loaded with fluids hence interfering with its proper contractility.

It's uncertain whether the poorer outcomes in this group are dependent on the valvular dysfunction or whether it's merely a surrogate marker of extensive comorbidities, particularly the amount of ventricular dysfunction ***(Wong et al., 2005).***

As was evident from the previous display of our study results, CABG alone did, to some extent, improve IMR over 6 months of follow-up. Although the overall postoperative clinical parameters demonstrated statistically-significant values in both groups, CABG combined with mitral repair achieved more improvement in the clinical follow-up parameters (EF %, Jet area and MR echo-grade and mean NYHA class) when compared to preoperative patient condition.

Some studies agree with our results and confirm the effectiveness of combining MV repair with CABG like ***Duarte et al., (1999),*** who reported more "step-up" improvement in the overall mean LVEF for CABG/repair versus CABG only from preoperative 39 % and 41 % to 54.5 % and 46 % respectively; ***Tolis (2002)*** 34 % and 31 % to 52.5 % and 43.2 %; ***Calafiore et al., (2004),*** from 27 % and 30 % to 57.1 % and 46 % ; and ***Kim et al., (2005),*** from 29 % and 33 % to 55 % and 43.5.

As regards improvement in the grade of mitral regurgitation, ***Duarte et al., (1999),*** found that moderate Ischemic MR improved to absent or mild in 42 % of the combined approach and 39 % in CABG alone to 76 % and 44 % respectively; ***Tolis (2002)*** from 33 % and 41 % to 82 % and 66 %; ***Calafiore et al., (2004),*** from 31 % and 39 % to 77 % and 51 %; and ***Kim et al., (2005),*** from 23 % and 36 % to 67 % and 54%.

Limitation of This Study

The most important limitations is the relatively-short period of postoperative follow-up, unavailable intraoperative trans-esophageal echocardiography and also the use of semi quantitative Doppler (jet area) to assess the degree of mitral regurgitation, Many studies advocate the use of quantitative Doppler (effective regurgitant orifice) to assess MR as this is less influenced by the loading condition and impaired function of the LV.

Conclusion

The current study concluded that, the presence of moderate (echo-grade 2+) ischemic MR in CAD patients scheduled for CABG surgery, does not increase perioperative morbidity or mortality. CABG with concomitant mitral repair is associated with better postoperative results as evident by the more favorable improvement in both patient's clinical symptomatology (NYHA Class) and echocardiographic parameters (MR echo-grade, LVEDD, LVESD, LVEF %, LA diameter, PASP, MR jet area) over the early (3 months) and late (6 months) postoperative follow-up periods. Moderate ischemic MR progresses to more severe grades in only a minority of patients (3%) in the early follow-up period following Combined CABG + MV repair compared to a higher incidence (13 %) in patients submitted to CABG only. And lastly, the higher number of preoperative infarctions, a larger LV size, lower ejection fraction, and failure to graft the right coronary artery territory can all lead to the persistence or progression of moderate ischemic MR following CABG operations.

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