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Right Ventricular Dysfunction after Coronary Artery Bypass Grafting Is a Reality of Unknown Cause and Significance

Muhammad-Mujtaba Ali Siddiqui, Anjum Jalal, Mubashir Sherwani, Muhammad Zubair Ahmad

CPE Institute of Cardiology, Multan, Pakistan

ABSTRACT

Objective: To evaluate the impact of coronary bypass surgery on the function of the right ventricle (RV) in patients with a pre-operative ejection fraction ≥ 35% who did not have any perioperative myocardial infarction.

Method: We performed a prospective study of 30 patients who underwent uneventful isolated coronary artery bypass grafting (CABG). All patients had echocardiography prior to surgery and 3 months postoperatively. Myocardial tissue Doppler velocities were used to measure left and right ventricular function. The right ventricular myocardial performance index (Tei) and the ratio between the velocities of the RV and left ventricle (LV) were also calculated.

Results: There was a significant improvement in left ventricular ejection fraction before and after CABG (P = .046). The tissue Doppler imaging (TDI) velocities from the LV remained unchanged, but highly significant reductions in right ventricular TDI velocities were observed (P <.001). The TDI peak systolic (S), early diastolic (E), and late diastolic (A) velocities had a reduction of 30%, 34.5%, and 20%, respectively. Similarly, a fall in RV to LV ratios of various TDI velocities was also observed. This was also accompanied by a significant rise in the RV Tei index. All of these findings are suggestive of significant RV dysfunction.

Conclusion: There is a marked impairment of RV function after CABG.

INTRODUCTION

Coronary artery bypass grafting (CABG) is a frequently performed procedure for the treatment of coronary artery disease. It is usually followed by improvement in symptoms and is proved to be a better treatment option than coronary stenting because of significantly better adjusted rates of long-term survival and freedom from re-intervention [Hannan 2005]. However, improvement of ventricular segmental wall motion has been variable [Righetti 1977]. There is evidence that the left ventricle (LV) diastolic function improves while left ventricular systolic function is preserved after CABG [Hedman 2005; Diller 2008]. By contrast, the systolic and diastolic function of the right ventricle (RV) appears to suffer from long lasting impairment. Diller et al observed that impaired RV function did not recover to preoperative levels over 18 months [Diller 2008]. Alam et al noted that there was a small improvement in tricuspid systolic velocities 1 year post-CABG, but they did not reach the preoperative values [Alam 2003]. On the contrary, the findings of Yadev et al have suggested that RV impairment after CABG is likely to be permanent [Yadev 2010].

In this study, we looked at the impact of CABG on RV function as well as the relative LV and RV function using tissue Doppler imaging (TDI), which is known to be a sensitive and effective method for assessing both LV and RV function [Alam 2000].

METHODS

This is a prospective study of 30 patients who underwent uneventful isolated CABG at the CPE Institute of Cardiology, Multan, Pakistan. The patients with conditions that might have resulted in erroneous evaluation of RV function or were likely to affect the RV function indirectly were excluded from the study. Therefore, patients who had significant LV dysfunction (ejection fraction <35%) and those who sustained perioperative myocardial infarction (MI) were excluded from the study. Each patient had an echocardiography within 1 month before the operation and then 90 days after the operation. The echocardiographies were done using conventional 2D, Pulse Wave (PW) Doppler and TDI techniques. The echo studies were all done by the same person, who is a certified cardiologist with sufficient experience in this field (M.S.). The echocardiographer remained completely blind to patients' clinical data. Myocardial tissue Doppler peak systolic (S), early diastolic (E), and late diastolic (A) velocities were measured in cm/sec with reference to the lateral and septal aspects of the mitral annular ring and the lateral angle of the tricuspid valve. The RV to LV ratio was calculated for each patient. This is the ratio of tricuspid annular velocity to the

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Correspondence: Anjum Jalal FCPS, FRCS-CTh, CPE, Institute of Cardiology, Abdali Road, Multan, Pakistan; +92 32 14891250; fax: +92 61 9201166 (e-mail: anjumjalal1@hotmail.com).

Table 1. Comparison of Preoperative and Postoperative Conventional Echocardiographic Characteristics by Paired Sample *t* Test (n = 30)*

Routine Echocardiography Variables	Preoperative	Postoperative	Mean Difference (MD)	Standard Deviation of MD	*P*
LV end diastolic dimension, mm	47 ± 4.4	47 ± 4.5	0.5	3.4	.42
LV end systolic dimension, mm	31 ± 7.0	33 ± 3.9	-2.2	6.6	.08
LV ejection fraction, %	52 ± 11	56 ± 5.4	-4.5	11.8	.05
Intraventricular septum in diastole, mm	10 ± 1	11 ± 1	-0.36	1.2	1.18
Left atrial area, mm^2	39 ± 3	38 ± 2	0.5	3.3	.41
Mitral E wave, cm/sec	81 ± 20	84 ± 18	-2.5	13	.30
Mitral A wave, cm/sec	76 ± 18	74 ± 17	2.6	13.5	.29
E:A ratio	1.0 ± 0.2	1.16 ± 0.33	-0.08	.25	.08

*LV indicates left ventricle; E, early diastolic; A, late diastolic

average of the septal and lateral mitral annular velocities. The isovolumetric relaxation time (IVRT), isovolumetric contraction time (IVCT), and ejection time (ET) were derived from TD tracings. The myocardial performance index (Tei) was calculated as the sum of IVRT and IVCT divided by ET (ie, Tei = [IVRT + IVCT] ÷ ET) [Gondi 2007]. The Tei is considered an independent measure of RV function.

The CABG operations were done by conventional method. Patients were given oral bromazepam (3 mg) the night before surgery. Anesthesia was induced with intravenous morphine (0.1 mg/kg), midazolam (0.05-0.1 mg/kg), and propofol (1.0-2.5 mg/kg). Atracuronium (1 mg/kg) was given as paralyzing agent before endotracheal intubation. The anesthesia was maintained with sevoflorane/isoflurane. The cardiopulmonary bypass (CPB) was established with aortic and 2-stage right atrial cannula. The CPB circuit was primed with crystalloid Ringer's solution. The heparin was administered in a dose of 300 U/kg. Systemic temperature was kept between 30°C and 32°C. The local cooling was done with ice-cold saline or local ice slush according to the preference of the operating surgeons. Cold blood cardioplegia was delivered through the ascending aorta and was repeated every 20 minutes. The initial dose of cardioplegia was 10-15 mL/kg, and the next dose was given as 5-7 mL/kg. Hemofiltration was used to maintain hematocrit between 20% and 27%.

Statistical analysis was performed using Excel (Microsoft, Redmond, WA, USA) and SPSS (Chicago, IL, USA). Paired sample *t* tests were applied to different variables. The mean

Table 2. Comparison of Preoperative and Postoperative Tissue Doppler Imaging (TDI) Characteristics by Paired Sample t Test (n = 30)

Myocardial TDI Velocity, cm/sec	Preoperative	Postoperative	Mean Difference (MD)	Standard Deviation of MD	*P*
Right ventricle					
Peak (S)	12.6 ± 2.5	8.7 ± 1.8	3.86	2.7	< .001
Early diastolic (E)	11 ± 2.4	8.83 ± 2.5	2.26	2.1	< .001
Late diastolic (A)	13.6 ± 3	8.9 ± 1.88	4.7	2.9	< .001
Left ventricle: septal					
Peak (S)	8 ± 2.2	7.8 ± 1.4	0.3	1.8	.39
Early diastolic (E)	8.2 ± 2	7.8 ± 1.5	0.36	1.6	.22
Late diastolic (A)	9 ± 2	8.5 ± 1.6	0.56	1.7	.08
Left ventricle: lateral					
Peak (S)	8.8 ± 2	8.1 ± 1.5	0.63	1.8	.06
Early diastolic (E)	10 ± 2	9.9 ± 2.6	0.2	2.4	.66
Late diastolic (A)	10 ± 1.8	9 ± 2	0.9	1.9	.01
RV:LV ratio					
Peak (S)	1.6 ± 0.37	1.14 ± 0.26	0.47	0.41	< .001
Early diastolic (E)	1.3 ± 0.3	1.1 ± 0.3	0.26	0.34	< .001
Late diastolic (A)	1.5 ± 0.42	1 ± 0.2	0.48	0.40	< .001
RV myocardial performance index (Tei)	0.43 ± 0.07	0.73 ± 0.1	-0.29	0.096	< .001

difference and the standard deviation of the mean difference were also calculated. A *P* value of .05 was considered significant.

The study had formal approval of Ethical Review Committee of the institute. Informed consent was given by all patients, and the study was conducted in strict compliance of the guidelines laid out in the Helsinki Convention.

RESULTS

The 30 patients included in the study consisted of 27 men and 3 women. The mean age was 55.8 years with a standard deviation of 8.76 years. Twenty-two patients suffered from Canadian Cardiovascular Society (CCS) Class III angina, and 8 patients had Class II angina. They received a total of 103 grafts (ie, 3.43 grafts per patient). These 103 grafts included 30 grafts to the left anterior descending arteries, 17 grafts to the diagonal arteries, 6 grafts to the ramus intermedius arteries, 23 grafts to the branches of the circumflex arteries, 23 grafts to the right coronary arteries, and 4 grafts to the posterior descending arteries. The left internal mammary artery (LIMA) was used in 25 patients (85%). No other arterial conduits were used. The mean cross-clamp time was 36 minutes (±12.35 minutes), and the mean cardiopulmonary bypass time was 48 minutes (±16.44 minutes). All patients were angina free after the surgery.

Table 1 shows that there was a significant improvement in LV ejection fraction before and after CABG ($P = .046$). There was no significant difference between TDI S velocities at the septal ($P = .39$) or lateral angle of mitral annulus ($P = .06$).

Table 2 shows that the TDI velocities from the LV remained unchanged. However, highly significant reductions in RV TDI velocities were observed. The TDI S velocity had 30% reduction ($P < .001$). The TDI A velocity had 34.5% reduction ($P < .001$), and the TDI E velocity had 20% reduction ($P < .001$). This was also associated with rise in RV Tei index from a mean of 0.43 to a mean of 0.73 ($P < .001$), which is suggestive of RV dysfunction.

A fall in RV to LV ratio following CABG surgery was also observed. There was 28% fall in the RV to LV ratio for TDI S velocity ($P < .001$). Similarly, the RV to LV ratio of TDI E velocity showed an 18.7% reduction ($P < .001$), and the RV to LV ratio of A velocity had a 31% reduction after CABG ($P < .001$).

DISCUSSION

A selective RV dysfunction after CABG was documented by Christakis et al nearly 25 years ago, and they attributed this to inadequate myocardial protection in cases of right coronary stenosis [Christakis 1985]. Similar observations have been made more recently [Alam 2003; Diller 2008; Yadev 2010], and the impairment of RV function seems to occur despite the adoption of newer techniques of myocardial preservation and avoidance of cardiopulmonary bypass [Pegg 2008].

It can be difficult to assess RV function by conventional 2D echocardiography. This is due to the anatomy of the RV and its complex motion during the cardiac cycle. Brooks et al have studied the use of a conductance catheter placed directly or indirectly through the RV for assessing RV contractility, but this is an invasive method and, as such, its use is limited to the perioperative period only [Brookes 1998].

By adjusting the pulsed-wave sample in line with the RV longitudinal excursion plane, TD can estimate RV long-axis function by assessing the low-frequency, high-amplitude motions of the tricuspid annulus against the stationary apex. This has been demonstrated to be an accurate correlate of radio nucleotide–determined RV ejection fraction [Meluzín 2001]. TD is also highly reproducible (5% inter-observer variation) and easily integrated into existing echocardiography protocols [Alam 1999].

TDI has been used for objective quantification of the RV function, and it has been noted that RV impairment occurs immediately after CABG. The clinical significance of depressed RV function following CABG has been assessed. Hedman et al found no relationship between RV dysfunction and improvement in exercise capacity 3 months following the CABG [Hedman 2004]. On the contrary, Meluzín et al have documented a very clear prognostic importance of RV dysfunction in non-operated patients of symptomatic cardiac failure. The patients with low tricuspid annulus acceleration, low RV systolic velocities, and low RV diastolic velocities were associated with increased risk of cardiac death or hospitalization. There was a 6-fold increase in the relative risk of hospitalization or cardiac death when all 3 features were present [Meluzín 2005]. It may be difficult to see the impact of RV dysfunction in post-CABG patients because the beneficial effects of revascularization on LV function may mask any adverse effects of RV dysfunction.

The cause for RV dysfunction after CABG is as yet poorly understood. Intuitively, one can consider local tissue damage due to direct exposure of RV free wall to the environment as a factor. Similarly, loss of pericardial restraint could be another possible cause. Other factors incriminated in this phenomenon include RV hypoperfusion during cold cardioplegic arrest [Christakis 1996], use of retrograde cardioplegia in severe diffuse disease of the right coronary artery (RCA) [Allen 1995], and pericardial adhesion formation [Wranne 1991]. However, the techniques of myocardial preservation do not seem to be responsible because RV dysfunction has been observed across the board in different strategies of myocardial management as well as off-pump surgery. Because it has been detected immediately after surgery, the development of pericardial adhesions does not seem be a causative factor. Moreover, prevention of tethering of RV free wall by using synthetic pericardial substitutes has failed to show any benefit [Lindström 2000]. We believe that RV dysfunction is a result of relatively less effective myocardial preservation due to several reasons including direct exposure of the RV to the atmosphere, more rapid increase in temperature, poor network of collaterals, and non-venting of venous blood returning to the RV. We are planning to repeat the study after developing a more aggressive approach to RV preservation.

In this study, we have observed a significant impairment of RV function following CABG. There was an absolute reduction in RV tissue Doppler velocity and also a reduction in the RV to LV tissue Doppler ratio, which indicated that this was not a manifestation of global myocardial injury. The fall in relative

RV velocities between preoperative and postoperative echocardiography was significantly large and consistent, suggesting a strong relationship between CABG and RV dysfunction.

CONCLUSION

There is a marked impairment of RV function after CABG.

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