



Significance of preoperative right ventricular function on mid-term outcomes after surgical ventricular restoration for ischemic cardiomyopathy

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Abstract

Objectives To analyze our surgical experiences with surgical ventricular restoration (SVR) for dilated ischemic cardiomyopathy (ICM) and to determine the significance of preoperative right ventricular (RV) function on outcomes.

Methods and results This study retrospectively analyzed 19 patients who underwent SVR between April 2010 and May 2016. Their mean age and New York Heart Association functional class were 62 ± 11 years and 2.9 ± 0.8 , respectively. The preoperative mean left ventricular (LV) end-systolic volume index and LV ejection fraction (LVEF) were 134 ± 56 mL/m² and $24 \pm 7\%$, respectively. The preoperative mean RV fractional area change (RVFAC) to quantify RV systolic function was $33 \pm 13\%$, as assessed by transthoracic echocardiography. The mean follow-up period was 47 ± 20 months. Three patients died of cardiac causes during the follow-up, with the 3-year and 5-year freedoms from cardiac-related death of 89% and 79%, respectively. Major adverse cardiac events (MACEs) occurred in ten patients, with the 3-year and 5-year MACE-free survival rates of 58% and 41%, respectively. RVFAC (risk ratio [RR]=0.92, 95% confidence interval [CI] 0.86–0.98, $p=0.01$) and LVEF (RR=0.83, 95% CI 0.68–0.97, $p=0.02$) were significant predictors of MACEs in the multivariate analysis. Patients with RVFAC of $<35\%$ had significantly poorer MACE-free survival rates (33% at 3 years) than those with RVFAC of $\geq 35\%$ (80% at 3 years).

Conclusion SVR for ICM provided acceptable freedom from cardiac-related death; however, MACEs commonly occurred and was associated with RV dysfunction.

Key words Surgical ventricular restoration · Ischemic cardiomyopathy · preoperative right ventricular function · RVFAC

Introduction

The impact of surgical ventricular restoration (SVR) on survival benefits is controversial as the Surgical Treatment for Ischemic Heart Failure (STICH) trial concluded that applying SVR to reduce left ventricular (LV) volume for

coronary artery bypass grafting (CABG) does not alleviate symptoms or improve exercise tolerance; moreover, it fails to reduce the death and cardiac rehospitalization rates [1]. Generally, a preoperative very large left ventricular (LV) volume and a retained large LV volume postoperatively with a small volume reduction rate are predictors of poor outcome after SVR for dilated ischemic cardiomyopathy (ICM). However, findings vary depending on the influence of preoperative LV volume on outcomes [2–7]. Moreover, providing an adequate LV systolic volume or LV end-systolic volume index (LVESVI) intraoperatively with an adequate volume reduction rate is difficult because the LV systolic volume is affected by the improvement of postoperative LV systolic function and preload and afterload, although LV diastolic volume may be regulated intraoperatively. Although LV ejection fraction (LVEF), New York Heart Association (NYHA) classification, LV shape, and mitral regurgitation

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(MR) are reported as predictors of poor outcome, they are also not conclusive. Thus, an alternative for assessing cardiac function and predicting outcomes after SVR accurately is necessary. Previously, we reported the significance of LV diastolic function on mid-term outcomes after SVR [8]. Right ventricular (RV) dysfunction is a well-known predictor in patients with heart failure. However, the influence of preoperative RV function on outcomes after SVR remains to be established. This study aimed to analyze our surgical experiences with SVR for ICM and to investigate the significance of preoperative RV function on outcomes.

Materials and methods

Patients

Between April 2010 and May 2016, 19 patients underwent elective SVR for ICM at Miyazaki Medical Association Hospital. The Institutional Review Board at Miyazaki Medical Association Hospital and Miyazaki University Hospital approved this retrospective study. The requirement for individual patient consent was waived because of the retrospective nature of the study. General informed consent was obtained from each patient preoperatively. SVR was indicated for ICM in patients with previous anterior wall myocardial infarction and severe hypokinesis of the left ventricle with an LVESVI of $> 80\text{--}100\text{ mL/m}^2$ and regional asynergy (either akinetic or dyskinetic without myocardial viability) exceeding 35% of the ventricular perimeter via left ventriculography and/or myocardial scintigraphy. To evaluate myocardial viability, electrocardiography, transthoracic echocardiography, left ventriculography, and myocardial scintigraphy were used; all patients underwent myocardial scintigraphy and 17 patients underwent left ventriculography. We did not use cardiac magnetic resonance imaging (MRI) to assess ventricle volume and function, scar extension, and myocardial fibrosis because this equipment was not available at Miyazaki Medical Association Hospital.

The optimal medical treatment using β -blockers, angiotensin-converting-enzyme inhibitors, diuretics, and aspirin was immediately re-instituted as soon as possible postoperatively.

Surgical procedures

Our operative technique has been previously described [8]. All patients were placed on an intra-aortic balloon pump (IABP) preoperatively. We performed a distal CABG anastomosis, first with an on-pump beating heart to minimize the aortic cross-clamp time, and then antegrade blood cardioplegic arrest was induced. After completing

mitral valve repair (MVR) and central CABG anastomosis, the left ventricle was incised and the exclusion line was determined. The balloon was filled at $50\text{--}60\text{ mL/m}^2$ to adjust the tightness of the Fontan stitch and/or patch size to prevent the excessive reduction of LV volume; the aortic cross-clamping was then released. Subsequently, patch closure was performed and the left ventriculotomy was closed under the on-pump beating-heart condition. The SVR techniques used in our study included the Dor [9] and septal anterior ventricular exclusion (SAVE) [10] procedures.

Statistical analysis

Results are expressed as means \pm standard deviations. Statistical analyses were performed using the *t* test or Wilcoxon signed-rank test for continuous variables and the Chi-square test for categorical variables. The correlation between RV fractional area change (RVFAC) and other preoperative echocardiographic parameters, preoperative LV volume, and LVEF was verified using Pearson's correlation test. The cumulative survival and freedom from major adverse cardiac events (MACEs) was calculated using the Kaplan–Meier method. MACEs were defined with cardiac-related death including sudden death or rehospitalization for heart failure requiring intravenous therapy. To predict the preoperative and surgical-related risk factors for MACEs, the preoperative LV end-diastolic volume index (LVEDVI), LVESVI, presence or absence of preoperative LVESVI of $\geq 94\text{ mL/m}^2$ [4], and LVEF, the transthoracic echocardiography (TTE) parameters, NYHA and operative procedures (Dor or SAVE procedure, presence or absence of concomitant surgical procedure with CABG or MVR) were included in the univariate Cox proportional hazards models. In a multivariate analysis, RVFAC, presence or absence of preoperative LVESVI of $\geq 94\text{ mL/m}^2$, and LVEF [11] were included in the Cox-proportional hazards models. Similarly, the postoperative LVEDVI, LVESVI, presence or absence of postoperative LVESVI of $\geq 70\text{ mL/m}^2$ [12], LVEF, and TTE parameters during follow-up were included in the univariate Cox proportional hazards models to predict the postoperative risk factors for MACEs. In a multivariate analysis, RVFAC, LV end-diastolic dimension (LVDd), and presence or absence of postoperative LVESVI of $\geq 70\text{ mL/m}^2$ were included in the Cox-proportional hazards models. Predicting the risk factors of cardiac-related death was not performed due to the small number of end-points.

Using a cut-off value of RVFAC $< 35\%$ vs. $\geq 35\%$ according to the 2010 Guidelines for the echocardiographic assessment of the right heart in adults [13], the cumulative survival and freedom from MACEs were

compared between groups using the Wilcoxon test. A value of $p < 0.05$ was considered statistically significant. All statistical analyses were performed using JMP version 13.2.1 (SAS Institute, Inc., Cary, NC, USA).

Results

Patient characteristics

Table 1 presents the preoperative characteristics. The mean patient age was 62 ± 11 (range 38–76) years. Seven patients were classified as NYHA class II (37%), 7 as class III (37%), and 5 as class IV (26%). According to the results of the left ventriculography or myocardial scintigraphy, the mean LVEDVI, LVESVI, and LVEF were 174 ± 74 (range 102–398) mL/m², 134 ± 56 (range 61–294) mL/m², and $24 \pm 7\%$ (range 14–41%), respectively. Three patients had an LVESVI of < 80 mL/m². However, they underwent SVR because one patient had an LV aneurysm with a giant thrombus and the other two had a low LVEF of 32% and 26%.

Preoperative transthoracic echocardiography parameters

Table 2 presents the preoperative TTE parameters. MR was classified as none (grade 0), mild (grade 1), moderate (grade 2), moderately severe (grade 3), or severe (grade 4) by “eyeballing” or by planimetry of the MR color flow jet area. We assessed RVFAC by tracing the RV endocardium both in systole and diastole from the annulus, along the free wall to the apex, and then back to the annulus, and along the interventricular septum to quantify RV function [13]. The early-to-late mitral valve flow ratio (E/A), the ratio between early inflow velocity and septal mitral annular early diastolic velocity

Table 2 Transthoracic echocardiogram parameters

Parameter	Preoperative	During follow-up	<i>p</i> value
LVDd (mm)	65 ± 6	60 ± 7	< 0.01
LVDs (mm)	55 ± 8	51 ± 8	< 0.01
LAD (mm)	46 ± 6	44 ± 7	0.2
LVEF (%)	30 ± 11	32 ± 10	0.4
MR grade	1.5 ± 1.0	0.8 ± 1.0	0.06
PAP (mmHg)	41 ± 17	–	
RVFAC (%)	33 ± 13	37 ± 14	0.2
E/A	2.4 ± 1.8	–	
E/e'	24 ± 11	–	
DT (ms)	153 ± 87	–	

LVDd left ventricular diastolic dimension, *LVDs* left ventricular systolic dimension, *LAD* left atrium dimension, *LVEF* left ventricular ejection fraction, *MR* mitral regurgitation, *PAP* pulmonary artery systolic pressure, *RVFAC* right ventricular fractional area change, *DT* deceleration time

(E/e'), and the deceleration time (DT) of early inflow velocity were also assessed to determine the LV diastolic property. The mean LVDd, LV end-systolic dimension (LVDs), left atrium dimension (LAD), LVEF, MR grade, and estimated pulmonary artery pressure (PAP) were 65 ± 6 mm, 55 ± 8 mm, 46 ± 6 mm, $30 \pm 11\%$, 1.5 ± 1.0 , and 41 ± 17 mmHg, respectively. The mean RVFAC, E/A , E/e' , and DT were $33 \pm 13\%$, 2.4 ± 1.8 , 24 ± 11 , and 153 ± 87 ms, respectively.

Correlation between RVFAC and preoperative transthoracic echocardiography parameters, preoperative LV volume index and LVEF

RVFAC was negatively correlated with preoperative PAP ($R^2 = 0.28$, $p = 0.04$), E/A ($R^2 = 0.34$, $p = 0.01$), and E/e'

Table 1 Preoperative characteristics

Variable	Total (n = 19)	No MACE (n = 9)	MACE (n = 10)	<i>p</i> value
Age (years)	62 ± 11	57 ± 15	61 ± 22	0.8
Male (n, %)	17 (89)	7 (78)	10 (100)	0.1
DM (n, %)	10 (53)	4 (44)	6 (60)	0.5
Hyperlipidemia (n, %)	18 (95)	8 (89)	10 (100)	0.3
Tobacco (n, %)	11 (58)	4 (45)	7 (70)	0.3
NYHA	2.9 ± 0.8	2.3 ± 0.5	3.0 ± 1.0	0.01
LVEDVI (mL/m ²)	174 ± 74	194 ± 94	164 ± 45	0.3
LVESVI (mL/m ²)	134 ± 56	143 ± 74	127 ± 32	0.5
LVESVI ≥ 94 mL/m ² (n, %)	15 (79)	6 (67)	9 (90)	0.2
LVEF (%)	24 ± 7	28 ± 7	21 ± 7	0.01
BNP (pg/mL)	650 ± 699	283 ± 241	762 ± 705	0.03

MACE major adverse cardiac event, *DM* diabetes mellitus, *NYHA* New York Heart Association classification, *LVEDVI* left ventricular end-diastolic volume index, *LVESVI* left ventricular end-systolic volume index, *LVEF* left ventricular ejection fraction, *BNP* B-type natriuretic peptide level

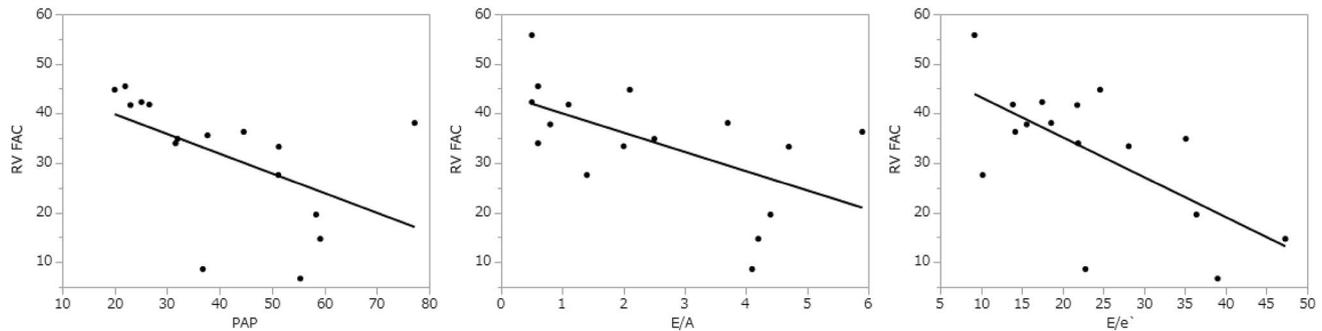


Fig. 1 Correlation between RVFAC and the preoperative transthoracic echocardiography parameters. RVFAC showed a negative correlation with preoperative PAP ($R^2=0.28$, $p=0.04$), E/A ($R^2=0.34$,

$p=0.01$), and E/e' ($R^2=0.42$, $p<0.01$). RVFAC right ventricular fractional area change, PAP estimated pulmonary artery pressure

($R^2=0.42$, $p<0.01$) (Fig. 1). However, it was not correlated with any other TTE parameter, preoperative LV volume index, or LVEF.

Operative data and postoperative LV volume changes

The Dor procedure was performed in 15 patients whose scar formation, targeted for surgical exclusion, was located mainly from the mid-anterior wall to the apex. Moreover, to restore the shape of the left ventricle, the SAVE procedure was performed in four patients whose scar formation, targeted for surgical exclusion, extended upward. Fourteen patients underwent CABG and received a mean of 3.4 ± 1.0 grafts. MVR was performed in 11 patients with mild (4), mild–moderate (4), moderate (2), and severe (1) MR.

Table 3 presents the postoperative LV volume changes. Postoperatively, LVEDVI and LVESVI decreased to 124 ± 47 (range 78–257) mL/m^2 and 83 ± 34 (range 39–161) mL/m^2 , respectively. In addition, LVEF improved to $34 \pm 9\%$ (range 20–50%). The LV end-diastolic and end-systolic volume reduction rates were 30 and 37%, respectively.

Table 3 Postoperative LV volume, LVEF, and volume reduction rate

Variable	Total ($n=19$)	No MACE ($n=9$)	MACE ($n=10$)	p value
LVEDVI (mL/m^2)	124 ± 47	130 ± 54	117 ± 31	0.6
Volume RR (%)	30 ± 14	33 ± 9	26 ± 16	0.3
LVESVI (mL/m^2)	83 ± 34	85 ± 38	79 ± 21	0.7
Volume RR (%)	37 ± 16	37 ± 14	36 ± 15	0.9
LVESVI ≥ 70 mL/m^2 (n , %)	13 (79)	5 (56)	8 (80)	0.3
LVEF (%)	34 ± 9	35 ± 9	33 ± 7	0.6

LV left ventricular, LVEF LV ejection fraction, MACE major adverse cardiac event, LVEDVI LV end-diastolic volume index, RR reduction rate, LVESVI LV end-systolic volume index

Hospital mortality

No hospital deaths occurred among the 19 patients.

Transthoracic echocardiography parameters during the follow-up period

All patients were followed up until March 2018, with a mean follow-up period of 47 ± 20 (range 5–97) months. Table 2 presents the changes in TTE parameters during the follow-up period in which data were gathered from all of the patients before death. Postoperative LVDd (from 65 ± 6 to 60 ± 7 mm; $p<0.01$) and LVDs (from 55 ± 8 to 51 ± 8 mm; $p<0.01$) significantly improved compared with the preoperative parameter values. The MR grade also improved, but not significantly (1.5 ± 1.0 to 0.8 ± 1.0 ; $p=0.06$). No differences were observed between preoperative and postoperative TTE parameters regarding LAD, LVEF, and RVFAC. We have not shown postoperative E/A , E/e' , and DT because 11 patients underwent MVR; we also could not measure estimated PAP because one quarter of the patients did not have any tricuspid valve regurgitation.

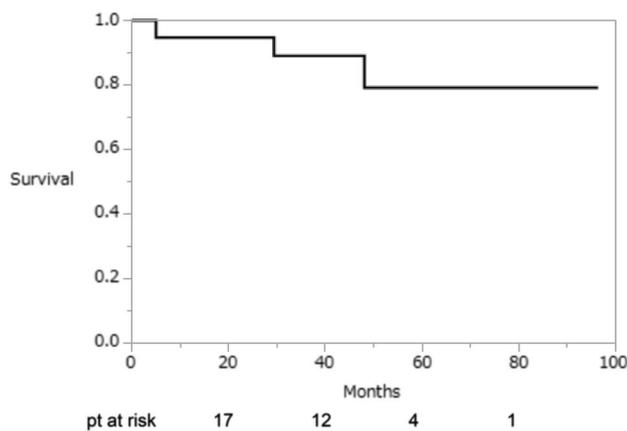


Fig. 2 The curve for freedom from cardiac-related death. The 3-year and 5-year freedoms from cardiac-related death were 89% and 79%, respectively

Clinical outcome

Four patients died during follow-up. Among them, three died of cardiac causes and one died of a non-cardiac cause. The 3- and 5-year overall survival rates were 89% and 63%, respectively. Figure 2 shows the curve for freedom from cardiac-related death. The 3- and 5-year freedoms from cardiac-related death were 89% and 79%, respectively.

MACEs occurred in ten patients; nine were heart failure requiring hospitalization including two cardiac-related deaths, and one was sudden death. Thus, the 3- and 5-year MACE-free survival rates were 58% and 41%, respectively.

The overall mean NYHA class, which included the three patients who died of cardiac causes and were categorized as NYHA class IV, improved from 2.9 ± 0.8 preoperatively to 2.3 ± 1.0 postoperatively. Four cases were classified as NYHA class I (21%), nine as class II (47%), three as class III (16%), and three as class IV (16%).

Comparison of characteristics, LV volume, and transthoracic echocardiography parameters between patients with and without MACEs

Tables 1, 3, and 4 show comparison of characteristics, LV volume, and TTE parameters between patients with and without MACEs. Patients with MACEs were associated with preoperative higher NYHA class, B-type natriuretic peptide level, *E/A*, and *E/e'*, and lower LVEF by left ventriculography and RVFAC. In addition, the LV dimension did not change and RVFAC remained low during follow-up in patients with MACEs, in contrast with the findings for those without MACEs.

Risk factor analysis for MACEs

Table 5 presents the univariate and multivariate Cox proportional hazards models for MACEs. Ten parameters, including RVFAC, were significant preoperative and surgical-related predictors of MACEs in the univariate analysis. In the multivariate analysis, RVFAC (risk ratio [RR] = 0.92, 95% confidence interval [CI] 0.86–0.98, $p=0.01$) and LVEF by left ventriculography (RR = 0.83, 95% CI 0.68–0.97, $p=0.02$) were significant preoperative predictors of MACE. Four

Table 4 Comparison of transthoracic echocardiogram parameters between patients with and without MACEs

Parameter	Preoperative			During follow-up		
	No MACE ($n=9$)	MACE ($n=10$)	p between groups	No MACE ($n=9$)	MACE ($n=10$)	p between groups
LVDd (mm)	$64 \pm 8^*$	65 ± 4	0.7	$55 \pm 7^*$	64 ± 3	<0.01
LVDs (mm)	$53 \pm 9^*$	56 ± 7	0.4	$46 \pm 7^*$	56 ± 5	<0.01
LAD (mm)	44 ± 5	48 ± 6	0.1	41 ± 7	48 ± 6	0.06
LVEF (%)	33 ± 10	27 ± 10	0.2	36 ± 10	29 ± 9	0.2
MR grade	1.1 ± 0.9	1.9 ± 0.9	0.1	0.6 ± 0.7	1.0 ± 1.2	0.4
PAP (mmHg)	32 ± 11	48 ± 16	0.06	–	–	–
RVFAC (%)	40 ± 7	27 ± 13	0.02	43 ± 9	32 ± 15	0.06
<i>E/A</i>	1.4 ± 1.6	3.5 ± 1.0	0.01	–	–	–
<i>E/e'</i>	15 ± 4	32 ± 9	<0.01	–	–	–
DT (ms)	149 ± 61	158 ± 103	0.8	–	–	–

MACE major adverse cardiac event, LVDd left ventricular diastolic dimension, LVDs left ventricular systolic dimension, LAD left atrium dimension, LVEF left ventricular ejection fraction, MR mitral regurgitation, PAP pulmonary artery systolic pressure, RVFAC right ventricular fractional area change, DT deceleration time

* p value <0.05 between the preoperative and during follow-up

Table 5 Univariate and multivariate risk factor analysis for MACEs

Variable	Univariate analysis			Multivariate analysis		
	RR	95% CI	<i>p</i> value	RR	95% CI	<i>p</i> value
Preoperative RVFAC	0.93	0.88–0.98	0.01	0.92	0.86–0.98	0.01
RVFAC <35%	4.06	1.11–18	<0.01			
LAD	1.17	1.02–1.36	0.02			
PAP	1.04	1.002–1.09	0.04			
<i>E/A</i>	1.53	1.07–2.23	0.02			
<i>E/e'</i>	1.14	1.05–1.24	<0.01			
NYHA			0.01			
LVESVI ≥ 94 mL/m ²	2.65	0.49–48	0.3	0.23	0.02–2.42	0.2
LVEF	0.87	0.73–0.98	0.02	0.83	0.68–0.97	0.02
BNP	1.001	1.0003–1.0002	<0.01			
MVR	6.55	1.22–121	0.03			
Postoperative LVESVI ≥ 70 mL/m ²	1.88	0.47–12	0.3	0.30	0.04–2.81	0.3
Follow-up LVDd	1.18	1.04–1.38	<0.01	1.28	1.07–1.59	<0.01
LVDs	1.14	1.02–1.27	0.01			
RVFAC	0.92	0.86–0.98	<0.01	0.94	0.87–0.99	0.04
RVFAC <35%	4.73	1.32–18	0.02			

MACE major adverse cardiac event, RR Risk ratio, CI confidence interval, RVFAC right ventricular fractional area change, LAD left atrium dimension, PAP pulmonary artery systolic pressure, NYHA New York Heart Association classification, LVESVI left ventricular end-systolic volume index, LVEF left ventricular ejection fraction, BNP B-type natriuretic peptide level, MVR mitral valve repair, LVDd left ventricular diastolic dimension, LVDs left ventricular systolic dimension

factors were significant postoperative predictors of MACEs in the univariate analysis, whereas RVFAC (RR = 0.94, 95% CI 0.87–0.99, *p* = 0.04) and LVDd (RR = 1.28, 95% CI 1.07–1.59, *p* < 0.01) were significant postoperative predictors of MACE in the multivariate analysis.

Comparison of outcomes in patients with preoperative RVFAC of ≥ 35% and < 35%

No significant differences were observed between patients with preoperative RVFAC of ≥ 35% (*n* = 10) and < 35% (*n* = 9) in terms of characteristics, LV volume, and TTE parameters, except preoperative NYHA class (2.5 ± 0.7 vs. 3.4 ± 0.7; *p* = 0.03), *E/e'* (17 ± 5 vs. 30 ± 11; *p* = 0.03), and RVFAC (42 ± 6% vs. 24 ± 11%; *p* < 0.01).

No significant difference in freedom from cardiac-related death was observed between the groups. The 3- and 5-year freedoms from cardiac-related death in patients with RVFAC of ≥ 35% were 89% and 67%, respectively, and those in patients with RVFAC of < 35% were 89% and 89%, respectively (*p* = 0.6). However, a significant difference in MACE-free survival was observed between groups. The 3- and 5-year MACEs-free survival rates in patients with RVFAC of ≥ 35% were 80% and 60%, respectively, and those in patients with RVFAC of < 35% were 33% and 22%, respectively (*p* = 0.02) (Fig. 3).

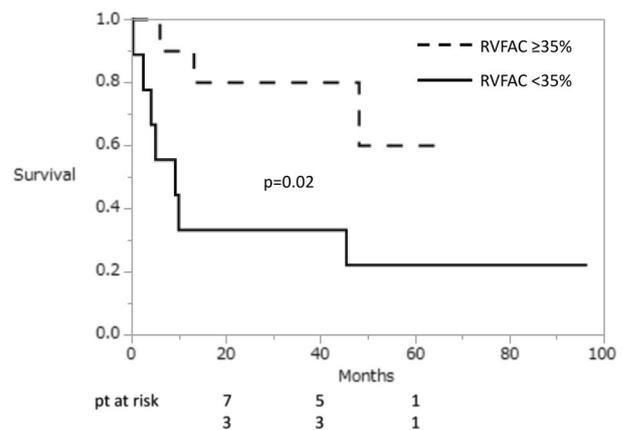


Fig. 3 The curve for MACE-free survival. The 3-year and 5-year MACE-free survival rates for patients with RVFAC ≥ 35% were 80% and 60%, respectively, and for patients with RVFAC < 35%, these rates were 33% and 22%, respectively (*p* = 0.02). MACE major adverse cardiac event, RVFAC right ventricular fractional area change

Discussion

Our results showed that SVR provided acceptable freedom from cardiac-related death; however, it was also associated with frequent MACEs. In addition to LVEF by left

ventriculography, preoperative RVFAC to quantify RV function was one of the predictive factors of MACEs after SVR.

RV dysfunction in cardiomyopathy does not simply result from pulmonary venous and arterial hypertension due to LV chronic dysfunction because RV systolic function is the result of a complex interaction with the remodeled and enlarged left ventricle, LV septal function, LV myocardial function, and PAP with or without MR [14]. RV dysfunction is a well-known predictor of chronic heart failure [15–17]. However, only limited data are available on the influence of RV function on outcomes after SVR.

In the subanalysis of the STICH trial, patients with moderate-to-severe RV dysfunction who received CABG and SVR had significantly higher mortality and cardiovascular hospitalization rates in the long-term follow-up period than those who received CABG alone [18]. Few studies also demonstrated that RV systolic dysfunction was associated not only with early outcomes [19] but also with late outcomes after SVR [14].

In this study, we demonstrated several interesting findings regarding the implications of RVFAC on outcomes. Firstly, SVR can be performed safely, even for patients with severe RV dysfunction. RV function is often impaired after cardiac surgery, owing to ineffective myocardial protection, air embolism, pulmonary vasoconstriction related to protamine, and the inflammatory response to extracorporeal circulation [14, 20]. This influence could be pronounced in patients with severe RV dysfunction and may exacerbate early outcomes after SVR. However, no hospital deaths occurred regardless of severe RV dysfunction in this study. Meticulous perioperative management using IABP might contribute to good early outcomes [21].

Secondly, preoperative RVFAC was negatively associated with LV diastolic properties such as PAP, E/A , and E/e' . This is in line with the findings of the studies by Garatti et al. and Kukulski et al. [14, 18]. Moreover, RVFAC did not significantly improve, and RVFAC during follow-up as well as RVFAC were predictors of MACEs. In general, SVR improved systolic function but worsened LV diastolic function to some extent [22, 23]. Thus, if the RV function remains too low to handle the increased afterload due to the increased LV filling pressure, SVR may worsen the outcomes in patients with ICM [19].

Thirdly, patients with MACEs retained larger LVDD and LVDs during follow-up compared with the findings in those without MACEs. Moreover, LVDD during follow-up was also a significant postoperative predictor of MACEs. As previously mentioned, preoperative RVFAC was negatively correlated with LV diastolic properties in this study. LV diastolic dysfunction is a strong predictor of cardiovascular events even after a cardiovascular surgery [24]. Additionally, some studies revealed that preoperative severe LV diastolic dysfunction was not associated

with LV reverse remodeling but was strongly related to worse outcomes after SVR [25, 26]. Although the correlation between the degree of myocardial viability and RV function was not investigated in this study, the increasing severity of RV systolic dysfunction may reflect a progressively more advanced LV remodeling [14, 18] and a lower amount of LV reserve [19].

Lastly, no significant differences in preoperative and postoperative LV volume were observed between patients with and without MACEs; preoperative and postoperative LVESVIs were not predictors of MACEs. Although we cannot precisely explain why LV volume did not impact outcomes, one plausible reason is simply the small number of patients in this study. Another reason may be that patients in this study had already an extremely large LV volume (LVESVI: 134 ± 56 mL/m²). The volume reduction effect by SVR has limits. The maximum values of preoperative LV sizes to achieve postoperative LVESVI of < 60 mL/m² are 65 mm for LVDD and 94 mL/m² for LVESVI [4]. Consequently, the postoperative LVESVI of 83 ± 34 mL/m² with a volume reduction rate of $37 \pm 16\%$ was still comparably larger in our study [4, 12]. In such situations, other factors like RV function or LV diastolic properties may enhance its effect on outcomes [8]. With regard to LVEF as a predictor of MACEs, preserving LVEF is critical because LV volume reduction by SVR may cause decreased stroke volume [27].

Hence, preoperative RV dysfunction could influence outcomes in patients after SVR, which is similar in patients with chronic heart failure. Consequently, patients with RV dysfunction could benefit from additional perioperative measures for RV protection, such as the use of IABP and/or inhaled nitric oxide [19]. Otherwise, refraining from SVR and avoiding conventional surgery, transplantation, or mechanical support should be considered [19, 28].

However, our study showed that no hospital mortality occurred and the freedom from cardiac-related death at 5 years was 79% in patients with RV dysfunction, although MACEs commonly occurred. Nevertheless, these findings seem acceptable considering their poor prognosis. Moreover, few studies indicate that patients with RV dysfunction comprise a significant proportion of those needing surgery for ICM [19, 29], such as in the present study (47% RV dysfunction in this study). Thus, considering the shortage of donors for transplantation and the inherent unresolved problems due to thromboembolism, bleeding, or infection of the mechanical support device, SVR may remain as an alternative even for patients with RV dysfunction. This can be achieved with adequate patient selection and proper assessment of RV function (i.e., an assessment of the septal viability by gadolinium-enhanced MRI [20]), meticulous perioperative management, and optimal management of RV dysfunction as well as LV dysfunction, using β -blockers.

Further studies are needed to ascertain the influence of RV dysfunction on outcomes in the treatment of ICM and to provide patients with optimal treatment regimens.

Limitations

There are several limitations that should be considered in this study. This was a retrospective study with a small number of patients and different surgical techniques, including SVR with or without CABG and/or MVR. Our limited surgical experience with ICM could have influenced the outcomes after SVR. However, no hospital death was observed, and the freedom from cardiac-related death was acceptable as compared with results from previous studies. We used RVFAC as the only measure to quantify RV function in this study; RVFAC is one of the recommended methods to quantitatively estimate the RV function with prognostic value [13]. However, combining more than one measures of RV function, such as tissue Doppler-derived tricuspid lateral annular systolic velocity and RV index of myocardial performance, may more reliably distinguish normal from abnormal function [13]. MRI is very useful in assessing myocardial anatomy, regional and global function, and extent of scar of both ventricles. However, MRI was not used. We could not surmise which chronic heart failure was considered as MACEs, either left or right or both when patients were readmitted due to heart failure because they did not always undergo a detailed examination using swan ganz catheter measurements. Moreover, this issue has not been considered in this study, which is one of the weak points of this study. However, we think that RVFAC must mirror not only RV function but also LV function as previously mentioned. Thus, identifying the accurate chronic heart failure, either left or right or both, is difficult, in contrast to RV failure with current continuous-flow left ventricular assist devices.

Conclusions

SVR for ICM provided acceptable freedom from cardiac-related death. However, MACEs commonly occurred, and RV dysfunction was associated with MACEs.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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