Pre-operative Speckle-tracking Imaging to Predict the Need for Right Ventricular Support in Patients Undergoing Left Ventricular Assist Device Implantation

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ABSTRACT

Background: Right ventricular (RV) dysfunction after left ventricular assist device (LVAD) implantation significantly complicates post-device management and has been shown to be associated with increased mortality. Pre-operative identification of patients who may develop post-LVAD RV dysfunction is challenging. This study was designed to evaluate pre-operative echocardiographic speckle tracking imaging as a predictor of post operative RV dysfunction. **Methods:** Thirty-nine patients who underwent Heartmate II LVAD placement in a single center were studied. Pre- and post-operative clinical, hemodynamic, laboratory, and echocardiographic data were prospectively collected as part of an ongoing institutional LVAD database. RV strain parameters were measured retrospectively using off-line speckle-tracking analysis software. **Results:** Twenty five of 39 LVAD recipients developed acute RV failure during the early post-operative period. RV function in 14 of these recipients improved with inotropes and judicious adjustment of LVAD parameters. Eleven patients, however, expired despite aggressive medical therapy including 7 patients who underwent placement of an RVAD. These 11 individuals were identified as having significantly lower global RV strain prior to device placement ($p<0.05$). Seventy two percent of the patients with a peak longitudinal systolic RV strain higher than -3%, expired. Twenty-four of 27 (88%) patients with a global RV strain of -3% or lower survived without need for an RVAD ($p<0.001$). Hemodynamic, laboratory and traditional echocardiographic data were not predictive of post-LVAD RV dysfunction or survival. Multivariate analysis showed RV longitudinal strain, especially global strain, to be the only significant predictor of severe RV dysfunction. **Conclusion:** Poor intrinsic RV myocardial function is associated with a higher mortality in LVAD patients. Speckle-tracking echocardiography imaging, particularly, peak systolic global RV strain appears to be promising in predicting LVAD patients who require RVAD.

Key words: Echocardiography, Heart Failure, Right heart function, Strain, Ventricular assist device.

BACKGROUND

Left ventricular assist devices (LVADs) are currently used either as a bridge to transplantation (BTT) or destination therapy (DT) in a carefully selected population of patients with refractory LV dysfunction. It has been estimated that between 30,000 and 100,000 individuals in the US might benefit from permanent LVAD support.[1-2] Right ventricular failure is common, occurs in 20%–30% of individuals undergoing LVAD support, and is associated with significant post-LVAD morbidity and mortality.[3-7] Therefore, identification of patients who will develop RV failure after LVAD implantation and adoption of timely interventions to prevent RV failure will likely improve outcomes after device placement. Unfortunately, pre-operative prediction of RV failure post LVAD

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DOI: 10.5530/jcdr.2015.1.1

remains challenging. Although several studies have proposed specific clinical, laboratory, hemodynamic, and echocardiographic parameters that may serve as risk factors for RV dysfunction after LVAD placement, none have proven to be reliable indices for predicting post-LVAD RV dysfunction.[8-10] Conventional echocardiography techniques that measure RV dimensions and ejection fraction are load dependent and may be significantly affected by common abnormalities including tricuspid regurgitation. Cardiac magnetic resonant imaging appears to be promising for evaluation of RV function. However, patients scheduled for LVAD placement often have contraindications to MR imaging such as the presence of implanted defibrillators.

The dimensionless "strain" index, which was introduced recently for the evaluation of left ventricular and right ventricular (RV) function, is relatively load-independent.[11,12] Speckle-tracking echocardiography, an angle independent technique for quantifying myocardial deformation, has been shown to be a reliable method for measuring LV or RV strain.[13-21] Previous studies have reported RV longitudinal systolic strain and strain rate predicts future right heart failure, clinical deterioration, and mortality in patients with pulmonary arterial hypertension.[22,23] However, there have been only limited data on the assessment of the RV function using strain-based measurement in LVAD patients.[24,25]

We hypothesized that intrinsic RV myocardial dysfunction is one of the most common causes of post-LVAD RV dysfunction. The purpose of this study was to determine whether pre-operative RV global strain by 2-D echocardiography speckle tracking imaging, can predict post-LVAD RV dysfunction.

METHODS

Patients

We studied forty-five consecutive patients who received a second-generation continuous-flow LVAD (Heart-Mate II, Thoratec Corporation, Pleasanton, California) at a single center from January 1st, 2010 to December 31st, 2011. Thirty-nine patients who underwent LVAD placement for either destination or bridge to transplant therapy and 10 age-matched controls were included. Six patients were excluded because of poor quality of echocardiograms with no adequate visualization of RV free wall endocardial borders in the apical 4-chamber view, precluding reliable evaluation of RV longitudinal strain. The study was approved by the institutional review board and all patients consented to the procedures.

Clinical and demographic data

Prospectively collected pre-operative clinical, echocardiographic, hemodynamic, and laboratory data as well as data concerning post-operative adverse events, right heart failure, and six month mortality were obtained from the medical records. The clinical variables obtained before LVAD implantation included demographics, medications, preimplant inotrope dependency, preoperative intra-aortic balloon pump, and co-morbidities. The pre-operative laboratory data included blood urea nitrogen, creatinine, total bilirubin, alanine aminotransferase, aspartate aminotransferase and other parameters typically obtained on the standard completed metabolic profile. The hemodynamic data included measurements of the right atrial pressure, right ventricular systolic and diastolic pressure, mean pulmonary artery pressure, pulmonary capillary wedge pressures, cardiac output, cardiac index, and pulmonary vascular resistance. Echocardiographic examinations were performed in all patients within two weeks before surgery (mean 6 ± 9 days) without intervening major acute events such as myocardial infarction or pulmonary embolism. The primary end point was death or implantation of an RVAD due to acute RV failure within 6 months after LVAD implantation. Right heart failure was defined using previously described criteria, including requiring continuous post-operative inotropic agents for >14 days; pulmonary vasodilator use (inhaled nitric oxide) >48 hours, or right-sided mechanical support. Patients who underwent LVAD placement were divided into 2 groups. Group 1 included those who required an RVAD due to RV dysfunction and/or who died within 6 months. Group 2 included those who survived with an LVAD for more than 6 months without the need for RVAD support.

The Michigan RV risk score[26] was calculated based on 4 variables with vasopressor use adding 4 points, creatinine >2.3 mg/dl adding 3 points, bilirubin > 2 mg/dl adding 2.5 points, and aspartate aminotransferase > 80 IU/dl adding 2 points. A higher score is associated with a greater risk for RV failure.

Echocardiographic measurements

Two-dimensional echocardiography was performed before LVAD implantation using a commercially available system (Siemens Sequoia, Mountview, CA). Echocardiographic images were analyzed using offline speckle-tracking analysis software (Axius Velocity Vector Imaging, Syngo, Siemens). The RV endocardial border in the apical 4-chamber view was traced with 10-15 points placed starting and ending at 1 cm below the tricuspid annulus with special attention to

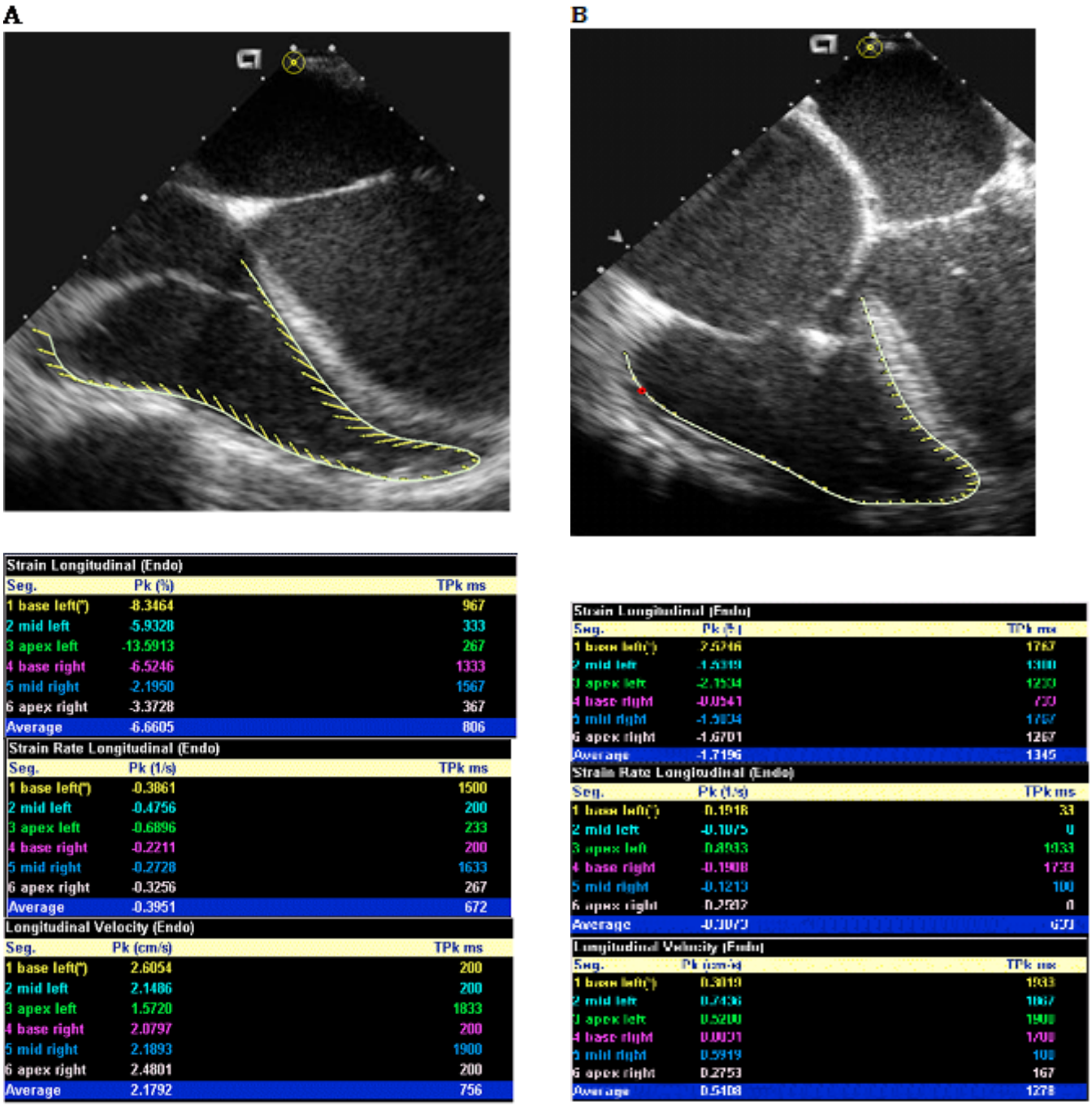


Figure 1: (A) An example of speckle tracking imaging of the RV in systole in a patient survived with LVAD without post-LVAD RV dysfunction or requiring RVAD. Note that there is a higher vector velocity and higher calculated absolute values of strain and strain rate. (B) An example of speckle tracking imaging in a patient with severe post-LVAD RV dysfunction who subsequently required RV assist device. Note that there is smaller amplitude of velocity vectors, reduced absolute value of strain and strain rate. LVAD: left ventricular assist device; RV: right ventricle.

a complete visualization of RV free wall. Images without complete visualization of RV free wall were excluded from the analysis. The RV free wall and septum were divided into 6 segments from the base to the apex. Global RV longitudinal myocardial velocity, strain, and strain rate were calculated by averaging the value of 6 segments (see Figure 1). RV free wall longitudinal myocardial strain was calculated by averaging the value of RV free wall apical, mid, and basal segments. Three consecutive heart cycles were recorded and averaged. For each patient, the mean value of 3 measurements was used for data analysis. Negative strain values indicate tissue shortening or contraction. The greater the negative value, the better the contraction or deformation. Traditional 2-D echocardiography LV

and RV dimensions and ejection fractions were obtained as well. Right ventricular systolic pressure was estimated from the peak continuous-wave Doppler velocity of the TR jet plus estimated right atrial pressure.[27] Interobserver variability was determined by a second independent blinded observer who measured the echocardiographic variables in 10 randomly selected patients. Intraobserver variabilty was determined by having the first observer who measured the data in all patients measure the same image on three occasions. Cardiac chamber size and function were measured according to the guidelines of the American Society of Echocardiography.[27,28]

Statistical analysis

Continuous variables were presented as mean ± standard deviation, whereas frequencies and percentages were used for categorical variables. Univariate analysis was performed using the Student t-test for normally distributed continuous variables, the Wilcoxon rank sum test for non-normally distributed continuous variables, and the chi-square test or Fisher's exact test for categorical variables to perform between-group comparisons for those patients with RVAD or died and those survived. Cox proportional hazards analysis were performed on univariable predictors of RV failure (entry criterion $p \leq 0.1$). A receiver-operating characteristic curve of RV strain was created, and the area under the curve (AUC) for the score was calculated. The AUCs were also calculated for conventional univariable predictors of RV failure (Michigan score, mean PA pressure, and RV ejection fraction). The AUC for RV strain was then compared to each of the other AUCs. Kaplan-Meier survival curves were created to evaluate post-LVAD survival. Survival between groups was analyzed by log rank for linear trend. Post-LVAD survival was defined as current LVAD support at the 6-month cutoff date or cardiac transplantation. Interobserver variability for echocardiographic parameters was assessed using the intraclass correlation coefficient (ICC). ICC was considered poor if <0.4, acceptable if between 0.4 and 0.75, good if between 0.75 and 0.9, and excellent if >0.9. $P<0.05$ was considered statistically significant. The data was analyzed by SPSS version 20.0.

RESULTS

Thirty-nine patients who underwent Heart-Mate II implantation and ten age-matched controls were included in the study. All patients received optimized medical therapy for heart failure treatment and all were followed up after their LVAD implantation for a median follow-up of 286 days (Inter Quartile range 78 to 436 days) or until orthotopic heart transplantation. Clinical characteristics and outcomes data are shown in (Table 1) for the 39 device treated patients. The mean age was 56 ± 15 years and 79% were men. Most patients were in NYHA class IV and had been receiving inotropic support (95%) prior to LVAD placement. The majority of patients had pulmonary hypertension (mean PA pressure of 39 ± 9 mmHg) with an increased mean wedge pressure (30 ± 9 mmHg) and a decreased cardiac index (2.0 ± 0.7 l/min/m^2). Nine (23%) patients underwent heart transplantation during the follow up period. Of 39 Heart-Mate II LVAD recipients, 25 (64%) developed some degree of clinical right heart failure post-operatively and required adjustment of the inotropes and/or addition of pulmonary vasodilators. Fourteen of these 25 patients ultimately recovered RV function, while 11 patients with intrinsic RV dysfunction as identified by significantly lower global RV peak myocardial velocity, strain, and strain rate prior to LVAD ($p<0.05$), eventually required RVAD placement and/or died despite aggresive medical therapy with inotropic agents and preload/afterload adjustments. Six (15%) patients died within 30 days and 11 (28%) died within 6 months. Seven of the 11 patients who died did so despite RVAD support.

There were no statistically significant differences in most clinical characteristics and laboratory data between the 28 LVAD survivors and the 11 patients who died (see Table 1). Mean PA pressure tends to be lower in the RVAD-death group but not statistically significant. Compared with normal controls, the LVAD groups had a significantly larger ventricular size (mean LV end diastolic dimension of 7.3 ± 0.9 cm vs. 4.6 ± 0.5 cm, $p<0.001$) and a significantly reduced ventricular ejection fraction (14% ± 6% vs. 59 ± 5%, $p<0.001$) (see Table 2 A). There was no statistically significant difference in ventricular size, ejection fraction, and RV systolic pressure between LVAD survivors group and LVAD/RVAD –death group (see Table 2B). RVEF tended to be lower in the RVAD-death group but this finding was not statistically significant.

Global longitudinal peak systolic RV myocardial velocities, strain and strain rate were significantly impaired in patients undergoing LVAD placement compared with normal controls ($p<0.001$). In contrast to conventional indices, patients who required RVAD or died had significantly lower global RV peak myocardial strain prior to LVAD placement as compared with the LVAD survivors ($p=0.004$). (Table 2) (Figure 1, 2) RV free wall longitudinal peak systolic myocardial strain was significantly higher in LVAD survivors than in the RVAD/death group (-7.1 ± 3.6% vs. -4.2 ± 3.7%, $p=0.03$).

Table 1: Baseline clinical and hemodynamic characteristics

Baseline characteristics	Total (n=39) Mean or n(%)	LVAD-survived (n=28)	RVAD/ Death(n=11)	P* value
Age (years)	56±15	56±15	57±16	0.78
Male	31(79%)	22(78%)	9 (82%)	0.99
Heart failure etiology				
Idiopathic dilated cardiomyopathy	16 (41%)	13 (46%)	3 (27%)	-
Ischemic cardiomyopathy	18 (46%)	12 (43%)	6 (55%)	0.99
Adriamycin cardiomyopathy	4 (10%)	2 (7%)	2 (18%)	-
Peripartum cardiomyopathy	1 (3%)	1 (4%)	0	-
NYHA functional class				
IV	37 (95%)	-	-	-
Inotropic therapy before LVAD implant	37(95%)	26 (93%)	11 (100%)	0.99
Aortic balloon pump support before implant	10	8	2	0.69
Reason for LVAD implant				
Cardiac Shock	29 (74%)	21 (75%)	8 (73%)	0.99
Ventricular Tachycardia	5 (13%)	5 (18%)	0	0.3
Destination therapy	21 (54%)	13 (46%)	8 (73%)	0.99
Pulmonary Hypertension	17 (89%)	12 (92%)	5 (83%)	0.99
Hemodynamic parameters				
Mean RA pressure (mmHg)	16±5	15±5	16±4	0.74
Mean PA pressure(mmHg)	39±9	40±9	35±6	0.07
PVR(Wood Units)	2.6±1.8	3±1.4	3±2.6	0.71
RV Systolic pressure	57±17	60±14	55±18	0.37
RV diastolic pressure	23±8	23±9	21±7	0.52
Mean wedge pressure(mmHg)	30±9	31±8	27±5	0.16
Cardiac output (l/min)	4±1.7	4±2	4±1.2	0.54
Cardiac index (l/min/m2)	2.0±0.7	2±0.8	2±0.4	0.62
Laboratory				
Blood Urea Nitrogen (mg/dl)	37±24	35±24	40±27	0.58
Creatinine (mg/dl)	1.5±0.7	1.5±0.7	1.3±0.5	0.11
Alanine aminotransferase (U/L)	60±135	38±43	113±240	0.12
Aspartate aminotransferase(U/L)	51±55	42±42	73±77	0.11
Total bilirubin (mg/dl)	1.9±2.6	1.2±0.8	2.3±3.7	0.55
Additional surgery with LVAD implantation				
None	30 (77%)	-	-	-
RVAD	3	-	-	-
Aortic valve replacement	3	-	-	-
Follow-up				
Length of hospital stay post-implant(days)	28±25	-	-	-
Heart Transplantation	9 (23%)	-	-	-

*** Compared LVAD-Survive group and LVAD-RVAD/Death group. LVAD:left ventricular assist device, RVAD:right ventricular assist device,RA:right atrium, PA:pulmonary artery, PVR: pulmonary vascular resistance, RV: right ventricle, NYHA: New York Heart Association.**

Table 2A: Comparison of ventricle size and function, right ventricular systolic pressure, right ventricular peak velocity, strain and strain rate in LVAD group and control

	Age	LVEDD (cm)	LVEF (%)	RVEDV (ml)	RVEF (%)	TR VelMax	RVSP (mmHg)	RV Peak Velocity (cm/s)	RV Strain (%)	RV Strain Rate(s^{-1})	RV Free Wall Strain (%)
Control group (N=10)	54±10	4.6±0.5	62±7	40±14	58±10	174±104	21±23	4.1±0.8	-21±3	-1.6±0.3	-
LVAD group (N=39)	56±15	7.3±0.9	16.3±6	75±52	29±13	279±72	45±15	2.1±1.1	-5.7±3	-0.7±0.3	-7.1±3.6
P value	0.68	<0.001	<0.001	0.04	<0.001	<0.01	<0.01	<0.001	<0.001	<0.001	-

Table 2B: Comparison of ventricle size and function, right ventricular systolic pressure, right ventricular peak velocity, strain and strain rate in LVAD-survive group and LVAD-RVAD/Death group

	Age	LVEDD (cm)	LVEF (%)	RVEDV (ml)	RVEF (%)	TR VelMax	RVSP (mmHg)	RV Peak Velocity (cm/s)	RV Strain (%)	RV Strain Rate(s^{-1})	RV Free Wall Strain (%)
LVAD-Survive (N=28)	56±15	7.3±0.9	16.3±6	76.1±55.6	31±13	281±78	45±15	2.4±1.1	-6.4±3	-0.8±0.3	-7.1±3.6
LVAD-RVAD/ Death (N=11)	57±16	7.2±1.2	16.7±8	72.1±38.6	22±12	274±58	44±13	1.3±0.6	-3.4±2	-0.5±0.4	-4.4±3.7
P value	0.78	0.86	0.88	0.83	0.07	0.79	0.85	0.006	0.004	0.03	0.03

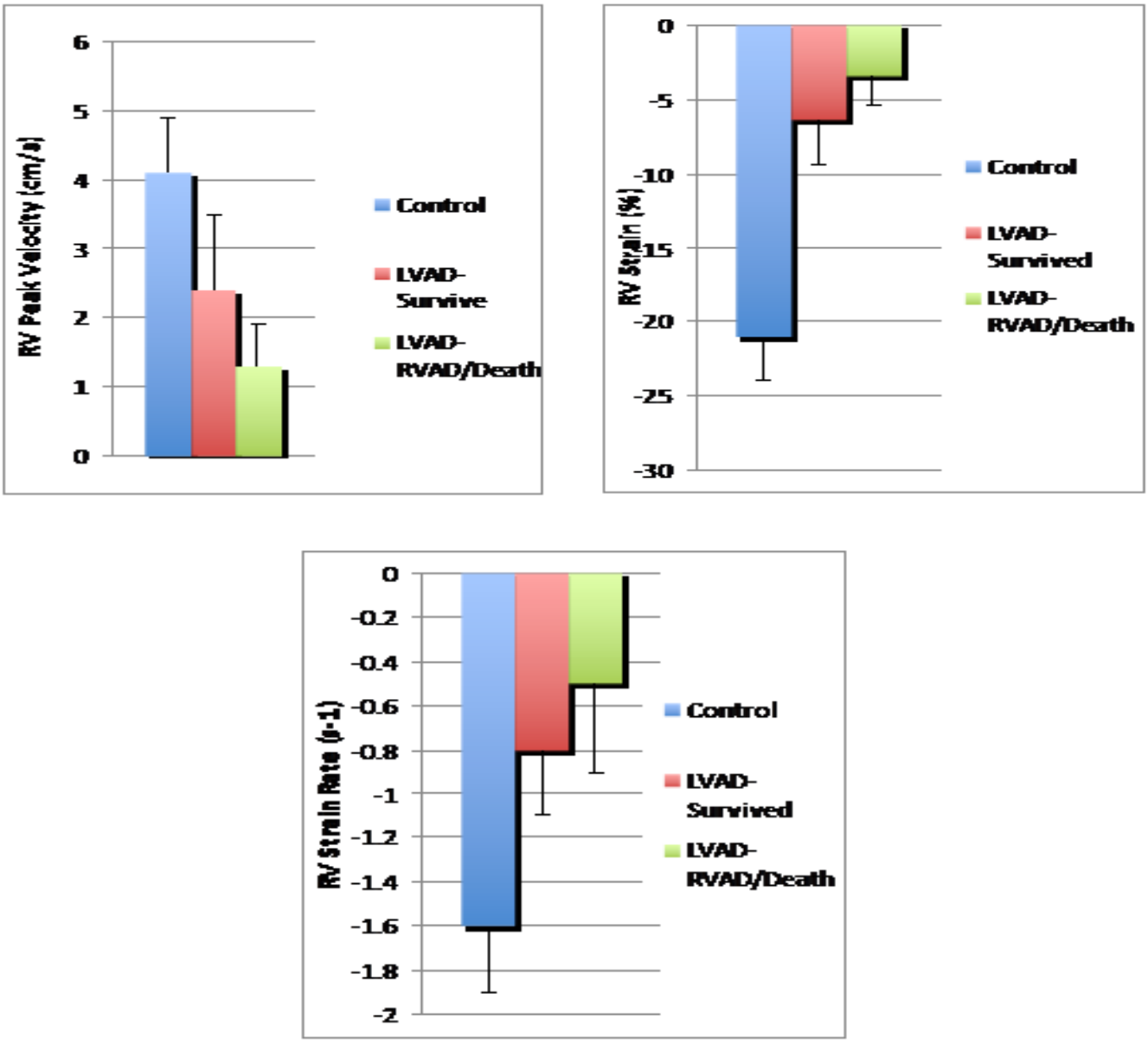


Figure 2: This figure showed (A) normal controls have RV strain with mean of -21%, LVAD-survived group of -5.8%, LVAD-RVAD/Death of -2.8%. (B) RV strain rate has a similar trend. (C) the same trend is shown in velocity. (Error bars represent standard deviation). LVAD: left ventricular assist device; RVAD: right ventricular assist device; RV: right ventricle.

Table 3: Multivariate Predictors

Variable	Hazard Ratio	P Value	95% CI
RV Strain	1.553	0.048	1.004-2.401
Mean PA pressure	0.965	0.342	0.897-1.038
RV ejection fraction	1.081	0.981	0.002-724.718
Michigan Score	0.941	0.787	0.607-1.46

RV: right ventricular PA: pulmonary artery

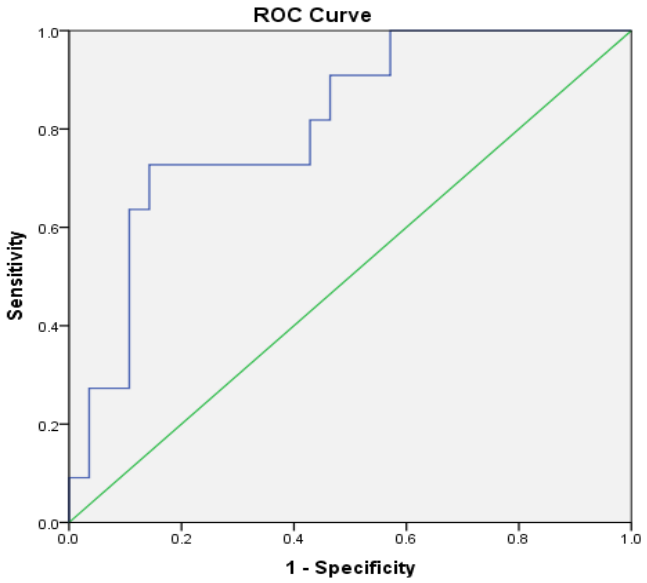


Figure 3: Receiver Operating Characteristic (ROC) curve of the RV strain predictor of RV failure

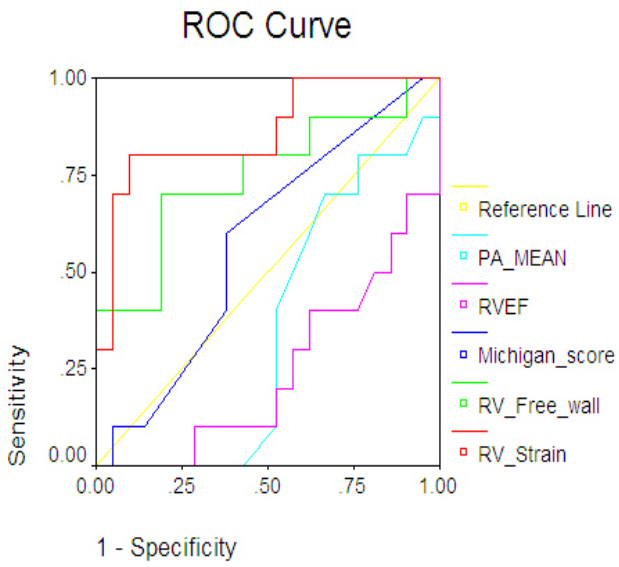


Figure 4: Receiver Operating Characteristic (ROC) curve and area under the ROC curve (AUC) of the RV strain and other univariable predictors of severe RV failure or death. PA_Mean: mean pulmonary artery pressure; RVEF: right ventricular ejection fraction; RV: right ventricle

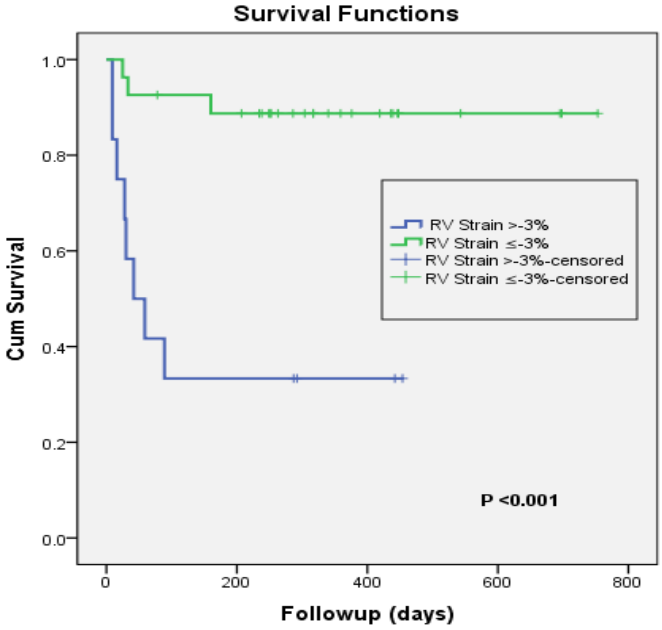


Figure 5: Kaplan-Meier survival of the 39 patients after LVAD implantation stratified according to the cut-off level of RV strain of -3%: Higher strain was an independent predictor of severe RV failure and death. CUM: cumulative, RV: right ventricle.

Table 4: Inter-observer Variability for Right Ventricular (RV) Parameters

Variable	Observer 1 (Mean ± SD)	Observer 2 (Mean ± SD)	Inter observer variability [ICC (95% CI)]
Strain	3.80 ± 2.46	3.72 ± 2.28	0.96 (0.83-0.99)
Strain rate	0.65 ± 0.31	0.55 ± 0.32	0.92 (0.65-0.98)
Velocity	2.15 ± 1.07	2.14 ± 1.64	0.93 (0.70-0.98)
RV end-diastolic volume	95.69 ± 52.35	99.17 ± 42.98	0.87 (0.44-0.97)
RV end-systolic volume	75.70 ± 47.71	80.09 ± 42.92	0.90 (0.62-0.98)
RV ejection fraction	0.24 ± 0.16	0.26 ± 0.17	0.85 (0.41-0.96)

Cox regression analysis revealed that RV global strain was the only independent predictor of RVF development after LVAD placement after the addition of mean PA pressure, RV ejection fraction, and Michigan score into the model. (Hazard ratio [HR] 1.6, 95% confidential interval [CI] 1.0 to 2.4, p=0.048). (see Table 3).

Using receiver-operating characteristic analysis, the optimal cut-off value for peak longitudinal systolic RV global strain in identifying a patient at risk for RV failure was -3% (sensitivity 73%, specificity 86%). Among all of the variables analyzed, RV strain showed the highest diagnostic accuracy (AUC 0.81) to predict RVAD or death, which is significantly higher than Michigan score, mean PA pressure and RV free wall strain ($p \leq 0.05$) (see Figure 3, 4). Kaplan-Meier survival analyses were performed to compare the survival between the two different groups using RV strain cutoff value of -3%. The corresponding 30-day survival rates after LVAD placement were 58% for RV strain > -3%, and 93% for RV strain ≤ -3%. The survival rates at 180 days were 33%, and 89% for the 2 groups (log-rank for linear trend $p<0.001$). (see Figure 5). The generated survival curves indicated an increased rate of mortality for patients with RV strain > -3%.

Interobserver variability for Velocity Vector Imaging was tested in 10 randomly selected patients by 2 independent observers. Interobserver variability was excellent for strain, strain rate, and velocity parameters (ICC range 0.92 to 0.96) and good for RV volume, ejection fraction parameters (ICC range 0.85 to 0.90). (see Table 4).

DISCUSSION

The significant finding of this study is that the RV dimensionless parameter – peak systolic longitudinal global RV strain derived from 2D echocardiographic speckle tracking imaging, appears to be a promising new parameter capable of predicting the risk of death, severe RV dysfunction or need for RVAD placement after LVAD implantation. To our knowledge, only one other study published[25] has ever looked at this parameter in this patient population. Grant's conclusion was that the free wall of the RV strain is a useful predictor of post-LVAD RV dysfunction in patients undergoing LVAD implantation. For the most part, we agree. Our study, however, demonstrated that the highest diagnostic accuracy with respect to predicting the need for an RVAD or death after LVAD implantation can be achieved not by measuring RV free wall strain but rather by measuring global myocardial longitudinal RV strain (AUC=0.81). In fact, the predictive accuracy of global myocardial longitudinal RV strain with an RV strain cutoff value of -3% is significantly higher than the Michigan score and mean PA pressure, as well ($p \leq 0.05$).

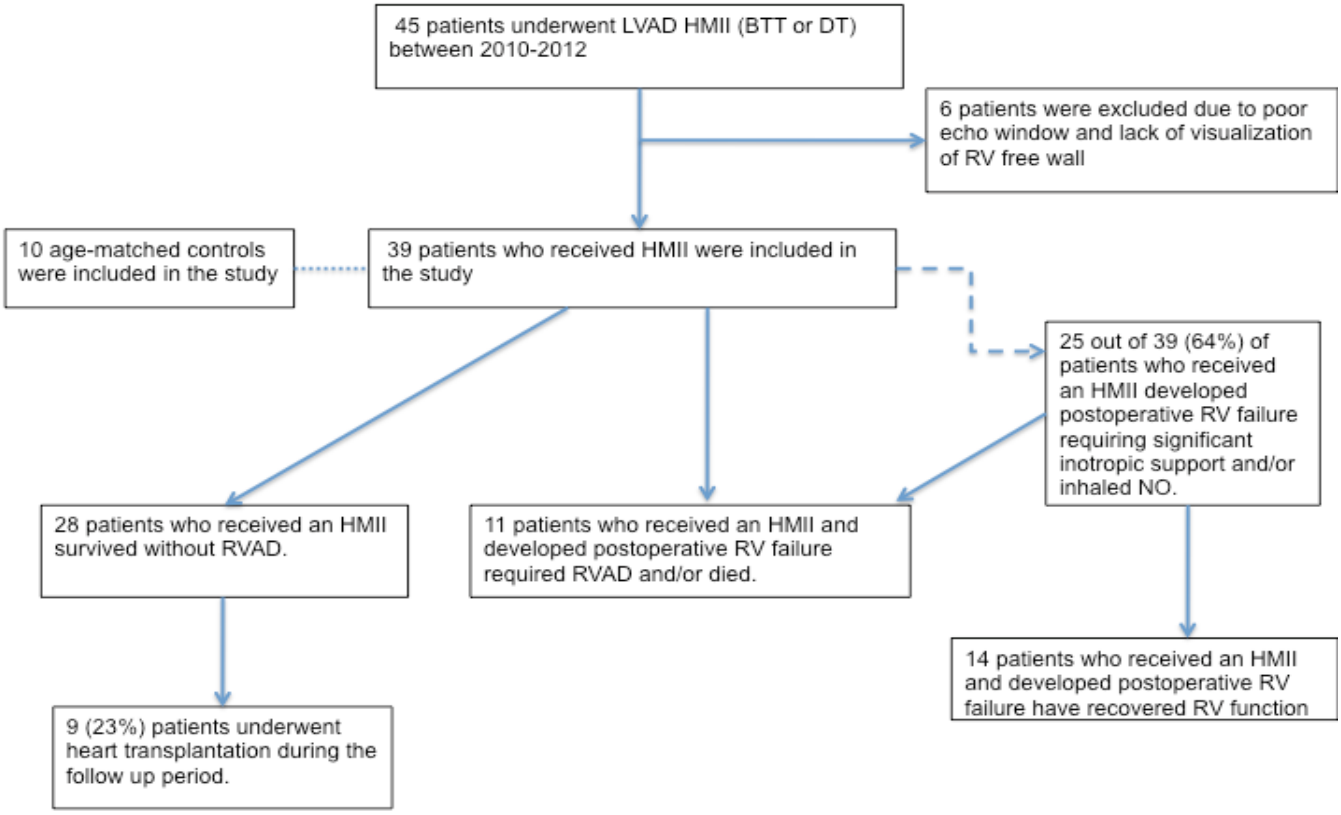
The mechanism of post-LVAD RV dysfunction is multi-factorial. Among our patients who developed acute RV failure immediately post LVAD implantation, 14 patients recovered from RV dysfunction after adjustment of LVAD parameters (RPM) or after the introduction of high dose inotropes, inhaled nitric oxide, or supplementation with intravenous thyroid hormone. Comparing the RV strains between the survived patients who had recovered and who had not recovered RV dysfunction, there is no significant difference, although both groups are significantly different from the RVAD/Died group. Eleven patients however, with intrinsic RV dysfunction, as identified by poor strain/strain rate, eventually died (including seven patients who required an RVAD) after failure of the above interventions. It is generally accepted that post-LVAD RV dysfunction may be secondary to either non-myocardial etiologies such as changes in pre-load and/or after-load often due to excess LVAD flow or unexpected volume shifts, or intrinsic myocardial etiologies such as poor RV contractility or changes in septal synchrony. In most cases both etiologies are probably in play to varying degrees. Of all potential etiologies of RV dysfunction, the most common may be excess decompression of the LV causing septal shift. Fortunately, this is easily addressed by simply optimizing LVAD flow rate usually under TEE guidance. Other common etiologies include pressure overload associated pulmonary hypertension and volume overload associated severe tricuspid regurgitation. The former can often be managed with inhaled nitric oxide or inhaled prostacyclin

while the later can generally be managed with diuretics. Intrinsic RV myocardial dysfunction may actually be the most difficult to address over the long run as both treatment options – inotropes and RVADs – are supportive but not curative. As such, intrinsic RV dysfunction is not uncommonly associated with high post-LVAD morbidity and mortality.

The high 30-day mortality of 15% in our study was similar to other studies.[26,29,30] The majority of our RVAD patients underwent relatively late "rescue" implantation of the RVAD. The timing of RVAD placement might partially explain the high mortality seen in this cohort at our facility and elsewhere (although comparison of cause of mortality across studies is difficult).[3,4,26,31] Earlier institution of RV support after LVAD placement may reduce or even eliminate the adverse impact of intrinsic RV dysfunction on other organ systems.[31-33] Assessing the risk for developing severe RV dysfunction after LVAD implantation allows the team to anticipate the need for early RVAD implantation and may improve the short and long term outcome of LVAD patients. In general, the assessment of intrinsic RV myocardial dysfunction has been clinically challenging. Traditional echocardiographic techniques measure RV dimension, ejection fraction, and tricuspid regurgitation, most of which are dependent on RV afterload and preload and often fluctuate significantly depending on loading conditions, are not reliable for assessing RV intrinsic contractility. Likewise, pre-operative clinical, hemodynamic, laboratory and traditional 2-D echocardiographic data are also of relatively limited use with respect to predicting post-LVAD RV failure, as was noted in our study as well.

As a dimensionless measure of global and regional myocardial deformation, strain has the advantage of distinguishing true contractility of the myocardium rather than translational motion, tethering or changes related to loading conditions, a potential concern with M-mode or 2-D derived measures of RV dimensions and ejection fraction.[34-35]

Furthermore, speckle-tracking echocardiography has the advantage of angle independence for quantifying myocardial deformation compared to tissue Doppler technique. Therefore, strain indices derived from speckle tracking imaging is most directly measuring the intrinsic myocardium function and it is less dependent on preload



Flow diagram of the study: RV: Right Ventricle; LVAD: Left Ventricular Assist Device; RVAD: Right Ventricular Assist Device; HMII: Heart-Mate II; BTT: bridge to transplantation; DT: destination therapy; NO: Nitric Oxide

and afterload. Sachdev and colleagues[22] reported that RV longitudinal systolic strain and strain rate values obtained using the same speckle tracking imaging used in this study, were decreased in patients with pulmonary arterial hypertension compared with the normal group and independently predicted future right heart failure, clinical deterioration, and mortality in patients with pulmonary arterial hypertension. Cameli *et al*[56] evaluated patients with refractory systolic heart failure referred for cardiac transplantation and found that TAPSE (tricuspid annular plane systolic excursion) and Doppler tissue imaging tricuspid systolic motion measurements were not useful indices of RV systolic function. Rather the measurement of RV longitudinal strain (especially free-wall RV longitudinal strain) provided a better estimate of RV systolic function. Our study further extends the utility of RV strain demonstrating that RV dysfunction post LVAD implantation can be predicted by this parameter.

Limitations

Our study has several limitations. First, this is single-center retrospective analysis of a relatively small cohort of patients. Second, acceptable images may not be obtainable in all patients. However, in such cases, Doppler-based strain assessment is a potential alternative option for such patients. Doppler-based strain, however, is more dependent on angle and frame rate and is generally felt to be inferior to speckle-based methods. This modality was not tested here. Third, our results were all performed with one software system. Potentially there may be variations between absolute values measured with different vendor's systems. Fourth, the definition of RV failure was neither prospectively nor strictly defined. This might account for the relatively high rate of RV dysfunction seen in our patient population as compared with other studies. Despite this limitation, those patients that did develop RV dysfunction were identifiable using global myocardial longitudinal RV strain. One could envision that if RV dysfunction were defined more rigidly, the difference would be even more apparent.

CONCLUSION

Poor intrinsic RV myocardial function is associated with a higher mortality in LVAD patients. RV strain indices using speckle-tracking imaging, particularly peak systolic global RV strain, appears to be promising in predicting which LVAD patients may develop postoperative RV dysfunction requiring RVAD placement.

ACKNOWLEDGEMENTS

The authors would like to thank Tony Martin, Donna Blicharz, Francesca Aiello for their contribution to the study.

DISCLOSURES

None of the authors have relationships with industry or financial associations related to this study to disclose.

CONFLICT OF INTEREST

The authors have no conflicts of interest to disclose.

ABBREVIATIONS

LV: Left Ventricle

LVAD: Left Ventricular Assist Device

PA: Pulmonary Arterial

RV: Right Ventricle

RVAD: Right Ventricular Assist Device

TR: Tricuspid Regurgitation.

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