Mitral Valve Repair Without Repair of Moderate Tricuspid Regurgitation

Michele De Bonis, MD, Elisabetta Lapenna, MD, Alberto Pozzoli, MD, Teodora Nisi, MD, Andrea Giacomini, MD, Mariachiara Calabrese, MD, Giovanni La Canna, MD, Federico Pappalardo, MD, Antonio Miceli, MD, Mattia Glauber, MD, Fabio Barili, MD, PhD, and Ottavio Alfieri, MD

Department of Cardiac Surgery, IRCCS San Raffaele Scientific Institute, Milan; Department of Cardiac Surgery, Fondazione Toscana Gabriele Monasterio, Massa; and Department of Cardiac Surgery, Santa Croce e Carle Hospital, Cuneo, Italy

Background. The objective of this study was to assess the fate at long term of mild-to-moderate functional tricuspid regurgitation (TR) left untreated at the time of mitral valve repair in patients with dilated cardiomyopathy.

Methods. We selected from our prospective hospital database 84 patients (age, 64 ± 9.6 years; ejection fraction, 0.31 ± 0.064) who underwent mitral repair for secondary mitral regurgitation in whom concomitant mild-to-moderate TR (nonlinear scale 1 to 4+) was left untreated. Tricuspid regurgitation was classified as mild in 61 patients (72.6%) and moderate in 23 patients (27.3%). Annular dilatation itself was not systematically measured and was not used as a trigger for tricuspid annuloplasty. Most of the patients were in New York Heart Association functional class III or IV (56 of 84; 66.7%).

Results. At a median follow-up of 7.3 years (interquartile range, 4.5 to 9.3), 17 patients (20.2%) had moderate-to-severe TR and 21 patients (25%) showed a

urrent guidelines recommend surgical treatment of secondary tricuspid regurgitation (TR) in patients with mild or moderate TR undergoing mitral valve (MV) surgery if significant dilatation of the tricuspid annulus is documented [1, 2]. Indeed, several observational series and small randomized studies have shown that in the presence of tricuspid annulus dilatation, not treating less than severe secondary TR may lead to progression of the tricuspid disease despite correction of the associated leftsided lesion [3-8]. The vast majority of those data, however, come from series that did not include patients with functional mitral regurgitation (MR) or included a very limited number of them [3, 5-7, 9]. Only a few reports describe the evolution of mild or moderate TR in the context of dilated cardiomyopathy (DCM), and results are usually limited to early or mid-term follow-up [10, 11]. In the past, the concept that less than severe secondary TR decreases after MV surgery alone was widely accepted [12, 13], and the size of the tricuspid annulus was not used

 $\ensuremath{\mathbb C}$ 2015 by The Society of Thoracic Surgeons Published by Elsevier

progression of at least two grades of their untreated preoperative TR. Freedom from moderate-to-severe TR or from progression of at least two grades of the baseline TR was 77% \pm 5% at 5 years and 56.7% \pm 8.4% at 10 years. Multivariate analysis identified preoperative right ventricular dysfunction (hazard ratio, 7.2; 95% confidence interval, 2.8 to 23; p = 0.001) and age (hazard ratio, 1; 95% confidence interval, 1.0 1.1; p = 0.03) as independent predictors of TR worsening.

Conclusions. A significant number of dilated cardiomyopathy patients with mild-to-moderate TR left untreated at the time of mitral repair show important TR at follow-up. In this setting, a more aggressive policy should be used taking into consideration the degree of annular dilatation and the function of the right ventricle and not simply the grade of TR.

> (Ann Thorac Surg 2015;100:2206–12) © 2015 by The Society of Thoracic Surgeons

to guide the decision-making process in terms of TR correction. Assessing the long-term results of this conservative approach, particularly in patients with DCM, may help to define the best strategy to adopt in this setting. Therefore, the purpose of this study was to evaluate the long-term fate of less than severe secondary TR in patients who underwent mitral repair for functional MR and in whom a tricuspid annuloplasty procedure was not concomitantly performed regardless of the size of the tricuspid annulus.

Material and Methods

Study Population

Two hospital databases (San Raffaele Hospital, Milan, and Fondazione Toscana Gabriele Monasterio, Massa, Italy) were used to select 84 patients with severe secondary MR and concomitant less than severe functional TR. All patients underwent MV repair between 1999 and 2008, and the associated functional TR was left untreated. Patients with primary or degenerative MR, acute MR caused by papillary muscle rupture, concomitant left ventricular (LV) reconstruction or aortic valve surgery, residual mild-to-moderate MR at discharge, or

Accepted for publication May 18, 2015.

Address correspondence to Dr De Bonis, Cardiac Surgery Department, IRCCS San Raffaele Scientific Institute, Via Olgettina 60, 20132 Milan, Italy; e-mail: michele.debonis@hsr.it.

preoperative implantable cardioverter-defibrillator (ICD) or pacemaker wires across the tricuspid valve and those who died in the hospital or within the first 6 postoperative months were excluded. The institutional ethic committee approved this study and waived individual consent for this retrospective analysis.

Echocardiographic Measurements

Preoperatively all patients underwent transesophageal echocardiography (TEE). Tricuspid regurgitation grade was measured by transthoracic echocardiogram (TTE) in the apical four-chamber view, the parasternal short-axis view at the level of the aortic valve, and the right ventricle (RV) inflow view. The nonlinear scale (1 to 4+) to assess the echocardiographic grade of regurgitation was adopted [11]. The vena contracta width at the narrowest portion of the regurgitant jet was measured in those patients in whom TR quantitation appeared to be more difficult. Secondary TR was considered less than severe when the regurgitant jet occupied less than 20% of the right atrial area and the vena contract width was less than 7 mm. The dilatation of the tricuspid annulus was usually assessed qualitatively, and precise annular dimensions were not routinely measured. Significant tethering of the tricuspid leaflet was defined as a coaptation depth of greater than 5 mm (distance between the tricuspid annular plane and the coaptation point of the tricuspid leaflets at the time of maximal systolic closure). Right ventricular function was assessed qualitatively in all patients. In addition, depending on the center and echocardiographer's preference, this qualitative evaluation was integrated by other quantitative variables such as the percent RV area change [14] and the peak systolic velocity of the tricuspid annulus assessed by tissue Doppler imaging. The peak systolic velocity of the tricuspid annulus was used as an index of global RV function by placing the sample volume of the pulse-wave tissue Doppler imaging on the tricuspid annulus at the site of attachment of the anterior leaflet in the two-dimensional four-chamber view. Peak RV systolic pressure as an estimate of pulmonary artery pressure was calculated by measuring peak tricuspid regurgitant velocity and using the modified Bernoulli equation, with 10 mm Hg added for the estimated right atrial pressure [15]. Pulmonary hypertension was defined as an RV systolic pressure greater than 40 mm Hg.

Surgery

All patients were operated on through a median sternotomy with standard cardiopulmonary bypass using moderate hypothermia. The MV was approached through a left atriotomy and repaired by means of an undersized annuloplasty. An edge-to-edge technique combined with moderately restrictive annuloplasty was preferred in San Raffaele Hospital when the tethering of the leaflets was more pronounced, as defined by a coaptation depth of at least 1 cm.

Follow-Up

Follow-up data were obtained by means of outpatient visit and TTE examination, or by means of telephone

interview with the patients and the referring cardiologists. At least one TTE was obtained at follow-up for all hospital survivors. Follow-up was 100% complete, and mean length was 7 ± 3 years (range, 0.7 to 14 years), with a median of 7.3 years (interquartile range, 4.5 to 9.3 years).

Statistical Analysis

Calculations were performed using SPSS version 22.0 (SPSS Inc, Chicago, IL) for Windows (Microsoft Corp, Redmond, WA) software package. Continuous data were expressed as mean + standard deviation or as median and interquartile range. Categorical data were reported as number and percentage. For continuous variables, differences were tested with the unpaired Student's t test, nonparametric Mann-Whitney U test (independent samples). or Wilcoxon signed-rank test (related samples). For categorical variables, the χ^2 test was used. Linear or logistic regression analysis was used for correlation of variables of interest as appropriate. Time-to-event data (survival and freedom from events) were analyzed by the Kaplan-Meier method, and differences among groups were evaluated with the log-rank test. Cox proportionalhazard regression was used to estimate the hazard ratios of potential predictors of TR of grade 3+ or greater or progression of the preoperative TR of at least two grades at follow-up. Variables with a probability value of less than 0.05 in univariate analysis were entered in a multivariable model.

Results

Clinical and Echocardiographic Data

Follow-up was obtained for all patients between December 1, 2013, and March 1, 2014. The study population comprised 84 patients. Mean age was 64 ± 9.6 years, and all patients had severe secondary MR and significant LV dysfunction (ejection fraction, 0.31 ± 0.064). New York Heart Association functional class III or IV was present in 56 of 84 patients (66.5%; Table 1). Concomitant TR was classified as absent or mild in 61 patients (72.6%) and moderate in 23 patients (27.3%) and was left untreated.

Mitral regurgitation was treated by means of an isolated undersized annuloplasty in 49 patients (58.3%), whereas the edge-to-edge technique was associated in the remaining 35 patients (41.7%). Rigid or semirigid rings were used in the vast majority of cases (82 of 84 patients, 97.7%) and included St. Jude Seguin (St. Jude Medical Inc, St. Paul, MN), Carpentier-Edwards Classic (Edwards Lifesciences, Irvine, CA), Edwards Geoform (Edwards Lifesciences), Sorin Memo 3D (Sorin Group, Saluggia, Italy), and Medtronic CG Future Band (Medtronic, Inc, Minneapolis, MN). Flexible rings (Medtronic Duran - Medtronic, Inc, Minneapolis, MN; St. Jude Tailor - St. Jude Medical Inc, St. Paul, MN) were implanted in only 2 patients (2.3%). Concomitant procedures were coronary artery bypass grafting (38 of 84 patients, 45.2%) and ablation of atrial fibrillation (13 of 84 patients, 15.4%).

Table 1. Clinical and Echocardiographic Preoperative Data at Time of Mitral Valve Surgery

Variables	Values	
Male/female	60/24	
Age (y)	64 ± 9.6	
Ischemic/idiopathic DCM (n, %)	56 (66.7)/28 (33.3)	
NYHA functional class		
П	28 (33.3)	
Ш	43 (51.2)	
IV	13 (15.5)	
Atrial fibrillation	17 (20.2)	
LVEF	0.31 ± 0.064	
LVEDD (mm)	66.9 ± 7.57	
LVESD (mm)	50.6 ± 7.29	
LVEDV (mL)	184.3 ± 52.7	
LVESV (mL)	129.5 ± 44.3	
SPAP (mm Hg)	44 ± 13.4	
RV dysfunction (n, %)	17 (20.2)	
SPAP >40 mm Hg (n, %)	36 (42.8)	
MR grade—TR grade (n, %)		
MR 4/4+—TR 0/4+	20 (23.8)	
MR 4/4+—TR 1+/4+	41 (48.8)	
MR 4/4+—TR 2+/4+	23 (27.3)	

dilated cardiomyopathy; LVEDD = left ventricular end-DCM = LVEDV = left ventricular end-diastolic voldiastolic diameter; LVEF = left ventricular ejection fraction; LVESD = leftume: LVESV = leftventricular end-systolic diameter; ventricular endsystolic volume; MR = mitral regurgitation; NYHA = NewYork Heart Association: RV = right ventricle; SPAP = systolicTR = tricuspid regurgitation. pulmonary artery pressure;

Thirty-seven patients (44%) died more than 6 months after surgery, for an overall survival of 74.2% \pm 4.8% at 5 years and 54.8% \pm 5.9% at 10 years. The cause of death was cardiac related in 22 of them (26.2%): congestive heart failure (12 patients), sudden deaths (3 patients), acute myocardial infarction (2 patients), multiorgan failure after heart transplantation (2 patients), endocarditis (1 patient), aortic dissection (1 patient), and cerebral bleeding after percutaneous transluminal coronary angioplasty (1 patient). Other noncardiac causes of death were cancer (8 patients), old age (4 patients), liver failure (1 patient), respiratory failure (1 patient), and Alzheimer disease (1 patient). Five- and 10-year freedom from cardiac death was 84.6% \pm 4% and 71.2% \pm 5.6% (Fig 1). Overall 12 patients had recurrent moderate-to-severe or severe MR. Four of them underwent reoperation (in 1 case because of endocarditis), 2 patients underwent heart transplantation, 4 died before reoperation, and 2 patients with moderateto-severe MR have not yet undergone reoperation.

All patients (100%) were evaluated during the initial part of follow-up by our heart failure outpatient clinic, with regular visits and a dedicated heart failure cardiologist. After 10 years we were still observing 40% of the patients. The others, owing to the particular advanced stage of cardiomyopathy, were seen by referring cardiologists, with adequate or optimal medical therapy.

As shown in Table 2, at follow-up significant improvements in LV function, LV dimensions, and systolic



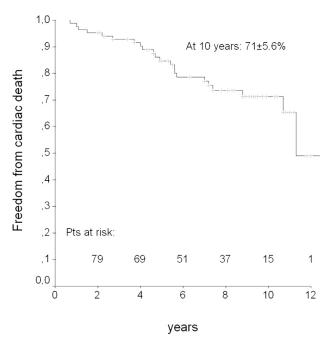


Fig 1. Freedom from cardiac death.

pulmonary artery pressure compared with baseline were documented. Also, the percentage of patients in New York Heart Association functional class III or IV decreased from 66.6% (56 of 84 patients) before operation to 25.9% (21 of 84 patients) at the last outpatient visit (p < 0.0001).

Evolution of Functional Tricuspid Regurgitation

At the last echocardiographic assessment, secondary TR was absent or mild in 39 patients (46.4%), moderate in 28 patients (33.3%), moderately severe in 12 patients (14.2%), and severe in 5 patients (5.9%). Overall, 17 patients (20.2%) had moderately severe or severe TR (Table 2). In 21 patients (25%), secondary TR worsened at least two

Table 2. Preoperative and Follow-Up Echocardiographic Data

/	1	8 1	
Variables	Preoperative	Follow-Up	p Value
LVEF	0.31 ± 0.064	0.36 ± 0.109	0.0001
LVEDD (mm)	66 ± 7.5	60 ± 8.4	0.0001
LVEDV (mL)	188 ± 56.2	170 ± 60.5	0.02
SPAP (mm Hg)	44 ± 13.7	40 ± 10.5	0.05
RV dysfunction (n, %)	17 (20.2)	25 (29.7)	0.2
TR grade (n, %)			0.0001
0/4+	20 (23.8)	3 (3.5)	
1+/4+	41 (48.8)	36 (42.8)	
2+/4+	23 (27.3)	28 (33.3)	
3+/4+		12 (14.3)	
4+/4+		5 (5.9)	

Table 3. Progression of Tricuspid Regurgitation in the 23 Patients With Tricuspid Regurgitation $\geq 3+$ at Follow-Up or With Tricuspid Regurgitation Worsening of at Least Two Grades Compared With Baseline

Preoperative TR	TR at Last Follow-Up	Number of Patients
0/4+	2+/4+	6
0/4+	3+/4+	1
0/4+	4+/4+	2
1+/4+	3+/4+	9
1+/4+	4+/4+	2
2+/4+	3+/4+	2
2+/4+	4+/+	1

 $TR = tricuspid \ regurgitation.$

grades (Table 3). Eighteen patients who died at follow-up had mild or moderate TR. The other 5 patients who died and the 2 patients who underwent transplantation had moderately severe or severe TR. Usually the mechanism of TR described at follow-up was persistent annular dilatation, although leaflet tethering (coaptation depth \geq 0.5 cm) was reported as an associate finding in 6 patients (7.1%). Freedom from moderately severe or severe TR was 85% \pm 4.2% at 5 years and 65% \pm 8.3% at 10 years (Fig 2). When the presence of moderately severe or severe TR at follow-up and worsening of at least two grades of preoperative TR were considered together, this combined end point was reached at follow-up by 23 of the 84 patients in the study (27.3%). Freedom from this combined end point was 77% \pm 5% at 5 years and 56.7% \pm 8.4% at 10 years (Fig 3).

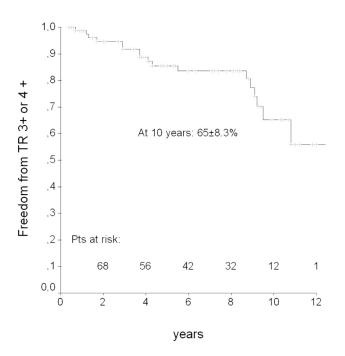


Fig 2. Freedom from tricuspid regurgitation (TR) \ge 3+ at last follow-up.

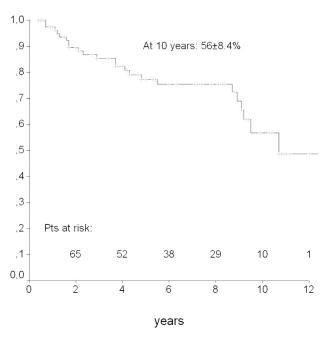


Fig 3. Freedom from the combined end point (follow-up tricuspid regurgitation $\geq 3+$ or progression of tricuspid regurgitation of at least two grades compared with baseline).

Preoperative Predictors of Tricuspid Regurgitation Worsening at Follow-Up

Table 4 reports the potential predictors of the combined end point moderately severe or severe TR or progression of the preoperative TR of at least two grades at follow-up. At univariate analysis, age (p = 0.02), preoperative RV dysfunction (p = 0.0001), and LV end-diastolic volume (p = 0.01) were significant risk factors. Multivariate analysis identified preoperative RV dysfunction (hazard ratio, 7.2; 95% confidence interval, 2.2 to 23; p = 0.001) and age (hazard ratio, 1; 95% confidence interval, 1.0 to 1.1; p = 0.03) as the only independent predictors of TR worsening. As shown in Table 4, the 95% confidence interval of the RV dysfunction hazard ratio is very wide (2.2 to 23), suggesting that for this specific predictor, further analysis in a high-number cohort is necessary. This is a preliminary analysis that needs to be confirmed.

Pacemaker or Implantable Cardioverter-Defibrillator With or Without Cardiac Resynchronization Therapy Implantation and Functional Tricuspid Regurgitation at Follow-Up

During follow-up, 38 patients (45.2%) underwent pacemaker or ICD implantation 7 months to 6 years after surgery. In particular, 6 patients (7.1%) received an ICD, 27 patients (32.1%) received an ICD with cardiac resynchronization therapy (CRT), and 5 patients (5.9%) received a pacemaker for atrioventricular block (4 patients) or after atrioventricular node ablation for refractory atrial fibrillation (1 patient). Of those 38 patients, 8 experienced moderately severe or severe TR or showed a progression of their preoperative TR of at least two

Preoperative Variables	Univariate		Multivariate	
	HR (95% CI)	p Value	HR (95% CI)	p Value
Age	1.0 (1.0–1.1)	0.02	1 (1.0–1.1)	0.03
Atrial fibrillation	0.7 (0.2–2.3)	0.6		
NYHA functional class III or IV	1.0 (0.4–2.6)	0.8		
LVEF	0.9 (0.9–1.0)	0.4		
LVEDV	1.0 (1.0-1.01)	0.01	1 (0.9–1)	0.2
SPAP >40 mm Hg	1.3 (0.5–3.1)	0.5		
RV dysfunction	8.3 (3.3-20.7)	0.0001	7.2 (2.2–23)	0.001

Table 4. Predictors of Tricuspid Regurgitation \geq 3+ or Tricuspid Regurgitation Progression of at Least Two Grades Compared WithBaseline

grades before pacemaker, ICD, or ICD-CRT implantation. Eight more patients (21%) experienced TR progression (as previously defined) between 12 months and 4 years after pacemaker, ICD, or ICD-CRT insertion. The effective impact of this procedure on worsening of TR remains uncertain because in most of the cases, the positioning of the transtricuspid lead increased the baseline degree of TR by no more than one grade. However, when the incidence of TR progression was compared in the two groups of patients with and without pacemaker, ICD, or ICD-CRT implantation, the difference was not statistically significant (8 of 38 [21%] versus 7 of 46 [15.2%], respectively; p = 0.6).

Patterns of Left Ventricular Remodeling and Influence on Tricuspid Regurgitation Progression

The pattern of LV remodeling after surgery influenced the evolution of functional TR. Reverse LV remodeling, defined as a decrease of at least 15% in the indexed LV end-systolic volume or in both the indexed LV endsystolic volume and LV end-diastolic volume at the last echocardiographic follow-up compared with the preoperative values, was documented in 39 patients (46.4%) of the study population (reverse LV remodeling group). Moderately severe or severe TR or a progression of the preoperative TR of at least two grades occurred in 5 patients of this group (12.8%), whereas it was documented in 18 patients of the group without reverse LV remodeling (18 of 45 patients; 40%; p = 0.01). Of the 23 patients with TR progression, 18 had no reverse remodeling of the LV (78.2%). Therefore at follow-up they had a lower LV ejection fraction (p = 0.005) and a higher rate of recurrent MR of at least grade 3+ (p = 0.003), pulmonary hypertension (p = 0.0001), and RV dysfunction (p = 0.0001) compared with those without TR worsening. Not surprisingly, New York Heart Association functional class III or IV was significantly more common in patients belonging to the group with TR progression (47.8% versus 18%; p = 0.01; Table 5).

Comment

The main finding of this study is that in DCM patients undergoing mitral repair for secondary MR, a preoperative condition of less than moderately severe TR, left untreated, may progress over the course of years in a significant proportion of patients. Therefore, deciding whether or not to perform a concomitant tricuspid

Table 5. Echocardiographic and Clinical Data at Last Follow-Up in the Tricuspid Regurgitation Progression and No Tric	cuspid
Regurgitation Progression Groups ^a	•

Variables	TR Progression $(n = 23)$	No TR Progression $(n = 61)$	p Value
	31 ± 10.49	38.4 ± 10.44	0.005
LVEDD (mm)	61.8 ± 10.65	60 ± 7.82	0.4
LVEDV (mL)	191.7 ± 67.76	162 ± 54.19	0.1
SPAP (mm Hg)	47.7 ± 10.81	$\textbf{36.7} \pm \textbf{8.56}$	0.0001
RV dysfunction (n, %)	15 (65.2)	10 (16.3)	0.0001
SPAP >40 mm Hg (n, %)	18 (78.2)	11 (18)	0.0001
Recurrence of MR \geq 3+ (n, %)	8 (34.7)	4 (6.5)	0.003
Atrial fibrillation (n, %)	8 (34.7)	13 (21.3)	0.3
NYHA functional class III or IV (n, %)	11 (47.8)	11 (18)	0.01

^a TR progression defined as TR \geq 3+ or TR progression of at least two grades compared with baseline.

annuloplasty at the time of MV surgery simply on the basis of the grade of TR should be avoided, particularly in the setting of functional MR. Current guidelines recommend performing concomitant tricuspid annuloplasty at the time of left-sided valve surgery even in patients with only mild functional TR, in the presence of tricuspid annular dilatation [1, 2] or prior evidence of right heart failure [2]. Despite that, many surgeons still prefer a conservative approach when concomitant TR is less than severe, regardless of the annular dimensions [16, 17]. One of the reasons behind this attitude is that the studies supporting a more aggressive approach are often singlecenter, retrospective series with various etiologies of MR and different criteria to define the dilatation of the tricuspid annulus [3, 5, 18]. The only randomized study published so far that confirms the beneficial effect of prophylactic tricuspid annuloplasty enrolled only 42 patients with all types of MR etiologies (functional, degenerative, endocarditis, prosthetic dysfunction, and MV repair failure) [7]. While waiting for more consistent and possibly randomized data, we believe that the results of our study might be useful to guide the decision-making process in the context of MR secondary to DCM. Indeed, in this setting, there are some peculiar conditions that might lead to TR progression. The natural history of the disease, the presence of a low ejection fraction and pulmonary hypertension, the high prevalence of RV dysfunction, the need for ICD or biventricular pacing, and the possibility of recurrence of MR are some of them. Previous studies have shown that a significant number of patients who underwent MV repair for MR secondary to ischemic or nonischemic DCM present a higher grade of their baseline TR, which was left untreated, at follow-up [7, 10, 11]. Those studies, however, are limited to early or mid-term follow-up, and some of them also include patients who received a concomitant tricuspid annuloplasty [10, 11]. In our series, the decision to avoid a concomitant tricuspid annuloplasty was based on a severity of TR that was not significant rather than on the degree of annular dilatation.

Our data show that in more than one quarter of the cases, uncorrected moderate or mild TR becomes at least moderately severe or shows a progression of at least two grades. Among the baseline characteristics of the patients, age and preoperative RV dysfunction were identified as independent risk factors for TR progression. Those patients seem to be more likely to develop significant late TR and, therefore, should be more aggressively treated with a concomitant tricuspid annuloplasty. In our series, most of the cases of progression or recurrence of significant TR occurred in patients without reverse LV remodeling at follow-up. That explains why patients with TR progression had significantly lower LV ejection fraction, higher MR recurrence, higher pulmonary hypertension, and RV dysfunction at follow-up compared with those without TR worsening. Our results are in line with previous studies that have reported an association between increased systolic pulmonary artery pressure and worse TR or higher rate of tricuspid repair failure [15].

As previously reported [9], pacemaker leads can be responsible for TR worsening, and this was probably (although not certainly) the case in 8 of our patients. Still, in the remaining 15 patients with progression of TR, worsening of TR occurred before pacemaker insertion or in the complete absence of any transtricuspid lead. As a matter of fact, the rate of TR progression was similar in patients with and without ICD-CRT or pacemaker implantation at follow-up. The fact that so many DCM patients require ICD-CRT insertion after MV surgery, potentially further jeopardizing the competence of the tricuspid valve, probably represents another reason for a more aggressive approach toward tricuspid valve repair.

Finally we also decided to enroll in this observational long-term study 20 patients with no TR at baseline. In fact, 45% of these patients showed a progression of TR of at least two grades at follow-up, confirming that other factors should be considered during the decision-making process.

Study Limitations

This study does have all the limitations of every retrospective analysis. The relatively small number of patients included needs to be underlined, but despite that, this series remains one of largest reported so far in the setting of DCM. We are absolutely aware of the fact that the jet area to right atrial area method that was used to quantify TR has important limitations, such as being influenced by loading conditions, machine settings, and eccentricity of the regurgitant jets. Measurements of RV systolic pressure may be limited by the angle of incidence encountered; therefore it could underestimate the Doppler profile of the TR jet and the TR grade. No consistent, accurate data were available preoperatively and at followup regarding right atrial and RV dimensions, tricuspid annular size, and degree of leaflet tethering.

Conclusions

In conclusion, a significant number of DCM patients with no TR or with mild-to-moderate TR left untreated at the time of mitral repair show important TR at follow-up. In this setting, a more aggressive policy should be used, taking into consideration the degree of annular dilatation and the function of the right ventricle and not simply the grade of TR.

References

- 1. Vahanian A, Alfieri O, Andreotti F, et al, Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology (ESC); European Association for Cardio-Thoracic Surgery (EACTS). Guidelines on the management of valvular heart disease (version 2012). Eur Heart J 2012;33:2451–96.
- 2. Nishimura RA, Otto CM, Bonow RO, et al, American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014;63:e57–185.

- **3.** Dreyfus GD, Corbi PJ, Chan KM, Bahrami T. Secondary tricuspid regurgitation or dilatation: which should be the criteria for surgical repair? Ann Thorac Surg 2005;79:127–32.
- 4. Colombo T, Russo C, Ciliberto GR, et al. Tricuspid regurgitation secondary to mitral valve disease: tricuspid annulus function as guide to tricuspid valve repair. Cardiovasc Surg 2001;9:369–77.
- 5. Van de Veire NR, Braun J, Delgado V, et al. Tricuspid annuloplasty prevents right ventricular dilatation and progression of tricuspid regurgitation in patients with tricuspid annular dilatation undergoing mitral valve repair. J Thorac Cardiovasc Surg 2011;141:1431–9.
- 6. Izumi C, Iga K, Konishi T. Progression of isolated tricuspid regurgitation late after mitral valve surgery for rheumatic mitral valve disease. J Heart Valve Dis 2002;11:353–6.
- 7. Benedetto U, Melina G, Angeloni E, et al. Prophylactic tricuspid annuloplasty in patients with dilated tricuspid annulus undergoing mitral valve surgery. J Thorac Cardiovasc Surg 2012;143:632–8.
- 8. Navia JL, Brozzi NA, Klein AL, et al. Moderate tricuspid regurgitation with left-sided degenerative heart valve disease: to repair or not to repair? Ann Thorac Surg 2012;93:59–67.
- 9. McCarthy PM, Bhudia SK, Rajeswaran J, et al. Tricuspid valve repair: durability and risk factors for failure. J Thorac Cardiovasc Surg 2004;127:674–85.
- **10.** De Bonis M, Lapenna E, Sorrentino F, et al. Evolution of tricuspid regurgitation after mitral valve repair for functional mitral regurgitation in dilated cardiomyopathy. Eur J Cardiothorac Surg 2008;33:600–6.

- **11.** Matsunaga A, Duran CM. Progression of tricuspid regurgitation after repaired functional ischemic mitral regurgitation. Circulation 2005;112(9 Suppl):I453–7.
- **12.** Braunwald NS, Ross J Jr, Morrow AG. Conservative management of tricuspid regurgitation in patients undergoing mitral valve replacement. Circulation 1967;35(4 Suppl): I63–9.
- Duran CM, Pomar JL, Colman T, Figueroa A, Revuelta JM, Ubago JL. Is tricuspid valve repair necessary? J Thorac Cardiovasc Surg 1980;80:849–60.
- Kaul S, Tei C, Hopkins JM, Shah PM. Assessment of right ventricular function using two-dimensional echocardiography. Am Heart J 1984;107:526–31.
- **15.** Fukuda S, Gillinov AM, McCarthy PM, et al. Determinants of recurrent or residual functional tricuspid regurgitation after tricuspid annuloplasty. Circulation 2006;114(1 Suppl): I582–7.
- **16.** Chan V, Burwash IG, Lam BK, et al. Clinical and echocardiographic impact of functional tricuspid regurgitation repair at the time of mitral valve replacement. Ann Thorac Surg 2009;88:1209–15.
- **17.** Yilmaz O, Suri RM, Dearani JA, et al. Functional tricuspid regurgitation at the time of mitral valve repair for degenerative leaflet prolapse: the case for a selective approach. J Thorac Cardiovasc Surg 2011;142:608–13.
- **18.** Calafiore AM, Gallina S, Iacò AL, et al. Mitral valve surgery for functional mitral regurgitation: should moderate-or-more tricuspid regurgitation be treated? a propensity score analysis. Ann Thorac Surg 2009;87:698–703.