Percutaneous Mitral Valve Repair Preserves Right Ventricular Function

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Background: Chronic mitral regurgitation (MR) often leads to diminished right ventricular (RV) function due to long-standing pressure and volume overload. Surgical intervention often adds to the preexisting RV dysfunction. Percutaneous mitral valve (MV) repair can reduce MR, but to what extent this affects the right ventricle is unknown.

Methods: Consecutive patients scheduled for percutaneous MV repair using the MitraClip system underwent transthoracic echocardiography at baseline and at 1- and 6-month follow-up. RV systolic function was evaluated using five echocardiographic parameters. RV afterload was evaluated using systolic pulmonary arterial pressure and the mean MV pressure gradient. Residual MR was defined as grade ≥ 3 and mitral stenosis (MS) as a mean MV pressure gradient ≥ 5 mm Hg.

Results: Sixty-eight patients (52% men; mean age, 75 $\pm$ 10 years) were included. Six months after MitraClip implantation, there were no significant changes in any of the RV parameters. MR decreased ($P < .01$) and the mean MV pressure gradient increased during follow-up (2.3 $\pm$ 1.4 mm Hg at baseline vs 4.5 $\pm$ 2.7 mm Hg at 6 months, $P < .01$). Patients with both residual MR and MS 6 months after MitraClip implantation showed significantly higher systolic pulmonary arterial pressure values ($P < .01$) and lower New York Heart Association functional classes ($P < .01$) compared with patients without residual MR or MS.

Conclusions: Percutaneous MV repair, in contrast to surgical repair or replacement, does not negatively affect RV function. After repair, RV afterload and New York Heart Association functional class are improved in the case of successful repair but adversely affected in the presence of both residual MR and MS. (J Am Soc Echocardiogr 2014;27:1098-106.)

Keywords: Percutaneous mitral valve repair, MitraClip, Right ventricular function, Mitral regurgitation, Mitral stenosis, Transthoracic echocardiography

Right ventricular (RV) dysfunction is an important predictor of survival and exercise capacity in various cardiovascular diseases.[1-4] In patients with chronic severe mitral regurgitation (MR), RV function may be impaired because of the long-standing pressure overload caused by the increase in left atrial pressure and changes in the pulmonary vasculature. This gives rise to remodeling of the right ventricle, which leads to decreased contractile performance and RV dysfunction that affect patient prognosis. If concomitant tricuspid regurgitation (TR) is present, this volume overload may further add to RV dysfunction.[5-9] Without intervention, symptomatic patients with severe MR have an annual mortality rate of $\geq$5%.[10]

After surgical repair or replacement of the mitral valve (MV), the right ventricle plays an important role in the postoperative course and functional recovery.[8,11,12] RV impairment after MV replacement significantly increases 5-year mortality compared with patients without right heart failure.[13] Other studies have shown that especially during the early postoperative period, there is a pronounced decrease in RV function, with only partial recovery even after long-term follow-up.[14-16]

Percutaneous MV repair is currently considered a feasible and safe procedure to reduce MR in patients not suitable for surgical MV repair or replacement.[17-23] Previous studies have shown the beneficial effect of percutaneous MV repair on left ventricular (LV) remodeling and clinical outcomes, along with a reduction in the rate of major adverse events compared with surgery.[17,24,25] However, data on how percutaneous MV repair affects RV function and RV remodeling are lacking. In the present study, RV function and pulmonary pressures after

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Abbreviations

DTI-S′ = Peak systolic velocity of the lateral tricuspid valve annulus

FAC = Fractional area change

IVA = Myocardial acceleration during isovolumetric contraction

LV = Left ventricular

MR = Mitral regurgitation

MS = Mitral stenosis

MV = Mitral valve

NYHA = New York Heart Association

RA = Right atrial

RIMP = Right ventricular index of myocardial performance

RV = Right ventricular

sPAP = Systolic pulmonary arterial pressure

TAPSE = Tricuspid annular plane systolic excursion

TR = Tricuspid regurgitation

percutaneous MV repair with the MitraClip system (Abbott Laboratories, Abbott Park, IL) were assessed in a cohort of patients with high surgical risk.

METHODS

Study Population

Data from consecutive patients who were referred to our tertiary center between May 2009 and September 2013 for percutaneous MV repair with the MitraClip system were prospectively collected in a dedicated database. All patients gave written informed consent to their data being collected and used, per the ethical guidelines of the institution. Before MitraClip implantation, all patients had symptomatic severe MR and were denied surgical treatment and accepted for percutaneous treatment by our multidisciplinary heart team. The MitraClip system and the implantation procedure have been described previously.[17] In this retrospective analysis, all patients in whom MitraClip implantation was not successful and those without analyzable baseline or follow-up echocardiograms were excluded.

Echocardiographic Examination

All included patients underwent transthoracic echocardiography before MitraClip implantation and after 1 and 6-month follow-up, performed by experienced and European Society of Cardiology–certified sonographers, using high-quality commercially available ultrasound systems (iE33 [Philips Medical Systems, Andover, MA] or Vivid 7 and 9 [GE Healthcare, Milwaukee, WI]). Echocardiographic recordings were made with a 1.6- to 3.2-MHz transducer (System 7; GE Healthcare), digitized, and analyzed offline. All views were obtained according to the recommendations of the American Society of Echocardiography.[26]

RV function was assessed using five different echocardiographic measurements: tricuspid annular plane systolic excursion (TAPSE), peak systolic velocity of the lateral tricuspid valve annulus (DTI-S′), the RV index of myocardial performance (RIMP), RV fractional area change (FAC), and myocardial acceleration during isovolumetric contraction (IVA).[26,27] TAPSE was measured by placing an M-mode cursor through the lateral tricuspid valve annulus in the apical four-chamber view and measuring the total systolic excursion distance of the tricuspid annulus. DTI-S′ was measured by spectral Doppler tissue imaging within an apical four-chamber view. The pulsed Doppler sample volume was placed in either the tricuspid annulus or the middle of the basal segment of the RV free wall, to enable measurement of the instantaneous spectrum of velocities and to allow the determination of peak velocities. RIMP was calculated as the sum of isovolumetric contraction and relaxation times divided by RV outflow ejection time. RIMP measurements were taken using the spectral tissue Doppler method, whereby all time intervals are measured from a single beat by pulsing the tricuspid annulus. RV FAC was calculated as (RV end-diastolic area − RV end-systolic area)/RV end-diastolic area × 100. These measurements were obtained by tracing the RV endocardium in both systole and diastole from the annulus, along the free wall (beneath the trabeculations) to the apex, and then back to the annulus, along the interventricular septum. IVA was measured by dividing the myocardial velocity during isovolumetric contraction by the time until the peak velocity of this wave and was measured by Doppler tissue imaging at the lateral tricuspid annulus. For the calculation of IVA, the onset of myocardial acceleration was at the zero crossing point of myocardial velocity during isovolumetric contraction.

The following cutoff values were used for the assessment of RV function on the basis of the echocardiographic indices: TAPSE < 16 mm, RIMP > 0.55, DTI-S′ < 10 cm/sec, RV FAC $< 35\%$, and IVA < 2.2 m/sec^2.[26,28] Furthermore, RV function was divided into normal and impaired categories using these cutoff values, and the presence of impaired RV function was defined when at least two major criteria were present or one major criterion combined with two minor criteria. The following parameters were considered major criteria for impaired RV function on the basis of echocardiographic indices: TAPSE, RIMP, and DTI-S′. Minor criteria for impaired RV function were RV FAC and IVA.[26,28]

Values of TAPSE are suggested to be load dependent, so these values were not taken into account if moderate or severe TR was present.[29]

Systolic pulmonary arterial pressure (sPAP) was calculated using RV systolic pressure derived from continuous-wave Doppler interrogation of TR, with the addition of right atrial (RA) pressure estimated with measurement of inferior vena cava size and collapsibility.[26] No patient had two-dimensional or Doppler evidence of pulmonary valve stenosis or RV outflow tract obstruction. TR was qualitatively graded using color-flow Doppler according to American Society of Echocardiography guidelines as follows: normal or trivial (grade 1), mild (grade 2), moderate (grade 3), or severe (grade 4).[26]

Other echocardiographic parameters included LV end-diastolic diameter, LV end-systolic diameter, LV end-diastolic volume, and LV end-systolic volume. LV ejection fraction was measured by using the biplane Simpson's method. By using the quantified length and area measurements, the left atrial and RA volumes were calculated. Atrial diastole was determined by selecting the last frame in ventricular systole before MV opening. The long-axis lengths of the left and right atria were defined by measuring the distance from the center of the mitral annulus to the posterior atrial wall. The atrial endocardial area was traced to exclude the atrial appendages and pulmonary or caval veins. For left atrial volume, we used the biplane area-length formula: volume $= (0.848 \times \text{area}_{4ch} \times \text{area}_{2ch})/[(\text{length}_{4ch} + \text{length}_{2ch})/2]$, where 4ch and 2ch are the four-chamber and two-chamber views, respectively.[30] For RA volume, the monoplane area-length formula was used: volume $= 0.848 \times (\text{area}_{4ch})^2/\text{length}_{4ch}$. RA area was measured in the apical four-chamber view by planimetry. RA area was traced at the end of ventricular systole (largest volume) from the lateral aspect of the tricuspid annulus to the septal aspect, excluding the area between the leaflets and annulus, following the RA endocardium, excluding the inferior and superior vena cava and RA appendage.[26] The mean MV pressure gradient was determined with continuous-wave Doppler. After MitraClip implantation, a mean MV pressure gradient ≥ 5 mm Hg, at a heart rate < 100 beats/min, was considered to indicate significant (iatrogenic) mitral stenosis (MS). Cardiac output was calculated using the LV outflow tract diameter, pulsed Doppler velocity-time integral measurements, and heart rate.

MR grade was based on qualitative and quantitative data by color Doppler and continuous-wave Doppler interrogation of the regurgitant