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ORIGINAL RESEARCH ARTICLE

Echocardiographic analysis of acute effects of percutaneous mitral annuloplasty on severity of secondary mitral regurgitation

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Abstract

Aims Percutaneous mitral annuloplasty (PMA) represents a new treatment option for secondary mitral regurgitation (SMR) being associated with higher morbidity and mortality. The present study was aimed to evaluate whether or not acute effects on SMR severity can quantitatively be assessed after PMA.

Methods and results PMA was performed in 30 patients (mean age 76 ± 9; 37% males) with moderate (n = 14) or severe (n = 16) SMR. Vena contracta (VC), left ventricular (LV) velocity-time-integral ratio ($VTI_{MV/LVOT}$), effective regurgitant orifice area (EROA) by two-dimensional proximal isovelocity surface area (PISA), regurgitant volume ($RVol_{PISA}$) and regurgitant fraction (RF_{PISA}) by PISA, $RVol_{volume}$ and RF_{volume} by LV volume analyses, and parameters describing LV morphology, function, and cardiac performance were assessed by transthoracic echocardiography prior to and after PMA. According to RF_{PISA}/RF_{volume}, 14 patients showed mild, 15 moderate, and 1 severe SMR after PMA. Mean RF, RVol, EROA, VC, and $VTI_{MV/LVOT}$ were lower directly after PMA (RF_{PISA}: 49% ± 11 vs. 34% ± 13, $P < 0.001$; RF_{volume}: 46% ± 10 vs. 34% ± 13, $P < 0.001$; $RVol_{PISA}$: 33 mL ± 13 vs. 25 mL ± 12, $P < 0.001$; $RVol_{volume}$: 28 mL ± 17 vs. 20 mL ± 14, $P < 0.05$; $EROA_{PISA}$: 0.24 cm^2 ± 0.1 vs. 0.19 cm^2 ± 0.1, $P < 0.05$; VC: 5.2 ± 0.1 vs. 4.1 ± 0.2, $P < 0.001$; $VTI_{MV/LVOT}$: 1.9 ± 0.4 vs. 1.6 ± 0.5, $P < 0.05$). Parameters of LV morphology, function, and cardiac performance did not change directly after PMA.

Conclusions PMA leads to a reduction of MR severity in >80% of SMR patients. Acute effects of PMA can quantitatively be assessed by transthoracic echocardiography.

Keywords Secondary mitral regurgitation; Percutaneous mitral annuloplasty; Transthoracic echocardiography

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Introduction

Mitral regurgitation (MR) is the second most common valvular heart disease in European countries.[1] Whereas components of the mitral apparatus are primarily affected in primary MR, secondary mitral regurgitation (SMR) is caused by alterations, for example, ring dilatation, of the left ventricle or left atrium, while mitral leaflets and chordae are structurally normal.[2,3] SMR is associated with an impaired prognosis showing increased mortality in patients after acute myocardial infarction and in heart failure patients.[4,5]

Treatment of patients with SMR is primarily based on optimal medical treatment of heart failure. Cardiac resynchronization therapy should be evaluated according to the current guidelines in selected patients.[3,6] If patients are still symptomatic after optimal conventional treatment, surgical mitral valve repair or interventional mitral valve therapy should be considered.[3,6] Surgical mitral annuloplasty should be considered in patients with an acceptable surgical risk, who have no indication or option for myocardial revascularization and/or concomitant valvular heart diseases.[3] However, surgical valve repair has never been demonstrated to alter the course of the primary disease or to improve long-term

mortality in SMR patients.[3,7] Interventional mitral valve therapy is indicated in high surgical risk patients with severe primary MR and is increasingly established in high surgical risk patients with severe SMR.[8,9] Interventional mitral valve therapy is mostly performed by the MitraClip procedure, followed by percutaneous mitral annuloplasty (PMA) using the Carillon® Mitral Contour System. Recent trials underline the current perception that treating SMR is important. However, contradictory findings to clinical outcomes are still debatable.[10,11]

The principle of PMA with the Carillon Mitral Contour Device is based on the stabilization and diminution of the posterior mitral annulus to improve the coaptation of the mitral leaflets. The circumference of the mitral annulus is aimed to be reduced by cinching the proximal and distal anchor after insertion of the device within the coronary sinus.

Previous studies about the Carillon Mitral Contour Device have shown that clinical symptoms and severity of MR have been improved over time.[12–15] In contrast to previous trials, the aim of the present study was to analyse the acute effects of percutaneous mitral annuloplasty with the Carillon Mitral Contour Device on SMR severity, which were quantitatively assessed by transthoracic echocardiography (TTE).

Methods

General aspects and study design

In the present retrospective study, percutaneous mitral annuloplasty (Carillon Mitral Contour System) was performed in 30 symptomatic adult patients with moderate or severe SMR, while TTE was performed at resting conditions 1 day prior to interventional therapy and at discharge (±3.5 days after intervention in average). Percutaneous mitral annuloplasty as well as baseline and follow-up TTE have been performed by experienced investigators at the Department of Cardiology at the University Hospital Leipzig between 2013 and 2018. All included patients were symptomatic and had at least moderate SMR after optimal medical treatment.[3,6] Indications for interventional therapy using PMA (Carillon Mitral Contour System) have been verified by decisions of the local heart team. Exclusion criteria were defined by the following: cardiogenic shock, previous acute myocardial infarction (<3 months), acute cardiac decompensation due to SMR, primary MR, and other indications for surgery—at least moderate aortic regurgitation and/or severe aortic stenosis and/or relevant mitral stenosis. All patients provided informed consent after full explanation of the purpose and order of all procedures. The study complies with the Declaration of Helsinki, and the study design was approved by the locally appointed ethics committee.

Echocardiography

Transthoracic echocardiography was performed using a GE Vivid 7, Vivid E9, or Vivid E95 system with a M5S phased array probe (GE Healthcare Vingmed Ultrasound AS, Horten, Norway). Echocardiographic analyses were performed using the EchoPac software (version 12.0.1, GE Healthcare Vingmed Ultrasound AS). All investigations and measurements were performed according to national and international recommendations.[3,16–19]

Assessment of left ventricular and left atrial morphology and function

Left ventricular (LV) end-diastolic (LVEDD) and end-systolic diameters (LVESD) were assessed by two-dimensional or M-mode measurements in parasternal long or short axis views, respectively. LV end-diastolic (LVEDV), LV end-systolic (LVESV), and LV total stroke volumes (SV_{tot}) and LV ejection fraction were determined by LV biplane planimetry using the modified Simpson's rule in the apical two-chamber and four-chamber views.[17] Systolic and diastolic sphericity indices (normal values <0.7) were defined as quotients of the LVEDV or LVESV in relation to a sphere, which diameter corresponds to the longitudinal axis of the left ventricle.[18] LV effective stroke volume (SV_{eff}) was assessed by LV outflow tract diameter (D_{LVOT}) proximal to the aortic valve annulus in the parasternal long axis view and the velocity time integral (VTI) of the LVOT pulsed-wave Doppler signal (VTI_{LVOT}) in the apical long axis view according to the equation: $SV_{eff} = 0.785 \times D_{LVOT} \times VTI_{LVOT}$. E/E′ was assessed according to the current recommendations and was used to estimate LV end-diastolic filling pressures.[18] Indexed left atrial (LA) end-diastolic and end-systolic volumes were assessed by LA planimetry in the apical two- and four-chamber views, and LA volume index > 34 mL/m^2 was defined as abnormal based on the current recommendations.[18]

Assessment of mitral valve morphology

Mitral annulus diameter (D_{MV}) was averaged by measurements in the apical long axis and four-chamber views during maximum expansion in early diastole. Coaptation depth was assessed as the smallest distance between the mitral annular plane and the coaptating leaflets in the centre of the mitral valve. Tenting area was determined by planimetry between the mitral annular plane and the mitral leaflets in the apical long axis view.[20]

Assessment of semi-quantitative and quantitative parameters for evaluation of secondary mitral regurgitation

Vena contracta (VC) was averaged by the smallest regurgitant jet width in apical long axis and four-chamber views. $VTI_{MV/LVOT}$ was assessed by VTI measurements of the pulsed-wave Doppler signal of the transmitral inflow and LV outflow. The regurgitant volume ($RVol_{PISA}$) and effective regurgitant orifice area (EROA) were determined by two-dimensional proximal isovelocity surface area (PISA). The regurgitant fraction (RF_{PISA}) was calculated by $RVol_{PISA}$ divided by SV_{tot}. Further, $RVol_{volume}$ was calculated by subtracting SV_{eff} (stroke volume via LVOT) from SV_{tot} (LV planimetry), and RF_{volume} was calculated by $RVol_{volume}$ divided by $SV_{tot.}$[3,19]

Parameters of cardiac performance

SV_{eff}, cardiac output (CO) (CO = SV_{eff} × heart rate), and cardiac index (CI = CO/body surface area) were determined to characterize cardiac performance. Global longitudinal peak systolic strain (GLPSS) was assessed by speckle tracking analysis of the apical long axis, two- and four-chamber views using a 17-segment model of the left ventricle.[17] Peak power index (PPI) was assessed according to the equation: PPI = (systolic blood pressure × LVOT area × maximum LVOT velocity (V_{max}))/LVEDV.[21] Preload recruitable stroke work (PRSW) was determined according to the equation: PRSW = stroke work/LVEDV $-$ K × LVEDV + (1 $-$ K) × LV mass. K is a factor that results from LV mass × 0.0004 + 0.6408, while LV mass was calculated by measuring LVEDD, end-diastolic interventricular septal wall thickness, and LV end-diastolic posterior wall thickness according to the current recommendations.[17,22] Stroke work (SW) describes the SV in relation to blood pressure (RR) conditions and was determined by the equation: SW = SV_{eff} × mean RR [(($RR_{systolic}$ $-$ $RR_{diastolic}$)/3) + $RR_{diastolic}$].[22] Total vascular resistance (TVR) was assessed by the equation: TVR = ($RR_{systolic}$ × 80)/CO.[23] End-systolic wall stress (WS) was determined by the equation: WS = (0.334 × $RR_{systolic}$ × LVESD)/(end-systolic LV posterior wall thickness × (1 + end-systolic LV posterior wall thickness/LVESD)).[24]

Statistical analysis

Data were expressed as mean ± standard deviation. Normality of distribution was tested by Kolmogorov–Smirnov test. Student's t-test and Wilcoxon matched-pairs test were used for comparison of baseline and follow-up parameters. Statistical significance was considered as $P < 0.05$ (confidence interval: 95%). Statistical analyses were performed using SPSS software, version 25 (IBM Deutschland GmbH, Ehningen, Germany).

Results

Patient demographics

Patient characteristics are summarized in *Table 1*. In 19 (63%) patients, SMR was caused by ischaemic cardiomyopathy, and 11 (37%) patients suffered from either dilatative cardiomyopathy or cardiomyopathy due to permanent atrial fibrillation (AF). According to an integrated evaluation using semi-quantitative (VC and $VTI_{MV/LVOT}$) and quantitative echocardiographic parameters (RF_{PISA} and RF_{volume}), 14 (47%) patients had moderate SMR and 16 (53%) patients had severe SMR prior to PMA. PMA was successfully performed in all patients. During the intervention, the cinching of the posterior mitral annulus was documented by guiding with TEE.

Echocardiographic analyses

According to RF_{PISA} and RF_{volume}, 14 (47%) patients showed mild, 15 (50%) moderate, and 1 (3%) severe SMR after interventional therapy. In 25/30 patients (83%), an improvement of SMR severity was achieved after PMA in comparison with baseline measurements (*Figure 1*). Mean RF, RVol, EROA, VC, and $VTI_{MV/LVOT}$ were reduced after treatment of SMR (RF_{PISA}: 49% ± 11 to 34% ± 13, $P < 0.001$; RF_{volume}: 46% ± 10 to 34% ± 13, $P < 0.001$; $RVol_{PISA}$: 33 mL ± 13 to 25 mL ± 12, $P < 0.001$; $RVol_{volume}$: 28 mL ± 17 to 20 mL ± 14, $P < 0.05$; $EROA_{PISA}$: 0.24 cm^2 ± 0.1 to 0.19 cm^2 ± 0.1, $P < 0.05$; VC: 5.2 ± 0.1 to 4.1 ± 0.2, $P < 0.001$; $VTI_{MV/LVOT}$: 1.9 ± 0.4 to 1.6 ± 0.5, $P < 0.05$; *Table 2* and *Figure 2*). *Figure 1* highlights

Table 1 Baseline characteristics

Baseline characteristics	Moderate or severe SMR (n = 30)
Male (%)	11 (37)
Female (%)	19 (63)
Age (years)	76 ± 9
Blood pressure (mmHg)	124 ± 19/65 ± 11
NYHA II (%)	5 (17)
NYHA III (%)	15 (50)
NYHA IV (%)	10 (33)
Coronary artery disease (%)	19 (63)
Diabetes mellitus (%)	10 (33)
Arterial hypertension (%)	25 (83)
Hyperlipidaemia (%)	20 (67)
Obesity (%)	3 (10)
Atrial fibrillation (%)	14 (47)
Pacemaker (%)	6 (20)

SMR, secondary mitral regurgitation; NYHA, New York Heart Association.

Figure 1 Example of a patient with (A and B) moderate secondary mitral regurgitation (SMR) prior to and (C and D) mild SMR after percutaneous mitral annuloplasty. Pre- and Postinterventional evaluations both approaches for the assessment of regurgitant volume (RVol) and regurgitant fraction (RF) [(A and C) proximal isovelocity surface area (PISA); (B and D) volume measurements by biplane left ventricular planimetry by Simpson's rule and Doppler echocardiography] as well as $VTI_{MV/LVOT}$ and vena contracta are considered. LVOT, left ventricular outflow tract; MV, mitral valve; SV_{tot}, total stroke volume; VTI, velocity time integral.

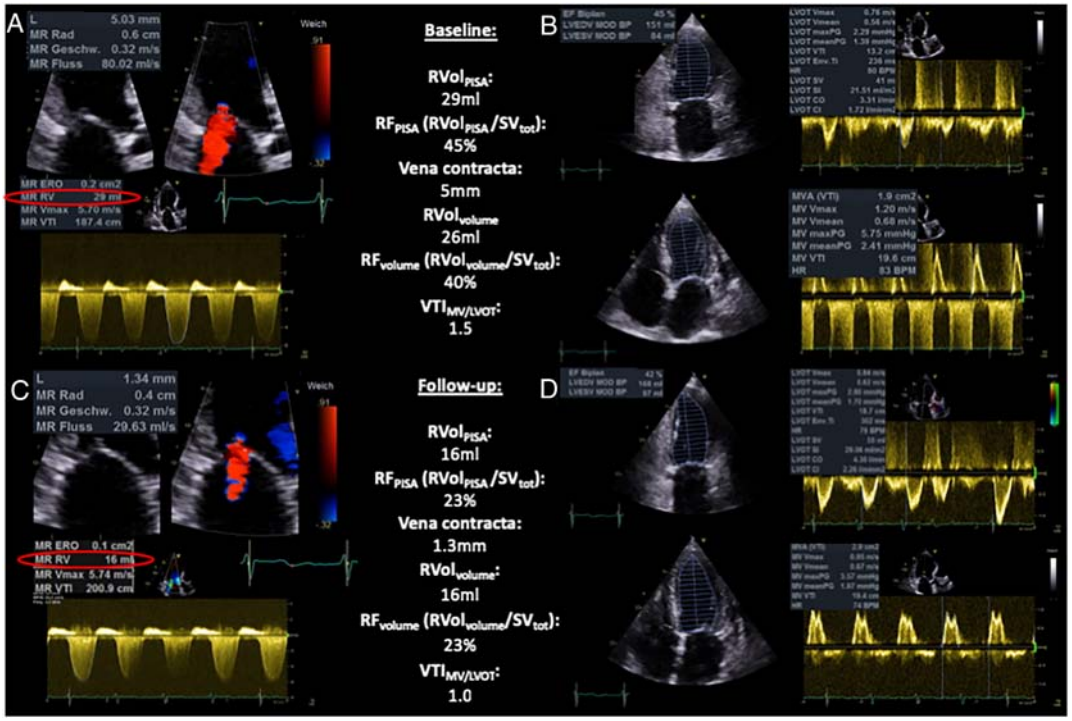

Table 2 Echocardiographic assessment of parameters describing mitral valve morphology and semi-quantitative and quantitative evaluation of secondary mitral regurgitation

Parameters	Mean ± SD prior to annuloplasty (n = 30)	Mean ± SD after annuloplasty (n = 30)	P (< 0.05)
D_{MV} (cm)	3.6 ± 0.6	3.4 ± 0.6	<0.001
Tenting area (cm^2)	2.2 ± 0.7	1.9 ± 0.6	<0.001
Coaptation depth (cm)	1.4 ± 0.4	1.3 ± 0.3	<0.05
$VTI_{MV/LVOT}$	1.9 ± 0.4	1.6 ± 0.5	<0.05
Vena contracta (mm)	5.2 ± 0.1	4.1 ± 0.2	<0.001
$EROA_{PISA}$ (cm^2)	0.24 ± 0.1	0.19 ± 0.1	<0.05
$RVol_{volume}$ (mL)	28 ± 17	20 ± 14	<0.05
RF_{volume} (%)	46 ± 10	34 ± 13	<0.001
$RVol_{PISA}$ (mL)	33 ± 13	25 ± 12	<0.001
RF_{PISA} (%)	49 ± 11	34 ± 13	<0.001

D, diameter; EROA, effective regurgitant orifice area; LVOT, left ventricular outflow tract; MV, mitral valve; PISA, proximal isovelocity surface area; RF, regurgitant fraction; RVol, regurgitant volume; SD, standard deviation; VTI, velocity time integral.

the practical approach of assessing RVol und RF by different echocardiographic approaches. Significant decreases were observed for D_{MV}, tenting area, and coaptation depth (*Table 2*). Parameters characterizing LV and LA morphology, LV remodelling, or cardiac performance did not change directly after PMA (*Tables 3* and *4*).

Subanalysis: non-improvers and improvers

In 5/30 patients (17%), no significant improvement of SMR severity was achieved after PMA. Four patients still showed moderate SMR and one patient severe SMR according to RF_{volume} and RF_{PISA} (RF_{PISA}: 46% ± 9 to 44% ± 10, $P > 0.05$; RF_{volume}: 43% ± 8 to 41% ± 10, $P > 0.05$; $RVol_{PISA}$: 31 mL ± 9 to 29 mL ± 8, $P > 0.05$; $RVol_{volume}$: 26 mL ± 8 to 27 mL ± 9, $P > 0.05$; $EROA_{PISA}$: 0.22 cm^2 ± 0.1 to 0.21 cm^2 ± 0.1, $P > 0.05$; VC: 4.9 ± 0.2 to 4.6 ± 0.2, $P > 0.05$; $VTI_{MV/LVOT}$: 1.8 ± 0.4 to 1.8 ± 0.3, $P > 0.05$). All of these patients had (paroxysmal) AF, whereas no significant differences were obtained with respect to other patients' characteristics.

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Figure 2 Comparison of semi-quantitative and quantitative parameters for evaluation of secondary mitral regurgitation prior to and after percutaneous mitral annuloplasty. (A) RF_{PISA}, (B) $RVol_{PISA}$, (C) vena contracta, and (D) $VTI_{MV/LVOT}$ were significantly lower after percutaneous mitral annuloplasty ($P < 0.05$). RF, regurgitant fraction; RVol, regurgitant volume; VTI, velocity time integral.

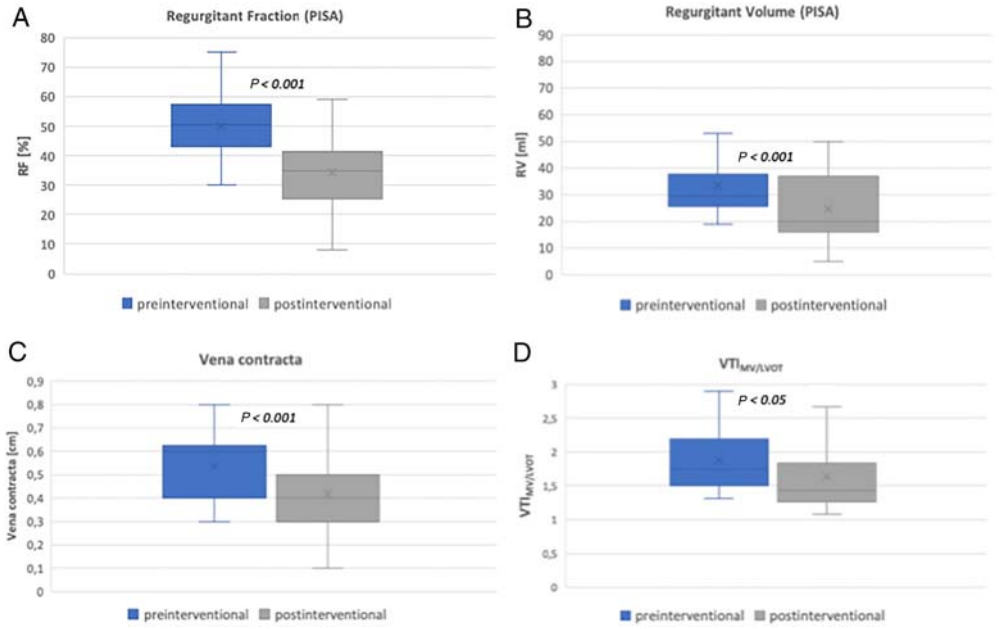

Table 3 Echocardiographic assessment of parameters describing left ventricular and left atrial morphology and function

Parameters	Mean ± SD prior to annuloplasty ($n = 30$)	Mean ± SD after annuloplasty ($n = 30$)	P (< 0.05)
LVEDD (cm)	5.9 ± 1.0	5.9 ± 1.0	0.79
LVESD (cm)	4.6 ± 1.1	4.5 ± 1.2	0.31
Sphericity $index_{diast}$	0.5 ± 0.2	0.5 ± 0.1	0.27
Sphericity $index_{syst}$	0.4 ± 0.2	0.4 ± 0.2	0.32
Indexed LVEDV (mL/m^2)	93 ± 38	95 ± 37	0.70
Indexed LVESV (mL/m^2)	57 ± 31	55 ± 33	0.60
LVEF (%)	42 ± 13	45 ± 15	0.06
SV_{tot} (biplane) (mL)	67 ± 24	72 ± 25	0.17
SV_{eff} (mL)	35 ± 14	45 ± 16	**<0.001**
Indexed LAEDV (mL)	47 ± 22	46 ± 21	0.58
Indexed LAESV (mL)	56 ± 20	55 ± 20	0.65
E/E′	18 ± 5	19 ± 7	0.72

LAEDV, left atrial end-diastolic volume; LAESV, left atrial end-systolic volume; LVEDD, left ventricular end-diastolic diameter; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; LVESV, left ventricular end-systolic volume; SD, standard deviation; SV_{eff}, effective stroke volume; SV_{tot}, total stroke volume.

Table 4 Echocardiographic assessment of parameters describing cardiac performance

Parameters	Mean ± SD prior to annuloplasty ($n = 30$)	Mean ± SD after annuloplasty ($n = 30$)	P (< 0.05)
Global longitudinal strain (%)	−12.2 ± 5.59	−12.3 ± 5.34	0.39
Cardiac output (L/min)	5.1 ± 1.87	5.4 ± 1.80	0.13
Cardiac index ($L/min/m^2$)	2.7 ± 0.88	2.9 ± 0.84	0.13
Total vascular resistance (WU)	2.2 ± 0.84	2.0 ± 0.68	0.06
Stroke work (mmHg × mL)	6234 ± 2435	6362 ± 2492	0.35
End-systolic wall stress (mmHg/cm)	256 ± 444	201 ± 369	0.09
Preload recruitable stroke work (mmHg)	64 ± 21	66 ± 20	0.35
Peak power index (mmHg/s)	406 ± 162	387 ± 157	0.25

SD, standard deviation.

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After exclusion of non-improvers, improvement of SMR severity was observed by the following: RF_{PISA}: 50% ± 10 to 31% ± 11, $P < 0.001$; RF_{volume}: 48% ± 9 to 31% ± 11, $P < 0.001$; $RVol_{PISA}$: 34 mL ± 10 to 22 mL ± 10, $P < 0.001$; $RVol_{volume}$: 31 mL ± 11 to 18 mL ± 10, $P < 0.01$; $EROA_{PISA}$: 0.25 cm^2 ± 0.1 to 0.17 cm^2 ± 0.1, $P < 0.05$; VC: 5.4 ± 0.2 to 3.8 ± 0.2, $P < 0.001$; $VTI_{MV/LVOT}$: 2.0 ± 0.4 to 1.4 ± 0.3, $P < 0.01$.

Safety

After PMA, small pericardial effusions were detected in four patients being haemodynamically not relevant in any patient. During the interventional procedure, compressions of branches of the coronary arteries were observed (n = 3, 10%), whereby the circumflex coronary artery was affected in two cases and the right posterolateral branch in one case. However, after readjustment of the device, no myocardial impairment could be documented by speckle tracking analyses in the intraprocedural TEE. In one patient with AF, pacemaker implantation was necessary due to bradyarrhythmia absoluta after PMA.

Discussion

In the present study:

(1) Percutaneous mitral annuloplasty leads to a reduction of MR severity in >80% of SMR patients directly after interventional therapy (±3.5 days in average).
(2) Reduction of SMR severity can be assessed by morphological (tenting area, D_{MV}, etc.), semi-quantitative (VC, $VTI_{MV/LVOT}$, RVol, etc.), and quantitative (RF) echocardiographic parameters directly after interventional therapy.
(3) Significant differences between both echocardiographic approaches of RVol and RF assessment (PISA and volume measurements) could not be observed.
(4) Percutaneous mitral annuloplasty with implantation of the Carillon Mitral Contour System seems to be a safe and effective procedure.

In contrast to previous trials, this is the first study investigating acute effects of percutaneous mitral annuloplasty. AMADEUS, TITAN, TITAN II, and REDUCE-FMR have analysed long-term effects of PMA showing similar results referring to values of D_{MV}, VC, EROA, and RVol after 1–12 months. However, information about RF and the haemodynamic relevance of SMR is not given in previous studies.[12–15] In comparison with REDUCE-FMR in the present study, only patients with at least moderate SMR were included.[15]

According to the current recommendations, an integrated approach for the assessment of MR is recommended, although PISA and VC are described as favoured approaches under certain circumstances, especially in patients with SMR.[3,16,19] However, PISA is subjected to multiple limitations leading to overestimation or underestimation of MR severity, particularly eccentric jet formations, crescent-shaped regurgitant orifices, or errors due to the PISA formula itself because of dynamic changes of the regurgitant orifice area during the heart cycle.[3,16,19] The assessment of RVol and EROA by PISA does actually not permit estimations of the haemodynamic relevance of SMR, which can only be evaluated by $RVol_{PISA}$ in relation to SV_{tot}. In the present study, severe SMR was characterized by RF considering LV volumes, SV_{tot}, and SV_{eff} and documenting moderate-to-severe and severe SMR prior to interventional therapy (mean $RVol_{PISA}$ of 33 mL ± 13 and mean EROA of 0.24 cm^2 ± 0.1). Both $RVol_{PISA}$ and EROA are in borderline ranges of moderate SMR according to the current recommendations.[3,16] Despite lower values of $RVol_{PISA}$ and EROA in comparison with MITRA-FR (mean EROA: 0.3 cm^2) and COAPT trials (mean EROA: 0.4 cm^2),[10,11] SMR severity can be assumed within comparable ranges with respect to quantification of haemodynamics. In SMR patients being recompensated by optimal medical treatment, it can be expected that CO and CI are within normal ranges prior to and after interventional therapy. The implausibility of echocardiographic parameters (SV_{tot}, SV_{eff}, RVol, and CI values) in recent trials[10,11] makes it extremely difficult to interpret these trials.[25,26]

The assessment of SV_{tot} and SV_{eff} by biplane LV planimetry and Doppler echocardiography enables another approach for calculation of $RVol_{volume}$ and RF_{volume}, especially in case of inconclusive findings by PISA, VC, and etc.[3,16,19] According to the findings of the present study, RVol and RF should be counterchecked by both approaches (LV planimetry/Doppler echocardiography and PISA) with respect to plausibility. However, minor overestimations of $RVol_{PISA}$ in comparison with $RVol_{volume}$ might presumably be due to different SMR jet formations.

Although VC and $VTI_{MV/LVOT}$ were significantly lower after PMA, post-interventional mean $VTI_{MV/LVOT}$ was still >1.4, describing severe MR.[27] The incongruence of assessing SMR severity by semi-quantitative parameters including parameters determined by PISA might be explained by methodological issues, different ultrasound settings, and anatomical variations of mitral ring geometry and LA size. Thus, the present study shows that careful assessment of SMR severity, preferably using RF, seems to be essential and should be considered in heart failure patients with reduced LV ejection fraction.

Prior to interventional therapy, LVEDD, LVESD, and indexed LVEDV and LVESV were higher probably due to LV remodelling. Decreased LV volumes in comparison with Lipiecki *et al*. and Schofer *et al*. could be explained by different

methods measuring LV diameters and LV volumes.[12,14] Further, increased indexed LA end-diastolic and LA end-systolic volumes were observed to be caused by volume overload due to SMR or probably LA remodelling due to atrial fibrilliation and hypertensive and/or ischaemic heart disease, respectively. After PMA, no significant differences of these parameters were observed. This is probably due to the nature of LV and LA reverse remodelling, which is a complex and long-term process being influenced by several haemodynamic and neurohumoral factors.[28] Follow-up investigations of the present cohort are needed to clarify whether or not LV and LA remodelling might be induced after reduction of SMR severity after PMA. However, Siminiak *et al*. have shown that LV reverse remodelling might be induced after PMA after several months.[13]

Subanalyses of non-improvers and improvers might point out that positive acute treatment effects could be observed in severe compared with moderate SMR. It can be assumed that acute non-improvers may be appropriate candidates for other mitral valve therapies, for example, concomitant MitraClip procedure. This sets the stage for follow-up studies that will determine the acute effects of combined or mitral valve procedures.

PMA with implantation of the Carillon Mitral Contour System seems to be a safe and effective procedure. Mild adverse events were observed in $<$20% of the patients. Device associated compressions of marginal branches are known as possible adverse events induced by percutaneous mitral annuloplasty.[12,14,15] Territories of marginal branches might have the highest risk of being affected by the procedure due to its anatomical localization adjacent to the coronary sinus. After percutaneous mitral annuloplasty, TTE should be performed to detect further adverse events, for example, pericardial effusion.[29]

Limitations

The power of the study is limited by the small number of patients, which is mainly due to the selected cohort of high-risk patients. Further, the study focuses on the analysis of acute effects of mitral annuloplasty on SMR severity. In the present study, several echocardiographic approaches are used to assess SMR severity. Data sets of another imaging modality, for example, cardiac magnetic resonance imaging, were usually not available because of several contraindications in these highly selected and often critically ill high-risk patients.

Conclusions

Percutaneous mitral annuloplasty (Carillon) leads to a reduction of SMR severity directly after interventional therapy. The present study proves that these acute effects of percutaneous mitral annuloplasty on SMR severity can conclusively be quantified by different echocardiographic approaches.

Conflict of interest

None declared.

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