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Authors: Piotr Scisło, Adam Rdzanek, Arkadiusz Pietrasik, Janusz Kochman, Grzegorz Opolski

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Short title: Heart after TMVR

Piotr Scisło, Adam Rdzanek, Arkadiusz Pietrasik, Janusz Kochman, Grzegorz Opolski

1st Chair and Department of Cardiology, Medical University of Warsaw, Warsaw, Poland

Correspondence to: Piotr Scisło, MD, PhD, 1st Chair and Department of Cardiology, Medical University of Warsaw, Banacha 1a St., 02-097 Warsaw, Poland, phone: +48 22 5991612, email: scislo@wum.edu.pl

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WHAT’S NEW?

Our research study analyzes the critical problem of mechanical and volumetric changes of the left atrium and both ventricles after successful transscatheter edge-to-edge mitral valve repair (TMVR). We analyze data derived from three-dimensional echocardiographic volumetric assessment and speckle-tracking strains of the left atrium (LA) and the left (LV) and right (RV) ventricles in the pre- and post-operative period following successful TMVR. We found positive volumetric remodeling of the LA and LV without influence on the LV ejection fraction. The procedures had no positive influence on basic RV parameters. Overall, we did not observe beneficial effects of TMVR on the mechanical strain of the heart. Furthermore, the subgroups of patients with a history of atrial fibrillation and coronary artery disease actually showed deteriorated mechanical strain. The important findings of our study need to be taken into consideration when implementing medical treatment following TMVR.
ABSTRACT

Introduction: The function of the heart after successful transcatheter edge-to-edge mitral valve repair (TMVR) remains not fully investigated.

Objectives: The aim of our study was to assess the direct impact of effective TMVR on the strains of the left atrium (LA) and the left (LV) and right (RV) ventricles in patients with functional mitral regurgitation (FMR) due to a coronary artery disease (CAD) or dilated cardiomyopathy.

Patients and methods: Out of a group that successfully underwent TMVR we selected twenty-eight for analysis. The remodeling of the LA, LV, RV and their strains were assessed.

Results: In the short-term follow-up, we found a positive change of the LA’s and LV’s volumes, RV’s dimensions but not the LV ejection fraction (LV-EF). No improvements of strains were observed in pre/post analysis of LV-s, RV-s and LA-s and LA-s Phase 3. We found a deterioration of LA-s Phase 1 and Phase 2 strains in patients with paroxysmal atrial fibrillation (AF) history: LA-s Phase 1 2.5 (5.47 - 0) \( P=0.01 \); LA-s Phase 2 2.2 (-0.82 - 5.47) vs \( P=0.049 \) and CAD: LA-s Phase 1 -3.1 (-0.75 - -6.61) \( P=0.004 \); LA-s Phase 2 (-3.7 - -7.48) \( P=0.01 \).

Conclusions: Our data indicates that no improvement of heart strains can be expected after successful TMVR in the short-term follow-up, while the function of the LA may even deteriorate in some subpopulations.
INTRODUCTION

The interventional repair of functional mitral regurgitation (FMR) is one of the options reserved for patients that are at too high of a risk to undergo cardiac surgery. [1] The most commonly used technique is transcatheter mitral valve repair (TMVR), which involves performing edge-to-edge leaflet stitching with a dedicated device (Mitraclip/Abbott or Pascal/Edwards). Although, the impact of TMVR was extensively reported in two clinical trials, some detailed questions remain unanswered. [2-4] We have knowledge of the impact of TMVR on heart strains in patients with degenerative valve disease. [5-7] However, only a few papers refer to larger FMR patient cohorts according to current standardized consensus. FMR causes persistent volume overload of the left atrium (LA). It leads to adverse remodeling of the LA, its dilatation and dysfunction.[8] Hemodynamic changes cause inflammation, myocyte hypertrophy and interstitial fibrosis of the LA wall. [8]

Currently, echocardiographic functional analysis of the heart is based on two main concepts. The first is based on a comparison of volume changes during the cardiac cycle (eg, ejection fraction of the left ventricle (LV)). Routinely, post-procedural LA/LV volume change is used to assess their remodeling and dysfunction. The second and more sophisticated concept is based on myocardium deformation analysis by speckle tracking. Summarized information from these two methods may give us a better perspective of post-procedural changes to the function of the heart.

Our study presents a holistic approach to the analysis of heart strain after successful TMVR in patients with severe FMR.
PATIENTS AND METHODS

Primary endpoint
To assess the direct impact of effective TMVR on the strains of the LA, LV and right ventricle (RV) in patients with FMR due to coronary artery disease (CAD)/dilated cardiomyopathy (DCM).

Secondary endpoint
To identify potential factors for strain change.

Model
The working model assumed the minimization of all factors other than TMVR that could affect tested parameters (eg, course of the primary disease). Focusing only on TMVR patients, we eliminated potential bias such as form surgical trauma.

Cohort
Retrospectively, we reviewed data of 83 consecutive patients referred to TMVR due to mitral regurgitation (MR) and presenting high risk for cardiac surgery. The eligibility criteria were as follows:

1. FMR successfully treated with TMVR (MitraClip) with reduction of mitral effective regurgitant orifice and volume, which changed the grade of MR by 1 class (from severe to moderate), with stenosis not higher then mild.

2. Availability of 2D and 3D echocardiography images proper for all segments analysis at baseline and follow-up (up-to 7-days); acquired in patients with optimal peri-operative hemodynamic status.

3. Sinus rhythm (SR) at the time of baseline and follow-up echocardiography.

4. Adequate echocardiographic image quality covering all LA, LV and RV walls.
Patients with degenerative MR, paced rhythm, chronic atrial fbrillation (CAF), cardiac surgery other than coronary-artery by-pass grafting (CABG), poor image quality and those with missing/suboptimal images were excluded.

Baseline demographics of the subjects and the information regarding Euroscore I, NYHA class, coronary artery disease (CAD), CABG, prior myocardial infarction (MI), implantation of cardioverter-defibrillator (ICD), history of paroxysmal and persistent AF (AF), chronic obstructive pulmonary disease (COPD) and diabetes mellitus type 2 (DM) was obtained.

For detailed analysis we divided cohort into following subgroups: SR – pts without history of AF, AF – pts with history of AF, CAD – pts with CAD, DCM – pts with dilatated cardiomyopathy.

The details of the TMVR with MitraClip procedure were described previously. [9,10]

All patients submitted a signed information and consent form and the study protocol was approved by the review boards of the participating institutions.

Echocardiography

The clinical characteristics of the cohort are presented in Table 1. The data was acquired on the Philips Epiq 7C/CVXi (Andover, MA, USA) system equipped with transthoracic s5-1/x5-1 and transesophageal x8-2t probes. Off-card analysis was made on Philips QLAB 13 /

Tomtec Autostrain Plug-ins / LV/RV/LA (Andover, MA, USA) software based on speckle tracking. In each patient, the following was measured:

1. LV strain in 3 orthogonal planes acquired in transthoracic echocardiography: apical 4 chamber, apical 3 chamber and apical 2 chamber. Longitudinal strain (LV-GLS) was calculated automatically.

2. RV global strain (free wall and septum) was measured in transthoracic, apical 4 chamber view.
3. LA strains were measured in transthoracic, apical 4 chamber view. Following the guidelines, we measured strains for all 3 LA cycles [8]:
   a. Phase 1 – Reservoir phase. The phase begins at the end of ventricular diastole (mitral closure) and continues until the mitral valve opening. It encompasses the time of left ventricular isovolumetric contraction, ejection, and isovolumetric relaxation
   b. Phase 2 – Conduit phase. From the mitral valve opening through diastasis until the onset of LA contraction in patients in sinus rhythm. In patients with AF it continues until the end of ventricular diastole (mitral valve closure).
   c. Phase 3 – Contraction phase. From the onset of LA contraction until the end of ventricular diastole (mitral valve closure) in patients with sinus rhythm.

For volumetric analysis, Philips software was used: QLAB 13 Dynamic Heart Model. Full dataset for volumetric analysis was acquired from the apical window. We measured LV 3D end-systolic (LV-EDV) and end-diastolic (LV-ESV) volumes and calculated the ejection fraction (LV-EF). From the same acquisition, we calculated the LA volume (LAV) and its index to patient’s surface area (LAVi). We used the vendor setup for Dynamic Heart Model. Automatic analyses were supervised by an experienced echocardiographer and corrected when needed.

**Statistical analysis**

All data was collected in a dedicated Microsoft Access (365) database. Data was tested for normality using Shapiro-Wilk test. Continuous variables were expressed as the mean (SD, lines) or median (interquartile range [IRQ], whiskers) as appropriate. Categorical variables were presented as number (percentage). Unpaired, continuous variables were compared using the t-test or nonparametric Mann–Whitney test, as appropriate. Paired variables (pre- versus post-procedural) were compared with the Wilcoxon signed rank test. Categorical variables
were compared using the Chi-square or Fisher's exact tests, as appropriate. Intraobserver variability for MR parameters was measured in a sample of 10 random patients using an interclass correlation coefficient. The MedCalc for Windows software, version 18.11 (MedCalc Software, Ostend, Belgium) was used for statistical analysis and all reported probability values were 2-tailed. A $P$-value less than 0.05 was considered significant.

**Ethics**

The study was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki, its later amendments, and the approval of the Review Board of the Medical University of Warsaw.

**RESULTS**

Twenty-eight patients met the eligibility criteria (Table 1, Table 2). The mean time of follow-up echocardiography was 2.5 (2-4) days.

**Left ventricle volumes and strain**

After the procedure, we observed a significant reduction of LV-EDV (ml, median (IQR), 12.5 (2.7-17.9) $P=0.007$) and LV-ESV (ml, median (IQR), 14.5 (3.0-20.0); $P=0.001$).

However, there were no significant changes of LVDd and LV-EF (Table 2.) We did not find any significant difference of the LV-GLS in pre- and post-procedural assessment (%, median (IQR), -0.76 (-2.25– -0.76); $P = 0.35$). The above observation extends to the subgroup analysis of AF, SR, CAD and DCM (Figure 1).

**Right ventricle diastolic dimension (RVDd) and strain (RV-s)**

After the procedure, there was significant reduction of the RVDd (cm, median (IQR) 0.2 (-0.06-0.3) 0.04) (Table 2). We did not find any significant differences between RV-s before and after the procedure (%, median (IQR), -2.2 (-7.52-2.92); $P = 0.09$). The above
observation extends to the subgroup analysis of SR, CAD and DCM, except AF’s where we found significant deterioration (%, median (IQR), -4 (-7.52– -1.04); P=0.007) (Figure 2).

**Left atrium volumes and strain**

After TMVR we observed reduction of LAV (ml, median (IQR), 18.5 (5.3–32.8); P<0.001) and LAVi (ml, median (IQR), 10.5 (2.4–23.3); P<0.001). LA pre- and post-procedural dimensions were not changed.

All phases of left atrial strain were analysed separately and globally, including subgroups of AF, SR, CAD and DCM.

a. Phase 1 – Reservoir phase (LA-s P1). Generally, we did not notice any significant differences between pre- and post-procedural LA-s P1 (%, median (IQR), 2.2 (-0.82– 5.87); P = 0.057; Figure 3). The above observation extends to the subgroup analysis of SR and DCM but not to AF (%, median (IQR), 2.5 (0–5.47); P=0.01) and CAD (%, median (IQR), 2.2 (-0.82–5.47) P=0.049).

b. Phase 2 – Conduit phase (LA-s P2). The conduit phase strains differed significantly in global assessment (%, median (IQR), -3.1 (-0.33– -7.48); P = 0.03). But that was determined by the significant increase of deteriorating values in the subgroups of AF (%, median (IQR), -3.1 (-0.75–6.61) P = 0.004) and CAD (% median (IQR), -3.7 (-7.48– -1.25) P = 0.01) (Figure 4).

c. Phase 3 – Contraction phase. We did not observe any significant differences in general and subgroup analysis of the strains in pre- vs post-procedural data (%, median (IQR), -0.14 (-2.58–3.65); P = 0.66) and its subgroups (Figure 5).

**DISCUSSION**

Many papers point to the prognostic value of heart strain analysis in patients with valve disease at 1-year follow-up. [11–14] In general, these papers analyzed mixed cohorts of
patients not only after transcatheter but also surgical repair. In contrast, our work sheds light on strain fluctuation in the perioperative period after transcatheter repair only. Moreover, we analyzed cohort of FMR patients, the study of whom is difficult due to several factors which have impact on clinical and echocardiographic status. In the natural history of CAD/DCM diseases, we observe the process of replacing myocytes by interstitial fibrosis. The increase of interstitial fibrosis compromises elasticity of the tissue and leads to impaired compliance. In parallel, with decreased amount of myocardiocytes, the contractility of cavities is depressed. Impaired compliance and decreased elasticity of the LA lead to its stiffness – in critical situations of even small volume overload, patients are more susceptible to developing hemodynamic failure, even pulmonary edema. [6] The mechanism is universal, and we find it both in the tissue of atria and in the ventricles. In CAD patients, the process is more localized due to regional ischemia, while in DCM patients it is more global.

**Ventricle parameters**

Normalization of LV’s parameters after valvular procedures depends on many factors. Data collected after successful mitral valve surgery due to degenerative regurgitation indicates improvement of the ejection fraction and strains. [12]

However, our cohort was different. The procedures were introduced in the final stage of heart failure in a course of CAD or DCM. As we anticipated, we observed positive remodeling of the LV – a decrease of LV-EDV/LV-ESV after the procedure due to reduction of primary LV volume overload. Contrary to volume reduction, we did not observe any changes of LV-EF, LVDd and LV-GLS after procedures. Similarly, we found positive remodeling of the RV but no improvement of its function was detected. These observations are juxtaposed with the results of the COAPT trial, which suggests the LV’s functional recovery after TMVR takes longer than 2 weeks. This implies no rapid change of pharmacotherapy after successful TMVR. [3] However, the studied procedures may have been made too late, in the end stage of
the disease, after the point-of-no-return when the interstitial fibrosis was too extensive. Our cohort may be more similar to the population of another large study – the MITRA-HF trial - which suggests this kind of therapy does not improve prognosis. [2] The baseline clinical characteristics of our cohort of patients supports this thesis. Our observations could imply that volumetrically significant mitral regurgitation is an effect of remodeling and that the disease is not valvular but myocardial in its origin and consequently the reduction of MR did not stop or reverse the primary disease. [15–18]

**Left atrial parameters**

Adverse LA remodeling is confirmed in patients with MR. [19] It is expected that effective mitral valve disease treatment may result in positive remodeling. [20] We observed short-term reverse LA remodeling (LAV, LAVi) due to preload reduction (post-procedural decrease of MR-vol.) and left-atrial shunt after intra-atrial septum access.

In our observations, the positive remodeling does not go together with functional recovery. Moustafa et al. proved that all phases of atrial strain were depressed in patients with primary chronic MR. [21] Toprak et al. found improvement of strains during LA systole and diastole after the mitral repair in a one-year follow-up. [22] The above observation was substantiated by the work of Ipek et al., but only for patients with normal/high LA strain (≥30%) at baseline.[6] Contrary to the previous authors, [22] they did not register any improvement of LA strain after TMVR in patients with depressed LA strain (<30%) at baseline.

Our work confirms these findings and even goes a step further. We found that in a group with a history of AF, the strain did not improve but deteriorated in the reservoir and conduit phase (Phase 1 and Phase 2, respectively). Similar findings were acquired in the CAD subgroup. However, the deterioration was found only during conduit phase (Phase 2). In this group of patients, the expected scale of degradation of the LA wall tissue may even be greater and the AF can be treated as a symptom of extensive wall damage. [23]
From the clinical point of view, the information may be useful for crafting of perioperative pharmacotherapy when withdrawal of therapy is potentially too aggressive.

Conclusion

In patients with severe FMR with depressed LV function treated with TMVR, we cannot expect improvement of the LA, LV and RV strains. Even worse, we should be prepared for deterioration in subgroups of AF and CAD. However, we observed positive early volumetric remodeling of LV and LA, but without influence on LV-EF. Our data supports the thesis that in the late stage of FMR with CAD/DCM, TMVR treatment is only a palliative procedure. We believe that current guidelines recommend TMVR too late, when there are no myocytes required for functional recovery, only the scar.

Limitations

Due to very restrictive eligibility criteria, we were in a position to only collect data on a small number of patients. However, even studies based on large clinical trials encountered difficulties in extending the research of similar groups (eg, EVEREST II randomized clinical trial based on 38 such subjects). [6, 22] We identified three main problems in collecting the data: (1) for comparative purposes, data should be acquired on the same equipment because sometimes strain analysis is vendor related, [14] (2) acquiring the data on the sinus rhythm can be a challenge for end-stage FMR due to CAD/DCM and (3) due to implanted ICD/CRT, we couldn’t assess the stage of interstitial fibrosis of the ventricles/LA by cardiac magnetic resonance to support the hypothesis of intervention after the point of no return.

Contribution statement: PS was the main author. PS and AR participated in designing the study. PS, AR and AP were responsible for conducting the study. All authors made substantial contribution to drafting, critical revisions and approval of the final version of the manuscript.
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Table 1. Baseline characteristics of study patients

| Characteristics                     | Value                          |
|-------------------------------------|-------------------------------|
| Patients, n                         | 28                            |
| Male sex, n (%)                     | 19 (68)                       |
| Age, y, median (IQR)                | 72.5 (67–77)                  |
| EuroSCORE II, median (IQR)          | 2.45 (2–3.4)                  |
| NYHA functional class, n (%)        |                               |
| II                                  | 12 (43)                       |
| >III                                | 16 (57)                       |
| NTPro-BNP, u, median (IQR)          | 3260 (1858–6066)              |
| History of AF, n (%)                | 20 (7)                        |
| CAD, n (%)                          | 16 (57)                       |
| DCM, n (%)                          | 12 (43)                       |
| Previous CABG, n (%)                | 8 (29)                        |
| Previous MI, n (%)                  | 14 (50)                       |
| ICD, n (%)                          | 16 (57)                       |
| COPD, n (%)                         | 6 (21)                        |
| Diabetes, n (%)                     | 12 (42)                       |

Abbreviations: CABG, coronary-artery bypass graft; CAD, coronary artery disease; COPD, chronic obstructive lung disease; DCM, dilatated cardiomyopathy; ICD, implanted cardioverter-defibrillator; IQR, interquartile range
Table 2. Change of the echocardiographic data (pre-post) after edge-to-edge transcatheter mitral valve repair.

| Parameter                      | Pre-post difference |  \( P \)  |
|-------------------------------|---------------------|-----------|
| MR-ERO, cm^2, median (IQR)    | 0.20 (0.18-0.26)    | <0.001    |
| MR-volume, ml, median (IQR)   | 25.5 (22.0-31.3)    | <0.001    |
| MVA, cm^2, median (IQR)       | 3.3 (2.9-4.0)       | <0.001    |
| MV gradient post, mmHg, median (IQR) | 3 (2-4) | n/a       |
| LV-EDV, ml, median (IQR)      | 12.5 (2.7-17.9)     | 0.007     |
| LV-ESV, ml, median (IQR)      | 14.5 (3.0-20.0)     | 0.001     |
| LV-EF, %, median (IQR)        | 2.0 (-0.6-5.6)      | 0.06      |
| LVDd, cm, median (IQR)        | 0.05 (-0.1-0.26)    | 0.29      |
| LAV, ml, median (IQR)         | 18.5 (5.3-32.8)     | <0.001    |
| LAVi, ml/m^2, median (IQR)    | 10.5 (2.4-23.3)     | <0.001    |
| LA, cm, median (IQR)          | 0.1 (-0.06-0.2)     | 0.38      |
| RV, cm, median (IQR)          | 0.2 (-0.06-0.3)     | 0.04      |

Abbreviations: IQR, interquartile range; LA, left atrium; LAV, left atrial volume; LAVi, indexed left atrial volume; LVDd, left ventricle diastolic dimension; LV-EDV, left ventricle end-diastolic volume; LV-EF, left ventricle ejection fraction; LV-ESV, left ventricle end-systolic volume; MR-ERO, mitral regurgitation effective regurgitant orifice; MR-volume, mitral regurgitation volume; MI, myocardial infarction; MVA, mitral valve area; MV gradient post – postprocedural mean mitral valve gradient; PAF, paroxysmal atrial fibrillation; RV, right ventricle.
FIGURE LEGENDS

**Figure 1.** Change of the left ventricle strain in whole cohort and subpopulations.

**Figure 2.** Change of the right ventricle strain in whole cohort and subpopulations.
Figure 3. Change of the left atrial strain Phase 1 in whole cohort and subpopulations.

Figure 4. Change of the left atrial strain Phase 2 in whole cohort and subpopulations.
Figure 5. Change of the left atrial strain Phase 3 in whole cohort and subpopulations.