Original Research

Optimizing MitraClip Outcomes: The Case for Routine Iatrogenic ASD Closure

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Abstract

Introduction: Iatrogenic atrial septal defect (iASD) resulting from MitraClip procedures may cause volume overload and deterioration of right ventricular (RV) function. The concurrent MitraClip procedure, along with an intervention to close iASD appears to yield a potentially favorable impact on the functioning of the right ventricle.

Aim of the study: The study aims to evaluate the effect of iASD closure with an occluder immediately after the MitraClip procedure on RV function, pulmonary resistance, and right ventricle-pulmonary artery coupling (RV-PAc).

Methods: The study group (ASDc) consisted of consecutive patients who underwent concomitant iASD closure with the Amplatzer occluder (n = 10). The control group (n = 9) comprised patients with iASD left untreated (CT group). RV assessment before MitraClip and during follow-up visits was based on transthoracic echocardiography (TTE).

Results: In the CT group, fractional area change (FAC) increased from 33.3 ± 15.6% to 38.2 ± 14.0%; *P* = .28, and in the ASDc group, from 38.9 ± 11.6% to 40.4 ± 13.7%; *P* = .76. In the CT group, tricuspid annular plane systolic excursion (TAPSE) decreased from 19.2 ± 4.3 mm to 17.3 ± 3.8 mm; *P* = .47, and in the ASDc group from 19.1 ± 6.8 mm to 16.5 ± 6.1 mm; *P* = .04. In the entire group, right ventricular systolic pressure (RVSP) dropped from 52.7 ± 16.0 mmHg to 45.1 ± 8.1 mmHg; *P* = .01. The reduction in RVSP was 11 mmHg in the ASDc group versus 4 mmHg in the CT group (*P* = .35). Pulmonary vascular resistance (PVR) itself did not change significantly before and after the procedure. RV-PAc increased respectively by 36% and 9.75% from baseline values in the ASDc and CT groups.

Conclusion: Closure of the iASD results in a greater reduction in RVSP but also TAPSE. RV-PAc, a parameter unaffected by RV preload, reveals notably improved hemodynamic conditions for RV performance after iASD closure.

Keywords

MitraClip, right ventricle, iatrogenic atrial septal defect, Amplatzer occluder, mitral regurgitation

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Introduction

Transcatheter reduction of mitral regurgitation is a well-established strategy in treating patients with secondary mitral regurgitation and heart failure symptoms.[1] Randomized trials have shown beneficial effects on symptom improvement, reduced hospitalization rates, and mortality.[2] However, some patients experience deterioration of right ventricular function. In the material published by Ledwoch et al,[3] deterioration of right ventricular (RV) function occurred in 20% of patients, with a significantly higher mortality rate in this group of patients compared to those with unchanged or improved RV function after MitraClip procedure. A potential factor with adverse effects on right ventricular function is the presence of a persistent iatrogenic atrial septal defect (iASD), which may be of particular significance when there is a change in the

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hemodynamic conditions in the left atrium associated with the creation of a bi-ostial valve with a reduced opening area, corresponding to the mitral stenosis.[4] Data on the significance of iASD in patients after the MitraClip procedure remain inconclusive. In the experimental model, creating an iASD with a significant left-right leak had little effect on RV function. The factors evaluated were the preload-dependent dP/dt ratio and the preload-independent end-systolic elastance and slope of the preload recruitable stroke work relation, which increased similarly during the dobutamine test in the group of animals regardless of the degree of a leak resulting from iASD.[5] On the other hand, Rommel et al evaluated the effect of chronic volume overload on RV function and its decrease due to the reduction of tricuspid regurgitation. The authors showed that reducing RV volume overload improved RV function and left ventricular filling. As a result, patients experienced an improvement in symptoms of heart failure as assessed by the New York Heart Association functional classification ($P < .01$), a reduction in peripheral edema ($P = .01$), as well as an increase in the 6-minute walk distance test by 20% at 1 month and by 22% at 6 months after the procedure ($P < .01$).[6]

In contrast, Alachkar showed significant RV dilatation in patients with a persistent left-right shunt. Still, during the 2-year follow-up period, the presence of iASD was not shown to translate into worse RV functional assessment parameters, that is, tricuspid annular plane systolic excursion (TAPSE) and, right ventricular systolic pressure (RVSP), as well as hospitalization rates and mortality.[7] On the other hand, in the MITHRAS cohort, patients with iASD had worse clinical outcomes due to more frequent hospitalization resulting from heart failure decompensation. At the same time, the closure of iASD performed 30 days after the MitraClip procedure had no advantage over a further conservative strategy.[8]

To date, it has not been evaluated whether iASD closure immediately after the MitraClip procedure can protect against deterioration of RV function and consequently has a favorable effect on prognosis.

This study evaluates the effect of iASD closure with an occluder immediately after the MitraClip procedure on RV function, pulmonary resistance, and right ventricle-pulmonary artery coupling (RV-PAc).

Material and Methods

Study Design

The current study is single-center, prospective, and unblinded observational study. The study aims to evaluate the effect of iASD closure with an occluder immediately after the MitraClip procedure on RV function, pulmonary resistance, and right ventricle-pulmonary artery coupling (RV-PAc). Every participant enrolled in the study underwent a percutaneous mitral regurgitation reduction procedure with MitraClip (Abbott, Menlo Park, CA, USA), and half had a concomitant iASD closure procedure with an occluder (Amplatzer™ Atrial Septal Occluder Device, Abbott, Chicago USA). The iASD closure procedure was performed in every second patient undergoing MitraClip irrespective of baseline clinical characteristics and preprocedural echocardiographic findings. The decision to perform the iASD closure was not randomized, but rather followed a predefined, alternating pattern of inclusion. The echocardiographic parameters to be analyzed were recorded during the examination performed 24 hours before the procedure and at follow-up visits after 3 and 6 months. The study was conducted under the principles of the Declaration of Helsinki, and the local Bioethics Committee approved the study protocol. Before the procedure, all patients read the details of the study and signed an informed consent.

Study Population

The study population consisted of 19 consecutive patients with heart failure of ischemic nature and severe secondary mitral regurgitation, diagnosed based on transthoracic echocardiography with an Effective Regurgitant Orifice (ERO) $\geq 0.3\ cm^2$, who were previously qualified for the MitraClip procedure based on the mitral valve morphological assessment by transthoracic and transesophageal echocardiography. Inclusion criteria included symptoms of heart failure classified as NYHA functional class II-IV and meeting the anatomical criteria for qualification for the MitraClip procedure. Additionally, patients were required to have at least minimal tricuspid regurgitation to be eligible for the study. Patients were excluded if their left ventricular ejection fraction (LVEF) was either below 20% or above 50%. All patients participating in the study received an ACE inhibitor or ARB, a beta-blocker, and a medication from the aldosterone antagonist class, while they did not receive SGLT2 inhibitors. Furthermore, the pulmonary hypertension observed in certain cases resulted from left-sided heart failure. Only patients who did not necessitate revascularization and received maximal tolerated pharmacological management for heart failure were considered eligible candidates for the MitraClip procedure. In cases where symptoms of heart failure persisted despite the administration of optimal pharmacological treatment, resynchronization pacemaker implantation had been previously employed as a therapeutic intervention (5 patients in ASDc group and 3 in CT group, $P = .46$). Of the enrolled patients, the study group (group ASDc) consisted of patients who underwent concomitant iASD closure with the Amplatzer occluder ($n = 10$), while the remaining patients ($n = 9$) with iASD defect left constituted the control group (CT group). The compared groups did not differ in age (66.9 ± 9.66 years vs 67.0 ± 9.84 in the ASDc and CT groups, respectively, $P = 1.00$). In the studied group, atrial fibrillation was present in 10 patients (62.5%): 2 (33.3%) in the ASDc group and 8 (80%) in the CT group, $P = .06$. Follow-up visits were conducted after 3 and 6 months. During the follow-up visits, transthoracic and transesophageal echocardiography, and cardiovascular functional tests (6-minute walk test and ergospirometry test) were performed.

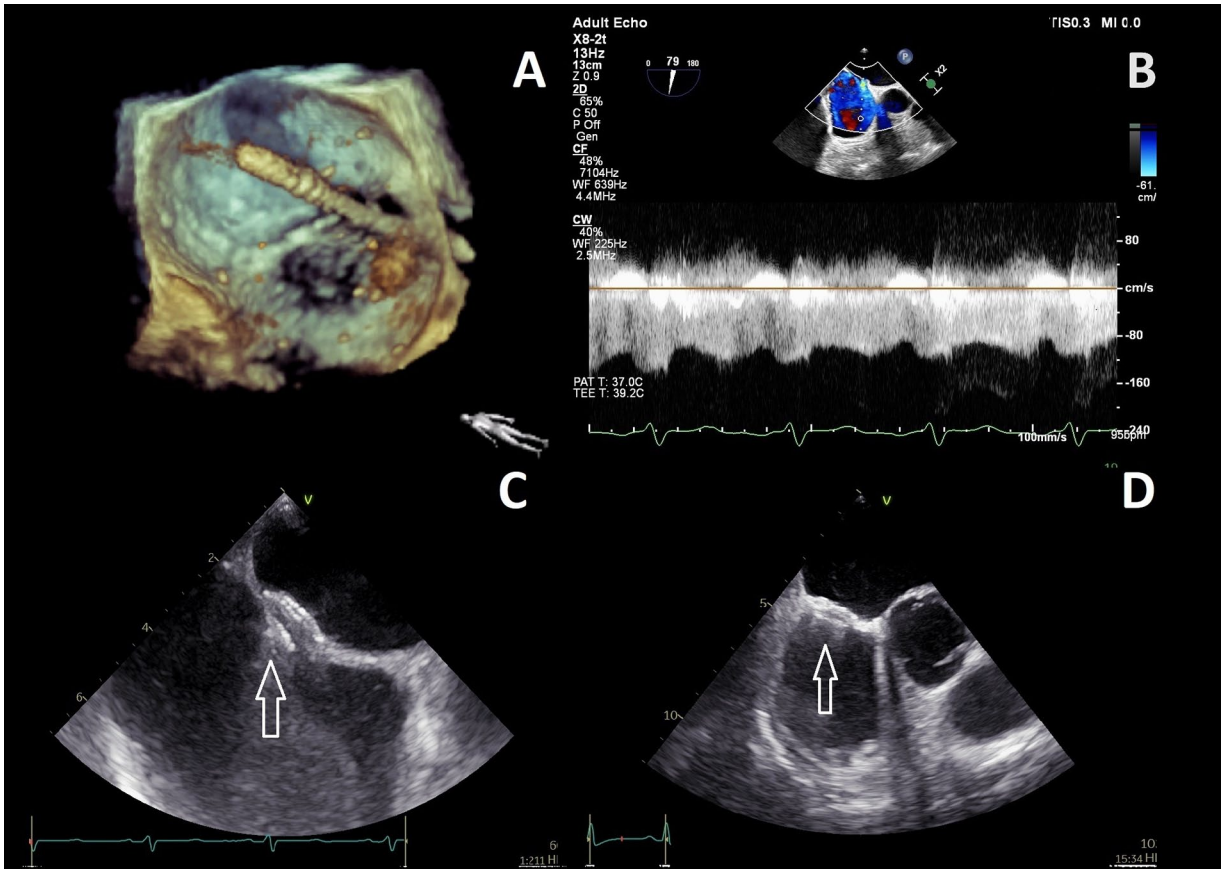

Figure 1. Development of iatrogenic atrial septal defect resulting from the MitraClip, along with its subsequent management involving the employment of the Amplatzer occluder for closure. (A) MitraClip system catheter passing through the atrial septum. (B) Doppler spectra of flow through an iatrogenic atrial septal defect. (C and D) Visualization of the implanted occluder on transesophageal echocardiography.
Arrows indicate the occluder.

Closure of the iASD

The iASD closure procedures were performed immediately after the implantation of the MitraClip. A guidewire was inserted into the left superior pulmonary vein through the MitraClip system vascular catheter. The catheter was then replaced with a catheter designed for Amplatzer occluder delivery. A 7 mm Amplatzer occluder was pledged to close the iASD in all cases. The process of iASD formation as a result of the MitraClip procedure, followed by its closure using the Amplatzer occluder, is shown in Figure 1. A stability test was performed before the device was released. Implantation of the occluder was performed under fluoroscopy and transesophageal echocardiography. The absence of flow through the inter-atrial septum was confirmed by transesophageal echocardiography using the color Doppler.

Echocardiography

RV assessment before MitraClip and during follow-up visits was based on transthoracic echocardiography (TTE). Those studies were performed on a GE VIVID E95 (General Electric Healthcare, Horten Norway) using a 2.5 MHz transducer. Images were recorded in standard views and according to the American Society of Echocardiography Guidelines. From the recorded 2D images and Doppler spectrum, information was collected on the right and left ventricular dimensions, right systolic function (TAPSE, fractional area change (FAC)), left ventricular ejection fraction (LV-EF), RVSP, pulmonary vascular resistance (PVR), echo-derived right ventricular efficiency (RVEe), RV-PAc and tricuspid regurgitation range. The PVR was calculated using the tricuspid regurgitation peak velocity to RV outflow tract velocity-time integral ratio, and RVEe was defined as TAPSE to PVR ratio. RV-PAc was defined as TAPSE to systolic pulmonary artery pressure (sPAP).

Statistical Analysis

Mean values and standard deviation were reported for continuous variables. Parameter values in both groups were compared using the Mann-Whitney test. Comparison analyses between measurements in the same patients were performed using the Wilcoxon matched-pairs signed rank test.

Table 1. Baseline characteristics of study and control groups.

Parameter	CT group Mean ± SD	ASDc group Mean ± SD	*P* level
Age (years)	67.0 ± 9.84	66.9 ± 9.66	1.00
Female, n (%)	1 (9.1%)	1 (12.5%)	.81
BMI	24.2 ± 8.7	26.8 ± 3.3	.42
Hypertension, n, (%)	6 (60%)	4 (44.4%)	.49
Diabetes, n (%)	2 (20%)	5 (55.6%)	.11
Heart failure NYHA II, n (%)	6 (60%)	5 (55.6%)	.85
Heart failure NYHA III, n (%)	4 (40%)	4 (44.4%)	
RV diameter in long parasternal axis (mm)	32.1 ± 7.4	35.8 ± 4.3	.16
RV diameter in four-chamber view (mm)	36.8 ± 8.2	34.7 ± 11.2	.65
FAC (%)	33.2 ± 15.6	38.9 ± 11.6	.36
TAPSE (mm)	19.2 ± 4.3	19.1 ± 6.8	.59
RVSP (mmHg)	50.4 ± 9.1	54.2 ± 20.2	1.00
PVR (WU)	3.3 ± 0.8	3.9 ± 1.8	.87
RV-PAc (mm/mmHg)	0.4 ± 0.1	0.4 ± 0.2	.27
RVEe (mm/WU)	6.9 ± 2.7	9.4 ± 5.5	.49
LV EDD (mm)	61.9 ± 6.3	65.3 ± 6.1	.35
LV ESD (mm)	52.2 ± 7.1	55.7 ± 6.6	.74
LV EDV (ml)	195.7 ± 80.1	197.5 ± 50.0	.49
LV ESV (ml)	134.7 ± 61.8	136.7 ± 36.3	.51
LV EF (%)	33.1 ± 9.0	31.2 ± 9.1	.74
NT-proBNP (ng/ml)	2058.8 ± 2239.4	2458.8 ± 1736.9	.45
6-min walking test distance (m)	344.6 ± 130.3	239.7 ± 148.3	.18
O_2 consumption (ml/kg)	14.2 ± 6.9	17.3 ± 6.2	.39

Abbreviations: EDD, end-diastolic dimension; EDV, end-diastolic volume; EF, ejection fraction; ESD, end-systolic dimension; ESV, end-systolic volume; FAC, fractional area change; LV, left ventricle; NT-proBNP, N-terminal pro-B-type natriuretic brain peptide; PVR, pulmonary vascular resistance; RV, right ventricle; RVEe, echo-derived right ventricular efficiency; RV-Pac, right ventricle-pulmonary artery coupling; RVSP, right ventricular systolic pressure; TAPSE, tricuspid annular plane systolic excursion.

A significance level P of $<.05$ was considered significant. Statistical calculations were performed using the JAMOVI Version 2.3 computer software (the Jamovi project (2023), Sydney, Australia).

Results

Selected echocardiographic parameters and cardiovascular functional evaluation results for the ASDc study group and the CT control group are listed in Table 1. Patients in both groups did not significantly differ in the degree of left and right ventricular dilatation and ejection fraction. Patients in the ASDc group had non-significantly higher N-terminal pro-B-type natriuretic brain peptide (NT-proBNP) values and worse results on the 6-minute walking test. After MitraClip, NT-proBNP decreased from 2458.8 ± 1736.9 ng/ml to 1939.1 ± 1858.0 ng/ml ($P=.18$) in the ASDc group, while it decreased from 2058.8 ± 2239.4 ng/ml to 1723.4 ± 1468.8 ng/ml ($P=.56$) in the CT group.

Baseline mitral regurgitation grade 4+ was present in 17, and grade 3+ in 2 patients. Significant differences were found in the quantitative assessment of mitral regurgitation. Effective regurgitant orifice (ERO) was significantly higher in the ASDc group compared to the CT group (0.3 ± 0.1 vs 0.4 ± 0.1, $P=.02$). The degree of disproportionality of the mitral regurgitation, expressed by the ERO to end-diastolic volume ratio, was also higher in the ASDc group but irrelevantly compared to the CT group (2.3 ± 1.1 vs 1.7 ± 0.5, $P=.32$). After treatment, 15 patients achieved a reduction of at least 2 degrees, 3 patients of 1 degree, and one had no reduction. There were no differences in the degree of mitral regurgitation reduction between the ASDc and CT groups ($P=.73$).

Among the included patients, dominated those with trace and mild tricuspid regurgitation. Severe tricuspid regurgitation rated at 4+ was present in only 2 patients before the MitraClip procedure, in both cases in the ASDc group. These patients experienced a reduction in tricuspid regurgitation to 2+ after the procedure. Tricuspid regurgitation increased after the MitraClip procedure in two cases, with tricuspid regurgitation rated 2+ at most after the procedure (Figure 2). A reduction in tricuspid regurgitation by at least 1+ was observed in 3 patients (50%) in the ASDc group and 3 patients (30%) in the CT group during the 6-month follow-up, with a P-value of .42. Although the difference was not statistically significant, there was a trend toward improvement in both groups, with a more pronounced effect observed in patients with closed iASD.

The results of the echocardiographic examination after 6 months are presented in Table 2. In the CT group, FAC increased from 33.3 ± 15.6% to 38.2 ± 14.0%; $P=.28$, and in the ASDc group, from 38.9 ± 11.6% to 40.4 ± 13.7%; $P=.76$. On the other hand, concerning TAPSE, a decreasing

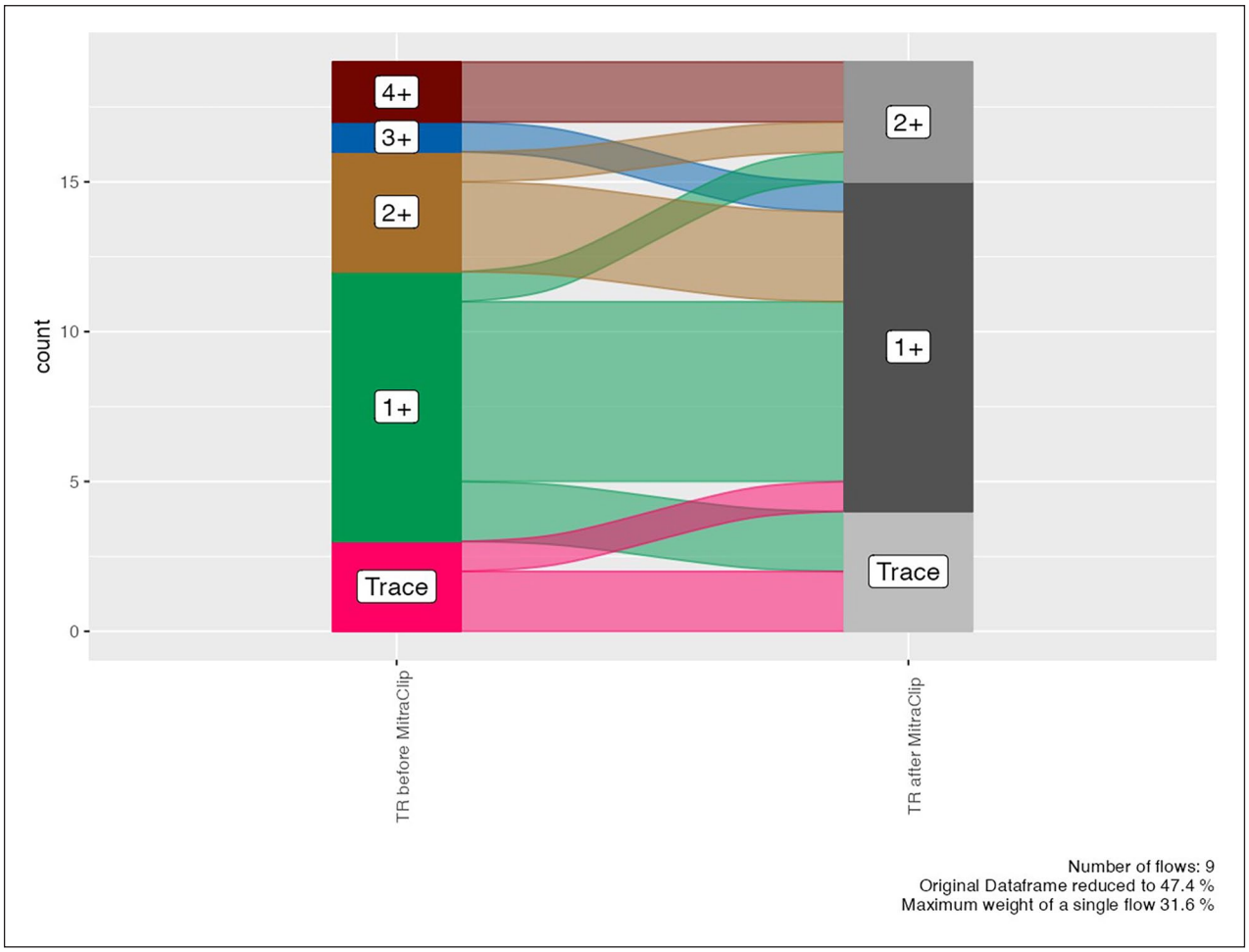

Figure 2. Change in tricuspid regurgitation after MitraClip procedure.
Abbreviation: TR, tricuspid regurgitation.

Table 2. Echocardiographic assessment at 6-month follow-up visit.

	CT group	ASDc group	
Parameter	Mean ± SD	Mean ± SD	*P* level
RV diameter in long parasternal axis (mm)	33.9 ± 5.9	30.5 ± 5.5	.52
RV diameter in four-chamber view (mm)	37.8 ± 10.9	30.3 ± 3.8	.10
FAC (%)	32.2 ± 11.5	44.3 ± 11.4	.23
TAPSE (mm)	16.6 ± 3.2	14.5 ± 5.3	.57
PVR (WU)	3.0 ± 0.7	3.3 ± 1.8	.63
RV-PAc (mm/mmHg)	0.4 ± 0.3	0.5 ± 0.1	.08
RVEe (mm/WU)	6.9 ± 3.8	5.8 ± 6.3	.65
RVSP (mmHg)	39.9 ± 8.6	43.8 ± 8.2	.46
LV EDD (mm)	63.3 ± 6.3	64.8 ± 1.7	.77
LV ESD (mm)	55.3 ± 6.7	56.8 ± 2.4	.62
LV EDV (ml)	172.1 ± 79.3	203.5 ± 50.7	.19
LV ESV (ml)	110.2 ± 36.7	133.5 ± 55.9	.63
LV EF (%)	30.8 ± 5.3	32.5 ± 10.7	.99
NT-proBNP (ng/ml)	2068 ± 1696	3086 ± 2940	.84
6-min walking test distance (m)	367 ± 99.2	425 ± 93.8	.23
O_2 consumption (ml/kg)	17.6 ± 4.6	17.9 ± 8.4	.76

Abbreviations: EDD, end-diastolic dimension; EDV, end-diastolic volume; EF, ejection fraction; ESD, end-systolic dimension; ESV, end-systolic volume; FAC, fractional area change; LV, left ventricle; NT-proBNP, N-terminal pro-B-type natriuretic brain peptide; PVR, pulmonary vascular resistance; RV, right ventricle; RVEe, echo-derived right ventricular efficiency; RV-Pac, right ventricle-pulmonary artery coupling; RVSP, right ventricular systolic pressure; TAPSE, tricuspid annular plane systolic excursion.

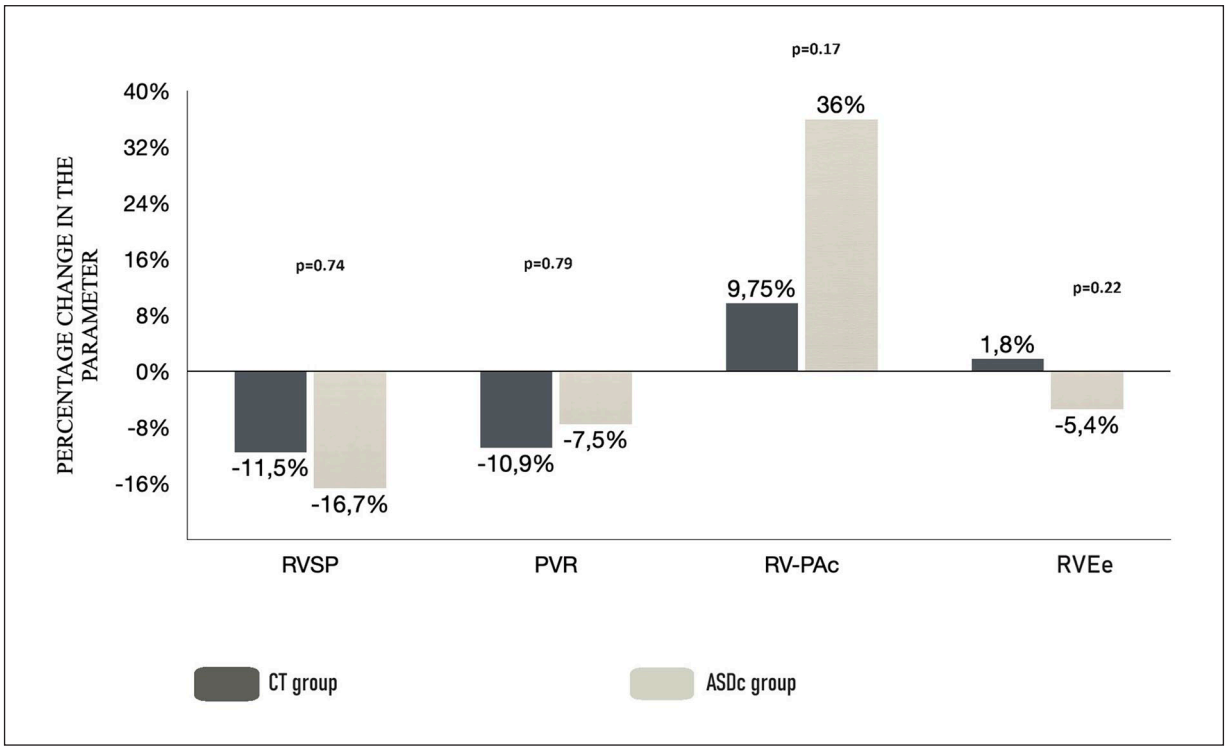

Figure 3. Percentage change in RVSP, PVR, TAPSE/sPAP, and RVEe.
Abbreviations: PVR, pulmonary vascular resistance; RVEe, echo-derived right ventricular efficiency; RVSP, right ventricular systolic pressure; TAPSE/sPAP, right ventricular-pulmonary artery coupling.

trend after the MitraClip procedure was observed. Such a trend was observed in both groups, but statistically significant differences were observed only in the ASDc group. In the CT group, TAPSE decreased from 19.2 ± 4.3 mm to 17.3 ± 3.8 mm; $P = .47$, and in the ASDc group from 19.1 ± 6.8 mm to 16.5 ± 6.1 mm, $P = .04$. However, after the procedure, there were no significant differences between the groups ($P = .59$). Also, the change in FAC and TAPSE did not correlate with the age of the patients. The Spaerman's rho correlation coefficient was .11 ($P = .67$) for FAC and −.28 ($P = .24$) for TAPSE, respectively.

The MitraClip procedure contributed to marked changes in RVSP. In the entire group, a decrease in RVSP was observed from 52.7 ± 16.0 mmHg before the procedure to 45.1 ± 8.1 mmHg after the procedure ($P = .01$). Values in both groups after MitraClip were similar (44.6 ± 9.7 mmHg vs 45.1 ± 6.0 mmHg; $P = .79$). However, in the ASDc group, the reduction in RVSP averaged 11 mmHg, while in the CT group, it was only 4 mmHg; nonetheless, these differences were insignificant ($P = .35$). Changes in RVSP before and after surgery did not correlate with patient age (Spareman's rho coefficient .06; $P = .81$).

PVR itself before and after the procedure did not change significantly. Before the procedure, PVR in the study population was 3.3 ± 1.3 WU, and the follow-up PVR was 3.0 ± 1.2 WU ($P = .28$). The mean PVR difference was 0.3 WU in the ASDc and CT groups ($P = .23$).

Before the procedure, RV-PAc was similar in both groups (0.4 ± 0.2 mm/mmHg in the ASDc group and 0.4 ± 0.1 mm/mmHg in the CT group; $P = .27$). This parameter's value increased slightly in the CT group to 0.4 ± 0.3 mm/mmHg; $P = .55$. An upward trend occurred in the ASDc group, where RV-PAc increased 36% from baseline values and was 0.5 ± 0.1, $P = .08$ in the follow-up.

In the CT group, RVEe was before MitraClip 6.9 ± 2.7 mm/WU and remained almost unchanged after the procedure (6.9 ± 3.8 mm/WU; $P = .74$). Meanwhile, in the ASDc group, a decreasing trend was evident for RVEe (mean change −0.4 mm/WU; $P = .63$). The percentage change in the values of parameters related to right ventricular function is shown in Figure 3. The percentage changes in parameters related to right ventricular function are presented in Figure 3. The change in RVSP was −11.5% for the CT group and −16.7% for the ASDc group, with a P-value of .74, indicating no statistically significant difference between the groups. The percentage change in PVR was −10.9% for the CT group and −7.5% for the ASDc group, with a P-value of .79, further confirming the absence of a significant difference. The change in RV-PAc was 9.75% for the CT group and 36% for the ASDc group, with a P-value of .17, suggesting a trend toward improvement in the ASDc group, although the difference was not statistically significant. Finally, the change in RVEe was 1.8% for the CT group and −5.4% for the ASDc group, with a P-value of .22, indicating no statistically significant difference.

The measurements of physical capacity within both groups exhibited no significant alteration. In the ASDc group, there was a non-significant increase in oxygen consumption from an initial value of 17.3 ± 6.2 to a final value

of 18.4 ± 6.1 ml/kg/min (P = .47). Conversely, in the CT group, oxygen consumption decreased from 15.7 ± 5.7 to 12.7 ± 5.4 ml/kg/min (P = .41), also without achieving statistical significance.

Discussion

The most important conclusion of the current study is the finding of the beneficial effect of the MitraClip procedure on RV hemodynamic conditions expressed by a decrease in RVSP. Such an effect occurred independently of the concomitant closure of the iASD; however, the placement of the occluder resulted in a greater reduction in RVSP and showed a clear incremental trend toward RV-PAc improvement. This is important because the persistence of high pulmonary pressure in patients leads to progressive RV dilatation, decreased RV contractility, systemic congestion, and secondary organ dysfunction.[9,10] Our results are consistent with the observations of Schueler et al,[11] who found less decrease in sPAP in the iASD group (−1.6 ± 14.1 mmHg vs 9.3 ± 17.4 mmHg; P = .02), worse clinical outcomes and increased mortality at 6-month follow-up.

However, despite the decrease in RVSP, the RV volume overload associated with iASD may have other adverse effects. In a meta-analysis by Maier et al,[12] persistent iASD observed in 28% of MitraClip patients after 12-month follow-up was associated with a higher risk of RV volume overload and more frequent rehospitalization than patients without persistent iASD. Hence, the influence of other parameters on prognosis after MitraClip is being evaluated. Of particular interest in this regard was RV-PAc. In a subanalysis of the COAPT study, RV-PAc was assessed as the ratio of RV free wall longitudinal strain derived from speckle-tracking echocardiography and noninvasively measured RV systolic pressure. At two-year follow-up, so defined RV- pulmonary artery uncoupling of ⩽0.5%/mm increased the risk of hospitalization for heart failure (HR: 1.87; 95% CI: 1.31-2.66; P = .0005), as well as for all-cause mortality alone (HR: 2.57; 95% CI: 1.54-4.29; P = .0003).[13] Also, Popolo Rubbio et al[14] using the same methodology to assess RV-PAc as in the current study, showed that TAPSE/sPAP ⩽ 0.36 remained a sustained predictor of mortality and hospitalization for heart failure at 1 year after MitraClip (hazard ratio: 3.87; 95% confidence interval: 1.83-8.22; P ⩽ .001). An analysis of 817 patients undergoing MitraClip procedure included in the multicentered EuroSMR registry showed an adverse effect of RV-pulmonary artery decoupling, expressed as TAPSE/sPAP ratio ⩽0.27 mm/mmHg, on both survivals at 1 year (70.2% vs 84.0%; P < .001) and 2 years (53.4% vs 73.1%; P < .001) after the procedure, but also a trend toward a lower rate of NYHA functional class I or II (56.5% vs 65.5%; P = .086).[15] R. Kazimierczyk et al[16] also demonstrated the prognostic value of this parameter assessed echocardiographically on a group of patients with pulmonary arterial hypertension, with a worse prognosis associated with a TAPSE/sPAP value of less than 0.25 mm/mmHg. In our study, the improvement in RV-PAc did not reach statistical significance, which is probably due to the small size of the group, but the 36% increase in the ASDc group compared to 9.75% in CT group indicates the importance of RV volume overload due to iASD, as well as the benefit of closing iASD immediately after the procedure. In comparing the outcomes of our present study with the findings from the MIHTHRAS study, where the advantages of iASD closure were not evident, a crucial determinant of the benefits associated with iASD closure appears to be the timing of the defect's closure.[8] It is noteworthy that patients undergoing MitraClip procedures typically exhibit varying degrees of RV dysfunction. Consequently, subjecting these individuals to additional volume overload, even for a relatively brief duration of several weeks, might impede the potential for enhancing RV function.

In our study, we did not observe improvements in the basic parameters of RV function, like FAC and TAPSE. However, the included patients had baseline TAPSE ⩾ 16 mm and FAC ⩾ 35%, within normal limits in most cases. Thus, no further increase in these parameters could be assumed. In addition, TAPSE is a parameter that depends on preload RV volume.[17] This explains the notably lower TAPSE values only in the iASD group. At the same time, this observation provides a rationale for believing that the blood volume flowing through the iASD after the MitraClip procedure is hemodynamically relevant.

Study Limitations

An obvious limitation of the paper is the small size of the 2 groups, so despite the percentage differences in results, it can only speak to the observed trends. The non-randomized approach to the iASD closure procedure further complicates the interpretation of the findings, as it introduces the potential for selection bias. It should also be noted that the qualifications of patients in the current study considered the RV assessment. Hence, there were no patients with baseline significant RV dilatation among the study participants, and TASE and FAC indicate preserved systolic function. Under such conditions, a left-right leak may have a lesser effect on the RV function and RV-PAc. This could limit the generalizability of the findings, as patients with more advanced RV dysfunction may not have been adequately represented. In addition, the results presented here relate only to the echocardiographic parameters, not to clinical outcomes. Thus, while the study demonstrates changes in RV function and pulmonary resistance, it does not provide evidence of the clinical impact of the intervention. Given these limitations, future research is needed to confirm these findings and expand our understanding of the potential benefits of iASD closure. Future research requires more extensive, multi-center randomized controlled trials, longer follow-up periods, and assessment of iASD closure impact on clinical outcomes such as mortality rates, hospitalizations, and quality of life. By addressing these limitations and exploring the proposed future directions, we can better understand the potential role of iASD closure in improving outcomes for patients undergoing MitraClip procedures.

Conclusions

The MitraClip procedure leads to a significant decline in RVSP, with a more pronounced reduction following the closure of iASD. The closure of the iASD seemingly exerts a more robust unfavorable influence on right ventricular (RV) function, evident from a greater reduction in tricuspid annular plane systolic excursion (TAPSE). Nonetheless, an evaluation of RV functional parameters unaffected by RV preload, such as RV-PAc, reveals notably improved hemodynamic conditions for RV performance after iASD closure. These outcomes underscore the advantages of direct iASD closure in protecting the RV against volume overload. However, given the limitations of this study, further investigation is needed to confirm these findings.

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Ethical Considerations

The study protocol was approved by the Bioethics Committee at the Medical University of Silesia in Katowice, Poland (KNW/0022/KB1/60/19 dated 04.06.2019).

Author Contributions

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