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# Clinical and echocardiographic predictors of the anterior mitral leaflet repair failure

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## Abstract

**Background:** Anterior mitral leaflet prolapse repair is a highly effective procedure, but despite excellent operative results still has an inferior long-term durability when compared to posterior leaflet repair.

**Methods:** We analysed mitral repair durability in 74 consecutive patients operated for anterior leaflet prolapse between 2010 and 2021. Their pre- and postoperative clinical, echocardiographic data and repair durability as well, were compared with 74 randomly assigned posterior leaflet prolapse patients who underwent valve repair during the same period.

**Results:** While groups were of similar age, patients with anterior leaflet prolapse had an inferior preoperative status in terms of functional reserve, atrial fibrillation, operative risk, ejection fraction and had more dilated left heart chambers as well. 1, 5, and 10-year freedom from repair failure was 87.1 ± 4.6%, 79.8 ± 6.5% and 50.7 ± 12.5% in the anterior, and 98.5 ± 1.5% respectively in the posterior leaflet group. Atrial fibrilation (hazard ratio [HR] 5.365; 95%; confidence interval [CI] 1.093–26.324 $p = .038$) and left ventricle end-systolic diameter (HR 1.160 95%; CI 1.037–1.299 $p = .010$) independently predicted anterior leaflet repair failure. Receiver Operating Curve analysis established left ventricle end-systolic diameter ≤42 mm as a cut-off value associated with improved anterior leaflet repair durability. Accordingly, 10-year repair durability in a subset of patients, with preserved left ventricle end-systolic diameter (≤42 mm) was 86.4 ± 7.8%.

**Conclusion:** Better long-term repair durability in patients with anterior mitral leaflet prolapse and preserved sinus rhytm and left-ventricle diameters justifies early reconstructive approach.

KEYWORDS

left ventricle, mitral valve repair, survival analysis

## 1 | INTRODUCTION

Mitral valve (MV) repair serves as the treatment-of-choice for degenerative mitral regurgitation (MR) due to superior outcomes.[1–5] Repair techniques for the posterior mitral leaflet (PML) prolapse are straightforward and easily reproducible, with proven safety and long-term durability.[6,7] On the other hand, anterior mitral leaflet (AML) prolapse repair is technically more demanding with inferior long-term results, and by itself represents an independent risk factor for repair failure.[7–9] Although a few repair techniques could affect long-term

durability,[8,10] literature is still modest in subtle analysis of repair failure in patients with AML prolapse. It is still unclear why those patients did worse in term of repair durability in spite of optimal intraoperative echocardiographic control results. To better understand the mechanism of AML repair failure, we set up following study objectives: (a) to analyze preoperative clinical and echocardiographic characteristics in patients with isolated AML prolapse and (b) to define possible predictors that are affecting repair durability in patients with AML prolapse.

## 2 | MATERIALS AND METHODS

### 2.1 | Patient selection and study protocol

This retrospective study included 148 patients who underwent mitral valve repair between January 2010 and March 2021. The test group was made of 74 consecutive patients with isolated AML prolapse repair. The control group consisted of 74 patients with isolated PML prolapse repair, selected as random quota sample according to the annual number of patients operated in the test group. We applied the following exclusion criteria: bileaflet prolapse, mitral pathology other than degenerative, history of myocardial infarction, concomitant aortic valve surgery and reoperative cardiac surgery as well. Eleven patients (14.8%) from the test, and four patients (5.4%) from the control group were unavailable for the follow-up. Therefore, the final number of the analyzed patients was 59 in the test, and 70 patients in the control group. Mean follow-up period was 41.05 ± 36.9 months (7 days—130.67 months; *M* = 30.16months). Adverse events were reported using the classification of Edmunds et al.[11] for reporting morbidity and mortality after cardiac valvular operations.

**TABLE 1** Basic operative data

| Variables[a] | AML (*n* = 59) | MPL (*n* = 70) | *p* Value |
|---|---|---|---|
| CCT, min | 82.6 ± 30.59 | 77.21 ± 25.02 | .273 |
| CPB, min | 112.64 ± 40.12 | 106.69 ± 35.57 | .373 |
| Concomitant procedure | | | |
| CABG | 11 (18.6) | 10 (14.3) | .505 |
| CABG, no of grafts | 2.27 | 1.88 | .005 |
| LV EF,[b] % | 47.27 ± 7.862 | 49.50 ± 9.846 | .572 |
| LV ESD,[b] mm | 39.09 ± 4.805 | 39.90 ± 7.708 | .761 |
| LV EDD,[b] mm | 57.73 ± 5.764 | 61.90 ± 11.13 | .288 |
| TR | 8 (13.6) | 11 (15.7) | .731 |
| Ring type | | | |
| SJM Saddle® | 36 (61) | 34 (48,6) | |
| SJM Seguin® | 3 (5.1) | 6 (8.6) | |
| CE Physio I® | 6 (10.2) | 8 (11.4) | |
| CE Classic® | 10 (16.9) | 11 (15.7) | |
| Medtronic Duran "AnCore"® | 4 (6.8) | 10 (14.3) | |
| SJM Tailor® | 0 | 1 (1.4) | |
| Ring size (mm) | 30.98 ± 3.319 | 31.37 ± 2.940 | .482 |

Abbreviations: CABG, coronary artery bypass grafting; CCT, cross clamping time; CE Physio I®, Edwards Lifescience, Irvine, California, USA; CPB, cardiopulmonary bypass; EDD, end-diastolic diameter; ESD, end-sistolic diameter; LV, left ventricle; SJM Saddle®, Saint Jude Medical St Paul, Minessota, USA; TR, tricuspid regudrgitation.

[a]Continuous data are shown as mean ± standard deviation and categoric as number (%).

[b]Patients with concomitant CABG surgery.

### 2.2 | Data acquisition and outcomes

Preoperative and operative data were retrospectively collected from patient files while the follow-up exam and the interview were performed at the hospital. Expirienced physicians did transthoracic echocardiography follow-up controls. Degree of MR was assessed semi-quantitatively. Follow-up echocardiographic parameters were collected according to the available baseline data set. The analysis of the repair failure and its predictors were done only in the test group while the follow-up results of the control group were only reported. The study end point - overall repair failure was expressed as residual severe MR or/and mitral valve replacement (MVR) due to repair failure (severe MR/MVR).

### 2.3 | Surgical technique

All patients were operated through a median sternotomy and underwent ring annuloplasty as well. A number of different reconstructive techniques were used for AML prolapse repair: "flip-over" technique in 19 patients (32.2%), neochordae implantation in 13 patients (22%), AML resection in 10 patients (16.9%), secondary chordae transposition in 13 patients (22%), papillary muscle repositioning in two patients (3.4%), chordal shortening and "edge-to-edge" technique in two patients. On the contrary, PML prolapse was treated with leaflet resection in all patients. Operative data are presented in Table 1. Patients with AML disease had more grafts per patient when compared with PML group, but we found no difference in preoperative left ventricle (LV) function between these coronary artery bypass grafting (CABG) subsets. Patients with concomitant CABG surgery were as well similar when compared with isolated mitral repair surgery patients in term of ejection fraction (EF) (42.27 ± 7.862% vs. 48.54 ± 11.803%, $p = .736$), LV end-sistolic diameter (LVESD) (39.09 ± 4.805 mm vs. 40.94 ± 7.156 mm, $p = .420$) and LV end-diastolic diameter (LVEDD): (57.73± 5.764 mm vs. 60.6 ± 8.211 mm, $p = .376$) in the AML prolapse as well as in the PML prolapse group: EF (49.5 ± 9.846 vs. 53.70 ± 6.892, $p = .09$), LVESD (39.90 ± 7.078 vs. 37.77 ± 5.166, $p = .256$) and LVEDD (61.90 ± 11.130 vs. 59.62 ± 5.621, $p = .316$). Therefore, we could

say that the asymptomatic coronary disease had no impact on preoperative LV function in our study.

### 2.4 | Statistical analyses

Categorical variables are represented as absolute numbers and percentages while the mean and standard deviations (mean ± SD) are used to describe continuous variables. Student *t*-test, Mann–Whitney test, Pearson's $\chi^2$ test, or Fisher's exact test, were used appropriately, to compare preoperative parameters between groups. We analyzed time-dependent variables by the Kaplan–Meier method and log-rank test. A multivariate analysis was used to determine repair failure predictors by logistic regression (method: forward stepwise— conditional). Receiver Operating Characteristic (ROC) curve analysis was used to establish best cut-off values for AML repair failure risk. Statistical analysis was performed using the Statistical Package for Social Science (SPSS) 19.0. The level of significance was set at $p < .05$.

## 3 | RESULTS

### 3.1 | Preoperative data

Baseline patient characteristics are presented in Table 2. AML group had more female patients, inferior functional reserve—New York Heart Association (NYHA) class, more atrial fibrillation (AF) and increased operative risk when compared with the PML prolapse group. Those patients also demonstrated poorer EF, larger left atrium (LA) and LV ESD. On the other side, there were more diabetic patients in the PML prolapse group.

### 3.2 | Mortality

Two patients died in the AML (2.7%), and one in the PML prolapse group (1.35%), making the overall hospital mortality of 2%. Valve related hospital mortality was 1.35% (one patient) in the AML prolapse group. This was a 74-year old female patient, who underwent MVR due to an early AML repair failure and died 40 days after the surgery. There were two late deaths in both groups and were no valve-related. 1, 5, and 10 years survival was 96.5 ± 2.5%, 87.10 ± 6.7% and 87.10 ± 6.7%%, respectively in AML, and 98.6 ± 1.4%, 93.5 ± 3.8%, 93.5 ± 3.8%, respectively in the PML prolapse group (log rank. $\chi^2 = 0.657$, $df = 1$, $p > .05$).

### 3.3 | Morbidity

Hospital and late morbidity were described in Table 3. Repair failure was by far, the most frequent adverse event in the AML prolapse group, and was present in 13 patients (22%). The second most common complication in both groups was hemorrhage and was presented as chronic pericardial tamponade. Two out of four CVI-s were of hemorrhagic origin as well. Thromboembolic events occurred in two patients in the AML prolapse group while there was only one mitral valve endocarditis in the PML prolapse group. The overall

**TABLE 2** Baseline demographic, clinical and echocardiographic data

| Variables[a] | AML (*n* = 59) | PML (*n* = 70) | *p* Value |
|---|---|---|---|
| Age, years | 54.78 ± 14.87 | 55.4 ± 12.11 | .781 |
| Gender | | | .027 |
| Male | 31 (52.5) | 50 (71.4) | |
| Female | 28 (47.5) | 20 (28.6) | |
| NYHA class | | | .0001 |
| I | 2 (3.4) | 9 (12.9) | |
| II | 29 (49.2) | 50 (71.4) | |
| III | 28 (47.5) | 10 (14.3) | |
| IV | 0 | 1 (0.8) | |
| Heart rhythm | | | .024 |
| Sinus | 42 (71.2) | 61 (87.1) | |
| AF | 17 (28.8) | 9 (12.9) | |
| Comorbidities | | | |
| HTA | 29 (49.2) | 42 (60) | .217 |
| HLP II | 17 (28.8) | 26 (37.1) | .317 |
| DM | 1 (1.7) | 8 (11.4) | .020 |
| COPD | 2 (3.4) | 2 (2.9) | .862 |
| Smoker | 22 (37.3) | 19 (27.1) | .218 |
| BSA, $m^2$ | 1.88 ± 0.19 | 1.95 ± 0.21 | .055 |
| ES | 4.42 ± 1.76 | 3.74 ± 1.56 | .021 |
| ES LOG | 3.94 ± 2.63 | 2.97 ± 1.88 | .019 |
| Echocardiography | | | |
| EF (%) | 48.31 ± 11.13 | 53.10 ± 7.45 | .006 |
| LA, mm | 49.49 ± 8.72 | 45.19 ± 6.78 | .002 |
| LV EDD, mm | 59.63 ± 7.82 | 59.94 ± 6.62 | .807 |
| LV ESD, mm | 40.59 ± 6.78 | 38.07 ± 5.47 | .021 |
| RVSP, mmHg | 44.34 ± 14.63 | 42.87 ± 13.72 | .558 |
| LV INF, mm | 9.52 ± 1.61 | 9.63 ± 1.37 | .666 |
| LV IVS, mm | 9.74 ± 1.45 | 9.85 ± 1.45 | .667 |

Abbreviations: AF, atrial fibrillation; BSA, body surface area; COPD, Chronic Obstructive Pulmonary Disease; DM, diabetes melitus; EDD, end-diastolic diameter; EF, ejection fraction; ES, Euros score; ESD, end-systolic diameter; HLP, hyperlipoproteinemia; HTA, hypertension; INF, inferior; IVS, inter ventricular septum; LOG, logistic; LV, left ventricle; NYHA, New York Heart Association; RVSP, right ventricle systolic pressure.

[a]Continuous data are shown as mean ± standard deviation and categoric as number (%).

incidence of postoperative complications was lower in the PML prolapse group (Log rank (Mantel–Cox) $\chi^2 = 6.795$, $df = 1$, $p = .009$). Overall long-term freedom from morbidity in patents with AL and with PL prolapse repair is shown in Figure 1.

### 3.4 | Repair durability

Echocardiographic follow-up exam in AML prolapse group demonstrated zero-to-mild MR in 30 patients (58.8%) and mild-to-moderate MR in 12 patients (23.5%). Severe MR was found in nine patients (17.7%), and four of them underwent valve replacement immediately upon the diagnosis of the residual MR. Therefore, the total number of eight patients underwent MVR due to repair failure while the another five patients were living with severe residual MR. Within reoperated patients, surgery dependent repair failure was found in four patients while other four had "de-novo" leaflet prolapse. 1, 5, and 10-year freedom from MVR was 87.1 ± 4.6%, 87.1 ± 4.6%, and 79.2 ± 8.6%, respectively (Figure 2A), while freedom from severe MR was 100%, 81.7 ± 5.6% and 64.1 ± 14.2%, respectively (Figure 2B). The study end-point, freedom from overall repair failure—severe MR/MVR in AL prolapse group at 1, 5, and 10 year was 87.1 ± 4.6%, 79.8 ± 6.5%, and 50.7 ± 12.5%, respectively (Figure 2C).

Freedom from overall repair failure in PML group at 1, 5, and 10-year was 98.5 ± 1.5% (Figure 2D) and was superior when compared to AML group (log rank [Mantel–Cox] = 15.005, $df = 1$, $p = .0001$). In PML prolapse group zero-to mild MR was present in 55 patients (90.2%), mild-to-moderate MR in six patients (8.6%), and there was no severe MR.

We found a difference in long-term AML repair durability among high and low-volume surgeons (log Rank (Mantel Cox) = 11,105, $df = 2$, $p = .004$). Univarate analysis identified the absence of sinus rhythm ($\chi^2$: likelihood ratio = 4.736, $df = 1$, $p = .03$), LVESD ($t$-test; $p = .003$), and LVEDD ($t$-test; $p = .02$) as preoperative predictors for AML repair failure. Forward stepwise conditional multivariate logistic regression model demonstrated that LVESD ($df = 1$, $p = .010$, Exp (B) = 1.160; 95% CI 1.037–1.299) and AF ($df = 1$, $p = .038$, Exp (B) = 5.365; 95% CI 1.093–26.324) were independent predictors for repair failure. Analysis of ROC curve coordinates established an ESD of 42 mm as a cut-off

**TABLE 3** Hospital and late morbidity overview in patients with AL and PL prolapse

| Complication | AML group | | PML group | |
|---|---|---|---|---|
| | Hospital | Late | Hospital | Late |
| Residual MR | 3 (5.1%) | 10 (16.9%) | 1 (1.4%) | 0 |
| Hemorrhagic | 4 (6.8%) | 2 (3.4%) | 6 (8.5%) | 0 |
| Thromboembolic | 0 | 0 | 0 | 0 |
| Endocarditis | 0 | 0 | 0 | 1 (1.4%) |
| Pleural effusion | 5 (8.5%) | 0 | 2 (2.9%) | 0 |
| CVI | 1 (1.7%) | 1 (1.7%) | 1 (1.4%) | 1 (1.4%) |
| Inotropes | 2 (3.4%) | N/A | 5 (7.1%) | N/A |
| AF | 1 (1.7%) | 3 (5.1%) | 4 (5.7%) | 1 (1.4%) |
| AV block | 2 (3.4%) | 1 (1.7%) | 1 (1.4%) | |
| Wound infection | 1 (1.79%) | 0 | 0 | 0 |

Abbreviations: AF, atrial fibrillation; CVI, cerebro vascular insult; MR, mitral regurgitation; PML, posterior mitral leaflet.

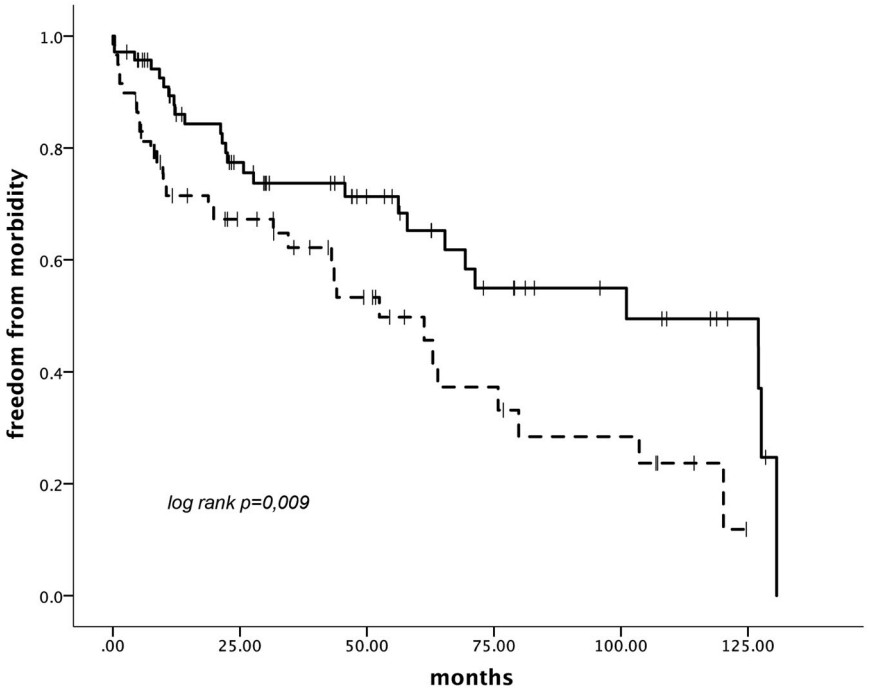
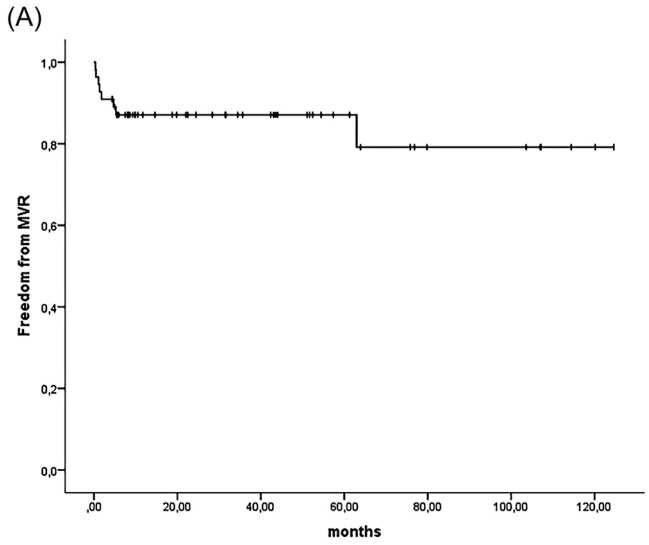
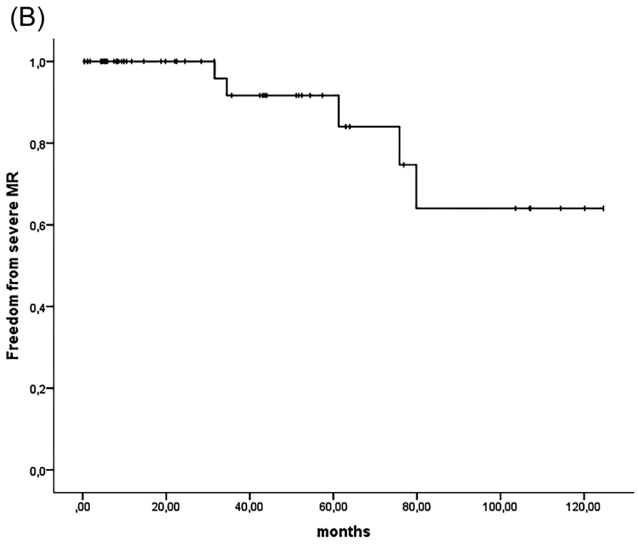


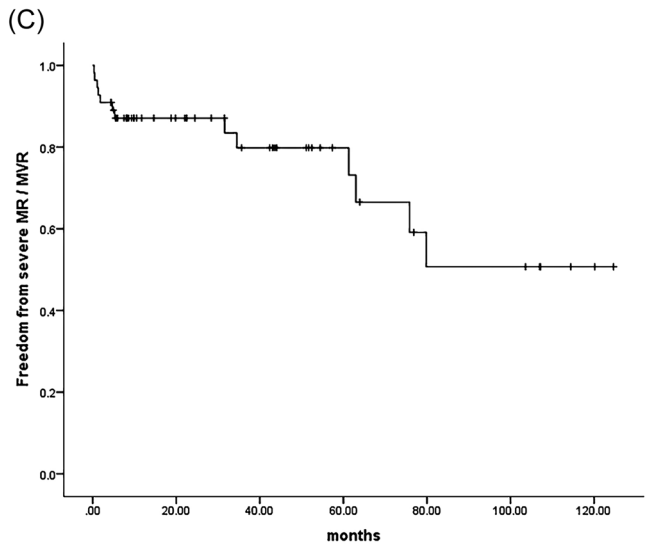

**FIGURE 1** Kaplan–Meyer analysis of overall long-term freedom from morbidity in patents with AL (dotted line) and with PL prolapse repair (solid line).



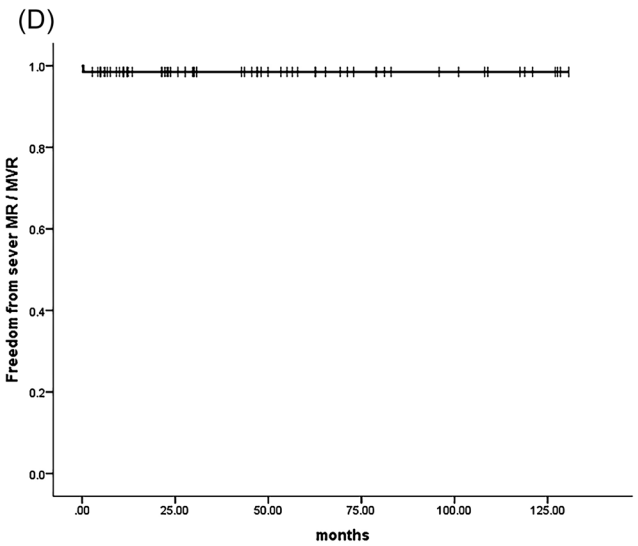

| months | | 20 | 40 | 60 | 80 | 100 | 120 |
|---|---|---|---|---|---|---|---|
| Number risks AL | at | 29 | 15 | 12 | 6 | 6 | 2 |




| Months | | 20 | 40 | 60 | 80 | 100 | 120 |
|---|---|---|---|---|---|---|---|
| Number risks AL | at | 54 | 29 | 16 | 6 | 6 | 2 |




| months | | 20 | 40 | 60 | 80 | 100 | 120 |
|---|---|---|---|---|---|---|---|
| Number risks | at | 29 | 21 | 12 | 6 | 6 | 2 |




| months | | 20 | 40 | 60 | 80 | 100 | 120 |
|---|---|---|---|---|---|---|---|
| Number risks | at | 46 | 32 | 21 | 13 | 10 | 5 |

**FIGURE 2** Survival curves. (A) Freedom from MVR, (B) freedom from severe MR and (C) freedom from overall repair failure—severe mitral regurgitation (MR)/mitral valve replacement (MVR) in AL group. (D) Freedom from overall repair failure—severe MR/MVR in PL group.

value for repair failure risk (Area under the curve 0.792; 95% CI 0.654–0.930, $p = .02$).

### 3.5 | Echocardiographic and functional status follow-up

We found a reduction of left atrial and LV diameters in both groups while EF remained unchanged (Table 4). There was also an LV wall thickness reduction in AML group. RVSP decreased only in the PML group, which is probably due to more functional mitral valve repairs. Such an improvement in heart function resulted in the NYHA functional status recovery in both groups (AML $Z = -5.580$, $p = .001$ and PML $Z = -6.970$, $p = .000$). 71.7% in AML and 95.4% in PML group were in NYHA class I, and 11.5% and 4.6% patients, respectively were in NYHA class II. Severe functional disability existed in AML group only, six patients (1.35%) were in NYHA III class, and one patient (1.35%) was in NYHA class IV.

**TABLE 4** Echocardiographic follow-up data in patients with AML and PML prolapse repair

| Variable[a] | AML pre-op *n* = 51 | AML post-op *n* = 51 | *p* Value | PML pre-op *n* = 66 | PML post-op *n* = 66 | *p* Value |
|---|---|---|---|---|---|---|
| EF, % | 48.82 ± 10.94 | 49.31 ± 0.96 | 0.569 | 53.97 ± 6.41 | 54.61 ± 6.27 | .402 |
| LA, mm | 48.35 ± 8.16 | 45.96 ± 8.31 | 0.014 | 44.72 ± 6.50 | 41.58 ± 4.91 | .000 |
| LV EDD, mm | 58.75 ± 7.67 | 54.67 ± 7.97 | 0.000 | 60.03 ± 6.38 | 53.21 ± 4.64 | .000 |
| LV ESD, mm | 40.04 ± 6.96 | 36.78 ± 8.86 | 0.000 | 37.82 ± 4.89 | 34.49 ± 5.22 | .000 |
| RVSP, mmHg | 42.98 ± 11.97 | 40.98 ± 9.98 | 0.269 | 41.55 ± 12.38 | 36.33 ± 4.94 | .003 |
| LV IVS, mm | 9.78 ± 1.47 | 9.11 ± 1.56 | 0.005 | 9.79 ± 1.42 | 9.89 ± 1.38 | .594 |
| LV INF, mm | 9.48 ± 1.71 | 8.85 ± 1.70 | 0.004 | 9.59 ± 1.27 | 9.80 ± 1.26 | .194 |

Abbreviations: AML, anterior mitral leaflet; EDD, end-diastolic diameter; EF, ejection fraction; ESD, end-systolic diameter; LA, left atrium; LV, left ventricle; INF, inferior; IVS, inter ventricular septum; PML, posterior mitral leaflet; RVSP, right ventricle systolic pressure.

[a]Data are shown as mean ± standard deviation.

## 4 | DISCUSSION

Our study analyses the reasons for inferior long-term results of the mitral repair in patients with degenerative AML prolapse. We examined baseline preoperative data and demonstrated inferior clinical and echocardiographic status in patients with AML prolapse compared with the PML prolapse group. Results were mostly in line with similar studies,[7,12] and such a difference is explained by a delayed surgery due to inferior AML repair durability. However, we couldn't find enough evidence to support such an assumption.

First, our groups were similar regarding the age. In the youngest age group (<40 years) we found more patients with AML prolapse (13 vs. 8) as well as a greater degree of LA enlargement (44.77± 8.10 mm vs. 40.63 ± 5.528 mm; $t = 1.27$, $df = 19$, $p = .219$) when compared with PML group. Although not significant, such a shift to a younger age indicates that an accelerated course of a disease rather than disease duration itself could be responsible for inferior preoperative status in patients with AML prolapse. Flail AML could have different path physiology and impact on left heart chambers than the posterior one. The PML prolapse regurgitation jet is directed toward the IAS, and its energy is absorbed by the outer support of the right atrium. On the contrary, the AML prolapse regurgitation jet is directed toward the free wall of the LA that has the weak outer support and probably could not absorb regurgitation jet energy so effectively, and therefore accelerates LA dilatation. Medical literature so far described lower pulmonary flow reversal in patients with PML prolapse when compared with other prolaps locations.[13] Such a jet also targets and overstresses myocytes in the carina area between pulmonary veins, were almost 36% of ectopic nodes responsible for the onset of the AF are located in.[14] Dilated LA along with AF accelerates vicious circle that enlarges mitral annulus and further increases the degree of MR, LV volume overload and LV impairment as well.[15] Clinically, these changes reflect through a higher degree of functional impairment that we found in patients with AML prolapse.

Repair durability in our AML cohort was inferior when compared with other centers[7,9,12,16] in terms of freedom from severe MR, MVR, and freedom from overall repair failure as well. However, our AML cohort has a higher degree of LV systolic impairment preoperatively (EF, LVESD) as well as a greater degree of LA dilatation (Figure 3). When we adjusted repair failure analyses in terms of ROC calculated LVESD cut-off value, we found a functional AML mitral repair in 86.4 ± 7.8% patients with up-to modest LVESD dilatation (<42 mm) at 10-year follow-up. On the contrary, in a subgroup of patients with LVESD ≥ 42 mm only 21.6 ± 17.5% repairs were functional 6 years after the surgery, and no functional repair was found at 8-year follow-up (Figure 3). Witkowski et al.[17] demonstrated that preoperative AF, EF ≤ 60%, LVESD ≥ 40 mm, atrial fibrilation, and global longitudinal strain (GLS) > −19.9% were predictors of long-term EF dysfunction (EF ≤ 50%) after MVP, while the GLS and LVESD were independent predictors. Song et al.[18] found that LVESD ≥ 41 mm and LV end-systolic volume ≥85 cc are predictors of immediate postoperative severe LV dysfunction after mitral valve repair. It is important to notice that both studies emphasises the dependence of the onset and severity of LV failure with LVESD enlargement.

The left ventricle is an important part of the mitral apparatus and reverse LV remodeling is the goal of mitral repair surgery. Reverse remodeling is a sign of LV function recovery, but also relieves the leaflet strain, preserves good coaptation line and regardless of the mitral pathology provides long-term repair durability. According to some in vitro studies,[19] anterior leaflet itself is generally exposed to a greater systolic stress than the posterior one. Volume overload in MR affects LV shape early, even in asymptomatic patients with preserved EF.[20] Sphericity continues to grow up with disease progression and exerts additional strain on already altered chordal tissue. Therefore, such a sum of an excessive strain load in patients with the degenerative AML disease and enlarged LVESD could precipitate disease dependent repair failure as “de-novo” chordal rupture and to some degree it could contribute in a procedure dependent repair failure as well.

### 4.1 | Limitations of the study

The study results represent our first 10-year experience with AL prolapse repair, and they are certainly influenced by the learning

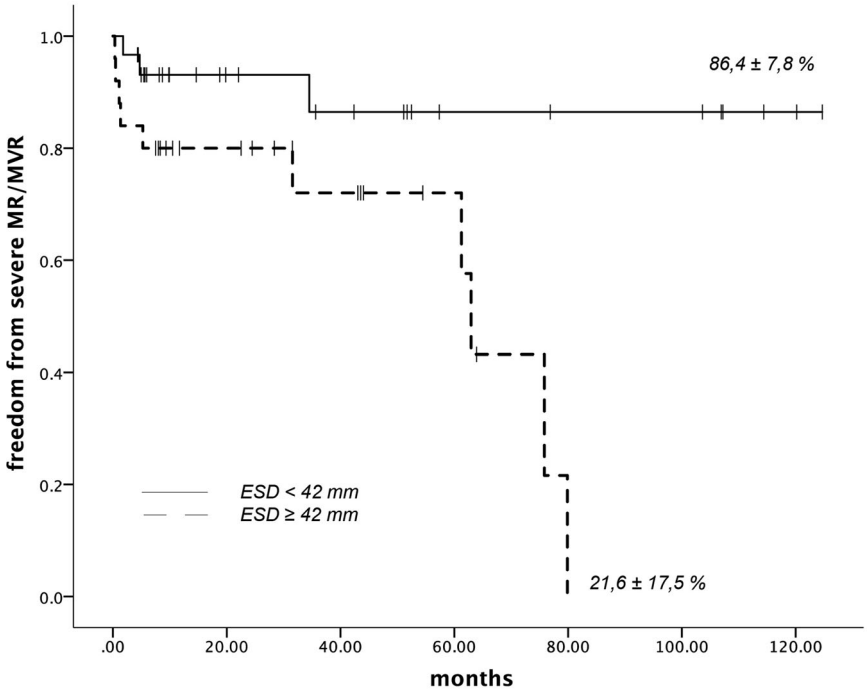



**FIGURE 3** Kaplan–Meyer analysis of overall long-term freedom from repair failure in anterior mitral leaflet group repair, regarding the left ventricle end-sistolic diameter (ESD) cut-off level of 42 mm

curve. However, main study limitations were small study groups and important number of patients lost from follow-up. Besides that there are some differences in compared groups. Patients with concomitant CABG had more more grafts per patient in AML when compared with PML group. Left ventricle ejection fraction was slightly lower in AML grop, although this difference was not statistically significant. Due to the retrospective study design we had to adjust echocardiographic follow-up parameters to retrospectively collected data. Therefore, we had to use semi-quantitative method to evaluate residual MR instead of more precise measurements. All of the above is decreasing to some degree the power of our analysis. However, we think that we are on the right path with our results, particularly in lights of recent studies that clearly defined very similar LVESD cut-off values as predictors for postrepair LV dysfunction. Having in mind that LV is an integral part of the mitral apparatus, its impact on leaflet coaptation is expected, even in degenerative mitral disease and especially in terms of AML prolapse complexity.

## 5 | CONCLUSION

With respect to these limitations, we can conclude that AML prolapse regurgitation jet probably has a different pathophysiology than the posterior one. It is probably, not just a "prolapse from an opposite site," and results in a greater degree of clinical impairment and LV dysfunction when compared to PML prolapse. Regurgitant jet pathophysiology rather than disease duration itself seems to be responsible for an accelerated disease course in these patients. Absence of sinus rhythm and LVESD are found to be independent preoperative risk factors for AML repair failure. Therefore, to provide optimal long-term repair durability, patients with flail AML should be approached in the same way as those with PML prolapse, and so be operated in early stages of the disease. Those patients have to be addressed to an experienced surgical team for the best patient benefit.

### AUTHOR CONTRIBUTIONS

Ivan Stojanovic, Bogdan R. Okiljevic, Radojicic Zoran, Novakovic Aleksandra, Marko Kaitovic, and Tomic Slobodan had major roles in the conception, design, planning, and carrying out the study. Ivan Stojanovic, Bogdan R. Okiljevic, Radojicic Zoran, Novakovic Aleksandra, Marko Kaitovic, and Tomic Slobodan also each contributed to the analysis of the data and the writing and editing of the manuscript.

### CONFLICT OF INTEREST

The authors declare no conflict of interest.

### ETHICS STATEMENT

This study was approved by the Institutional Review Board.

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