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STRUCTURAL

ORIGINAL RESEARCH

Impact of Recurrent Mitral Regurgitation Following Mitral Transcatheter Edge-to-Edge Repair on Long-Term Outcomes

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ABSTRACT

BACKGROUND Residual mitral regurgitation (MR) after mitral transcatheter edge-to-edge repair (M-TEER) is a major determinant of outcome. However, the prognostic impact of recurrent mitral regurgitation (recMR) after initially successful M-TEER remains unclear.

OBJECTIVES This study aimed to evaluate the association between recMR after successful M-TEER and long-term mortality across degenerative mitral regurgitation (DMR) and functional mitral regurgitation (FMR).

METHODS This retrospective cohort included 461 consecutive patients with MR undergoing successful M-TEER (residual mitral regurgitation ≤2+ at discharge) between 2008 and 2021. *recMR* was defined as ≥1-grade increase in mitral regurgitation severity at follow-up compared with discharge. The primary endpoint was all-cause mortality.

RESULTS Among 461 patients with MR undergoing M_TEER (mean age 75.0 ± 9.1 years; 60.7% male [280 of 461]; 29.3% [135 of 461] had DMR), recMR occurred in 31.7% (146 of 461) (FMR in 31.9% [104 of 326]; DMR in 31.1% [42 of 135]). The median follow-up time was 3.44 years (Q1-Q3: 1.51-3.99 years). In a 12-month landmark analysis, absence of recMR was associated with improved survival (log-rank, $P = 0.0088$). At 4 years, all-cause mortality was 44.8% (95% CI: 38.7%-50.3%) in patients without recMR and 56.3% (95% CI: 47.2%-63.9%) in those with recMR. In multivariable Cox regression, recMR independently predicted higher mortality (HR: 1.43; 95% CI: 1.06-1.93; $P = 0.019$). The survival difference was significant in patients with DMR (log-rank, $P = 0.014$) but not with FMR ($P = 0.11$).

CONCLUSIONS recMR is common and independently associated with increased mortality after M-TEER, with a more pronounced effect in patients with DMR. Identification of patients at risk may inform closer surveillance and timely reintervention. (JACC Cardiovasc Interv. 2026;19:2743-2755) © 2026 by the American College of Cardiology Foundation.

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ABBREVIATIONS AND ACRONYMS

6-MWD = 6-minute walking distance

AFib = atrial fibrillation

DMR = degenerative mitral regurgitation

FMR = functional mitral regurgitation

LVEDD = left ventricular end-diastolic diameter

LVEF = left ventricular ejection fraction

MPG = mitral pressure gradient

MR = mitral regurgitation

M-TEER = mitral transcatheter edge-to-edge repair

recMR = recurrent mitral regurgitation

resMR = residual mitral regurgitation

TTE = transthoracic echocardiography

Mitral regurgitation (MR) is common and presents a substantial burden of heart failure events, impaired quality of life, and excess mortality.[1] For patients with degenerative MR (DMR) or functional MR (FMR) who are at high or prohibitive surgical risk, mitral transcatheter edge-to-edge repair (M-TEER) is an established therapeutic alternative.[2]

Across studies, the immediate postprocedural residual MR (resMR) grade is one of the strongest and most consistent predictors of long-term outcomes after M-TEER.[3-5] Consequently, procedural strategy prioritizes maximal feasible reduction of resMR as the dominant treatment goal. This entails a careful trade-off between more complete MR reduction (eg, additional clip implantation) and the risk of inducing an elevated postprocedural transmitral gradient.

Despite index procedure-associated device success, a subset of patients experience recurrent MR (recMR), because of mechanisms that may include ongoing ventricular or annular remodeling, aggravated leaflet degeneration, progressive tethering, or device-related factors.[6] Unstable results may deteriorate the initial prognostic benefit of successful index procedures. This specific patient subset may exhibit outcomes comparable to, or worse than, those with suboptimal immediate results.

The objectives of this study were to determine the incidence and prognostic value of recMR after successful M-TEER, to define the independent association of recMR with mid- and long-term mortality, and to identify clinical and procedural determinants, including potential effect modification by MR etiology (degenerative vs functional).

METHODS

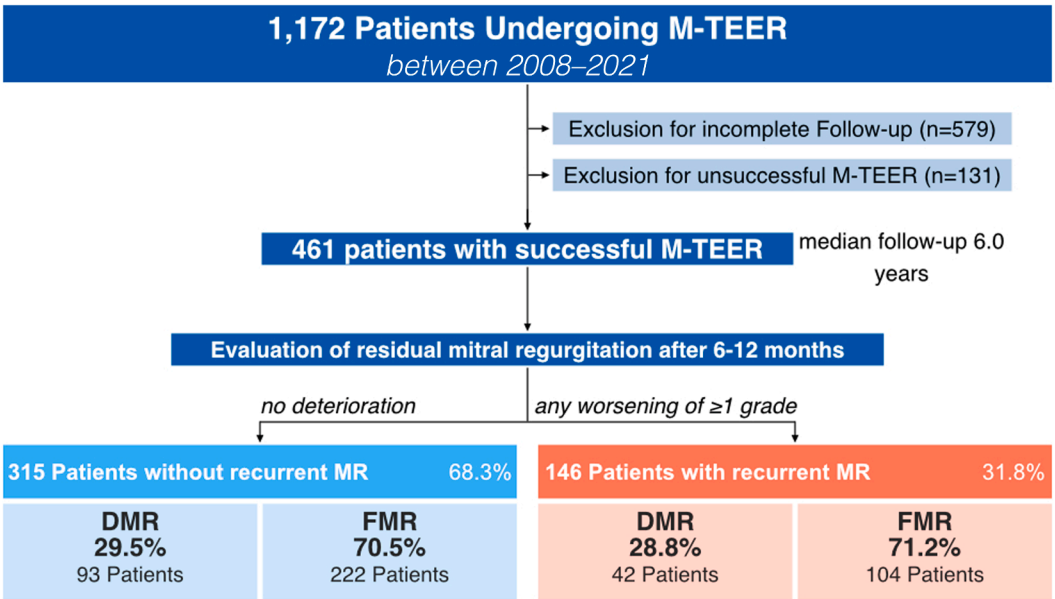
STUDY POPULATION. This retrospective single-center study included 1,172 consecutive patients with MR undergoing M-TEER at the University Heart & Vascular Center Hamburg, Germany, from 2008 to 2021. Because resMR at discharge is an established predictor of prognosis after M-TEER, the present study evaluated the prognostic impact of recMR after initially successful procedures. Therefore, only patients with resMR ≤2+ at discharge were included whereas patients with resMR ≥3+ were excluded. Patients with postprocedural resMR ≥3+ at discharge (n = 131), missing etiology (n = 1), or insufficient follow-up at 6 or 12 months (n = 579) were excluded, yielding a final cohort of 461 patients (Figure 1). All patients were deemed unsuitable for surgery by an interdisciplinary heart team and provided written informed consent before treatment with the MitraClip (Abbott) or PASCAL device (Edwards Lifesciences). M-TEER procedures were performed according to institutional standards. All patients gave written informed consent before study inclusion. The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the Hamburg Chamber of Physicians.

recMR AND ENDPOINT DEFINITION. An *optimal immediate result* was defined as resMR ≤2+ at discharge, consistent with prior reports.[7] *recMR* was defined as a ≥1-grade increase in MR severity at the 6- or 12-month follow-up compared with discharge. Given the lack of a uniform definition of MR recurrence after M-TEER, *recMR* was defined according to relative changes in postprocedural MR severity rather than absolute MR thresholds, as used in prior studies and analysis (Figure 1).[6,8]

Etiology was adjudicated as DMR or FMR for prespecified subgroup analyses. The primary endpoint was all-cause mortality. Secondary endpoints included recMR, functional status assessed by NYHA functional class, and 6-minute walking distance (6-MWD).

ECHOCARDIOGRAPHIC EVALUATION AND FOLLOW-UP. Comprehensive transthoracic echocardiography (TTE) assessments were performed as part of routine clinical practice pre- and postprocedure, at discharge, and at 3, 6, and 12 months by experienced echocardiographers. MR severity was assessed using an integrative, multiparametric echocardiographic approach in accordance with current guideline recommendations,[2,9,10] incorporating qualitative, semiquantitative, and quantitative parameters derived from 2-dimensional TTE and Doppler imaging. Because of the limitations of individual parameters, MR severity was determined by integrating multiple indices. In cases of discordance, additional parameters, including ventricular and left atrial dimensions, and clinical context, were considered to assign the final MR grade. Postprocedural MR severity was assessed by TTE at discharge under stable hemodynamic conditions and served as the reference for procedural success and recMR. Clinical follow-up at 3, 6, and 12 months included medical history, heart failure assessment, laboratory testing, 6-MWD, and NYHA functional class. Structured telephone follow-up and available health records were used when in-person visits were not feasible. The median

FIGURE 1 Study Flow Diagram



The study population consisted of 461 patients who underwent M-TEER. *Recurrent MR* was defined as an increase of ≥1 grade in mitral regurgitation severity at the 12-month follow-up compared with discharge. *Non-recurrent MR* was defined as either no change or a reduction in grade of residual MR severity at the 12-month follow-up compared with discharge. Among the 461 patients, 146 patients (32%) experienced recurrent MR, with 42 (29%) of these patients having DMR and 104 (71%) having FMR. DMR = degenerative mitral regurgitation; FMR = functional mitral regurgitation; MR = mitral regurgitation; M-TEER = mitral transcatheter edge-to-edge repair.

observational time was 6.0 years (maximum 13.6 years). Outcome analyses were administratively censored at 5 years because of a low event rate beyond year 6.

STATISTICAL ANALYSIS. Continuous variables are presented as mean ± SD or median with the frist and third quartiles (Q1-Q3; 25th-75th percentiles) for skewed data. Categorical variables are expressed as count and percentage. Group comparisons were performed using Student's *t*-test or Wilcoxon rank sum test for continuous variables and the chi-square test for categorical variables, as appropriate. The primary outcome was all-cause mortality. Time-to-event analyses were performed using Cox proportional hazards regression with HRs and 95% CIs. Baseline characteristics and outcomes were analyzed overall and according to recMR status. Included and excluded patients were compared to assess potential selection bias. Multivariable Cox regression models were constructed using a theory-driven approach, including clinically relevant covariates associated with mortality after M-TEER, including age, severely reduced left ventricular ejection fraction (LVEF ≤30%), severe renal dysfunction (estimated glomerular filtration rate [eGFR] <30 mL/min per 1.73 m^2), and atrial fibrillation (AFib).[11-13] Model assumptions were assessed for all regression analyses. Multicollinearity was evaluated using variance inflation factors, and proportional hazards assumptions were assessed using Schoenfeld residuals. All fitted models met the predefined assumptions. Follow-up time was summarized as median with IQR and maximum follow-up. Survival probabilities were estimated using the Kaplan-Meier method with 95% CIs. Because recMR was defined according to 6- or 12-month follow-up imaging, survival analyses used a 12-month landmark approach. Patients were then followed from the landmark onward for all-cause mortality. NYHA functional class trajectories were analyzed using a cumulative link mixed model with patient ID as a random intercept. Fixed effects included time, recMR group, and their interaction. Estimated marginal means and Tukey-adjusted pairwise comparisons were calculated. Multivariable Cox regression analyses were additionally performed to identify baseline predictors of recMR using candidate covariates derived from prior

studies identifying them as modifiers of M-TEER procedural outcomes and MR recurrence.[6,14,15] Variables with >20% missingness were excluded from multivariable analyses. Sensitivity analysis using inverse probability weighting was performed to assess residual confounding. Stabilized weights were estimated using logistic regression including baseline covariates with ≤21% missingness, followed by weighted Cox regression analysis. All statistical tests were 2-sided, and *P* value <0.05 was considered statistically significant. Analyses were performed using R 4.3.0 (R Foundation for Statistical Computing).

RESULTS

BASELINE CHARACTERISTICS. Among 461 patients (mean age 75.0 ± 9.1 years; 60.7% male [280 of 461]), most presented with advanced symptoms (NYHA functional class III-IV in 92.4% [426 of 461]) and high surgical risk (median Society of Thoracic Surgeons Predicted Risk of Mortality [STS-PROM] score 3.6% [Q1-Q3: 2.2%-5.6%]). Severe MR (≥3+) was present in 99.3% of patients (458 of 461), and FMR accounted for 70.7% (326 of 461) and DMR for 29.3% (135 of 461). Comorbidities were common, including coronary artery disease in 65.1% of patients (295 of 453), AFib in 67.9% (309 of 455), and prior heart failure hospital stay in 77.2% (156 of 202). The median LVEF was 42.0% (Q1-Q3: 29.2%-53.8%), with 26.8% of patients (123 of 461) experiencing severely reduced LVEF (≤30%). Severe chronic kidney disease (eGFR <30 mL/min per 1.73 m^2) was present in 12.1% (52 of 430). Additional baseline characteristics and echocardiographic parameters stratified by etiology and recMR are summarized in **Table 1**. The median follow-up time was 3.44 years (Q1-Q3: 1.51-3.99 years).

Comparisons between included and excluded patients are provided in Supplemental Table 1. Excluded patients were older, had higher STS-PROM scores, and more frequently presented with advanced MR severity, anemia, and impaired renal function, whereas other baseline characteristics were similar. Missingness analyses suggested no major deviation from randomness for baseline and procedural variables. However, inclusion of follow-up variables provided evidence against missing completely at random (Supplemental Table 2).

PROCEDURAL OUTCOMES. Procedural characteristics are summarized in **Table 2**. By study design, all patients had resMR ≤2+ at discharge. resMR at discharge was ≤1+ in 64.4% of patients (297 of 461) and 2+ in 35.6% (164 of 461). Numerically, patients with DMR more frequently had resMR 2+ at discharge than did those with FMR (43.0% [58 of 135] vs 32.5% [106 of 326]; $P = 0.064$) (Supplemental Table 3). A median of 1 device (Q1-Q3: 1.0-2.0 devices) was implanted. One device was used in 64.4% of all patients (297 of 461), 2 devices in 31.2% (144 of 461), and ≥3 devices in 4.3% (20 of 461).

Major periprocedural complications were infrequent. Overall procedural complications occurred in 5.0% of patients (23 of 461), vascular access site complications in 8.5% (39 of 457), bleeding requiring transfusion in 5.6% (26 of 461), and in-hospital bleeding events in 6.5% (30 of 461). No patient required prolonged mechanical ventilation, conversion to surgery, or died during hospitalization. The median length of intensive care unit stay was 1.0 day (Q1-Q3: 1.0-1.0 day), and the median length of total hospital stay was 8.5 days (Q1-Q3: 7.0-13.8 days) (**Table 2**). Procedural outcomes according to recMR status are provided in Supplemental Table 4.

INCIDENCE OF recMR AND ITS IMPACT ON FUNCTIONAL STATUS. At the 6- to 12-month follow-up, recMR occurred in 31.7% of patients (146 of 461) and was similar between FMR and DMR (31.9% [104 of 326] vs 31.1% [42 of 135], respectively) (**Figure 1**). In DMR, recMR was associated with higher baseline systolic pulmonary artery pressure (51.0 mm Hg [Q1-Q3: 37.2-60.2 mm Hg] vs 38.5 mm Hg [Q1-Q3: 31.0-51.8 mm Hg]; $P = 0.035$), whereas in FMR, recMR was associated with a larger left ventricular end-diastolic diameter (LVEDD) (66.0 mm [Q1-Q3: 60.4-73.5 mm] vs 63.0 mm [Q1-Q3: 56.0-70.0 mm]; $P = 0.0010$) and a higher baseline mitral pressure gradient (MPG) (2.0 mm Hg [Q1-Q3: 1.4-3.0 mm Hg] vs 2.0 mm Hg [Q1-Q3: 1.0-2.0 mm Hg]; $P = 0.024$) (**Table 1**). Symptomatic improvement after M-TEER was observed in both groups but was more sustained in patients without recMR. At 12 months, NYHA functional class ≤II was present in 67.6% of patients without recMR (213 of 315) and 60.0% of those with recMR (114 of 191). Corresponding rates at 24 months were 60.0% (114 of 191) and 56.4% (44 of 78), respectively (**Figure 2**). Mixed model analyses demonstrated a significant interaction between recMR status and time, indicating greater symptomatic improvement from baseline to 12-month follow-up in patients without recMR (OR: 2.02; 95% CI: 1.08-3.74; $P = 0.027$) (Supplemental Figure 1). Available functional capacity measures showed similar findings. 6-MWD improved in both groups with numerically greater improvement in patients without recMR (**Table 3**). N-terminal pro-B-type natriuretic peptide

TABLE 1 Study Baseline Characteristics According to recMR Status and Etiology

		DMR (n = 135)			FMR (n = 326)		
	All Patients (N = 461)	Non-recMR (n = 93)	recMR (n = 42)	*P* Value	Non-recMR (n = 222)	recMR (n = 104)	*P* Value
Clinical data							
Male	280/461 (60.7)	46/93 (49.5)	25/42 (59.5)	0.369	143/222 (64.4)	66/104 (63.5)	0.965
Age, y	76.1 (70.3-81.3) NA: 0	80.0 (72.9-84.2) NA: 0	80.9 (76.3-83.0) NA: 0	0.339	74.5 (69.5-79.9) NA: 0	73.7 (68.8-80.2) NA: 0	0.793
BMI, kg/m^2	24.9 (22.9-27.5) NA: 9	25.3 (23.0.9-27.9) NA: 2	25.4 (23.2-26.4) NA: 1	0.831	24.9 (23.2-27.7) NA: 5	24.2 (22.1-26.6) NA: 1	0.040
STS-PROM score, %	3.6 (2.2-5.6) NA: 84	4.4 (2.6-6.1) NA: 18	4.2 (2.6-5.7) NA: 8	0.852	3.7 (2.2-5.7) NA: 43	3.1 (1.9-4.3) NA: 15	0.036
NYHA functional class				0.769			0.567
III	316/450 (70.2)	65/90 (72.2)	28/39 (71.8)		149/217 (68.7)	74/104 (71.2)	
IV	100/450 (22.2)	17/90 (18.9)	9/39 (23.1)		53/217 (24.4)	21/104 (20.2)	
Cardiovascular risk factors							
AHT	328/451 (72.7)	73/92 (79.3)	31/41 (75.6)	0.799	157/216 (72.2)	67/102 (65.7)	0.252
Dyslipidemia	181/436 (41.5)	31/87 (35.6)	17/40 (42.5)	0.586	84/208 (40.4)	49/101 (48.5)	0.218
Nicotine abuse	105/321 (32.7)	15/60 (25.0)	10/28 (35.7)	0.433	59/160 (36.9)	21/73 (28.8)	0.289
DM	118/458 (25.8)	15/90 (16.7)	9/42 (21.4)	0.676	66/222 (29.7)	28/104 (26.9)	0.696
Comorbidities							
CAD	295/453 (65.1)	55/92 (59.8)	21/41 (51.2)	0.464	151/218 (69.3)	68/102 (66.7)	0.736
ICM	185/405 (45.7)	23/84 (27.4)	7/37 (18.9)	0.444	112/192 (58.3)	43/92 (46.7)	0.087
Prior MI	135/445 (30.3)	19/91 (20.9)	8/41 (19.5)	0.999	70/215 (32.6)	38/98 (38.8)	0.345
Prior PCI	188/455 (42.2)	37/90 (41.1)	10/41 (24.4)	0.098	99/214 (46.3)	42/100 (42.0)	0.558
Prior CABG	121/445 (27.2)	25/92 (27.2)	9/40 (22.5)	0.728	57/214 (26.6)	30/99 (30.3)	0.591
COPD	75/461 (16.3)	18/93 (19.4)	8/42 (19.0)	0.999	39/222 (17.6)	10/104 (9.6)	0.088
Prior stroke	61/446 (13.7)	10/92 (10.9)	3/41 (7.3)	0.754	31/213 (14.6)	17/100 (17.0)	0.695
AFib	309/455 (67.9)	71/93 (76.3)	28/42 (66.7)	0.334	147/219 (67.1)	63/101 (62.4)	0.481
Anemia	262/449 (58.4)	50/88 (56.8)	30/41 (73.2)	0.113	128/218 (58.7)	54/102 (52.9)	0.395
eGFR, <30 mL/min per 1.73 m^2	52/430 (12.1)	8/81 (9.9)	3/36 (8.3)	0.999	30/211 (14.2)	11/102 (10.8)	0.506
NT-proBNP level, pg/mL	3,271 (1,610-6,595) NA: 37	2,148 (1,149-4,160) NA: 10	3,102 (1,089-4,803) NA: 4	0.690	3,923 (1,807-7,188) NA: 14	3,589 (1,814-6,942) NA: 9	0.574
Echocardiographic data							
LVEF, %	42.0 (29.2-53.8) NA: 40	55.1 (49.5-62.7) NA: 14	56.1 (48.8-60.2) NA: 4	0.756	36.3 (27.3-46.8) NA: 16	36.3 (25.8-45.6) NA: 6	0.458
LVEDD, mm	63.0 (56.0-70.0) NA 85	57.0 (50.0-65.0) NA: 20	57.0 (52.0-65.0) NA: 9	0.495	63.0 (56.0-70.0) NA: 35	66.0 (60.4-73.5) NA: 21	0.011
LVESD, mm	50.0 (41.0-60.0) NA: 136	41.0 (36.0-47.5) NA: 26	42.0 (37.0-51.0) NA: 14	0.440	53.0 (45.0-62.0) NA: 65	54.0 (47.0-63.0) NA: 31	0.141
mMPG, mm Hg	2.0 (1.3-3.0) NA: 88	2.0 (1.9-4.0) NA: 16	2.0 (1.6-3.0) NA: 12	0.628	2.0 (1.4-3.0) NA: 47	2.0 (1.0-2.0) NA: 13	0.024
sPAP, mm Hg	39.0 (31.0-51.0) NA: 80	38.5 (31.0-51.8) NA: 19	51.0 (37.2-60.2) NA: 10	0.010	40.0 (30.5-51.0) NA: 35	36.5 (31.0-45.0) NA: 16	0.089
TAPSE, mm	17.0 (14.0-20.0) NA: 324	19.0 (15.0-22.0) NA: 72	18.0 (17.7-27.0) NA: 33	0.496	16.0 (14.0-20.0) NA: 146	16.5 (15.0-19.6) NA: 73	0.964

Values are median (Q1-Q3) or n/total n (%).

AFib = atrial fibrillation; AHT = arterial hypertension; BMI = body mass index; CABG = coronary artery bypass graft; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; DM = diabetes mellitus; DMR = degenerative mitral regurgitation; FMR = functional mitral regurgitation; GFR = glomerular filtration rate; ICM = ischemic cardiomyopathy; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; LVESD = left ventricular end-systolic diameter; MI = myocardial infarction; mMPG = mean mitral pressure gradient; MR = mitral regurgitation; NA = not available; NT-proBNP = N-terminal pro-B-type natriuretic peptide; PCI = percutaneous coronary intervention; recMR = recurrent mitral regurgitation; sPAP = systolic pulmonary artery pressure; STS-PROM score = Society of Thoracic Surgeons Predicted Risk of Mortality score; TAPSE = tricuspid annular plane systolic excursion.

levels also declined in both groups after M-TEER. Additional clinical and functional outcome measures stratified by recMR are presented in Supplemental Table 5.

PROGNOSTIC IMPACT OF recMR. The prognostic impact of recMR after initially successful M-TEER is summarized in **Central Illustration**. Using a 12-month landmark analysis, patients without recMR showed

TABLE 2 Procedural Outcomes According to recMR Status and Etiology

	All Patients (N = 461)	DMR (n = 135)		P Value	FMR (n = 326)		P Value
		Non-recMR (n = 93)	recMR (n = 42)		Non-recMR (n = 222)	recMR (n = 104)	
Procedural characteristics							
No. of devices implanted							
≥2 devices	164/461 (35.6)	34/93 (36.6)	17/42 (40.5)	0.808	86/222 (38.7)	27/104 (26.0)	0.033
Procedural duration, min	155.0 (115.0-200.0) NA: 47	145.0 (95.0-202.5) NA: 20	160.0 (120.0-200.0) NA: 5	0.662	155.0 (117.0-200.0) NA: 22	165.0 (124.2-200.0) NA: 10	0.446
Fluoroscopy time, min	29.0 (20.0-43.5) NA: 31	29.5 (20.0-46.2) NA: 7	26.2 (16.7-34.3) NA: 2	0.191	29.0 (20.7-41.1) NA: 16	29.4 (22.0-45.1) NA: 6	0.676
Device time, min	58.0 (37.0-90.0) NA: 391	54.0 (36.5-89.2) NA: 25	63.5 (33.8-94.0) NA: 10	0.518	58.0 (37.0-90.0) NA: 73	57.0 (37.5-91.0) NA: 29	0.889
resMR at discharge							
resMR 2+	164/461 (35.6)	50/93 (53.8)	8/42 (19.0)	<0.001	90/222 (40.5)	16/104 (15.4)	<0.001
Length of hospital stay (d)	8.5 (7.0-13.8) NA: 391	10.0 (7.2-14.5) NA: 79	7.0 (6.2-9.2) NA: 36	0.081	8.0 (7.0-13.0) NA: 190	10.5 (6.0-16.0) NA: 86	0.685
Complications							
Procedural complications	23/461 (5.0)	4/93 (4.3)	2/42 (4.8)	0.999	10/222 (4.5)	7/104 (6.7)	0.565
Puncture site complication	39/457 (8.5)	14/92 (15.2)	3/42 (7.1)	0.306	15/219 (6.8)	7/104 (6.7)	0.999
Bleeding requiring transfusion	26/461 (5.6)	10/93 (10.8)	1/42 (2.4)	0.172	12/222 (5.4)	3/104 (2.9)	0.403
In-hospital bleeding	30/461 (6.5)	9/93 (9.7)	3/42 (7.1)	0.753	13/222 (5.9)	5/104 (4.8)	0.900
Length of ICU stay, d	1.0 (1.0-1.0) NA: 43	1.0 (1.0-1.0) NA: 11	1.0 (1.0-1.0) NA: 4	0.455	1.0 (1.0-1.0) NA: 8	1.0 (1.0-1.0) NA: 8	0.469
Mechanical ventilation >24 h	0/458 (0.0)						
Death within 30 d (%)	0/461 (0.0)						

Values are median (Q1-Q3) or n/total n (%).
ICU = intensive care unit; other abbreviations as in Table 1.

superior survival from 12 months onward than did those with recMR (log-rank, $P = 0.0088$) (Figure 3A). At 4 years, all-cause mortality was 44.8% (95% CI: 38.7%-50.3%) in patients without recMR and 56.3% (95% CI: 47.2%-63.9%) in those with recMR. This association was more pronounced in DMR (log-rank, $P = 0.014$) (Figure 3B), whereas a similar trend in FMR did not reach statistical significance (log-rank, $P = 0.11$) (Figure 3C). In DMR, 4-year all-cause mortality was 38.0% (95% CI: 26.4%-47.8%) without recMR and 60.3% (95% CI: 41.1%-73.2%) with recMR. In FMR, corresponding rates were 47.5% (95% CI: 40.2%-53.9%) and 55.1% (95% CI: 44.1%-63.9%), respectively. In multivariable Cox regression analysis, recMR remained independently associated with increased all-cause mortality after adjustment for age, severely reduced LVEF (≤30%), anemia, severe renal dysfunction (eGFR <30 mL/min per 1.73 m^2), and AFib (adjusted HR: 1.43; 95% CI: 1.06-1.93; $P = 0.019$) (Table 4). Landmark survival analysis stratified by residual MR grade is provided in Supplemental Figure 2.

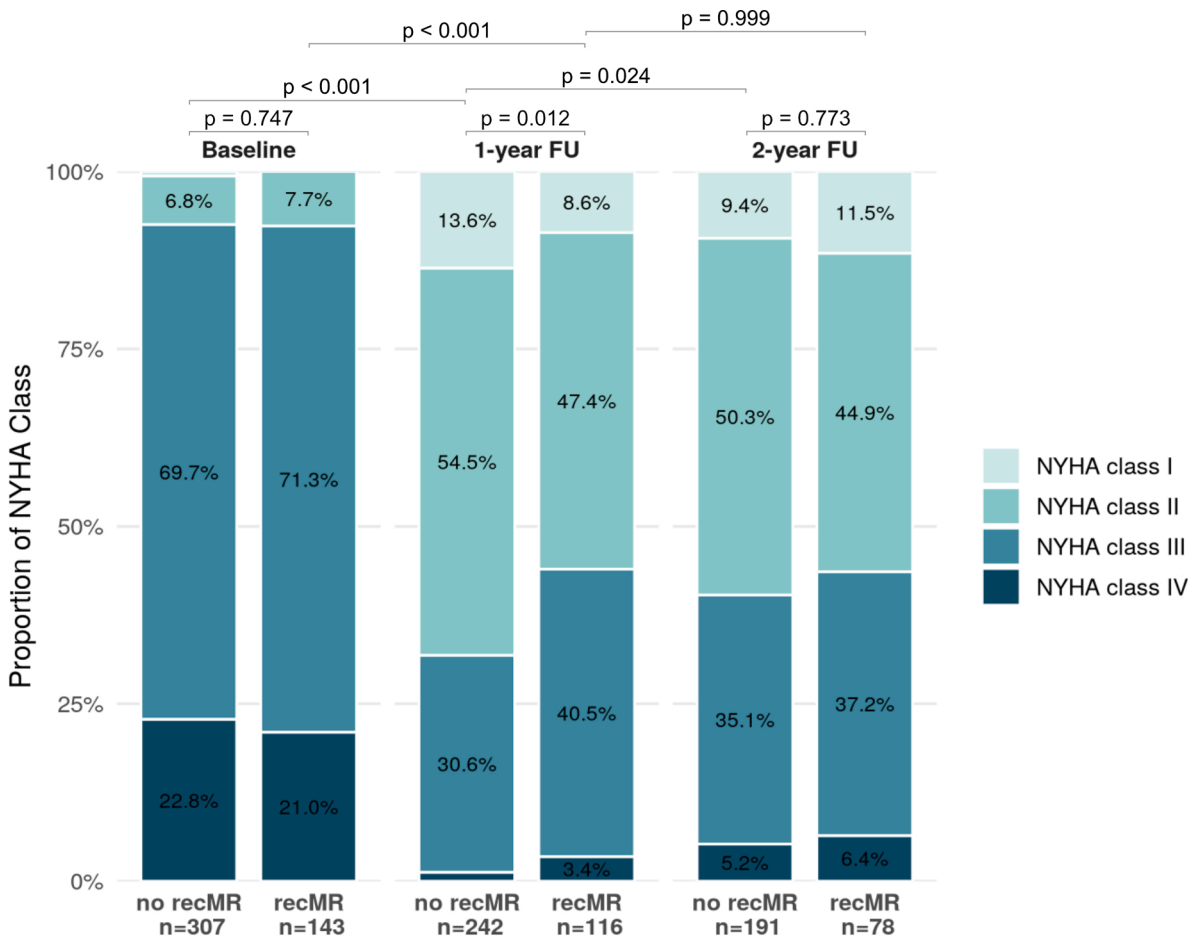
During follow-up, mitral valve reinterventions were performed in 8.9% of patients (41 of 461), including surgical mitral valve procedures in 2.4% (11 of 461). Reinterventions occurred more frequently in patients with recMR than in those without recMR (13.0% [19 of 146] vs 7.0% [22 of 315]; $P = 0.052$) (Supplemental Table 6).

PREDICTORS OF DEVELOPING recMR. In multivariable logistic regression, larger LVEDD (per mm: OR: 1.03; 95% CI: 1.01-1.06; $P = 0.019$) was associated with higher odds of recMR. Higher MPG (per mm Hg: OR: 0.72; 95% CI: 0.57-0.90; $P = 0.005$), ischemic cardiomyopathy (OR: 0.55; 95% CI: 0.32-0.95; $P = 0.033$), and implantation of 2 vs 1 device (OR: 0.49; 95% CI: 0.27-0.85; $P = 0.014$) were associated with lower odds of recMR (Table 5).

DISCUSSION

In this single-center cohort of 461 patients with successful M-TEER (resMR ≤2+ at discharge), recMR was common (32% at 6-12 months) and independently associated with increased long-term mortality. This association was most pronounced in DMR, whereas no significant survival difference was observed in FMR. Baseline and procedural correlates of recMR included larger LVEDD, whereas higher baseline MPG and the use of 2 devices were associated with a lower likelihood of recMR.

FIGURE 2 NYHA Functional Class at Baseline and Follow-Up According to recMR Status



RecMR was not correlated with the severity of NYHA functional class at baseline ($P = 0.747$). Regardless of the presence of recMR, patients exhibited a significant improvement in NYHA functional class. In addition, a significant difference was observed in the extent of improvement between patients with recMR and those without recMR during the 1-year FU period ($P = 0.012$). FU = follow-up; recMR = recurrent mitral regurgitation.

PREVALENCE AND DEFINITIONS OF recMR. Our recurrence rate aligns with prior reports, although comparisons are limited by heterogeneous recMR definitions. Prior registries reported recMR rates of approximately 20% to 30% after initially successful M-TEER, with variability across DMR and FMR.[16] Other contemporary data sets reported lower 12-month recurrence (approximately 9%).[6] Differences in recurrence definitions, device technology, and operator experience may contribute to this variability.

The Endovascular Valve Edge-to-Edge Repair Trial (EVEREST) II trial documented resMR ≥3+ in 12% at 5 years, though differentiating early failure from later recurrence is challenging.[17] Prior studies variably defined recMR on the basis of absolute MR thresholds (eg, ≥2+ or ≥3+), whereas our definition focused on dynamic changes relative to the postprocedural result.[6,18,19] We adopted a clinically pragmatic definition of recMR, any ≥1-grade increase in MR severity between discharge and 6 to 12 months, to capture the full spectrum of early deterioration after an initially successful intervention, and this may partly explain the higher observed recurrence rate in our cohort. These findings underscore the need for harmonized definitions of recMR balancing clinical relevance and reproducibility.

MECHANISMS AND PREDICTORS OF recMR. recMR after initially successful M-TEER is likely multifactorial. In FMR, progressive left ventricular or annular remodeling with ongoing leaflet tethering can deteriorate the regurgitant orifice despite an initially adequate edge-to-edge approximation.[20] In DMR,

TABLE 3 Clinical and Functional Outcomes According to recMR Status and Etiology

		DMR (n = 135)			FMR (n = 326)		
	All Patients (N = 461)	Non-recMR (n = 93)	recMR (n = 42)	*P* Value	Non-recMR (n = 222)	recMR (n = 104)	*P* Value
NYHA functional class				0.769			0.567
III	316/450 (70.2)	65/90 (72.2)	28/39 (71.8)		149/217 (68.7)	74/104 (71.2)	
IV	100/450 (22.2)	17/90 (18.9)	9/39 (23.1)		53/217 (24.4)	21/104 (20.2)	
NYHA functional class at 12-mo follow-up				0.194			0.331
I	43/358 (12.0)	7/73 (9.6)	1/33 (3.0)		26/169 (15.4)	9/83 (10.8)	
II	187/358 (52.2)	38/73 (52.1)	14/33 (42.4)		94/169 (55.6)	41/83 (49.4)	
III	121/358 (33.8)	28/73 (38.4)	17/33 (51.5)		46/169 (27.2)	30/83 (36.1)	
IV	7/358 (2.0)	0/73 (0)	1/33 (3.0)		3/169 (1.8)	3/83 (3.6)	
NYHA functional class at 24-mo follow-up				0.877			0.598
I	27/269 (10.0)	7/56 (12.5)	3/22 (13.6)		11/135 (8.1)	6/56 (10.7)	
II	131/269 (48.7)	23/56 (41.1)	9/22 (40.9)		73/135 (54.1)	26/56 (46.4)	
III	96/269 (35.7)	23/56 (41.1)	10/22 (45.5)		44/135 (32.6)	19/56 (33.6)	
IV	15/269 (5.6)	3/56 (5.4)	0/22 (0)		7/135 (5.2)	5/56 (8.9)	
6-MWD, m	207.0 (89.5-316.8) NA: 157	205.0 (120.0-318.8) NA: 35	200.0 (74.0-300.0) NA: 13	0.889	207.0 (92.5-315.8) NA: 76	222.0 (80.0-307.5) NA: 33	0.641
6-MWD at 12-mo follow-up, m	270.0 (150.0-390.0) NA: 199	240.0 (150.0-375.0) NA: 44	180.0 (60.0-289.5) NA: 16	0.074	330.0 (180.0-420.0) NA: 93	277.5 (165.0-390.0) NA: 46	0.308
6-MWD at 24-mo follow-up, m	300.0 (150.0-405.0) NA: 297	285.0 (180.0-390.0) NA: 57	165.0 (120.0-285.0) NA: 30	0.102	360.0 (175.0-420.0) NA: 139	240.0 (130.0-420.0) NA: 71	0.309
4-y all-cause mortality, n (Kaplan-Meier estimate; 95% CI)	-	32 (38%; 26.4-47.8)	23 (60.3%; 41.1-73.2)	-	101 (47.5%; 40.2-53.9)	56 (55.1%; 44.1-63.9)	-

Values are presented as median (Q1-Q3) or n/total n (%), or number of events (Kaplan-Meier estimate, 95%-CI), as appropiate.
6-MWD = 6-minute walking distance; other abbreviations as in Table 1.

ongoing leaflet degeneration and calcification may predominate. Device-related factors such as partial leaflet detachment or suboptimal grasp may further contribute to recurrence.[20-23]

In our cohort, larger LVEDD was associated with recMR, likely reflecting greater coaptation gaps and tethering forces that reduce repair durability.[24]

TABLE 4 Multivariable Cox Regression Analysis of Predictors of All-Cause Mortality

	HR (95% CI)	*P* Value
recMR	1.43 (1.06-1.93)	0.019
Age	1.00 (0.99-1.02)	0.600
LVEF <30%	1.35 (0.96-1.90)	0.083
Anemia	1.80 (1.32-2.47)	<0.001
eGFR <30 mL/min per 1.73 m^2	1.14 (0.73-1.76)	0.567
AFib	0.94 (0.68-1.28)	0.684

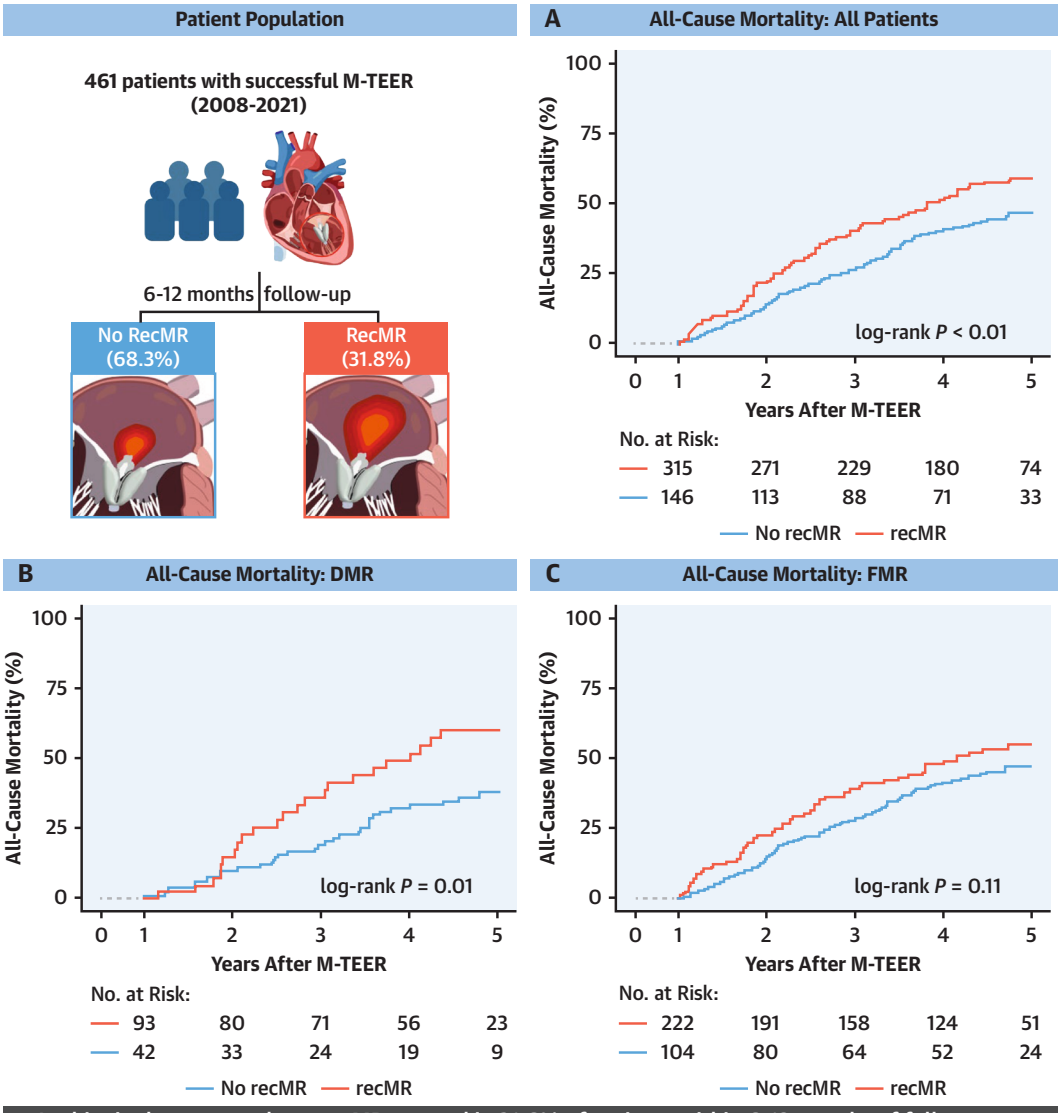
Multivariable Cox regression model constructed using a theory-driven approach including clinically relevant covariates selected a priori on the basis of their known association with mortality after M-TEER.
M-TEER = mitral transcatheter edge-to-edge repair; other abbreviations as in Table 1.

Conversely, higher baseline MPG was associated with lower odds of recMR, possibly marking advanced remodeling. These findings suggest that more advanced ventricular remodeling and reduced leaflet apposition predispose to recurrence, whereas higher baseline gradients reflect tighter coaptation and were protective against recMR. Finally, implantation of 2 devices was linked to greater stability, aligning with biomechanical reasoning by providing additional support to the leaflets.[25] These findings are consistent with a mechanism linking advanced left ventricular remodeling to recurrence and suggest that more extensive device implantation may mitigate residual leaflet malcoaptation.

IMPACT ON LONG-TERM OUTCOMES. Prior analyses, including the Cardiovascular Outcome Assessment of the MitraClip Percutaneous Therapy for Heart Failure Patients with Funtional Mitral Regurgitation (COAPT) trial, demonstrated that the degree of resMR after M-TEER is a key determinant of prognosis.[26] In contrast, the present study specifically focused on the prognostic relevance of recMR after initially successful

CENTRAL ILLUSTRATION Study Overview and Main Findings

Prognostic Impact of Recurrent MR and Etiology Following Successful M-TEER

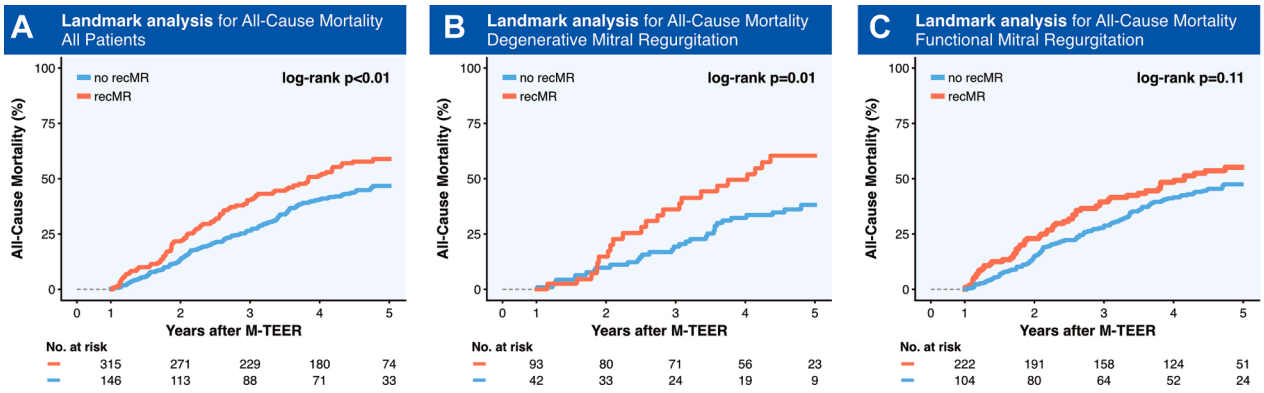


- In this single-center cohort, recMR occured in 31.6% of patients within 6-12 months of follow-up, with comparable recurrence rates across MR etiologies.
- Symptomatic improvement after M-TEER was observed in both groups.
- RecMR was independently associated with increased long-term mortality (adjusted HR: 1.43; $P = 0.019$). This finding was seen in patients with DMR (4-year all-cause mortality: no recMR 38.0% vs recMR 60.3%; $P = 0.014$). whereas no statistically significant survival difference was observed in FMR (4-year all-cause mortality: no recMR 47.5% vs recMR 55.1%; $P = 0.11$).
- Optimized follow-up strategies are essential for early detection and timely management of recMR, while durable MR reduction remains the central therapeutic objective after M-TEER.

Pauschinger C, et al. JACC Cardiovasc Interv. 2026;19(20):2743–2755.

Among 461 patients with initially successful M-TEER (residual MR ≤2+ at discharge), recMR developed in 31.6% of patients during the 6- to 12-month follow-up, with comparable recurrence rates across MR etiologies. Kaplan-Meier landmark analysis demonstrates higher long-term all-cause mortality in patients with recMR in the overall cohort (A), with a more pronounced association in DMR (B), whereas no statistically significant survival difference was observed in FMR (C). DMR = degenerative mitral regurgitation; FMR = functional mitral regurgitation; MR = mitral regurgitation; M-TEER = mitral transcatheter edge-to-edge repair; recMR = recurrent mitral regurgitation.

FIGURE 3 Impact of recMR on All-Cause Mortality



(A) Patients without recMR showed superior survival from 12 months onward than did those with recMR ($P = 0.0088$). (B) Among patients with degenerative mitral regurgitation, those with recMR demonstrated significantly reduced survival ($P = 0.014$). (C) In contrast, recMR in patients with functional mitral regurgitation did not significantly impact survival ($P = 0.11$). M-TEER = mitral valve transcatheter edge-to edge repair; recMR = recurrent mitral regurgitation.

MR reduction (resMR ≤2+ at discharge). Although resMR reflects procedural success, recMR reflects the durability of the repair over time, a dimension that has been less well characterized. Importantly, our study differs conceptually from prior work by isolating a population with initially successful MR reduction and examining subsequent changes in MR severity. This approach allows differentiation between procedural success and durability of repair. Although early resMR has consistently been shown to predict outcomes, the clinical impact of recMR after an initially favorable result remains less well defined. Our findings suggest that loss of MR reduction over time may represent a distinct and clinically relevant risk factor. From a 12-month landmark onward, absence of recMR was associated with improved survival, particularly in DMR, where valvular pathology is the principal driver of risk and M-TEER directly targets the underlying disease mechanism. In DMR, recurrence may reflect loss of an initially effective intervention or progression of valvular pathology. In contrast, in FMR, MR is largely a consequence of atrial or ventricular remodeling and clinical outcomes are closely linked to the severity and progression of the underlying ventricular disease. In this setting, optimization of guideline-directed medical therapy and reverse-remodeling strategies influence outcomes, potentially attenuating the relative impact of recMR on long-term mortality.[27] Data regarding longitudinal changes in guideline-directed medical therapy were not available in the present study and may have influenced the observed association. This etiology-specific heterogeneity supports an individualized post-procedural management approach.

Despite the observed association between recMR and long-term mortality, symptomatic improvement after M-TEER was observed in both groups, although patients without recMR demonstrated greater early improvement. Mixed model analyses showed significantly greater symptomatic improvement from baseline to 12-month follow-up in patients without recMR; however, differences in symptomatic status became less apparent at later follow-up.

TABLE 5 Predictors of the Occurrence of Recurrent Mitral Regurgitation

	OR (95% CI)	*P* Value
Mean mitral gradient	0.72 (0.57-0.90)	0.005
Dyslipidemia	1.54 (0.91-2.61)	0.109
Ischemic cardiomyopathy	0.55 (0.32-0.95)	0.033
Left ventricular end-diastolic diameter	1.03 (1.01-1.06)	0.019
≥2 devices	0.49 (0.22-0.85)	0.014

CLINICAL IMPLICATIONS AND FUTURE DIRECTIONS. Given the prevalence and prognostic impact of recMR, our study underscores the need for careful patient selection and individualized treatment strategies. Although M-TEER has emerged as a viable option for high-risk patients, recMR remains a critical challenge, especially in DMR patients. Where anatomically feasible, durable MR minimization, even at the expense of a borderline postprocedural gradient, may be justified, particularly in DMR, given the adverse prognostic implications of recMR.[3,28,29]

FOLLOW-UP STRATEGIES. Patients with recMR after M-TEER experience worse outcomes than do those with durable MR reduction, underscoring the

importance of optimized follow-up strategies for early detection and management.

Current guidelines recommend regular clinical evaluation, serial TTE, multidisciplinary heart team evaluation, optimization of guideline-directed medical therapy, and consideration of repeat intervention when appropriate.[2] However, these guidelines do not provide specific recommendations for patients at a high risk of recMR.

Emerging evidence suggests that intensified follow-up may improve outcomes in this subgroup. For example, Bartkowiak et al[30] showed that routine transesophageal echocardiography at 6 months post-M-TEER identified clinically relevant complications often missed by TTE alone. This highlights the potential value of structured, protocol-driven surveillance strategies beyond standard care.

Given the procedural complexity and risk of recurrence, follow-up at specialized heart valve centers—where comprehensive imaging, multidisciplinary expertise, and timely intervention are available—should be considered. Further research is needed to refine risk-adapted surveillance protocols.

STUDY LIMITATIONS. This retrospective single-center analysis lacked an independent echocardiographic core laboratory. Echocardiographic assessments were performed in routine clinical practice, and interobserver variability and blinding to prior MR assessments were not systematically evaluated. Therefore, misclassification of semiquantitative MR grading cannot be excluded, which may affect the precision of MR severity assessment particularly in the post-M-TEER setting. Our definition of recMR (≥1-grade increase) was broader than that in some studies, which may have led to higher recurrence estimates.

Mechanisms underlying recMR were not systematically adjudicated, including differentiation between device-related factors (eg single-leaflet device attachment, loss of leaflet insertion, or suboptimal grasp) and progressive ventricular or annular remodeling, limiting mechanistic interpretation and potential etiologic comparisons. The study period spanned more than a decade (2008-2021), during which device technology and procedural experience evolved substantially and may have influenced recurrence rates and outcomes. In addition, the analytic cohort was restricted to patients with available etiology classification and sufficient follow-up. Excluded patients differed in several baseline characteristics, introducing the possibility of selection bias and limiting generalizability.

Our findings apply specifically to patients with initially successful M-TEER (resMR ≤2+ at discharge) and should not be extrapolated to patients with severe resMR, whose prognosis is already known to be strongly influenced by the immediate post-procedural result.

Finally, structured reintervention pathways were not systematically assessed, limiting insights into management strategies after recMR.

CONCLUSIONS

recMR after initially successful M-TEER may be associated with increased mortality, with a more pronounced signal in DMR patients, underscoring that not only the immediate procedural result but also the durability of MR reduction is critical for long-term outcomes. Durable MR reduction therefore remains the core therapeutic target. Specialized heart valve centers play a crucial role in risk-adapted surveillance, enabling early detection of recurrence and timely reintervention.

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PERSPECTIVES

WHAT IS KNOWN? Residual mitral regurgitation is one of the strongest predictors of mortality after M-TEER. In contrast, recurrent mitral regurgitation (recMR) after an initially successful procedure remains insufficiently studied and poorly understood.

WHAT IS NEW? This study shows that recMR independently predicts long-term mortality after initially successful M-TEER, with the strongest impact observed in degenerative MR. These findings highlight the importance of achieving durable MR reduction and suggest that intensified follow-up at specialized heart valve centers may improve clinical outcomes.

WHAT IS NEXT? Future research should focus on refining patient selection, refining procedural techniques, and enhancing postprocedural surveillance strategies to identify and manage high-risk patients early and preserve the long-term therapeutic benefits of M-TEER.

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KEY WORDS mitral regurgitation, mitral transcatheter edge-to-edge repair, recurrent mitral regurgitation

APPENDIX For supplemental tables and figures, please see the online version of this paper.