Transcatheter edge-to-edge repair in acute mitral regurgitation following acute myocardial infarction: Recent advances

Dan Haberman[1], Shani Dahan[2], Lion Poles[1], David Marmor[3], Mony Shuvy[3]

[1]Heart Center, Kaplan Medical Center, Rehovot, Israel; Affiliated with the Hebrew University of Jerusalem, Faculty of Medicine, Jerusalem, Israel
[2]Department of Cardiology, Assuta Ashdod Medical Center; Affiliated to the Ben Gurion University of the Negev, Ashdod, Israel
[3]Jesselson Integrated Heart Center, Shaare Zedek Medical Center, Faculty of Medicine, Hebrew University of Jerusalem, Jerusalem, Israel

Correspondence to:
Dan Haberman, MD,
Heart Center, Kaplan Medical Center, Rehovot, Israel;
Affiliated with the Hebrew University of Jerusalem, Faculty of Medicine,
Pasternak St. P.O.Box 1,
Rehovot 76100, Israel,
phone: +972 8 944 13 35,
e-mail: haberdan@gmail.com

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ABSTRACT

Acute mitral regurgitation (MR) is not a rare finding following acute myocardial infarction (AMI). It may develop due to papillary muscle rupture (primary MR) or due to rapid remodeling of the infarcted areas leading to geometric changes and leaflets tethering (secondary or functional MR). The clinical presentation can be catastrophic, with pulmonary edema and refractory cardiogenic shock. Acute MR is a potentially life-threatening complication and is linked to worse clinical outcomes. Until recently, medical treatment or mitral valve surgery were the only established treatment options for these patients. However, there is growing evidence for the benefits of safe and effective trans-catheter interventions in this condition, specifically transcatheter edge-to-edge repair (TEER). We aimed to review the current role of TEER in post-MI acute MR patients, focusing on different etiologies.

Key words: mitral regurgitation, myocardial infarction, cardiogenic shock, papillary muscle rupture, trans-catheter edge-to-edge repair

INTRODUCTION

Acute mitral regurgitation (MR) is not a rare finding following acute myocardial infarction (AMI) and is an independent predictor of long-term cardiovascular outcomes [1]. It varies in its mechanism, severity, clinical hemodynamic consequences, and prognosis.

Acute MR may represent a medical emergency and a life-threatening complication. The sudden increase in left atrial volume due to the regurgitant jet, superimposed on a previously normal non-compliant left atrium, dramatically increases filling pressure. This often leads to the abrupt development of flush pulmonary edema. Moreover, because a large fraction of the blood ejected by the non-dilated left ventricle (LV) goes backward across the mitral valve, effective stroke volume is acutely reduced. This may manifest as low cardiac output state ranging from weakness to cardiogenic shock. Consequently, the neurohormonal response leads to a compensatory increase in vascular resistance, which further exacerbates the regurgitation, creating a vicious cycle. Unless rapidly diagnosed and treated, this dreaded complication is associated with high morbidity and mortality.

Acute MR is often misdiagnosed on physical examination, especially in severely deteriorated hemodynamic patients. The pathophysiology involves rapid equalization of left ventricular and left atrial pressure in mid-systole. This, together with low systemic blood pressure and heart sounds obscured by respiratory distress, often leads to a soft or absent murmur [2]. When suspected, the diagnosis can be usually made by transthoracic or transesophageal echocardiography [3]. The combination of pulmonary edema and cardiogenic shock with a hyperdynamic LV on echocardiography should raise suspicion for acute MR, even if the flow across the mitral valve is laminar by color Doppler. Further thorough echocardiographic evaluation, including continuous wave Doppler of mitral inflow and pulsed Doppler of pulmo-

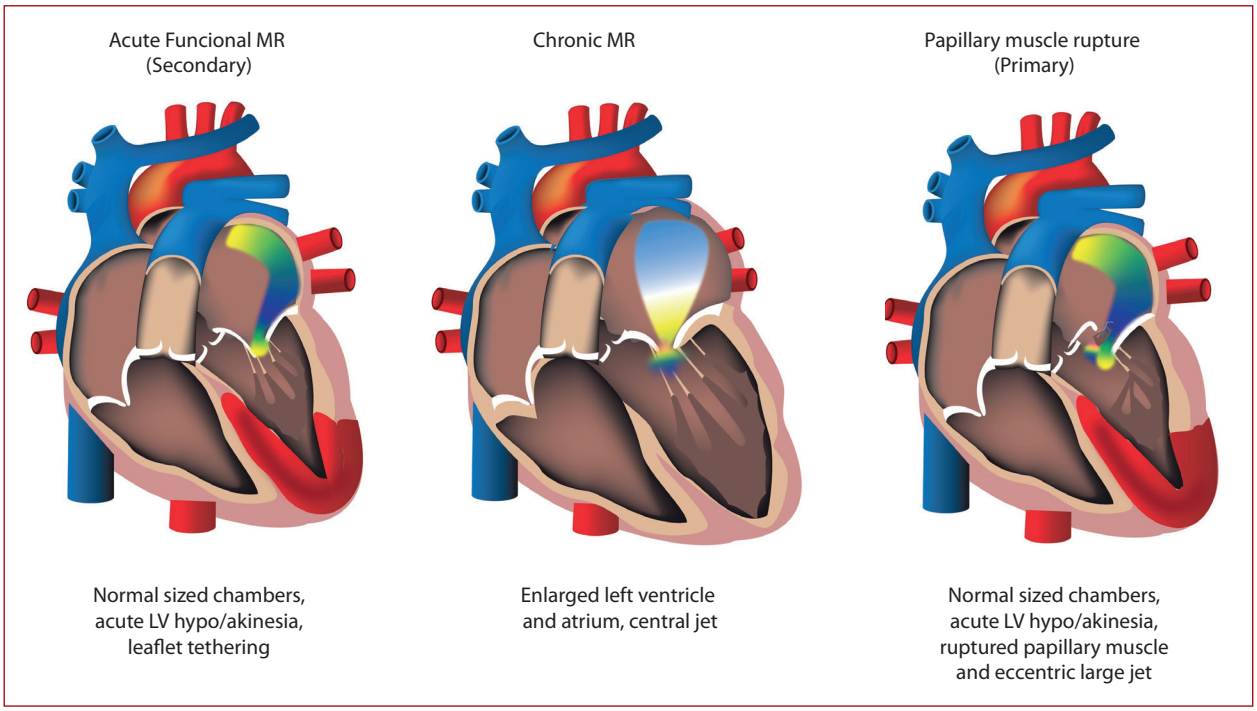

Figure 1. Mitral regurgitation mechanism — chronic, acute functional MR and papillary muscle rupture.
Schematic representation comparing three mechanisms — chronic MR, acute functional MR, and papillary muscle rupture ("acute primary"). In acute functional MR, a secondary mechanism, the left atrium and ventricle are in normal size, and LV infarction is visible causing leaflet tethering. In chronic MR, the left atrium and ventricle are enlarged to compensate for the pressure and volume overload, the annulus is dilatated, and the central jet is seen. In papillary muscle rupture, LV infarction is seen, ruptured muscle with a chord attached to the free leaflet is causing a wide eccentric jet.

Abbreviations: MR, mitral regurgitation; LV, left ventricle

nary veins, is often key to echocardiographic diagnosis of severe acute MR [4].

Identifying the etiology of acute MR is the cornerstone in care and management of these unstable patients. Sudden disruption of the mitral apparatus following acute MI can develop due to papillary muscle rupture (the primary etiology) or due to leaflet tethering related to the abrupt onset of regional or global left ventricular dysfunction (secondary etiology, also known as functional ischemic MR), as shown in Figure 1 [5]. Until recently, the only available therapeutic intervention for patients with severe acute MR in the setting of AMI was surgical repair of the valve.

In recent years, mitral valve transcatheter edge-to-edge repair (TEER) has become increasingly common in treating severe mitral regurgitation. TEER is based on a surgical technique introduced in the early 1990s [6], aiming to approximate the anterior and posterior leaflets of the mitral valve at the origin of the regurgitant jet. Initially, TEER was approved for patients with severe degenerative MR and high surgical risk [7]. In the recent European valvular diseases' guidelines, TEER is recommended for symptomatic patients with severe chronic primary MR, eligible for TEER, and with high surgical risk as a IIb indication [8]. Following the results of the Cardiovascular Outcomes Assessment of the MitraClip Percutaneous Therapy for Heart Failure Patients with Functional Mitral Regurgitation (COAPT) trial [9], TEER is also recommended for patients with severe refractory functional MR. TEER was recently upgraded to a IIa indication for selected patients with symptomatic severe secondary MR who fulfill the COAPT criteria, suggesting an increased chance of responding. TEER is also indicated as a IIb indication for those high-risk patients who do not fulfill the criteria, after careful evaluation and heart team discussion [8].

This review discusses the recent advances in the management and care of patients with acute MR following AMI, focusing on the safety and effectiveness of TEER in this patient population.

PRIMARY ETIOLOGY

Papillary muscle rupture (PMR) is the principal primary etiology of acute MR in the post-MI setting. Complete rupture of the papillary muscle is uncommon, occurring in 1%–3% of patients with AMI but is associated with severe clinical presentation, rapid deterioration, and poor prognosis. About half of these patients present with pulmonary congestion which may deteriorate rapidly to cardiogenic shock [10]. It usually occurs within 5 days following AMI with a mortality rate as high as 80% without urgent intervention [11].

The incidence of PMR, similar to other mechanical complications of AMI, has declined in the reperfusion era [12]. However, in-hospital mortality remains high with rates of 20%–40% [13]. PMR occurs more frequently in older individ-

uals and those with prior MI [14]. The posteromedial, rather than the anterolateral, papillary muscle is usually involved, due to its single blood supply from the right coronary or circumflex arteries.

Acute management of these patients requires hemodynamic stabilization and treatment of pulmonary edema. In hemodynamically stable patients, intravenous nitroglycerin or nitroprusside can be used for afterload reduction, along with diuretics for symptomatic congestion relief [15]. In unstable patients, initial medical treatment may include vasopressors as-hemodynamic support. The use of positive pressure ventilation can have additional benefits due to improved gas exchange and reduced LV preload and afterload. However, high positive end-expiratory pressure may impair hemodynamics as well, and thus many patients will eventually require invasive ventilation. Further stabilization with mechanical support devices (MCS) may be warranted, especially in patients undergoing surgery. The use of an intra-aortic balloon pump (IABP) is recommended by clinical guidelines in the case of mechanical complications of MI mainly due to afterload reduction which may further decrease MR and increase cardiac output. The use of IABP was not shown to improve survival, but patients with mechanical complications were excluded from clinical trials [16]. The data regarding other percutaneous mechanical support devices and veno-arterial extracorporeal membrane oxygenation (VA-ECMO) are still limited [17].

Surgical therapy remains the mainstay of treatment in patients with severe primary MR due to PMR, however, surgical risk may be extremely high in some patients. In the SHOCK Trial Registry (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock), only 38% of patients were eligible for mitral valve surgery [18].

In a more recent retrospective analysis of AMI admissions, data derived from the National Inpatient Sample (NIS) showed that only 58% of patients with PMR underwent mitral valve surgery. Older patients and those with comorbidities did not undergo surgery due to prohibitive surgical risk [13].

The outcome data on surgery in acute MR due to PMR is limited. In large case series, 79% of patients underwent mitral valve replacement, the intraoperative mortality was 4.2%, and in-hospital mortality was 25%. EuroSCORE II, complete PMR, and intraoperative IABP were identified as predictors of in-hospital mortality [19].

Current guidelines recommend medical therapy and mitral valve surgery, if feasible, for acute post-MI MR, but do not mention percutaneous therapies at this time [17, 20–22]. Mitral valve replacement is generally the preferred surgical technique in this situation because of its predictability and durability. However, repair should be done if possible, which is more likely in partial PMR. Coronary artery bypass graft (CABG) should be considered as a concomitant surgery for patients with PMR and obstructive coronary disease who failed to receive complete revascularization during PCI [23].

TEER with Mitraclip or Pascal devices is a well-established therapeutic modality for patients with chronic significant MR of both primary and secondary (functional) etiology. Data regarding such procedures in acute settings are limited to case series and registries. Current experience with percutaneous edge-to-edge mitral valve repair in acute primary MR is summarized in Table 1.

Most case studies report on patients in their 5th to 7th decade of life, who present with myocardial infarction and within days of presentation develop rapid deterioration with pulmonary edema and cardiogenic shock. Other characteristic features include high systolic pulmonary artery pressure, left atrial pressure, and the presence of v-wave on invasive hemodynamics.

These patients were treated successfully with 1 to 3 clips with good results and full clinical recovery [24–32]. Two case series were recently published; So et al. [33] performed an institutional review and found 8 patients who underwent emergency TEER, 4 of which were related to post-MI acute MR. Seventy-five percent had chordal rapture, 75% required MCS, and 5 of 8 cases achieved procedural success. Another case series by Chang et al. [34] reported 5 patients with post-MI acute MR in severe clinical condition, cardiogenic shock, and supported by MCS. TEER was performed within 3 days, and procedural success was achieved in all cases. However, only one patient survived to discharge from the hospital.

SECONDARY ETIOLOGY

Ischemic MR is a common and important complication after AMI. It is estimated that the prevalence of ischemic MR after MI is up to 50% [2, 35]. It is known to worsen patient prognosis even with mild to moderate severity. There is considerable clinical heterogeneity in ischemic MR, with some patients being asymptomatic while in others MR may progress rapidly and cause serious clinical deterioration leading to acute heart failure and death [36].

Ischemic cardiomyopathy may be associated with LV remodeling and geometric changes, systolic leaflet tethering, reduced LV closing forces, and mitral annular dilatation. All these changes together result in leaflet mal-coaptation with restricted and incomplete mitral leaflet closure [37]. After MI, the mitral leaflets are elongated as a compensatory mechanism to prevent MR. Additional processes, including leaflet thickening with fibrotic changes, are associated with failure of this compensatory process [38]. This is the most common scenario leading to functional MR. Another less frequent scenario is acute ischemic MR due to acute transient ischemia, with reduced perfusion to either the myocardial walls or papillary muscles. The exact mechanism of acute ischemic MR (acute functional MR) is not fully understood. Recent data suggest cellular and molecular changes in the leaflets themselves. In acute

Table 1. Case studies of papillary muscle rupture treated with edge-to-edge mitral valve repair

Title	Reference	Patients	Cause and mechanism of acute MR	Presentation	Procedure results	Early outcomes	Follow-up
Successful Percutaneous Mitral Valve Repair with the MitraClip System of Acute Mitral Regurgitation due to Papillary Muscle Rupture as Complication of Acute Myocardial Infarction	Bilge M, Alemdar R, Yasar AS. CCI 2014 [S1]	A 60 years-old female PMH: HTN, DM, colorectal carcinoma stable and chronic renal failure	Posterolateral STEMI. PCI to proximal CX and marginal branch, rupture of anterolateral papillary muscle	Pulmonary edema, cardiogenic shock. Mechanical ventilation and IABP. LVEF, 45%; sPAP, 65 mm Hg	7th day, single clip with reduction to trace MR	4 days after MitraClip: minor hemorrhagic stroke	30-day follow-up: MR grade 1+
MitraClip for Papillary Muscle Rupture in Patient with Cardiogenic Shock	Wolff R, Cohen G, Peterson C, et al. CJC 2014 [S2]	A 68-year-old male, late arrival STEMI with Q waves in lateral and posterior leads STS score, 64%; EuroSCORE II, 75%	Latecomer lateral STEMI, with occlusion of the large first obtuse marginal artery Complete rupture of the anterolateral papillary muscle with flail of A2. LVEF, 25%	Cardiogenic shock and ventricular arrhythmia. IABP and inotropes. Mean LAP, 37 mm Hg; v-wave, 55 mm Hg	Device success 2 clips (A2–P2); MR grade 2+; v-wave, 30 mm Hg	MR grade 1–2+, LVEF 30%, LVEDD 51 mm, LVESD 46 mm, normal RV function	3-month: NYHA II, MR grade 1–2+ LVEF 38%, LVEDD 62 mm LVESD 50 mm 6-month: NYHA II
MitraClip Implantation After Acute Ischemic Papillary Muscle Rupture in a Patient with Prolonged Cardiogenic Shock	Bahlmann E, Frerker C, Kreidel F. et al. Ann Thorac Surg. 2015 [S3]	A 77-year-old male, s/p CABG presented with dyspnea and 3-day back pain. Lateral NSTEMI. Log EuroSCORE, 78%	Lateral NSTEMI. Subtotal SVG to the circumflex artery. No PCI performed. Complete rupture of the posterior papillary muscle	Cardiogenic shock and pulmonary edema. IABP and inotropes. Mean LAP, 21 mm Hg; mean PAP, 24 mm Hg; CO, 4.6 l/min; CI, 2.2 l/min/m^2	Device success, 3 clips; MR grade, 0; mean LAP, 22 mm Hg; mean PAP, 26 mmHg; CO, 6.8 l/min; CI, 3.2 l/min/m^2	Alive at 16th postop day	N/A
Percutaneous Mitral Valve Repair with Mitraclip System in a Patient With Acute Mitral Regurgitation After Myocardial Infarction	Rodríguez-Santamarta M, Estévez-Loureiro R, Gualis J, et al. Rev Esp Cardio 2015 [S4]	A 76-year-old male; STS score, 6.7%; Log EuroSCORE, 29.1%	Inferolateral STEMI, successful primary PCI of the proximal circumflex artery, Ischemic asymmetric posterior leaflet tethering, A2–P2 and A3–P3	Pulmonary edema	MR grade (4th day), 1+; 2 clips (A2–P2; lateral to the first one); MR grade, 1+; MV MG < 5 mm Hg	Alive, NYHA I	NYHA I
Acute Mitral Regurgitation Secondary to Papillary Muscle Tear Is Transcatheter Edge-to-Edge Mitral Valve Repair a New Paradigm?	Valle JA, Miyasaka RL, Carroll JD. Circ Cardiovascular interv. 2017 [S5]	A 84-year-old male, LVEF, mildly reduced	Inferior STEMI, successful primary PCI of saphenous vein graft to the right coronary artery. Partial tear of the the posteromedial papillary muscle with flail of A2–A3	Cardiogenic shock, mean LAP, 29 mm Hg; v-wave, 59 mm Hg	3 clips in a "zipper" approach; MR grade, 1+; MV MG, 5 mm Hg; mean LAP, 14 mm Hg; v-wave, 20 mm Hg	Alive	6-week: NYHA, II; MR grade, 1–2+
Use of MitraClip for Postmyocardial Infarction Mitral Regurgitation Secondary to Papillary Muscle Dysfunction	Yasin M, Nanjundappa A, Annie FH, et al. Cureus 2018 [S6]	A 68-year-old male	Inferior NSTEMI, rupture of the posteromedial papillary muscle with flail of the posterior leaflet	Cardiogenic shock, pulmonary edema, v-a ECMO	MR grade (3rd day), 1+; device success	Alive, 5 day after admission: 2 clips (A2–P2, P1-P2), MR. grade: 1+	30-day; MR grade, 1+
Edge-to-edge mitral valve repair for acute mitral valve regurgitation due to papillary muscle rupture: a case report	Papadopoulos K, Chrissoheris M, Nikolaou I, et al. Eur Heart J Case Rep 2019 [S7]	A 85-year-old female, Log EuroSCORE, 43%; STS score, 13%; LVEF, 40%	Anterior STEMI, successful primary PCI of the intermediate artery, Partial rupture of the anterolateral papillary muscle with flail of A1 A2 and P1	Cardiogenic shock, pulmonary edema, IABP and inotropes	Device success, 2 clips (A2–P2, A1-P1) with a "zipping" of the lateral commissure, MR grade, 1–2+; MV area, 2.1 cm^2; MV MG, 6 mm Hg	Alive (postop day 7)	20-month: NYHA II; MR grade, 2+; MV MG, 6 mm Hg

Table 1 (cont.). Case studies of papillary muscle rupture treated with edge-to-edge mitral valve repair

Title	Reference	Patients	Cause and mechanism of acute MR	Presentation	Procedure results	Early outcomes	Follow-up
Successful MitraClip XTR for Torrential Mitral Regurgitation Secondary to Papillary Muscle Rupture as a Complication of Acute Myocardial Infarction	Villablanca PA, Wang DD, Lynch D, et al. Structural heart. 2019 [S8]	A 70-year-old male, LVEF, 60%; STS score, 14.3%	Lateral NSTEMI, successful primary PCI of the proximal and mid-circumflex arteries. Complete rupture of The posteromedial papillary muscle with flail of P2–P3	Cardiogenic shock and pulmonary edema, Impella CP, then exchanged with IABP plus inotropes, mean LAP, 22 mm Hg; V wave, 60 mm Hg; CO, 3.7 l/min; CI, 1.8 l/min/m^2	1 XTR clip (A2–P2); MR grade; 1+; MV MG; 1 mm Hg; mean LAP, 10 mm Hg; V wave, 12 mm Hg; CO, 4.9 l/min; CI, 2.8 l/min/m^2	Alive (postop day 3)	6-month: NYHA I; MR grade, 1+
Transcatheter Mitral Valve Edge-to-Edge Repair with the New MitraClip XTR System for Acute Mitral Regurgitation Caused by Papillary Muscle Rupture	Komatsu I, Cohen EA, Cohen GN, et al. Can J Cardiol. 2019 [S9]	A 55-year-old male, LVEF, 55%	Inferior STEMI, successful primary PCI of culprit single-vessel disease, the posteromedial papillary muscle rupture with anteriorly directed eccentric jet, Coaptation gap, 1 cm; MV area, 6.2 cm^2; MV MG, 3 mm Hg	Pulmonary edema, Cardiogenic shock, Acute kidney injury, IABP, Vasopressors, V wave: 50 mm Hg	2 clip XTR (A2–P2); MR grade, 1–2+; MV MG, 3 mm Hg; MV area, 2.94 cm^2; V wave, 17 mm Hg	Alive	3-month: MR grade, 2+ (eccentric); no HF symptoms
Transcatheter edge-to-edge repair for acute mitral regurgitation with cardiogenic shock secondary to mechanical complication	So CY, Kang G, Lee JC, et al. Cardiovasc Revasc Med. 2022. [S10]ability to wean off MCS, all-cause and cardiovascular mortality at 30-day.\ nRESULTS: Eight patients were identified from institutional review. Detail anatomical analysis found that patients with mechanical MV complications related to myocardial infarction had a lower transseptal height achieved during MitraClip (3.6 ± 0.1 cm vs 4.3 ± 0.3 cm, $P = 0.03$)	24 patients: mean age: 73 ± 12.9; M:F17:7; mean LVEF, 50 ± 11.2%	Chordal rupture, 50%; papillary muscle rupture, 50%; anterolateral, 41.7%; posteromedial, 25%; STEMI, 58.3%	MCS usage: 75%, IABP: 44.4%, Impella: 27.8% ECMO: 16.7%, ECPELLA: 11.1%, MV gradient, pre-MC: 3 ± 1.2. symptoms onset to MC, days: 11 ± 8.2	MV gradient; post-MC, 3.9 ± 1.9; number of clips, median, 2	Final MR >2+ 13%; ≤2+ 87%; final MV gradient ≤5 mm Hg 81.3%; >5 mmHg 18.7%	Ability to wean off MCS: 94.4%; device success, 68.8%; 30-day CV mortality, 4.5%; 30-day all-cause mortality, 9.1%
Transcatheter Edge-to-Edge Repair for Acute Mitral Regurgitation due to Postinfarction Papillary Muscle Rupture	Chang CW, Romero S, Price MJ. JSCAI 2022 [S11]	5 patients: mean age: 75 ± 12.7; M:F, 2:3	MR severity >4+ (100%); preprocedural LVEF, 62.4% ± 14.5%	Cardiogenic shock, IABP: 100%, IABP+VA-ECMO: 20%. EuroSCORE II, mean: 31.8 ± 6.4	Number of clips, mean, 2 ± 0.7	Postprocedural LVEF, 46.4% ± 18.4%; MR severity after MitraClip, trace (60%); mild (20%); moderate (20%)	Hospital outcome: discharged (20%), deceased (80%)

Supplementary references are available at https://journals.viamedica.pl/kardiologia_polska

Abbreviations: CABG, coronary artery bypass graft; CAD, coronary artery disease; CHF, congestive heart failure; CO, cardiac output; CI, cardiac index; CS, cardiogenic shock; ECMO, extracorporeal membrane oxygenation; EF, ejection fraction; FU, follow-up; IABP, intra-aortic balloon pump; INTERMACS, interagency registry for mechanically assisted circulatory support; MCS, mechanical circulatory support; MI, myocardial infarction; MR, mitral regurgitation; MV, mitral valve; NYHA, New York Heart Association functional class; PCI, percutaneous coronary intervention; LV, left ventricle; STEMI, ST-segment elevation myocardial infarction; TEER, trans-catheter edge-to-edge repair.

MR, the mitral apparatus remains dynamic, and valvular tenting is modest but linked to severity [39].

Tethering of leaflets is observed both in inferior-posterior or MI and in anterior MI. Experimental data showed that antero-apical MI, involving all apical segments, can mechanically displace papillary muscles, causing MR even without basal and mid-inferior wall motion abnormalities [40]. The vicious cycle of ischemia and MR lead to adverse remodeling and eventually to LV dilatation and heart failure. Interestingly, in ischemic MR animal models, only early MR repair (up to 1 month after the induction of MI) prevented adverse LV remodeling, suggesting that there is a point of no return for MR repair [41].

Early revascularization in this patient population has the potential to improve MR degree both due to ischemia alleviation and potential rescued myocardium which is related to LV remodeling [5]. Early reperfusion of myocardial infarction has reduced mortality and mechanical complications including ischemic MR [10].

The literature about the prevalence of functional MR with different culprit coronary arteries is controversial. Some studies suggest that inferior-posterior or lateral MI are more likely to be associated with functional MR, while others suggest the anterior wall as being more frequently involved. It is known that clinical outcomes with functional MR are worse for anterior-wall MIs [42].

The use of trans-catheter treatment options for patients with ischemic MR is rapidly growing, TEER is a well-established therapeutic modality for stable patients with chronic functional MR. Patients with acute condition were excluded from clinical trials and most registries. However, the data for treatment of acute functional MR post-MI are more established than for acute primary MR. The data are limited to case studies, case series, and registries [43–47] which are currently not mentioned in clinical guidelines. Current experience with percutaneous edge-to-edge mitral valve repair in acute secondary MR is listed in Table 2.

Our group published a large case series, in which 20 patients with acute functional MR following MI were treated with TEER (mean age 68 ± 10 years, 70% females, 40% in cardiogenic shock). Procedural success was 95% with MR reduction, improvement of hemodynamic parameters, and overall 30-day survival of 90% [44]. Additional series was published that included 44 patients with post-MI acute functional MR [46] (mean age 70 ± 11 years, with 32 % on MCS). Procedural success was 87% with 91% 30-day survival. The next study from the IREMMI group included 93 patients and investigated the role of cardiogenic shock in the same clinical condition. The mean age of patients was 70 ± 11 years, with 54% in cardiogenic shock at the time on TEER, of those 66% with MCS. Procedural success was high and did not differ between the two groups. Thirty-day mortality was low and numerically higher in the CS group (10% vs. 2.3%) but was not statistically significant. The combined event of mortality and/or rehospitalization was comparable (26% vs. 28%) [48].

Another article from the IREMMI group focused on the effect of TEER on LV function. One hundred and five patients treated with TEER for acute functional MR following MI were divided into two groups based on a cut-off of the left ventricular ejection fraction (LVEF) 35%. The mortality rates in the two groups were comparable up to 1 year (11% of LVEF <35% and 7%) so was the 3-month re-hospitalization rate (19% for LVEF < 35% and 12%). The authors concluded that AMI patients with severe LV function may benefit from TEER and should not be excluded [49].

Ultimately, the largest study so far in the field from our group compared 3 treatment options: conservative (medical) treatment, surgery, and percutaneous (TEER), as shown in Figure 2 [47]. Four hundred and seventy-one patients with severe functional MR following AMI were included in a retrospective registry from 21 centers (mean age 73 ± 11, 43% females). Two hundred and sixty-six were managed conservatively and 205 underwent mitral valve intervention based on the physician's clinical decision. Patients in the intervention group were in worse clinical condition but had lower in-hospital (11% vs. 27%; P <0.01) and 1-year mortality (16% vs. 36%; P <0.01) compared with those treated conservatively (Figure 2). In the mitral valve intervention group, 106 patients were treated with mitral valve surgery (2/3 replacement and 1/3 repair), and 99 underwent TEER. Patients in the TEER group were older, had more prior MI and CABG, and presented in worse clinical condition. Patients in the surgical groups were treated earlier (MI to intervention; 12 days vs. 19 days; P <0.01). The procedural success did not differ between the two groups (93 vs. 92%; P = 0.53), but surgery had more complications (34% vs. 6%; P <0.01) and higher in-hospital mortality (16% vs. 6%; P <0.01) and 1-year mortality (31% vs. 17%; P = 0.04). Interestingly, after excluding patients who died in the hospital, no difference in 1-year mortality was observed.

The above data suggest that TEER is feasible and safe in patients with acute MR following MI, especially when considering the severe clinical condition of these patients and their high surgical risk. In this subgroup of patients, both mitral valve surgery and TEER achieved MR reduction, hemodynamic improvement, and lower 1-year mortality and rehospitalizations when compared with conservative treatment. However, we cannot generalize the results on longer-term outcomes to date, as only 1-year outcome results have been published. Moreover, one should keep in mind that ischemic MR is a complex lesion with multiple contributing mechanisms, and the optimal treatment may vary in individual patients.

CONCLUSIONS

Acute MR following AMI is an unfortunate and fairly common clinical situation with high morbidity and mortality. The etiology of MR and patient characteristics have great implications for clinical presentation and treatment options. TEER is an emerging treatment option in this clinical scenario that should be taken into consideration along

Table 2. Case studies and registries of acute functional MR treated with edge-to-edge mitral valve repair

Title	Reference	Patients	Cause and mechanism of acute MR	Presentation	Procedure results	Early outcomes	Follow-up
Percutaneous Mitral Valve Repair for Acute Mitral Regurgitation after an Acute Myocardial Infarction	Estévez-Loureiro R, Arzamendi D, Freixa X, et al. J Am Coll Cardiol. 2015. [S12]	5 patients: mean age, 68; median EuroSCORE II 29% and 3of 5 in CS	Functional. 2 STEMI, 3 NSTEMI	Cardiogenic shock, IABP	Device success Complications	1 death in a week	Median FU 317 days, 80%; NYHA 1–2; 80% MR <:2
Salvage MitraClip in severe secondary mitral regurgitation complicating acute myocardial infarction: data from a multicenter international study	Haberman D, Taramasso M, Czarnecki A, et al. Eur J Heart Fail. 2019. [S13]	20 patients: mean age, 68.1; F:M, 14:20	Functional: STEMI, 13; Anterior wall, 11; Inferior-lateral wall, 9; Multi-vessel; CAD, 17 (85%)	All patients had moderate to severe MR (3–4+); mean EF, 35.9 ± 12.5%; severe LV dysfunction, 7 (35%); cardiogenic shock and IABP/vasopressors, 8 (40%); mechanical ventilation, 6 (30%)	MR reduction to 1+ was achieved in 12 patients and to 2+ in 7 pts; pulmonary artery pressure reduction; left atrial v-wave reduction	1 patient died after a leaflet tear and urgent MVR surgery, 1 patient had an access site hematoma, 95% were discharged after the procedure, 1 patient died out of hospital, after 3 weeks due to unknown cause	Median FU, 15 months; functional capacity improvement in 17 pts (85%) – NYHA I/II, EF and pulmonary artery pressure remained unchanged
Transcatheter Mitral Valve Repair in Cardiogenic Shock and Mitral Regurgitation	Jung RG, Simard T, Kovach C, et al. JACC Cardiovasc Interv. 2021. [S14]	141 patients: mean age, 68.9 ± 12.1; male, 78 (55.3%); history of HF, 115 (81.6%); NYHA (n = 135): II, 5 (3.7%); III, 32 (23.7%); IV, 98% (72.6%)	Mechanism: functional: 106 (75.2%); degenerative, 33 (23.4%); both, 2 (1.4%)	STS score (n = 117), 16.1 ± 16.6; INTERMACS score (n = 88), 3.1 ± 1.0; SCAI cardiogenic shock class: B, 18 (12.8%); C, 71 (50.4%); D, 42 (29.8%); E, 10 (7.1%); Intubated, 51 (36.2%)	Device success procedural change MR by ≥1 grade and an absolute grade of ≤2+ on FU	In-hospital mortality, 22 (15.6%)	90-day mortality (n = 129), 38 (29.5%); 1-year mortality (n = 129), 55 (42.6%); CHF admission (n = 141), 26 (18.4%)
Transcatheter mitral valve repair in patients with acute myocardial infarction: insights from the European Registry of MitraClip in acute mitral regurgitation following an acute myocardial infarction (EREMMI)	Estevez-Loureiro R, Adamo M, Arzamendi D, et al. EuroIntervention. 2020 [S15]	44 patients: mean age: 70 ± 10.8; male: 63.6%; NYHA: II, 1 (2.3%); II, 6 (13.6%); III, 9 (20.5%); IV, 28% (63.6%); EuroSCORE II, 15.1 (6.2%–23.2%)	Functional (post-acute MI)	MR severity: 3, 10.3%; 4, 89.7%	6 months FU: MR reduction: 0-1: 31%, 2: 41.4%, 3: 17.2%, 4: 10.3%. NYHA improvement: I: 13.8%, II: 62.1%, III: 17.2%, IV: 6.9%	30 days: death: 4 (9.1%); re-admission due to HF, 0; cardiac surgery, 1 (2.3%); major adverse events, 5 (11.4)	6 months: death, 8 (18.2%); re-admission due to HF, 8 (18.2%); cardiac surgery, 3 (6.8%); major adverse events, 16 (36.4%)
Conservative, surgical, and percutaneous treatment for mitral regurgitation shortly after acute myocardial infarction	Haberman D, Estévez-Loureiro R, Benito-Gonzalez T et al. Eur Heart J. 2022 [S16]	99 patients: mean age, 71 ± 10; female, 51 (51%); EuroSCORE II, 10 (7–21); Killip class ≥3, 66 (67%); prior MI, 55 (56%); prior CABG, 28 (27%); MR grade 4+, 80 (81%)	STEMI presentation, 71 (72%); anterior wall involvement, 35 (36%)	PCI, 94 (94%); LVEF, 35% ± 11%; cardiogenic shock: 51 (52%); mechanical ventilation, 39 (39%); vasoactive medication, 39 (39%); IABP, 33 (33%); ECMO, 5 (5%); MCS, 34 (34%); MR grade, 0	MR >2 at discharge: 8 (8%), Major complications: 6 (6%)	Procedure success, 92 (93%); in-hospital mortality, 6 (6%)	Mortality at 3 months, 10 (10%); rehospitalization at 3 months, 13 (13%); 1-year mortality, 16 (17%)

Supplementary references are available at https://journals.viamedica.pl/kardiologia_polska

Abbreviations: see Table 1

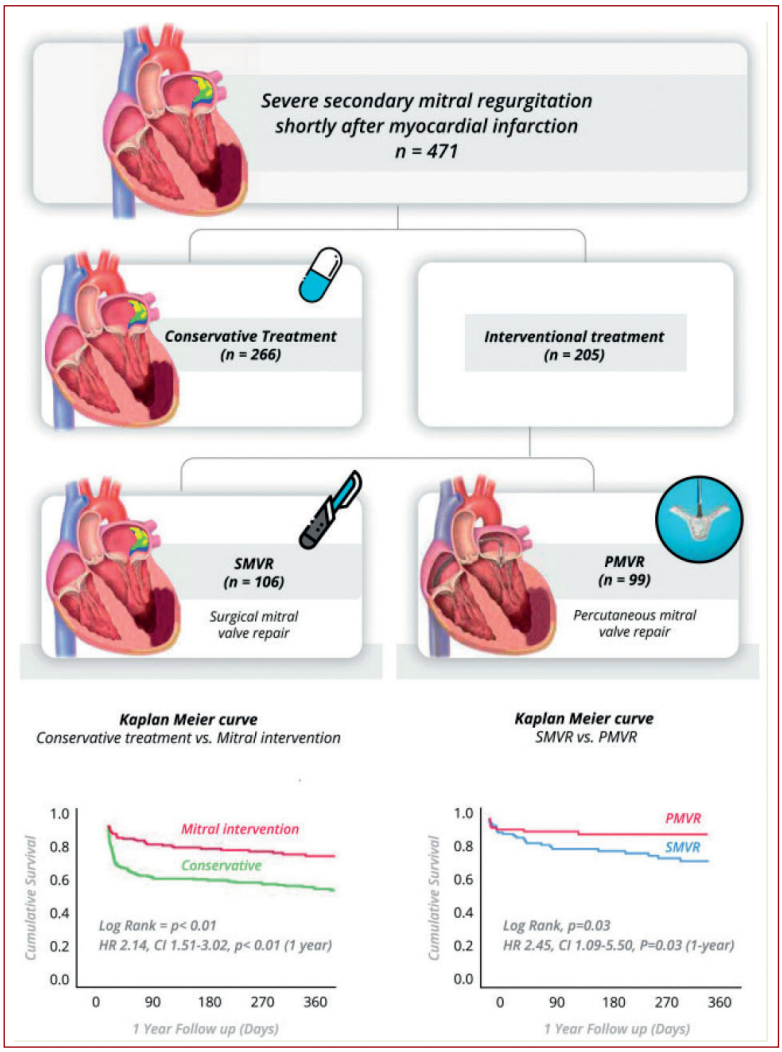

Figure 2. Three treatment options for severe secondary MR following acute MI. Re-published with permission from Haberman D et al. [47]. The registry included 471 patients with significant mitral regurgitation within 90 days after acute myocardial infarction who remained symptomatic on optimal medical therapy. Overall, patients who were treated by mitral intervention, which may serve as an alternative for surgical mitral valve repair/replacement (SMVR) in this high-risk population, had better survival than patients treated conservatively. Among patients treated with mitral intervention, patients treated with percutaneous mitral valve repair (PMVR) had a promising survival rate.

Abbreviation: MI, myocardial infarction; other — see Figure 1

with medical and surgical treatment during heart team discussion and decisions. In fact, the concept of "urgent TEER" may be a game changer, as it allows rapid hemodynamic improvement as a bridge to recovery in selected decompensated high-risk patients. Still, patient selection is crucial for the success of this procedure, and additional studies including randomized trials are required for the establishment of this concept.

FUTURE PERSPECTIVES AND OPEN QUESTIONS

Importantly, the impact of TEER after MI was evaluated in high-risk decompensated patients and therefore cannot be implemented in stable patients. In addition, the preferred timing of the procedure is not defined, and it is unclear which clinical and anatomical criteria should be used to decide if a patient is suitable for TEER. A suggested algorithm is presented in Figure 3. Other important aspects that should be acknowledged are the availability of TEER in the acute setting, experience of the operators, and economic costs. Finally, the use of new devices, like the ECMO and the Impella, should be considered for temporary hemodynamic support in post-MI acute MR patients, to allow a safe TEER procedure.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

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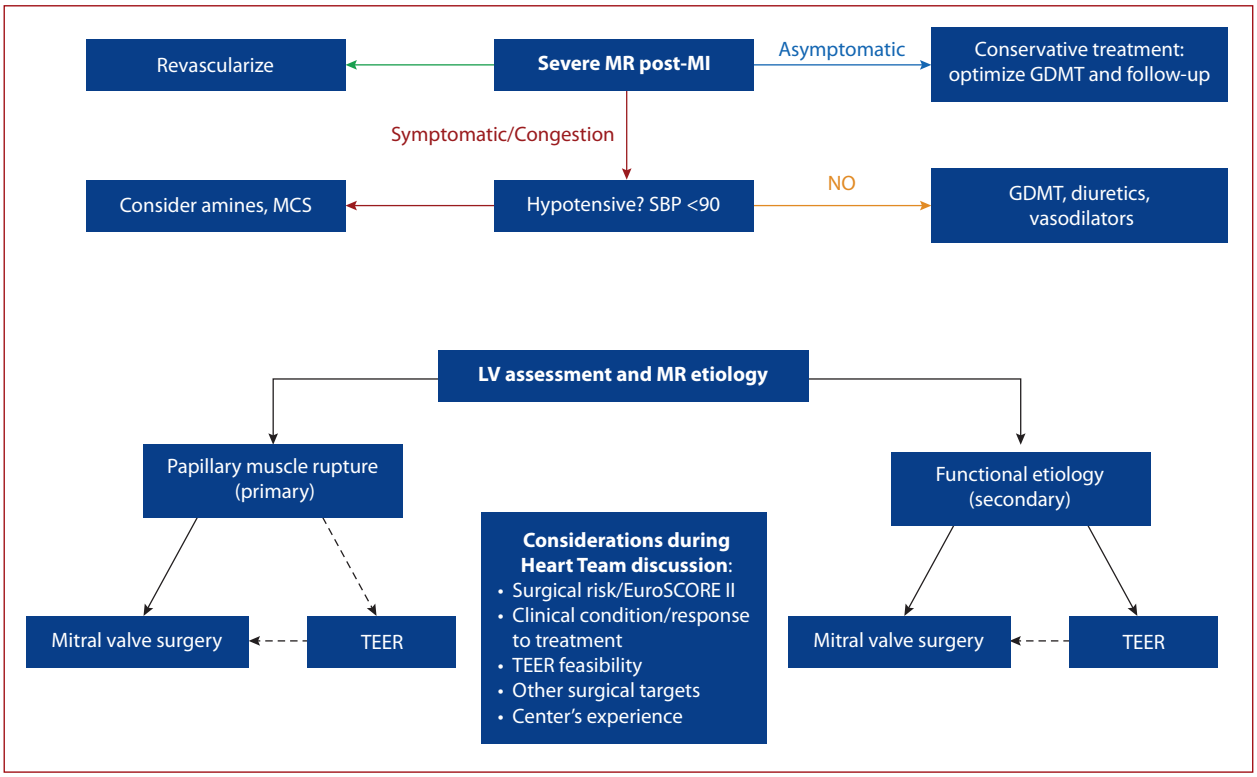

Figure 3. Proposed management algorithm for the treatment of post-MI, severe MR

Abbreviations: GDMT, guideline-directed medical treatment; MCS, mechanical circulatory support; TEER, trans-catheter edge-to-edge repair; other — see Figures 1 and 2

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