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Authors: Dan Haberman, Shani Dahan, Lion Poles, David Marmor, Mony Shuvy

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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 to the Hebrew University of Jerusalem, The Faculty of Medicine, Jerusalem, Israel
[2]Department of cardiology, Assuta Ashdod medical Center, affiliated with Ben Gurion University of the Negev, Ashdod, Israel
[3]Jesselson Integrated Heart Centre, 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 to the Hebrew University of Jerusalem,
The Faculty of Medicine, Jerusalem, Israel
e-mail: haberdan@gmail.com

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-threating 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: myocardial infarction, mitral regurgitation, trans-catheter edge-to-edge repair (TEER), cardiogenic shock, papillary muscle rupture

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 a low systemic blood pressure and obscured heart sounds by respiratory distress, often lead 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 through echocardiographic evaluation, including continuous wave Doppler of mitral inflow and pulsed Doppler of pulmonary veins, are often keys to echocardiographic diagnosis of severe acute MR [4].

Identifying the etiology of acute MR is the cornerstone in the 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 leaflets 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 to treat 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 high surgical risk as 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 IIa indication for selected patients with symptomatic severe secondary MR who fulfil 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 fulfil 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 principle 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 individuals and in 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 the circumflex artery.

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 a hemodynamic support. The use of positive pressure ventilation can have additional benefits due to improved gas exchange and reducing 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 case of mechanical complication 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) is still limited [17].

Surgical treatments remain 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 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 is 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 patients at their 5th to 7th decade of life, presenting 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 institutional review and found 8 patients who underwent emergency TEER, 4 of which were related to post-MI acute MR. 75% had chordal rapture, 75% required MCS and 5 out 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 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 of ischemic MR, where some patients may be asymptomatic, and 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 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 the papillary muscles. The exact mechanism of acute ischemic MR (acute functional MR) is not fully understood, recent data suggest cellular and molecular changes of the leaflets themselves. In acute MR, the mitral apparatus remains dynamic and valvular tenting is modest, but linked to severity [39].

Tethering of leaflets is observed both in infero-posterior 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 from most registries. However, the data for treatment of acute functional MR post-MI is more established than for acute primary MR. The data is 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 twenty 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-days survival of 90% [44]. Additional series was published that included forty-four patients with post-MI acute functional MR [46]. Mean age 70 ± 11 years, with 32 % on MCS. Procedural success was 87% with 91% 30-days survival. The next study from the IREMMI group included ninety-three patients, investigating the role of cardiogenic shock in the same clinical condition. Mean age 70 ± 11 years, 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. 30-days 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 paper from the IREMMI group focused on the effect of TEER on LV function.

105 patients treated with TEER for acute functional MR following MI were divided into two group based on a cut-off of LVEF 35%. Mortality rates comparing the two groups were

comparable up to 1 year (11% of LVEF <35% and 7%) as well as 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 the group compared 3 treatments options: conservative (medical) treatment, surgery and percutaneous (TEER), as shown in Figure 2 [47]. 471 patients with severe functional MR following AMI were included in a retrospective registry from 21 centers, mean age 73 ± 11, 43% females. 266 were managed conservatively and 205 underwent mitral valve intervention based on the physician's clinical decision. Patients in the intervention group were 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 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 different in 1-year mortality was observed. The data detailed above suggests that TEER is feasible and safe in patient 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 these results on longer term outcomes as to-date, 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 as along with patients' characteristics have great implication on clinical presentation and treatment options. TEER is an emerging treatment option in this clinical scenario that should be taken into consideration along 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 PERSPECTIVE 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 in order to decide if the 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, the experience of the operators, and the 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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REFERENCES

1. López-Pérez M, Estévez-Loureiro R, López-Sainz A, et al. Long-term prognostic value of mitral regurgitation in patients with ST-segment elevation myocardial infarction treated by primary percutaneous coronary intervention. Am J Cardiol. 2014; 113(6): 907–912, doi: 10.1016/j.amjcard.2013.11.050, indexed in Pubmed: 24461770.
2. Bursi F, Enriquez-Sarano M, Nkomo VT, et al. Heart failure and death after myocardial infarction in the community: the emerging role of mitral regurgitation. Circulation.

2005; 111(3): 295–301, doi: 10.1161/01.CIR.0000151097.30779.04, indexed in Pubmed: 15655133.

3. Stout KK, Verrier ED. Acute valvular regurgitation. Circulation. 2009; 119(25): 3232–3241, doi: 10.1161/CIRCULATIONAHA.108.782292, indexed in Pubmed: 19564568.
4. Schiller NB, Foster E, Redberg RF. Transesophageal echocardiography in the evaluation of mitral regurgitation. The twenty-four signs of severe mitral regurgitation. Cardiol Clin. 1993; 11(3): 399–408, indexed in Pubmed: 8402769.
5. Nishino S, Watanabe N, Kimura T, et al. The course of ischemic mitral regurgitation in acute myocardial infarction after primary percutaneous coronary intervention: from emergency room to long-term follow-up. Circ Cardiovasc Imaging. 2016; 9(8): e004841, doi: 10.1161/CIRCIMAGING.116.004841, indexed in Pubmed: 27516478.
6. Maisano F, Torracca L, Oppizzi M, et al. The edge-to-edge technique: a simplified method to correct mitral insufficiency1. Eur J Cardiothorac Surg. 1998; 13(3): 240–246, doi: 10.1016/s1010-7940(98)00014-1, indexed in Pubmed: 9628372.
7. Feldman T, Foster E, Glower DD, et al. Percutaneous repair or surgery for mitral regurgitation. N Engl J Med. 2011; 364(15): 1395–1406, doi: 10.1056/NEJMoa1009355, indexed in Pubmed: 21463154.
8. Vahanian A, Beyersdorf F, Praz F, et al. 2021 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J. 2022; 43(7): 561–632, doi: 10.1093/eurheartj/ehab395, indexed in Pubmed: 34453165.
9. Stone GW, Lindenfeld J, Abraham WT, et al. Transcatheter mitral-valve repair in patients with heart failure. N Engl J Med. 2018; 379(24): 2307–2318, doi: 10.1056/NEJMoa1806640, indexed in Pubmed: 30280640.
10. Elbadawi A, Elgendy IY, Mahmoud K, et al. Temporal trends and outcomes of mechanical complications in patients with acute myocardial infarction. JACC Cardiovasc Interv. 2019; 12(18): 1825–1836, doi: 10.1016/j.jcin.2019.04.039, indexed in Pubmed: 31537282.
11. Kishon Y, Oh JK, Schaff HV, et al. Mitral valve operation in postinfarction rupture of a papillary muscle: immediate results and long-term follow-up of 22 patients. Mayo Clin Proc. 1992; 67(11): 1023–1030, doi: 10.1016/s0025-6196(12)61116-1, indexed in Pubmed: 1434862.
12. French JK, Hellkamp AS, Armstrong PW, et al. Mechanical complications after percutaneous coronary intervention in ST-elevation myocardial infarction (from

APEX-AMI). Am J Cardiol. 2010; 105(1): 59–63, doi: 10.1016/j.amjcard.2009.08.653, indexed in Pubmed: 20102891.

13. Bhardwaj B, Sidhu G, Balla S, et al. Outcomes and hospital utilization in patients with papillary muscle rupture associated with acute myocardial infarction. Am J Cardiol. 2020; 125(7): 1020–1025, doi: 10.1016/j.amjcard.2019.12.051, indexed in Pubmed: 31973809.
14. Calvo FE, Figueras J, Cortadellas J, et al. Severe mitral regurgitation complicating acute myocardial infarction. Clinical and angiographic differences between patients with and without papillary muscle rupture. Eur Heart J. 1997; 18(10): 1606–1610, doi: 10.1093/oxfordjournals.eurheartj.a015140, indexed in Pubmed: 9347271.
15. van Diepen S, Katz JN, Albert NM, et al. Contemporary management of cardiogenic shock: a scientific statement from the American Heart Association. Circulation. 2017; 136(16): e232–e268, doi: 10.1161/CIR.0000000000000525, indexed in Pubmed: 28923988.
16. Thiele H, Zeymer U, Neumann FJ, et al. Intraaortic balloon support for myocardial infarction with cardiogenic shock. N Engl J Med. 2012; 367(14): 1287–1296, doi: 10.1056/NEJMoa1208410, indexed in Pubmed: 22920912.
17. Ekanem E, Gattani R, Bakhshi H, et al. Combined venoarterial ECMO and impella-cp circulatory support for cardiogenic shock due to papillary muscle rupture. JACC Case Rep. 2020; 2(14): 2169–2172, doi: 10.1016/j.jaccas.2020.08.007, indexed in Pubmed: 34317131.
18. Thompson CR, Buller CE, Sleeper LA, et al. Cardiogenic shock due to acute severe mitral regurgitation complicating acute myocardial infarction: a report from the SHOCK Trial Registry. SHould we use emergently revascularize Occluded Coronaries in cardiogenic shocK? J Am Coll Cardiol. 2000; 36(3 Suppl A): 1104–1109, doi: 10.1016/s0735-1097(00)00846-9, indexed in Pubmed: 10985712.
19. Bouma W, Wijdh-den Hamer IJ, Koene BM, et al. Predictors of in-hospital mortality after mitral valve surgery for post-myocardial infarction papillary muscle rupture. J Cardiothorac Surg. 2014; 9: 171, doi: 10.1186/s13019-014-0171-z, indexed in Pubmed: 25322911.
20. Wojakowski W, Chmielak Z, Widenka K, et al. Transcatheter mitral valve repair and replacement. Expert consensus statement of the Polish Cardiac Society and the Polish Society of Cardiothoracic Surgeons. Kardiol Pol. 2021; 79(10): 1165–1177, doi: 10.33963/KP.a2021.0116, indexed in Pubmed: 34599502.

21. Otto C, Nishimura R, Bonow R, et al. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: Executive Summary: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021; 143(5), doi: 10.1161/cir.0000000000000932.
22. Damluji AA, van Diepen S, Katz JN, et al. Mechanical Complications of Acute Myocardial Infarction: A Scientific Statement From the American Heart Association. Circulation. 2021; 144(2): e16–e35, doi: 10.1161/CIR.0000000000000985, indexed in Pubmed: 34126755.
23. Schroeter T, Lehmann S, Misfeld M, et al. Clinical outcome after mitral valve surgery due to ischemic papillary muscle rupture. Ann Thorac Surg. 2013; 95(3): 820–824, doi: 10.1016/j.athoracsur.2012.10.050, indexed in Pubmed: 23219255.
24. Bilge M, Alemdar R, Yasar AS. Successful percutaneous mitral valve repair with the MitraClip system of acute mitral regurgitation due to papillary muscle rupture as complication of acute myocardial infarction. Catheter Cardiovasc Interv. 2014; 83(1): E137–E140, doi: 10.1002/ccd.24960, indexed in Pubmed: 23592592.
25. Wolff R, Cohen G, Peterson C, et al. MitraClip for papillary muscle rupture in patient with cardiogenic shock. Can J Cardiol. 2014; 30(11): 1461.e13–1461.e14, doi: 10.1016/j.cjca.2014.07.015, indexed in Pubmed: 25442448.
26. Bahlmann E, Frerker C, Kreidel F, et al. MitraClip implantation after acute ischemic papillary muscle rupture in a patient with prolonged cardiogenic shock. Ann Thorac Surg. 2015; 99(2): e41–e42, doi: 10.1016/j.athoracsur.2014.09.075, indexed in Pubmed: 25639443.
27. Rodríguez-Santamarta M, Estévez-Loureiro R, Gualis J, et al. Percutaneous mitral valve repair with MitraClip system in a patient with acute mitral regurgitation after myocardial infarction. Rev Esp Cardiol (Engl Ed). 2015; 68(3): 259–261, doi: 10.1016/j.rec.2014.10.009, indexed in Pubmed: 25612473.
28. Valle JA, Miyasaka RL, Carroll JD. Acute mitral regurgitation secondary to papillary muscle tear: is transcatheter edge-to-edge mitral valve repair a new paradigm? Circ Cardiovasc Interv. 2017; 10(6), doi: 10.1161/CIRCINTERVENTIONS.117.005050, indexed in Pubmed: 28607001.
29. Yasin M, Nanjundappa A, Annie FH, et al. Use of MitraClip for postmyocardial infarction mitral regurgitation secondary to papillary muscle dysfunction. Cureus. 2018; 10(7): e3065, doi: 10.7759/cureus.3065, indexed in Pubmed: 30280061.

30. Papadopoulos K, Chrissoheris M, Nikolaou I, et al. Edge-to-edge mitral valve repair for acute mitral valve regurgitation due to papillary muscle rupture: a case report. Eur Heart J Case Rep. 2019; 3(1): ytz001, doi: 10.1093/ehjcr/ytz001, indexed in Pubmed: 31020246.
31. Villablanca P, Wang D, Lynch D, et al. Successful MitraClip XTR for Torrential Mitral Regurgitation Secondary to Papillary Muscle Rupture as a Complication of Acute Myocardial Infarction. Structural Heart. 2019; 3(4): 352–355, doi: 10.1080/24748706.2019.1618513.
32. Komatsu I, Cohen EA, Cohen GN, et al. Transcatheter mitral valve edge-to-edge repair with the new MitraClip XTR system for acute mitral regurgitation caused by papillary muscle rupture. Can J Cardiol. 2019; 35(11): 1604.e5–1604.e7, doi: 10.1016/j.cjca.2019.06.024, indexed in Pubmed: 31604669.
33. So CY, Kang G, Lee JC, et al. Transcatheter edge-to-edge repair for acute mitral regurgitation with cardiogenic shock secondary to mechanical complication. Cardiovasc Revasc Med. 2022 [Epub ahead of print], doi: 10.1016/j.carrev.2022.07.003, indexed in Pubmed: 35882600.
34. Chang CW, Romero S, Price M. Transcatheter edge-to-edge repair for acute mitral regurgitation due to postinfarction papillary muscle rupture. JSCAI. 2022; 1(5): 100431, doi: 10.1016/j.jscai.2022.100431.
35. Naser N, Dzubur A, Kusljugic Z, et al. Echocardiographic assessment of ischaemic mitral regurgitation, mechanism, severity, impact on treatment strategy and long term outcome. Acta Inform Med. 2016; 24(3): 172–177, doi: 10.5455/aim.2016.24.172-177, indexed in Pubmed: 27482130.
36. Sannino A, Grayburn PA. Ischemic mitral regurgitation after acute myocardial infarction in the percutaneous coronary intervention era. Circ Cardiovasc Imaging. 2016; 9(8): e005323, doi: 10.1161/CIRCIMAGING.116.005323, indexed in Pubmed: 27516481.
37. Hung JW. Ischemic (functional) mitral regurgitation. Cardiol Clin. 2013; 31(2): 231–236, doi: 10.1016/j.ccl.2013.04.003, indexed in Pubmed: 23743075.
38. Dal-Bianco JP, Aikawa E, Bischoff J, et al. Active adaptation of the tethered mitral valve: insights into a compensatory mechanism for functional mitral regurgitation. Circulation. 2009; 120(4): 334–342, doi: 10.1161/CIRCULATIONAHA.108.846782, indexed in Pubmed: 19597052.

39. Kimura T, Roger VL, Watanabe N, et al. The unique mechanism of functional mitral regurgitation in acute myocardial infarction: a prospective dynamic 4D quantitative echocardiographic study. Eur Heart J Cardiovasc Imaging. 2019; 20(4): 396–406, doi: 10.1093/ehjci/jey177, indexed in Pubmed: 30517693.
40. Yosefy C, Beeri R, Guerrero JL, et al. Mitral regurgitation after anteroapical myocardial infarction: new mechanistic insights. Circulation. 2011; 123(14): 1529–1536, doi: 10.1161/CIRCULATIONAHA.110.977843, indexed in Pubmed: 21444880.
41. Beaudoin J, Levine RA, Guerrero JL, et al. Late repair of ischemic mitral regurgitation does not prevent left ventricular remodeling: importance of timing for beneficial repair. Circulation. 2013; 128(11 Suppl 1): S248–S252, doi: 10.1161/CIRCULATIONAHA.112.000124, indexed in Pubmed: 24030415.
42. Mentias A, Raza MQ, Barakat AF, et al. Outcomes of ischaemic mitral regurgitation in anterior versus inferior ST elevation myocardial infarction. Open Heart. 2016; 3(2): e000493, doi: 10.1136/openhrt-2016-000493, indexed in Pubmed: 27933193.
43. Estévez-Loureiro R, Arzamendi D, Freixa X, et al. Percutaneous Mitral Valve Repair for Acute Mitral Regurgitation After an Acute Myocardial Infarction. J Am Coll Cardiol. 2015; 66(1): 91–92, doi: 10.1016/j.jacc.2015.03.597, indexed in Pubmed: 26139064.
44. Haberman D, Taramasso M, Czarnecki A, et al. Salvage MitraClip in severe secondary mitral regurgitation complicating acute myocardial infarction: data from a multicentre international study. Eur J Heart Fail. 2019; 21(9): 1161–1164, doi: 10.1002/ejhf.1565, indexed in Pubmed: 31380580.
45. Jung RG, Simard T, Kovach C, et al. Transcatheter mitral valve repair in cardiogenic shock and mitral regurgitation: a patient-level, multicenter analysis. JACC Cardiovasc Interv. 2021; 14(1): 1–11, doi: 10.1016/j.jcin.2020.08.037, indexed in Pubmed: 33069653.
46. Estevez-Loureiro R, Adamo M, Arzamendi D, et al. 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). EuroIntervention. 2020; 15(14): 1248–1250, doi: 10.4244/EIJ-D-19-00653, indexed in Pubmed: 31738168.
47. Haberman D, Estévez-Loureiro R, Benito-Gonzalez T, et al. Conservative, surgical, and percutaneous treatment for mitral regurgitation shortly after acute myocardial

infarction. Eur Heart J. 2022; 43(7): 641–650, doi: 10.1093/eurheartj/ehab496, indexed in Pubmed: 34463727.

48. Estévez-Loureiro R, Shuvy M, Taramasso M, et al. Use of MitraClip for mitral valve repair in patients with acute mitral regurgitation following acute myocardial infarction: Effect of cardiogenic shock on outcomes (IREMMI Registry). Catheter Cardiovasc Interv. 2021; 97(6): 1259–1267, doi: 10.1002/ccd.29552, indexed in Pubmed: 33600072.

49. Haberman D, Estévez-Loureiro R, Benito-Gonzalez T, et al. Safety and feasibility of MitraClip implantation in patients with acute mitral regurgitation after recent myocardial infarction and severe left ventricle dysfunction. J Clin Med. 2021; 10(9), doi: 10.3390/jcm10091819, indexed in Pubmed: 33921996.

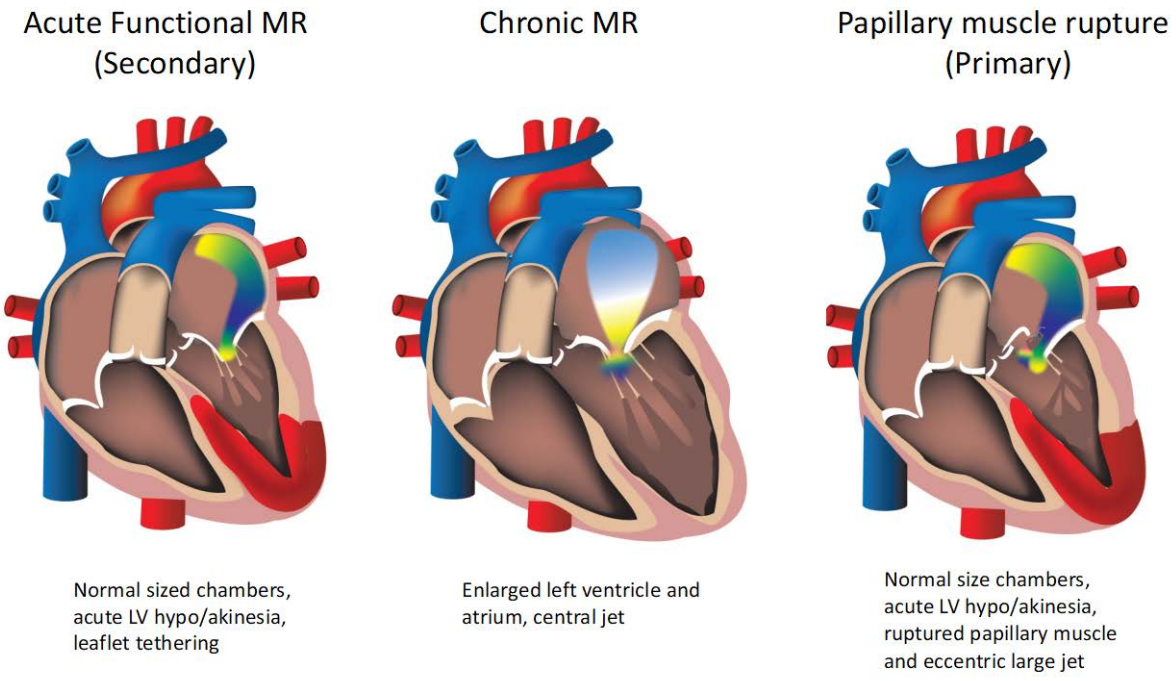

Figure 1. Mitral regurgitation mechanism — chronic, acute functional MR and papillary muscle rupture.

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 in 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 central jet is seen. In papillary muscle rupture, LV infarction is seen, ruptured muscle with chord attached to free leaflet causing a wide eccentric jet

Abbreviations: MR, mitral regurgitation; LV, left ventricle

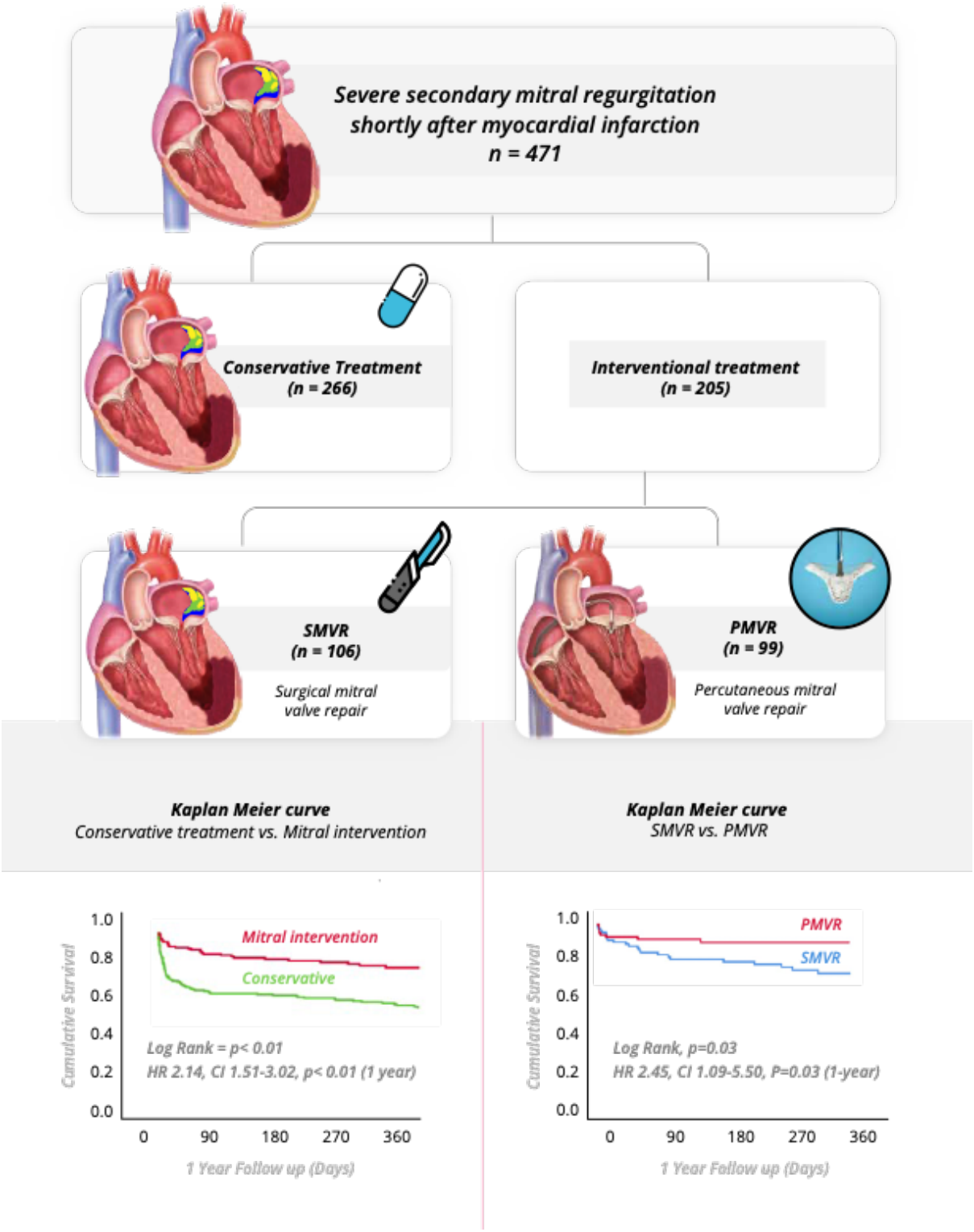

Figure 2. Three Treatment Options for Severe Secondary MR following Acute MI With permission from Haberman D et al. [47].

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 treated by mitral intervention had better survival over patients treated conservatively. Among patients treated with mitral intervention, patient treated with percutaneous mitral valve repair (PMVR) had a promising survival which may serve as alternative for surgical mitral valve repair/replacement (SMVR) in this high-risk population

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

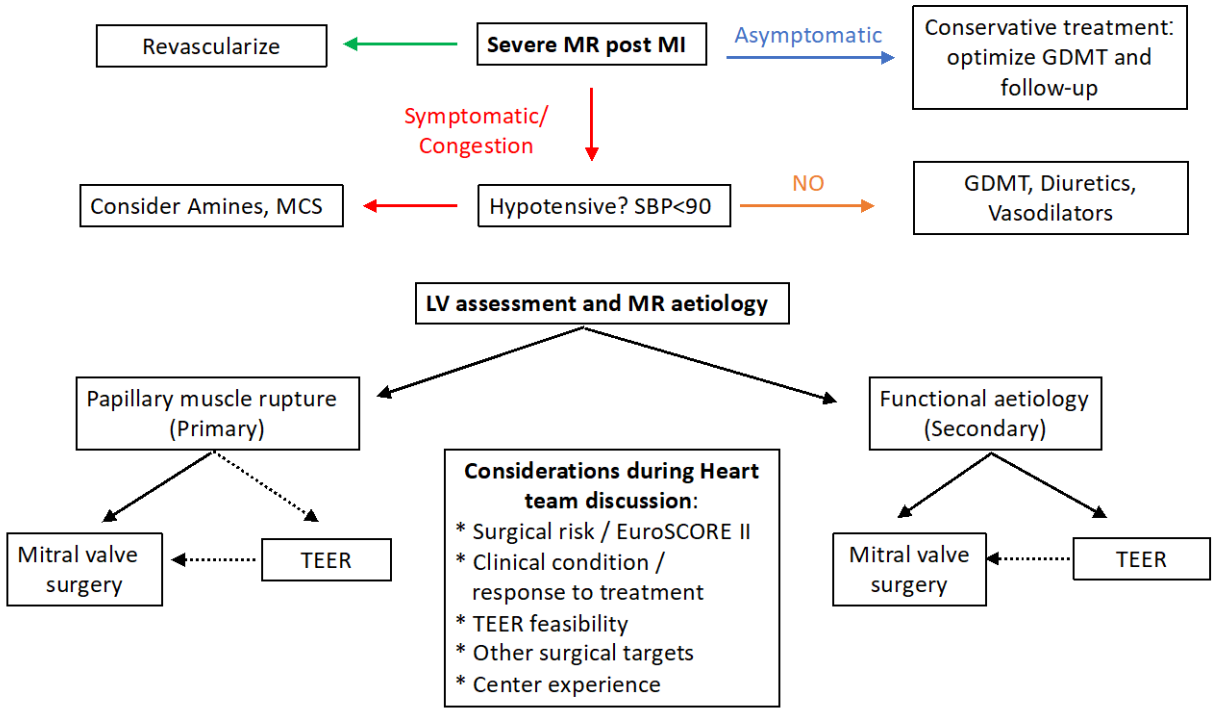

Figure 3. Proposed management algorithm for the treatment of post MI, severe MR

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

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]	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.	68-year-old male, late arrival STEMI with Q waves in	Latecomer lateral STEMI, with occlusion of large first obtuse marginal artery.	Cardiogenic shock and ventricular arrhythmias.	Device success 2 clips (A2–P2); MR grade 2+;	MR grade 1–2+, LVEF 30%, LVEDD 51 mm, LVESD	3-month: NYHA II, MR grade 1–2+ LVEF 38%,

	CJC 2014 [S2]	lateral and posterior leads STS score, 64%; EuroSCORE II, 75%	Complete rupture of anterolateral papillary muscle with flail of A2. LVEF, 25%	IABP and inotropes. mean LAP, 37 mm Hg; v-wave, 55 mm Hg	v-wave, 30 mm Hg	46 mm, normal RV function	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]	77-year-old male, s/p CABG presented with dyspnea and 3 days back pain. Lateral NSTEMI. Log EuroSCORE, 78%	Lateral NSTEMI. Subtotal SVG to 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	Rodríguez-Santamarta	76-year-old male; STS	Inferolateral STEMI, successful	Pulmonary edema	MR grade (4th day), 1+; 2	Alive, NYHA I	NYHA I

Mitraclip System in a Patient With Acute Mitral Regurgitation After Myocardial Infarction	M, Estévez-Loureiro R, Gualis J, et al. Rev Esp Cardio 2015 [S4]	score, 6.7%; Log EuroSCORE, 29.1%	primary PCI of proximal circumflex artery, Ischemic asymmetric posterior leaflet tethering, A2–P2 and A3–P3		clips (A2–P2; lateral to the first one); MR grade, 1+; MV MG < 5 mm Hg		
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]	4-year-old male, LVEF, mildly reduced	Inferior STEMI, successful primary PCI of saphenous vein graft to right coronary artery. Partial tear of 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	Alive	6-week: NYHA, II; MR grade, 1–2+

					mm Hg; v-wave, 20 mm Hg		
Use of MitraClip for Postmyocardial Infarction Mitral Regurgitation Secondary to Papillary Muscle Dysfunction	Yasin M, Nanjundappa A, Annie FH, et al. Cureus 2018 [S6]	68-year-old male	Inferior NSTEMI, rupture of posteromedial papillary muscle with flail of 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]	-year-old female, og EuroSCORE, 43%; STS score, 13%; LVEF, 40%	Anterior STEMI, successful primary PCI of intermediate artery, Partial rupture of the anterolateral papillary muscle with flail of A1	Cardiogenic shock, pulmonary edema, IABP and inotropes	Device success, 2 clips (A2–P2, A1-P1) with a “zipping” of the lateral commissure,	Alive (postop day 7)	20-month: NYHA II; MR grade, 2+; MV MG, 6 mm Hg

			A2 and P1		MR grade, 1–2+; MV area, 2.1 cm^2; MV MG, 6 mm Hg		
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]	0-year-old male, LVEF, 60%; TS score, 14.3%	Lateral NSTEMI, successful primary PCI of proximal and mid-circumflex artery. Complete rupture of 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]	5-year-old male, LVEF, 55%	Inferior STEMI, successful primary PCI of culprit single-vessel disease, 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 mmHg	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	So CY, Kang G, Lee JC, et al. Cardiovasc	24 patients: mean age: 73 ± 12.9; M:F, 17:7; mean LVEF, 50 ± 11.2%	Chordal rupture, 50%; papillary muscle rupture, 50%; anterolateral, 41.7%;	MCS usage: 75%, IABP: 44.4%, Impella: 27.8% ECMO: 16.7%, ECPELLA:	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	Ability to wean off MCS: 94.4%; device success, 68.8%; 30-day CV mortality,

mechanical complication	Revasc Med. 2022. [S10]		posteromedial, 25%; STEMI, 58.3%	11.1%, MV gradient, pre-MC: 3+/-1.2. symptoms onset to MC, days: 11+/-8.2		81.3%; >5 mmHg 18.7%	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-elevation myocardial infarction; TEER, trans-catheter edge-to-edge repair.

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, euroSCORE Killip class	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	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;	All patients had moderate to severe MR (3–4+);	MR reduction to 1+ was achieved in 12 patients	1 patient died after a leaflet tear and urgent MVR	Median FU, 15 months; functional capacity improvement

acute myocardial infarction: data from a multicentre international study			Multi-vessel; CAD, 17 (85%)	mean EF, 35.9 ± 12.5%; severe LV dysfunction, 7 (35%); cardiogenic shock and IABP/vasopressors, 8 (40%); mechanical ventilation, 6 (30%)	and to 2+ in 7 pts;pulmonary artery pressure reduction; left atrial v-wave reduction	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	in 17 pts (85%) – NYHA I/II, EF and pulmonary artery pressure remained unchanged
Transcatheter Mitral Valve Repair in	Jung RG, Simard T, Kovach C, et al. JACC Cardiovasc	141 patients: mean age, 68.9± 12.1;	Mechanism: functional: 106 (75.2%);	STS score (n = 117), 16.1 ± 16.6;	Device success (procedural	In-hospital mortality, 22 (15.6%)	90-day mortality (n = 129), 38

Cardiogenic Shock and Mitral Regurgitation	Interv. 2021. [S14]	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%)	degenerative, 33 (23.4%); both, 2 (1.4%)	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%)	change MR by ≥1 grade and an absolute grade of ≤2+ on FU		(29.5%); 1-yr 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	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	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%.	30 days: death: 4 (9.1%); re-admission due to HF, 0; cardiac surgery, 1 (2.3%);	6 months: death, 8 (18.2%); re-admission due to HF, 8 (18.2%); cardiac surgery, 3

following an acute myocardial infarction (EREMMI)		(20.5%); IV, 28% (63.6%); EuroSCORE II, 15.1 (6.2%–23.2%)			NYHA improvement:I: 13.8%, II: 62.1%, III: 17.2%, IV: 6.9%	major adverse events, 5 (11.4)	(6.8%); major adverse events, 16 (36.4)
Conservative, surgical, and percutaneous treatment for mitral regurgitation shortly after acutemyocardial 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	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%)

				(5%); MCS, 34 (34%); MR grade, 0			

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

Abbreviations: see Table 1