GAWIN, Konrad, ZAWIŚLAK, Wiktoria, IGNASIAK, Anita, CISOWSKI, Michał, DĄBROWSKA, Maria, RYCHLICA, Kacper, CHOLEWIŃSKA-RYCHLICA, Jolanta, MADURA, Paulina and MROZIK-GAŁECKA, Daria. Mechanical complications of acute myocardial infarction - a review of current literature. Quality in Sport. 2026;49:67968. eISSN 2450-3118.
https://doi.org/10.12775/QS.2026.49.67968
https://apcz.umk.pl/QS/article/view/67968

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 l.p. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 04.01.2026. Revised: 15.01.2026. Accepted: 15.01.2026. Published: 18.01.2026.

Mechanical complications of acute myocardial infarction - a review of current literature

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Abstract

Mechanical complications of acute myocardial infarction (AMI) include papillary muscle rupture and acute mitral regurgitation, ventricular septal rupture, free wall rupture and pseudoaneurysm. The incidence of mechanical complications of AMI has decreased significantly in the era of widespread early reperfusion and primary percutaneous intervention, but they are still associated with high mortality. Diagnosis and prompt implementation of interventions (hemodynamic stabilization, surgical, transcatheter) remain crucial for survival. However, due to the small number of high-quality clinical trials, the quality of care often depends on local procedures and resources. This review article summarizes the available data on the main mechanical complications after AMI, based on the latest guidelines and current review articles and studies from 1991 to 2022.

Purpose of research

The purpose of this review is to discuss mechanical complications of AMI.. Due to their potentially asymptomatic course, it is important to emphasize accurate diagnosis and implementation of rapid treatment if they are suspected to occur during or after AMI.

Materials and methods

The study was conducted through a search across PubMed and Google Scholar databases, using keywords such as "mechanical complications," "myocardial infarction," "left ventricle rupture," "free wall rupture," "papillary muscle rupture," and "pseudoaneurysm." The analysis focused on scientific literature with validated insights into the pathophysiology, clinical presentation, diagnostic methods, and treatment strategies of AMI mechanical complications.

Results and conclusions

The potentially fatal consequences of AMI mechanical complications dictate a proactive approach in the treatment of patients with a history of AMI. Routine follow-up examinations, comprehensive cardiac imaging, and high clinical alertness are essential for early diagnosis. By providing meticulous post-MI care, healthcare professionals can improve patient outcomes and reduce mortality.

Keywords: acute myocardial infarction, mechanical complications, free wall rupture, ventricular septal rupture, acute mitral regurgitation, pseudoaneurysm, acute heart failure, cardiogenic shock

Introduction

The most common mechanical complications of AMI are papillary muscle rupture and secondary acute mitral regurgitation, ventricular septal rupture, pseudoaneurysm, and free wall rupture. They are characterized by high mortality, reaching up to 73%. (1) They usually occur in the first week after the infarction, more often in acute coronary syndromes with ST segment elevation (STEMI) than in acute coronary syndromes without ST segment elevation (NSTEMI). Despite a decrease in frequency due to the widespread use of early reperfusion, their occurrence is associated with high mortality; therefore, they require rapid diagnosis, hemodynamic stabilization, and often immediate surgical or catheter-based intervention.

1. Papillary muscle rupture and acute mitral regurgitation

Epidemiology

Papillary muscle rupture (PMR) is a rare complication of AMI, with a higher incidence in the pre-reperfusion era, currently complicating approximately 0.05%–0.26% of AMI cases. Despite its rarity, PMR accounts for up to 5% of deaths in the post-infarction period (2, 3) . Hospital mortality remains high, ranging from 10% to 50% in cases of complete PMR (3).

Pathogenesis

The mitral valve is supported by two papillary muscles connected by chordae tendineae: the anterior-lateral and posterior-medial muscles. A PMR can be partial or complete. A partial rupture of only a fragment of the muscle results in less valve regurgitation. Partial ruptures are better tolerated hemodynamically compared to complete ruptures involving the entire muscle, which cause rapid deterioration of the clinical condition. The rupture most often affects the posterior-medial papillary muscle (usually supplied exclusively by the right coronary artery) in inferior wall infarction. The anterior-lateral papillary muscle, supplied by multiple branches of the left coronary artery, rarely undergoes critical ischemia and rupture. PMR leads to mitral valve regurgitation and retrograde blood flow, resulting in heart failure.

Diagnosis

Risk factors for PMR include: STEMI, female gender, older age, chronic heart failure, chronic renal failure, and delayed diagnosis of myocardial infarction.

PMR typically occurs 2-7 days after an AMI and classically manifests as acute heart failure during this period in patients after an inferior wall infarction. PMR is characterized by a varied clinical picture: from asymptomatic to life-threatening cardiogenic shock and pulmonary edema. Acute mitral regurgitation can result in left- or right-ventricular heart failure. (4) Acute mitral regurgitation is not always accompanied by an audible murmur over the heart due to the decreasing pressure gradient between the left ventricle and the left atrium.

Transthoracic or transesophageal echocardiography is the test of choice. It allows visualization of papillary muscle dysfunction, increased reverse flow velocity across the mitral valve, abnormal valve leaflet motion, or a mobile mass attached to the chordae tendineae. The sensitivity of transthoracic echocardiography in detecting structural abnormalities ranges from 65% to 85%, while the sensitivity of transesophageal echocardiography ranges from 92% to 100%. (5) Rapid equalization of pressures between the left ventricle and left atrium often reduces the velocity of regurgitant flow through the mitral valve and makes Doppler assessment harder. Echocardiography is also crucial for differentiating between other mechanical complications of AMI.

Management

Treatment involves the use of nitroglycerin or sodium nitroprusside infusion to reduce afterload in hemodynamically stable patients. In hemodynamically unstable patients with cardiogenic shock, pressor amines such as norepinephrine or dopamine may be used, but there are no clinical studies confirming the effectiveness of such intervention. The use of intra-aortic balloon pump may contribute to improving cardiac output, increasing coronary flow, and reducing afterload. (6)

Definitive treatment involves urgent repair or replacement of the mitral valve — cardiac surgery is usually necessary; in selected patients, catheter-based therapy is considered (rarely and experimentally). (7) Mortality in PMR is significantly lower in patients who undergo surgery compared to those who receive conservative treatment. (8) In patients undergoing surgical mitral valve replacement, simultaneous aortic-coronary bypass (CABG) should be considered. Depending on the patient's clinical condition, surgery may be postponed for 6-8 weeks to allow the necrotic area to organize and improve surgical conditions.

Prognosis

The long-term clinical prognosis in operated patients is relatively good. Without surgical intervention, the prognosis is poor, with high early mortality due to the risk of cardiogenic shock. Even after surgery, mortality remains significant (in different publications, mortality can range from several dozen to >30–50% depending on the series and severity of shock), especially in elderly patients with multiple comorbidities. (3, 9) The prognosis after surgical intervention is better in the case of simultaneous CABG (10). The outcome depends on rapid diagnosis, preoperative hemodynamic status, the patient's general health, and the availability of mechanical support.

2. Ventricular septal rupture

Epidemiology

Currently, the incidence of ventricular septal rupture (VSR) has decreased significantly: from approximately 1%–3% before widespread early reperfusion to 0.17% - 0.44% of all infarcts according to current estimates, although the values vary depending on the population and time of the study. (11) Nevertheless, VSR remains a complication with a high mortality rate, reaching 100% in cases of conservative treatment alone, due to progressive heart failure and hemodynamic disturbances (12). VSR is more common in STEMI than in NSTEMI. (13)

Pathogenesis

VSR usually occurs within 48 hours after the infarction, but may occur up to 2 weeks later. Necrosis and disintegration of the septal muscle fibers lead to the formation of an acute post-infarction defect in the septum. (14) A defect appearing several weeks after the infarction is a chronic rupture. Ventricular septal rupture occurs most often in cases of full-thickness infarction. VSR can be located: anterior, if there is an infarction in the area of the anterior descending artery, or posterior, in the case of a lower wall infarction. Defects in the anterior location are usually simpler, while those in the posterior location are more complex. The defect may be single or occur in the form of numerous smaller defects. (15)

Risk factors for VSD include: STEMI, older age, female gender, chronic heart failure, LAD occlusion, and delayed reperfusion.

Diagnosis

The typical clinical presentation of VSD is sudden heart failure or cardiogenic shock with symptoms of hypoperfusion. A characteristic symptom of VSR is a loud holosystolic murmur best heard at the left sternal border, which, however, may be not audible in cases of large leakage or severe cardiogenic shock. A heart murmur may occur as an isolated symptom of VSD. There may also be worsening symptoms of congestive heart failure, pulmonary edema, cardiogenic shock, and signs of multi-organ failure, such as increased liver enzymes, elevated creatinine, and lactate levels (16).

As with other mechanical complications, the basis for diagnosis is transthoracic echocardiography with color Doppler imaging to visualize left-to-right flow. If the condition of a patient with AMI suddenly worsens, transthoracic echocardiography is indicated, with particular attention to imaging the interventricular septum in various projections. Transesophageal echocardiography may be used in cases of poor transthoracic imaging conditions. Other diagnostic methods that allow for more accurate imaging of the size and location of the defect include ECG-gated computed tomography, cardiac magnetic resonance imaging, and ventriculography.

Management

Conservative management in the preoperative period includes the administration of positive inotropic drugs such as dobutamine or milrinone to increase cardiac output. In patients with hypotension, noradrenaline infusion can be used, but this may increase left-to-right shunting. In cases of high blood pressure and increased peripheral resistance, vasodilators such as nitroglycerin or sodium nitroprusside and/ or intra-aortic balloon pump can be used to reduce afterload. Supplemental oxygen therapy or mechanical ventilation may be necessary.

The treatment of choice is surgical closure of the defect, which remains the only effective intervention, but is associated with high postoperative mortality. (16) The duration and technique of the operation depend on the size and location of the defect and the condition of the tissues. The operation involves excision of the necrotic tissue and closure of the resulting defect with sutures in the case of smaller defects, or with a Teflon patch in the case of larger defects. If the infarct area involves the apex, amputation of the apex may be performed. In posterior defects the conditions for surgery are much more unfavorable due to more difficult surgical access and greater tissue sensitivity. If the patient is stable, it may be beneficial to postpone the surgery,

allowing connective tissue to form at the site of the defect, which provides better conditions for surgery.

An alternative procedure is transcatheter closure of the defect (device closure) in selected patients, especially when there is a high surgical risk or as a bridge therapy; the data on the results are varied. (16).

Prognosis

VSR is associated with very high mortality without treatment (many patients die within days). Even with surgical treatment, 30-day mortality remains high (20–60% or more in various series); outcomes are better with planned repair in stable patients than with urgent surgery during severe shock. Percutaneous closure of the defect may reduce short-term mortality in selected patients. (16)

3. Free wall rupture

Epidemiology

Free wall rupture is a very serious, albeit rare, complication of AMI. It occurs in less than 1% of cases, but is associated with high mortality. The widespread use of interventional techniques for treating AMI, as well as early fibrinolysis, has reduced the incidence of acute free wall rupture to approximately 0.01%–0.02% in cases of invasive treatment. (17) In approximately 50% of cases, free wall rupture manifests as sudden cardiac death before admission to hospital, which is why mortality data are incomplete. (17) In 40% of cases, rupture occurs within the first 24 hours after AMI, and in 85% of cases within the first week. After 10 days, i.e., after the healing process has begun, ruptures occur very rarely. Risk factors for free wall rupture include: first ischemic episode, hypertension, age >70 years, anterior location of the infarction, female gender, and delayed reperfusion. Free wall rupture is the third most common cause of death in the period after hospitalization for STEMI, after cardiogenic shock and acute heart failure.

Pathogenesis

There are three types of free wall rupture according to the Becker and van Mantgem classification. Type I is an acute, large rupture usually occurring within 24 hours after the infarction, with massive bleeding into the pericardial sac and tamponade, presenting as cardiogenic shock or sudden cardiac death. Type II is subacute myocardial damage, often incomplete, with slow bleeding into the pericardial sac and gradually increasing tamponade, which may initially be

asymptomatic. Type III is a chronic rupture limited by a clot with the formation of a cardiac pseudoaneurysm, often asymptomatic, usually occurring more than 5 days after the infarction. The location of the free wall rupture depends on the coronary artery supplying the ischemic area. Rupture most often occurs in the left ventricle rather than the right ventricle, in areas of severe ischemia. Early ruptures may also be associated with the use of fibrinolytic therapy. In the pre-reperfusion era, acute blockage of coronary blood flow typically led to full-thickness myocardial necrosis, a histopathological process that usually took 3 to 5 days. Although acute reperfusion strategies effectively prevent extensive transmural necrosis, reperfusion-related injury after AMI can lead to ruptures occurring within the first 24 to 48 hours after symptom onset.

Diagnosis

If symptoms of hemodynamic failure occur during or after the diagnosis of AMI, the possibility of mechanical complications should always be considered. If acute rupture occurs within 24 hours of the infarction, with massive bleeding into the pericardial sac, death from massive tamponade can occur within minutes. In the case of subacute rupture, 1-3 days after the infarction, with less damage to the heart muscle, symptoms include chest pain resistant to pharmacological treatment, hypotension, and developing cardiogenic shock. Pericardial tamponade manifests itself as Beck's triad: hypotension, increased jugular venous filling, and muffled heart sounds. Chronic rupture is observed late and is often asymptomatic.

The basis for diagnosis is echocardiography. The diagnosis is confirmed by the presence of a defect in the free wall of the heart and a direct connection between the ventricle and the pericardial sac. Less certain evidence of free wall rupture is the presence of fluid or clots in the pericardial sac or thinning of the heart wall. In case of diagnostic uncertainty, ventriculography may be the decisive test, but it is an invasive procedure associated with complications and delays cardiac surgery. Transesophageal echocardiography, computed tomography, or cardiac magnetic resonance imaging can provide accurate data on the location and extent of the defect. It is important to differentiate free wall rupture from other mechanical complications after AMI: ventricular septal rupture, PMR with acute mitral regurgitation, and pseudoaneurysm.

Management

The treatment of choice is cardiac surgery with resection of the necrotic tissue and reconstruction of the heart muscle; emergency surgery is crucial, especially in the case of acute rupture. If free wall rupture is diagnosed before coronary angiography, it should be considered whether it would

be beneficial to perform it before surgery. Invasive coronary artery diagnosis in hemodynamically stable patients with free wall rupture can provide valuable data on the condition of the coronary arteries in the context of CABG performed simultaneously with free wall rupture repair. In the case of tamponade diagnosis in the course of free wall rupture, pericardiocentesis may lead to rapid control of cardiogenic shock symptoms, but may be ineffective in the presence of numerous clots in the pericardial sac (18). Intra-aortic balloon pump or ECMO may be used for hemodynamic stabilization. In stable patients, monitoring of vital signs is crucial.

The goal of surgical treatment is to permanently close the defect in the free wall of the heart and prevent the formation of a pseudoaneurysm. There are two main techniques for repairing the defect: suture and sutureless, which are comparable in effectiveness. (19) Suture techniques may include: linear closure, patch closure, excision of necrotic tissue, triple patch closure. Sutureless techniques are based on the use of surgical glue or collagen sponge, but cannot be used in the presence of active bleeding.

An alternative to surgery in patients with poor prognosis may be the use of catheter-based techniques. Percutaneous closure of free wall rupture using the Ampflitzer system is also possible in patients at high surgical risk due to severe left ventricular dysfunction, hemodynamic instability, advanced age, and severe chronic diseases.

Prognosis

In approximately 15-30% of patients, free wall rupture leads to death. (20) In the case of conservative treatment, mortality is very high, reaching 90%. (21)

4. Pseudoaneurysm

Epidemiology

Pseudaneurysm is a rare but fatal complication of myocardial infarction. Pseudaneurysms tend to grow and rupture, but may remain undiagnosed for several months or years after the infarction and be detected incidentally in imaging studies in stable patients. (22)

Pathogenesis

Unlike true aneurysms, which are the result of thinning, dyskinesia or akinesia and bulging of full thickness of the heart wall, pseudoaneurysms form when a rupture in the free wall of the left ventricle is confined by pleural adhesions. The wall of a pseudoaneurysm does not contain the

full muscular layer of the ventricle (unlike a true aneurysm), which carries a high risk of late rupture. (23) Pseudoaneurysms are most commonly located on the posterior and lateral walls of the left ventricle. (24)

Diagnosis

The clinical picture is non-specific and may include chest pain, shortness of breath, symptoms of heart failure, arrhythmias, or a sudden episode of pain/hemodynamic disturbances when the pseudoaneurysm ruptures. (23) Pseudoaneurysms may also be asymptomatic for many months or years. (22)

Transesophageal and transthoracic echocardiography, cardiac computed tomography (CTA), and cardiac magnetic resonance imaging (MRI) help in the diagnosis and differentiation of pseudoaneurysm from true aneurysm. A pseudoaneurysm is characterized by a narrow neck and does not involve the entire thickness of the heart wall (endocardium, myocardium, and pericardium). (23)

Management

Due to the high risk of rupture, urgent or elective cardiac surgery is usually recommended in most patients — closure of the defect and correction of the wall. In high-risk patients or those with small, stable pseudoaneurysms, a wait-and-see approach may be considered, but the risk of late rupture and death remains significant. (23) Percutaneous closure of a pseudoaneurysm is also possible, but data on this treatment method are scarce and incomplete (25).

Prognosis

Due to its rarity, data on the prognosis of pseudoaneurysms are incomplete. Based on available studies, postoperative mortality appears to be relatively low. (26)

Table 1. Summary of Mechanical Complications of Acute Myocardial Infarction.

Complication	**Incidence**	**Typical clinical picture**	**Echocardiography**	**Management**	**Postoperative mortality**
Papillary muscle rupture and acute mitral regurgitation	0.05%–0.26% AMI	Acute heart failure 2–7 days after inferior wall infarction	Reverse flow through the mitral valve and a displaced mass connected to the chordae tendineae.	Hemodynamic stabilization, surgical valve repair.	10-50%
Ventricular septal	0,17%- 0,44% AMI	Acute heart failure or	Left-to-right shunt.	Hemodynamic	20-60%

rupture		cardiogenic shock within 48 hours after AMI, holosystolic murmur at the left edge of the sternum.		stabilization, surgical or transcatheter closure of the defect; delayed closure to allow for better operating conditions.	
Free wall rupture	<1% AMI	Sudden cardiac death or progressive tamponade	Tamponade and flow through the free wall of the heart.	Pericardiocentesis and emergency surgery.	15-30%
Pseudoaneurysm	<1% AMI	Months/years of asymptomatic course or non-specific symptoms.	Aneurysm with a narrow neck connecting it to the ventricle.	Surgical closure of the defect	Relatively low

Discussion

Widespread use of early reperfusion (PCI, thrombolysis in appropriate cases) has reduced the incidence of mechanical complications, but has not eliminated them completely. In some cases, mechanical complications such as VSR may be a consequence of reperfusion. Therefore, every patient with severe transmural infarction should be monitored for possible mechanical complications. (2) Sudden hemodynamic deterioration, acute exacerbation of dyspnea, new heart murmur, or signs of tamponade require immediate echocardiography with Doppler imaging. Rapid diagnosis determines the possibility of implementing life-saving therapy. (2) Transthoracic echocardiography remains the first-line examination; in critical conditions, circulatory support devices (IABP, Impella, ECMO) enable stabilization prior to cardiac surgery.

Effective management requires coordination between the intensive care team, interventional cardiologists, echocardiographers, and cardiac surgeons. Every hour of delay significantly worsens the prognosis. Current research focuses on improving catheter-based techniques (e.g., closure of VSR and pseudoaneurysms with Amplatzer devices, or percutaneous mitral clips for PMR in inoperable patients) and on developing criteria for choosing between surgical and minimally invasive intervention. CABG performed simultaneously with surgery may be beneficial, especially in patients with multivessel disease.

Disclosure

The authors declare no conflict of interest in relation to this study.

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All authors have read and agreed with the published version of the manuscript.

Funding Statement

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Consent Statement

Not applicable.

Data Availability Statement

Not applicable.

Conflict of interest

The authors declare no conflict of interests.

Declaration of the use of generative AI and AI-assisted technologies in the writing process. In preparing this work, the authors used ChatGPT for the purpose of improving language and readability, text formatting, and verification of bibliographic styles. After using this tool/service, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

References

1. Dąbek, J., Skorus, P. R., Potyka, K., Potyka, M., Stachoń, K. & Gąsior, Z. (2019). Mechanical complications of acute myocardial infarction, including risk factors, treatment, and patient survival in a 10-year clinical follow-up. Annales Academiae Medicae Silesiensis, 73, 119–129. https://doi.org/10.18794/aams/99256

2. Ibáñez B, Heusch G, Ovize M, Van de Werf F. Mechanical Complications of Acute Myocardial Infarction: A Scientific Statement From the American Heart Association. *Circulation.* 2021;144(24):e136–e152. DOI:10.1161/CIR.0000000000001013.

3. Bhardwaj B, Sidhu G, Balla S, Kumar V, Kumar A, Aggarwal K, Dohrmann ML, Alpert MA. Outcomes and hospital utilization in patients with papillary muscle rupture associated with acute myocardial infarction. *Am J Cardiol*. 2020;125:1020–1025. doi: 10.1016/j.amjcard.2019.12.051

4. Pereira J, Essa M, Sugeng L. Double Rupture of a Tricuspid Papillary Muscle and Ventricular Septum: A Rare Combination after Myocardial Infarction. CASE (Phila). 2019 Jan 25;3(2):85-88. doi: 10.1016/j.case.2018.11.009. PMID: 31049486; PMCID: PMC6479214.

5. Sochowski RA, Chan KL, Ascah KJ, Bedard P. Comparison of accuracy of transesophageal versus transthoracic echocardiography for the detection of mitral valve prolapse with ruptured chordae tendineae (flail mitral leaflet). Am J Cardiol. 1991 Jun 1;67(15):1251-5. doi: 10.1016/0002-9149(91)90936-f. PMID: 2035450.

6. Tehrani BN, Truesdell AG, Psotka MA, Rosner C, Singh R, Sinha SS, Damluji AA, Batchelor WB. A standardized and comprehensive approach to the management of cardiogenic shock. *JACC Heart Fail*. 2020;8:879–891. doi: 10.1016/j.jchf.2020.09.005

7. Massimi G, Matteucci M, Kowalewski M, Ronco D, Chiarini G, De Piero ME, Lo Coco V, Maessen JG, Beghi C, Lorusso R. Operative strategies for acute mitral regurgitation as a mechanical complication of myocardial infarction. Ann Cardiothorac Surg. 2022 May;11(3):328-336. doi: 10.21037/acs-2022-ami-0016. PMID: 35733709; PMCID: PMC9207701.

8. Pahuja M, Ranka S, Chauhan K, Patel A, Chehab O, Elmoghrabi A, Mony S, Ando T, Mishra T, Singh M, Abubaker H, Yassin A, Glazier JJ, Afonso L, Kapur NK, Burkhoff D. Rupture of Papillary Muscle and Chordae Tendinae Complicating STEMI: A Call for Action. ASAIO J. 2021 Aug 1;67(8):907-916. doi: 10.1097/MAT.0000000000001299. PMID: 33093383.

9. French, John K., et al. "Mechanical complications after percutaneous coronary intervention in ST-elevation myocardial infarction (from APEX-AMI)." *The American journal of cardiology* 105.1 (2010): 59-63. https://doi.org/10.1016/j.amjcard.2009.08.653

10. Massimi G, Ronco D, De Bonis M, Kowalewski M, Formica F, Russo CF, Sponga S, Vendramin I, Falcetta G, Fischlein T, Troise G, Trumello C, Actis Dato G, Carrozzini M, Shah SH, Coco VL, Villa E, Scrofani R, Torchio F, Antona C, Kalisnik JM, D'Alessandro S, Pettinari M, Sardari Nia P, Lodo V, Colli A, Ruhparwar A, Thielmann M, Meyns B, Khouqeer FA, Fino C, Simon C, Kowalowka A, Deja MA, Beghi C, Matteucci M, Lorusso R. Surgical treatment for post-infarction papillary muscle rupture: a multicenter study. Eur J Cardiothorac Surg. 2022 Jan 24;61(2):469-476. doi: 10.1093/ejcts/ezab469. PMID: 34718501

11. Treille de Grandsaigne H, Bouisset F, Porterie J, Biendel C, Marcheix B, Lairez O, et al. Incidence, management, and prognosis of post-ischemic ventricular septal defect: insights from a 12-year tertiary center experience. *Front Cardiovasc Med* 2022;9:1066308. https://doi.org/10.3389/fcvm.2022.1066308

12. Menon, V, Webb, J, Hillis, L. et al. Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: a report from the SHOCK Trial Registry. JACC. 2000 Sep, 36 (3_Supplement_1) 1110–1116.
https://doi.org/10.1016/S0735-1097 (00)00878-0

13. Elbadawi A., Elgendy IY., Mahmoud K., Barakat AF., Mentias A., Mohamed AH., et al. Temporal trends and outcomes of mechanical complications in patients with acute myocardial infarction. *JACC Cardiovasc Interv* 2019;12:1825–36. https://doi.org/10.1016/j.jcin.2019.04.039

14. Prabhu S.D., Frangogiannis N.G. The Biological Basis for Cardiac Repair After Myocardial Infarction: From Inflammation to Fibrosis. Circ. Res. 2016; 119(1): 91–112, doi: 10.1161/CIRCRESAHA.116.303577.

15. Edwards BS, Edwards WD, Edwards JE. Ventricular septal rupture complicating acute myocardial infarction: identification of simple and complex types in 53 autopsied hearts. *Am J Cardiol* 1984;54:1201–5. https://doi.org/10.1016/S0002-9149(84)80067-3

16. Cubeddu, R, Lorusso, R, Ronco, D. et al. Ventricular Septal Rupture After Myocardial Infarction: JACC Focus Seminar 3/5. JACC. 2024 May, 83 (19) 1886–1901.
https://doi.org/10.1016/j.jacc.2024.01.041

17. José López-Sendón, Enrique P. Gurfinkel, Esteban Lopez de Sa, Giancarlo Agnelli, Joel M. Gore, Phillippe Gabriel Steg, Kim A. Eagle, Jose Ruiz Cantador, Gordon Fitzgerald, Christopher B. Granger, for the Global Registry of Acute Coronary Events (GRACE) Investigators, Factors related to heart rupture in acute coronary syndromes in the Global Registry of Acute Coronary Events, *European Heart Journal*, Volume 31, Issue 12, June 2010, Pages 1449–1456, https://doi.org/10.1093/eurheartj/ehq061

18. Matteo Matteucci, Dario Fina, Federica Jiritano, Paolo Meani, W Matthijs Blankesteijn, Giuseppe Maria Raffa, Mariusz Kowaleski, Samuel Heuts, Cesare Beghi, Jos Maessen, Roberto Lorusso, Treatment strategies for post-infarction left ventricular free-wall rupture, *European*

Heart Journal. Acute Cardiovascular Care, Volume 8, Issue 4, 1 June 2019, Pages 379–387, https://doi.org/10.1177/2048872619840876

19. Matteucci, Matteo, et al. "Sutured and sutureless repair of postinfarction left ventricular free-wall rupture: a systematic review." *European Journal of Cardio-Thoracic Surgery* 56.5 (2019): 840-848. DOI: 10.1093/ejcts/ezz101

20. Karamitsos TD, Ferreira V, Banerjee R, et al. Contained left ventricular rupture after acute myocardial infarction revealed by cardiovascular magnetic resonance imaging. Circulation. 2012; 125(18): 2278–2280, doi: 10.1161/CIRCULATIONAHA.111.068619, indexed in Pubmed: 22566351.

21. Blinc, Ales, et al. "Subacute rupture of the left ventricular free wall after acute myocardial infarction: three cases of long-term survival without emergency surgical repair." *Chest* 109.2 (1996): 565-567.
https://doi.org/10.1378/chest.109.2.565

22. Yeo TC, Malouf JF, Oh JK, Seward JB. Clinical profile and outcome in 52 patients with cardiac pseudoaneurysm. *Ann Intern Med*. 1998;128:299–305. doi: 10.7326/0003-4819-128-4-199802150-00010

23. Sheikh WR, Sehgal P, Verma A, Haldar M, Jaiswal S. Left ventricular pseudoaneurysm post myocardial infarction. Int J Crit Illn Inj Sci. 2019 Jan-Mar;9(1):43-45. doi: 10.4103/IJCIIS.IJCIIS_42_18. PMID: 30989068; PMCID: PMC6423930.

24. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. *J Am Coll Cardiol*. 1998;32:557–561. doi: 10.1016/s0735-1097(98)00290-3

25. Costa MA, Pencina M, Nikolic S, Engels T, Templin B, Abraham WT. The PARACHUTE IV trial design and rationale: percutaneous ventricular restoration using the parachute device in patients with ischemic heart failure and dilated left ventricles. *Am Heart J*. 2013;165:531–536. doi: 10.1016/j.ahj.2012.12.022

26. Zhong Z, Song W, Zheng S, Liu S. Surgical and Conservative Treatment of Post-infarction Left Ventricular Pseudoaneurysm. Front Cardiovasc Med. 2022 Jan 27;9:801511. doi: 10.3389/fcvm.2022.801511. PMID: 35155628; PMCID: PMC8829002.