Journal of Advances in Medicine and Medical Research

Volume 35, Issue 17, Page 32-43, 2023; Article no.JAMMR.101475
ISSN: 2456-8899
(Past name: British Journal of Medicine and Medical Research, Past ISSN: 2231-0614, NLM ID: 101570965)

A Review of Medical Emergencies Related to the Embolic, Inflammatory, and Mechanical Complications in Patients with Acute Myocardial Infarction

Hassan Nabhani [a*]

[a] *Cleveland Clinic Abu Dhabi, United Arab Emirates.*

Author's contribution

The sole author designed, analyzed, interpreted and prepared the manuscript.

Article Information

DOI: 10.9734/JAMMR/2023/v35i175101

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/101475

Systematic Review Article

Received: 10/04/2023
Accepted: 12/06/2023
Published: 29/06/2023

ABSTRACT

Although the incidence of coronary heart disease (CHD) has decreased in the United States, acute myocardial infarction (AMI) remains a major medical problem. Many patients still need emergency care in the emergency department (ED) due to complications due to mechanical, inflammatory, and embolic factors. This summary aims to provide a brief summary of the available evidence in clinical medicine regarding the evaluation and management of mechanical, inflammatory, and embolic complications after myocardial infarction. Although 30-day mortality after AMI has decreased in recent years, it remains high at 7.8% due to many subacute complications that occur within a few weeks. Mechanical problems such as ventricular free wall rupture, ventricular septal rupture, mitral regurgitation, and left ventricular aneurysm formation have a high risk of morbidity. The complications of MI can vary in severity and can affect different organs and systems of the

**Corresponding author: E-mail: drhassannabhani@gmail.com;*

body. Heart attacks can disrupt the normal electrical activity of the heart, leading to irregular heart rhythms, such as ventricular tachycardia or ventricular fibrillation. A heart attack can weaken the heart muscle, making it difficult for the heart to pump blood effectively. This can lead to heart failure, a condition where the heart is unable to meet the body's demand for blood and oxygen. In severe cases, a heart attack can cause cardiogenic shock, a condition characterized by a significant drop in blood pressure and inadequate blood flow to vital organs. This is a life-threatening situation that requires immediate medical attention. Sometimes, a heart attack can cause a rupture in the wall separating the heart's chambers (ventricular septum), leading to the formation of a VSD. This condition allows blood to flow between the ventricles, decreasing the heart's ability to pump blood effectively.

Mechanical, inflammatory, and embolic complications associated with AMI can have a significant impact on morbidity and mortality. Physicians should check for existing conditions when considering other diagnoses. In addition to monitoring disease progression and physical examination, electrocardiography and point-of-care echocardiography allow rapid and noninvasive assessment of the underlying pathophysiology. Management may vary depending on the particular presentation and etiology; however, relationships with cardiologists and cardiologists are recommended.

Keywords: Emergency medicine; embolic complications; acute myocardial infarction; complicated myocardial infarction.

1. INTRODUCTION

Atherosclerotic cardiovascular disease is the leading cause of death worldwide [1] Although the incidence of coronary heart disease (CHD) has decreased in the United States In the United States, studies show that the incidence of myocardial infarction (AMI) is not decreasing, at approximately 208 per 100,000 person-years, of which it is approximately 22%. ST-segment elevation myocardial infarction (STEMI) [2]. Although there is an improvement in 30-day mortality after AMI, it remains as high as 7 days 8% [2].

Ischemic complications can affect the kidneys, leading to renal failure, and embolic strokes from arrhythmias can affect the brain and heart with the development of left ventricular (LV) aneurysms [7–9]. Ischemic complications may also be associated with stent thrombosis and secondary heart failure with increased morbidity [10,11]. In the 1980s the name of myocardial infarction was changed to include ECG evidence of MI. Q wave MI replaces the old definition of transmural MI and is used for MI involving all three layers of the myocardium, causing pathological Q waves in both leads on the ECG. Non-transmural MIs are referred to as non-Q-wave MIs on the assumption that the Q wave will not appear on the ECG unless the entire myocardial thickness is included [12-31]. However, clinical trials in subsequent years have not confirmed that Q-wave MI is equivalent to transmural MI. Thus, in the 1990s, ST-segment elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI) were considered the preferred terms [3-6].

2. METHODS

Methods included include published literature, case reports, retrospective and prospective studies, systematic reviews and meta-analyses, clinical trials, and other reviews. Search is limited to English publications. Initially, more than 600 articles were identified and, with the agreement of the authors, important articles focusing on emergency medical information and guidelines were selected as medicine. Finally, 177 emergency treatment and referral programs were included. As for descriptive analysis, individual survey data were not collected.

3. LITERATURE REVIEW

3.1 Ventricular Free Wall Rupture

3.1.1 Etiology

Left ventricular free-wall rupture is a critical mechanical complication that can arise from an acute myocardial infarction (heart attack). It primarily occurs in individuals with ST-elevation myocardial infarction (STEMI), although it can also be caused by infections or trauma. Timely diagnosis relies on maintaining a high level of suspicion and employing transthoracic echocardiography to aid in the assessment. VFWR can be characterized as simple or

complex and may involve the inner or posterior wall of the left ventricle (LV). Simple VFWRs provide direct access to the defect, while complex VFWRs exhibit convoluted anatomical bundles that connect with the primary tear.

Although posterior and posterior AMIs are more prone to white wall ruptures, they are less common in anterior AMIs because they are more common. Subacute VFWRs are often associated with inferior infarcts [72-81]. The distribution of infarcted arteries causing VFWR was as follows: circumflex coronary artery (LCX) in 40% of patients, left anterior descending artery (LAD) in 42%, and right coronary artery (RCA) in 18%.

3.1.2 Risk factors

Risk factors for VFWR include coexistence, age over 55 years (usually 65-70 years), high blood pressure, large infarcts, single artery (usually total occlusion), transmural infarcts, pseudoaneurysm formation, delayed or incomplete revascularization. Patients without a history of angina are more likely to develop VFWR after their first AMI, suggesting a limited risk of coronary artery disease.

3.1.3 Diagnosis and presentation

Early detection of VFWR is important to patient survival. The clinical presentation may vary, with some patients experiencing prolonged or short-term angina in the days before rupture. Physical exertion and persistent ischemia can cause VFWR. Patients may also have sexual dysfunction, shortness of breath, or hypotension suggestive of VFWR and cardiac arrest [32-49]. Electrocardiography (ECG) has limited specificity in diagnosing VFWR, but some findings such as abnormal electrical activity or electrical changes may be seen. Echocardiography plays an important role in the diagnosis by showing signs of pericardial effusion and cardiac tamponade [50-71].

Chest radiography and computed tomography angiography may provide additional information but are not usually required for diagnosis.

3.1.4 Prognosis

The prognosis of VFWR remains poor, with a mortality rate ranging from 75% to 94%. Subacute VFWR is less likely to cause rapid hemodynamic deterioration. Effective surgical treatment was associated with a 48-year survival. Between 5% and 76% and most well-treated patients have long survival [82-112].

3.2 Ventricular Septal Rupture (VSR)

3.2.1 Causes

VSR can be classified as simple or complex. Simple VSRs are characterized by a single disorder in the interventricular system, while complex VSRs involve multiple pathways connecting the ventricles that develop after septal rupture. The location of the VSR can provide insight into subtypes such as the complex forms associated with posteroinferior infarcts and the simpler forms seen in posterior-anterior infarcts.

3.2.2 Diagnosis and presentation

Acute mitral regurgitation (MR) usually occurs 2-7 days after myocardial infarction (AMI) with an average duration of 13 hours. The clinical presentation varies according to the severity of MR. In the case of fulminant MR due to papillary muscle rupture, patients may develop pulmonary edema, hypotension, and cardiogenic shock. However, the physical examination may be nonspecific and some patients with mild to severe MR may have a nonspecific complaint. However, a diastolic murmur towards the left axilla and a pan systolic murmur at the highest apex can cause MR [114,120].

Transthoracic echocardiography (TTE) can provide valuable information about the size and direction of the regurgitation jet, visualize the anterior surface and evidence of mal coaptation, and assess biventricular function.

In the case of incomplete TTE, transesophageal echocardiography (TEE) is required, especially if MR is suspicious despite incomplete TTE results.

3.2.3 Timely management

Early diagnosis and surgical treatment of MR are important to improve survival. Current management includes the management of pulmonary edema, which may require mechanical ventilation (NIV) or endotracheal intubation. Medical therapy aims to advance the anterior left ventricle and reduce MR.

Vasodilators such as sodium nitroprusside or nitroglycerin are used to reduce afterload, while inotropes such as dobutamine improve inotropy

and vasodilation. Consider supplementing any device with devices such as an intra aortic balloon pump (IABP), Impella, or extracorporeal membrane oxygenation (ECMO). The decision for surgical treatment or percutaneous intervention depends on the severity and underlying etiology of MR and requires the cooperation of specialist physicians and interventional cardiologists [125,126].

3.2.4 Prognosis

Chronic MR after AMI has a poor prognosis if left untreated. Mortality is approximately 75% in 24 hours and increases to 95% in 2 weeks. Surgical treatment has an operative mortality of approximately 39% and in-hospital mortality of 55%.

3.3 Pericarditis after Myocardial Infarction (PMIP)

3.3.1 Etiology

It is caused due to the inflammation of the perimyocardium after myocardial infarction. This causes inflammation and an autoimmune response in exposed individuals. The inflammation may appear as simple pericarditis or progress to more serious conditions such as cardiac tamponade, pleural effusion, and/or pleuropericarditis.

PMIP can be divided into two types depending on time. Previously, early PMIP was estimated to occur in 10-20% of patients after AMI, but with the advent of thrombolytic therapy, this rate has dropped to about 6% [118,119].

The incidence of late PMIP, formerly known as DS, is 3-4%, but modern studies show the incidence to be less than 1%.

3.3.2 Risk factors

There are many risk factors that can contribute to the development of PMIP. These include delayed reperfusion, higher cardiovascular biomarker levels, smaller size, younger age, anterior ischemia, right ventricular and inferior infarction, and decreased left ventricular ejection fraction [121–124].

3.3.3 Clinical diagnosis and presentation

Acute pericarditis, while often resolving on its own, can lead to notable complications. It is important to distinguish between acute idiopathic pericarditis and pericarditis resulting from cardiac injury, as they have distinct underlying mechanisms, complication profiles, and natural progressions. Measuring serum troponin I levels may help identify patients with elevated levels who paradoxically face a lower risk of treatment failure, recurrences, constrictive pericarditis, tamponade, or death. Patients who exhibit a positive response to anti-inflammatory medications typically have a favorable prognosis. Nevertheless, there is a possibility of experiencing complications such as cardiac tamponade, constrictive pericarditis, and recurrent episodes [128-133].

3.3.4 Management

Although specific information on PMIP management is limited, treatment guidelines can be determined from studies of acute pericarditis. For early PMIP, which usually resolves on its own, it is recommended not to use non-steroidal anti-inflammatory drugs (NSAIDs) other than once-daily aspirin for secondary prevention during the first 7-10 days after AMI.

Acetaminophen is the drug of choice for patients with severe symptoms requiring sedation, and high-dose aspirin is considered second-line [123].

Management of advanced PMIP usually includes supportive care and drug therapy with NSAIDs, colchicine and corticosteroids [115–117]. Because of the need for antiplatelet therapy and the potential interaction of other NSAIDs with myocardial damage and scar formation, aspirin is the first choice for PMIP patients [115]. Indomethacin should be avoided as it reduces blood flow [113]. High-dose aspirin (800 mg orally every 6-8 hours for 7-10 days, then tapering down to 800 mg weekly for 3 weeks) has been shown to be effective in most cases in research [127].

Ibuprofen has been shown to increase blood flow in the arteries and can be administered at a weekly dose of 400 to 800 mg for a period of 3-4 weeks, with doses of 600 to 800 mg every 6 to 8 hours [124]. It is important to provide gastrointestinal protection in all patients treated with NSAIDs, usually proton pump inhibitors [115].

Low-dose corticosteroid therapy (eg, prednisone 0.2-0.5 mg/kg/day for 4 weeks followed by

tapering) has been shown to be effective in patients who discontinue aspirin/NSAIDs or who do not respond to treatment.

If steroids are used, the dose should be reduced to prevent the recurrence of PMIP. In the case of PMIP complicated by cardiac tamponade, pericardiocentesis is required [115,117].

3.4 Other Complications

Many subacute complications can occur after acute myocardial infarction (AMI). These complications include ischemic stroke, kidney failure, heart failure, and cardiac arrhythmias. Some individuals may experience recurring chest pain or angina even after the heart attack has occurred. This can be caused by partial blockages in the coronary arteries or persistent narrowing of the blood vessels supplying the heart. Following a heart attack, inflammation of the pericardium (the sac surrounding the heart) can occur, causing chest pain, fever, and other symptoms. Pericarditis can sometimes lead to complications such as pericardial effusion (accumulation of fluid in the pericardium) or constrictive pericarditis (thickening and stiffening of the pericardium). Sometimes, a heart attack can cause a rupture in the wall separating the heart's chambers (ventricular septum), leading to the formation of a VSD. This condition allows blood to flow between the ventricles, decreasing the heart's ability to pump blood effectively.

4. CONCLUSION

These complications include all embolic, ischemic, arrhythmic, and inflammatory processes that occur in the weeks after AMI. Ventricular white wall rupture, ventricular septum rupture, acute mitral valve insufficiency, left ventricular aneurysm, pericarditis after myocardial infarction, etc. may appear in the form. Effective management requires a good understanding of the underlying pathophysiology and collaboration with cardiology and cardiac surgery specialists is recommended when needed.

CONSENT AND ETHICAL APPROVAL

It is not applicable.

ACKNOWLEDGEMENTS

The idea for this manuscript and its writing and editing were contributed by WTD, TM, BL, and AK. Dr. William Brady approved the concept and structure of this review. No grants or funding were utilized for this manuscript, and it has not been presented in abstract form. The publication of this review has been approved by all authors and the responsible authorities where the work was conducted. It is an original work that has not been published elsewhere and will not be submitted elsewhere without proper consent. The views and opinions expressed in this review do not reflect those of the U.S. government, Department of Defense, U.S. Army, U.S. Air Force, or SAUSHEC EM Residency Program.

COMPETING INTERESTS

Author has declared that no competing interests exist.

REFERENCES

1. Barquera S, Pedroza-Tobias A, Medina C, Hernandez-Barrera L, Bibbins-Domingo K, Lozano R, et al. Global overview of the epidemiology of atherosclerotic cardiovascular disease. Arch Med Res. 2015;46(5):328–38.
2. Yeh RW, Sidney S, Chandra M, Sorel M, Selby JV, Go AS. Population trends in the incidence and outcomes of acute myocardial infarction. N Engl J Med. 2010;362(23): 2155–65.
3. French JK, Hellkamp AS, Armstrong PW, Cohen E, Kleiman NS, O'Connor CM, et al. Mechanical complications after percutaneous coronary intervention in ST1180 T. Montrief et al. / American Journal of Emergency Medicine 37 (2019) 1175–1183 elevation myocardial infarction (from APEX-AMI). Am J Cardiol. 2010; 105(1):59–63.
4. Kutty RS, Jones N, Moorjani N. Mechanical complications of acute myocardial infarction. Cardiol Clin 2013;31(4):519–31 [vii–viii].
5. Aronson D, Goldsher N, Zukermann R, Kapeliovich M, Lessick J, Mutlak D, et al. Ischemic mitral regurgitation and risk of heart failure after myocardial infarction. Arch Intern Med 2006;166(21): 2362–8.
6. Schmitt J, Duray G, Gersh BJ, Hohnloser SH. Atrial fibrillation in acute myocardial infarction: a systematic review of the incidence, clinical features, and prognostic implications. Eur Heart J. 2009;30(9): 1038–45.

7. Dutta M, Hanna E, Das P, Steinhubl SR. Incidence and prevention of ischemic stroke following myocardial infarction: review of current literature. Cerebrovasc Dis. 2006;22(5–6):331–9.
8. Putaala J, Nieminen T. Stroke risk period after acute myocardial infarction revised. J Am Heart Assoc. 2018;7(22):e011200.
9. Shacham Y, Steinvil A, Arbel Y. Acute kidney injury among ST-elevation myocardial infarction patients treated by primary percutaneous coronary intervention: a multifactorial entity. J Nephrol. 2016;29(2):169–74.
10. Beinart R, Abu Sham'a R, Segev A, Hod H, Guetta V, Shechter M, et al. The incidence and clinical predictors of early stent thrombosis in patients with acute coronary syndrome. Am Heart J. 2010;159(1): 118–24.
11. Torabi A, Cleland JG, Rigby AS, Sherwi N. Development and course of heart failure after myocardial infarction in younger and older people. J Geriatr Cardiol. 2014; 11(1):1–12.
12. Wehrens XH, Doevendans PA. Cardiac rupture complicates myocardial infarction. Int J Cardiol. 2004;95(2–3):285–92.
13. Oliva PB, Hammill SC, Edwards WD. Cardiac rupture, a clinically predictable complication of acute myocardial infarction: report of 70 cases with clinicopathologic correlations. J Am Coll Cardiol. 1993; 22(3):720–6.
14. Keeley EC, de Lemos JA. Free wall rupture in the elderly: deleterious effect of fibrinolytic therapy on the ageing heart. Eur Heart J. 2005;26(17):1693–4.
15. Moreno R, Lopez de Sa E, Lopez-Sendon JL, Garcia E, Soriano J, Abeytua M, et al. Frequency of left ventricular free-wall rupture in patients with acute myocardial infarction treated with primary angioplasty. Am J Cardiol. 2000;85(6):757–60, [A8].
16. Yip HK, Wu CJ, Chang HW, Wang CP, Cheng CI, Chua S, et al. Cardiac rupture complicating acute myocardial infarction in the direct percutaneous coronary intervention reperfusion era. Chest. 2003;124(2):565–71.
17. Hutchins KD, Skurnick J, Lavenhar M, Natarajan GA. Cardiac rupture in acute myocardial infarction: a reassessment. Am J Forensic Med Pathol. 2002;23(1):78–82.
18. Slater J, Brown RJ, Antonelli TA, Menon V, Boland J, Col J, et al. Cardiogenic shock due to cardiac free-wall rupture or tamponade after acute myocardial infarction: a report from the SHOCK Trial Registry. Should we emergently revascularize occluded coronaries for cardiogenic shock? J Am Coll Cardiol. 2000;36(3 Suppl A):1117–22.
19. Purcaro A, Costantini C, Ciampani N, Mazzanti M, Silenzi C, Gili A, et al. Diagnostic criteria and management of subacute ventricular free wall rupture complicating acute myocardial infarction. Am J Cardiol. 1997;80(4):397–405.
20. Ng R, Yeghiazarians Y. Post myocardial infarction cardiogenic shock: a review of current therapies. J Intensive Care Med. 2013;28(3):151–65.
21. Crenshaw BS, Granger CB, Birnbaum Y, Pieper KS, Morris DC, Kleiman NS, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. Circulation. 2000;101(1):27–32.
22. Pollak H, Diez W, Spiel R, Enenkel W, Mlczoch J. Early diagnosis of subacute free wall rupture complicating acute myocardial infarction. Eur Heart J. 1993;14(5): 640–8.
23. Mann JM, Roberts WC. Rupture of the left ventricular free wall during acute myocardial infarction: analysis of 138 necropsy patients and comparison with 50 necropsy patients with acute myocardial infarction without rupture. Am J Cardiol. 1988;62(13):847–59.
24. Batts KP, Ackermann DM, Edwards WD. Postinfarction rupture of the left ventricular free wall: clinicopathologic correlates in 100 consecutive autopsy cases. Hum Pathol. 1990;21(5):530–5.
25. Haddadin S, Milano AD, Faggian G, Morjan M, Patelli F, Golia G, et al. Surgical treatment of postinfarction left ventricular free wall rupture. J Card Surg. 2009;24(6):624–31.
26. Blinc A, Noc M, Pohar B, Cernic N, Horvat M. Subacute rupture of the left ventricular free wall after acute myocardial infarction. Three cases of long-term survival without emergency surgical repair. Chest. 1996;109(2):565–7.
27. Markowicz-Pawlus E, Nozynski J, Duszanska A, Hawranek M, Jarski P, Kalarus Z. The impact of a previous history of ischaemic episodes on the occurrence

of left ventricular free wall rupture in the setting of myocardial infarction. Kardiol Pol. 2012;70(7):713–7.
28. Figueras J, Curos A, Cortadellas J, Sans M, Soler-Soler J. Relevance of electrocardiographic findings, heart failure, and infarct site in assessing risk and timing of left ventricular free wall rupture during acute myocardial infarction. Am J Cardiol. 1995;76(8):543–7.
29. Melchior T, Hildebrant P, Kober L, Jensen G, Torp-Pedersen C. Do diabetes mellitus and systemic hypertension predispose to left ventricular free wall rupture in acute myocardial infarction? Am J Cardiol. 1997;80(9):1224–5.
30. Figueras J, Cortadellas J, Calvo F, Soler-Soler J. Relevance of delayed hospital admission on development of cardiac rupture during acute myocardial infarction: study in 225 patients with free wall, septal or papillary muscle rupture. J Am Coll Cardiol. 1998;32(1):135–9.
31. Pretre R, Ye Q, Grunenfelder J, Zund G, Turina MI. Role of myocardial revascularization in postinfarction ventricular septal rupture. Ann Thorac Surg. 2000;69(1): 51–5.
32. Lopez-Sendon J, Gonzalez A, Lopez de Sa E, Coma-Canella I, Roldan I, Dominguez F, et al. Diagnosis of subacute ventricular wall rupture after acute myocardial infarction: sensitivity and specificity of clinical, hemodynamic and echocardiographic criteria. J Am Coll Cardiol. 1992;19(6):1145–53.
33. Pollak H, Mlczoch J. Effect of nitrates on the frequency of left ventricular free wall rupture complicating acute myocardial infarction: a case-controlled study. Am Heart J. 1994;128(3):466–71.
34. Shiyovich A, Nesher L. Contained left ventricular free wall rupture following myocardial infarction. Case Rep Crit Care. 2012;2012:467810.
35. Herlitz J, Samuelsson SO, Richter A, Hjalmarson A. Prediction of rupture in acute myocardial infarction. Clin Cardiol. 1988;11(2):63–9.
36. Pohjola-Sintonen S, Muller JE, Stone PH, Willich SN, Antman EM, Davis VG, et al. Ventricular septal and free wall rupture complicating acute myocardial infarction: experience in the Multicenter Investigation of Limitation of Infarct Size. Am Heart J. 1989;117(4): 809–18.
37. Yoshikawa T, Inoue S, Abe S, Akaishi M, Mitamura H, Ogawa S, et al. Acute myocardial infarction without warning: clinical characteristics and significance of preinfarction angina. Cardiology. 1993; 82(1):42–7.
38. Cheriex EC, de Swart H, Dijkman LW, Havenith MG, Maessen JG, Engelen DJ, et al. Myocardial rupture after myocardial infarction is related to the perfusion status of the infarct-related coronary artery. Am Heart J. 1995;129(4):644–50.
39. Figueras J, Cortadellas J, Soler-Soler J. Left ventricular free wall rupture: clinical presentation and management. Heart. 2000;83(5):499–504.
40. Birnbaum Y, Chamoun AJ, Anzuini A, Lick SD, Ahmad M, Uretsky BF. Ventricular free wall rupture following acute myocardial infarction. Coron Artery Dis. 2003;14(6): 463–70.
41. Mahilmaran A, Nayar PG, Sheshadri M, Sudarsana G, Abraham KA. Left ventricular pseudoaneurysm caused by coronary spasm, myocardial infarction, and myocardial rupture. Tex Heart Inst J. 2002;29(2):122–5.
42. Figueras J, Curos A, Cortadellas J, Soler-Soler J. Reliability of electromechanical dissociation in the diagnosis of left ventricular free wall rupture in acute myocardial infarction. Am Heart J. 1996;131(5):861–4.
43. Che J, Li G, Chen K, Liu T. Post-MI free wall rupture syndrome. Case report, literature review, and new terminology. Clin Case Rep. 2016;4(6):576–83.
44. Roberts JD, Mong KW, Sussex B. Successful management of left ventricular free wall rupture. Can J Cardiol. 2007;23(8):672–4.
45. Honasoge AP, Dubbs SB. Rapid fire: pericardial effusion and tamponade. Emerg Med Clin North Am. 2018;36(3):557–65.
46. Honda S, Asaumi Y, Yamane T, Nagai T, Miyagi T, Noguchi T, et al. Trends in the clinical and pathological characteristics of cardiac rupture in patients with acute myocardial infarction over 35 years. J Am Heart Assoc. 2014;3(5):e000984.
47. Wehrens XH, Doevendans PA, Widdershoven JW, Dassen WR, Prenger K, Wellens HJ, et al. Usefulness of sinus tachycardia and ST-segment elevation in V(5) to identify impending left ventricular free wall rupture in inferior wall myocardial infarction. Am J Cardiol. 2001;88(4):414–7.

48. Alerhand S, Carter JM. What echocardiographic findings suggest a pericardial effusion is causing tamponade? Am J Emerg Med. 2019;37(2):321–6.
49. Perez-Casares A, Cesar S, Brunet-Garcia L, Sanchez-de-Toledo J. Echocardiographic evaluation of pericardial effusion and cardiac tamponade. Front Pediatr. 2017;5:79.
50. Eisenberg MJ, Dunn MM, Kanth N, Gamsu G, Schiller NB. Diagnostic value of chest radiography for pericardial effusion. J Am Coll Cardiol. 1993;22(2):588–93.
51. Onoda N, Nonami A, Yabe T, Doi YL, Fujita Y, Yamamoto S, et al. Postinfarct cardiac free wall rupture detected by multidetector computed tomography. J Cardiol Cases. 2012;5(3):e147-e9.
52. Hoshino A, Yokoya S, Enomoto S, Kawahito H, Kurata H, Nakahara Y, et al. [Survivor of blow out type of free wall rupture: multislice computed tomographic detection of myocardial rupture in a case of small myocardial infarction]. J Cardio.l 2007;49 (2):97–102.
53. Brenes JA, Keifer T, Karim RM, Shroff GR. Adjuvant role of CT in the diagnosis of post-infarction left ventricular free-wall rupture. Cardiol Res. 2012;3(6):284–7.
54. Mantovani V, Vanoli D, Chelazzi P, Lepore V, Ferrarese S, Sala A. Post-infarction cardiac rupture: surgical treatment. Eur J Cardiothorac Surg. 2002;22(5):777–80.
55. Kumar R, Sinha A, Lin MJ, Uchino R, Butryn T, O'Mara MS, et al. Complications of pericardiocentesis: a clinical synopsis. Int J Crit Illn Inj Sci. 2015;5(3):206–12.
56. Figueras J, Alcalde O, Barrabes JA, Serra V, Alguersuari J, Cortadellas J, et al. Changes in hospital mortality rates in 425 patients with acute ST-elevation myocardial infarction and cardiac rupture over a 30-year period. Circulation. 2008;118(25):2783–9.
57. Zoffoli G, Battaglia F, Venturini A, Asta A, Terrini A, Zanchettin C, et al. A novel approach to ventricular rupture: clinical needs and surgical technique. Ann Thorac Surg. 2012;93(3):1002–3.
58. Flajsig I, Castells y Cuch E, Mayosky AA, Rodriguez R, Calbet JM, Saura E, et al. Surgical treatment of left ventricular free wall rupture after myocardial infarction: case series. Croat Med J. 2002;43(6): 643–8.
59. Hochman JS, Buller CE, Sleeper LA, Boland J, Dzavik V, Sanborn TA, et al. Cardiogenic shock complicating acute myocardial infarction—etiologies, management, and outcome: a report from the SHOCK Trial Registry. SHould we emergently revascularize Occluded Coronaries for cardiogenic shocK? J Am Coll Cardiol. 2000;36(3 Suppl A): 1063–70.
60. Radford MJ, Johnson RA, Daggett WM, Jr., Fallon JT, Buckley MJ, Gold HK, et al. Ventricular septal rupture: a review of clinical and physiologic features and an analysis of survival. Circulation. 1981; 64(3):545–53.
61. Holmes DR, Jr., Bates ER, Kleiman NS, Sadowski Z, Horgan JH, Morris DC, et al. Contemporary reperfusion therapy for cardiogenic shock: the GUSTO-I trial experience. The GUSTO-I Investigators. Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries. J Am Coll Cardiol. 1995;26(3):668–74.
62. Skillington PD, Davies RH, Luff AJ, Williams JD, Dawkins KD, Conway N, et al. Surgical treatment for infarct-related ventricular septal defects. Improved early results combined with analysis of late functional status. J Thorac Cardiovasc Surg. 1990;99(5):798–808.
63. Hutchins GM. Rupture of the interventricular septum complicating myocardial infarction: pathological analysis of 10 patients with clinically diagnosed perforations. Am Heart J. 1979;97(2): 165–73.
64. Daggett WM, Buckley MJ, Akins CW, Leinbach RC, Gold HK, Block PC, et al. Improved results of surgical management of postinfarction ventricular septal rupture. Ann Surg. 1982;196(3):269–77.
65. Serpytis P, Karvelyte N, Serpytis R, Kalinauskas G, Rucinskas K, Samalavicius R, et al. Post-infarction ventricular septal defect: risk factors and early outcomes. Hellenic J Cardiol. 2015;56(1): 66–71.
66. Skehan JD, Carey C, Norrell MS, de Belder M, Balcon R, Mills PG. Patterns of coronary artery disease in post-infarction ventricular septal rupture. Br Heart J. 1989;62(4):268–72.
67. Toma M, Fu Y, Ezekowitz JA, McAlister FA, Westerhout CM, Granger CB, et al. Does silent myocardial infarction add prognostic value in ST-elevation myocardial infarction patients without a history of prior myocardial infarction?

Insights from the Assessment of Pexelizumab in Acute Myocardial Infarction (APEX-AMI) Trial. Am Heart J. 2010;160(4):671–7.
68. Menon V, Webb JG, Hillis LD, Sleeper LA, Abboud R, Dzavik V, et al. Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: a report from the SHOCK Trial Registry. SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK? J Am Coll Cardiol. 2000;36(3 Suppl A): 1110–6.
69. Glancy DL, Khuri BN, Mustapha JA, Menon PV, Hanna EB. Myocardial infarction with ventricular septal rupture and cardiogenic shock. Proc (Bayl Univ Med Cent). 2015;28(4):512–3.
70. Reeder GS. Identification and treatment of complications of myocardial infarction. Mayo Clin Proc. 1995;70(9):880–4.
71. Topaz O, Taylor AL. Interventricular septal rupture complicating acute myocardial infarction: from pathophysiologic features to the role of invasive and noninvasive diagnostic modalities in current management. Am J Med. 1992;93(6): 683–8.
72. Lemery R, Smith HC, Giuliani ER, Gersh BJ. Prognosis in rupture of the ventricular septum after acute myocardial infarction and role of early surgical intervention. Am J Cardiol. 1992;70(2):147–51.
73. Maganti K, Rigolin VH, Sarano ME, Bonow RO. Valvular heart disease: diagnosis and management. Mayo Clin Proc. 2010;85(5):483–500.
74. Jones BM, Kapadia SR, Smedira NG, Robich M, Tuzcu EM, Menon V, et al. Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. Eur Heart J. 2014;35(31):2060–8.
75. Vlodaver Z, Edwards JE. Rupture of ventricular septum or papillary muscle complicating myocardial infarction. Circulation. 1977;55(5):815–22.
76. Vargas-Barron J, Molina-Carrion M, Romero-Cardenas A, Roldan FJ, Medrano GA, Avila-Casado C, et al. Risk factors, echocardiographic patterns, and outcomes in patients with acute ventricular septal rupture during myocardial infarction. Am J Cardiol. 2005;95(10): 1153–8.
77. Evrin T, Unluer EE, Kuday E, Bayata S, Surum N, Eser U, et al. Bedside echocardiography in acute myocardial infarction patients with hemodynamic deterioration. J Natl Med Assoc. 2018;110(4):396–8.
78. Smyllie JH, Sutherland GR, Geuskens R, Dawkins K, Conway N, Roelandt JR. Doppler color flow mapping in the diagnosis of ventricular septal rupture and acute mitral regurgitation after myocardial infarction. J Am Coll Cardiol. 1990;15(6):1449–55.
79. Fortin DF, Sheikh KH, Kisslo J. The utility of echocardiography in the diagnostic strategy of postinfarction ventricular septal rupture: a comparison of twodimensional echocardiography versus Doppler color flow imaging. Am Heart J. 1991;121(1 Pt 1):25–32.
80. Awasthy N, Radhakrishnan S. Stepwise evaluation of left to right shunts by echocardiography. Indian Heart J. 2013;65(2):201–18.
81. Konstantinides S, Geibel A, Kasper W, Just H. Noninvasive estimation of right ventricular systolic pressure in postinfarction ventricular septal rupture: an assessment of two Doppler echocardiographic methods. Crit Care Med. 1997;25(7):1167–74.
82. Murday A. Optimal management of acute ventricular septal rupture. Heart. 2003;89 (12):1462–6.
83. Malhotra A, Patel K, Sharma P, Wadhawa V, Madan T, Khandeparkar J, et al. Techniques, timing & prognosis of post infarct ventricular septal repair: a re-look at old dogmas. Braz J Cardiovasc Surg. 2017;32(3):147–55.
84. Tariq S, Aronow WS. Use of inotropic agents in treatment of systolic heart failure. Int J Mol Sci 2015;16(12):29060–8.
85. Bayram M, De Luca L, Massie MB, Gheorghiade M. Reassessment of dobutamine, dopamine, and milrinone in the management of acute heart failure syndromes. Am J Cardiol. 2005;96(6A):47G–58G.
86. George I, Xydas S, Topkara VK, Ferdinando C, Barnwell EC, Gableman L, et al. Clinical indication for use and outcomes after inhaled nitric oxide therapy. Ann Thorac Surg. 2006;82(6): 2161–9.
87. Kettner J, Sramko M, Holek M, Pirk J, Kautzner J. Utility of intra-aortic balloon pump support for ventricular septal rupture and acute mitral regurgitation complicating

acute myocardial infarction. Am J Cardiol. 2013;112(11):1709–13.
88. Rob D, Spunda R, Lindner J, Rohn V, Kunstyr J, Balik M, et al. A rationale for early extracorporeal membrane oxygenation in patients with postinfarction ventricular septal rupture complicated by cardiogenic shock. Eur J Heart Fail. 2017;19 Suppl 2:97–103.
89. Sheu JJ, Tsaii TH, Lee FY, Fang HY, Sun CK, Leu S, et al. Early extracorporeal membrane oxygenator-assisted primary percutaneous coronary intervention improved 30-day clinical outcomes in patients with ST-segment elevation myocardial infarction complicated with profound cardiogenic shock. Crit Care Med. 2010;38(9): 1810–7.
90. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database. Ann Thorac Surg. 2012;94(2): 436–43 [discussion 43-4].
91. La Torre MW, Centofanti P, Attisani M, Patane F, Rinaldi M. Posterior ventricular septal defect in presence of cardiogenic shock: early implantation of the Impella recover LP 5.0 as a bridge to surgery. Tex Heart Inst J. 2011;38(1):42–9.
92. Poulsen SH, Praestholm M, Munk K, Wierup P, Egeblad H, Nielsen-Kudsk JE. Ventricular septal rupture complicating acute myocardial infarction: clinical characteristics and contemporary outcome. Ann Thorac Surg. 2008;85(5):1591–6.
93. Jeppsson A, Liden H, Johnsson P, Hartford M, Radegran K. Surgical repair of post-infarction ventricular septal defects: a national experience. Eur J Cardiothorac Surg. 2005;27(2):216–21.
94. Thompson CR, Buller CE, Sleeper LA, Antonelli TA, Webb JG, Jaber WA, 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 revascularized Occluded Coronaries in cardiogenic shock? J Am Coll Cardiol. 2000;36(3 Suppl A):1104–9.
95. Kishon Y, Oh JK, Schaff HV, Mullany CJ, Tajik AJ, Gersh BJ. 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):1023–30.
96. Tcheng JE, Jackman JD, Jr., Nelson CL, Gardner LH, Smith LR, Rankin JS, et al. Outcome of patients sustaining acute ischemic mitral regurgitation during myocardial infarction. Ann Intern Med. 1992;117(1):18–24.
97. Glasson JR, Komeda M, Daughters GT, Bolger AF, Karlsson MO, Foppiano LE, et al. Early systolic mitral leaflet “loitering” during acute ischemic mitral regurgitation. J Thorac Cardiovasc Surg. 1998;116(2):193–205.
98. Lai DT, Tibayan FA, Myrmel T, Timek TA, Dagum P, Daughters GT, et al. Mechanistic insights into posterior mitral leaflet inter-scallop malcoaptation during acute ischemic mitral regurgitation. Circulation. 2002;106(12 Suppl 1):I40-I5.
99. Kimura T, Roger VL, Watanabe N, Barros-Gomes S, Topilsky Y, Nishino S, 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. 2018.
100. Lamas GA, Mitchell GF, Flaker GC, Smith SC, Jr., Gersh BJ, Basta L, et al. Clinical significance of mitral regurgitation after acute myocardial infarction. Survival and Ventricular Enlargement Investigators. Circulation. 1997;96(3):827–33.
101. Voci P, Bilotta F, Caretta Q, Mercanti C, Marino B. Papillary muscle perfusion pattern. A hypothesis for ischemic papillary muscle dysfunction. Circulation. 1995;91 (6):1714–8.
102. Jain SK, Larsen TR, Darda S, Saba S, David S. A forgotten devil; rupture of mitral valve papillary muscle. Am J Case Rep. 2013;14:38–42.
103. Stout KK, Verrier ED. Acute valvular regurgitation. Circulation. 2009;119(25): 3232–41.
104. Raman S, Pipavath S. Images in clinical medicine. Asymmetric edema of the upper lung due to mitral valvular dysfunction. N Engl J Med. 2009;361(5):e6.
105. Gueret P, Khalife K, Jobic Y, Fillipi E, Isaaz K, Tassan-Mangina S, et al. Echocardiographic assessment of the incidence of mechanical complications during the early phase of myocardial infarction in the reperfusion era: a French multicentre prospective registry. Arch Cardiovasc Dis. 2008;101(1):41–7.
106. Czarnecki A, Thakrar A, Fang T, Lytwyn M, Ahmadie R, Pascoe E, et al. Acute severe

mitral regurgitation: consideration of papillary muscle architecture. Cardiovasc Ultrasound. 2008;6:5.
107. Chevalier P, Burri H, Fahrat F, Cucherat M, Jegaden O, Obadia JF, et al. Perioperative outcome and long-term survival of surgery for acute post-infarction mitral regurgitation. Eur J Cardiothorac Surg. 2004;26(2):330–5.
108. Chen EP, Bittner HB, Davis Jr RD, Van Trigt 3rd P. Milrinone improves pulmonary hemodynamics and right ventricular function in chronic pulmonary hypertension. Ann Thorac Surg. 1997;63(3):814–21.
109. Dekker AL, Reesink KD, van der Veen FH, van Ommen GV, Geskes GG, Soemers AC, et al. Intra-aortic balloon pumping in acute mitral regurgitation reduces aortic impedance and regurgitant fraction. Shock. 2003;19(4):334–8.
110. Arnaiz-Garcia ME, Dalmau-Sorli MJ, Gonzalez-Santos JM, Perez-Losada ME, SastreRincon JA, Hernandez-Hernandez J, et al. Veno-arterial extracorporeal membrane oxygenation as a bridge for enabling surgery in a patient under cardiogenic shock due to acute mitral prosthesis dysfunction. J Saudi Heart Assoc. 2018;30(2):140–2.
111. Staudacher DL, Bode C, Wengenmayer T. Severe mitral regurgitation requiring ECMO therapy treated by interventional valve reconstruction using the Mitra Clip. Catheter Cardiovasc Interv. 2015;85(1): 170–5.
112. Kim TS, Na CY, Baek JH, Kim JH, Oh SS. Preoperative extracorporeal membrane oxygenation for severe ischemic mitral regurgitation — 2 case reports. Korean J Thorac Cardiovasc Surg. 2011;44(3): 236–9.
113. Dixon SR, Henriques JP, Mauri L, Sjauw K, Civitello A, Kar B, et al. A prospective feasibility trial investigating the use of the Impella 2.5 system in patients undergoing high-risk percutaneous coronary intervention (the PROTECT I Trial): initial U.S. experience. JACC Cardiovasc Interv. 2009;2(2):91–6.
114. Wei JY, Hutchins GM, Bulkley BH. Papillary muscle rupture in fatal acute myocardial infarction: a potentially treatable form of cardiogenic shock. Ann Intern Med. 1979;90(2):149–52.
115. Khandaker MH, Espinosa RE, Nishimura RA, Sinak LJ, Hayes SN, Melduni RM, et al. Pericardial disease: diagnosis and management. Mayo Clin Proc. 2010;85(6): 572–93. 1182 T. Montrief et al. / American Journal of Emergency Medicine. 2019;37:1175–1183.
116. Imazio M, Hoit BD. Post-cardiac injury syndromes. An emerging cause of pericardial diseases. Int J Cardiol. 2013;168(2):648–52.
117. Indik JH, Alpert JS. Post-myocardial infarction pericarditis. Curr Treat Options Cardiovasc Med. 2000;2(4):351–6.
118. Welin L, Vedin A, Wilhelmsson C. Characteristics, prevalence, and prognosis of postmyocardial infarction syndrome. Br Heart J 1983;50(2):140–5.
119. Lichstein E, Arsura E, Hollander G, Greengart A, Sanders M. Current incidence of postmyocardial infarction (Dressler's) syndrome. Am J Cardiol. 1982;50(6):1269–71.
120. Dressler W. The post-myocardial-infarction syndrome: a report on forty-four cases. AMA Arch Intern Med. 1959;103(1): 28–42.
121. Lador A, Hasdai D, Mager A, Porter A, Goldenberg I, Shlomo N, et al. Incidence and prognosis of pericarditis after ST-elevation myocardial infarction (from the acute coronary syndrome Israeli survey 2000 to 2013 registry database). Am J Cardiol. 2018;121(6):690–4.
122. Correale E, Maggioni AP, Romano S, Ricciardiello V, Battista R, Salvarola G, et al. Comparison of frequency, diagnostic and prognostic significance of pericardial involvement in acute myocardial infarction treated with and without thrombolytics. Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI). Am J Cardiol. 1993;71(16): 1377–81.
123. Wall TC, Califf RM, Harrelson-Woodlief L, Mark DB, Honan M, Abbotsmith CW, et al. The usefulness of a pericardial friction rub after thrombolytic therapy during acute myocardial infarction in predicting the amount of myocardial damage. The TAMI Study Group. Am J Cardiol. 1990; 66(20):1418–21.
124. Mehrzad R, Spodick DH. Pericardial involvement in diseases of the heart and other contiguous structures: part I: pericardial involvement in infarct pericarditis and pericardial involvement following myocardial infarction. Cardiology. 2012;121(3):164–76.

125. Wessman DE, Stafford CM. The postcardiac injury syndrome: case report and review of the literature. South Med J. 2006;99(3):309–14.
126. Imazio M, Cooper LT. Management of myopericarditis. Expert Rev Cardiovasc Ther. 2013;11(2):193–201.
127. Imazio M, Spodick DH, Brucato A, Trinchero R, Adler Y. Controversial issues in the management of pericardial diseases. Circulation. 2010;121(7):916–28.
128. Oliva PB, Hammill SC, Talano JV. T wave changes are consistent with epicardial involvement in acute myocardial infarction. Observations in patients with a postinfarction pericardial effusion without clinically recognized postinfarction pericarditis. J Am Coll Cardiol. 1994;24(4):1073–7.
129. Oliva PB, Hammill SC, Edwards WD. Electrocardiographic diagnosis of postinfarction regional pericarditis. Ancillary observations regarding the effect of reperfusion on the rapidity and amplitude of T wave inversion after acute myocardial infarction. Circulation. 1993; 88(3):896–904.
130. Figueras J, Juncal A, Carballo J, Cortadellas J, Soler JS. Nature and progression of pericardial effusion in patients with a first myocardial infarction: relationship to age and free wall rupture. Am Heart J 2002; 144(2):251–8
131. Pujari SH, Agasthi P. Left Ventricular Rupture. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [Cited 2023 Jun 9]. Available:http://www.ncbi.nlm.nih.gov/books/NBK559271/
132. Koklu E, Arslan S, Yuksel IO, Bayar N, Yilmaz GM, Kucukseymen S. Management of Left Ventricular Free Wall Rupture Associated with Acute Myocardial Infarction. J Acute Med. 2017;7(1):31–4.
133. Amir O, Smith R, Nishikawa A, Gregoric ID, Smart FW. Left Ventricular Free Wall Rupture in Acute Myocardial Infarction. Tex Heart Inst J. 2005; 32(3):424–6.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/101475