JACC: CASE REPORTS VOL. 2, NO. 10, 2020

MINI-FOCUS ISSUE: SCAI

BEGINNER

CASE REPORT: CLINICAL CASE

Acute Myocardial Infarction and Papillary Muscle Rupture in the COVID-19 Era

Auras R. Atreya, MD, MPH, Kris Kawamoto, MD, Prasanthi Yelavarthy, MD, Mansoor A. Arain, MD, David G. Cohen, MD, Brett L. Wanamaker, MD, Ashraf Abou El Ela, MD, Matthew A. Romano, MD, Paul M. Grossman, MD

ABSTRACT

Mechanical complications of acute myocardial infarction are infrequent in the modern era of primary percutaneous coronary intervention, but they are associated with high mortality rates. Papillary muscle rupture with acute severe mitral regurgitation is one such life-threatening complication that requires early detection and urgent surgical intervention. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:1637–41) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 52-year-old firefighter presented with shortness of breath, chest pressure, and altered mental status. The patient was unaccompanied and, because of his altered mental status, was unable to provide a history of symptom duration. Initial evaluation revealed normal body temperature, severe respiratory distress (respiratory rate of 30 breaths/min, peripheral oxygen saturation of 93% on 15 l/min O_2), blood pressure of 87/41 mm Hg, heart rate of 89 beats/min, and rales at the lung bases bilaterally. A murmur could not be auscultated. An electrocardiogram showed sinus rhythm with ST-segment depression in the precordial leads suggestive of posterior myocardial injury (Figure 1).

LEARNING OBJECTIVES

- To diagnose ruptured papillary muscle in the cardiac catheterization laboratory.
- To understand the pathophysiology of acute MR and understand noninvasive and invasive therapies for stabilization of hemodynamics.

PAST MEDICAL HISTORY

The patient had a history of hyperlipidemia, prediabetes, gout, and well-controlled hypertension.

DIFFERENTIAL DIAGNOSIS

Given the patient's severe respiratory distress, electrocardiographic abnormalities, and his occupation, there was moderate suspicion of coronavirus disease-2019 (COVID-19). The differential diagnosis also included posterior ST-segment elevation myocardial infarction (STEMI) with pulmonary edema.

From the Division of Cardiovascular Medicine and Cardiac Surgery, Frankel Cardiovascular Center, Michigan Medicine, University of Michigan, Ann Arbor, Michigan. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Case Reports* author instructions page.

Manuscript received June 4, 2020; accepted June 26, 2020.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2020.06.037

ABBREVIATIONS AND ACRONYMS

AMI = acute myocardial infarction

COVID-19 = coronavirus disease-2019

IABP = intra-aortic balloon pump

MR = mitral regurgitation

STEMI = ST-segment elevation myocardial infarction

TEE = transesophageal echocardiogram

VA ECMO = venoarterial extra corporeal membrane oxygenation

INVESTIGATIONS

The chest radiogram showed a diffuse interstitial abnormality suggestive of pulmonary edema (Figure 2). The patient was brought on an emergency basis to a negative pressure cardiac catheterization laboratory, and a decision was made to intubate the patient because of his worsening respiratory distress. While he was being intubated, copious pink, frothy fluid emanated into the oropharynx and made endotracheal tube placement extremely difficult; intubation took 10 min. During this time, he progressed to pulseless electrical activity arrest, and cardiopulmonary resuscitation was initiated. As cardiopulmonary resuscitation was being performed, left femoral arterial and venous access was obtained for venoarterial extracorporeal membrane oxygenation (VA ECMO), and mechanical circulatory support was initiated. Shortly thereafter, his heart rhythm transitioned to ventricular fibrillation. Sinus rhythm was restored following 200-J external defibrillation.

Once he was stabilized on VA ECMO, diagnostic coronary angiography revealed right-dominant circulation with thrombotic occlusion of the middle left circumflex artery (Figure 3). Out of concern for acute severe ischemic mitral regurgitation (MR), a biplane left ventriculogram was performed that showed a normal ejection fraction with inferolateral hypokinesis (Video 1). Severe MR was noted, with concern for a ruptured papillary muscle. Transesophageal echocardiogram (TEE) confirmed the diagnosis of severe MR with flail medial anterior and posterior mitral leaflets, consistent with a posterior medial papillary muscle rupture (Video 2). TEE also revealed insignificant left ventricular forward flow into the aorta with only intermittent aortic valve opening. A high-sensitivity troponin assay obtained in the emergency department was 386 pg/ml (reference 0 to 19 pg/ml), and his creatine phosphokinase level was 181 IU/l (reference 38 to 240 IU/l). The peak high-sensitivity troponin level was 2,151 pg/ml, and the creatine phosphokinase level was 4,122 IU/l on day 5 after presentation. A reverse transcription polymerase chain reaction assay for severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) had a negative result.

MANAGEMENT

Right-sided heart catheterization revealed pulmonary capillary wedge pressure of 29 mm Hg and mean pulmonary artery pressure of 30 mm Hg on VA ECMO. Intra-aortic balloon pump (IABP) counterpulsation was initiated to help reduce afterload and improve forward flow in the setting of severe MR and VA ECMO. There was significant augmentation of diastolic blood pressure after IABP implantation. In the intensive care unit, therapeutic hypothermia was

FIGURE 1 Electrocardiogram Shows Significant ST-Segment Depression in Precordial Leads, Suggestive of Posterior Injury

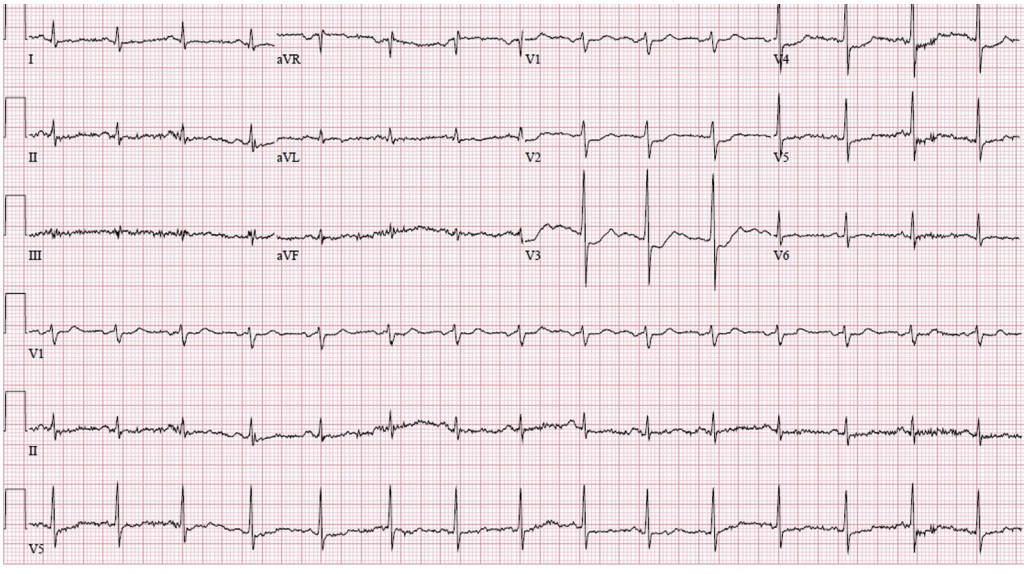


FIGURE 2 Chest Radiograph With Diffuse Interstitial Abnormality, Consistent With Pulmonary Edema

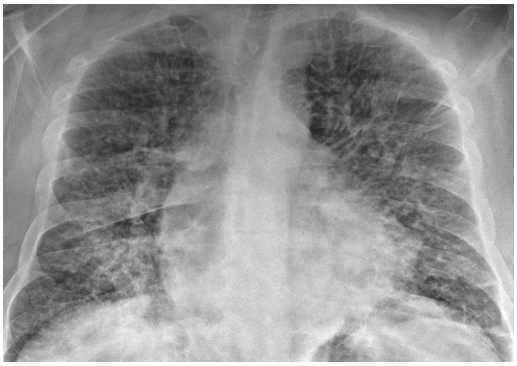
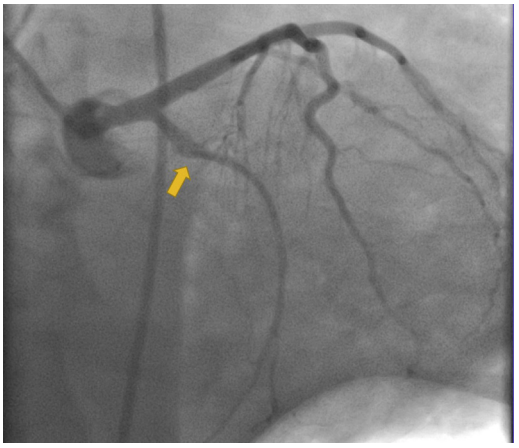


FIGURE 3 Coronary Angiogram Showing Thrombotic Occlusion of the Left Circumflex Artery After the Origin of the Obtuse Marginal Branch



The **arrow** shows the occlusion.

achieved through the ECMO circuit. He underwent mitral valve replacement with a No. 29 Edwards Magna mitral tissue valve (Edwards Lifesciences, Irvine, California) 2 days after his initial presentation (**Figure 4**, Video 3).

DISCUSSION

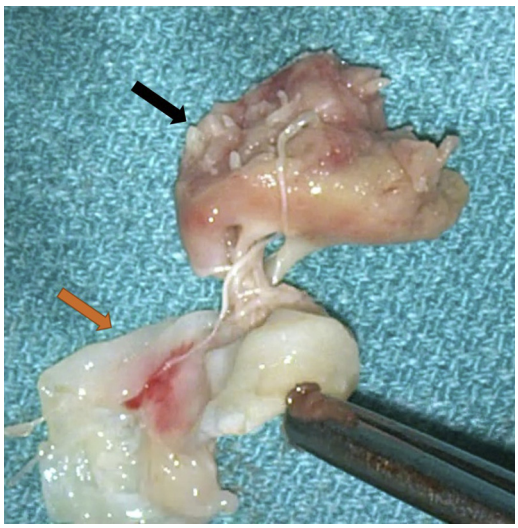
Mechanical complications of acute myocardial infarction (AMI) include left ventricular free wall rupture, interventricular septal rupture, and acute MR secondary to papillary muscle dysfunction or rupture. The incidence of mechanical complications in the pre-fibrinolytic era was as high as 6% and has significantly decreased to <1% in the reperfusion era (1). A recent analysis of nearly 4 million patients hospitalized with STEMI between 2003 and 2015 reported an incidence of 0.27% for all mechanical complications: 0.21% had interventricular septal rupture, 0.05% had papillary muscle rupture, and 0.01% had free wall rupture (2).

Papillary muscle rupture usually occurs 2 to 7 days after AMI and typically is a complication of inferior STEMI (3). The posteromedial papillary muscle is more likely to be supplied by a single coronary artery (either branches of the right posterior descending or right posterolateral artery or the left coronary obtuse marginal arteries) compared with the anterolateral papillary muscle, which receives a dual blood supply from the left anterior descending artery and the left circumflex artery (4). It is hypothesized that papillary muscle rupture usually occurs in the setting of small infarcts related to single-vessel coronary disease because limited myocardial injury allows for preserved left ventricular function and greater shearing force at the site of potential rupture (3).

Acute severe MR secondary to papillary muscle rupture and flail mitral valve leaflet manifests with rapid pulmonary edema and often is complicated by cardiogenic shock. Because the healthy left atrium is noncompliant, these patients rapidly experience elevated left atrial pressures with predominant backward flow of blood into the pulmonary veins with pulmonary edema and hypoxemia (5). Simultaneously, decreased forward flow into the systemic circulation results in cardiogenic shock and a vasoconstrictor response. Given the rapid equalization of left ventricular and left atrial pressures in patients with acute severe MR, a systolic murmur on auscultation is classically absent. The diagnosis requires a high degree of suspicion and prompt evaluation with echocardiography or contrast left ventriculography.

Emergency medical and surgical treatment is required when papillary muscle rupture occurs. Mechanical ventilation for management of pulmonary edema and afterload reduction with vasodilator therapy and IABP counterpulsation are used for temporary hemodynamic stabilization until surgical intervention is performed. Afterload reduction with an IABP improves forward flow and has been shown to increase blood pressure and cardiac output and reduce pulmonary capillary wedge pressure (5). Another option for hemodynamic support is the TandemHeart (LivaNova, London, United Kingdom) left ventricular assist device that serves to decompress the left atrium and left ventricle and improve

FIGURE 4 Surgical Specimen of Ruptured Papillary Muscle



The photograph shows ruptured posteromedial papillary muscle **(black arrow)** and mitral valve chordal apparatus **(orange arrow).**

cardiac output by returning blood to the systemic circulation (6). The Impella left ventricular assist device (Abiomed, Danvers, Massachusetts) is contraindicated in patients with a ruptured papillary muscle because of hyperdynamic left ventricular contraction and catheter interaction with the ruptured chordal apparatus. In our patient, the TEE obtained during VA ECMO and before IABP catheter insertion revealed nearly absent forward flow through the aortic valve, with the entire stroke volume directed toward the left atrium and pulmonary veins. These findings are consistent with increased afterload conditions created by the VA ECMO circuit in addition to severe MR. Following IABP implantation, there was considerable augmentation of blood pressure, a finding indicating adequate flow from the left ventricle into the aorta.

AMI complicated by acute severe MR portends a very poor prognosis with in-hospital mortality approaching 70% when surgery is not performed (7). The timing of surgery remains controversial. Studies have shown lower operative mortality when surgery is delayed for more than 3 months after AMI (3). However, this finding reflects survival bias. Earlier surgical intervention increases operative mortality, but it improves long-term mortality. Patients who survive the perioperative period after mitral valve surgery have a good prognosis, with heart failure-free survival rates nearly identical to those in patients presenting with AMI who do not have a mechanical complication (8).

With the ongoing COVID-19 pandemic, there have been reported delays in access to care (9). Delays and lack of prompt pharmacologic or invasive reperfusion therapies are bound to increase mechanical complications of AMI, so emergency department physicians and cardiologists need to be aware of atypical presentations of AMI and possible mechanical complications. As demonstrated in this case, although the differential diagnosis of COVID-19 infection could have delayed his cardiovascular diagnosis, a decision to transport the patient to the cardiac catheterization laboratory was critical to achieve resuscitation from cardiac arrest and facilitate investigations to diagnose papillary muscle rupture rapidly. Additionally, hospitals need to be ready to respond to such emergencies with adequate personnel, personal protective equipment, and appropriate facilities (10).

FOLLOW-UP

Although an initial computed tomographic scan of the head following cardiac catheterization laboratory resuscitation did not reveal ischemic brain injury, myoclonic seizures developed in this patient, and subsequent computed tomographic scans revealed loss of gray-white differentiation consistent with severe global hypoxic injury. Given the lack of improvement in clinical status, care was withdrawn by the patient's family, and the patient died on hospital day 9.

CONCLUSIONS

This case highlights the importance of early recognition of mechanical complications associated with STEMI and prompt initiation of resuscitative and therapeutic measures, within the confines of modern-day COVID-19-driven institutional protocols.

ADDRESS FOR CORRESPONDENCE: Dr. Auras R. Atreya, Frankel Cardiovascular Center, University of Michigan, 1500 East Medical Center Drive Ann Arbor, Michigan 48109. E-mail: auras@med.umich.edu. Twitter: @AurasAtreya.

REFERENCES

1. Figueras J, Alcalde O, Barrabes JA, 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;118:2783–9.

2. Elbadawi A, Elgendy IY, Mahmoud K, et al. Temporal trends and outcomes of mechanical complications in patients with acute myocardial infarction. J Am Coll Cardiol Intv 2019;12:1825–36.

3. Nishimura RA, Schaff HV, Shub C, et al. Papillary muscle rupture complicating acute myocardial infarction: analysis of 17 patients. Am J Cardiol 1983;51:373–7.

4. James TN. Anatomy of the coronary arteries in health and disease. Circulation 1965;32:1020–33.

5. Stout KK, Verrier ED. Acute valvular regurgitation. Circulation 2009;119:3232–41.

6. DiVita M, Visveswaran GK, Makam K, et al. Emergent TandemHeart-ECMO for acute severe mitral regurgitation with cardiogenic shock and hypoxaemia: a case series. Eur Heart J Case Rep 2020;4:1–6.

7. Lavie CJ, Gersh BJ. Mechanical and electrical complications of acute myocardial infarction. Mayo Clin Proc 1990;65:709–30.

8. Russo A, Suri RM, Grigioni F, et al. Clinical outcome after surgical correction of mitral regurgitation due to papillary muscle rupture. Circulation 2008;118:1528–34.

9. Rodriguez-Leor O, Cid-Alvarez B. STEMI care during COVID-19: losing sight of the forest for the trees. J Am Coll Cardiol Case Rep 2020;2:1625–7.

10. Truesdell M, Guttman P, Clarke B, et al. Conversion of positive pressure cardiac catheterization and electrophysiology laboratories to a novel 2-zone negative pressure system during COVID-19 pandemic. J Cardiovasc Electrophysiol 2020 May 23 [E-pub ahead of print].

KEY WORDS acute coronary syndrome, COVID-19, mitral regurgitation, papillary muscle rupture, percutaneous coronary intervention, ST-segment elevation myocardial infarction, transesophageal echocardiography

APPENDIX For supplemental videos, please see the online version of this paper.