

Flail Mitral Leaflet Post-Myocardial Infarction - A Lethal Mechanical Complication

Dinakaran Umashankar *, Christian Toquica Gahona, Quang Dat Ha, Roopeessh Vempati, Muhammad Sanusi, Utheja Dasari, Michele DeGregorio

Trinity Health Oakland Hospital, Pontiac, Michigan

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***Corresponding author:** Dinakaran Umashankar, Trinity Health Oakland Hospital, Pontiac, Michigan

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ABSTRACT/BACKGROUND

Flail mitral leaflet (FML) caused by papillary muscle rupture is an uncommon mechanical complication following myocardial infarction (MI), with an incidence of less than 1%.^[1] It typically occurs 2-7 days post-infarction, and its clinical ramifications can range from mild symptoms to acute heart failure or cardiogenic shock.^[2,3] The mortality rate associated with this complication is approximately 71%^[4] in patients who do not undergo valvular surgery, highlighting the critical need for immediate intervention. Although infrequent, FML remains a life-threatening mechanical complication of MI, with its incidence decreasing in recent years due to advancements in reperfusion strategies. We present a case that underscores the importance of prompt recognition and management to optimize patient outcomes.

Keywords: Flail mitral leaflet; Muscle; Myocardial infarction

CASE PRESENTATION

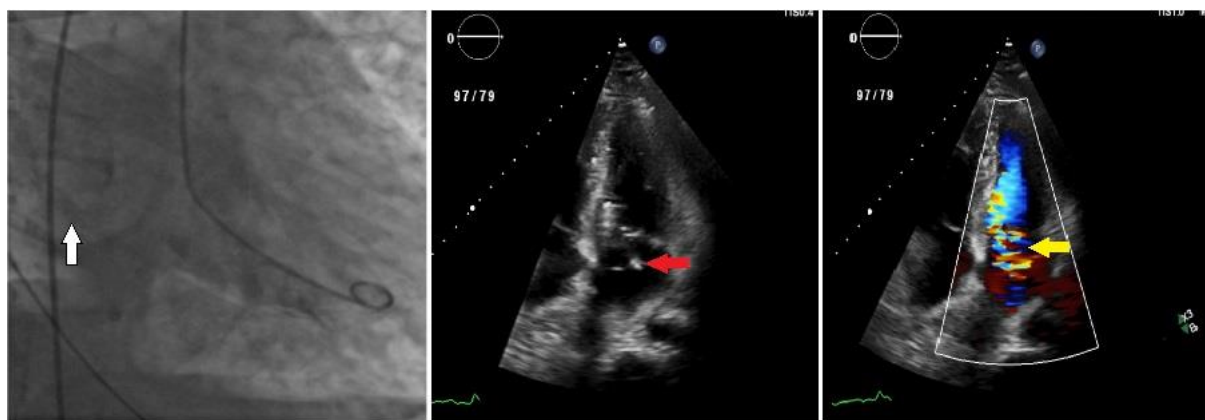
An 80-year-old male with a history of renal cell carcinoma and metastatic bone disease is undergoing chemotherapy with Belzutifan. He also has paroxysmal atrial fibrillation, chronic provoked deep vein thrombosis in the right lower extremity [2022] on anticoagulation with apixaban, hypertension, hyperlipidemia, and chronic obstructive pulmonary disease. The patient presented with easy fatigability for several days since starting a new chemotherapy regimen two weeks ago with belzutifan and shortness of breath, classified as NYHA Class IV for one day. Upon presentation, vital signs indicated a pulse rate of 108/min, blood pressure of 79/50 mmHg, respiratory rate of 27/min, and oxygen saturation of 93% while on 6L oxygen therapy. The patient was initiated on norepinephrine support due to low blood pressure after no improvement from a 500 cc 0.9% normal saline fluid bolus. The physical examination revealed elevated jugular venous distension (JVD) and bilateral crackles upon lung auscultation. The ECG showed ST elevation in inferior leads, accompanied by reciprocal changes in the precordial leads. High-sensitivity troponin-I was noted to be elevated at 1723 ng/L,

while B-type natriuretic peptide was elevated at 641 pg/mL. The chest x-ray suggested pulmonary edema with perihilar interstitial opacities and cephalization of blood vessels.

The patient received a loading dose of aspirin and ticagrelor. Since he was already on apixaban, heparin was deferred, and the emergency STEMI protocol was activated. The patient experienced cardiopulmonary arrest, with the rhythm showing pulseless electrical activity. Cardiopulmonary resuscitation was initiated, and a return of spontaneous circulation was achieved after approximately 11 minutes. The patient was then transferred to the cardiac catheterization lab.

A coronary angiogram revealed a long right coronary artery supplying the septal muscle, showing 99% stenosis, and a short left circumflex artery; a drug-eluting stent was successfully placed. A left ventricular assist device was implanted for mechanical circulatory support. Post-catheterization echocardiography revealed an LVEF of 56%, with a flail anterolateral mitral valve leaflet and acute moderate to severe mitral regurgitation characterized by an eccentric jet. The inferior wall appeared akinetic, while the anterior wall seemed hyperdynamic.

An emergency cardiothoracic surgery consultation was requested, but mitral valve surgery was postponed due to the patient's terminal illness and his hemodynamic instability while receiving triple vasopressor support with norepinephrine, epinephrine, and phenylephrine. The patient's condition continued to worsen, and he was placed on vasopressin support in addition to the three other vasopressors; however, his state further declined, leading to his eventual cardiac arrest.



Panel A:
Left ventriculogram showing grade II-III angiographic mitral regurgitation (white arrow).

Panel B:
Apical four chamber view showing a flail anterior mitral valve leaflet (Red arrow)

Panel C:
Apical four chamber view color Doppler with evidence of a large posteriorly directed mitral regurgitation jet (yellow arrow).

DISCUSSION

The integrity of the mitral valve relies on a delicate interplay among the valve leaflets, chordae tendineae, and papillary muscles. Ischemia or infarction of the papillary muscles—usually affecting the posteromedial papillary muscle due to its single blood supply from the posterior descending artery—can lead to severe mitral regurgitation (MR), with clinical implications ranging from mild symptoms to acute heart failure or cardiogenic shock.^[2,3]

Flail mitral leaflets (FML) are characterized by the prolapse of one or both mitral valve leaflets into the left atrium during systole, resulting from disruption of the chordae tendineae or papillary muscles. If not promptly addressed, this condition can significantly exacerbate the hemodynamic burden by causing sudden, severe MR, leading to pulmonary edema, decreased cardiac output, and high mortality.^[3,5]

FML following myocardial infarction (MI) is a rare but catastrophic event, typically arising from ischemic injury to the papillary muscles. Transthoracic or transesophageal echocardiography serves as the cornerstone of diagnosis in suspected cases of mitral valve prolapse (MVP). Echocardiography not only confirms the presence of a flail leaflet but also quantifies the severity of MR, identifies associated complications, and informs the urgency of intervention.^[6] Prompt surgical management, whether through mitral valve repair or replacement, is crucial for improving outcomes in patients with mitral regurgitation (MR) or mitral valve disease (MVD). Conservative medical management of heart failure and cardiogenic shock alone might not suffice due to the severity of MR and its effects on cardiac function,^[5,6] as the mortality rate is high—around 71%^[3]—in patients not undergoing valvular surgery, with a perioperative mortality rate (within 30 days) of 27.3%.^[7]

The decline in the incidence of mechanical complications like FML has been linked to advancements in the timely revascularization of acute MI through percutaneous coronary interventions (PCI) and thrombolysis. Although the occurrence of papillary muscle rupture causing MR has decreased in the reperfusion era (range, 0.05%–0.26%),^[8] late presentation or insufficient reperfusion therapy continues to pose a risk for these complications.^[9] This case underscores the vital role of advanced imaging and the importance of prompt intervention in achieving optimal patient outcomes. It also highlights the significance of the medical community's expertise in the early identification of mechanical complications in patients who present with sudden hemodynamic instability following myocardial infarction (MI).

CONCLUSION

This case underscores the critical nature of flail mitral leaflets as a life-threatening complication of myocardial infarction. The diagnosis necessitates a high index of suspicion, especially in patients experiencing acute dyspnea, pulmonary edema, or cardiogenic shock following MI. Transthoracic and transesophageal echocardiography remain essential tools for the prompt identification and evaluation of mitral regurgitation severity. Early surgical intervention, combined with optimal medical therapy, reduces mortality and improves patient outcomes.

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