# Cardiogenic Shock Post-percutaneous Coronary Intervention for Myocardial Infarction

A 78-year-old Chinese man with history of ischaemic heart disease, status post coronary artery bypass grafting, as well as sick sinus syndrome with pacemaker inserted, presented to the emergency department for constant central squeezing chest pain associated with diaphoresis. He has known ischaemic heart disease diagnosed in 1998. Coronary angiogram then showed triple vessel disease (stenoses of the left main coronary artery, 25%; proximal left anterior descending artery, 100%; proximal left circumflex artery, 25%, first obtuse marginal artery, 90%; second obtuse marginal artery, 70%; third obtuse marginal artery, 100%; proximal right coronary artery, 25%; mid right coronary artery, 75%; right posterior descending artery, 10%; right posterolateral artery, 90%). He subsequently underwent coronary artery grafting in 1998, with left internal mammary artery to the left anterior descending artery, as well as saphenous venous grafts to the first obtuse marginal branch and the right posterior descending artery. The left ventricular ejection fraction post-surgery was quantitated to be 63%.

He presented with central chest pain of 1-day duration, starting at 1900 hours on the day of admission. On admission to hospital, electrocardiogram showed T wave inversion in inferior leads (Fig. 1A). Cardiac enzymes showed an upward trend consistent with myocardial necrosis. Creatine kinase myocardial b (CKMB) fraction rose from 41.4 ug/L to 111.0 ug/L over 15 hours, while troponin I rose from undetectable levels on admission to >80.000 ug/L over the same period. He was treated as a non-ST elevation myocardial infarction with a thrombolysis in myocardial infarction (TIMI) risk score of 5.

On the second day of admission, a coronary angiogram was performed in view of ongoing chest pain. Angiogram done 14 hours from onset of chest pain, showed native triple vessel disease and patent left internal mammary and saphenous vein grafts. The occluded right posterolateral artery was deemed the culprit lesion (Fig. 1B) and balloon angioplasty was performed with successful revascularisation, as well as symptom resolution (Fig. 1C).

Eight hours after procedure, the patient developed acute pulmonary oedema and severe hypotension. He was in cardiogenic shock. A new holosystolic murmur was also heard loudest over the apex. However, there was no thrill palpable in the precordium.

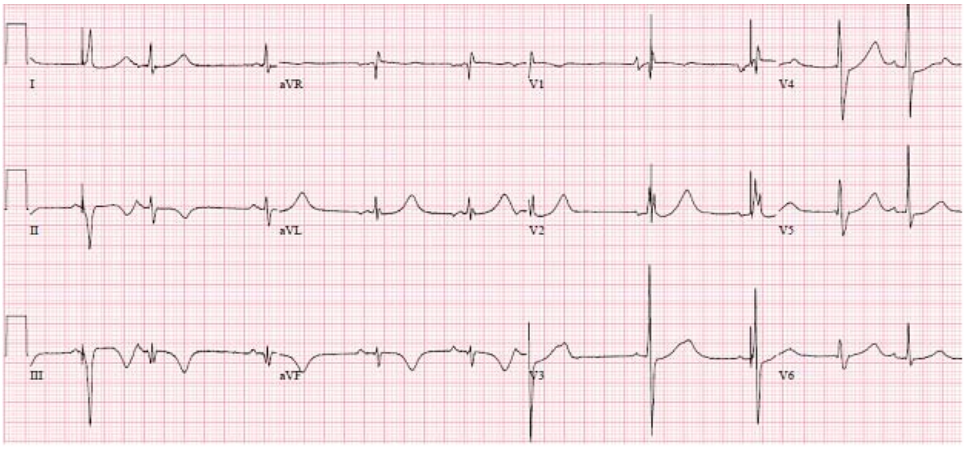


Fig. 1(A). Electrocardiogram shows new T wave inversions in the inferior leads.

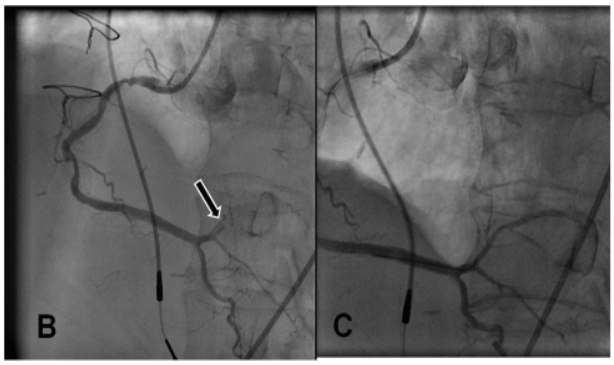


Fig. 1(B). Coronary angiogram shows occlusion of the right posterolateral artery which is the culprit vessel. (C). Post balloon angioplasty coronary angiogram shows successful revascularisation of right posterolateral artery.

Taking into account the temporal sequence of events and the evolution of physical signs, what is the most likely cause of acute heart failure and cardiogenic shock in this patient?

A. Ventricular septal rupture with acute intra-cardiac shunt.

B. Ventricular free wall rupture.

C. Acute papillary rupture resulting in severe mitral regurgitation.

D. Re-occlusion of the culprit vessel.

Echocardiography showed partially ruptured posteromedial papillary muscle (arrow, Fig. 1D) with posteriorly-directed, eccentric mitral regurgitation (MR) (Fig. 1E). There was a linear echo-lucency demonstrating transection of the muscular body (arrow, Fig. 1F). This resulted in flail anterior mitral valve leaflet (arrow, Fig. 1G). In view of cardiogenic shock, intra-aortic balloon pump was inserted as a bridging therapy to definitive surgery. However, due to high surgical risk, the patient's family declined surgical intervention. The patient was managed conservatively and demised 15 hours later despite maximal inotropic support.

## Discussion

Development of a new systolic murmur post myocardial infarction is an important clinical sign with significant bearings on the subsequent management of the patient. Considerations include that of ventricular septum rupture or acute mitral regurgitation. In this patient, echocardiogram showed partially ruptured posteromedial papillary muscle with a flail anterior leaflet of the mitral valve. This resulted in a posteriorly directed, eccentric mitral regurgitation.

Papillary rupture resulting in acute mitral regurgitation after myocardial infarction is an uncommon but largely fatal complication. In a study of mechanical complications of myocardial infarction, 697 consecutive patients admitted for ST elevation myocardial infarction (STEMI)were studied. Twenty-seven of the patients (3.9%) developed mechanical complications, namely ventricular free wall rupture (n = 20), ventricular septum rupture (n = 5), papillary muscle rupture (n = 1) or combination of the above (n = 1). It was found that in the setting of mechanical complications post-myocardial infarction, mortality increased from 10.5% to 55.6%.[1] In an analysis of 22 necropsy patients with papillary rupture post-myocardial infarction, the posteromedial muscle is involved 3 times more often than the anterolateral muscle.[2] This is due to the posteromedial muscle having a more tenuous blood supply from the right coronary artery compared with the anterolateral papillary muscle, which often receives dual blood supply from the left anterior descending artery as well as the circumflex artery.

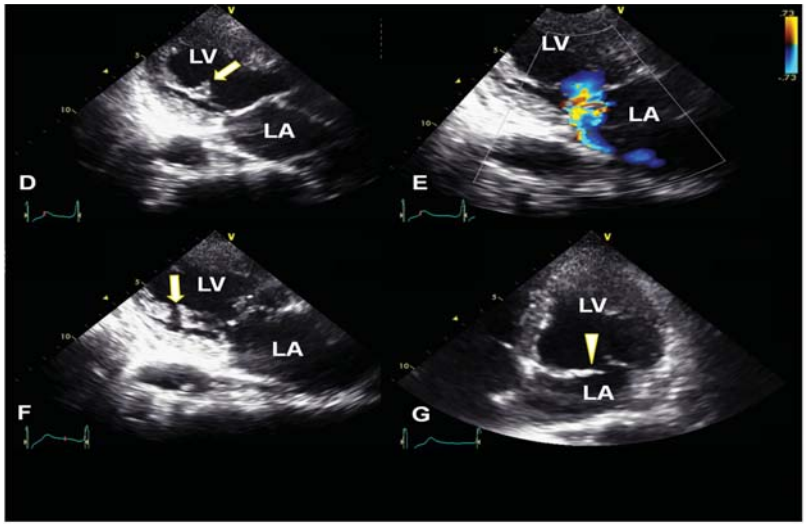


Fig. 1(D). Echocardiogram (parasternal long axis view) shows partially ruptured posteromedial papillary muscle. (LA: left atrium; LV: left ventricle). (E). Echocardiogram (apical 2-chamber view) shows posteriorly directed eccentric severe mitral regurgitant jet. (F) Echocardiogram (apical 2-chamber view) shows linear echo-lucency demonstrating transection of the muscular body of the posteromedial papillary muscle. (G) Echocardiogram (modified apical 4-chamber view) shows flail anterior mitral valve leaflet.

Answer: (C)

Urgent transthoracic echocardiogram was performed in our patient. Often this portable and readily available imaging modality in the coronary care unit is useful in rapidly delineating the mechanical cause of cardiogenic shock. Indeed in this case, a new onset posteriorly directed eccentric mitral regurgitation jet was identified. This may be secondary to a flail or prolapsed anterior mitral leaflet or a restricted posterior leaflet. The transthoracic echocardiography was able to identify clearly a flail anterior leaflet. Although more commonly, rupture of the posterolateral papillary muscle would result in a flail posterior mitral leaflet with an anteriorly directed regurgitant jet, this was not the case here. In our patient, rupture of the posteromedial papillary muscle resulted in a more unusual flail anterior leaflet. This is because both papillary muscles have chordae attached to both leaflets of the mitral valve.[3,4] As such, leaflet involvement does not necessarily indicate pathology in the corresponding papillary muscle.

Intra-aortic balloon counter pulsation in the presence of acute mitral regurgitation has been shown in animal models to increase cardiac output, increase cerebral perfusion and decrease aortic impedance. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines for ST elevation myocardial infarction put intra-aortic balloon counter pulsation as a class I recommendation for patients with (i) Hypotension unresponsive to other interventions; (ii) Low cardiac output state; (iii) Cardiogenic shock not rapidly reversed with pharmacological therapy, while awaiting revascularisation and (iv) Recurrent ischaemic chest pain with signs of haemodynamic instability, poor LV function, or large areas of myocardium at risk while awaiting revascularisation. However, in the latest IABP-SHOCK II trial on intra-aortic balloon counter pulsation in post-myocardial infarction cardiogenic shock, there was no significant benefit of intra-aortic balloon counter pulsation when compared against optimal medical therapy for patients whom early revascularisation has been planned.[5] Mechanical complications of myocardial infarction resulting in cardiogenic shock were not specifically studied.

Lastly, both the cardiac enzymes, CKMB/ Troponin I levels in our patient were very high and out of proportion to infarct territory, as documented on ECG and from the culprit vessel on the coronary angiogram done emergently. This could lead us to suspect myocardial or papillary rupture contributing to the cardiac enzyme elevation, instead of solely infarct-related artery myocardial necrosis. This was consistent with a study of 19 patients who suffered myocardial rupture post infarction and underwent surgical repair in an Israeli hospital.[6] In these 19 patients, there was an extreme elevation of sensitive markers, including cardiac enzymes and coagulation markers.

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