A Sealed Myocardial Rupture Complicating Silent Myocardial Infarction

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Abstract

A case is presented of a 78-years-old patient with known coronary artery disease who was admitted with intermittent vomiting and extreme weakness lasting for few hours and an ECG tracing of an inferior wall ST-segment elevation MI. An echocardiogram revealed an elongated thrombotic mass occupying the entire space between the two pericardial layers. His clinical hemodynamic status was rapidly improved with fluid resuscitation. The patient underwent an urgent coronary angiography and referred for surgery where the thrombotic pericardial content was removed and the rupture myocardial site was successfully sealed with a pericardial patch.

Keywords: Coronary artery disease; Cardiac tamponade; Cardiogenic shock

Introduction

Myocardial rupture is usually the result of thrombotic occlusion of an epicardial coronary artery. Prognosis is poor unless urgent surgery is undertaken immediately or by chance, a pericardial patch forms a closed envelope sealing the rupture site and avoiding cardiac tamponade. We report on a 78-years-old patient admitted with an inferior wall ST-segment elevation MI. An echocardiogram revealed a thrombotic mass occupying the entire space between the two pericardial layers. The patient was referred for an urgent surgery where the pericardial content was removed and the rupture myocardial site was successfully sealed with a pericardial patch.

Case Presentation

A 78-years-old male, with a history of remote Percutaneous Coronary Intervention (PCI) and severe Parkinsonism, was presented to the emergency department due to intermittent vomiting of few hours’ duration and extreme weakness. No prior cardiac symptoms of chest pain or dyspnea were reported. On admission he was found cachectic, hypoxic, conscious and hypotensive. When seen for the first time, his blood pressure was 75/50 mmHg and the pulse rate was 60 beats per minute. Physical examination revealed increased jugular pulse and cold extremities. An ECG tracing showed a lactate level of 90 mg/dl accompanied by metabolic acidosis. This was compatible with an inferior ST-Segment Elevation Myocardial Infarction (STEMI) showing prominent ST-segment displacements of 6 mm to 7 mm involving the inferior and lateral leads. Laboratory workup showed a lactate level of 90 mg/dl accompanied by metabolic acidosis. This was related to a profound hypo-perfusion state. Cardiac biomarkers including troponin-T levels were indicative of acute myocardial infarction. He was treated with fluid resuscitation with a good hemodynamic response. At this stage, an urgent transthoracic echocardiography was performed, demonstrating severe LV dysfunction, with a large mural thrombus extending from the apical segment towards the anterior and infero-apical segments and protruding into the left-ventricular cavity (Figure 1 and 2). Doppler-flow was consistent with typical respiratory variability across the mitral valve suggestive of cardiac tamponade. Sub-costal view revealed a large elongated thrombus between the two pericardial layers extending from apex to the base, obliterating the entire right ventricular cavity and compressing the right atrium. The thrombotic mass reached 4 cm thickness. The possibility of a sealed myocardial rupture was promptly raised and with a presumed diagnosis of a myocardial wall rupture secondary to myocardial infarction, the patient was first stabilized in the emergency room by continuous fluids administration and then referred for an urgent coronary angiography which demonstrated a diffusely diseased right coronary artery with grade 2 TIMI flow. The patient was then transferred to the operating room, where large thrombi were removed from the pericardial space. An inferior free wall rupture was sealed with a pericardial patch. A month later, while he is hemodynamically stable but mechanically ventilated through a tracheostomy due to severe deconditioning state, the patient was transferred to a respiratory rehabilitation institute.
Discussion

Despite of the increasing clinical awareness, the potentially lethal consequences of acute mechanical complications are worrisome. The development of complications such as myocardial rupture or acute VSD following MI has decreased significantly to less than 1% since the routine implementation of primary PCI for the treatment of acute STEMI [1,2]. Even though, once myocardial rupture occurs, an eminent massive hemopericardium is expected, rapidly leading to tamponade and death in most of the cases.

Here we report an unusual case of left ventricular free wall rupture in a 78 years-old male. The patient underwent an urgent off-pump operation with relatively favorable outcome. Generally, surgical repair is of proven efficacy for the treatment of myocardial rupture survivors. This harmful generally lethal complication occurs in approximately 0.5% of MI patients [1,2]. Successful surgical management varied widely and an operative mortality ranging from 12% to 30% has been reported in small selected groups of patients [3,4]. Long-term survival may never be published in large scale. Despite high operative mortality and in view of the low survival rate without surgical repair, the lack of an effective medical alternative makes surgical repair the mainstay of current management for these patients. Prompt recognition of myocardial rupture is a key to salvaging this life threatening complication and to improve survival. In this regard, a further decline in the prevalence of rupture events may be expected to emerge when prompt emergent mechanical revascularization coupled with an aggressive therapeutic strategies are adopted among patients presenting with acute coronary syndromes.

References