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PERICARDIUM: THE FORGOTTEN SPACE DURING ACUTE MYOCARDIAL INFARCTION

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□ **Abstract—Background: Acute pericardial pathologies, such as pericardial effusion, pericarditis, and cardiac tamponade, have been reported rarely in patients presenting as ST-elevation myocardial infarction (STEMI). We present a series of 3 patients with STEMI, where an undiagnosed pericardial effusion led to pericardial tamponade and subsequent cardiocirculatory collapse. Case Reports: This is a case series of 3 patients, all women, aged 72, 64, and 54 years who presented to the emergency department with chest pain or syncope and were found to have STEMI with hemodynamic instability. They were taken to the catheterization laboratory for urgent coronary revascularization requiring mechanical circulatory support (intra-aortic balloon pump or impella). During catheterization, all 3 patients were diagnosed with large pericardial effusion using hemodynamic parameters and bedside transesophageal echocardiogram. Commonly ignored, pericardial tamponade and acute large pericardial effusion can be the cause of cardiocirculatory collapse. Two of the 3 patients survived with aggressive interventions requiring pericardial drains, long-term mechanical circulatory support, and effective postoperative rehabilitation. Why Should an Emergency Physician Be Aware of This?: It is important for treating clinicians, including emergency physicians, intensivist, and cardiologist, to consider the differential of a cardiac tamponade due to a pericardial effusion as a potential cause for hypotension in patients with an acute STEMI.**

□ **Keywords—pericardium; myocardial infarction; pericardial tamponade**

INTRODUCTION

Acute pericardial pathologies, such as pericardial effusion (PE) and pericarditis, can complicate transmural myocardial infarction (MI). Current practices, with early revascularization, have made this complication rare. Moreover, an extreme form of pericardial disease, pericardial tamponade, post-MI, is reported even more rarely (0.12%) (1). We present a series of 3 patients who presented to the emergency department (ED) with ST segment elevation myocardial infarction (STEMI) and an undiagnosed PE led to pericardial tamponade and subsequent cardiocirculatory collapse.

Case 1

A 72-year-old woman with no known medical history had a syncopal event in the field. She was found by emergency medical services to be diaphoretic, having agonal breathing requiring emergent intubation. In the ED, she was hypotensive to 70/55 mm Hg. Electrocardiogram (ECG) showed ST elevation in lead I and aVL with reciprocal ST depression in II, III, and aVF (Figure 1). The patient was taken emergently to the cardiac catheterization laboratory with the diagnosis of an anterior wall MI complicated with cardiogenic shock. In the ED, the patient was started on i.v. norepinephrine and was loaded with i.v. heparin.

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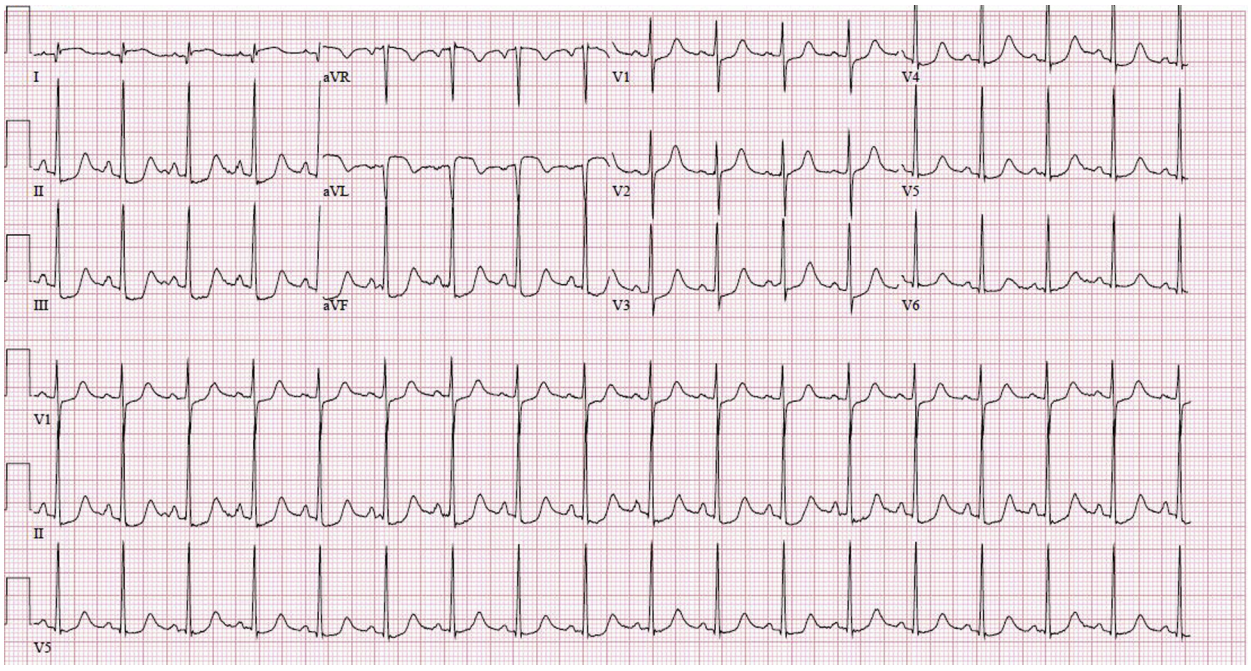

Figure 1. Electrocardiogram tracing showing ST elevation in lead I, aVL with reciprocal ST depression in II, III, and aVF.

Coronary angiography revealed high-degree (70%) tubular stenosis of the proximal left anterior descending (LAD) with thrombolysis in myocardial infarction (TIMI) 3 flow into the distal vessel. Due to persistent hypotension, an intraortic balloon pump (IABP) was placed from the femoral access. Revascularization of the proximal LAD with excellent final TIMI 3 flow was performed with placement of drug-eluting stent to LAD. The procedure was performed with adjunctive i.v. abciximab. Left ventriculography post revascularization revealed a hyperdynamic, underfilled left ventricle. Right heart catheterization after revascularization revealed an unexpected high right atrial pressure (21 mm Hg) with equalization of right ventricular end diastolic pressure (RVEDP) and pulmonary capillary wedge pressure (PCWP). A pulmonary artery saturation of 36% with a systemic saturation of 96% was drawn. An emergent transesophageal echocardiogram (TEE) revealed the presence of a large circumferential PE (>2 cm) with thrombus, and restriction of right atrial and right ventricular filling (Figure 2). In light of these findings, an emergent pericardiocentesis was performed, and approximately 2 L bloody fluid was drained over almost 2 h. Abciximab was stopped. She was resuscitated with 5 U packed red blood cell transfusions and 8 U platelets. Considering the presence of irregular, long, high-degree LAD stenosis, post-MI pericarditis further exacerbated by abciximab was highly suspected. She was admitted to the coronary care unit with the pericardial drain and IABP. The balloon pump was discontinued the following day. Her hospital course was complicated by atrial fibrillation, bilateral diffuse embolic stroke, and acute kidney injury. She received aggressive rehabilitation therapy, returned to her baseline activity with no residual neurologic deficits, and continues to follow-up in the cardiology clinic.

Case 2

A 64-year-old woman with no significant medical history presented to the ED complaining of chest and back pain for 2 days. She was noted to be hypotensive to 90/50 mm Hg. Initial ECG revealed ST-elevation in II, III, aVF, and V4–V6 with reciprocal ST depression in V1 and V2 (Figure 3). An STEMI alert was initiated. Due to complaints of back pain, an acute aortic syndrome was excluded based on computed tomographic (CT) imaging of the chest, abdomen, and pelvis. Emergent coronary angiography revealed 80% diffuse stenosis throughout the left main extending to the proximal, mid, and distal LAD, with 80% disease in proximal left circumflex and tandem 85–95% stenosis in right coronary artery (RCA) with a 70% eccentric stenosis at the proximal right posterior descending artery. All of these vessels had TIMI 3 flow, except for a 100% occlusion of a moderate-sized posterior left ventricular (PLV) branch. A hand injection left ventriculogram in the right anterior oblique (RAO) projection showed normal ejection fraction with hypokinesis of the basal inferior wall. Considering the presence of multivessel disease, emergent cardiothoracic surgery consult was obtained and a decision to intervene on the RCA-PLV branch was made.