CLINICAL VIGNETTE

Free Wall Rupture: A Rare Complication of Myocardial Infarction

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Clinical Presentation

An 81-year-old female was brought to the ED by paramedics for chest pain with nausea and vomiting. She was clammy and diaphoretic in the field and EKG showed ST elevations. She was given aspirin and sublingual nitroglycerin which improved her chest discomfort. Her past medical history includes hypertension, hyperlipidemia and mildly elevated coronary artery calcification score. She also had elevated A1C and mild to moderate bilateral carotid stenosis.

Initial ED vital signs included blood pressure of 96/64, pulse of 77, and O2 saturation of 99% on room air. She was alert and in no acute distress. Cardiac exam showed normal heart sounds without murmurs. Chest was clear and her skin was warm and dry. Initial labs included: WBC 13.8, creatinine of 1.3, glucose 161, BNP 325 and troponin 22.5ng/mL. EKG showed normal sinus rhythm with less than 1 mm ST elevations in the anterior leads, lateral 0.5 mm ST depressions with T wave inversions. She was taken to the cardiac catheterization laboratory, where she became unresponsive. Code Blue was called for pulseless asystolic cardiac arrest. CPR was initiated and ACLS protocols were followed. Bedside echocardiogram showed a large pericardial effusion. She underwent emergent pericardiocentesis and 500 mL of fresh blood was removed. Despite prolonged efforts, she could not be resuscitated and was pronounced about 1 hour after code blue initiation.

Discussion

This patient had acute myocardial infarction complicated by left ventricular free wall rupture. Free wall rupture, rupture of the interventricular septum, and acute mitral regurgitation due to papillary muscle rupture are three mechanical complications of acute myocardial infarctions. Interventricular septal rupture is the most common mechanical complication in patients with ST elevation myocardial infarction (STEMI) and free wall rupture is the least common. Table 1 presents the rates of the three types of rupture in STEMI and non-STEMI (NSTEMI) patients, from the National Inpatient Sample database of 9,000,000 MI hospitalizations between 2003-2015.[1]

Table 1

COMPLICATION	RATE IN STEMI	RATE IN NSTEMI
Ventricular septal rupture	0.21%	0.04%
Papillary muscle rupture	0.05%	0.01%
Free wall rupture	0.01%	0.01%

These complications can be fatal or lead to cardiogenic shock. They are caused by rupture or tearing of necrotic myocardial tissue. The incidence of these mechanical complications is approximately 3 per 1000 patients in the setting of an acute myocardial infarction in the reperfusion era, with most occurring in STEMI patients.[2] Prior to the widespread use of reperfusion, rupture of the free wall occurred in approximately 2 percent of acute MIs.[3]

Rupture of the myocardium involves the left ventricle more often than the right ventricle. Patients with had early, less than 72 hours free wall rupture, more commonly had anterior infarction sites with an abrupt tear in the infarcted myocardial wall. Patients with late rupture, greater than 4 days from MI had infarct expansion.[4]

Risk factors for myocardial rupture, in studies from the fibrinolytic era, include: no prior history of angina or MI; ST elevation on initial EKG; and peak CK-MB level > 150 IU/L. Patients with all 3 of these risk factors had 9.2 times higher risk of myocardial rupture.[5] Other risk factors include first MI, large transmural MIs, anterior MI, female gender, and age >70 years. Protective factors include left ventricular hypertrophy and the presence of extensive collaterals.[6] Over 50% of free wall ruptures occur within the first 5 days after a myocardial infarction and over 90% occur within the first 14 days.[7]

Clinical presentation can be variable. Patients with a silent MI who have cardiac rupture present with sudden death. Patients with known MI can have complete rupture or incomplete/ subacute rupture, which occurs when organized clot and pericardial tissue seal the perforation site.[8] Complete rupture leads to pericardial tamponade from hemopericardium with sudden right sided congestive heart failure and shock with progression to pulseless electrical activity and death.[9] In contrast, incomplete rupture may be present as pericardial chest pain, nausea, restlessness, acute hypotension, with or without EKG features of pericarditis.

Diagnosis is made by echocardiography which shows a large pericardial effusion and signs of tamponade. Initial treatment includes emergency pericardiocentesis, fluids, inotropic support, and vasopressor support if indicated.

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