

<b>ID/CC</b>	A 50-year-old male who was admitted to the CCU 3 days ago following an MI presents with <b>hypotension</b> .
<b>HPI</b>	The patient was thrombolized post-MI and was recovering well. He also complained of a mild fever but no chills or rigors.
<b>PE</b>	VS: tachycardia; weak, thready pulse; tachypnea; <b>hypotension</b> . PE: pallor; cool, moist skin; mild cyanosis of lips and digits; > 10-mmHg fall in arterial pressure with inspiration ( <b>PULSUS PARADOXUS</b> ); <b>heart sounds muffled</b> and <b>JVP elevated</b> ; lungs clear bilaterally.
<b>Labs</b>	Elevated cardiac enzymes (CK-MB, troponin) as a result of recent acute MI.
<b>Imaging</b>	Echo: diastolic compression of the right ventricle; pericardial effusion.
<b>Gross Pathology</b>	Rupture of the left ventricular wall with hemopericardium.
<b>Micro Pathology</b>	Ischemic coagulative necrosis of the affected myocardium, consisting of multiple erythrocytes and dead, anucleated myocytes.
<b>Treatment</b>	Emergency pericardiocentesis; treat shock by infusing fluid and isoproterenol; surgical repair of cardiac rupture subsequent to stabilization.
<b>Discussion</b>	Cardiac rupture most typically develops 3 to 10 days after the initial onset of the infarction secondary to rupture of necrotic cardiac muscle; there is usually little warning before the sudden collapse, which is associated with acute cardiac tamponade and electromechanical dissociation. Papillary muscle rupture may also occur following an acute MI, resulting in mitral regurgitation and left ventricular failure.