

Repair of contained ventricular rupture with infected intrapericardial thrombus

Running head: Infected ventricular rupture

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Ischemia-mediated ventricular rupture is a rarely encountered process in the era of early percutaneous revascularization. Contained rupture, or pseudoaneurysm often occurs as a result of pericardial adhesions. Even more uncommon is the presence of associated infection involving the rupture. We present the case of a patient with an infected intrapericardial thrombus in the setting of a left ventricular free wall rupture who underwent successful repair.

Introduction:

Non-traumatic left ventricular rupture is thought to be a result of inadequate coronary collateral circulation yielding profound ischemia, with eventual necrosis or scar formation, to a region of myocardium. This entity is infrequent given the aggressive, early percutaneous reperfusion techniques currently employed. Rarer so is the direct involvement of bacterial infection with the rupture. The case of a 64 year old patient with recent percutaneous coronary intervention found to have an infected intrapericardial thrombus in the setting of a left ventricular free wall rupture is discussed below.

Case Report:

A 64 year old man without prior medical history experienced an infero-lateral ST-segment elevation myocardial infarction. He was treated with drug eluting stent placement to the right coronary artery which was burdened by a large, acute thrombus. On post-procedure day two he complained of angina and returned to the angiography suite wherein drug eluting stents were placed in the circumflex artery. Subsequent transthoracic echocardiogram was unremarkable and demonstrated an ejection fraction of 55 percent. He was discharged on aspirin, atorvastatin, metoprolol, and ticagrelor.

He returned to our institution five weeks later complaining of 48 hours of malaise, fevers, dyspnea, and chest pressure. He was tachycardic and hypotensive but intermittently responsive to intravenous fluid resuscitation. Blood cultures obtained for concern of sepsis resulted in gram positive cocci growth. A CT of the chest noted a left ventricular irregularity and adjacent thrombus (figure 1). Transthoracic echocardiogram revealed an ejection fraction of 35 percent, a large pericardial effusion, an aneurysmal-appearing inferolateral left ventricular wall with intrapericardial thrombus, and akinesis of the inferolateral wall. These findings were better delineated via intraoperative transesophageal echocardiography (figure 2).

Emergent surgical intervention was performed with a median sternotomy, aorto-bicaval cannulation and antegrade cardioplegia. Dense intrapericardial adhesions were encountered and lysed

revealing a large pseudoaneurysm sac containing murky, seropurulent fluid. A culture of this fluid and surrounding tissue was obtained. After debridement of the pseudoaneurysm sac, a 4 centimeter defect in the inferolateral wall below the mitral valve was observed (figure 3). The defect was closed with a slightly oversized circular bovine pericardial patch secured to healthy endocardium with a running suture. The pseudoaneurysm edges were closed in a linear fashion using two layers of suture buttressed with felt strips. Post-repair transesophageal echocardiography demonstrated no ventricular geometry distortions or mitral valvular dysfunction (figure 2).

The patient required an intra-aortic balloon pump for 48 hours. He remained vasoplegic with septic-type physiology requiring vasopressin and norepinephrine during this time. Final blood and surgical fluid/tissue cultures revealed *Peptostreptococcus asaccharolyticus*. Infectious disease consultation supported a dental source and the patient was treated with six weeks of intravenous ertapenem. The patient was discharged in good condition on postoperative day 15.

Conclusion:

Left ventricular pseudoaneurysm is a rare complication following myocardial infarction in the current era due to early percutaneous revascularization. Previous reports have demonstrated the inferior or posterolateral ventricular walls as the predominant sites.^{1,2} Pseudoaneurysms represent rupture of ischemic myocardium contained by the formation of intrapericardial adhesions. Patients with ventricular free wall rupture yield a high mortality given the resulting acute cardiac tamponade.^{1,2} The current patient likely developed a post-infarction pericarditis which permitted his ability to present to surgery. A lack of coronary arterial collateralization likely predisposed the current patient to ventricular rupture.

Surgical repair is warranted given the propensity for complications such as worsening heart failure and tamponade. Prior studies have revealed mortality rates for medically managed pseudoaneurysms near 50 percent. Surgical therapy carries a risk of mortality around ten percent although this increases in the presence of concomitant mitral valve intervention.^{1,2} With regard to the current

patient, one may advocate for a period of observation to allow for potential maturation of necrotic myocardium and delineation of viable ventricular wall for repair. Nevertheless, given the large myocardial defect, ongoing heart failure symptoms, acute decline in ejection fraction, and persistent hypotension it was felt that emergent repair was necessary. Concern for infection should further support emergent surgical repair over medical management.

The finding of an infected thrombus was surprising as the majority of limited reports available describe infection of true left ventricular aneurysms, often with associated mural thrombi.³ Even less common are publications on contained rupture and pseudoaneurysm with coexisting infection.⁴⁻⁸ A single report describes a case similar to the present patient wherein a successful repair of a posterolateral left ventricular rupture was achieved in the setting of purulent pericarditis rather than an infected intrapericardial clot. Both the current case and the aforementioned patient yielded peptostreptococcus.⁸ As staphylococcus and streptococcus species predominate as the organisms in the referenced reports, anaerobic bacteria have also been documented as sources of intrapericardial infection; however, the majority are gram negative.⁹ The presence of the anaerobic, gram positive peptostreptococcus outside of recent dental work or clinically observed dental infection represents a rare, but possible source.

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Figure Legends:

Figure 1. Coronal computed tomography image demonstrating left ventricular wall defect and intrapericardial thrombus. Asterisk = left ventricular cavity, arrow = left ventricle free wall defect, arrowhead = thrombus.

Figure 2. Pre- and post-repair transesophageal echocardiography images. Leftward image: asterisk = left ventricular cavity, arrow = ventricular wall defect, arrowhead = mitral valve. Rightward image: asterisk = left ventricular cavity, arrow = ventricular wall patch closure, arrowhead = mitral valve.

Figure 3. Intraoperative photograph demonstrating left ventricular wall defect. The heart is oriented with the apex retracted cephalad. Arrow = left ventricular wall & mitral valve chordae, arrowhead = pseudoaneurysm wall.