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Left Ventricular Pseudoaneurysm and Left Ventricular Thrombus in a Patient Presenting with an Acute ST-Elevation Myocardial Infarction

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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 73-year-old
Final Diagnosis: Pseudoaneurysm • thrombus
Symptoms: Chest pain • sycope
Medication: —
Clinical Procedure: —
Specialty: General and Internal Medicine

Objective: Unusual clinical course

Background: ST-elevation myocardial infarction (STEMI), when associated with acute left ventricular (LV) free-wall rupture, is often a lethal complication, and if not followed by sudden death, the rupture may be contained by the parietal pericardium and a local thrombus, leading to the formation of a left ventricular (LV) pseudoaneurysm. The incidence of LV pseudoaneurysm after STEMI is ~ 0.3%.

Case Report: A 73-year-old man who presented with an acute syncopal episode and intermittent chest pain for 7 days was found to have an anterolateral myocardial infarction (MI) with lateral wall rupture and pseudoaneurysm formation. He had an LV thrombosis in the LV aneurysm. While this increased his risk of thromboembolic events, it likely stopped the evolution of the rupture and stabilized the pericardial effusion size. The patient underwent coronary artery bypass grafting (CABG), thrombectomy, and lateral wall repair.

Conclusions: Left ventricular pseudoaneurysm and left ventricular thrombus in a patient presenting with an acute ST-elevation myocardial infarction is a rare complication of myocardial infraction, with an incidence of <1%. It is often a lethal complication and requires stabilization and repair if not followed by sudden death.

Keywords: Thrombectomy • ST Elevation Myocardial Infarction • Ventricular Remodeling

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/934272

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Background

Acute myocardial infarction has 3 major mechanical complications: left ventricular (LV) free-wall rupture, interventricular septum rupture, and acute mitral valve regurgitation (MVR) [1,2]. ST-elevation myocardial infarction (STEMI) is often a lethal complication when associated with acute LV free-wall rupture. If not followed by sudden death, the rupture may be contained by the parietal pericardium and local thrombus, leading to the formation of a left ventricular (LV) pseudoaneurysm. The incidence of LV pseudoaneurysm after STEMI is 0.2% to 0.3% [3]. Untreated pseudoaneurysms have a 30-45% risk of rupture, carrying a mortality rate of almost 50% despite medical treatment, and 24-40% with surgical treatment [4]. Thus, it is essential to accurately diagnose LV pseudoaneurysms and to manage them to improve patient outcomes.

Case Report

The patient was a 73-year-old man with a past medical history of insulin-dependent diabetes and obesity. He reported a sudden syncopal episode while using the restroom, with no initial symptoms. Upon arrival to the Emergency Department (ED), although initially hypertensive, he suddenly became hypotensive and reported feeling back pain. His vitals were remarkable for blood pressure of 65/40 mmHg, heart rate of 86 beats per minute, SaO2 95% on 4 liters/minute oxygen via nasal cannula, and temperature of 36.7°C. His physical examination was remarkable for visible and palpable double systolic apical impulse in the fifth intercostal space of the left midclavicular line and pericardial rub, with the first component representing the normal apical outward movement and the second the bulging of the aneurysm during peak ventricular pressure later in systole. His serology was notable for anemia with hemoglobin of 11.3 g/dL (grams per deciliter) (normal range of 11.9-15.1 g/dL), leukocytosis with white blood cell count 32.6×10^3/uL (normal range of $4.4\text{-}10.5\times10^3$/uL), acute kidney injury with creatinine of 2.96 mg/dL, and troponins were elevated at 38.03 nanograms per milliliter (ng/mL) (normal range of 0 and 0.4 ng/mL) using a cardiac troponin I assay. On further questioning, he reported having chest pain approximately 5 days prior to his presentation. He described the pain as substernal, non-radiating, and lasting for about 8 h before subsiding on its own. He did not present at the onset of symptoms given the current Covid pandemic.

EKG showed diffuse ST elevations in the inferior and lateral leads, consistent with STEMI (**Figure 1**). Although he had STEMI, given his lack of angina, there was a concern for an artifact, and a repeat EKG was ordered. A bedside transthoracic echocardiogram (TTE) showed an ejection fraction (EF) of 25-29%, diffuse hypokinesis, and a moderate circumferential pericardial effusion with density consistent with an organized clot. There were no signs of mitral regurgitation or tamponade on the initial echo. A hyperechoic mobile structure was noted in the left ventricle (LV) concerning for a thrombus or vegetation, and an LV apical aneurysm with contained rupture (**Figure 2A, 2B**) was found. Aortic dissection could not be ruled out, so a computed tomography angiography of the chest (CTA) was ordered,

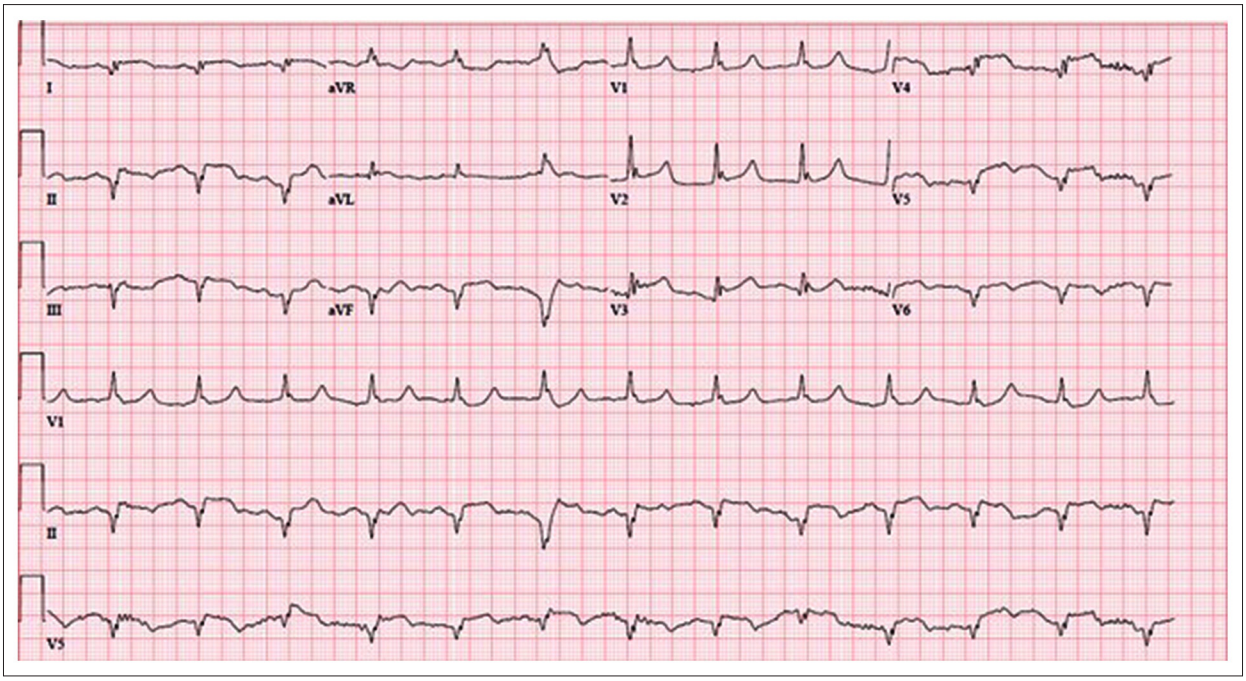

Figure 1. Acute myocardial infract with diffuse ST elevation.

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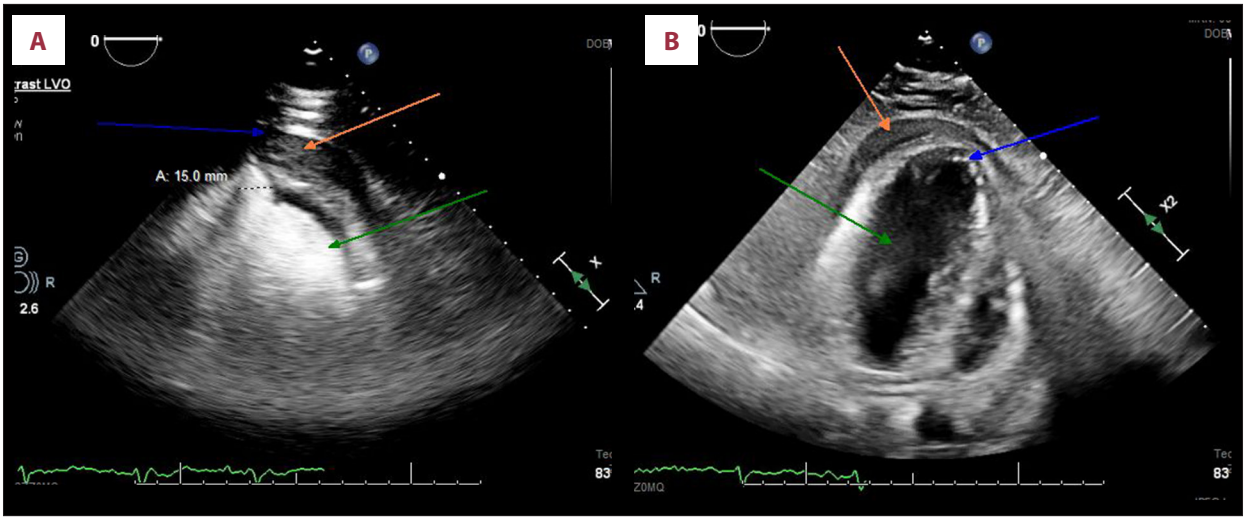

Figure 2. Apical 4-chamber view echocardiogram. Orange arrow showing pericardial effusion. Blue arrow showing aneurysmal segment with site of suspected contained rupture and neck of aneurysmal segment measurement. (**A**) Green arrow showing LV filled with contrast, (**B**) green arrow showing LV without contrast.

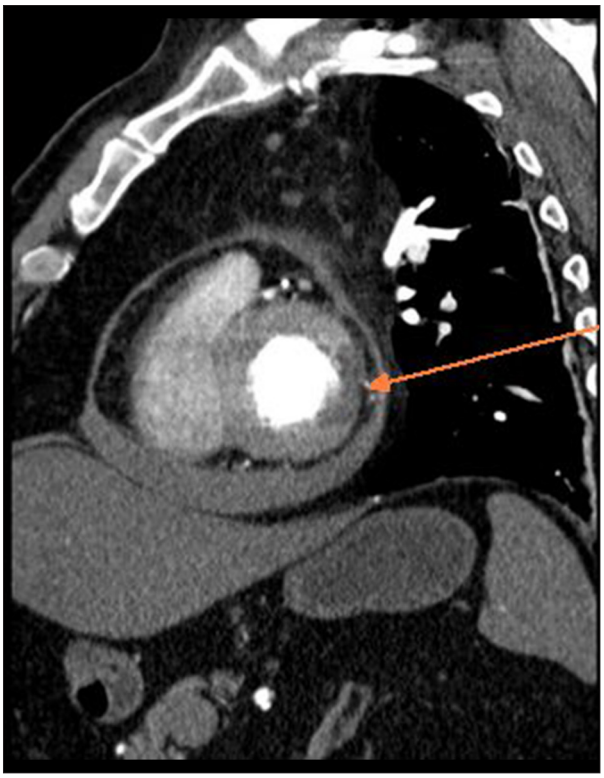

Figure 3. CT scan showing change in lateral wall contrast perfusion concerning for possible ischemia/infarct.

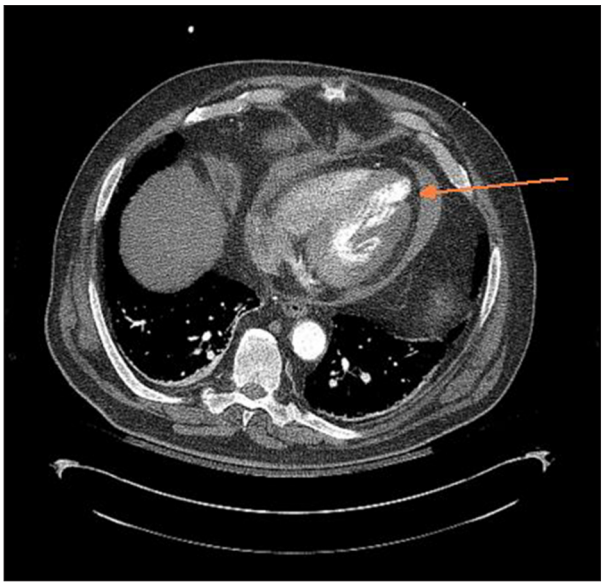

Figure 4. Small pericardial effusion, with moderate internal density. This is nonspecific but can be seen in the setting of an exudative pericarditis. Arrow pointing to area with notable defect concerning for possible contained rupture.

which showed no aortic dissection, small exudative pericardial effusion, and findings suspicious for apical infarction and LV pseudoaneurysm, as suggested by thinning myocardium at the apex (**Figures 3, 4**). A repeat EKG returned unchanged, and he was taken to the cardiac catheterization lab emergently. He was diagnosed with multivessel coronary artery disease, showing a dominant left system, left main normal, and left circumflex with mild disease. The obtuse marginal branch (OM) 1 branch had small luminal changes, an occluded OM 2 branch at the ostium, and OM 3 branch with 90% stenosis in the mid-segment. The left posterior descending artery (PDA) branch showed 90% stenosis from ostium to mid-segment. The left anterior descending (LAD) had 80% stenosis from the middle to distal segment and 90% stenosis at the apex. He also had an ejection fraction of 25-30% with confirmed left ventricular thrombus, as well as

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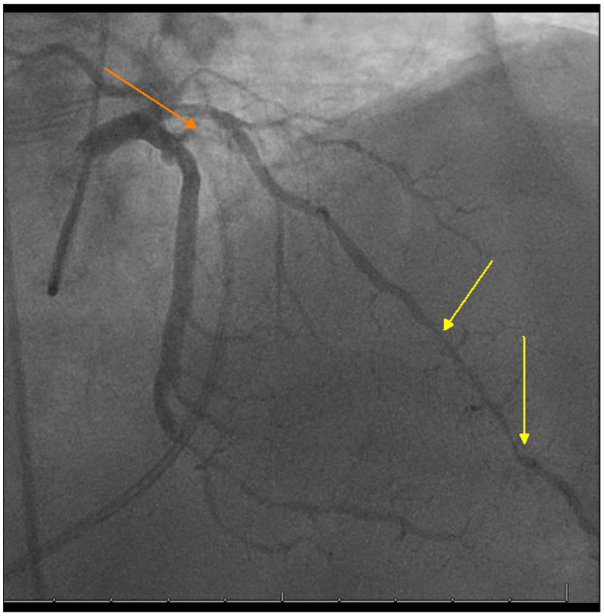

Figure 5. Orange arrow showing complete occlusion of obtuse marginal branch. Yellow arrows showing severely diseased middle and distal segments of left anterior descending artery.

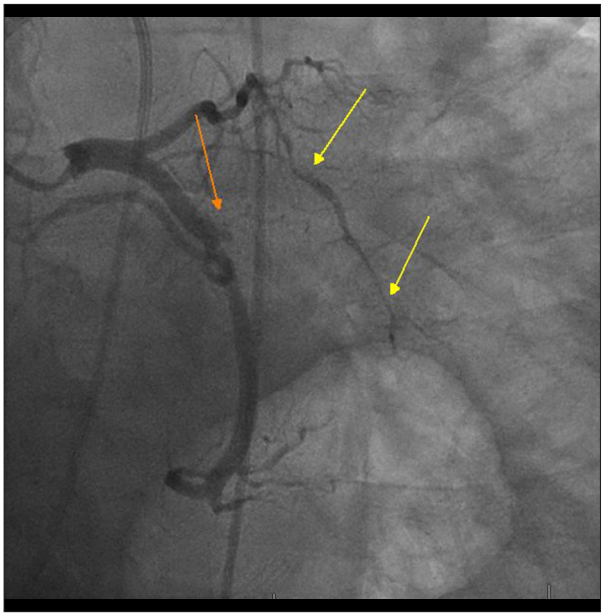

Figure 6. Orange arrow showing complete occlusion of the obtuse marginal branch. Yellow arrows showing severely diseased middle and distal segments of left anterior descending artery.

apical aneurysm with contained rupture (**Figures 5, 6**). No percutaneous coronary intervention (PCI) was done, but an intra-aortic balloon pump was placed for cardiogenic shock. The patient was intubated and transferred to the cardiac care unit.

He had a cardiac index of 2 L/min/m^2 and required vasopressor support with norepinephrine bitartrate. He underwent coronary artery bypass grafting (CABG), a thrombectomy of a fresh clot from the terminal OM, and repair of a tear in the lateral wall of the LV with a bovine pericardial patch. Strips are sewn to the lateral wall of the LV just cephalad to the terminal OM, and retrieval of embolic material from the LV via aortotomy. He remained in the coronary care unit until he became hemodynamically stable and was off pressors, and was extubated. Postoperatively, he had atrial fibrillation. There remained 1 mobile LV thrombus, so he was on a heparin drip and bridged to warfarin with an international normalized ratio (INR) goal of 2-3. He was stable, and a repeat TTE prior to discharge showed an EF of 25-29%. He was discharged on aspirin, warfarin, high-intensity atorvastatin, metoprolol succinate, and bumetanide, and he was fitted with a LifeVest wearable defibrillator. The patient made an excellent recovery, and reported that he was doing well at his outpatient follow-up appointments.

Discussion

We discuss the case of a 73-year-old man with a history of hypertension, hyperlipidemia, and diabetes who presented to our hospital with acute syncopal episodes and intermittent chest pain for the last 7 days. He was found to have an anterolateral MI with lateral wall rupture and pseudoaneurysm formation.

If it occurs, myocardial rupture usually occurs within the first 3-5 days after the infarction in half of the cases (early rupture) and within the first 2 weeks in 90% (late rupture) [5]. Left false ventricular aneurysm, also known as pseudoaneurysm, is a rare and catastrophic pathology. It most commonly presents as a complication of myocardial infarction (MI) in about 4.7% of MI cases [6]. It occurs when an ischemic left ventricular wall rupture is contained by a hematoma communicating with the ventricular cavity. The ruptured wall can become sealed by an adherent pericardium, organizing hematoma, thrombus, or scar tissue and has the appearance of an aneurysm [7]. The wall of the pseudoaneurysm consists only of fibrous tissue or pericardium. It lacks the true layers of the ventricle (endocardium and myocardium) [8]. The expected sequela of a false aneurysm is the rupture of the adherent sac [9]. The mechanism of underlying ventricular wall rupture is still controversial, but it has been suggested to be due to the dissociation of the non-contractile infarcted region from surrounding healthy and contractile regions [10].

LV pseudoaneurysm may be filled with thrombus, with a potential risk of systemic embolism. A thrombus can form in the chamber of the aneurysm and gradually increase in size. An LV thrombus reduces the continuous impact of blood flow on the wall of the pseudoaneurysm. Eventually, the thrombus can fill

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the aneurysm and prevent blood from flowing into the aneurysm from the left ventricle. In 1 report, 13% of the patients with a left ventricular pseudoaneurysm had systemic embolism as the initial clinical presentation [11]. A ventricular thrombus was seen in the current patient, but the thrombus formation likely resulted in a good prognosis for him as it reduced blood flow out of the aneurysm free-wall rupture into the pericardium. No anticoagulation therapy was used initially, and only anti-platelet therapy was used. After surgical repair, he was started on anticoagulation therapy to avoid the possible risk of thromboembolism due to residual thrombus in the LV.

Clinical findings of ventricular rupture are usually nonspecific, such as chest pain, congestive heart failure, thromboembolic events, arrhythmias, cyanosis, hypotension, syncope, bradycardia, pulsus paradoxus, elevated venous pressures, very feeble heart sounds, electromechanical dissociation, and cardiogenic shock [12]. Sudden death is the least frequent presentation.

Risk factors for ventricular wall rupture are older age, female sex, pre-existing hypertension, absence of left ventricular hypertrophy, and first-time myocardial infarction [13]. Also, patients with free-wall rupture are less likely to have diabetes or a history of prior MI. It is presumed that people with no prior history of myocardial infarction are less likely to have developed collateral circulations that protect the myocardium in the setting of acute vessel occlusion, as mentioned in the SHOCK trial registry [14].

Diagnosing LVFWR and pseudoaneurysm can be difficult. Transthoracic echocardiography (TTE) or transesophageal echocardiography (TEE) is usually the preferred imaging test, as it is readily available and fast. The most common finding is pericardial effusion, and if the effusion is absent, LV rupture is almost excluded. However, TTE or TEE may sometimes be unrevealing. Cardiac MRI is an excellent diagnostic test that identifies the contained cardiac rupture and distinguishing pseudoaneurysm from true aneurysms and provides clear anatomical features to guide surgical interventions [15].

Most patients with LVFWR are hemodynamically unstable. Thus, cardiac computed tomography (CT) chest scanning is the next-best imaging option in these patients, as it is more widely available, allows visualization of the LV myocardium, and shows segments that are difficult to see on echocardiography. Usually, a multimodality imaging approach that incorporates more than 1 imaging test is necessary for diagnosis [5].

Left ventricular free-wall rupture (LVFWR) is a life-threatening condition that has gradually disappeared due to the great improvement in percutaneous coronary intervention (PCI) techniques and early intervention.

According to recently published studies on the COVID-19 pandemic outcomes in patients with STEMI, there was a significant increase in the late presentation and decreased rate of primary PCI. Also, mechanical complications in patients with acute myocardial infarction have increased in the COVID-19 era due to delays in seeking medical care after the onset of symptoms and the late presentation of the patients. Our patient reported avoiding early medical care because of increasing fear of acquiring Coronavirus infection in the hospital.

Immediate surgery is the treatment of choice because there is a 30-45% risk of rupture when a pseudoaneurysm is left untreated [16]. Once the diagnosis is established and LV pseudoaneurysms detected, surgery is recommended within the first 3 months after MI [17]. In this case, CABG, thrombectomy, and repair of the tear in the lateral wall of the LV were performed. Although postoperative mortality rates are 7-30% in some case reports [18], our patient is still functioning well and receiving optimal medical treatment and regular follow-up visits as an outpatient.

Conclusions

In conclusion, this is an atypical case of rare LV free-wall rupture and pseudoaneurysm formation after acute STEMI. Our patient presented 5 days after onset of chest pain and delayed seeking medical care due to fear of the Coronavirus. On presentation, he had multimodality imaging tests and was diagnosed promptly. In addition to the LVFWR and PSA, he had an LV thrombosis in the LV aneurysm. While this increased his risk of thromboembolism events, it stopped the evolution of the rupture and stabilized the pericardial effusion size. Our patient had CABG, thrombectomy, and lateral wall repair. This treatment in the acute phase saved our patient's life.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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