Contained Free Wall Rupture after Myocardial Infarction

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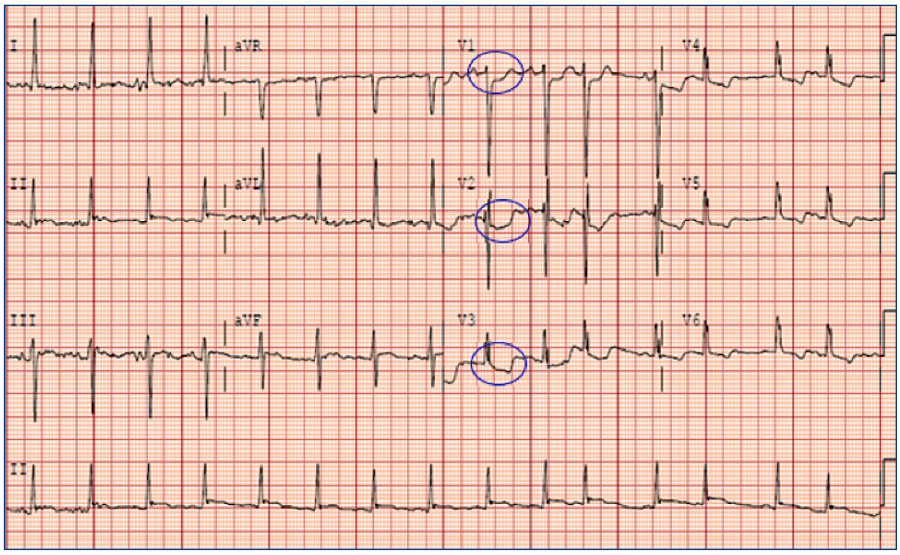
CASE SUMMARY

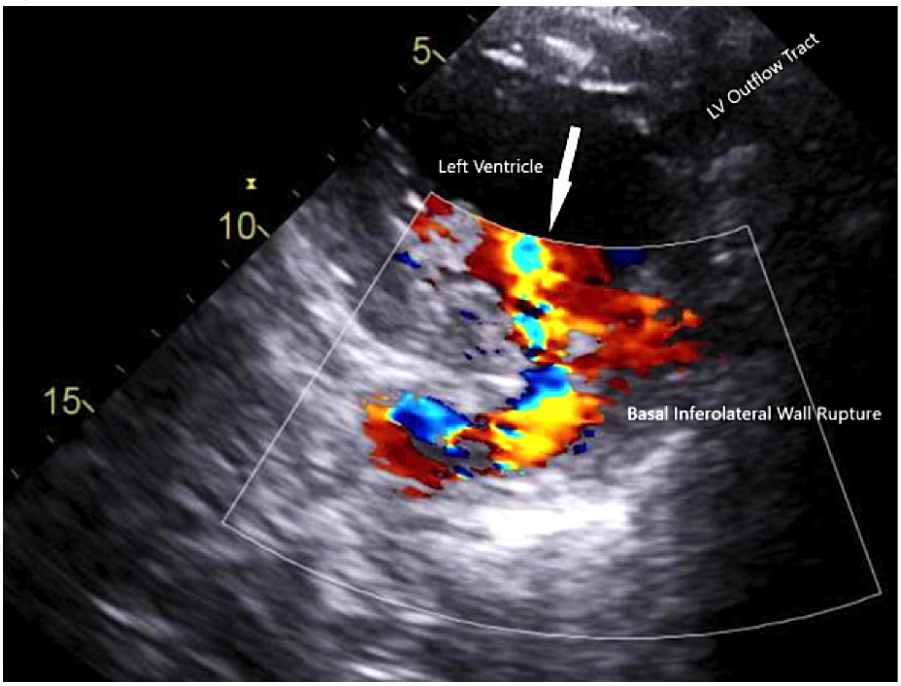
A 78-year-old man with Alzheimer's disease, coronary artery disease and anteroapical infarction 18 years prior resulting in triple-vessel Coronary Artery Bypass Grafting (CABG), presented via ambulance after his wife had found him stumbling in his bathroom. His only complaint leading up to this episode was right-shoulder pain. Initially vital signs were stable, but over the course of four hours he developed tachypnea and hypoxia. Electrocardiogram demonstrated new ST-depressions in precordial leads V1-3 (**Figure 1**). Troponin was elevated to 15.18 ng/mL. Patient was administered loading doses of both aspirin and ticagrelor given concern for posterior wall infarction. On Bipap, patient became hypotensive to 84/61. The patient's hemodynamic deterioration prompted an urgent bedside echocardiogram. A contained left ventricular free wall rupture was visualized (**Figure 2**).

Figure 1. EKG on Presentation



*New ST-depressions in the precordial anterior leads (V1-V3), circled in blue

Figure 2. Bedside Transthoracic Echocardiogram, Parasternal Long Axis



*Left ventricular wall rupture with significant structures labeled. White arrow indicating ventricular wall defect and direction of flow.

DISCUSSION

Free wall rupture is one of the more feared complications of myocardial infarction (MI), occurring classically within the first week of MI. A pseudoaneurysm forms when the rupture is contained by pericardium or scar tissue. Pseudoaneurysms can also be caused by trauma or surgery. However, MI is reported as the etiology more than half of the time.[1] When infarction is the culprit, the inferior or posterolateral wall are the most common areas for pseudoaneurysm formation.[2] If a pseudoaneurysm forms following an MI, it generally has a narrow neck with an abrupt disfiguration of the left ventricle.[3]

Figure 2 fits the discussed descriptors for pseudoaneurysm formation following an MI. The rupture opening is narrow at the basal inferolateral wall causing compression of the left ventricle and obscuring normal landmarks. Our patient most likely experienced a silent MI in the week leading to his presentation. Surgical management of ventricular rupture following infarction is complicated because the defect is difficult to reach at the base of the heart and is surrounded by necrotic tissue. This patient had been loaded with dual antiplatelet therapy (DAPT) making any surgical intervention more hazardous. Given recent administration of dual antiplatelet therapy, his progressive Alzheimer's disease, and the overall high risk for surgical mortality; immediate surgical management was not recommended. The patient and his family decided to pursue comfort measures only with no further escalation of care.

References

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