Getting over a Broken Heart: Intramyocardial Dissecting Hematoma as Late Presentation of Myocardial Infarction

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INTRODUCTION

Well-described mechanical complications of myocardial infarction (MI) include ventricular septal rupture, papillary muscle rupture with secondary severe mitral regurgitation, and ventricular free wall rupture with either rapid progression of hemodynamic instability and death or containment with pseudoaneurysm formation. Intramyocardial dissecting hematoma is a rare mechanical complication of MI; increased familiarity with echocardiography and multimodality cardiac imaging is essential for a rapid diagnosis and subsequent management.

CASE PRESENTATION

A 42-year-old man presented to the emergency department with a 2-week history of shortness of breath on exertion and new-onset cough. Before this presentation, he sought medical attention and received a presumptive diagnosis of pneumonia and was treated with oral antibiotics, with no alleviation of his symptoms. His symptoms progressed further, with development of orthopnea, paroxysmal nocturnal dyspnea, and peripheral edema in the preceding 3 days. He denied any history of chest pain, palpitations, presyncope, syncope, or claudication. His medical history was unremarkable, with no coronary artery disease, valvular heart disease, or congestive heart failure. He had no known cardiac risk factors and was not taking any regular medications. Before the recent illness, he was physically active and enjoyed recreational biking and running. He worked as a welder, infrequently smoked marijuana, and had a remote history of cocaine use 7 years prior. There was no family history of cardiovascular disease or sudden death.

Physical examination revealed a comfortable patient in no acute distress. Initial evaluation of vital signs revealed a regular heart rate of 99 beats/min and blood pressure of 122/74 mm Hg that was equal bilaterally. He was afebrile, and his oxygen saturation was 93% on room air. Jugular venous pressure was elevated at 6 cm above the sternal angle with a normal waveform and a positive hepatojugular reflux. Carotid pulse was of normal volume and contour, with no audible bruits. Precordial palpation revealed an enlarged apical impulse, which was laterally and inferiorly shifted by one interspace. There were no heaves or thrills palpable. Cardiac auscultation revealed normal S_1 and S_2, with an audible S_3 at the apex. There were no other additional sounds or murmurs. Respiratory examination revealed clear and equal breath sounds bilaterally, with the presence of bibasilar crackles at both lung bases. Peripheral pulses were palpable, and there was minimal bilateral pitting edema at the ankles. Abdominal examination was unremarkable, with no audible abdominal or femoral bruits. Initial blood work revealed a normal complete blood count and normal renal and coagulation profiles. Serum cardiac troponin I level was mildly elevated at 0.075 μg/L (upper limit of normal <0.045 μg/L). Chest radiography showed cardiomegaly with mild interstitial edema. Twelve-lead electrocardiography (Figure 1) showed normal sinus rhythm, with q waves in leads V_1 to V_6 with 1 mm of ST-segment elevation and biphasic T waves. Because of suspicion of a late presentation extensive anterior MI, the patient was transferred to the coronary care unit for ongoing management.

Urgent echocardiography (Figure 2, Videos 1 and 2) was performed and showed a severely dilated left ventricle with severely depressed systolic function and an ejection fraction that was <20%, with multiple regional wall motion abnormalities, including akinesis of the entire apex, anterior wall, inferior wall, and interventricular septum. No hemodynamically significant valvular dysfunction was present. The right ventricle was of normal size, with mild systolic dysfunction. There was a well-delineated echogenic layer separating the mid–left ventricular (LV) cavity from the apex. Between the apex and this echogenic structure, there was a mixture of echolucent and echogenic components, without evidence of communication with the mid-LV cavity by both Doppler color flow and microbubble contrast (Definity; Lantheus Medical Imaging, North Billerica, MA). No pericardial effusion or ventricular septal defects were present (Figure 3, Video 3). The suspected diagnosis was late-presentation extensive anterior MI with the rare mechanical complication of an intramyocardial dissecting hematoma involving the entire apex, with early thrombus formation within the dissection plane.

Coronary angiography (Figure 4, Video 4) revealed single-vessel disease with subtotal occlusion of the proximal left anterior descending coronary artery with visible intraluminal thrombus and mild disease in the left circumflex artery and the right coronary artery. Both computed tomographic (CT) angiography and cardiac magnetic resonance imaging (MRI) were performed for further characterization of the mechanical complication. The initial CT angiogram, obtained on the second day of hospitalization, revealed a severely dilated left ventricle with a crescentic low-attenuation filling defect in the apical portion of the LV cavity. There was a thin rim of myocardium overlying the apex, with no definite full-thickness rupture identified. On the luminal side of the filling defect, there was a rim of myocardium evident to suggest that the filling defect was intramyocardial. Given the superior tissue characterization, cardiac MRI with gadolinium

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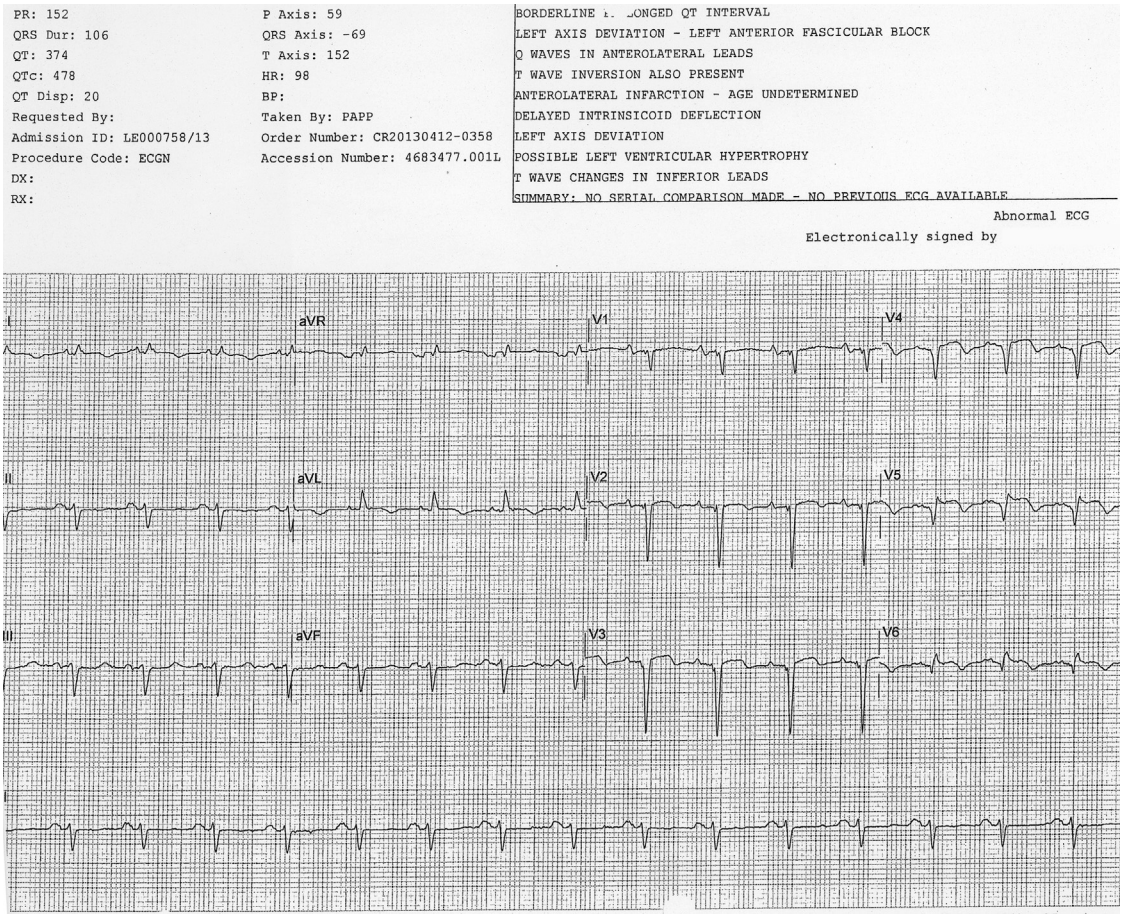

Figure 1 Twelve-lead electrocardiogram demonstrating sinus rhythm, left axis deviation, and Q waves across the precordial leads. Evolving T waves and ST-segment deviation are seen in leads V_3 to V_6.

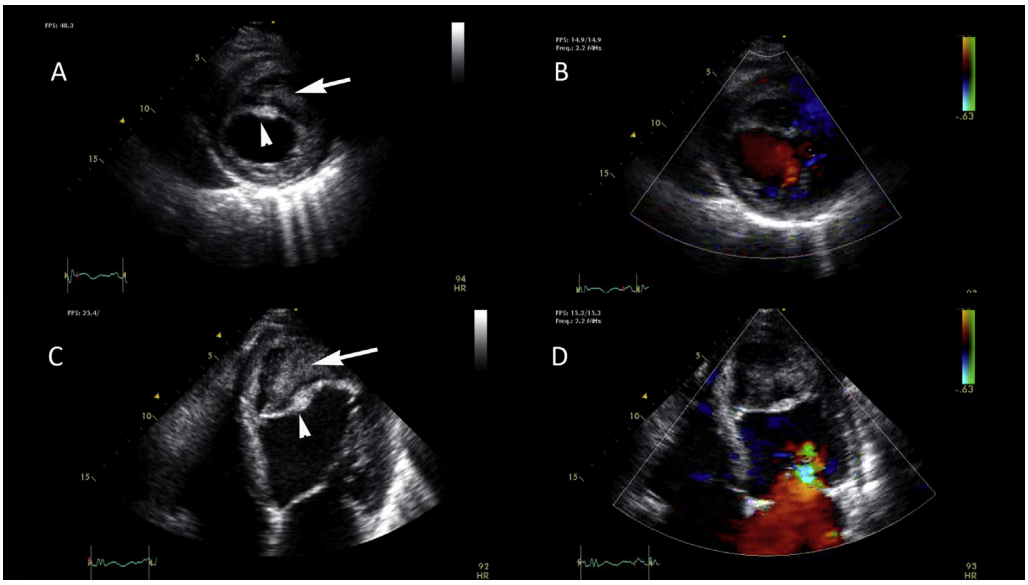

Figure 2 Transthoracic echocardiogram from parasternal **(A,B)** and apical **(C,D)** windows. A layer of myocardium is seen (*arrowhead*), starting in the distal third of the myocardium. Distal to this layer, there is mobile echodensity (*arrow*), consistent with thrombus. Color Doppler images **(B,D)** show no obvious flow within the distal cavity at the Nyquist limit of 63 cm/sec.