Intramyocardial Dissecting Hematoma in Patients with Ischemic Cardiomyopathy: Role of Multimodality Imaging in Three Patients Treated Conservatively

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INTRODUCTION

Intramyocardial dissecting hematoma (IDH) is a rare form of cardiac rupture that can occur as a complication following acute myocardial infarction (MI) or during the remodeling process. It is usually caused by a hemorrhagic dissection through the myocardium.[1,2] It consists of blood infiltration into and through the myocardial wall, which maintains endocardial and epicardial integrity. Formation of IDH may result from rupture of intramyocardial vessels into the interstitial space, decreased tensile strength of the infarcted area, and acute increase of coronary capillary perfusion pressure.[3,4] Before the advent of noninvasive imaging techniques, the diagnosis of IDH was made only by necropsy. It can develop in the left ventricular (LV) free wall, the right ventricle, or the interventricular septum.[5] We describe three cases, focusing on the utility of echocardiography and the use of other noninvasive imaging modalities. All of our patients were treated conservatively.

CASE PRESENTATION

A 59-year-old man with underlying ischemic dilated cardiomyopathy and New York Heart Association class II symptoms presented with chest pain precipitated by exertion, with worsening effort tolerance, orthopnea, paroxysmal nocturnal dyspnea, and bilateral leg swelling. Electrocardiography revealed biphasic T waves in lead V_3, deep T-wave inversion in leads V_4,V_5, and V_6, and Q waves and T-wave inversion in the inferior leads (Figure 1A). Therefore, chest pain was thought to be secondary to MI. Diagnostic procedures included cardiac catheterization, which showed single-vessel disease of the left anterior descending coronary artery (Figure 1B). Echocardiography was performed, showing a severely depressed LV ejection fraction (LVEF) of 21%, as well as extensive apical, mid anteroseptal, and mid inferior wall akinesia. A thickened and pulsatile LV cavity with dyskinetic motion surrounded by a thin endomyocardial border was visualized, suggesting contained rupture of an infarction with hematoma (Figures 1C and D, Videos 1, 2, and 3). Color Doppler interrogation revealed no flow between the left ventricular cavity and layers of myocardium, which showed no connection between the echo-free space and the left ventricle. There was no color flow seen in the left ventricle, because of the low-flow state and thrombus formation. No pericardial effusion or evidence of epicardial disruption was seen. Cardiac magnetic resonance imaging showed normal basal wall thickness, severe hypokinesia of the mid anterior and septal walls, and an akinetic apical segment. An intramyocardial dissection cavity was seen extending from the LV mid inferoseptal wall to the apical segments, with a large thrombus within the cavity. There was delayed gadolinium enhancement of the mid anterior and anteroseptal walls with extension to the apical segments consistent with infarction of the left anterior descending coronary artery territory. The dissection flap also showed late gadolinium enhancement. The diagnosis of recent anterior MI complicated by large apical intramyocardial dissecting hematoma was made, and the patient was treated conservatively.

Our second case was a 49-year-old man with underlying ischemic dilated cardiomyopathy admitted with deteriorating effort tolerance, pedal edema, and chest discomfort. At initial presentation, vital signs were stable (heart rate 84 beats/min, blood pressure 113/77 mm Hg, oxygen saturation 90% on room air). Cardiovascular examination revealed normal first and second heart sounds, without murmurs. Fine rales were heard in both lung bases. No coronary angiography was performed, because it was decided at the time that the severely dilated left ventricle pointed to nonviable myocardium. Two-dimensional echocardiography demonstrated dilated LV dimensions. The calculated LV internal diastolic dimension was 70 mm, and LV internal systolic dimension was 65 mm. All cardiac valves appeared normal, and there was no evidence of pericardial effusion. A biplane Simpson–calculated LVEF of 20%–25% showed depressed LV systolic function. Echocardiography also revealed akinetic motion of the mid septal to apical segments, a mobile endocardial flap, and echo-free spaces over the apical segment filled with spontaneous echocardiographic contrast. The same finding also was seen on three-dimensional echocardiography (Figures 2A and 2B, Videos 4 and 5). Color Doppler flow mapping showed no flow through the echo-free space and between the left ventricle and the endocardial flap. Three months later, subsequent echocardiography showed increased echogenicity over the apex, consistent with focal thrombosis (Figure 2C, Video 6). There was no change in LVEF, LV dimension, regional wall motion, or any valvular lesions with color Doppler flow mapping.

Our last patient was a 54-year-old woman admitted to our institution with worsening shortness of breath. Electrocardiography showed sinus rhythm and pathologic Q waves over leads V_1 to V_3, suggestive of anterior wall MI. Coronary angiography demonstrated total occlusion of the left anterior descending coronary artery and mild disease over the circumflex coronary artery and the right coronary artery. Bedside echocardiography revealed akinesis of the apical and lower

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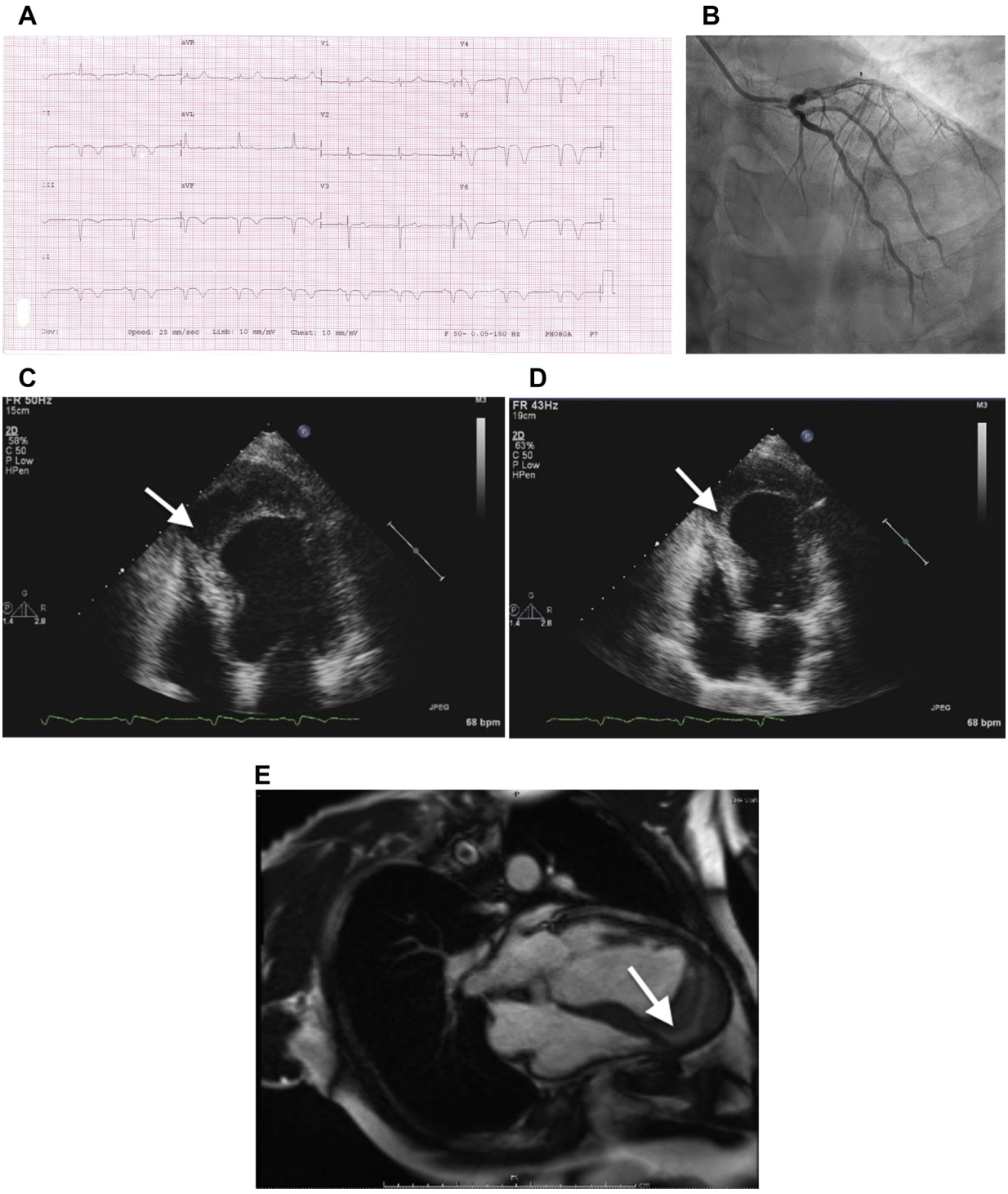

Figure 1 **(A)** Electrocardiogram at presentation shows biphasic T wave in lead V_3, deep T-wave inversion in leads V_4, V_5, and V_6, and Q waves with T-wave inversion in the inferior leads, consistent with angiographic findings. **(B)** Coronary angiogram shows moderate stenosis at proximal segment of the left anterior descending coronary artery, severe stenosis at midsegment, and complete total occlusion in the distal segment. **(C-E)** Transthoracic two-dimensional echocardiographic and cardiac magnetic resonance views of IDH. **(C,D)** At presentation, apical four-chamber view showing dissecting echo-free cavity (*arrow*). **(E)** An IDH was confirmed by gadolinium-enhanced magnetic resonance imaging, revealing a large thrombus (*arrow*) within the apical intramyocardial dissection cavity containing the hematoma in the apical segment.

anterior ventricular septum with formation of an echo-dense thick apical aneurysm, expanded and compressing the right ventricular chamber, with pulsatile systolic expansion (Figure 3A, Videos 7 and 8). Color Doppler assessment demonstrated no flow between the left ventricle and layers of myocardium, thus showing IDH. Right ventricular function was severely depressed, with tricuspid annular plane