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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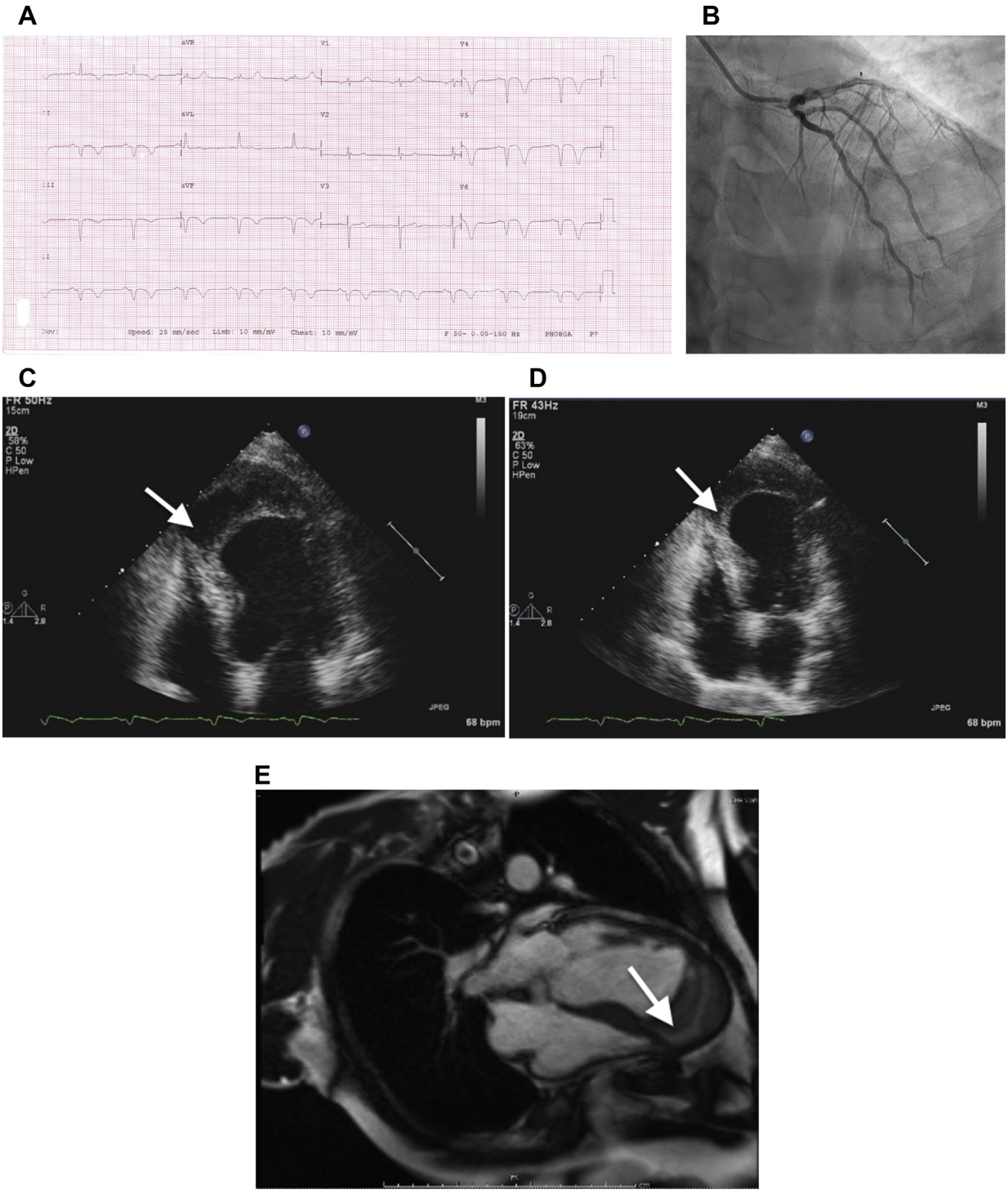

Figure 1 **(A)** Electrocardiogram at presentation shows biphasic T wave in lead V_3, deep T-wave inversion in leads V_4, V_5, and V_6, and Q waves with T-wave inversion in the inferior leads, consistent with angiographic findings. **(B)** Coronary angiogram shows moderate stenosis at proximal segment 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

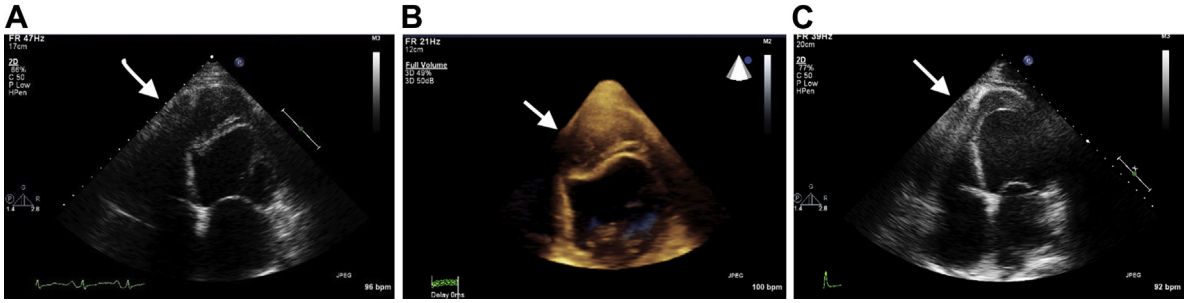

Figure 2 **(A)** Transthoracic two-dimensional echocardiographic views of intramyocardial dissection (*arrow*). **(B)** Transthoracic three-dimensional echocardiography showing a mobile endocardial flap with intramyocardial dissection cavity with spontaneous echocardiographic contrast (*arrow*). **(C)** Increased echogenicity over the apex (*arrow*), consistent with thrombus formation at 3-month follow-up.

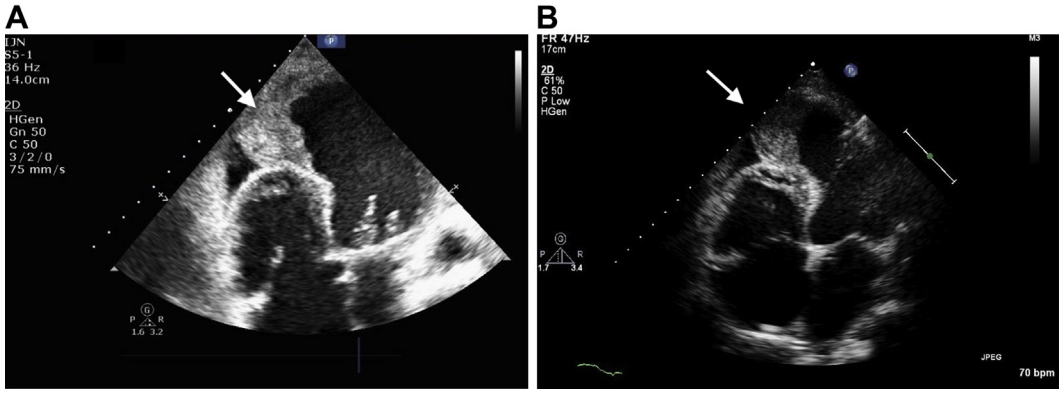

Figure 3 **(A)** Thick apical aneurysm (*arrow*), expanded and compressing the right ventricular chamber with pulsatile systolic expansion and high echo density suggestive of IDH. **(B)** Subsequent echocardiography 3 months later showing no significant changes compared to previous echo (*arrow*).

systolic excursion of 0.8 cm and tricuspid annular systolic tissue velocity of 5.4 cm/sec. Two days after admission, the patient experienced cardiac arrest, and cardiopulmonary resuscitation was performed for 10 min. She was intubated and required inotropic support. Three days after resuscitation, she was extubated and then 2 days later received an implantable cardioverter-defibrillator. The patient made an uneventful recovery with no further recurrences of arrhythmias. This patient was also managed conservatively. Subsequent echocardiography repeated during her follow-up 3 months later showed no change compared with the last examination during hospitalization (Figure 3B, Videos 9 and 10). Clinical follow-up was uneventful.

All three of our patients were treated conservatively after discussion with our cardiothoracic surgeons, the patients, and their family members and remained hemodynamically stable. Furthermore, all three patients were also reluctant to undergo surgery.

DISCUSSION

IDH is infrequent complications of MI and the subsequent remodeling process of an ischemic dilated cardiomyopathy.[6] The underlying mechanism was thought to be hemorrhagic dissection among the spiral myocardial fibers, creating a neocavitation of the left ventricle enclosed by myocardium.[7] Usually, MI leads to myocardial rupture, which leads to massive infiltration and collection of blood into and through the myocardial wall. Pathologic descriptions most frequently demonstrate a linear or direct tear through the myocardium (79% of cases), whereas in the second type of rupture (9% of cases), there is infiltration of blood within the ventricular wall.[8]

In other words, IDH occurs when the LV muscle is separated by hemorrhagic channels. The endocardium and epicardium remain intact, but the hematoma is entrapped within the myocardium. The hematoma formation is most likely due to (1) rupture of intramyocardial vessels into the media, (2) decreased tensile strength of the infarcted area, and (3) acute increase of coronary capillary perfusion pressure.[5] The appearance of IDH is different from that of laminated thrombus, because in IDH there is a free space between the epicardial and endocardial layers, with hypoechogenicity of blood during the initial stage.

IDH is seen in patients with MI, severe thoracic injury (e.g., after a motor vehicle accident), or with the application of a stabilizer device for off-pump coronary revascularization. Previously, this complication has been difficult to diagnose and was usually diagnosed postmortem or intraoperatively. Differential diagnosis includes pseudoaneurysm, intracavitary thrombosis, and prominent ventricular trabeculations.[8] With the advent of multimodality noninvasive imaging techniques, it is possible to diagnose IDH earlier and with greater confidence. Echocardiographic diagnosis of septal and/or free wall IDH is based on the presence of at least three of the following signs: (1) the formation of one or more neocavitations within the tissue with an echo-lucent center, (2) a thinned and mobile endomyocardial border surrounding the cavitary defect, (3) ventricular myocardium identified in the regions outside of the cystic areas, (4) changes in the echogenicity of the neocavitation suggesting blood content, (5) partial or complete absorption of the cystic structure, (6) continuity between the dissecting hematoma and one of the ventricular cavities, (7) communication between the two ventricular chambers through the myocardial dissection, and (8) Doppler recording of flow within the dissected myocardium.[9]

The management of IDH is dependent on the size of the hematoma, hemodynamic stability, and comorbidities. Several reports have shown good prognosis and resolution in patients with fewer comorbidities, stable hemodynamics, and small hematomas.[9]

CONCLUSION

Echocardiography and other imaging modalities, such as magnetic resonance imaging and computed tomography, are useful in the diagnosis of IDH. In terms of management, conservative treatment is a viable option for patients with reduced LVEFs and dilated cardiomyopathy.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.case.2017.05.004.

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