Left Ventricular Intramyocardial Dissecting Hematoma

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INTRODUCTION

Myocardial dissection or intramyocardial dissecting hematoma (IDH) is a rare mechanical complication of myocardial infarction.[1] It is a hemorrhagic dissociation between the helical fibers of the myocardium that some studies describe as a type of incomplete cardiac rupture or a form of cardiac rupture. There are limited reports on the management of myocardial dissection, and the clinical outcomes vary from asymptomatic remission to cardiac death. Here we report a case of myocardial dissection diagnosed using echocardiography.

CASE PRESENTATION

A 69-year-old man had a 2-week history of persistent dyspnea on exertion and was aware of chest and back pain; however, he believed these symptoms were due to fatigue from work and ignored them. Three days before admission, he lost consciousness at work but awoke immediately. Subsequently, as the dyspnea worsened, he went to see his local doctor. He was transferred to our hospital because of ST elevation on a 12-lead electrocardiogram and positive troponin on blood tests. Past medical history includes a history of radiation therapy for prostate cancer and outpatient imaging follow-up for descending thoracic aortic aneurysm. He was taking antihypertensive medications and statins. He had no history of smoking or drinking, and no allergies were noted. His mother had died in her 70s due to cardiac disease.

Physical examination on admission revealed the following: height, 168 cm; weight, 75.4 kg; body mass index, 26.7 kg/m^2; blood pressure, 101/79 mm Hg; and pulse rate regular at 87 beats/minute. Lung auscultation was clear, and there was no heart murmur. No pedal edema was noted, and peripheries were warm to touch. A 12-lead electrocardiogram showed sinus rhythm with a heart rate of 89 beats/minute and ST-segment elevation with abnormal Q waves in V2-V5 with right bundle branch block, suggesting recent anterolateral myocardial infarction (Figure 1A). Chest radiography revealed a cardiothoracic ratio of 57% with slight pulmonary congestion and pleural effusion (Figure 1B). The levels of troponin and brain natriuretic peptide were elevated at 30.77 ng/mL (upper limit of normal, <0.04 ng/mL) and

1,163 pg/mL (upper limit of normal, <20 pg/mL), respectively. The creatinine level was 1.16 mg/dL, and the estimated glomerular filtration rate was 48.9 mL/minute/m^2, reflecting decreased renal function.

Urgent transthoracic echocardiography in the emergency room revealed left ventricular (LV) end-diastolic and end-systolic dimensions of 51 and 40 mm, respectively, and the LV ejection fraction was 25%, showing depressed LV systolic function. The anteroseptal and anterior-to-lateral walls were akinetic (Figure 2A and B). There was mild mitral regurgitation, but no other significant valvular disease was seen. No pericardial effusion was observed. The four-chamber view of the apex showed no obvious mobile thrombus in the heart, but there was a hypoechoic area from the septum to the apex, which was suspected to be a mural thrombus or hematoma (Figure 2C, Video 1). In short-axis image, a hypoechoic area in the anterior wall region indicated that the myocardium was moving outward in systole and inward in diastole (Video 2). There were no findings of blood inflow to the hypoechoic area of the LV apex on color Doppler imaging (Figure 3A and B, Video 3). When the parasternal long-axis image was tilted to look at the anterior papillary muscle side, a hypoechoic region was observed as seen in the short-axis view (Figure 4A and B, Video 4). There was no obvious shunt flow from the LV to the right ventricle (Video 5). We concluded that the findings reflected IDH (Table 1)[2] based on the presence of the following signs: (1) the formation of cavitation 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, and (4) changes in the echogenicity of the cavitation suggesting blood content. Cardiac gadolinium-enhanced magnetic resonance imaging showed extensive late gadolinium enhancement from the anterior wall to the septum and endocardial lateral wall, with hypointense areas consistent with the hypoechoic areas on echocardiography (Figure 5A and B, Video 6). Coronary angiography revealed complete occlusion of the left anterior descending branch segment 6 (Figure 6A). In addition, there was a 75% stenosis in segment 13 of the circumflex branch (Figure 6B) and mild disease over the right coronary artery (Figure 6C). On the basis of the results of these tests, we diagnosed subacute myocardial infarction and IDH. Fortunately, the hemodynamic status was maintained, and we decided to continue medical treatment for heart failure. While administering continuous intravenous heparin, we carefully followed up on the myocardial dissection with frequent echocardiographic examinations to determine whether it had progressed to cardiac rupture. It did not progress to cardiac rupture or ventricular septal perforation, and serial echocardiography revealed resolution of intracavitary thrombus on systemic anticoagulation. The hypoechoic area in the anterior wall region disappeared on echocardiography 1 month later, leaving an apical aneurysmal change of the left ventricle, and the LV ejection fraction was 14% (Figure 7A–C, Video 7). During hospitalization, sustained ventricular tachycardia occurred, necessitating cardioverter-defibrillator implantation. The patient was discharged on day 68 after medication adjustment and rehabilitation.

DISCUSSION

Intramyocardial dissecting hematomas are rare mechanical complications that occur after acute myocardial infarction or during the remodeling process. The mechanism is thought to be a hemorrhagic dissociation between the helical fibers of the myocardium, resulting in the formation of a new lumen surrounded by myocardium.[3,4]

Hematoma formation is thought to result from (1) the rupture of intramyocardial vessels into the tunica media, (2) decreased tensile strength of the infarct site, and (3) acute increase in perfusion pressure in the coronary capillaries.[1] It is most likely to appear within a few hours to a week after onset, but reports of IHD are limited to case reports and case series. Some studies have referred to it as a type of incomplete cardiac rupture or a form of cardiac rupture.[5,6]

The differential diagnoses for IDH include pseudoaneurysm, intracardiac thrombosis, and prominent ventricular trabeculations. The detection of LV apical thrombus is suggestive for Loeffler endocarditis when the myocardial contractility is preserved.[7] With the advent of multimodal noninvasive imaging techniques, IDH can now be diagnosed earlier and with greater reliability.[8,9] Echocardiographic diagnosis of septal and/or free wall IDH is based on the presence of at least three of the signs in Table 1.[2] In a hypoechoic area of the IHD region, the myocardium moves outward in systole and inward in diastole. Contrast agents should be used, if available, to improve endocardial delineation when LV endocardial segments are poorly visualized in apical views.[10,11] For rapid diagnosis, familiarity with echocardiography and multimodal cardiac imaging is essential, and these interventions and patient management should be performed by a multidisciplinary team involving cardiologists, radiologists, and cardiac surgeons.[12]

The management of IDH depends on multiple factors including the age of the patient, hemodynamic stability, size of the hematoma, presence of ventricular septal defect, LV function, and pericardial effusion. The independent risk factors for mortality were low ejection fraction (<35%), age ≥ 60 years, and anterior myocardial infarction.[13] Intramyocardial dissecting hematoma limited to the apex has a high probability of spontaneous reabsorption, and an initial conservative approach may be reasonable.[2] In the absence of a complete tear of the myocardium, conservative management is the recommendation of choice because of the high risk of surgery and the possibility that the myocardium may heal over time. Surgical repair should be considered in cases with rapid progression, progression to cardiac rupture, or the need for surgical revascularization. Anticoagulation
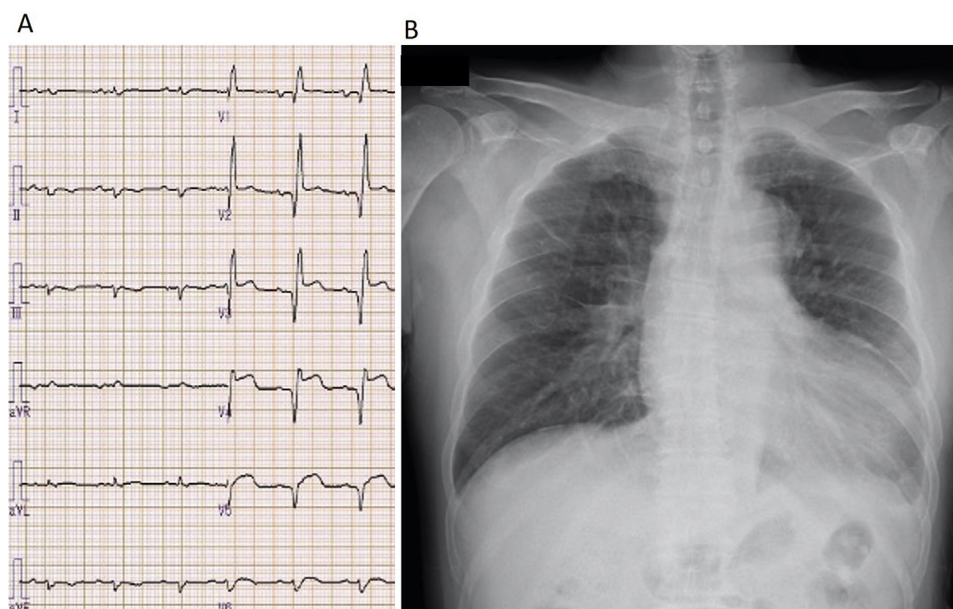

Figure 1 (A) A 12-lead electrocardiogram showed sinus rhythm with a heart rate of 89 beats/minute and ST-segment elevation with abnormal Q waves in V2-V5 with right bundle branch block. **(B)** Chest radiography revealed a cardiothoracic ratio of 57% with slight pulmonary congestion and pleural effusion.
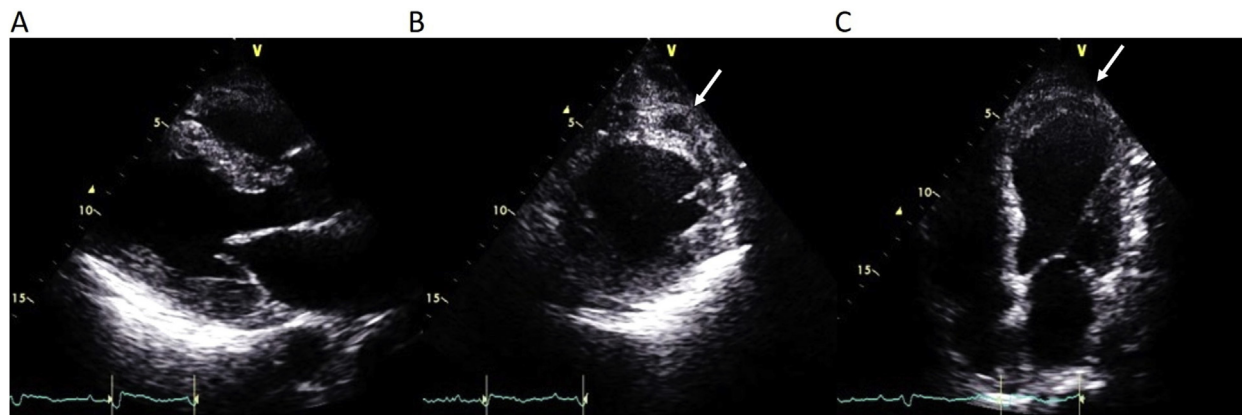
Figure 2 **(A)** Parasternal and **(B)** short-axis view of the left ventricle revealed the anteroseptal and anterior-to-lateral walls were akinetic. **(C)** The four-chamber view of the apex showed no obvious mobile thrombus in the heart, but there was a hypoechoic area from the septum to the apex (*arrows*).
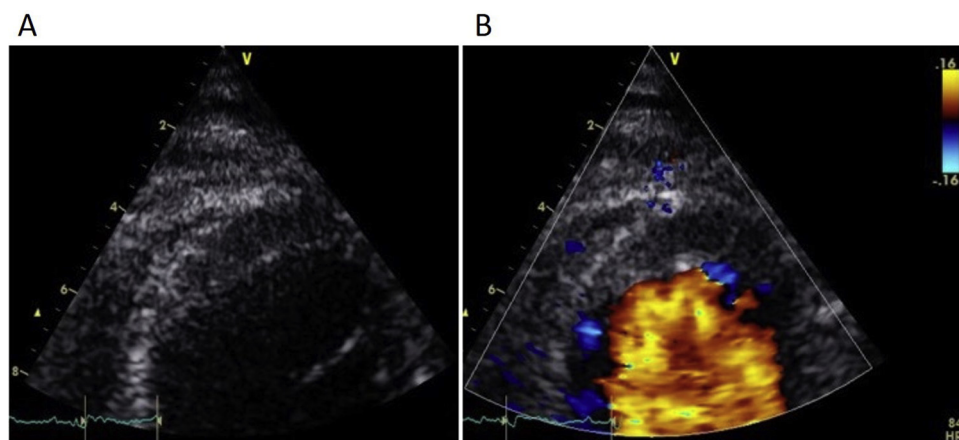

Figure 3 The four-chamber view of the LV apex showed no obvious mobile thrombus **(A)**. There were no findings of blood inflow to the hypoechoic area of the LV apex on color Doppler imaging **(B)**.
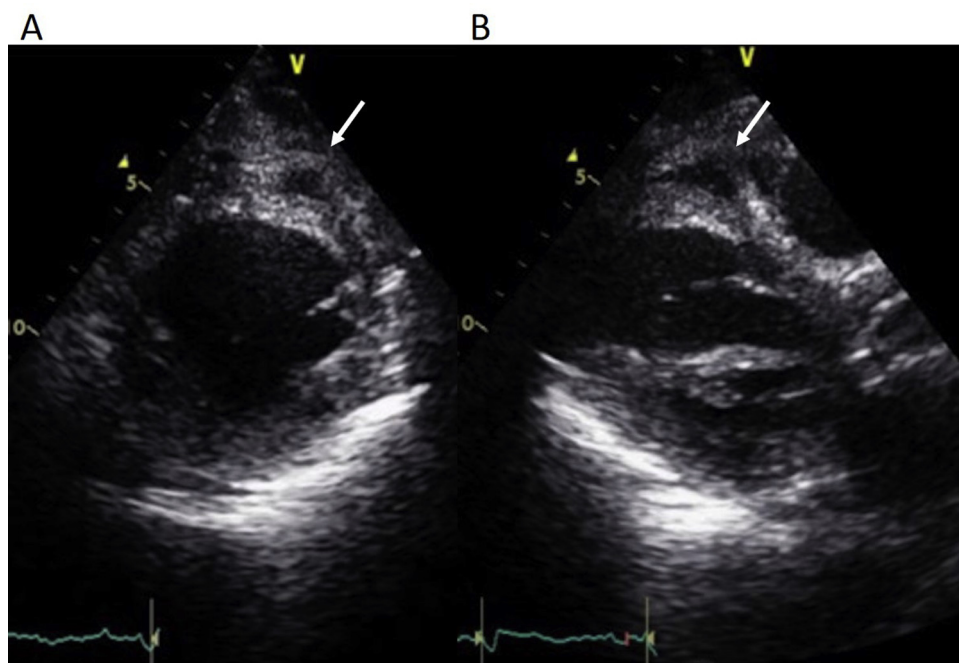

Figure 4 In short-axis image **(A)**, there was a hypoechoic area in the septum to anterior wall region. When the parasternal long-axis image was tilted to look at the anterior papillary muscle side **(B)**, a hypoechoic region (*arrow*) was observed as seen in the short-axis image.

Table 1 Signs in echocardiographic diagnosis of septal and/or free wall IDH

The formation of one or more neocavitations within the tissue with an echo-lucent center
A thinned and mobile endomyocardial border surrounding the cavitary defect
Ventricular myocardium identified in the regions outside of the cystic areas
Changes in the echogenicity of the neocavitation suggesting blood content
Partial or complete absorption of the cystic structure
Continuity between the dissecting hematoma and one of the ventricular cavities
Communication between the two ventricular chambers through the myocardial dissection
Doppler recording of flow within the dissected myocardium

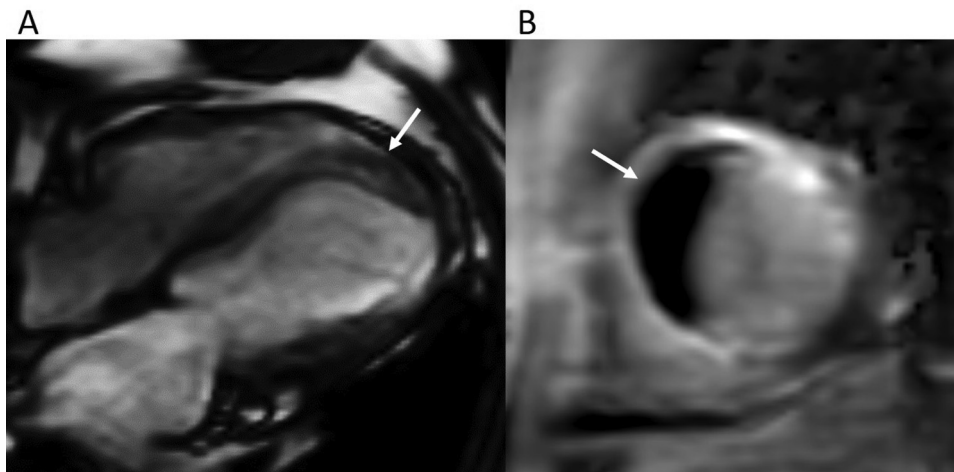IDH can be diagnosed based on the presence of at least three of the signs.[2]

Figure 5 Cardiovascular magnetic resonance imaging (CMR) from four-chamber **(A)** and mid-LV level of short-axis **(B)** views after gadolinium injection. Gadolinium-enhanced CMR showed extensive late gadolinium enhancement from the anterior wall to the septum, with hypointense areas consistent with the hypoechogenic areas (*arrow*) on echocardiography.
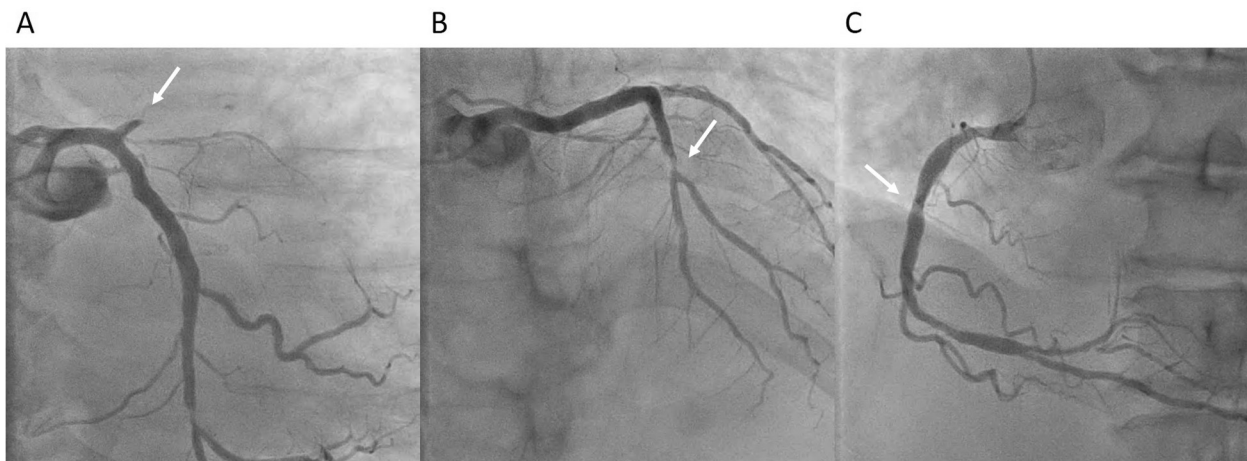

Figure 6 **(A)** Coronary angiography reveals complete occlusion of the left anterior descending branch segment 6 (*arrow*). **(B)** There is a 75% stenosis in segment 13 of the circumflex branch (*arrow*) and **(C)** mild disease (*arrow*) over the right coronary artery.
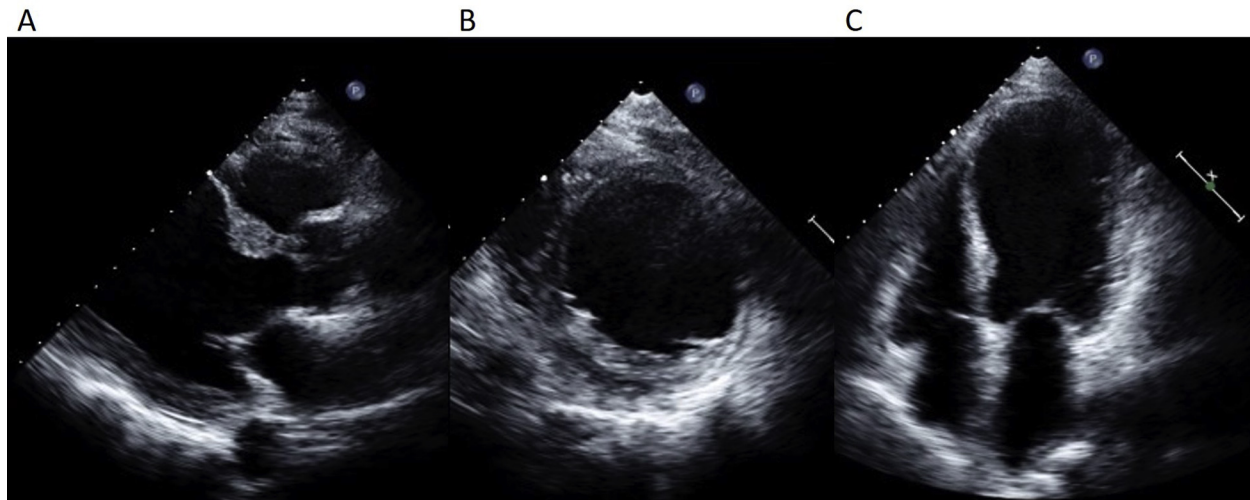
Figure 7 The hematoma gradually and spontaneously regressed, leaving an apical aneurysmal change of the LV in the parasternal long-axis **(A)**, short-axis **(B)** and apical four-chamber images **(C)**.

can be considered to reduce the risk of thromboembolism from within the left ventricle, but its use must be balanced against the risk of progression of dissection and bleeding into the pericardial space. Patients with IDH are at high risk of ongoing morbidity and mortality, including ventricular arrhythmias due to scarred myocardium, recurrent congestive heart failure, reduced cardiac output, LV remodeling and development of functional mitral regurgitation, and sudden cardiac death.[14] In the current case, the hemodynamic status was maintained without cardiac rupture; therefore, conservative treatment was sufficient, and antithrombotic therapy was continued with frequent echocardiographic follow-up. The patient developed ventricular arrhythmia but was discharged after implantable cardioverter-defibrillator implantation.

CONCLUSION

We encountered a case in which IDH was diagnosed using two-dimensional echocardiography. In the IDH region, there is formation of cavitation within the tissue with an echo-lucent center and the myocardium moves outward in systole and inward in diastole. Intramyocardial dissecting hematoma is a rare complication following acute myocardial infarction, and it is important to diagnose it using multimodal imaging, including echocardiography, and to frequently evaluate the status of the dissection and hematoma by echocardiography to enable the provision of appropriate treatment.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.case.2021.07.016.

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