

Early Spontaneous Remission of Intramyocardial Dissecting Hematoma

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Abstract

Intramyocardial dissecting hematoma is a rare but potentially fatal complication of myocardial infarction. The decision to adopt a surgical or conservative strategy may depend on the clinical and hemodynamic stability of patients. Regardless, the precise and temporal assessment of the structure of hematoma is imperative. We herein report the first case of a patient with early spontaneous remission of intramyocardial dissecting hematoma successfully managed by a conservative approach with multimodality imaging.

Key words: intramyocardial dissecting hematoma, complication, acute myocardial infarction

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Introduction

Intramyocardial dissecting hematoma (IDH) is an unusual form of myocardial rupture that can occur as a complication of myocardial infarction. Although conservative treatment may be chosen in some cases with hemodynamic stability, the temporal changes in the hematoma vary from patient to patient. We herein report the first case of IDH, with serial assessment of early spontaneous remission obtained by multimodality imaging.

Case Report

A 51-year-old man with a history of hypertension, type 2 diabetes mellitus, and smoking (30 pack-years) presented with dyspnea on effort and nocturnal cough for 3 weeks. His complaints started after a sudden onset of chest discomfort with cold sweat and vomiting, and gradually worsened.

Twelve-lead electrocardiography showed a QS pattern, poor R progression, and ST elevation with inverted T waves in the anterior precordial leads. Chest radiograph showed cardiac enlargement and pulmonary congestion. The levels of brain natriuretic peptide and troponin I were elevated

(1,653.7 pg/mL and 0.24 ng/mL, respectively), while the creatinine kinase level was normal (195 IU/L). These data were consistent with a diagnosis of congestive heart failure due to a recent anterior myocardial infarction.

Transthoracic echocardiogram at admission revealed a left ventricular ejection fraction of 19% with thinning and akinesis of the apical, anterior, and septal walls. The four-chamber view demonstrated a pulsatile mass with heterogeneous echogenicity at the infarcted apical-septal region (Fig. 1A, Movie S1). On a color-doppler examination, no significant flow was detected in the mass. The mass was interpreted as a subacute intramural thrombus, and combination therapy with an anticoagulant and an antiplatelet agent was started.

However, an echocardiogram on day 3 after admission showed apical mass enlargement with considerable movement, and the uninterrupted endocardium appeared to delineate the mass on the left ventricular cavity (Fig. 1B). Follow-up echocardiogram images led to the diagnosis of IDH.

Cardiac magnetic resonance imaging (MRI) was performed for further evaluation. High-intensity myocardium on T2-weighted images and delayed enhancement on late gadolinium-enhanced MRI in the antero-septal region re-

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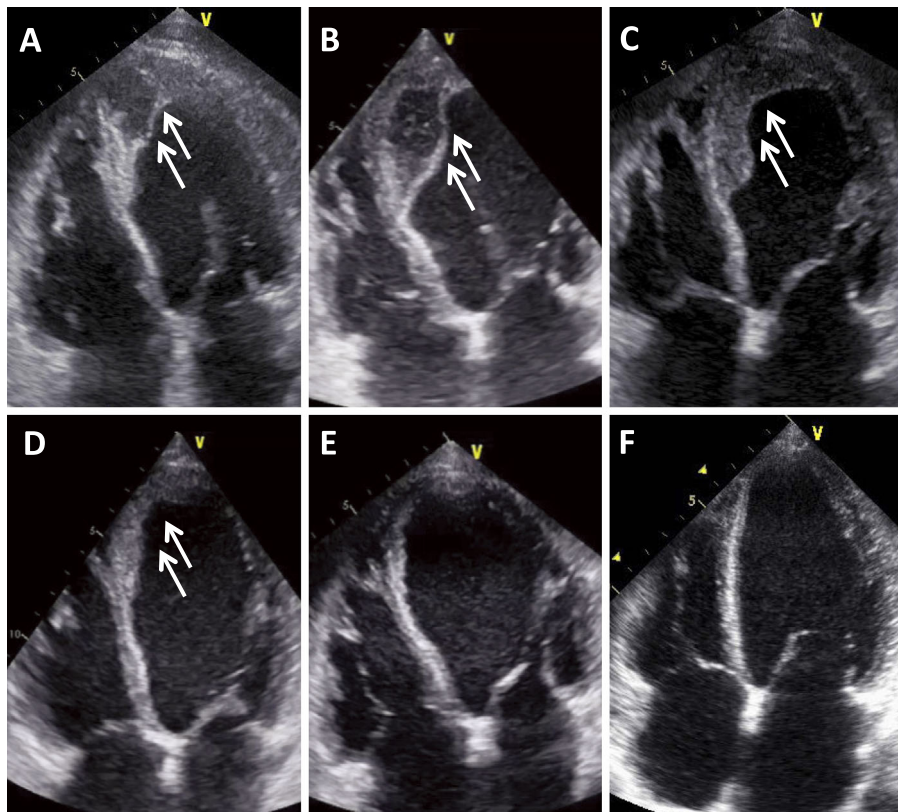


Figure 1. A series of follow-up transthoracic echocardiographic images of an intramyocardial dissecting hematoma (white arrows) in a four-chamber view. (A) On admission. (B) At Day 3, because of the enlargement of the hematoma, antithrombotic agents were discontinued. (C and D) At Day 9 and 15, gradual remission of the hematoma was seen. (E) At Day 23 (before coronary artery bypass grafting surgery), the hematoma had disappeared. (F) One month after coronary artery bypass grafting, no recurrence of the hematoma was seen.

flected recent myocardial infarction. Akinetic motion and wall thinning in the infarcted area were similar to the findings on echocardiogram. High-intensity areas on both dark-blood T1 and T2 images at the apex were consistent with a relatively new and unorganized hematoma. T2-weighted images demonstrated an endocardial layer and an infarcted myocardium, which delineated the hematoma between the left ventricular cavity and epicardial border (Fig. 2A). On cine MRI with T2 images using true fast imaging with a steady-state precession sequence, the delineated hematoma and its pulsatile motion with systolic expansion were clearly shown (Movie S2). The endocardial layer of the hematoma was uninterrupted and smoothly continued to the low-intensity endocardium in the mid-septal and lateral region. Furthermore, late gadolinium-enhanced MRI using phase-sensitive inversion recovery revealed the enhanced myocardium on the side of not only the epicardium but also the endocardium (Fig. 2B). These findings suggested that the hematoma was located within the infarcted myocardium and validated our diagnosis of IDH.

Because the patient's hemodynamic status was stable and there was neither an endomyocardial tearing nor a left ventricular pseudoaneurysm with IDH, we initially adopted a conservative approach by discontinuing the antithrombotic

agent to prevent enlargement of the hematoma. Thereafter, the hematoma gradually shrank in size each day, and we finally confirmed its disappearance on Day 23 after admission (Fig. 1C-E).

Coronary angiography revealed a 90% stenosis with a radiolucent area in the proximal portion of the left anterior descending artery and a 90% stenosis in the middle portion of the left circumflex artery and the proximal portion of the right coronary artery. Collateral flow into left anterior descending artery was not detected. There was no feeding artery to the hematoma. The patient underwent coronary artery bypass grafting on Day 25 after admission and was discharged in an ambulatory state on the 10th postoperative day. There was no recurrence of the hematoma even after the antiplatelet therapy was restarted (Fig. 1F, 2C-D).

Discussion

IDH is an infrequent form of myocardial rupture, complicated by myocardial infarction, trauma, and heart surgery (1, 2). Rupture of injured microvasculature into the interstitium, embrittlement of the infarcted myocardium, and increased coronary capillary perfusion pressure due to microvascular obstruction are considered to be underlying

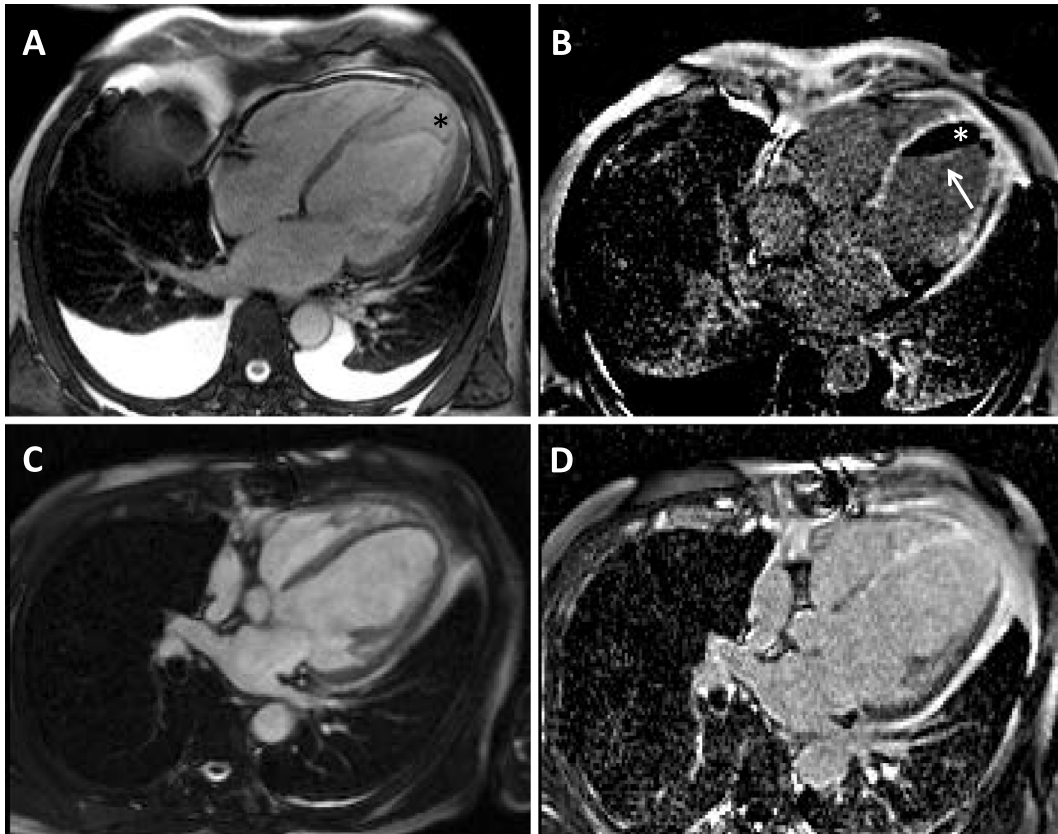


Figure 2. Cardiac magnetic resonance views in a four-chamber orientation on Day 3 (A and B) and one month after coronary artery bypass grafting (CABG) (C and D) using T2-weighted imaging (left) and late-gadolinium enhancement (right). (A) The hematoma (asterisks) was surrounded by the non-ruptured endocardial flap as the inner layer and by the infarcted myocardium with high intensity at the apical-septal lesion as the outer layer. (B) The infarcted myocardium was brightly enhanced, and the enhancement was also seen on the side of the inner layer (white arrow). (C and D) There were no findings indicative of recurrence of the hematoma.

mechanisms (3, 4). The differential diagnosis includes intramural thrombus, pseudoaneurysm, and ventricular trabeculations. A clear identification of the endocardial and epicardial layers surrounding the hematoma is very important to establish a diagnosis of IDH (2, 5). Echocardiogram is a noninvasive and repeatable examination. However, in this case, cardiac MRI provided a more precise structural image of IDH, complementing the echocardiographic diagnosis.

The prognosis of IDH might depend on the clinical condition and stability of the patient, and IDH itself. Surgical treatment of IDH has been reported to achieve good outcomes (1). However, there are growing reports of successful medical management with or without spontaneous remission of IDH (3, 6, 7). As seen in this case, early spontaneous remission might be achievable by adopting a conservative approach in cases of IDH without any flow between other spaces, including the ventricular cavity and pseudoaneurysm. Furthermore, if antithrombotic medicine is strongly suspected of causing the enlargement of hematoma, as in this case, these agents should be discontinued until the hematoma is resolved.

IDH is a rare but potentially fatal complication of myocardial infarction. Both a precise structural understanding

and serial assessment with multi-modality imaging are essential for the management of IDH.

The authors state that they have no Conflict of Interest (COI).

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