Newly developed intramyocardial dissection hematoma: a rare complication of myocardial infarction

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Abstract

Intramyocardial dissection hematoma (IDH) is an extremely rare complication of myocardial infarction (MI). Echocardiography is the initial imaging modality for IDH in patients with MI. We reported the case of a 74-year-old patient admitted because of abdominal pain and remarkably increased white cell count. He had anterior-wall MI and underwent percutaneous device intervention 8-months ago, his B-cell acute lymphoblastic leukemia (ALL) was recently diagnosed. Two dimensional echocardiography demonstrated decreased lower-anterior interventricular septum akinesis with the formation of an apical left ventricular aneurysm, and a cavitation with echolucent center formed within the tissue of the apical area was detected. Three-dimensional echocardiography with the true-view effect displayed a clearer image of dissecting flap resembling a sheet of tissue and fibers and bundles in the cavitation suggested dissected myocardium. Compared to the echocardiography performed 3-days ago in his community hospital, the intimal flap with the cavitary lesion in the apical region was newly developed. Therefore, the diagnosis of newly developed IDH was made. Considering hematological malignancy and hemodynamic instability, the patient was managed with conservative pharmacological treatments.

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INTRODUCTION

The left ventricle (LV) intramyocardial dissection hematoma (IDH) or hemorrhagic dissection that is not well-documented due to its scarcity is an extremely rare sequela of myocardial infarction, percutaneous device intervention, or trauma. It is often speculated that pathophysiological mechanism is due to intramyocardial blood vessel rupture into the adjacent spiral fibers of the myocardium although the exact underlying mechanism is unknown. The presenting symptoms of IDH varies based on limited number of existing literatures, and how to manage the therapeutic interventions of IDH remains controversial as well. Hereby, we present a coincidently detected case of IDH who were admitted due to initial presentation of abdominal pain and remarkably increased white cell count (WBC).

CASE DESCRIPTION

A 74-year-old man who had persistent upper lower-abdominal pain and remarkably increased white blood cell count (WBC) for a week without fever was admitted to our hospital. The patient had a known history of coronary artery disease (CAD), anterior-wall myocardial infarction (MI), hypertension, and heart failure (HF). Prior to the admission, the patient underwent percutaneous coronary intervention due to CAD and anterior-wall MI 8-months ago. After that, patients were frequently present with exertional dyspnea explained by the reduced left ventricular (LV) ejection fraction (LVEF). Otherwise, the patient was absent of chest pain, headache, dizziness, or cough on admission.

Moreover, the physical exam of the patient on admission was as follows: blood pressure 140/90 mmHg; heart rate 82 bpm; New York Heart association (NYHA) functional class III-IV. Cardiopulmonary auscultations were not prominent. Furthermore, twelve-lead electrocardiogram demonstrated ectopic rhythm, elevated ST-segment with abnormal Q wave in leads V1–V4, suggesting previous anterior wall MI (Fig 1a).

Laboratory test on admission revealed the following: WBC count 11.2x10^9/L (62% immature cells, normal reference range [3.5-9.5]x10^9/L); Neutrophil 1.17x10^9/L; lymphocyte 9.4x10^9/L; hemoglobin 107 g/L; platelets 28x10^9/L; D-dimer 3671 ng/mL, terminal pro-b-type natriuretic peptide 2414 pg/mL (<125 pg/mL). Subsequently, the patient’s bone marrow biopsy (Fig 1b) and abdominal computed tomography (CT) confirmed B-cell acute lymphoblastic leukemia (ALL) and splenic infarction, respectively.
Two-dimensional echocardiography demonstrated LV systolic dysfunction (EF 39%) as well as the apical and lower-anterior interventricular septum akinesis with the formation of an apical left ventricular aneurysm. Besides, a cavitation with echolucent center formed within the tissue of the apical area was detected (Fig 2). Moreover, the single mobile, linear-shaped endocardial border with a noticeable tear was surrounding the cavitation in the apical region (Video 1). When further Doppler echocardiography was applied to the cavitary lesion, laminar flow communication between the cavitary lesion and true left ventricle in the apical region was detected. Bearing all these findings in mind, three-dimensional echocardiography zooming into the left ventricle with the true-view effect provided a clearer image of dissecting flap resembling a sheet of tissue, additionally, fibers and bundles in the cavitation suggested dissected myocardium was clearly observed (Fig 3, Video 2). Besides, the intimal flap with the cavitary lesion in the apical region was not present based on the reports of the echocardiography performed 3-days ago in his community hospital, suggesting above mentioned findings in the apex rather newly developed within these days. Lastly, bi-atrial enlargement with mild mitral and moderate tricuspid regurgitation with increased pulmonary arterial systolic pressure (45mmHg) should also be noted. Taken all together, the diagnosis of LV intramyocardial dissection hematoma (IDH) was confirmed. Considering hematological malignancy and hemodynamic instability, the patient was managed with conservative pharmacological treatments.

Despite exhaustive medical and supportive management, his condition progressively deteriorated. Death as a consequence of LV failure occurred 40 days after admission.

DISCUSSION

The IDH or hemorrhagic dissection that is not well-documented due to its scarcity is an extremely rare sequela of percutaneous device intervention or trauma next to MI, which accounted for nearly 1% of complications of MI. It is often speculated that pathophysiological mechanism is due to intramyocardial blood vessel rupture into the adjacent spiral fibers of the myocardium resulting in decreased tensile force, subsequently increased tissue fragility of the infarcted area, and abruptly increased coronary capillary perfusing pressure, which might overall contribute to the formation of IDH. Although the exact underlying mechanism is still not well-established. Echocardiography remains the initial imaging modality for cardiac assessment in suspected patients with IDH due to its advantage of convenience, non-invasiveness, and cost-effectiveness. Until recently, most commonly recommended echocardiographic standards of IDH were first proposed and summarized by Vergas-Barron et al in 2008. And, echocardiographic hallmarks of IDH based on the existing cardiac remodeling resulting from MI, included echo-lucent centered neo-cavitation with or without alterations of echogenicity, a mobile endomyocardial border that communicates true versus false ventricle surrounding the cavitation, and associated doppler signal communicating between true and false ventricles, etc, which are all in line with echocardiographic findings of the current case report. Nevertheless, pseudoaneurysm, intracavitary thrombose, and prominent trabeculations as differential diagnosis of IDH should be well-recognized. Apart from echocardiography, the diagnostic approach of multimodality-imaging involving cardiac CT or magnetic resonance as the gold standard for the IDH diagnosis is reassured. However, multimodality-imaging approach in real clinical practice is often challenging due to extra cost and hemodynamic instability of the patients. Thereby, other imaging modality was not performed on our patient mainly due to hemodynamical instability.

Conservative management involving anticoagulant therapy and targeting the ongoing morbidity of the patients is the therapeutic mainstream of IDH. As the majority of patients with IDH were present with more or less hemodynamic instability. Particularly, for patients with relatively small IDH limited to apex, it is likely that the IDH might resolve itself via conservative management alone. However, if patients were present with signs of exacerbated progression towards cardiac rupture in the absence of cardiogenic shock, surgical intervention should be recommended. As such, cardiovascular conservative management was kept on the patient while waiting for the chemotherapy initiation in the current case. Although anticoagulant therapy was not given to the patient taking ALL related low platelets into account. However, the causal-effect relationship between B-cell ALL with IDH in the current case was precluded. Whether B-cell ALL contribute
to the pathogenesis of IDH or not, and how the B-cell ALL modify the future prognosis of IDH is unknown at the moment. Even though, a prior meta-analysis of summarizing existing 42 cases with IDH showed that LVEF < 35%, age ≥ 60 years, anterior-wall MI were associated with increased mortality in patients with IDH irrespective of surgical or conservative management. Nevertheless, the current case illustrated that IDH might occur in patients with MI after long-term follow-up despite its rare incident rate. Especially, when it is complicated with HF or malignant conditions, clinical presentation might not be prominent. In this regard, combined two- and three-dimensional echocardiography might play a crucial role in aspects of the differential diagnosis, management, and follow-up the patients with IDH.

Disclosures
Conflicts of interest: None of the authors have any conflicts of interest to report.

REFERENCES


Figure legends

Fig 1. a. 12-lead electrocardiogram shows ectopic rhythm, elevated ST-segment with abnormal Q wave in leads V1–V4, b. Diagnostic bone marrow smear shows several lymphoblasts with a high nuclear cytoplasmatic ratio and variably condensed chromatin.

Fig 2. Two-dimensional echocardiography shows the cavitation in the apical area and the linear-shaped endocardial border (red arrow) with small tear (white arrow).

Fig 3. Three-dimensional echocardiography with true-view effect observes the long intima tear, fibers and bundles in the cavitation.