MINI-FOCUS ISSUE: IMAGING

ADVANCED

CASE REPORT: CLINICAL CASE

Left Ventricular Intramyocardial Dissecting Hematomas



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ABSTRACT

Intramyocardial dissection is a rare complication of myocardial infarction, trauma, and percutaneous intervention. It is usually caused by hemorrhagic dissection among the spiral myocardial fibers. We hereby report the case of a patient with left ventricular intramyocardial dissection who presented with acute decompensated heart failure. (Level of Difficulty: Advanced.) (J Am Coll Cardiol Case Rep 2021;3:94–8) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

45-year-old man, an active tobacco smoker with a known history of diabetes mellitus and hypertension, presented with a recent

onset of New York Heart Association (NYHA) functional class IV heart failure. He had sudden-onset chest discomfort 1 month before, which resolved spontaneously within a day.

LEARNING OBJECTIVES

- To recognize that intramyocardial dissecting hematoma is a rare complication of myocardial infarction.
- To understand that delayed presentation can mimic left-ventricular clot on transthoracic echocardiogram.
- To comprehend that the distinction from intracavitary thrombus relies on the clear identification of the endocardial layer surrounding the neoformation and its systolic expansion.
- To realize that management of IDH depends on multiple factors including age of the patient, hemodynamic stability, size of hematoma, presence of ventricular septal defect, LV function, and pericardial effusion.

PHYSICAL EXAMINATION

The patient had tachycardia (pulse rate of 120 beats/min) with low-volume pulse, blood pressure of 80/60 mm Hg, elevated jugular venous pressure (prominent "a" and "v" waves), anasarca, S3 gallop, and hepatomegaly. There was no significant medical history relevant to the current presentation.

DIFFERENTIAL DIAGNOSIS

In view of his history of chest pain 1 month earlier, we kept the possibility of an acute coronary syndrome, now presenting with decompensated heart failure, as our first differential diagnosis. Another possible differential diagnosis could have been a longstanding ischemic cardiomyopathy with severe biventricular dysfunction.

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Manuscript received April 1, 2020; revised manuscript received July 7, 2020, accepted July 21, 2020.

INVESTIGATIONS

The electrocardiogram (ECG) showed sinus rhythm with right bundle branch block and low-voltage QRS complexes in limb leads and T inversion in V₁ to V₆ (Figure 1).Two-dimensional echocardiography showed left-ventricular (LV) dysfunction (ejection fraction [EF] ~25%) with akinetic interventricular septum (IVS). There was a large intramyocardial dissecting hematoma (IDH) within the LV cavity, with a mobile endocardial flap (Figures 2 to 5, Videos 1, 2, 3, 4, 5, and 6). The 3-dimensional echocardiogram delineated the mobile endocardial flap and dissected myocardial flap clearly (Figure 6). The contrast (Definity, Lantheus Medical Imaging, North Bellerica, Massachusetts) echo opacified the true LV cavity, with no leak of contrast into the neocavity (Figure 7, Video 7). Cine magnetic resonance (MR) sequences confirmed the diagnosis of intramyocardial dissection with multiple layers of hematoma and thrombus (Figures 8 and 9, Videos 8 and 9).

MANAGEMENT

Our final diagnosis was LV dissection with intramyocardial hematoma and severe LV dysfunction with acute decompensated heart failure. After initial stabilization with inotropes and diuretics, the heart team was consulted. Owing to the complexity of dissection and a prohibitive risk for surgery (EuroSCORE II 65.8%), hematoma evacuation and ventricular wall repair would have been challenging, and therefore the patient was kept on conservative management. He was discharged 2 weeks later on diuretics, ramipril, atorvastatin, and a single antiplatelet (aspirin).

DISCUSSION

IDH is a rare complication of myocardial infarction (MI), chest trauma, and percutaneous intervention. It can develop in the LV free wall, the right ventricle, or the IVS (1). 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 (2).

IDH consists of a cavity filled with blood, the outer wall of which is the myocardium and pericardium, and the inner wall, facing the ventricular cavity, is part of the myocardium and endocardium. Differential diagnosis includes pseudoaneurysm, intracavitary thrombus, or prominent ventricular

ND ACRONYMS

EF = eiection fraction

IDH = intramyocardial dissecting hematoma

IHD = ischemic heart disease

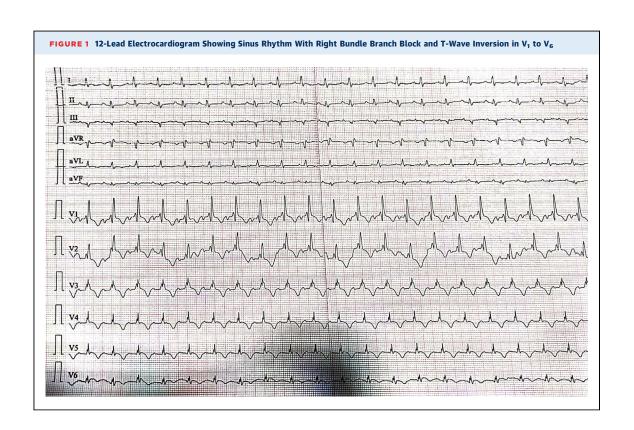
IVS = interventricular septum

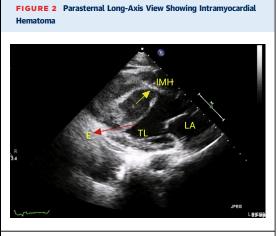
LV = left ventricle

MI = myocardial infarction

MR = magnetic resonance

NYHA = New York Heart Association





E = endocardial flap; IMH = intramyocardial hematoma; LA = left atrium; TL = true left-ventricular cavity.

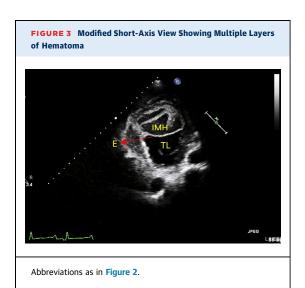
M = myocardial flap; other abbreviations as in Figure 2.

trabeculations. Establishing the integrity of epicardium differentiates IDH from pseudoaneurysm, as pseudoaneurysm comprises a complete rupture of the myocardial wall contained by the pericardium. The distinction from intracavitary thrombus relies on the clear identification of the endocardial layer surrounding the neoformation and its systolic expansion. A completely irregular shape of the ventricular wall with flow within it is the hallmark of prominent trabeculations (3).

In our patient, the most likely cause of LV dissection was MI, as he had multiple risk factors (diabetes, hypertension, and smoking) for ischemic heart disease (IHD). The finding of akinetic anterior wall segments on echo and cardiac MR confirmed our diagnosis of IHD.

In more than 90% of cases, rupture occurs after the first MI and has a strong correlation with single-vessel disease, reflecting lack of collateral circulation (4). Risk factors include anterior-wall infarct, large transmural infarction, age ≥60 years, hypertension, female sex, single-vessel disease, and absence of previous cardiac events (5).

Echocardiographic features of IDH include the formation of 1 or more neocavitations within the tissue with an echolucent center; a thinned and mobile endomyocardial border surrounding the cavitary defect; ventricular myocardium identified in the regions outside of the cystic areas; andchanges in the echogenicity of the neocavitation, suggesting blood content (6).



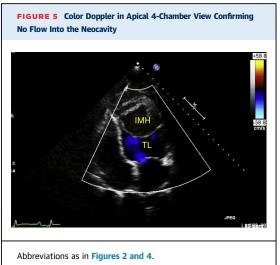
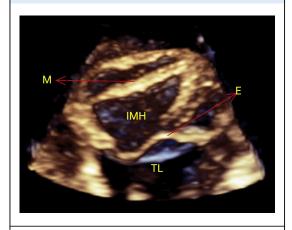


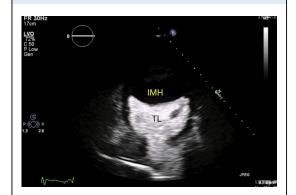
FIGURE 6 3-Dimensional Echo Showing the Mobile Endocardial Flap With Intramyocardial Dissecting Hematoma



Abbreviations as in Figures 2 and 4.

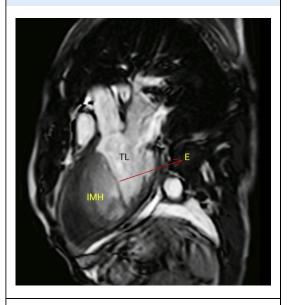
The management of IDH depends on multiple factors including age of the patient, hemodynamic stability, size of the hematoma, presence of ventricular septal defect, LV function, and pericardial effusion. IDH limited to the apex has a high probability of spontaneous reabsorption, and an initial conservative approach may be reasonable (6). Those patients with expansion of dissection on serial echocardiogram; ventricular septal defect; and with compromised hemodynamics, and low EF in anterior wall MI should undergo surgery (7). There have been case reports of surgical management that involved median sternotomy, cardiopulmonary bypass, and clot evacuation with subsequent ventricular wall repair; however, such procedures were done in patients who were not

FIGURE 7 Contrast Echo Differentiating the True Left Ventricular Cavity From the Neocavity



Abbreviations as in Figure 2.

FIGURE 8 Cardiac Magnetic Resonance Demonstrating Intramyocardial Dissecting Hematoma With Thrombus



Abbreviations as in Figure 2.

in cardiogenic shock (8). In our case, although the patient presented in acute decompensated heart failure with severe LV dysfunction, he had significant hemodynamic improvement with conservative management, and, also, such an extensive dissecting hematoma was beyond repair.

In a case series by Leitman et al. (7) published in 2018, 42 cases of IDH have been diagnosed and published thus far. In this series, in-hospital mortality was 23%, and the strongest independent risk factors for mortality were low EF (<35%), age \geq 60 years, and anterior-wall MI. Late presentation (>24 h after onset of symptoms) was also associated with increased mortality.

FOLLOW-UP

At present, at 6 months of follow-up, the patient is symptomatically better (NYHA functional class II), with no further extension of the dissecting hematoma.

CONCLUSIONS

IDH is a rare complication of MI and a diagnostic challenge. Management is based on individual clinical and imaging parameters. Low EF, age ≥60 years, anterior-wall MI, cardiogenic shock, and late diagnosis are predictors of in-hospital mortality. Conservative management is the usual approach in cases with small hematomas localized to the apex, whereas cases with hemodynamic instability and with expanding hematomas may need surgical repair.

FIGURE 9 Late Gadolinium Enhancement Magnetic Resonance Showing the Extensive Myocardial Scar

LGE
IMH
IMH

LGE = late gadolinium enhancement; other abbreviations as in Figures 2 and 8.

ACKNOWLEDGMENTS The authors thank Dr. K.H. Srinivas and Dr. Prabhavathi for their guidance and Dr. C.N. Manjunath for funding this paper with the institute's research grant.

AUTHOR DISCLOSURES

This work was funded by a research grant from Sri Jayadeva Institute of Cardiovascular Sciences & Research, Bengaluru, Karnataka, India.

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS acute heart failure, cardiac magnetic resonance, contrast agent, dissection, myocardial infarction

APPENDIX For supplemental videos, please see the online version of this paper.