JACC: CASE REPORTS VOL. 4, NO. 21, 2022

CASE REPORT

INTERMEDIATE

CLINICAL CASE SERIES

Intramyocardial Dissecting Hematoma

A Mechanical Complication Needing Surgical Therapy?

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ABSTRACT

Intramyocardial dissecting hematoma is a form of cardiac rupture caused by myocardial infarction, percutaneous coronary intervention, or trauma. It is a cavity between myocardial fibers caused by partial rupture of the ventricular wall. Therapeutic management, including the timing for surgical approach, has not been standardized. We present a case series describing 4 patients. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:1443–1448)

Intramyocardial dissecting hematoma (IDH) is the partial rupture of the ventricular wall forming a cavity filled with blood between the myocardial layers. It is a rare complication of acute myocardial infarction, and it can progress to complete myocardial rupture.[1] Transthoracic echocardiography (TTE) plays a role in its early diagnosis and differentiation from mural thrombus and myocardial pseudoaneurysm. Recently, a multi-imaging approach with cardiac tomography (CT) and cardiac magnetic resonance has proved to be useful for diagnosis.[2-4]

We describe a series of 4 IDH cases registered in our hospital during the past 7 years. Among a contemporary series of 814 patients with ST-segment elevation myocardial infarction (STEMI), only 3 patients presented with IDH associated with STEMI (0.37%).

LEARNING OBJECTIVES

- To recognize that IDH is a rare complication of myocardial infarction with high mortality.
- To clarify that a differential diagnosis should be made with pseudoaneurysm and mural thrombus.
- To acknowledge the use of multiple imaging techniques as an appropriate approach for confirmation and differential diagnosis.
- To recognize that hemodynamic instability, pericardial effusion, and dissection growth should be indications for urgent surgical treatment.

CASE DESCRIPTIONS AND CLINICAL MANAGEMENT

PATIENT 1. A 60-year-old man, an active smoker and with a history of hypertension, presented after 48 hours of a nonreperfused anterior STEMI with recent onset of New York Heart Association functional class III heart failure. Physical examination revealed pulse rate of 103 beats/min, blood pressure 130/80 mm Hg, and elevated jugular venous pressure with S3 gallop.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received May 9, 2022; revised manuscript received July 7, 2022, accepted July 13, 2022.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2022.07.025

ABBREVIATIONS AND ACRONYMS

CT = computed tomography

IDH = intramyocardial dissecting hematoma

STEMI = ST-segment elevation myocardial infarction

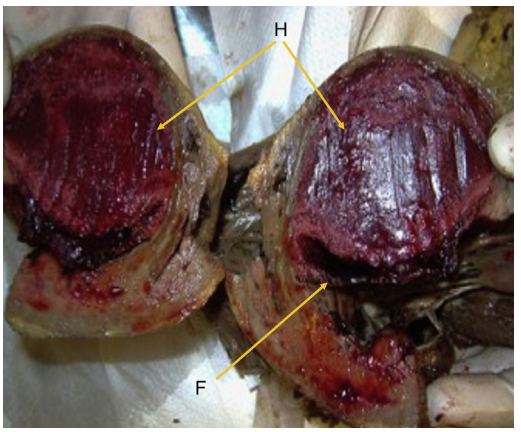
TTE = transthoracic echocardiography

An electrocardiogram showed sinus rhythm and Q-wave from leads V_1 to V_6, and coronary angiography evidenced occlusion of the anterior descending artery. TTE exhibited anterior wall akinesia and intramyocardial dissection flap involving apical segments and the anterior and inferior septum, defining a neocavity with images compatible with thrombus (Figure 1). Total rupture of the ventricular wall was ruled out because of the absence of pericardial effusion.

A conservative approach was decided owing to clinical stability. On the fourth day of hospitalization, the patient experienced ventricular tachycardia, which was electrically reversed. Control TTE showed flap progression with neocavity growth (Video 1). He experienced cardiorespiratory arrest and died within a few hours. Necropsy confirmed acute anteroseptal STEMI and IDH with associated complete wall rupture as the cause of death (Figure 2).

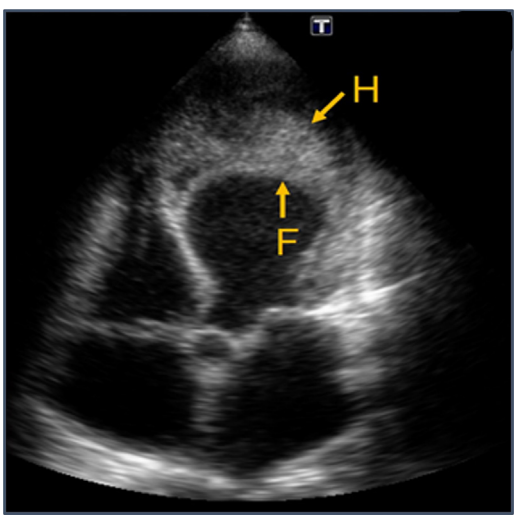
PATIENT 2. A 64-year-old woman with hypertension and dyslipidemia was admitted with STEMI and ongoing chest pain and basal bilateral rales. Her blood pressure was 125/85 mm Hg, and heart rate was 115 beats/min. Left main coronary artery and anterior descending artery angioplasty was performed 10 hours after the onset of symptom. TTE revealed anterior septal akinesia with mobile flap that extended from the mid-anterior septum to the apical septal segment, determining an anechoic cavity. A complete ventricular septal defect was ruled out because of the absence of blood flow through the septum. IDH was confirmed by CT (Figure 3). An initial conservative approach was decided, but a second TTE showing IDH progression and a new-onset pericardial effusion with moderate active flow confirmed a complete left ventricular free wall rupture (Video 2). Emergency cardiac surgery (endoventricular Gore-Tex patch placement) was indicated

FIGURE 2 Macroscopic View of Autopsy Specimen: Patient 1



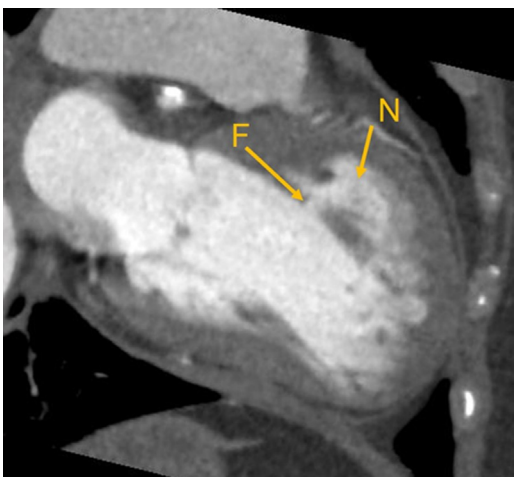
Intraparietal hematoma is seen. Abbreviations as in Figure 1.

FIGURE 1 Apical 4-Chamber View: Patient 1



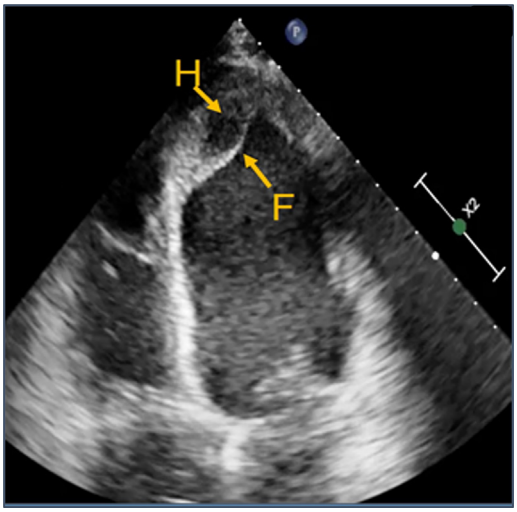
Intramyocardial dissection flap is shown delimiting the hematoma. F = flap; H = hematoma.

FIGURE 3 Computed Tomography View: Patient 2



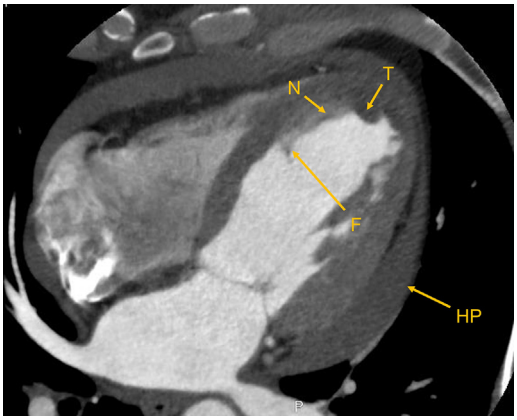
Evidence of intramyocardial dissection flap delineating a neocavity is seen. F = flap; N = neocavity.

FIGURE 4 Apical 4-Chamber View: Patient 3



Endocardial flap and intramyocardial hematoma are seen. Abbreviations as in Figure 1.

FIGURE 5 Computed Tomography View: Patient 4



Intramyocardial hematoma with thrombus, and hemopericardium, are seen. T = thrombus; HP = hemopericardium; other abbreviations as in Figure 2.

on day 9 after the patient's admission. The immediate postoperative outcome was favorable. However, the patient experienced septic shock associated with pneumonia, leading to death on postoperative day 16.

PATIENT 3. A 44-year-old man with diabetes, an active smoker, and with chronic heart failure with biventricular reduced ejection fraction of coronary etiology, was admitted with hypotension (80/60 mm Hg), tachycardia (104 beats/min), dyspnea, cold skin and extremities, and elevated serum lactate (3.8 mmol/L). Cardiogenic shock was diagnosed. Therapy was started with diuretic, inotropic, and vasodilator agents, and a TTE showed a linear and hyperrefringent band from the apical septum to the apical lateral segment, defining a cavity between it and the myocardial wall with heterogeneous content compatible with thrombus (**Figure 4**; Video 3). Other ventricular mechanical complications were ruled out. The final diagnosis was IDH associated with dilated cardiomyopathy, without confirmation of recent myocardial infarction. The patient experienced refractory shock and died in a few hours.

PATIENT 4. A 53-year-old man with hypertension, a current smoker, was admitted because of an anterior STEMI. The patient presented with ongoing chest pain, blood pressure 110/70 mm Hg, heart rate 83 beats/min, and no clinical signs of heart failure. An electrocardiogram showed ST-segment elevation in leads V_1 to V_5. He was treated with a pharmacoinvasive strategy, undergoing angioplasty of the anterior descending artery 7 hours after the onset of symptoms. Initial TTE revealed a separation between the subepicardium and subendocardium at the apical level suggestive of IDH. Differential diagnosis with apical intracavitary thrombus was done by identifying the endocardial layer and its systolic expansion (Video 4). The patient experienced pulseless ventricular tachycardia 24 hours after admission, which was successfully reversed. CT confirmed IDH, with the presence of hemopericardium (**Figure 5**), leading to a decision for an urgent surgical procedure. Pericardial blood collection drainage was performed, but no external rupture was evident on surgical exploration. Epistenocardic pericarditis with hemorrhagic pericardial effusion was considered as a differential diagnosis. A few hours after surgery, the patient experienced pulseless electrical activity and died. Necropsy reported severe hemopericardium with cardiac tamponade as the cause of death, along with IDH of the anterior left ventricular wall, including involvement from adjacent pericardial adipose tissue compatible with apical microperforation, confirmed by light microscopy (**Figures 6 to 7**).

DISCUSSION

IDH is an entity with very low incidence and high mortality. Previous case reports have described that low left ventricular ejection fraction, late

JACC: CASE REPORTS, VOL. 4, NO. 21, 2022
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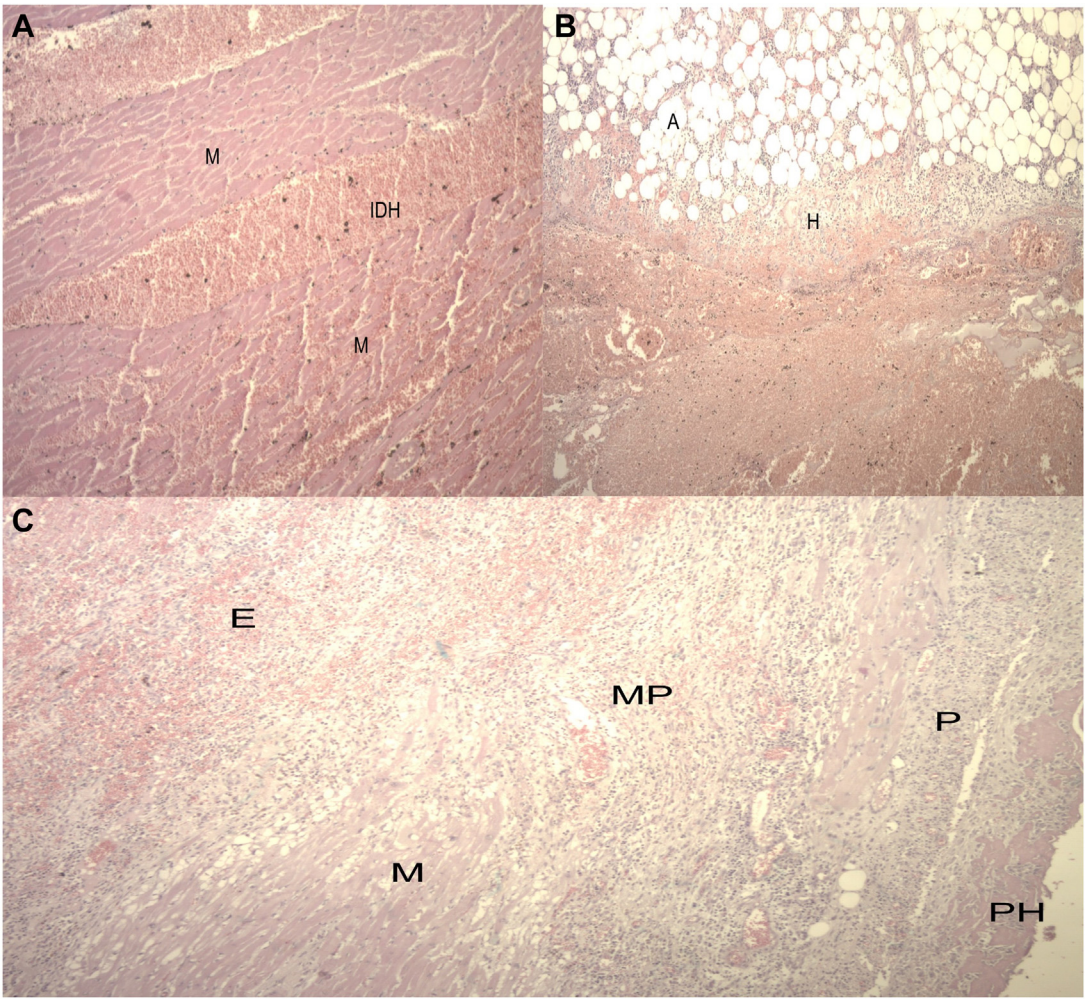
FIGURE 6 Microscopic Views of Autopsy Specimens: Patient 4



(A) Hematoxylin and eosin stain, ×10 image shows IDH area. **(B)** Hematoxylin and eosin stain, ×4 image shows hemorrhage in pericardial adipose tissue compatible with apical microperforation. **(C)** Hematoxylin and eosin stain, ×4 image shows myocardial microperforation area with signs of hemopericardium. A = adipose tissue; E = endocardium; H = hemorrhage; IDH = intramyocardial dissection; M = myocardium; MP = microperforation; P = pericardium; HP = hemopericardium.

diagnosis, pericardial effusion, and age over 60 years are associated with worse prognosis.[5]

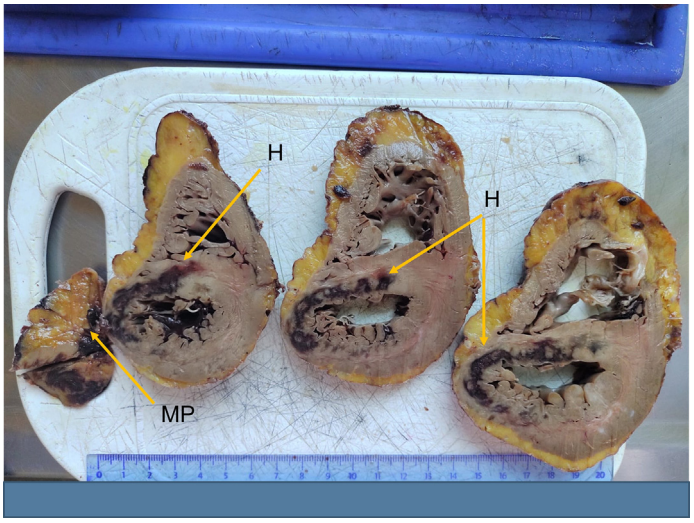
Therapeutic management guidelines have not yet been standardized.

It should be emphasized that the clinical evolution was torpid in all of our patients after an initial conservative approach was decided on, leading to death. Based on our experience, it could be suggested that an early surgical approach might improve clinical outcomes. Recent reports have shown that in very high-risk surgical patients, clinical and hemodynamic stability seems to allow a conservative approach with good midterm results. In this setting, urgent surgical repair should be considered in the presence of hemodynamic instability or expanding hematomas with pericardial effusion. [2,5,6]

None of our patients had any valvular disease of ischemic cause or other ventricular mechanical complications. Differential diagnosis with intracavitary thrombus remains crucial to avoid anticoagulation therapy, which could have a negative effect on IDH.

From our 4-case IDH series, we also highlight that each case was approached by a heart team (including cardiothoracic surgeons), using a multi-imaging

FIGURE 7 Macroscopic View of Autopsy Specimens: Patient 4



Hemorrhage in the apical pericardial adipose tissue, and area of intramyocardial dissection in the anterior left ventricle wall, are seen. H = hematoma; MP = microperforation.

strategy. Clinical follow-up included serial echocardiographic and/or tomographic imaging studies in all patients, and surgical or pathologic documentation in 3 of the 4 cases described (Table 1).

CONCLUSIONS

The therapeutic approach for IDH remains a challenge. An early surgical approach may be considered to improve clinical outcomes, being able to select a conservative strategy for very high-risk surgical patients with clinical and hemodynamic stability. Further evidence is needed to define the best therapeutic strategy.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

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

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TABLE 1 Patients with Intramyocardial Dissecting Hematoma and Their Characteristics

	Patient 1	Patient 2	Patient 3	Patient 4
Age (y)	60	64	44	53
Sex	Male	Female	Male	Male
Risk factors	Hypertension, smoking	Hypertension, dyslipidemia	Smoking, diabetes	Hypertension, smoking
AMI	Yes	Yes	Not confirmed	Yes
Time from AMI onset to reperfusion	–	10	–	7 h
Affected ventricular wall	Anterior	Anterior	Anterior	Anterior
Single-vessel disease	No	No	Unknown	Yes
LVEF (%)	31	30	17	38
Hemodynamic instability during hospitalization	Yes	No	Yes	Yes
Time from admission to hemodynamic instability (h)	120	-	3	10
Surgical management	No	Yes	No	Yes
Necropsy	Yes	No	No	Yes

AMI = acute myocardial infarction; LVEF = left ventricular ejection fraction.

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KEY WORDS cardiovascular disease, computed tomography, dissection, echocardiography, myocardial ischemia

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