

Dissection intramyocardial a rare complication of myocardial infraction: Case report

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Abstract

Intramyocardial dissecting hematoma (IDH) after acute myocardial infarction (MI) is a rare form of subacute cardiac rupture, resulting from hemorrhagic separation of helical fibers within the myocardium, which has been described in most studies as an incomplete cardiac rupture or a specific type of cardiac rupture. The management of myocardial dissection is variable and poorly documented, with clinical outcomes ranging from asymptomatic recovery to fatal cardiac events. We present here a case of myocardial dissection diagnosed by echocardiography.

Keywords: Word Keys: Intramyocardial; Dissection; Hematoma; Myocardial Infraction; Evolution; Prognostic

1. Introduction

Myocardial dissection, also known as intramyocardial dissecting hematoma (IDH), is a rare mechanical repercussion of a heart attack. Some studies categorize it as a type of incomplete cardiac rupture or a type of cardiac rupture caused by the hemorrhagic separation between the helical fibers of the myocardium. There are not many studies on myocardial dissection treatment, and clinical outcomes range from asymptomatic remission to cardiac death. We report a case of myocardial dissection identified by echocardiography.

2. Presentation of the case

A 79-year-old man presented with persistent dyspnea and atypical back chest pain for over a week. However, as the symptoms persisted, he sought medical attention. He was transferred to our hospital because of ST-segment elevation on the electrocardiogram and a positive troponin level. The patient had type 2 diabetes and no history of hypertension or smoking. On admission, a physical examination revealed normal pulmonary auscultation, no heart murmur, and no edema. The electrocardiogram revealed sinus rhythm with QS in the Antero septal apical segments (Figure 1). Chest X-ray revealed mild pulmonary congestion, but no pleural effusion. Troponin levels (300 N) were elevated.

Echocardiography was performed, showing a severely depressed LV ejection fraction (LVEF) of 26%, as well as extensive apical, mid-anteroseptal wall akinesia. A thickened and pulsatile LV cavity with dyskinetic motion surrounded by a thin endomyocardial border was visualized, suggesting a contained rupture of infarction with hematoma (Figure 2: A, B, and C, Videos 1, 2, and 3). Color Doppler interrogation revealed no flow between the left ventricular cavity and layers of the myocardium, which showed no connection between the echo-free space and the left ventricle. No color flow was observed in the left ventricle, due to low flow and thrombus formation. No pericardial effusion or signs of epicardial rupture were observed. Furthermore, MRI was not available in our context. A diagnosis of recent anterior infarction complicated by a large apical intramyocardial dissecting hematoma was made, and the patient was treated conservatively after concertation with the cardiovascular surgeons.

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The evolution was marked by clinical and hemodynamic stability without complication and the patient was discharged from the hospital within a few days without incident.

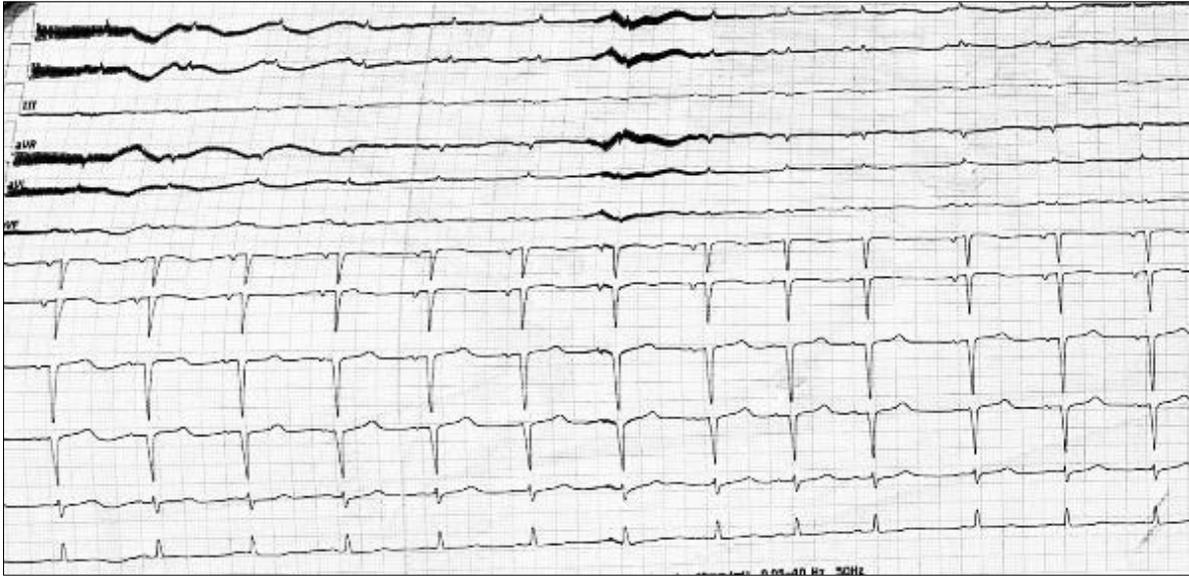


Figure 1 Electrocardiogram on arrival, showing deep q waves and ST elevation from V1 to V4

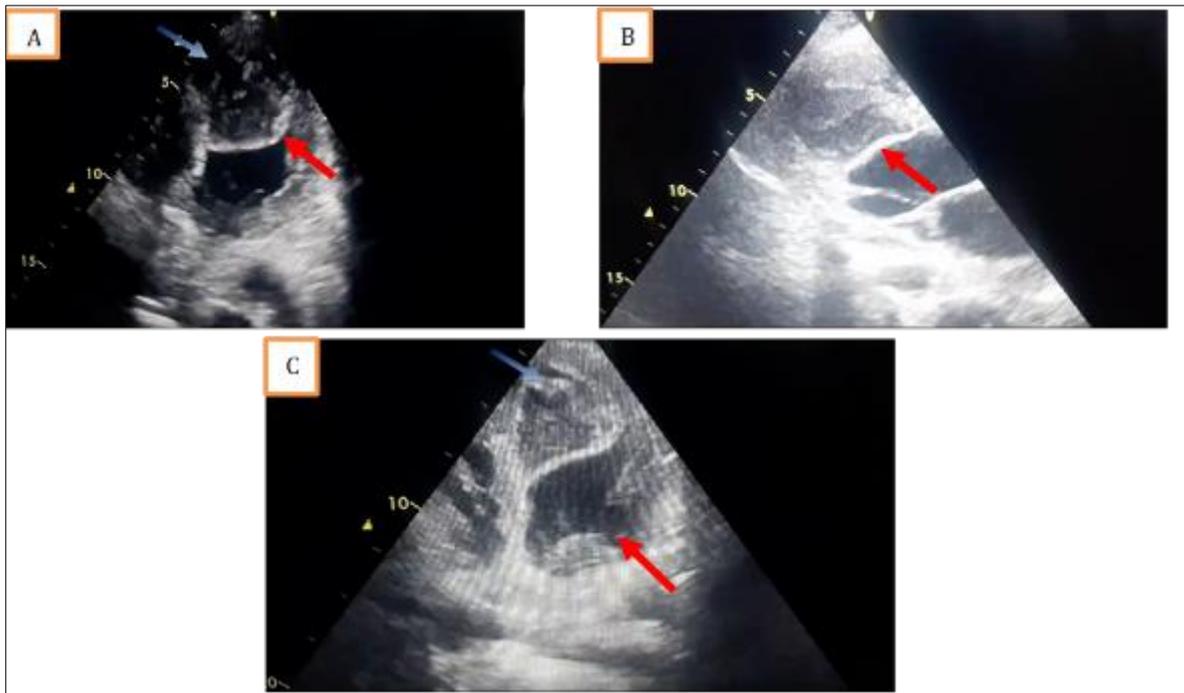


Figure 2 (A, B and C) In the apical four chamber view, first echocardiography revealed a mass abutting the left ventricular anterolateral to mid wall. This mass was initially thought to be a left ventricular clot Echo-lucent neocavitation including haematoma (Blue arrow) below a thin and movable layer of endomyocardium (Red arrow).

3. Discussion

Cardiac rupture, responsible for 0.8% of cardiac deaths after myocardial infarction, is a rare catastrophic event (1). It is defined by a linear tear in the myocardium from the free wall to the pericardial space or through the interventricular septum to the right ventricle, responsible for significant blood infiltration into and through the myocardial wall (1-2).

In addition, there are two types of cardiac rupture in the septum and free wall of the left ventricle: a simple tear and a complicated hemorrhagic dissection leading to cardiac death. The majority of cardiac ruptures are simple (79% of left ventricular free wall ruptures and 53% of septal ruptures). An intramyocardial dissecting hematoma is undulating or serpentine (3).

The mechanism of intramyocardial dissecting hematoma may be related to poor reperfusion. Necrosis results from a lack of blood flow, leading to cell membrane tearing, capillary rupture, and hemorrhage. Hypoxia-induced endothelial rupture is the main cause of hemorrhage development. The structural basis of myocardial dissection is the helical structure of the heart (3-4), which favors the propagation of bleeding along spiral myofibrils and provides a mechanism for intramyocardial dissecting hematoma. Reperfusion can potentially worsen hemorrhage by causing endothelial junction leakage, injury, and extravasation of red blood cells into tissue interstices.

In over 90% of cases, rupture occurs after the first infarction and correlates strongly with the monotruncal coronary artery reflecting the absence of collateral circulation (5). The most risk factors are anterior wall infarction, large transmural infarction, age 60, hypertension, female gender, coronary involvement, and absence of previous cardiac history (6).

Before the 1980s, the only cases described were found during necropsy. After 1981 the first case was diagnosed in a living patient who underwent surgery. Since then this condition has almost always been diagnosed via a two-dimensional echocardiogram. The presence of at least three of the following signs on echocardiography suggests the diagnosis of septal and/or free wall IDH (7-8-9):

- The formation of one or more neo-cavitations in the tissue that have an echo-lucent center
- A thinned, mobile endomyocardial tire related to the cavitary defect,
- The identification of ventricular myocardium in regions outside the cystic regions,
- Changes in the echogenicity of the neo cavitation suggesting blood content
- Partial or complete absorption of the cystic structure
- Communication between the two ventricular chambers via myocardial dissection,
- Flow measurement using Doppler within the dissected myocardium

However, MRI has become the gold standard for diagnosing IDH. The cine steady-state free-precession sequence provides superb imaging of the left ventricle, the usual anatomical structure of intramyocardial dissecting hematoma, dissecting endocardial flap, and connection with the right ventricle. Delayed enhancement images show a typical appearance of intramyocardial hematoma surrounded with a bright rim of hyperintense infarcted myocardium and dissecting endocardial flap.

Predictors of mortality in patients with intramyocardial dissecting hematoma				
	Age>60	MI	Anterior MI	EF<35% P<0.02
Age>60		35% P=0.03		44% P<0.02
MI	*35% P=0.03			40% P=0.03
Anterior MI				40% P=0.04
EF<35%	44% P<0.02	40% P=0.03	40% P=0.04	42% P=0.007

Figure 3 Multivariate analysis results. Color-coded map. Red color = mortality risk > 40%. Orange = mortality risk > than 35% up to 40%. Yellow = mortality risk 35% or less.

Multivariate analysis (Figure 3) showed that the major independent predictor of mortality in patients with IDH was EF < 35%. When associated with age > 60 or with MI and especially with late diagnosis (>24 hours since symptoms), mortality reached 50% (14)

Management of IDH depends on multiple factors, including patient age, hemodynamic stability, hematoma size, presence of ventricular septal defect, left ventricular function, and pericardial effusion. Independent risk factors for mortality were low ejection fraction (<35%), age ≥ 60 years, and previous myocardial infarction. In a case series by Leitman et al (7) published in 2018, 42 cases of HDI have been diagnosed and published to date, in-hospital mortality was 23% with late presentation (>24 h after symptom onset) was also associated with increased mortality. In our patient, the main risk factors were: low LVEF (26%), age >60 years, and previous MI.

Jesus Vargas-Barron (10) described 15 patients, and HDI was a complication of myocardial infarction in 2 patients with inferior infarction and 13 patients with anterior infarction. It showed that intramyocardial dissecting hematoma confined to the apex has a high probability of spontaneous reabsorption and that an initial conservative approach may be reasonable. On the other hand, Pliam et al analyzed (6) cases of intramyocardial dissecting hematomas. Eight were diagnosed post-mortem after conservative treatment and died a few weeks later. The remaining seven patients were diagnosed during their lifetime: five were treated surgically with good short and medium-term results, and the other two received medical treatment (one survived, the other died). In 1998, Nilkanth (8) reported a case of intramyocardial dissecting hematoma treated by surgical resection with good long-term survival. Subsequently, Nakata et al (8) Jiménez et al(9) and Vargas-Barron et al(10) described three cases of intramyocardial dissection treated conservatively with good results and acceptable medium- and long-term survival.

In our patient, we opted for a conservative approach because of the good clinical and hemodynamic tolerance, the limited experience in the surgical treatment of these patients, and the large size of the dissected area. We believe that spontaneous reabsorption of the intramyocardial hematoma can occur not only in patients with intact left ventricular endocardium but also in the presence of an intramyocardial tear with the passage of fluid from the left ventricle to the neocavity, as is the case in our patient.

Intramyocardial dissecting hematoma confined to the apex has a high probability of spontaneous reabsorption, and an initial conservative approach may be reasonable in the absence of a complete myocardial tear, 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 of rapid progression, progression to cardiac rupture, or the need for surgical revascularization. Anticoagulation may be considered to reduce the risk of thromboembolism within the left ventricle, but its use must be weighed against the risk of dissection progression and hemorrhage into the pericardial space (11-12)

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, left ventricular remodeling and development of functional mitral regurgitation, and sudden cardiac death. Nevertheless, we believe that the form of therapy should be based on the individual patient's condition and the experience of each surgical center in managing this type of pathology (13-14)

4. Conclusion

DH is a rare complication of MI that can be difficult to diagnose. Individual clinical and imaging aspects are used to guide management. In-hospital mortality is predicted with low EF, age > 60 years, anterior-wall MI, cardiogenic shock, and late diagnosis. In situations of minor apical hematomas, conservative therapy is usually used, however, cases of hemodynamic instability and developing hematomas may require surgical correction.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to disclosed.

Statement of informed consent

Patient perspective and informed consent.

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