Intramyocardial dissecting haematoma: a rare complication of acute myocardial infarction

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Intramyocardial dissecting haematoma (IDH) is a rare complication of myocardial infarction, with very scarce reports in medical literature. Before the advent of non-invasive imaging techniques, the diagnosis of IDH was only made by necropsy. It can develop in the left ventricular free wall, the right ventricle, or the interventricular septum.

We present a case of a patient with an IDH after acute anterolateral myocardial infarction, focusing on the utility of echocardiography in the diagnosis and follow-up of this unusual complication.

By this imaging modality, it was possible to see the various acoustic densities of the progressive clotting of the intramyocardial haematoma, its extension through the haemorrhagic dissection, as well as its independency in relation to ventricular cavities and extracardiac space by confirming intact epicardial and endocardial layers.

Based on this report, we believe that serial two-dimensional echocardiography, added, when necessary, by the use of contrast agents is the non-invasive method ideally suited to confirm the diagnosis and monitor its evolution at the patient’s bedside.

**KEYWORDS**
Intramyocardial dissecting haematoma; Myocardial infarction; Echocardiography

**Introduction**

Intramyocardial dissecting haematoma (IDH) is a rare and unusual form of myocardial rupture complicating acute myocardial infarction. This entity has also been described to occur spontaneously, after trauma to the chest or after reperfusion procedures.1,2 It consists of blood infiltration into and through the myocardial wall, which maintains endocardial and epicardial integrity. Formation of an intramyocardial haematoma 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.3,4 Before the advent of non-invasive imaging techniques, the diagnosis of IDH was only made by necropsy. It can develop in the left ventricular free wall, the right ventricle, or the interventricular septum.7 We describe a case of IDH after acute anterolateral myocardial infarction, focusing on the utility of echocardiography in the diagnosis and follow-up of this rare complication.

**Case report**

A 67-year-old white male with grade IV peripheral arteriopathy, and left hemiparesis caused by an ischaemic stroke 6 months earlier was admitted to the urgency department with complaints of chest pain, general malaise, and anorexia lasting for several days. At initial presentation, his vital signs were 84 bpm pulse rate, 113/73 mm Hg blood pressure, 96% oxygen saturation, and 36° of tympanic temperature. Cardiovascular examination revealed normal first and second heart sounds, without murmurs. Fine rales were heard in both lung bases. The ECG showed sinus rhythm, QS pattern in V3–V6, and ST elevation with inverted T waves in V2–V6 (Figure 1). The transthoracic echocardiogram at admission revealed a 35% ejection fraction with akinesis of the apex and mid-apical segments of the anterior and septal walls. At the apical-septal region, a pulsatile cavity with systolic expansion surrounded by a thin endomyocardial border was visualized. Colour-Doppler interrogation did not demonstrate any flow within that structure. There was no pericardial effusion or evidence of epicardial disruption (Figure 2A; Supplementary data online, Video S1). Initial cardiac biomarkers were slightly increased with peak troponin T of 0.07 ng/mL. These findings were consistent with acute ST
elevation anterior myocardial infarction complicated with probable IDH. A conservative approach was assumed and the patient was admitted to the coronary care unit, on double platelet antiaggregation with acetylsalicylic acid and clopidogrel, and anticoagulation therapy with enoxaparin 1 mg/kg b.i.d. subcutaneously.

Two days after admission and after starting anticoagulation therapy, the patient suffered a new ischaemic stroke, documented by brain CT scan, that resulted in global aphasia and right hemiparesis.

A subsequent echocardiogram, performed soon after this last event, showed progression of the dissection to the apical-lateral and mid-inferolateral segments and increased echogenicity of some parts of the cavity, consistent with focal thrombosis (Figure 2B and C; Supplementary data online, Video S2). Intravenous contrast (Sonovue®, Bracco,

**Figure 1** Twelve-lead electrocardiogram showing sinus rhythm, QS pattern in V3-V6 and ST elevation with inverted T waves in V2-V6, consistent with evolving anterior myocardial infarction.

**Figure 2** Transthoracic 2D-echocardiographic views of intramyocardial dissecting haematoma. (A) At presentation, parasternal long-axis view showing dissecting echo-free cavity (arrow). (B and C) Two days later, parasternal long-axis and subcostal short-axis views revealing increased echogenicity within the cavity, consistent with partially organized thrombi (arrow). (D) After contrast injection, (Sonovue®) integrity of the endocardial border is clearly shown (arrows). LA, left atrium; LV, left ventricle; Ao, aorta; C, dissecting cavity.
Italy) was also performed revealing integrity of the endocardial border and reinforcing the diagnostic hypothesis of IDH (Figure 2D; Supplementary data online, Video S3).

A conservative approach was taken due to the patient serious comorbidities and death occurred 10 days after admission.

Discussion

IDH is a very infrequent complication of the acute phase of myocardial infarction, with very scarce reports in medical literature.5 The underlying mechanism is a haemorrhage dissecting among the spiral myocardial fibres creating a necocavititation limited by the myocardium.6 Initially contained within the myocardial wall, the haematoma may expand, rupturing into adjacent structures, or spontaneously resolve.6 The diagnosis has commonly been made at surgery, post-mortem examination, or by echocardiography.

Until recent years, it was believed that the prognosis of intramyocardial dissection was invariably fatal in the short to midterm in those patients who did not undergo surgery. However, there are growing reports of a good outcome with conservative treatment, especially in patients with clinical and haemodynamic stability, in which echocardiographic monitoring shows a progressive clotting of the dissecting haematoma.7–9 In the case presented, in spite of double antiplatelet therapy and anticoagulation, the IDH evolved to partial thrombosis, so we believe that its extension was not related to the pharmacological treatment.

Differential diagnosis includes pseudoaneurysm,10 intracavitary thrombosis,3 or prominent ventricular trabeculations.11 IDH can easily be differentiated from pseudoaneurysm by establishing integrity of epicardium, since pseudoaneurysm comprises a complete rupture of the myocardial wall contained by the pericardium. The distinction with intracavitary thrombosis 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.

This case is paradigmatic of the utility of echocardiography to the diagnosis and follow-up of this kind of lesions clearly documenting the evolving nature of intramyocardial haematoma. By this imaging modality, it was possible to see the various acoustic densities of the progressive clotting of the intramyocardial haematoma, its extension through the haemorrhagic dissection, as well as its independency in relation to ventricular cavities and extracardiac space by confirming intact epicardial and endocardial layers.

Besides echocardiography, chest-computed tomography and cardiac magnetic resonance imaging can also have a role in the diagnosis by demonstrating intact myocardium around an intramyocardial cavity.5 But based on this report, we believe that serial two-dimensional echocardiography, added, if necessary, by the use of contrast agents is the imaging tool ideally suited to identify and follow-up this entity at the patient’s bedside.

Supplementary data

Supplementary data are available at European Journal of Echocardiography online.

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