CLINICAL IMAGE

Apical intramyocardial dissecting hematoma: a rare complication of acute myocardial infarction

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A 55-year-old woman with a history of smoking and hypertension was urgently admitted to the hospital due to antero-lateral ST-segment elevation myocardial infarction complicated by pulmonary edema and acute heart failure (HF). On admission, the patient was alert but with progressive dyspnea and fatigue. Physical examination revealed irregular pulse of 150 bpm, blood pressure of 127/82 mm Hg, and lung crackles. Electrocardiography showed atrial fibrillation with Q waves in leads II, III, aVF, and ST-segment elevations in leads V₂ through V₆. Urgent transthoracic echocardiography (TTE) showed thickening and dyskinesis of the apical region of the left ventricle (LV) (FIGURE 1A). Urgent coronary angiography showed total occlusion of the left anterior descending coronary artery and right coronary artery (FIGURE 1B and 1C). The right coronary artery was successfully reopened and stented; however, attempts to open the left anterior descending coronary artery were unsuccessful.

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After standard pharmacological treatment, the patient stabilized and sinus rhythm was restored. Cardiac necrosis markers were significantly increased (peak high-sensitivity troponin T, 3090 pg/ml; reference range <14 pg/ml; creatine kinase MB isoenzyme, 93 U/l; reference range <24 U/l). Repeated TTE revealed severely reduced LV ejection fraction, estimated at 20% to 25%, accompanied by a restrictive inflow profile and, surprisingly, a mass or "neochamber" dissecting the apical myocardium. It was separated from the LV cavity with a linear echo of a free-floating membrane. The pericardium was free of effusion. Color Doppler study did not show any communication between the LV, the "neochamber," the right ventricle, and the pericardium. Suspicion

of LV apical dissection / hematoma / aneurysm was raised.

Urgent cardiac magnetic resonance (CMR) confirmed an apical chamber with hematoma, a thin subepicardial myocardium showing late enhancement due to necrosis, a free-floating subendocardial dissection flap separating it from the LV cavity and bilateral pleural effusion (FIGURE 1D and 1E).

Enoxaparin treatment, initially administered due to atrial fibrillation, was stopped. Due to the extensive myocardial infarction and severely depressed LV function, the Heart Team decided on conservative treatment. In the following 2 weeks, the fluid filling the apical cavity gradually thrombosed and tendency for reabsorption was noted.

No significant arrhythmia was documented, the patient gradually recovered and was discharged after 3 weeks. Five-month follow-up documented stable NYHA II class on standard HF medication. TTE showed a streaky, low-signal structure in the apex and hematoma was not present anymore (FIGURE 1F). Control CMR showed a transmural scar in the thinner apex and apical segments with small residual intramural hematoma (FIGURE 1G and 1H). The LVEF was estimated at 35%.

The intramyocardial dissecting hematoma is a very rare presentation of cardiac rupture observed in approximately 0.45% of patients with ST-segment elevation myocardial infarction and is related to severe ischemia and the no-reflow phenomenon.¹ The reason can be swelling of endothelial cells causing gaps in the endothelial lining. Reperfusion of the microvascular obstruction may lead to extravasation of red blood cells causing a hemorrhagic dissection among the myocardial

FIGURE 1

A - echocardiography in the apical 4-chamber view showing echo-free cavity dissecting the apical myocardium (white arrow); B, C – coronary angiography showing total occlusion of the left anterior descending and right coronary artery (white arrows); D - first cardiac magnetic resonance (CMR)-cine gradient echo (GE) sequence, 4-chamber view-showing an apical "neochamber" (red arrow), a thin subepicardial myocardium (blue arrow), and a free-floating subendocardial dissection flap (yellow arrow) separating the "neochamber" from the left ventricular (LV) cavity. Bilateral pleural effusion is visible. E – the first CMR—T1--weighted GE sequence with high inversion time of 600 ms, 4-chamber view- showing large hematoma inside the "neochamber" of the LV (red arrow); F – follow-up echocardiography documenting regression of the apical hematoma; G - follow-up CMR-cine GE sequence—confirming the disappearance of LV "neochamber" and thin myocardium of the apical segments; H – follow-up CMR-late enhancement GE sequence, 4-chamber view-showing small residual hematoma (red arrow) inside the apical myocardial scar (blue arrow)

















fibers. The process may further aggravate ischemic myocardial necrosis and remodeling.¹⁻⁴

Intramyocardial hematoma may be treated conservatively or by a high-risk surgical removal, depending on the individual Heart Team decision. Surgery is needed if the separating membrane and outer wall layer are thin with predisposition to rupture as well as in case of significant valvular dysfunctions or blood flow obstruction due to a mass effect.⁵

TTE can raise the suspicion of hematoma or allow for the assessment of intracavitary communication or presence of pericardial bleeding; however, the image may be misleading, suggesting apical thrombus. As a consequence, the antithrombotic treatment may cause deadly tamponade. CMR can precisely visualize the size of a rupture, presence of microvascular obstruction, intramyocardial hemorrhage, necrosis, or scar. Our case illustrates that a spontaneous healing of the dissection is possible, and shows the great importance of detailed multimodality imaging, both in the acute setting and during the follow-up.^{1,2,4}

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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