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CASE REPORT

An Apical Mass of the Left Ventricle After a Myocardial Infarction: Imaging Contribution

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Abstract

Intramyocardial dissecting hematoma (IDH) is a rare complication of myocardial infarction (MI). It can affect the left ventricular free wall, the right ventricle, or the interventricular septum. We report a case of a 58-year-old man with an IDH following an acute anterior wall myocardial infarction detected by echocardiography and confirmed by Cardiac magnetic resonance (CMR).

Keywords: Intramyocardial dissecting hematoma, Myocardial infarction, Echocardiography, Cardiac magnetic resonance

1. Introduction

Intramyocardial dissecting hematoma (IDH) is a rare and unusual complication of myocardial infarction and hence management uncertainties.

This entity can be seen in several contexts and may lead to serious prognostic and therapeutic implications [1,2].

We report a case of IDH after acute anterior wall myocardial infarction detected by cross-sectional imaging.

2. Case report

A 58-year-old smoker man was admitted to the emergency department complaining from chest pain, dyspnea, and general malaise for the last 10 h. At initial presentation, his vital signs were 110 bpm pulse rate, 110/70 mm Hg blood pressure, and 88% oxygen saturation. The cardiovascular examination was normal. Fine rales were heard up to mid-lung fields. The electrocardiogram showed sinus rhythm, QS pattern and ST elevation in leads V1−V6.

The diagnosis of anterior myocardial infarction was confirmed, the patient received antithrombotic treatment with oral loading doses 300 mg clopidogrel, 150 mg aspirin and subcutaneous enoxaparin, intravenous 40 mg furosemid and he underwent failed fibrinolysis with intravenous tenecteplase. He was transferred to the cardiology department for further exploration. At admission, the patient was hemodynamically stable. The transthoracic echocardiogram at admission revealed a 20% left ventricle (LV) ejection fraction with a large akinesis of the apex and the anterior and septal walls. The pericardial effusion of low abundance opposite the anterior and lateral wall of the right ventricle and a hyper-echoic image measuring 59 × 32 mm facing the tip of the LV were detected (Fig. 1a). The color-Doppler mode did not demonstrate any flow within that structure. We highly suspected an apical thrombus.

The patient was pain free and given the doubt about this apical thrombus and the lack of viability we decided to manage the patient medically and to do further imaging exams.

A CT scan performed urgently showed a focal enlargement of the anterior and apical walls of the LV measuring 45 × 66 mm with intracavitary bulging. The spontaneous density was 41 HU, not

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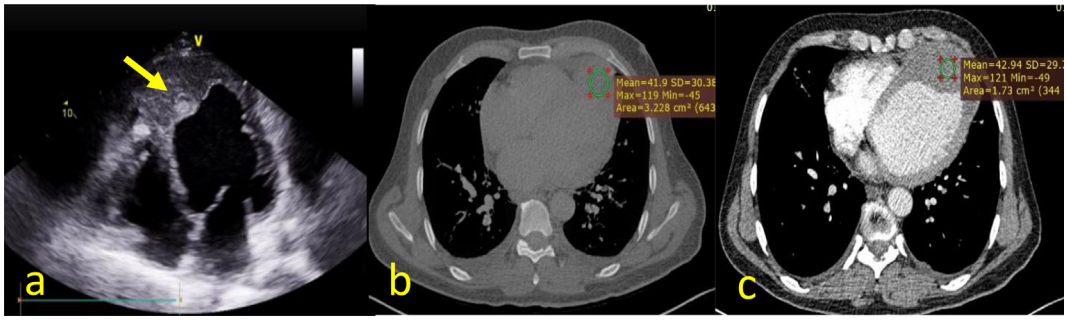

*Fig. 1. (a): **Transthoracic 2D echocardiogram; (b, c): CT scan without (b) and with (c) injection**: a. Hyper-echoic image facing the tip of the left ventricle (LV) (yellow arrow). b, c: A spontaneous hyperdense formation Non-enhanced after contrast injection associated with a bilateral pericardial and pleural effusion blade.*

raised after injection of contrast agent evoking an intramural hematoma (Fig. 1-b-c).

The cardiac magnetic resonance (CMR) has been indicated as it has a better contrast resolution. It showed a dissecting hematoma of the anterior myocardial wall in signal vacuum on the EG and hypersignal T1 sequences associated with two thrombi in the LV, one of which is a wall thrombus lining the bottom of the hematoma. We noted a late subendocardial and transmural elevation in places in antero-septal, infero-septal, middle and apical LV walls due to ischemia in the territory of the left anterior descending artery (LAD). The outer edge of the hematoma was bounded by the infarcted myocardial tissue (Fig. 2). There were no signs of viability in the anterior wall.

The patient was hemodynamically stable during his hospitalization and he was managed medically

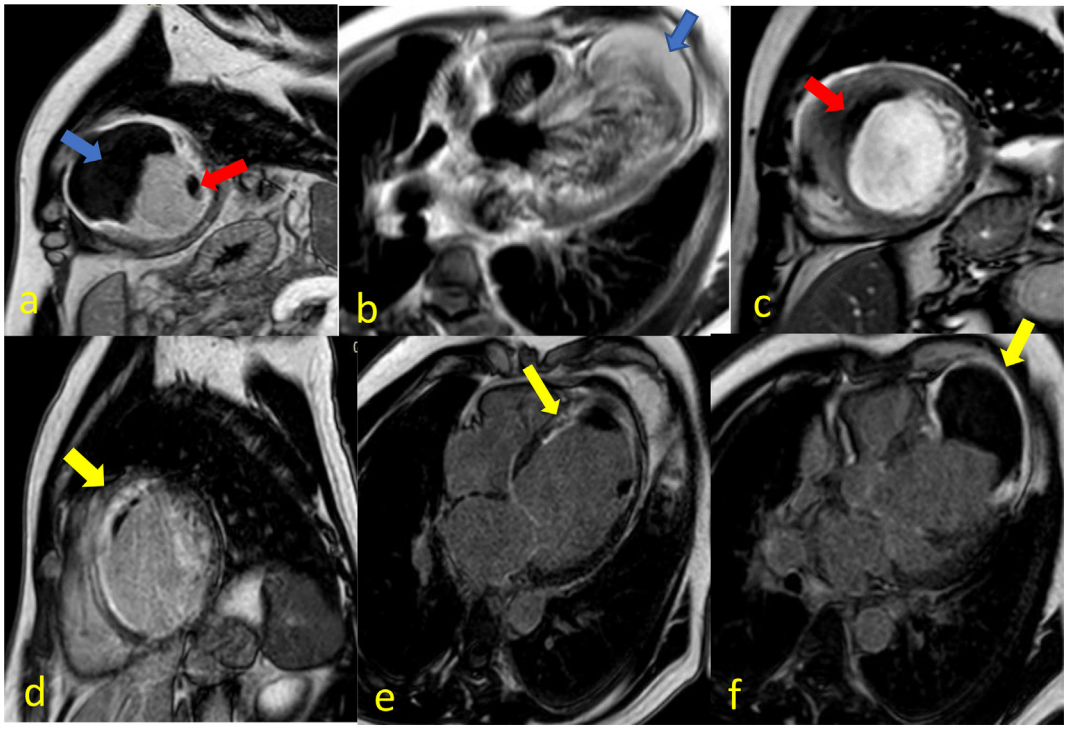

*Fig. 2. **(a, d, e, f) Late Gadolinium Enhancement (LGE); (b) T1-Weighted Spin Echo Sequence; (c) First-Pass Gadolinium Infusion Sequence; (a,c,d) minor axis of the LV; (b,e,f) axial 4 cavities**: Dissecting hematoma of the anterior myocardial wall in signal vacuum on EG and hypersignal T1 sequences (blue arrows). Two thrombi in the LV, one of which is a wall thrombus lining the bottom of the hematoma (red arrows). Late sub-endocardial and transmural elevation in places (yellow arrows) in antero-septal, infero-septal, middle and apical LV walls due to ischemia in the territory of the left anterior descending artery (LAD). The outer edge of the hematoma is bounded by the infarcted myocardial tissue.*

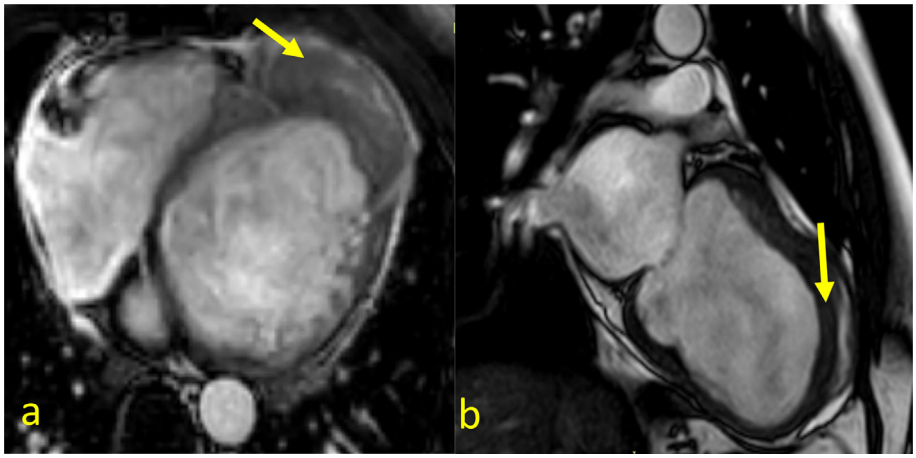

Fig. 3. Control MRI after 20 days: SSFP (steady-state free-precession) Cine Sequence: Axial 4 cavities (a), (b) long axis of the LV: Partial regression of dissecting intra myocardial hematoma (yellow arrows).

given the lack of viability. The CMR control after 20 days showed a partial regression of dissecting intra myocardial hematoma (Fig. 3). So, the patient was discharged. The Clinical follow-up was uneventful. The subsequent echocardiography repeated during the follow-up 3 months later showed a slight improvement of the LV ejection fraction with an increased echogenicity over the apex, consistent with a focal thrombosis (Fig. 4).

3. Discussion

The diagnosis of the IDH is often a challenge [3]. Usually the diagnosis is made at surgery, post-mortem examination, or by non-invasive imaging techniques such as echocardiography [4]. Indeed, during the recent years, echocardiography has permitted clinical suspicion, which is usually confirmed with CMR [3]. The pathogenesis of IDH involves a hemorrhage dissecting among the myocardial fibers creating a new cavity limited by the myocardium. Evolving, the hematoma may expand; rupturing into adjacent structures, or spontaneously resolve [3]. Serial echocardiography is helpful in determining its evolving nature and may guide the outcomes and necessity of surgical treatment. Color Doppler ultrasound is able to detect the presence of a communication with the endocardial or pericardial cavities [2]. Echocardiographic features of the IDH in anterior Myocardial Infarction are made of a non-homogenous neo-cavitation, often a pulsatile cavity with systolic expansion delineated with an endocardial flap [3]. Small pericardial effusion can be noted and it may be a sign of evolving myocardial rupture [3].

The gold standard for the diagnosis of IDH is CMR. Cine steady-state free-precession sequence provides excellent visualization of the LV, dissecting endocardial flap, the typical anatomical structure of IDH, and communication with the RV.

The T1 and T2 sequences are sensitive to blood products and often help in the diagnosis. The T1-

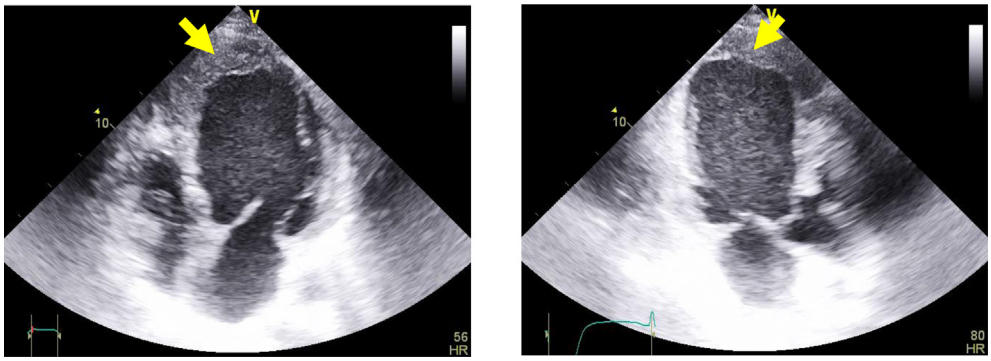

Fig. 4. Subsequent 2D trans-thoracic echocardiography 3 months later showing Increased echogenicity over the apex (yellow arrows), consistent with thrombus formation.

weighted image typically shows hyperintense lesion in the affected region corresponding to blood products due to subacute hemorrhage. The T2-weighted image shows hyperintense foci corresponding to edema or to fat. It is the Fat suppression technique which helps to differentiate edema from fat. The Delayed enhancement Images show the IDH surrounded with a bright rim of hyperintense infarcted myocardium and dissecting endocardial flap [5]. CMR is also a powerful investigational tool that is capable of revealing the underlying interactions of hemorrhage and therefore demonstrates its critical role in ischemia-reperfusion injury [5]. Previously it was believed that the prognosis of IDH was fatal in the short to midterm in those patients who did not undergo surgery. However, now outcome with conservative treatment is better especially in patients with clinical and hemodynamic stability [6,7].

The differential diagnosis of IDH includes prominent ventricular trabeculations, intracavitary thrombi and pseudoaneurysms [8,9].

We believe that sectional imaging in particular MRI is the gold standard to identify, confirm and follow-up this entity.

4. Conclusion

IDH after acute myocardial infarction is a rare form of subacute cardiac rupture. It is a diagnostic challenge. A high level of suspicion is needed in ultrasound. Nonetheless, CMR is undoubtedly the imaging modality to confirm the presence of intramyocardial hematoma to insure the management of these patients.

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Conflict of Interest

None declared.

Author contribution

Conception and design of Study: Selma Charfeddine, Wiem Feki. Literature review: Selma Charfeddine, Wiem Feki, Rania Hammami, Emna Daoud. Acquisition of data: Selma Charfeddine, Wiem Feki. Analysis and interpretation of data: Selma Charfeddine, Wiem Feki, Emna Daoud. Research investigation and analysis: Selma Charfeddine, Wiem Feki, Imen Maaloul. Data collection: Selma Charfeddine, Wiem Feki. Drafting of manuscript: Selma Charfeddine, Wiem Feki. Revising and editing the manuscript critically for important intellectual contents: Rania Hammami, Emna Daoud. Data preparation and presentation: Selma Charfeddine, Wiem Feki. Supervision of the research: Rania Hammami, Emna Daoud. Research coordination and management: Selma Charfeddine, Wiem Feki.

References

[1] Roslan A, Jauhari Aktifanus AT, Hakim N, Megat Samsudin WN, Khairuddin A. Intramyocardial dissecting hematoma in patients with ischemic cardiomyopathy: role of multimodality imaging in three patients treated conservatively. CASE (Phila) août 2017;1(4):159–62. https://doi.org/10.1016/j.case.2017.05.004.

[2] Harpaz D, Kriwisky M, Cohen AJ, Medalion B, Rozenman Y. Unusual form of cardiac rupture: sealed subacute left ventricular free wall rupture, evolving to intramyocardial dissecting hematoma and to pseudoaneurysm formation-a case report and review of the literature. J Am Soc Echocardiogr mars 2001;14(3):219–27. https://doi.org/10.1067/mje.2001.110780.

[3] Leitman M, Tyomkin V, Sternik L, Copel L, Goitein O, Vered Z. Intramyocardial dissecting hematoma: two case reports and a meta-analysis of the literature. Echocardiography févr 2018;35(2):260–6. https://doi.org/10.1111/echo.13796.

[4] Mart CR, Kaza AK. Postoperative dissecting ventricular septal hematoma: recognition and treatment. ISRN Pediatr 2011; 2011:534940. https://doi.org/10.5402/2011/534940.

[5] Ghugre NR, Pop M, Thomas R, Newbigging S, Qi X, Barry J, et al. Hemorrhage promotes inflammation and myocardial damage following acute myocardial infarction: insights from a novel preclinical model and cardiovascular magnetic resonance. J Cardiovasc Magn Reson 4 juill 2017;19(1):50. https://doi.org/10.1186/s12968-017-0361-7.

[6] Dias V, Cabral S, Gomes C, Antunes N, Sousa C, Vieira M, et al. Intramyocardial dissecting haematoma: a rare complication of acute myocardial infarction. Eur J Echocardiogr juin 2009;10(4):585–7. https://doi.org/10.1093/ejechocard/jep027.

[7] Tanoue K, Sata N, Amitani S, Yamashita T, Moriyama Y, Miyahara K. Interventricular septal dissection after acute myocardial infarction. Ann Thorac Surg août 2006;82(2):751. https://doi.org/10.1016/j.athoracsur.2005.05.032.

[8] GaliutoL,NataleL,LocorotondoG,BarchettaS,MastrantuonoM, Rebuzzi AG, et al. Images in cardiovascular medicine. Intramyocardial spontaneous hematoma mimicking an acute myocardial infarction. Circulation 9 oct 2007;116(15):e371–372. https://doi.org/10.1161/CIRCULATIONAHA.107.699793.

[9] Faludi R, Tóth L, Komócsi A, Varga-Szemes A, Papp L, Simor T. Chronic postinfarction pseudo-pseudoaneurysm diagnosed by cardiac MRI. J Magn Reson Imag déc 2007;26(6): 1656–8. https://doi.org/10.1002/jmri.21165.