ESC European Society of Cardiology

European Heart Journal - Case Reports (2022) **6**, 1–8
https://doi.org/10.1093/ehjcr/ytac295

CASE REPORT
Cardiac imaging

Conservatively treated intramyocardial dissecting haematoma of the interventricular septum as a rare complication of acute myocardial infarction: a case report

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Received 1 November 2021; first decision 14 January 2022; accepted 13 July 2022; online publish-ahead-of-print 23 July 2022

Background Intramyocardial dissecting haematoma (IDH) is a rare and potentially life-threatening complication of acute coronary syndrome. So far only isolated case reports and case series have been published.

Case summary We report the case of a late presenting myocardial infarction (MI) complicated by IDH of the ventricular septum, following a successful percutaneous coronary intervention (PCI). The clinically inapparent septal mass was discovered during the routine transthoracic echocardiography and the final diagnosis of haematoma was made by magnetic resonance imaging. The patient remained clinically stable, and septal mass on repeated echocardiography showed gradual regression.

Discussion This report suggests that IDH can spontaneously resolve without surgical intervention. An urgent echocardiogram should be used to assess the vitality of the myocardial tissue, especially with late presenting MI with deep Q-waves on the electrocardiogram strip. Conservative treatment in haemodynamically stable patients with IDH following MI and PCI is a feasible solution.

Keywords Case report • Intramyocardial dissecting haematoma • Myocardial infarction • Cardiac imaging

ESC Curriculum 2.2 Echocardiographyt • 2.3 Cardiac magnetic resonance • 3.4 Coronary angiography • 2.1 Imaging modalities • 3.2 Acute coronary syndrome

Learning points

- Late presenting myocardial infarction is associated with the formation of an intramyocardial haematoma.
- Serial echocardiography is useful in monitoring patients with intramyocardial haematoma; however, magnetic resonance imaging is the gold-standard method of assessment.
- Conservative management is a feasible choice in the treatment of haemodynamically stable patients, as complete regression sometimes occurs in a matter of weeks.

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Handling Editor: Francesca Musella
Peer-reviewers: Mohammed Al-Hijji and Mariama Akodad
Compliance Editor: Zhiyu Liu
Supplementary Material Editor: Gonçalo Costa

Introduction

Intramyocardial dissecting haematoma (IDH) is a rare complication of myocardial infarction (MI), percutaneous coronary intervention (PCI), coronary artery bypass surgery, and cardiac surgery. The dissection between the spiral myocardial fibres is formed by the influx of blood, creating neocavitation that is entirely contained within an integrated myocardial wall.[1] It occurs most commonly in the free wall of the left ventricle, the interventricular septum (IVS), and the free wall of the right ventricle.[2] This uncommon complication is usually detected by transthoracic echocardiography (TTE) and a definitive diagnosis is made by magnetic resonance imaging (MRI). Management of these patients is based on individual clinical judgment and can be conservative or surgical.

We present a patient with conservatively treated IDH after acute MI, underscoring the importance of using non-invasive imaging modalities for diagnosis and follow-up.

Timeline

Time	Event
20 January 2020	Persistent chest pain of medium intensity which started 6 h before admission
	PCI was performed
21 January 2020	TTE showed a septal mass which partially suppressed the right ventricular cavity
	Multi-slice computed tomography (MSCT) confirmed the mass in the ventricular septum
23 January 2020	MRI showed an intramyocardial haematoma of the ventricular septum
24 January to 13 February 2020	Repeated echocardiography showed a spontaneous gradual regression of the haematoma
13 February 2021	Patient was discharged
19 February 2021	TTE follow-up revealed a decrease in haematoma size
21 February 2021	MRI follow-up showed a smaller haematoma in the late subacute phase
8 April 2021	MRI and TTE follow-up showed almost complete regression of the septal haematoma
29 May 2021	Coronary computed tomography angiography (CCTA) at 3 months follow-up showed no in-stent stenosis or left anterior descending artery (LAD) compression

Case presentation

A 60-year-old male with known hypertension, dyslipidaemia, and positive family history of heart disease presented to our hospital with persistent moderate-intensity chest pain that began 6 h before ambulance arrival. There were no abnormalities on respiratory examination and cardiac auscultation. Blood pressure was 150/100 mmHg. The initial electrocardiogram showed sinus rhythm, heart rate of 80 beats/min, ST-elevation of 3 mm, and formed Q-waves in the anterior and lateral wall leads (*Figure 1*). The high-sensitivity troponin test detected significantly elevated troponin levels. The patient stated that he had episodes of brief, dull pain in his left shoulder for the past month. In 2007, he lost his sight in both eyes due to hereditary retinopathy.

The patient was diagnosed with ST-elevation MI and was urgently transported to the catheterization laboratory. He was previously loaded with dual antiplatelet therapy: 300 mg aspirin and 180 mg ticagrelor. The proximal segment of the LAD was occluded (*Figure 2A*) and two drug-eluting stents (Xience Pro 3 × 23 and Synergy 2.5 × 8) were implanted. The Runthrough NS Floppy guidewire was used. A 70% stenosis of the medial segment of the right coronary artery remained untreated. After PCI, the Thrombolysis in Myocardial Infarction (TIMI) 3 flow was achieved with no visible signs of complications (*Figure 2B*). The patient was then transported to our coronary unit, where he continued to receive dual antiplatelet therapy, a proton-pump inhibitor, and a high-intensity dose of statin. He was symptom-free.

A routine TTE was performed the following day before discharge. It revealed a spindle-shaped mass in the hypokinetic/akinetic IVS, parallel to the axis of the septum, ∼35 mm in size, on both parasternal long-axis view and apical four-chamber view, and with a central echolucency (*Figure 3A*). The main differential diagnoses were an intraventricular thrombus, myocardial tumour, and a septal haematoma. Since a workhorse wire with a soft, atraumatic, low-weight tip designed to reduce distal perforations was used and there was no contrast extravasation to the pericardial space or intracardial cavity during or after the PCI, the possibility of coronary artery perforation caused by the guide wire was dismissed.

Cardiac images by MSCT were obtained on the same day. We could not distinguish between a haematoma and an intracavitary thrombus with certainty because it was not possible to achieve a satisfactory characterization of the myocardium and the formation on both TTE and MSCT (*Figure 3*). In the absence of enoxaparine, a lower dose of 0.4 mL/24 h of nadroparin was carefully administrated.

Coronary computed tomography angiography was performed to assess the flow through the LAD and possible vessel compression. There was no evidence of in-stent stenosis, but a significant narrowing of the artery distal to the deployed stents was observed (*Figure 3B*).

An MRI was performed on the fourth day of hospitalization. There was an intraseptal mass with a hypointense signal to the myocardium on all sequences. The initial diagnosis of haematoma was based on the intraseptal location, and the absence of late gadolinium enhancement (LGE) within the change itself (*Figure 4*). Nadroparin was immediately discontinued from therapy. Because the patient was clinically stable and TTE monitoring showed no progression of the haematoma, a joint decision was made by cardiologists and a cardiac surgeon to continue conservative management. Because of the CCTA report, we believed that there was a risk of stent thrombosis, so we opted for a more potent P2Y12 inhibitor and did not de-escalate to clopidogrel. The patient was discharged in stable condition after 25 days.

Cardiac MRI was repeated after 2 weeks and revealed a smaller IDH with a hyperintense signal to the myocardium due to methaemoglobin. Magnetic resonance imaging and TTE scans after

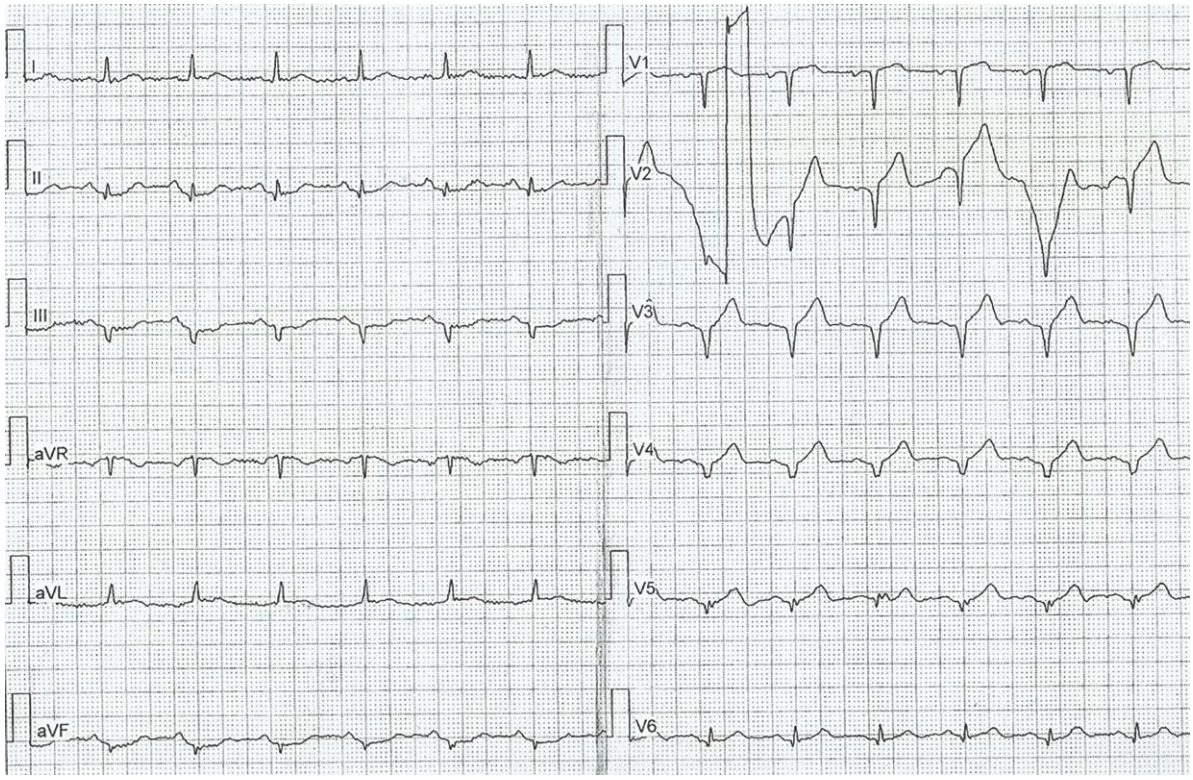

Figure 1 ST-elevation of 3 mm and formed Q-waves in the anterior and lateral wall leads.

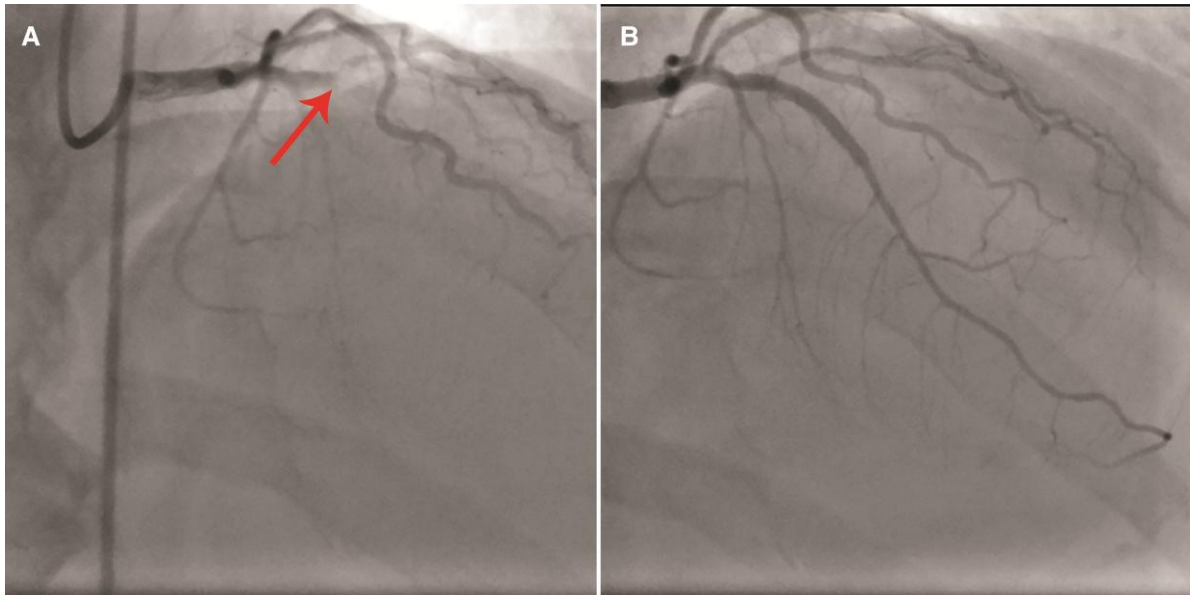

Figure 2 (*A*) The proximal segment of the left anterior descending artery was occluded. (*B*) Two drug-eluting stents were implanted. TIMI 3 flow was achieved, without visible signs of complications.

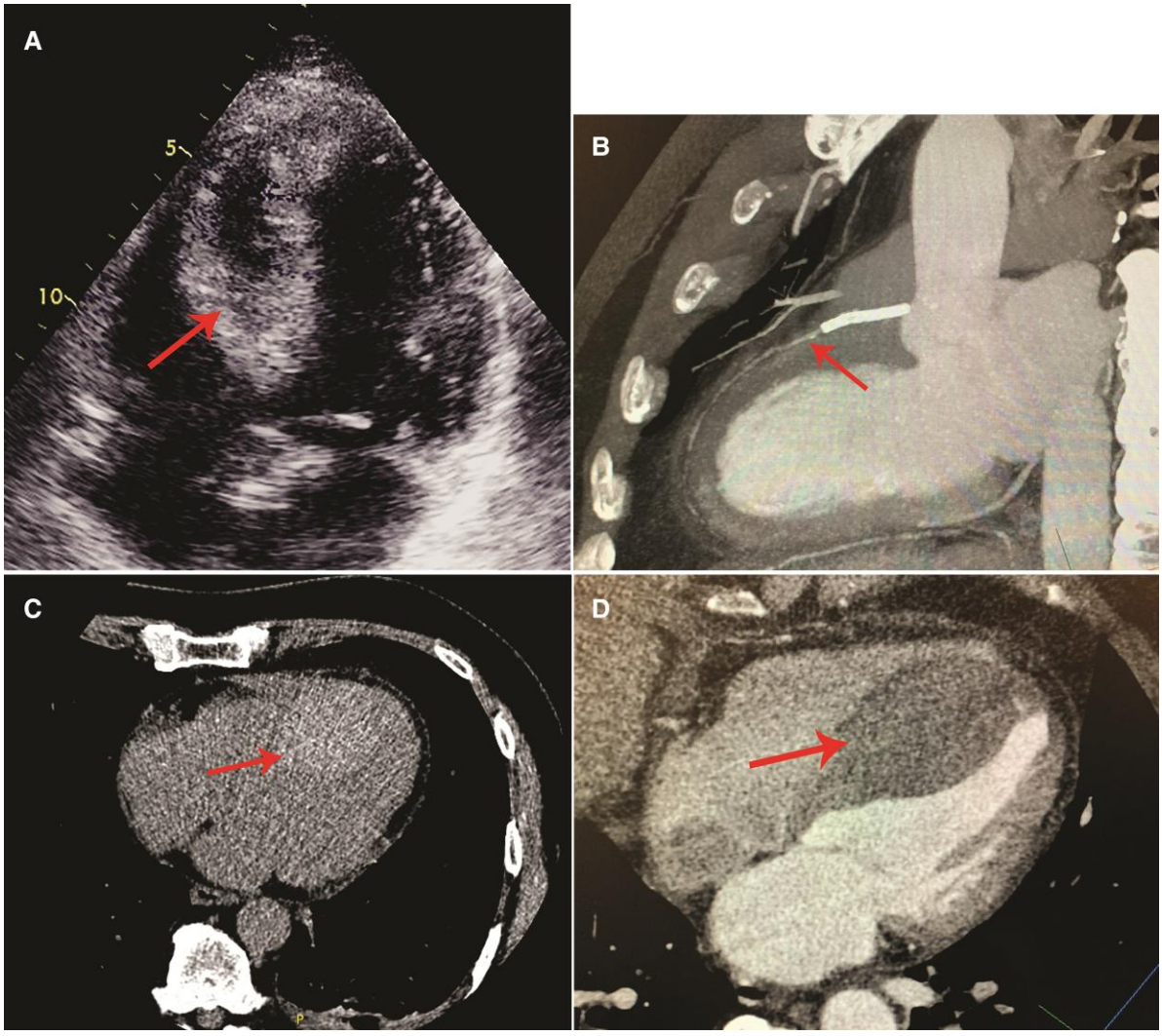

Figure 3 (*A*) Transthoracic echocardiography; (*B*) coronary computed tomography angiography; (*C*) native computed tomography scan, axial view; (*D*) coronary computed tomography angiography, four-chamber view. (*A*) Transthoracic echocardiography revealed a spindle-shaped mass in the hypokinetic/akinetic interventricular septum, with a central echo-lucency; (*B*) coronary computed tomography angiography revealed significant narrowing of the left anterior descending artery distal to the applied stents due to spasm; (*C*) spindle-shaped mass in the interventricular septum, hyperdense on native computed tomography; (*D*) without enhancement on post-contrast computed tomography.

2 months indicated that the haematoma had almost completely resolved, with a hypointense signal to the myocardium due to haemosiderin (*Figure 5*).

At 3 months follow-up, a repeat CCTA was performed because the patient refused a follow-up angiography. There was no in-stent stenosis or residual haematoma (*Figure 6*). The patient continued to take 100 mg of aspirin once daily and 90 mg of ticagrelor twice a day and remained symptom-free.

Discussion

The incidence of intramyocardial haematoma has not been well documented, thus our knowledge is mainly based on isolated case reports.[3]

Vargas-Barrón *et al*. described five types of cardiac rupture: a simple rupture, a complex rupture, a subepicardial aneurysm, pseudoaneurysm, and intramyocardial haematoma. Intramyocardial dissecting haematoma is characterized by massive blood infiltration into and through the myocardial wall. The endocardium and epicardium are both intact, and the haematoma is completely contained within the myocardium.[4]

According to post-mortem studies, post-infarction cardiac rupture usually develops within the first week, and very often within the first 24 h. This serious complication is more common in transmural and extensive infarcts. The majority of ruptures occurs during anterior wall MI, frequently near the IVS.[4]

An acute increase in coronary capillary pressure following reperfusion is thought to cause rupture of fine vasculature previously

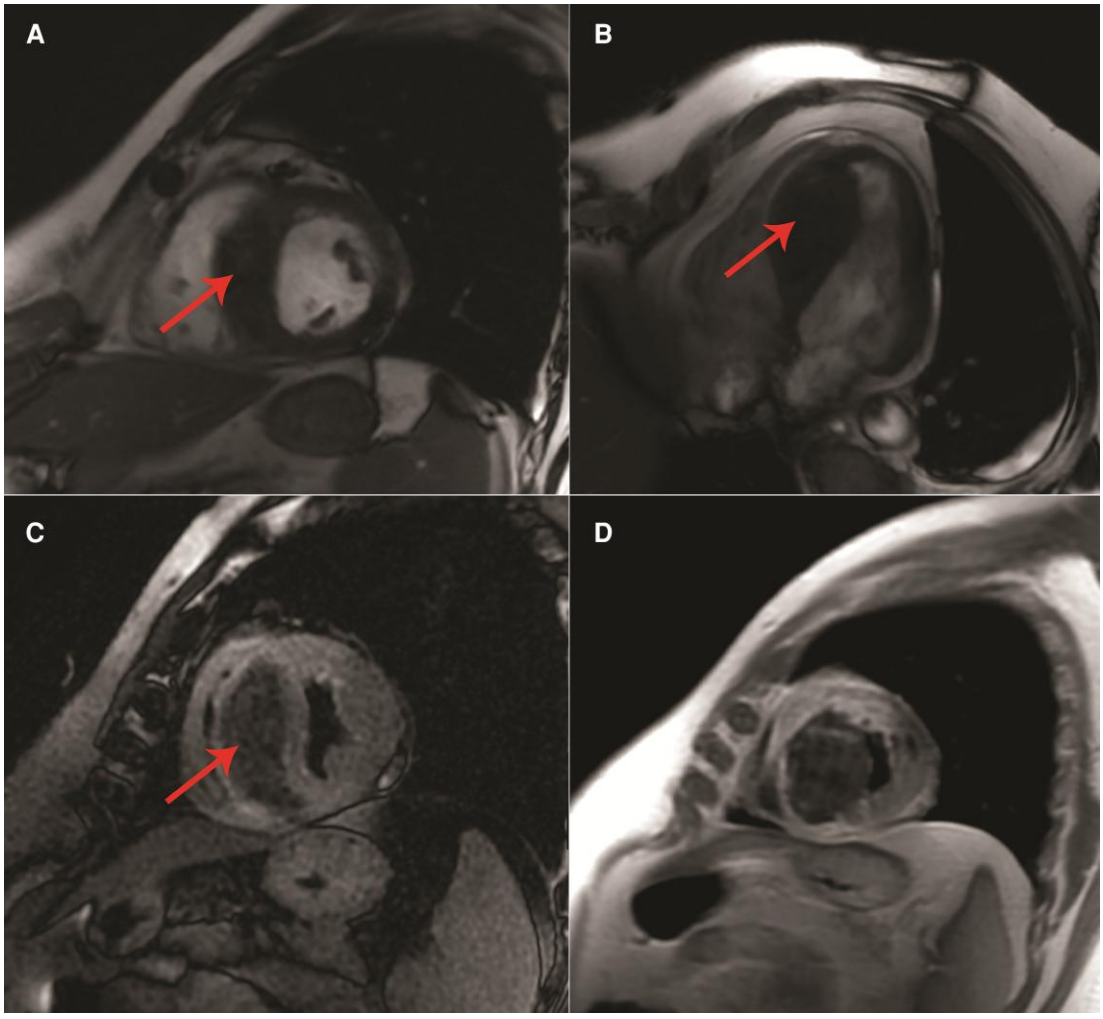

Figure 4 (*A*) Cine bright blood sequence, short-axis view; (*B*) Cine bright blood sequence, four-chamber view; (*C*) T2-weighted black blood triple inversion recovery sequence, short-axis view; (*D*) T1-weighted black blood double inversion recovery LGE sequence, short-axis view. On all sequences, we can see intraseptal mass that has hypointense signal to the myocardium. (*D*) There is no LGE of mass. LGE around mass is present in anteroseptal wall.

exposed to ischaemia and intramural blood dissection through weakened infarcted myocardial tissue.[5] Hypoxia is necessary to initiate the process of microvascular damage, and the duration of ischaemia determines its extent. All patients with IMH have microvascular obstruction.[6]

According to the research conducted by Spinelli *et al.*,[7] IDH is associated with coronary 'no reflow' (TIMI flow ≤ 2), as well as longer 'pain-to-balloon' time.

The first imaging modality that should raise suspicion for IDH is echocardiography, and the presence of at least three of the following signs can be used to diagnose IDH:[8]

(1) the formation of one or more neocavitations within the tissue with an echo-lucent centre;
(2) thinned and mobile endomyocardial border surrounding the cavitation;
(3) ventricular myocardium identified in the regions outside of the cystic areas;
(4) changes in the echogenicity of the neocavitation compared with normal myocardium suggesting blood content;
(5) partial or complete absorption of the cystic structure;
(6) continuity between the dissecting haematoma and one of the ventricular cavities;
(7) communication between the two ventricular chambers through the myocardial dissection;
(8) Doppler recording of flow within the dissected myocardium.

However, it is common for clinicians to still face uncertainty and rule out a tumour, a thrombus, and other types of cardiac rupture.[9] Therefore, the use of CT or MRI is recommended in clinically stable patients.[10] In the first week after reperfusion, cardiac MRI is considered to be the gold-standard method of

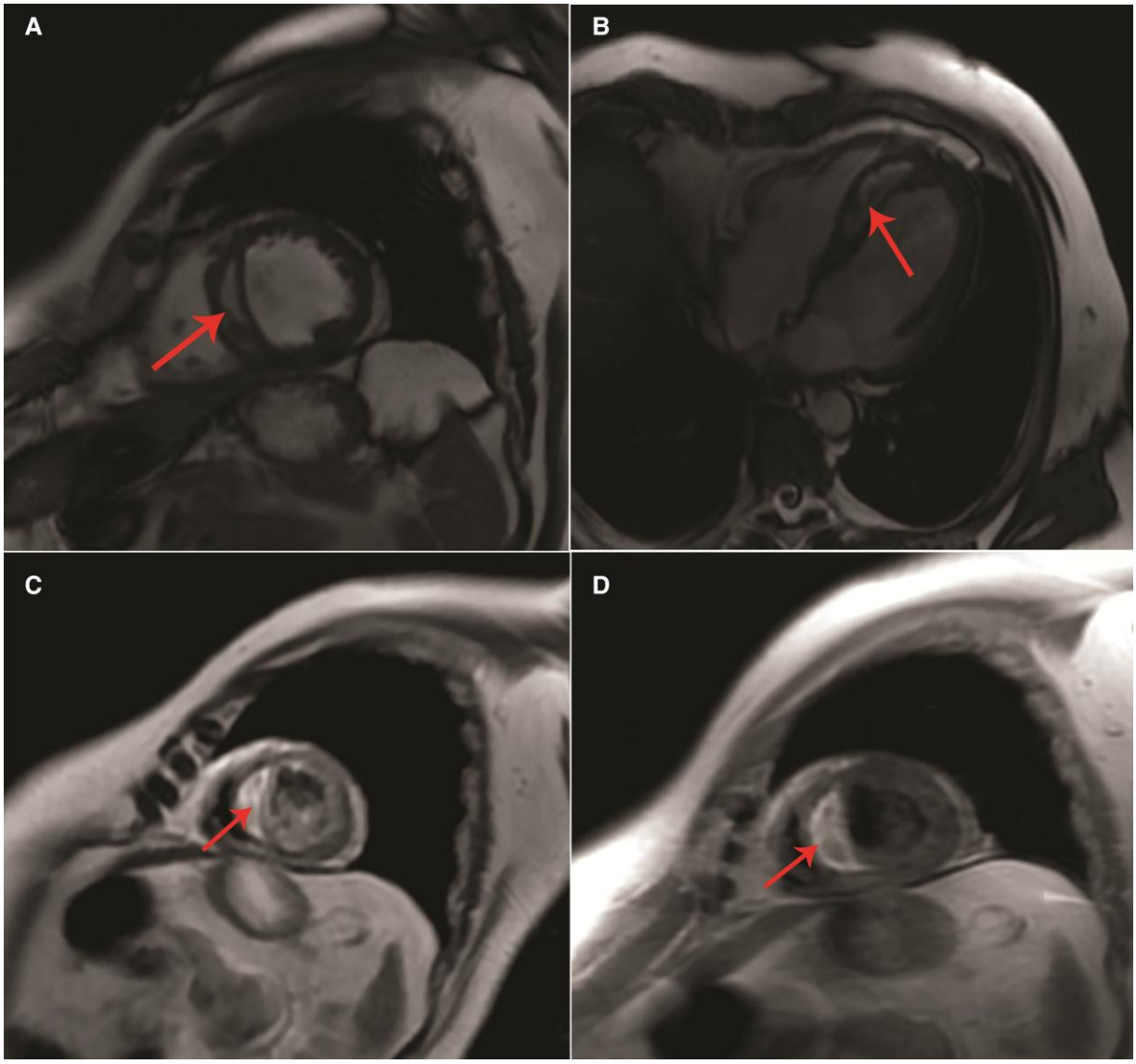

Figure 5 (*A*) Cine bright blood sequence, short-axis view; (*B*) Cine bright blood sequence, four-chamber view; (*C*) T2-weighted black blood double inversion recovery sequence, short-axis view; (*D*) T1-weighted black blood double inversion recovery sequence, short-axis view. After 2 weeks, haematoma is showing decrease in size on all sequences and hyperintense signal to the myocardium due to methaemoglobin.

assessment. Various MRI sequences (T1, T2, and T2*) can be used to assess intramyocardial haemorrhage, but T2* appears to be the most sensitive. In the late subacute phase (>7 days), elevated extracellular levels of methaemoglobin (due to the degradation of RBC) produce a very hyperintense signal on T1-weighted cardiac MRI, as seen at our initial follow-up. Eventually, deoxygenation leads to lysis of the erythrocyte membrane, exposing the iron-breakdown products ferritin and haemosiderin. Iron deposition can be detected by T2-weighted or T2*-weighted cardiac MRI in the first 4 weeks after the treatment.[11]

The optimal IDH therapy is controversial and an individual approach is recommended. Haematomas can be lethal owing to adverse haemodynamic effects (e.g. coronary artery compression), arrhythmias, and bleeding from epicardial rupture. Patients with expansion of the dissection on serial echocardiographic studies, those with ventricular septal defect (VSD) and impaired haemodynamics, and low EF, especially in anterior MI, should undergo surgery.[12] However, there have been reports of spontaneous haematoma resorption in haemodynamically stable individuals without dangers predictors.[13]

The development of pharmacological therapies aimed at protecting the microvasculature or altering the way reperfusion is established could prevent microvascular injury and reduce the risk of intramyocardial haemorrhage.[14,15] Furthermore, the striking similarities between haemorrhagic transformation

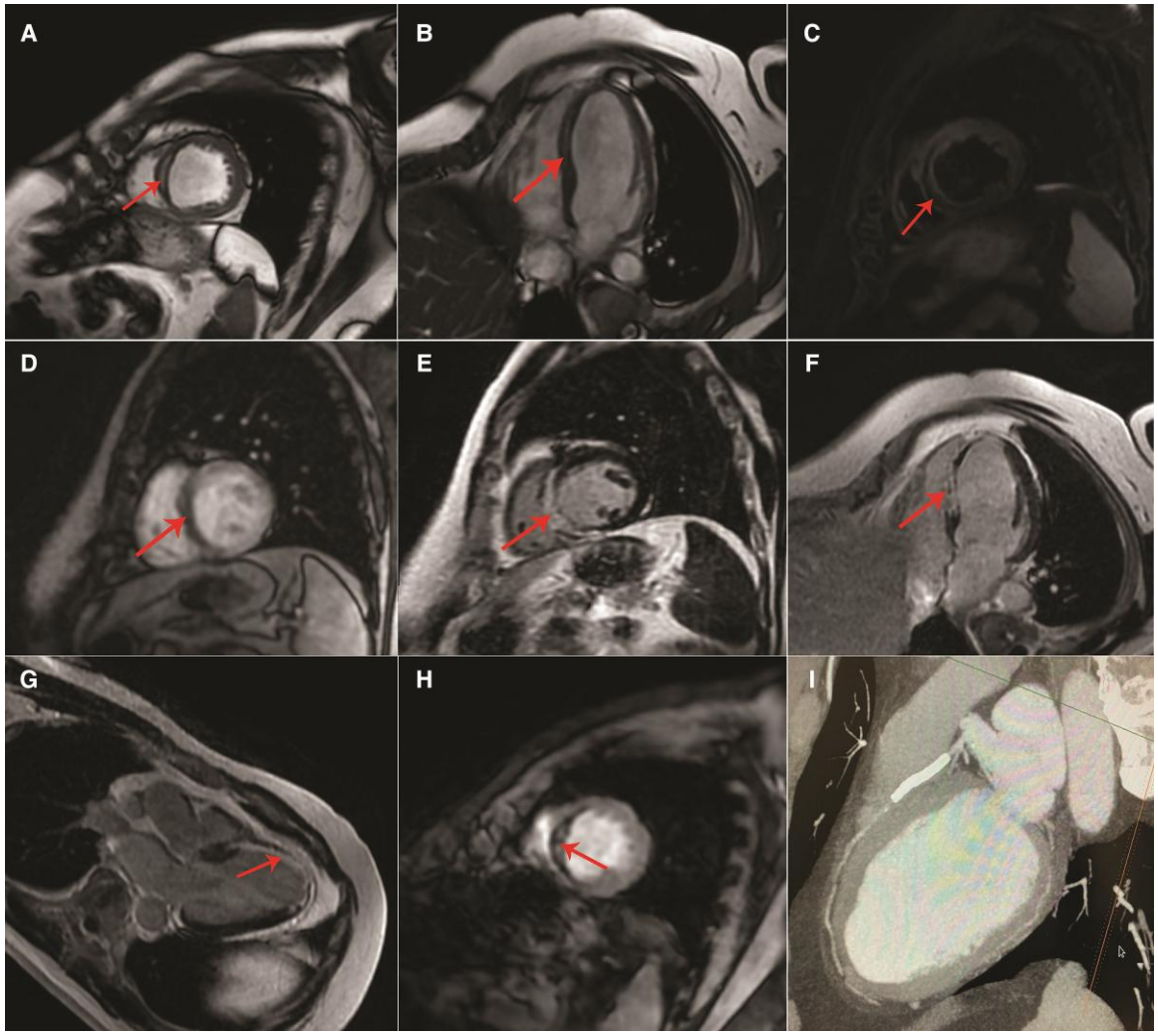

Figure 6 (*A*) Cine bright blood sequence, short-axis view; (*B*) Cine bright blood sequence, four-chamber view; (*C*) T2-weighted black blood triple inversion recovery sequence, short-axis view; (*D*) resting first pass perfusion imaging, short-axis view; (*E*) T1-weighted LGE sequence, short-axis view; (*F*) T1-weighted LGE sequence, four-chamber view; (*G*) T1-weighted LGE sequence, three-chamber view; (*H*) T2*-weighted sequence, short-axis view; (*I*) coronary computed tomography angiography. After 2 months: (*A* and *B*) significant decreasing of haematoma in size; (*C* and *H*) haematoma has hypointense signal to the myocardium due to haemosiderin; (*D*) haematoma is showing reduced signal intensity on resting first pass perfusion imaging; (*E–G*) transmural LGE of anteroseptal wall, in left anterior descending artery territory; (*I*) significant stenosis is not visible in this image, as it was in *Figure 2B*.

following ischaemic stroke and intramyocardial haemorrhage suggest that interdisciplinary research and exchange of ideas could aid in the development of novel treatments.[11]

Conclusion

Intramyocardial dissecting haematoma is a rare and sometimes fatal complication of MI, hence it should be borne in mind when examining a cardiac mass. Transthoracic echocardiography should be used as the initial imaging modality. However, if the diagnosis is uncertain, a cardiac MRI should be performed. Conservative management can be a good choice in treating haemodynamically stable patients, as complete regression sometimes occurs in a matter of weeks.

Lead author biography

Dr Svetlana Apostolović is an interventional cardiologist and a professor of Internal Medicine at the Faculty of Medicine, University of Nis, Serbia. Her main research fields include acute coronary syndromes, heart failure, and cardiac imaging. She is a president of the CSS working group for cardiac pharmacotherapy, a member of the Cardiology Society of Serbia, a member of the Academy of Medical Sciences, and a Fellow of the ESC (FESC).

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Acknowledgements

The authors would like to thank the physicians, nurses, and other healthcare workers who were in charge of this patient's care, as well as the Department of Radiology, University Clinical Center of Niš, Serbia for obtaining and elaborating the multi-slice computed tomography, coronary computed tomography angiography, and cardiac magnetic resonance images.

Slide sets: A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

Funding: The authors received no financial support for the research, authorship, and publication of this article.

References

1. Wei Y, Zhang B. Progressive intramyocardial dissecting hematoma in the apex of the left ventricle after percutaneous coronary intervention. *Quant Imaging Med Surg* 2020;**10**:2370–2373.
2. Zhao Y, He YH, Liu WX, Sun L, Han JC, Man TT, Gu XY, Chen Z, Wen ZY, Henein MY. Intramyocardial dissecting hematoma after acute myocardial infarction-echocardiographic features and clinical outcome. *Echocardiography* 2016; **33**:962–969.
3. Lee TJ, Roslan A, Teh KC, Ghazi A. Intramyocardial dissecting haematoma mimicking left ventricular clot, a rare complication of myocardial infarction: a case report. *Eur Heart J Case Rep* 2019;**3**:ytz056.
4. Vargas-Barrón J, Roldán FJ, Romero-Cárdenas Á, Vázquez-Antona CA. Intramyocardial dissecting hematoma and postinfarction cardiac rupture. *Echocardiography* 2013;**30**:106–113.
5. Mahmoud O, Mehra V. Two rare complications of myocardial infarction: a case report. *Eur Heart J Case Rep* 2019;**3**:1–4.
6. Konijnenberg LSF, Damman P, Duncker DJ, Kloner RA, Nijveldt R, van Geuns RM, Berry C, Riksen NP, Escaned J, van Royen N. Pathophysiology and diagnosis of coronary microvascular dysfunction in ST-elevation myocardial infarction. *Cardiovasc Res* 2020;**116**:787–805.
7. Spinelli L, Stabile E, Giugliano G, Morisco C, Giudice CA, Imbriaco M, Santoro M, Esposito G, Trimarco B. Intramyocardial dissecting hematoma in anterior wall ST elevation myocardial infarction: impact on left ventricular remodeling and prognosis. *Int J Cardiovasc Imaging* 2018;**34**:201–210.
8. Vargas-Barrón J, Roldán FJ, Romero-Cárdenas A, Molina-Carrión M, Vázquez-Antona CA, Zabalgoitia M, Martínez Rios MA, Pérez JE. Dissecting intramyocardial hematoma: clinical presentation, pathophysiology, outcomes and delineation by echocardiography. *Echocardiography* 2009;**26**:254–261.
9. Mousavi N, Cheezum MK, Aghayev A, Padera R, Vita T, Steigner M, Hulten E, Bittencourt MS, Dorbala S, Di Carli MF, Kwong RY, Dunne R, Blankstein R. Assessment of cardiac masses by cardiac magnetic resonance imaging: histological correlation and clinical outcomes. *J Am Heart Assoc* 2019;**8**:e007829.
10. Sakrana AA, Alzubaidi SAA, Shahat AM, Mahmoud AS, Naeim HA. Cardiovascular magnetic resonance and echocardiographic findings of a large thrombosed intramyocardial dissecting hematoma: a case report and a brief review of literature. *BJR Case Rep* 2020;**6**:20200028.
11. Betgem RP, de Waard GA, Nijveldt R, Beek AM, Escaned J, van Royen N. Intramyocardial haemorrhage after acute myocardial infarction. *Nat Rev Cardiol* 2014;**12**:156–167.
12. 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* 2018;**35**:260–266.
13. Miura A, Uemura Y, Takemoto K, Uchikawa T, Koyasu M, Ishikawa S, Mitsuda T, Imai R, Iwamiya S, Ozaki Y, Watanabe T, Watarai M, Ishii H, Murohara T. Early spontaneous remission of intramyocardial dissecting hematoma. *Intern Med* 2017;**56**: 1067–1070.
14. Jolicoeur EM, Dendukuri N, Belisle P, Range G, Souteyrand G, Bouisset F, Bouisset F, Zemour G, Delarche N, Harbaoui B, Schampaert E, Kouz S, Cayla G, Roubille F, Boueri Z, Mansour S, Marcaggi X, Tardif JC, McGillion M, Tanguay JF, Brophy J, Yu CW, Berry C, Carrick D, Høfsten DE, Engstrøm T, Kober L, Kelbæk H, Belle L. Immediate vs delayed stenting in ST-elevation myocardial infarction: rationale and design of the international PRIMACY Bayesian randomized controlled trial. *Can J Cardiol* 2020;**36**:1805–1814.
15. Gömöri K, Szabados T, Kenyeres É, Pipis J, Földesi I, Siska A, Dormán G, Ferdinandy P, Görbe A, Bencsik P. Cardioprotective effect of novel matrix metalloproteinase inhibitors. *Int J Mol Sci* 2020;**21**:6990. Published 2020 Sep 23.