CASE REPORT

Old complication, new presentation: a case of the spleen taking the bullet

Alexander Isted,¹ Francesco Fiorini,¹ Paula Mota²

¹School of Medicine, King's College London, London, UK
²Department of Cardiology, William Harvey Hospital, Kent, UK

Correspondence to
 Alexander Isted,
 alexander.isted@kcl.ac.uk

Accepted 12 June 2015

SUMMARY

We report a case of a 43-year-old man who presented to the accident and emergency department with acute abdominal pain. Ultrasound investigation showed non-specific splenic pathology and treatment for a splenic abscess was started. The patient later described a history of episodic, cardiac-sounding chest pain occurring at rest, the most recent case of which (6 days prior to admission) had been unusually severe. ECG showed anterior Q waves and aneurysm-type ST-T segment changes. Echocardiography, coupled with a CT scan of the abdomen, revealed the aetiology. The patient had sustained an anterolateral myocardial infarction, which had led to mural thrombus formation and secondary embolisation to the spleen, with no other end organ damage detected. The patient responded well to conservative management of the splenic infarct and initiation on the acute coronary syndrome pathway. Coronary angiography showed mild disease of the proximal left anterior descending artery. Cardiac MRI at 1 month confirmed an ischaemic aetiology.

BACKGROUND

Systemic embolisation of left ventricular mural thrombosis is a well-established event following myocardial infarction (MI), which has become far less common in the era of early revascularisation.¹ The clinical sequelae are highly variable depending on the end organs compromised by emboli, however, stroke is particularly notable due to the potentially catastrophic impact on morbidity and mortality. Left ventricular mural thromboembolic splenic infarction is rare² and, as in the case that we present, relatively fortuitous, considering the possible alternative sites of infarction.

CASE PRESENTATION

A 43-year-old man presented to accident and emergency department with a 24 h history of severe and sudden onset abdominal pain, diffuse across the epigastrium, left hypochondrium and left flank. The pain had been unremitting and building in severity over the course of the day. The patient reported vomiting on the previous day. Medical history consisted entirely of a single episode of pericarditis a year prior. Angiography performed at the time had been reported as normal. Family history was unremarkable. Premorbidly, the patient was a fully independent labourer with a 25 pack-year smoking history and an approximate alcohol intake of 18 units per week. He denied ever using recreational drugs. Physical examination revealed marked tenderness throughout the abdominal

regions described. The patient was mildly tachycardic at 106 bpm but afebrile.

INVESTIGATIONS

Chest X-ray revealed mild cardiomegaly. Abdominal ultrasound excluded gross pathology of the liver, gallbladder and pancreas, but demonstrated a focal region of hypoechogenicity measuring 67×60×35 mm in the subdiaphragmatic area of the spleen. Alongside elevated inflammatory markers, this was thought to be consistent with a splenic abscess, and intravenous piperacillin-tazobactam was given.

Later in his admission, further questioning revealed several episodes of chest pain over the preceding 2 months, which the patient had not sought medical attention for. All episodes had awoken him from sleep and were self-resolving over several hours, although one episode, 6 days prior to admission, was reported as the most severe and most protracted. This pain was symptomatically distinct from both the pain on admission and the pericarditis pain experienced the year before, and very suggestive of a cardiac nature: diffuse, crushing, retrosternal pain radiating to the anterior aspect of the jaw, associated with profuse sweating. At this point, an ECG was performed ([figure 1](#)). It demonstrated signs of old infarct (dome-shaped ST segment elevation, biphasic T waves and abnormal Q waves) in the anterolateral leads—features absent from his ECG from a year previously. Cardiac troponins were subsequently found to be elevated at 806 ng/L (>50 ng/L). A CT scan of the abdomen, coupled with transthoracic echocardiography, revealed a common aetiology. The abdominal CT scan ([figure 2A](#)) highlighted the intrasplenic collection identified on ultrasound, bordered by a large area of hypoperfusion, indicative of splenic infarction. A left-sided pleural effusion was also noted, thought to be secondary to the splenic collection. Echocardiography demonstrated markedly impaired left ventricular function (left ventricular ejection fraction (LVEF) <35%), with apical hypokinesis and two large left ventricular thrombi in situ, each approximately 1.5×1.5 cm in size ([figure 3A](#)). The summation of these findings was strongly suggestive of anterior MI leading to embolisation of left ventricular mural thrombi and subsequent splenic infarction.

DIFFERENTIAL DIAGNOSIS

The constellation of investigation findings made an MI the most likely diagnosis, but the patient's risk profile, most notably his young age and previously

CrossMark

To cite: Isted A, Fiorini F, Mota P. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-210299

Figure 1 ECG demonstrated dome-shaped ST segment elevation, biphasic T waves and abnormal Q waves in the anterolateral territories.

normal angiography, warranted exploration of possible non-atherosclerotic aetiologies. The key differential diagnoses to exclude were an acquired prothrombotic state and myocarditis, either viral or autoimmune.

TREATMENT

The patient was started on acute coronary syndrome protocol, including therapeutic-dose anticoagulation. Owing to the delay to presentation following the initial MI, primary percutaneous

coronary intervention was no longer indicated. The splenic lesion was not considered amenable to surgery and was managed with conservative supportive care.

OUTCOME AND FOLLOW-UP

Features of the presentation, moderately atypical for atherosclerotic disease, justified exclusion of an inflammatory cause. Thrombophilia screen was negative. Serology was positive for antinuclear antibody in a speckled pattern and the extractable

Figure 2 The splenic infarction seen in (A) four transverse view CT scan descending from the superior (i) to inferior (iv) aspects of the spleen; and (B) a transverse view true fast MRI with steady-state free precession single shot (arrow).

Figure 3 (A) Transthoracic apical two-chamber view echocardiogram showing two bright round echodense masses seen in the apex (asterisks). (B) Vertical long axis (two-chamber) view late gadolinium enhancement image showing a near full-thickness rim of subendocardial hyperenhancement in an apical distribution (arrows) surrounding an apical thrombus (asterisk). (C) Short-axis view late gadolinium delayed-enhancement image showing near full-thickness rim of subendocardial hyperenhancement in distribution of the left anterior descending artery (arrows).

nuclear antigen screen revealed a markedly elevated anti-ribonucleoprotein (anti-RNP) and mildly elevated anti-Smith (anti-Sm) autoantibody levels. The remaining serology results were all negative. These findings were consistent with a non-specific autoimmune process, possibly undifferentiated connective tissue disorder. To exclude acute myocarditis mimicking MI, a cardiac MRI was performed. The MRI demonstrated severely impaired LVEF with regions of ventricular wall thinning and akinesia, and mild left atrial dilation. The right atrium and ventricle appeared normal. In the distribution of the left anterior descending artery, there was extensive, nearly transmural, fibrosis, extending peripherally from the endocardium in a pattern consistent with an ischaemic aetiology (figure 3B, C). Overlaying this, in the apex, there was a laminar thrombus measuring 26×19×6 mm (figure 3B). Alongside the normal pericardium and lack of active inflammation, the conclusive diagnosis of MI was confirmed, excluding myocarditis. Absence of myocardial oedema indicated that the MI was chronic. The scan also identified the extensive infarction of the spleen (figure 2B). An elective angiogram was later performed, which demonstrated a small smooth tubular narrowing of the proximal long anterior descending artery and distal filling defects. On reviewing the previous angiogram with a higher suspicion of disease, minor atheroma was already present.

DISCUSSION

Systemic embolisation is a relatively acute complication post-MI and 90% of patients who develop it do so within the first 2 weeks.³ Incidence has declined significantly since the advent of reperfusion therapy.^{4–7} Early anticoagulation is effective in preventing mural thrombus formation and embolisation of existing thrombi,^{1–8} but patients with MI presenting late are at significant risk. Splenic infarction has a variable presentation, most commonly with acute onset left hypochondrial pain, and additional symptoms may include fever, nausea and/or vomiting, pleuritic pain and referred pain to the shoulder from diaphragmatic irritation (Kehr's sign). A third of cases are clinically asymptomatic.⁹ Splenic infarction is typically self-resolving with conservative management, although subcapsular haemorrhage and splenic abscess formation can occur. Indication for surgical splenectomy is confined to patients with complications or those whose symptoms do not abate.¹⁰ Our patient was discharged with oral anticoagulation, statin therapy, optimisation of heart dysfunction management and advice for smoking cessation.

Learning points

- ▶ This case demonstrates the need for diligence in eliciting any history of chest pain.
- ▶ Clinical suspicion of embolic events should be high within the first 2 weeks following myocardial infarction.
- ▶ ECGs must be performed in patients presenting with non-specific abdominal pain, in order to exclude atypical presentations of cardiac disease.

Contributors AI and FF obtained informed written consent from the patient, reviewed patient notes and images, and authored the initial drafts of the manuscript. PM led the team in caring for the patient, and contributed to the writing of subsequent and final drafts.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Loh E, Sutton MS, Wun CC, *et al.* Ventricular dysfunction and the risk of stroke after myocardial infarction. *N Engl J Med* 1997;336:251–7.
- 2 Jaroch MT, Broughan TA, Hermann RE. The natural history of splenic infarction. *Surgery* 1986;100:743–50.
- 3 Komrad MS, Coffey CE, Coffey KS, *et al.* Myocardial infarction and stroke. *Neurology* 1984;34:1403–9.
- 4 Nihoyannopoulos P, Smith GC, Maseri A, *et al.* The natural history of left ventricular thrombus in myocardial infarction: a rationale in support of masterly inactivity. *J Am Coll Cardiol* 1989;14:903–11.
- 5 Chiarella F, Santoro E, Domenicucci S, *et al.* PredischARGE two-dimensional echocardiographic evaluation of left ventricular thrombosis after acute myocardial infarction in the GISSI-3 study. *Am J Cardiol* 1998;81:822–7.
- 6 Rehan A, Kanwar M, Rosman H, *et al.* Incidence of post myocardial infarction left ventricular thrombus formation in the era of primary percutaneous intervention and glycoprotein IIb/IIIa inhibitors. A prospective observational study. *Cardiovasc Ultrasound* 2006;4:20.
- 7 Gianstefani S, Douiri A, Delithanasi I, *et al.* Incidence and predictors of early left ventricular thrombus after ST-elevation myocardial infarction in the contemporary era of primary percutaneous coronary intervention. *Am J Cardiol* 2014;113:1111–16.
- 8 Vandvik PO, Lincoff AM, Gore JM, *et al.* Primary and secondary prevention of cardiovascular disease: antithrombotic therapy and prevention of thrombosis, 9th ed: American College of chest physicians evidence-based clinical practice guidelines. *Chest* 2012;141(2 Suppl):e637S–68S.
- 9 Antopolsky M, Hiller N, Salameh S, *et al.* Splenic infarction: 10 years of experience. *Am J Emerg Med* 2009;27:262–5.
- 10 Hazanov N, Attali M, Somin M, *et al.* Splenic embolus: 13 cases from a single medical department. *Isr Med Assoc J* 2006;8:694–7.

Copyright 2015 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow