Reminder of important clinical lesson

Acute ischaemic ventricular inferoseptal defect in a pyretic patient: a diagnostic challenge

Martina Muscat, Etienne Laverse, Zaka Haq, Anwar Ahmad Chahal, Javed Akhtar

Cardiology Department, Queens Hospital, London, UK

Correspondence to Dr Etienne Laverse, etienne.laverse@imperial.ac.uk

Summary

A 78-year-old Caucasian male presented with a 5-day progressive history of fever, vomiting and dyspnoea. Initial clinical examination revealed fever, sinus tachycardia, hypotension, peripheral cyanosis and a systolic murmur. Investigations revealed a multiple organ dysfunction syndrome. A repeat trans-thoracic echocardiogram showed a basal inferoseptum aneurysm associated with an acquired ventricular septal defect not demonstrated on initial scan. An intra-aortic balloon pump was urgently inserted and he underwent surgical repair of the defect, 7 days after his initial presentation. He unfortunately died from postoperative complications.

BACKGROUND

Ventricular septal defect (VSD) is an important and often fatal consequence of myocardial infarction (MI). We present an atypical case of a postinfarct VSD with a late presentation. In the era of primary coronary intervention and advancements in peri MI therapy, the incidence of complications, such as VSD, has decreased significantly. Delayed or indeed misdiagnosis can occur as a result of low clinical suspicion and an unfamiliarity with atypical presentations. This case serves as an important reminder of potentially fatal sequelae of MI and VSD, thus allowing for prompt recognition and early intervention.

CASE PRESENTATION

A 78-year-old man presented with a 5-day progressive history of vomiting, dyspnoea and fever. He denied any history of chest pain. He had no significant medical history or family history and was on no regular medications. He had smoked variable amounts of cigarettes in the past.

On examination, he was febrile, tachycardic, hypotensive and peripherally cyanosed. There was a systolic murmur in the left lower sternal area. The patient deteriorated over 4 days with a multiple organ dysfunction syndrome and was managed in the critical care unit.

INVESTIGATIONS

Blood tests revealed raised inflammatory markers (white cell count of $15 \times 10^9/l$ and C reactive protein of 110 mg/l), a compensated metabolic acidosis, deranged liver function and evidence of acute kidney injury. The troponin I was 3.11 ng/ml (<0.04 ng/ml).

The ECG showed sinus tachycardia with T wave flattening in the inferior leads and T wave inversion in lead V6 (figure 1). There were no ST segment changes or pathological Q waves and therefore no ECG evidence of transmural infarction. The chest radiograph had a small right-sided pleural effusion with no obvious pulmonary congestion.

Preoperative coronary angiography demonstrated a dominant right coronary artery (RCA) with complete mid-

vessel occlusion after the right ventricular branch. The left coronary artery showed a severe segment of atheroma in the mid left anterior descending (LAD) artery. The circumflex was a small calibre vessel with no significant flow limiting lesions (figure 2).

An initial technically difficult echocardiogram (due to tachycardia and patient being in extremis) showed an inferoseptal ventricular aneurysm, with no colour flow seen to cross between the left and right circulations (figure 3). A repeat echocardiogram, 3 days later, demonstrated a basal inferoseptal aneurysm and a definite left to right shunt (figure 4). A preoperative transoesophageal echocardiogram (TOE) confirmed a large VSD and an associated inferior wall infarction.

TREATMENT

The ultimate treatment for the patient with VSD was surgical repair with a bovine pericardial patch. He also underwent concomitant coronary artery bypass grafting to the LAD and RCA.

OUTCOME AND FOLLOW-UP

The patient's postoperative TOE showed no residual VSD or valve dysfunction. The patient was managed in the intensive care unit postoperatively and unfortunately died from multi-organ failure including, haemorrhage, acute renal injury and failure to wean off inotropes.

DISCUSSION

Acquired VSD or ventricular septal rupture results from sudden occlusion of a coronary artery with an associated transmural infarction of the ventricular septum and adjacent ventricles.¹ Decreased cardiac output and left to right shunting leads to progressive decompensation.¹ The incidence of postischaemic VSD was 1 to 2% in the prethrombolytic era, which has improved to 0.2%.²⁻⁴ There is a high incidence on day 1 and days 3-5 following an acute MI.⁴

Early recognition of VSD can avoid clinical deterioration by early use of mechanical support to maintain

Figure 1 12-lead ECG at presentation, showing sinus rhythm with inferior T wave flattening and T wave inversion in V6.

Figure 2 Coronary angiogram 2D still showing occluded right coronary artery (solid arrow) and severe mid-vessel left anterior descending stenosis (dashed arrow).

haemodynamic stability⁵ and allow definitive lifesaving surgical intervention. The intra-aortic balloon pump works by counterpulsation that is, inflates in ventricular diastole and deflates in ventricular systole. This decreases the after-load of the left ventricle, improves coronary and renal perfusion and augments diastolic blood pressure.³

The diagnosis of acute MI was initially missed in this atypical case because of the signs and symptoms of sepsis, lack of chest symptoms and absence of ST changes or pathological Q waves on ECG. It is likely the patient completed his infarct several days before presenting with resolution of the electrical changes on the ECG. Moreover, the troponin I was only moderately raised in the presence

of acute renal injury and suspected sepsis. Furthermore, there was no clinical or radiological evidence of pulmonary oedema. His multiple organ dysfunction syndrome was all secondary to cardiogenic shock, which is the strongest predictor of early mortality.² MI activates the innate immune system in a similar way to infection. The acute inflammatory response can give the clinical picture of sepsis with fever postinfarct.⁶

Septal ruptures are said to occur more commonly in men with single coronary vessel disease presenting with their first anterior MI.⁷ Postinfarct VSDs manifest clinically by haemodynamic deterioration, a new systolic murmur and clinical presentation consistent with acute MI. Previous

Figure 3 A modified parasternal short axis view showing the basal inferoseptal aneurysm.

Figure 4 A modified subcostal view demonstrating trans-septal colour flow.

history of angina may lead to myocardial preconditioning and collateral formation, which may protect the myocardium from septal rupture after an acute MI.⁸

The overall mortality remains high in the reperfusion era.² Without surgical intervention, the survival is less than 10% after 1 month³ and it still has a high operative

mortality. Medical management has a 30 day mortality approaching 100%.¹ Right ventricular dysfunction, inferior VSD and shock predict poor outcome,¹ as does a short interval between MI and septal rupture.⁷

The preoperative TOE confirmed the VSD. In comparison to trans-thoracic echocardiography (TTE), TOE offers

high resolution imaging and evaluation of the entire ventricular septum.⁹ Two dimensional and colour Doppler echocardiography are sensitive but their main limitation is the presence of small acoustic windows in some patients.⁹

Surgical repair is the ultimate treatment of VSDs. However, optimal medical management prior to surgery is important. This involves reducing left ventricular filling pressures and afterload with the use of diuretics, inotropic support and haemo-filtration in oliguric cases.² Hospital survival for surgically treated patients in the Should We Emergently Revascularise Occluded Coronaries in Cardiogenic Shock trial and Global Utilisation of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial was 19 and 53%, respectively.² In the GUSTO trial, patients with VSD with cardiogenic shock had a mortality of 77%.

The timing of surgery is of crucial significance and there is a general consensus for early surgery in all suitable patients.^{3,4} Therefore as the incidence of VSDs decrease in the era of primary coronary intervention, it is all the more important that clinical suspicion and ability to recognise VSDs remain high, so that early intervention can offer the best chance of survival in this often fatal sequela of MI.

Learning points

- ▶ Acquired VSD results from sudden occlusion of a coronary artery with an associated transmural infarction of the ventricular septum.
- ▶ Cardiac injury can mimic infection as it leads to activation of the innate immune response.
- ▶ Early recognition of VSD can avoid clinical deterioration by early use of mechanical and pharmacological support to maintain haemodynamic stability.
- ▶ TTE is a non-invasive technique to help in diagnosing VSDs but failure to demonstrate a shunt on a TTE does not exclude a VSD and a TOE should be performed.
- ▶ Surgery is the definitive treatment. Inferior wall VSDs are technically more difficult to repair than anterior or posterior wall VSDs.

Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Sibal AK**, Prasad S, Alison P, *et al.* Acute ischaemic ventricular septal defect—a formidable surgical challenge. *Heart Lung Circ* 2010;**19**:71–4.
2. **Poulsen SH**, Praestholm M, Munk K, *et al.* Ventricular septal rupture complicating acute myocardial infarction: clinical characteristics and contemporary outcome. *Ann Thorac Surg* 2008;**85**:1591–6.
3. **Mantovani V**, Mariscalco G, Leva C, *et al.* Surgical repair of post-infarction ventricular septal defect: 19 years of experience. *Int J Cardiol* 2006;**108**:202–6.
4. **Maltais S**, Ibrahim R, Basmadjian AJ, *et al.* Postinfarction ventricular septal defects: towards a new treatment algorithm? *Ann Thorac Surg* 2009;**87**:687–92.
5. **Patanè F**, Grassi R, Zucchetti MC, *et al.* The use of Impella Recover in the treatment of post-infarction ventricular septal defect: a new case report. *Int J Cardiol* 2010;**144**:313–5.
6. **Ertl G**, Frantz S. Healing after myocardial infarction. *Cardiovasc Res* 2005;**66**:22–32.
7. **Deja MA**, Szostek J, Widenka K, *et al.* Post infarction ventricular septal defect - can we do better? *Eur J Cardiothorac Surg* 2000;**18**:194–201.
8. **Brush JE**, Cabin HS, Wohlgelemler D, *et al.* Ventricular septal rupture following a clinically unrecognized myocardial infarction. *Am Heart J* 1985;**110**:667–70.
9. **Ballal RS**, Sanyal RS, Nanda NC, *et al.* Usefulness of transesophageal echocardiography in the diagnosis of ventricular septal rupture secondary to acute myocardial infarction. *Am J Cardiol* 1993;**71**:367–70.

This pdf has been created automatically from the final edited text and images.

Copyright 2011 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.

Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Muscat M, Laverse E, Haq Z, Chahal AA, Akhtar J. Acute ischaemic ventricular inferoseptal defect in a pyretic patient: a diagnostic challenge. *BMJ Case Reports* 2011;10:1136/bcr.07.2011.4452, Published XXX

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