CASE REPORT

Left ventricular pseudoaneurysm formation in a patient presenting with a subacute myocardial infarction

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SUMMARY

Left ventricular pseudoaneurysm is a rare mechanical complication of acute myocardial infarction. In the present case, an 80-year-old man presenting with a subacute non-ST segment elevation myocardial infarction was found to have an occluded second obtuse marginal branch of the left circumflex coronary artery. Following the implantation of two drug-eluting stents, the patient developed no-reflow phenomenon. Coronary angiography 6 weeks later revealed persistence of the no-reflow phenomenon. During the left ventriculogram, a massive pseudoaneurysm was diagnosed and the patient successfully underwent emergency surgery. The persistence of no-reflow was likely due to the fact that the myocardial territory supplied by the infarct-related artery was completely necrosed resulting in persistent flow impairment through the vessel.

the diagnosis of new-onset atrial fibrillation, which spontaneously converted to sinus rhythm an hour later. A subsequent ECG revealed sinus rhythm with inferior Q-waves in leads III and aVF (figure 1). High-sensitivity Troponin T (hsTropT) was elevated at 1443 ng/L (normal range <14 ng/L), but the creatine kinase (CK) was within normal range at 129 U/L (normal range <190 U/L). Chest X-ray revealed Kerley B lines and small bilateral pleural effusions consistent with a diagnosis of decompensated heart failure. A diagnosis of subacute NSTEMI complicated by decompensated heart failure and new-onset paroxysmal atrial fibrillation was made. The patient was treated with 300 mg aspirin, 5000 IU unfractionated heparin and 40 mg intravenous furosemide and was transferred to our hospital for emergent coronary angiography.

BACKGROUND

A ventricular free-wall rupture is an infrequent complication of acute myocardial infarction, and when not followed by sudden death, it is referred to as subacute ventricular rupture. The latter can eventually lead to the formation of a left ventricular (LV) pseudoaneurysm. Herein, we describe the case of an 80-year-old man presenting with a subacute non-ST segment elevation myocardial infarction who subsequently developed a LV pseudoaneurysm.

CASE PRESENTATION

An 80-year-old man with a history of arterial hypertension and 40 pack-years smoking was referred by a peripheral hospital for coronary angiography because of a subacute non-ST segment elevation myocardial infarction (NSTEMI).

The patient initially presented 7 days prior to his general practitioner complaining of retrosternal chest pain and was given a diagnosis of bronchitis and was sent home with a prescription for bronchodilators. The patient nevertheless continued to experience recurrent episodes of retrosternal chest pain and weight gain of 5 kg and, therefore, he presented a week later to the emergency department of his local hospital. On clinical examination, his pulse was irregularly irregular with a heart rate of 132 beats per minute, auscultation revealed bilateral basal crepitations and he was noted to have bilateral leg oedema consistent with decompensated heart failure. An ECG confirmed

INVESTIGATIONS

Coronary angiography revealed two-vessel coronary artery disease with an occlusion of the second obtuse marginal (OM2) branch of the left circumflex coronary artery (LCX) (figure 2). In addition, significant stenoses were observed in the first diagonal branch of the left anterior descending (LAD) coronary artery and the distal segment of the LAD. The systolic LV function was preserved with an ejection fraction of 55% with inferoapical and inferomedial wall akinesia. Percutaneous coronary intervention (PCI) of the OM2 branch of the LCX was performed via right radial access. Following the administration of 5000 IU of unfractionated heparin, the lesion was crossed with a BMW Universal II guidewire and was predilated with a Ryujin Plus 2.0/20 mm semicompliant balloon catheter. Thereafter, a Resolute Onyx 2.5/38 mm stent was implanted in the OM2 branch of the LCX (figure 3). Following implantation of the stent, contrast injection revealed thrombolysis in myocardial infarction (TIMI) I flow, suggesting either no-reflow phenomenon or a dissection distal to the stent (see online supplementary video 1). Intracoronary verapamil was administered but flow improved only marginally from TIMI one to TIMI 2. At this point, it was decided to implant a second overlapping stent distal to the first stent (Resolute Onyx 2.25/12 mm) (figure 4). However, contrast injection following implantation of the second stent showed no improvement in TIMI II flow congruent with no-flow phenomenon (see online supplementary video 2). At this point, it was decided to terminate the

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Figure 1 12-Lead ECG showing Q-waves in leads III and aVF.

procedure and transfer the patient to the coronary care unit. During the hospitalisation, the CK peaked at 84 IU/L and the hsTropT peaked at 1581 ng/L. The patient continued to have paroxysmal atrial fibrillation, which converted to sinus rhythm following a loading dose of amiodarone. Triple therapy with aspirin 100 mg once daily, clopidogrel 75 mg once daily and apixiban 2.5 mg two times per day was commenced together with pantoprazole 40 mg and a maintenance dose of amiodarone 200 mg. The patient was transferred back to the peripheral hospital on day 3 with a plan to perform staged PCI of the diagonal branch and distal LAD in 6 weeks.

The patient remained hospitalised in the peripheral hospital for a further 8 days for treatment of decompensated heart failure. During this time, he also suffered an upper gastrointestinal bleed resulting in a 2.1 g/dL drop in haemoglobin. It was decided to stop aspirin and to treat with rivaroxiban lifelong and clopidogrel for 6 months and the haemoglobin remained stable thereafter. The patient underwent transthoracic echocardiography, which revealed a small circular haemodynamically insignificant pericardial effusion, which was interpreted to have occurred as a consequence of postinfarction pericarditis (pericarditis epistenocardica). The patient was discharged with an appointment for two subsequent

Figure 2 The second obtuse marginal branch of the left circumflex coronary artery is occluded (arrow).

Figure 3 A 2.5×38 mm drug-eluting stent was implanted into the second obtuse marginal branch of the left circumflex coronary artery.

elective follow-up transthoracic echocardiograms, which were performed 1 and 2 weeks postdischarge, respectively. The latter showed persistence of the small pericardial effusion, which was nonetheless reported to have decreased in size.

Four weeks following the last echocardiogram, the patient presented for the staged PCI procedure. On admission, the patient reported feeling unwell and complained of progressively worsening shortness of breath, which was occurring at rest. Coronary angiography revealed patent stents in the OM2 branch of the LCX, but surprisingly, the TIMI II flow had persisted and

Figure 4 A 2.25×12 mm drug-eluting stent was implanted distal to the first stent in an effort to improve blood flow in the second obtuse marginal branch of the left circumflex coronary artery.

Figure 5 A large defect in the posterior wall of the left ventricle (LV) near the apex under the posterior leaflet of the mitral valve (A). A polytetrafluoroethylene patch was sutured over the LV defect (B).

had not improved since the index procedure 6 weeks previously (see online supplementary video 3). Left ventriculography revealed a massive LV pseudoaneurysm into which the pigtail catheter had prolapsed (see online supplementary video 4). At this point, a transthoracic echocardiogram was performed and the cardiac surgeons were called. The echocardiogram confirmed the large LV pseudoaneurysm.

TREATMENT

Preoperative transoesophageal echocardiography showed a large aneurysm in the posterior wall of the LV near the apex and under the posterior leaflet of the mitral valve. The patient ultimately underwent on-pump cardiac surgery comprising a two-vessel coronary artery bypass operation (left internal mammary artery to proximal LAD with jump graft to the distal LAD), an aneurysmectomy and a polytetrafluoroethylene patch was sutured over the LV defect (figure 5). In addition, a bipolar MAZE procedure was performed to treat the paroxysmal atrial fibrillation. The patient had an uneventful recovery and was discharged to cardiac rehabilitation 10 days following the successful operation.

OUTCOME AND FOLLOW-UP

The patient made an excellent recovery, reported that he was doing well at his outpatients appointment 1 year after the operation.

DISCUSSION

LV pseudoaneurysm is a rare mechanical complication of acute myocardial infarction and occurs when cardiac rupture is contained by adherent pericardium or scar tissue.¹ Unlike a true LV aneurysm, a LV pseudoaneurysm contains no endocardium or myocardium. Free intrapericardial rupture typically results in cardiac tamponade and death (acute LV rupture). Less frequently, cardiac rupture is contained and LV pseudoaneurysm formation occurs (subacute LV rupture).¹ A recent long-term follow-up study over 30 years including 6678 patients with consecutive STEMI, of whom 425 had an LV rupture, showed that the prevalence of LV rupture has reduced from 6.2% during the period between 1977 and 1982 to 3.2% between 2001 and 2006.² This reduction in the prevalence of LV rupture coincided with the increasing use of reperfusion therapies such as thrombolysis in the 1980s and primary PCI in the 1990s, in addition to the increasing use of medications such as beta-blockers, ACE inhibitors, mineralocorticoid antagonists and antiplatelet agents throughout the 30-year period.² The mortality rate from LV rupture nevertheless remains very high ranging between 75% and 96%.² The present case is unique in that the myocardial territory supplied by the infarct-related artery (ie, the OM2 of the LCX) was completely necrosed, thereby disrupting the run off though the infarct related artery resulting in persistent flow impairment through the vessel.

Learning points

- ▶ The diagnosis of left ventricular (LV) pseudoaneurysm is difficult as most patients present with non-specific symptoms such as shortness of breath and lethargy and therefore a high index of suspicion is needed to make the diagnosis.
- ▶ It is important to consider the diagnosis of LV rupture among patients with high-risk features such as age >55 years, first transmural myocardial infarction, persistent ST-segment elevation or chest pain and cardiogenic shock or sudden pulseless electric activity arrest.
- ▶ The constellation of symptoms, signs and imaging findings from echocardiography and ventriculography is important to make the diagnosis.
- ▶ While the prevalence of LV rupture has reduced over the past 30 years due to the increasing use of reperfusion therapies, beta-blockers and ACE inhibitors and mortality rates remain high.

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