CASE REPORT

Spontaneous closure of ventricular septal perforation following percutaneous coronary intervention for acute myocardial infarction

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SUMMARY

We report on an 84-year-old woman with anteroapical acute myocardial infarction. Emergency coronary angiography revealed the occlusion of proximal left anterior descending artery without collateral circulation, and percutaneous coronary intervention was performed. Two drug eluting stents were implanted, and the procedure was concluded with thrombolysis in myocardial infarction grade 3 without complications. Postoperatively, no murmur was audible on auscultation and no shunt flow was observed on transthoracic echocardiography (TTE), and normal blood pressure was maintained. On day 2, however, the patient's vital signs deteriorated to a state of shock and systolic murmur appeared at the apical region. TTE showed a left-to-right shunt in the apical septal region, and ventricular septal perforation was diagnosed. Although emergency surgery was considered, the patient's vital signs improved the following day. The disappearance of the cardiac murmur and the shunt was confirmed. The clinical course was uneventful thereafter, and the patient was discharged.

BACKGROUND

Ventricular septal perforation (VSP) is a fatal complication associated with myocardial infarction. Without appropriate treatment, 33% of the patients will die within 24 hours and 65% within 1 week.¹ It is generally considered that emergency surgery is necessary to treat this condition. Spontaneous closure of postinfarction VSP was reported to be extremely rare, and we were able to identify only five case reports of this condition.^{2–6} It is discussed in these reports that spontaneous closures of VSP are associated with findings, including apical aneurysm formation, thrombus occlusion,² thrombus in the right ventricular partially occluded⁶ and small size VSP.^{2–4} We experienced spontaneous closure of VSP within 24 hours after onset which occurred a lot earlier than the reported cases. The underlying mechanisms of the closure remain unclear, and data on more cases need to be accumulated.

CASE PRESENTATION

The case presented in this report was an 84-year-old woman with a 24-year history of hypertension. In February 2017, the patient suddenly experienced chest pain at rest, which improved after resting for approximately half an hour. Thereafter, she had breathlessness with light exertion, because of

which she visited our department the following day. Upon arrival, her blood pressure was maintained at 111/63 mmHg, but percutaneous oxygen saturation was low at 90% in room air, and moist rales were audible throughout the lung fields. Cardiac auscultation revealed pathological third heart sound without clear murmurs. Blood test findings revealed elevated myocardial enzyme levels with creatine kinase/creatinine kinase-MB (CK/CK-MB) at 2753/215 IU/L and troponin I at 62.9 ng/L. Chest X-rays indicated pulmonary oedema (figure 1A), and a 12-lead electrocardiography revealed Q wave and ST elevation in leads V1–4 (figure 2A). On transthoracic echocardiography (TTE), decreased anteroapical wall movement was noted, with a low left ventricular ejection of 35% (figure 3A, B). No clear shunt flow was noted. On the basis of these findings, the patient was diagnosed with anteroapical ST elevation acute myocardial infarction equivalent to Killip class 3. Emergency intra-aortic balloon pumping (IABP) and coronary angiography (CAG) were performed on the same day. CAG revealed 90% stenosis at #6 (ostium) and occlusion at #7 (figure 3C), and percutaneous coronary intervention was performed. Two Resolute IntegrityTM stents (Medtronic, Minneapolis, Minnesota, USA) were placed, and the procedure was concluded with thrombolysis myocardial infarction grade 3 flow without complications (figure 3D). Peak CK/CK-MB was at 3469/390 IU/L, and the value quickly declined to the normal range. IABP was continued postoperatively, and the blood pressure was maintained at approximately 120/70 mmHg. Heart failure treatment was started with non-invasive positive pressure ventilation (NPPV) and diuretics. No cardiac murmurs were audible, and TTE did not reveal any left-to-right shunts. On hospital day 2, although there was no obvious change in ECG (figure 2B), the patient's vital parameters deteriorated to a state of shock with a systolic blood pressure of approximately 70 mmHg, even with IABP 1:1 backup. Auscultation revealed a systolic murmur with an apical thrill that was not detected at admission.

TTE did not reveal mitral valve insufficiency due to papillary muscle rupture or pericardial effusion. However, a left-to-right shunt of approximately 2.7 mm was noted in the region of the apical septal akinesis (figure 4A, B), and VSP was diagnosed.

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Figure 1 Chest X-rays on admission indicated pulmonary oedema (A), and at discharge, pulmonary congestion improved (B).

IABP was continued with dobutamine. Although emergency surgery was considered, the patient's vital parameters improved the following day with a blood pressure of 120/60 mmHg. The disappearance of the cardiac murmur and the left-to-right shunt was confirmed (figure 4C, D). Following this, IABP was continued until hospital day 7. Heart failure treatment was implemented with NPPV, diuretics and dobutamine. The patient's acute heart failure subsequently improved (figure 1B), with no reappearance of cardiac murmurs and left-to-right shunts.

OUTCOME AND FOLLOW-UP

The clinical course was uneventful thereafter, and the patient was discharged.

DISCUSSION

VSP was first reported by Latham in 1847.⁷ In 1957, Cooley was the first to report on successful surgery for VSP.⁸ Although VSPs were considered to occur as complications in 1–2% cases of acute myocardial infarction,⁹ recent reports have indicated that improvements in treatment methods for myocardial infarction have resulted in this rate dropping to 0.17–0.37%.^{10–13} Without appropriate treatment, mortality within 24 hours occurs in 33% patients and within 1 week in 65% patients.¹ Risk factors include initial infarction, old age, female sex, hypertension, the absence of collateral circulation, thrombolytic therapy, chronic kidney disease and congestive heart failure.^{10 12 14–16} Most of these risk factors were present in our patient.

Figure 2 A 12-lead electrocardiography on admission revealed Q wave and ST elevation in leads V1–4 (A). On hospital day 2, when the ventricular septal perforation was confirmed, ECG did not change markedly (B). aVR, augmented Vector Right; aVL, augmented Vector Left; aVF, augmented Vector Foot.

Figure 3 Transthoracic echocardiography on admission revealed decreased anteroapical wall movement with a low left ventricular ejection of 35% (A, B). Coronary angiography revealed 90% stenosis at #6 (ostium) and occlusion at #7 (C), and percutaneous coronary intervention was performed. Two Resolute Integrity stents were placed, and the procedure was concluded with TIMI3 flow without complications (D). A: Systolic phase. B: Diastolic phase.

Because a new cardiac murmur appeared with worsened vital signs following myocardial infarction, we considered acute mitral valve insufficiency due to papillary muscle rupture, left ventricular free wall rupture and VSP for the differential diagnosis. TTE indicated no papillary muscle rupture, mitral valve insufficiency or any new pericardial effusion, suggesting left ventricular free wall rupture. Because a left-to-right shunt flow was observed in the region of the apical septal akinesis, VSP was diagnosed.

In accordance with the areas perfused by the coronary artery, VSP often occurs in the apical side in cases of anteroapical myocardial infarction and in the base side for cases of inferior and lateral wall myocardial infarction.^{17 18} As our patient exhibited anteroapical myocardial infarction, the VSP observed in the apical region is consistent with the expected findings.

There have been very few reports of spontaneous closure of VSP due to myocardial infarction, and we were able to identify only five reported cases.²⁻⁶ The discussions on spontaneous closure in these reports indicated findings, including apical aneurysm formation, thrombus occlusion,² thrombus in the right ventricular partially occluded⁶ and a small size VSP.^{2 4}

We also considered evaluating the degree of shunt formation in our patient based on the left ventriculography and intracardiac oxygen saturation. However, we were unable to perform such testing as her vital parameters were extremely unstable at VSP onset. Despite this, the fact that the perforation associated with the left-to-right shunt had a diameter of approximately 2.7 mm on TTE suggested that it was small. The region in which VSP appeared (apical septum) was akinetic, and it appeared that the decreased wall movement had caused a tendency for thrombus

formation because of decreased blood flow. The deterioration of the vital parameters of the patient to a state of shock and akinesis along with the consequent decrease in blood in areas already suffering from low blood flow may have combined in a complex manner to promote thrombus formation, resulting in its spontaneous closure.

Haemodynamics is considered to be dependent on the VSP defect size.¹⁷ However, the patient went into shock despite the VSP size (2.7 mm) being smaller than that in other cases.^{2 4}

We think this was because the cardiac infarction site was extensive and accompanied by severely decreased left ventricular function such that even a small defect size could cause haemodynamic instability. However, because there are no reports supporting this reasoning, further investigation is required.

IABP had been performed before CAG in our patient. IABP mechanically decreases afterload by increasing the cardiac output. Therefore, in cases of VSP, its routine concomitant use is recommended.¹⁷ The question of whether this leads to spontaneous closure remains unclear because of the absence of reports describing this until now; however, it appears to have had a role in contributing to haemodynamic stability.

In these four cases,²⁻⁵ a period ranging from several months to several years was required for closure to occur. In contrast, spontaneous closure occurred in our patient within approximately 24 hours. This reason may be because the perforation size was smaller than that in other cases^{2 4} reported in the literature and that blood pressure had been maintained in the four cases,²⁻⁵ with none being in a state of shock. It is possible that the decreased blood flow rate caused by the state of shock promoted thrombus formation to a greater extent than in the other reported cases.

Figure 4 Transthoracic echocardiography showed a left-to-right shunt with a 2.7 mm diameter defect in the region of the apical septal akinesis (A, B), and a ventricular septal perforation was diagnosed. The disappearance of the cardiac murmur and the left-to-right shunt was confirmed on hospital day 3 the next day ventricular septal perforation was confirmed (C, D). LA, left atrium; LV, left ventricular; RA, right atrium; RV, right ventricular.

There have been no previous cases of spontaneous closure occurring within such a short period of time, and the reason remains unclear.

In this report, we presented a rare, valuable case of VSP. Reports of cases of VSP that exhibit spontaneous closure are extremely rare and much of the mechanisms underlying spontaneous closure remain unclear. As there are limited reports in the literature supporting the inferences made in our case, data on more cases need to be gathered.

Learning points

- ▶ Ventricular septal perforation (VSP) is a fatal complication-associated myocardial infarction, and careful observation is necessary. Especially, careful auscultation and transthoracic echocardiography are important.
- ▶ There are possibilities that VSP closed spontaneously.
- ▶ It is speculated that apical aneurysm formation, thrombus occlusion and a small size VSP are the mechanisms of VSP spontaneous closure.

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