Embolic Myocardial Infarction and Left Ventricular Rupture Due to Mitral Valve Endocarditis

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A left ventricular rupture due to embolic myocardial infarction is extremely rare. A 72-year-old woman developed an acute embolic myocardial infarction and mitral regurgitation due to infective endocarditis. Two days after the infarction, a left ventricular free wall rupture occurred after transesophageal echo examination. She received an epicardial patch and mitral valve replacement. Perioperatively, an intra-aortic balloon pump and long-term antibiotics were used. The postoperative course was uneventful, and she is doing well 10 months after surgery. (Ann Thorac Cardiovasc Surg 2007; 13: 206–208)

Key words: left ventricular rupture, embolic myocardial infarction, infective endocarditis

Introduction

Systemic embolization, including that of the coronary arteries, is not rare in infective endocarditis (IE). However, transmural infarction and left ventricular rupture due to coronary embolism are quite rare.

Case

A 72-year-old woman, who had a history of exodontias and non-steroidal anti-inflammatory drug (NSAID) use for backache, developed sudden onset acute myocardial infarction (AMI) and mitral regurgitation (MR). An electrocardiogram (ECG) showed poor R wave progression in V2–6 and inverted T wave in II, III, aVf, and V1–4. Transthoracic echo (TTE) demonstrated grade III MR and vegetation on the anterior leaflet. Hypokinesis was observed in the posterior-inferior and apical anterior of the left ventricle. Coronary angiography (CAG) demonstrated a distal left anterior descending coronary occlusion with otherwise normal coronary arteries (Fig. 1). Blood cultures identified gram-positive cocci (a few days later, Streptococcus constellatus was identified).

The patient was diagnosed with acute embolic myocardial infarction and IE-associated MR. We initiated conservative treatment with diuretics and antibiotics and planned an elective mitral valve operation.

Two days after the AMI attack, she underwent transesophageal echo (TEE). Fifteen minutes after TEE, she felt chest discomfort and vomited. Her blood pressure and heart rate dropped suddenly, and she entered shock status. No significant change of cardiac motion or pericardial effusion was seen by transthoracic echocardiogram. An ECG demonstrated significant ST degradation in V3 and V4 versus pre-TEE. Immediately, an intra-aortic balloon pump (IABP) was inserted, after which her hemodynamic status improved. A new coronary problem was ruled out by another emergent CAG. After the CAG (2 h after shock), a substantial amount of pericardial effusion was noted by TTE. Left ventricular free wall rupture (LVFWR) was suspected. She was transferred to the operating room.

At operation: 500 mL of bloody fluid was drained from the pericardium. Active bleeding had already stopped. Thick thrombus and pericardial adhesions had sealed a left ventricular rupture. After extracorporeal circulation was established, the thrombus was removed. There were vegetations on both the anterior and posterior leaflets of
the mitral valve (maximum size 6×14 mm), and a chordal rupture was noted in the posterior leaflet. The mitral valve was replaced by a bileaflet mechanical prosthesis. The adherent pericardium (2×2 cm area) on LVFWR was left as it was (Fig. 2). To reinforce the LVFWR area, an autopericardial patch (5×7 cm) was widely and continuously sutured. Fibrin glue was inserted between the epicardium and the pericardial patch.

Postoperatively, IABP was used for 7 days to reduce the LV after load. The antibiotics, penicillin G (for 24 days) and gentamicin sulfate (for the initial 7 days) were administered. She recovered soon uneventfully and remains well 10 months after surgery.

Discussion

The incidence of coronary embolism in IE has been reported to be 12%. However, it is extremely uncommon for a coronary embolism to develop into transmural infarction and/or LVFWR.

Most coronary embolism occur in the LAD. The take-off and downward courses of the LAD are more favorable for embolization than those of the right and left circumflex coronaries, which run at 90 degree angles from the parent trunks.

Mycotic coronary or ventricular aneurysm may cause cardiac rupture in a patient with IE. Mycotic aneurysm occurring in the region of a myocardial infarction carries with it a sevenfold higher risk of cardiac rupture compared with an without abscess. However, we anticipated that this case was not associated with a mycotic aneurysm of either the coronary or ventricular wall, since the LVFWR occurred too early after embolism and CAG did not show a coronary aneurysm. The recognised risk factors of LVFWR are older age, female sex, previous hypertension, a first lateral or anterior wall AMI, no previous angina, no LV hypertrophy, and transmural infarction. Other risk factors for LVFWR are the use of NSAIDs.
and/or corticosteroid at the time of AMI. In an autopsy study, Silverman reported that half the cases of LVFWR had received NSAIDs and/or corticosteroid. Anti-inflammatory agents may lead to LVFWR by interfering with healing or leading to infarct expansion.

Classical LVFWR usually produces symptoms within the first 24 hours after an AMI and almost always by the end of the first week. We guessed that LVFWR was induced by temporary increased blood pressure during TEE examination. In most cases, sudden hemodynamic collapse is followed quickly by electromechanical dissociation and death. Furthermore, sudden hypotension and bradycardia, often with cyanosis and loss of consciousness, are frequent indicators of impending rupture. Clinical manifestations depend on the amount and rate of pericardial bleeding. Rarely, an adherent thrombus or pericardial adhesions will seal a ventricular rupture.

In this case, when the circulation collapsed, the ECG showed an ST change and pericardial effusion could not be identified by TTE. So, we initially suspected a possible new MI event or vasovagal reflex. We therefore inserted IABP immediately and underwent emergency CAG. The TTE after the CAG eventually demonstrated a substantial amount of pericardial effusion. This indicated that the oozing-type LV rupture may not be diagnosed by echocardiogram at a very early phase. The ECG changes reflect the slowly outward-advancing intramyocardial hemorrhage, while the pleuritic pain and emesis are caused by the distension and irritation of the epicardium.

The definitive treatment for LVFWR is emergency surgical repair; more often, a pericardial patch is placed with either biological glue or epicardial sutures. Other surgical techniques include infarctectomy with patch placement and ventricular wall reconstruction. Opening of the left ventricle may be indicated only when an intraventricular defect like a septal rupture co-exist.

IABP support is a widely accepted treatment for ventricular rupture complicating AMI. Systolic unloading, with the reduction of both afterload and wall tension by IABP, is effective for minimizing postoperative bleeding and the possible risk of re-rupture.

Although we did not perform coronary bypass grafting, this procedure may be beneficial, particularly if a target vessel was defined by preoperative CAG. Percutaneous coronary intervention is another possible revascularization option. Thrombolysis, which may lead to intra-cranial hemorrhage, should generally be avoided.

Conclusion

We treated a patient with coronary embolism due to an IE on the mitral position, who developed LVFWR after TEE examination two days after AMI attack. She recovered well with an epicardial patch, mitral valve replacement, IABP support, and long-term antibiotics.

References