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CE - LETTER TO THE EDITOR

Ventricular septal defect after percutaneous coronary intervention in acute myocardial infarction

A clinical study of two cases

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We present a report concerning two patients who developed a ventricular septal defect (VSD) complicating an acute myocardial infarction (MI) admitted to our intensive care unit last year.

Clinical cases

A 77-year-old, diabetic woman, without a prior history of hypertension and cardiac disease was admitted to our department for anterior acute MI about 6 h after the onset of symptoms. The ECG showed ST elevation in anterior leads. The Troponin I level was 88 ng/ml. She underwent emergency coronary arteriography, which showed three vessels disease. The culprit lesion was a sub-occlusive stenosis with thrombus in the mid-left anterior descending artery—a type B2 lesion, according to the AHA/ACC classification—with TIMI I distal flow. A primary angioplasty was performed with implantation of a TITAN 2 2.5 × 16 at 12 atm, after a pre-dilatation of the lesion with a balloon Fire Star 2 × 15 at 12 atm. The final result was TIMI II-III flow without any residual narrowing. Moreover, an eccentric 90%, focal stenosis of RCA and proximal stenosis of MO2, were detected.

Echocardiogram showed an apical aneurysm and an ejection fraction of about 45%, without mitral insufficiency. Diastolic flow velocities were: peak $E = 0.92$ m/s, with a deceleration time of 260 ms and peak $A = 1.07$ m/s (E/A ratio $= 0.90$).

The patient was treated with Aspirin, Clopidogrel, ACE inhibitors and Metoprolol.

Six hours after PCI, Troponin I was decreased to 56 ng/ml. Moreover, the ST elevations were reduced.

After about 24 h, the patient suddenly complained of dyspnoea. The heart rate was 105 beats/min, blood pressure was 95/60 mmHg. Physical examination revealed tachypnoea, pulmonary rales and a 3/6 Levine holosystolic murmur on the left sternal border. She was also observed to be oliguric. The ECG again showed ST segment elevation in the anterior leads, and the Troponin I was 22 ng/ml.

The beta blocker medication was discontinued, and inotropic agents (Dobutamine and Dopamine at 5–10 mcg/Kg/m), oxygen and Furosemide were administered. Transthoracic echocardiography confirmed abnormalities of regional motion, furthermore the ejection fraction tended to increase to 50%. Evaluation of diastolic function revealed an inversion of E/A ratio (>1). In addition the right ventricle was mildly dilated, but the ratio of right/left ventricular diastolic diameters remained <2/3; right ventricular systolic function was preserved (tricuspid annular systolic motion = 25 mm).

Colour-Doppler examination demonstrated a moderate left to right shunt through a small defect in the apical segment of interventricular septum (Fig. 1). In the absence of tricuspidal regurgitation, a pulmonary arterial pressure was not detectable.

The patient was promptly transferred to cardiac surgery where an intra-aortic balloon pump (IABP) was first inserted, to allow further stabilization, and an emergency operation was then carried out. This included a coronary artery bypass graft to the RCA and concomitant surgical

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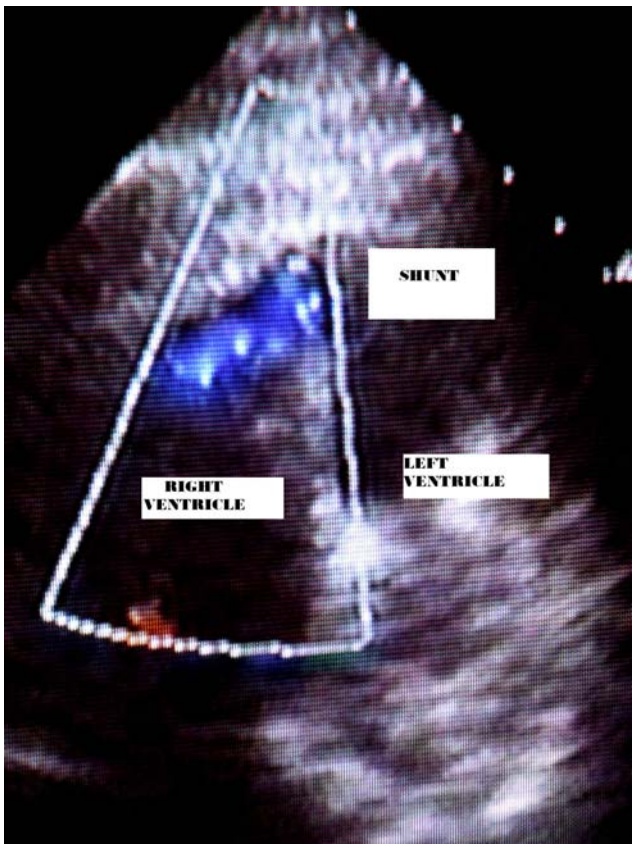

Fig. 1 Colour-Doppler echocardiography shows moderate interventricular shunt, from apical four-chamber view

repair of a large VSD (2.5 cm in diameter) through a left anterior ventriculotomy. A large area of the septum, the apex and part of the anterior free wall were clearly infarcted with extremely friable tissues and therefore the technique of "Infarct exclusion" (proposed by David [1]) was utilised using a large pericardial patch. Weaning from cardiopulmonary bypass was achieved easily with the assistance of the IABP. In the intensive care unit, the IABP and catecholamine infusion were necessary for 5 days. The subsequent recovery was slow but uneventful, and the patient was discharged for rehabilitation. At 5 months follow-up the patient was in satisfactory condition.

The second patient was an 80-year-old man, without a medical history of cardiovascular disease, who was transferred to our catheter laboratory to undergo coronary angiography because of recurrent chest pain 60 min after thrombolysis for an acute anterior ST elevation myocardial infarction.

Coronary angiography disclosed a sub-occlusive stenosis with thrombus burden in the mid-left anterior descending artery—type B1 lesion according to AHA/ACC classification—without additional significant stenosis in the other branches. The whole LAD had TIMI II distal flow. Using a 6 Fr FL guiding catheter and a BMW guide wire (0.014 in.), the lesion in the mid-left anterior descending artery was crossed easily without any complications. A Pronto^{TM} V3 catheter was used for thrombus extraction. After partial thrombectomy but with persistence of haziness, an intracoronary eptifibatide bolus was given. The lesion was directly stented using a 3.0 × 13 mm cobalt chromium Coroflex Blue stent at 16 atm with final TIMI II-III distal flow. Post procedural IV eptifibatide infusion was continued.

Soon after PCI, echocardiography evidenced normal chamber sizes with akinesis of the distal septum and apex. An ejection fraction of 44% was detected. Moreover, diastolic flow examination revealed an *E*/*A* ratio <1 (peak $E = 0.8$ m/s and peak $A = 1.2$ m/s). A mild mitral regurgitation was also detected. He was treated with Aspirin, Clopidogrel, low doses of beta blocker and ACE inhibitors. A few hours after the procedure, the patient suddenly manifested pulmonary oedema and cardiogenic shock. A new onset systolic murmur suggested the presence of a septal defect. An echocardiogram was performed promptly. This indicated dilatation of the right ventricle (whose area from an apical view was almost equal to that of left ventricle) and from a mild tricuspidal insufficiency that was calculated to have a right atrio-ventricular gradient of about 30 mmHg. Left ventricular size and systolic and diastolic functions were substantially unmodified. A left to right shunt was evidenced through a septal defect.

The patient died after a little while, despite Dopamine, Dobutamine and an adrenaline infusion rather than intra-aortic balloon counterpulsation.

Comments

Post myocardial infarction ventricular septal defect formation is a rare but catastrophic complication, being responsible for 5% of the overall deaths in patients with an acute MI [2–5]. In the pre-thrombolytic era, the range of incidence varied from 1 to 3% [2, 4]. At present, thrombolysis [6] and PCI [7] have decreased the incidence of septal rupture to about 0.2%. Early reperfusion probably reduces the extent of myocardial necrosis, which is the most important predictive factor of mechanical complications. In the SHOCK trial registry, ventricular septal rupture was responsible for about 4% of the cases of cardiogenic shock complicating myocardial infarction [5]. Women and elderly patients are more susceptible to septal rupture [2, 4–6]. Other risk factors include hypertension, no history of previous MI or angina, lack of collateral circulation and delayed treatment after onset of symptoms [2, 7, 8]. A septal rupture usually appears in the first week after acute MI, however, most cases occur within 24 h [2, 7]. Typical clinical manifestations are sudden cardiogenic

shock and pulmonary oedema. The physical examination reveals a loud systolic murmur often associated with a palpable thrill. Echocardiography is a superb tool for confirming of the presence of an interventricular septal rupture in patients with acute myocardial infarction [9]. Despite dramatic improvements in diagnosis and therapy, the overall in-hospital mortality, with medical treatment, remains extremely high (range 73.8–87%) [7, 8]. When surgery is performed before onset of circulatory collapse, survival is about 50%[7], otherwise a perioperative mortality of more than 80% has been observed in patients with cardiogenic shock complicating septal defect [5, 8, 10–12]. Consequently, surgical therapy should be performed as soon as possible, after rapid stabilization with inotropes and intraortic counterpulsation when necessary [11–13]. Because concomitant myocardial revascularization improves outcome, coronary angiography should always be performed before surgery.

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