Myocardial infarction and left ventricular free wall rupture in a patient with a prior pericardiectomy

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A 59-year-old man with an inferolateral myocardial infarction and cardiogenic shock was found to have extensive intrathoracic hemorrhage in communication with the left ventricle. His remote pericardiectomy precluded hemopericardium and tamponade, and permitted the establishment of an unusual diagnosis and subsequent closure of the site of myocardial perforation.

Key Words: *Acute myocardial infarction; Cardiac tamponade; Cardiogenic shock; Myocardial free wall rupture; Pericardiectomy*

Left ventricular free wall rupture (LVFWR) is a rare complication of acute myocardial infarction (AMI), occurring in approximately 2% of cases (1), and is often fatal because of the development of hemopericardium and tamponade. We describe a patient in whom free wall rupture occurred in the setting of previous pericardiectomy. Despite ongoing mediastinal hemorrhage, the diagnosis was established at cardiac catheterization and by computed tomographic scanning of the thorax, and surgical repair was performed.

CASE PRESENTATION

A previously well 59-year-old man suddenly developed left shoulder pain, followed by intense central chest pain with bilateral arm radiation and diaphoresis while picking up garbage. When seen at a local nursing station, his heart rate was 140 beats/min and his blood pressure was 80/50 mmHg. An electrocardiogram showed sinus rhythm with complete right bundle branch block. There was ST segment elevation in the inferolateral leads and ST segment depression in leads V1 through V4.

His cardiac risk factors included cigarette smoking, borderline hypertension and diet-controlled diabetes. He had undergone pericardiectomy for tuberculous pericarditis in 1970. He had been taking no medications.

He was treated with acetylsalicylic acid. Thrombolytic therapy was not administered because of a concern about the possibility of aortic dissection. On arrival to the emergency department at the St Boniface General Hospital (Winnipeg, Manitoba), his chest discomfort persisted. His heart rate was 146 beats/min and his blood pressure was 86/54 mmHg. There were no signs of congestive heart failure and no heart murmurs. The arterial pulses were intact.

The peak creatine phosphokinase and troponin T levels were 1202 U/L and 5.97 µg/L, respectively. A chest x-ray revealed right pleural effusion with an unusual dense area in the region of the right hilum.

Infarctus du myocarde et rupture de la paroi ventriculaire gauche libre chez un patient ayant déjà subi une péricardectomie

Un homme de 59 ans, victime d'un infarctus du myocarde inférolatéral suivi d'un choc cardiogénique, a présenté une hémorragie intrathoracique profuse à partir du ventricule gauche. Ses antécédents de péricardectomie écartaient la possibilité d'hémopéricarde et de tamponnade et ont permis d'établir ce diagnostic inhabituel et de corriger ensuite la perforation myocardique.

A contrast left ventriculogram revealed extravasation of contrast through the posterolateral wall of the left ventricle. Coronary angiography revealed 70% to 75% occlusion of the mid-distal circumflex artery. The mid-distal right coronary artery and the left anterior descending artery had insignificant stenosis of 40% to 50%.

A transient episode of cardiac arrest with pulseless electrical activity required endotracheal intubation, inotropic support and insertion of an intra-aortic balloon pump. His hemoglobin level fell from 112 g/L to 67 g/L, and an urgent blood transfusion was initiated.

Emergency transthoracic and transesophageal echocardiography were performed. The acoustic window was limited. An echo-free space with several immobile septi was identified anterior to the right ventricle. Cardiac rupture was suspected. A computed tomographic scan of the thorax confirmed extensive mediastinal hematoma in communication with the left ventricle.

The cardiac surgery department was consulted and the patient was taken directly to the operating room. A defect of approximately 1 cm² in the posterobasal wall of the left ventricle was repaired. Coronary bypass grafting was not attempted. He was transferred to the surgical intensive care unit on intravenous inotropic agents and 1:1 balloon pump augmentation. He was admitted to the surgical intensive care unit, but died on the fifth postoperative day.

DISCUSSION

Myocardial rupture is a complication of AMI that directly causes death in 8% of patients (2). A rare but catastrophic form of this complication is LVFWR, the incidence of which appears to have lessened in patients undergoing primary percutaneous intervention for AMI (3). LVFWR is more common in women and in older patients, as well as in patients with hypertension and a first lateral or anterior wall AMI (2-7). The MI causing a free wall rupture is often relatively small. Steroid use and late thrombolysis do not appear to increase the risk of LVFWR (8,9).

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Figure 1 Computed tomographic scan from the coronal view (**top**) and the short-axis view (**bottom**). Contrast can be seen filling the left ventricle (LV). There was a perforation in the free wall of the LV (arrow), with extensive hemorrhage along the left posterolateral aspect of the heart to the diaphragmatic surface and into the right pleural space. Considerable cardiac compression was noted from the right mediastinum, with compression predominantly of the right atrium and left atrium. A Anterior; Ao Aorta; P Posterior; RV Right ventricle

Classic LVFWR usually produces symptoms within the first 24 h of an AMI, and almost always by the end of the first week (10). The clinical manifestations of LVFWR depend on both the amount and the rate of intrapericardial bleeding. In most cases, sudden hemodynamic collapse is followed by electromechanical dissociation and death. In some cases, the intrapericardial hematoma seals the site of myocardial perforation and may lead to the formation of a left ventricular pseudoaneurysm (11). A subacute variant of LVFWR, marked by slow, repetitive bleeding, occurs in approximately one-third of cases (6,12).

Several studies have tried to identify the premonitory signs and symptoms of fatal LVFWR (5,6,12,13). Prodromal manifestations

Figure 3 During surgery, there was extensive mediastinal hematoma (not shown). A perforation (arrow) of approximately 1 cm² was identified in the posterobasal wall of the left ventricle and was repaired

include persistent chest pain (often erroneously attributed to ischemia), intractable vomiting, restlessness and ST segment elevation that persists for 72 h after the onset of chest pain (6,13). The classic signs of cardiac tamponade, including pulsus paradoxus and diastolic pressure equalization, are usually absent (12). Electromechanical dissociation may occur, but it has limited diagnostic value (5).

Echocardiography is the procedure of choice for the diagnosis of LVFWR and usually reveals a pericardial effusion with intrapericardial echoes consistent with hematoma. Occasionally, diastolic collapse of the right ventricle can be seen. Echocardiography has a diagnostic sensitivity of 100% and a specificity of 93% (12,14). Although hemopericardium following AMI may rarely result from the administration of thrombolytic therapy and/or glycoprotein IIb/IIIa inhibitors in patients without myocardial rupture, LVFWR should always be considered in a patient with an AMI, otherwise unexplained hypotension and pericardial effusion.

In our patient, who had a previous pericardiectomy, the extracardiac hemorrhage could clearly not be contained by the pericardium; instead, it communicated directly with the mediastinum and the right pleural space (producing progressive anemia, low intracardiac filling pressures and vigorous contraction of both ventricles). The presentation of this complication of AMI in this patient with an absent pericardium (ie, with hemorrhagic shock rather than cardiac tamponade) facilitated the completion of diagnostic studies and successful surgical repair.

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REFERENCES

- Moreno R, López de Sá E, López-Sendón JL, et al. Frequency of left ventricular free-wall rupture in patients with acute myocardial infarction treated with primary angioplasty. *Am J Cardiol* 2000;85:757-60,A8.
- Reddy SG, Roberts WC. Frequency of rupture of the left ventricular free wall or ventricular septum among necropsy cases of fatal acute myocardial infarction since introduction of coronary care units. *Am J Cardiol* 1989;63:906-11.
- Yip HK, Wu CJ, Chang HW, et al. Cardiac rupture complicating acute myocardial infarction in the direct percutaneous coronary intervention reperfusion era. *Chest* 2003;124:565-71.
- Batts KP, Ackermann DM, Edwards WD. Postinfarction rupture of the left ventricular free wall: Clinicopathologic correlates in 100 consecutive autopsy cases. *Hum Pathol* 1990;21:530-5.

5. Figueras J, Curós A, Cortadellas J, Soler-Soler J. Reliability of electromechanical dissociation in the diagnosis of left ventricular free wall rupture in acute myocardial infarction. *Am Heart J* 1996;131:861-4.
 6. Raitt MH, Kraft CD, Gardner CJ, Pearlman AS, Otto CM. Subacute ventricular free wall rupture complicating myocardial infarction. *Am Heart J* 1993;126:946-55.
 7. Sutherland FW, Guell FJ, Pathi VL, Naik SK. Postinfarction ventricular free wall rupture: Strategies for diagnosis and treatment. *Ann Thorac Surg* 1996;61:1281-5.
 8. Giugliano GR, Giugliano RP, Gibson CM, Kuntz RE. Meta-analysis of corticosteroid treatment in acute myocardial infarction. *Am J Cardiol* 2003;91:1055-9.
 9. Becker RC, Charlesworth A, Wilcox RG, et al. Cardiac rupture associated with thrombolytic therapy: Impact of time to treatment in the Late Assessment of Thrombolytic Efficacy (LATE) study. *J Am Coll Cardiol* 1995;25:1063-8.
 10. Purcaro A, Costantini C, Ciampani N, et al. Diagnostic criteria and management of subacute ventricular free wall rupture complicating acute myocardial infarction. *Am J Cardiol* 1997;80:397-405.
 11. Mahilmaran A, Nayar PG, Sheshadri M, Sudarsana G, Abraham KA. Left ventricular pseudoaneurysm caused by coronary spasm, myocardial infarction, and myocardial rupture. *Tex Heart Inst J* 2002;29:122-5.
 12. López-Sendón J, González A, López de Sá E, et al. Diagnosis of subacute ventricular wall rupture after acute myocardial infarction: Sensitivity and specificity of clinical, hemodynamic and echocardiographic criteria. *J Am Coll Cardiol* 1992;19:1145-53.
 13. Oliva PB, Hammill SC, Edwards WD. Cardiac rupture, a clinically predictable complication of acute myocardial infarction: Report of 70 cases with clinicopathologic correlations. *J Am Coll Cardiol* 1993;22:720-6.
 14. Mittle S, Makaryus AN, Mangion J. Role of contrast echocardiography in the assessment of myocardial rupture. *Echocardiography* 2003;20:77-81.
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