Successful management of left ventricular free wall rupture

Jason D Roberts MD, Kam W Mong MD FRCSC, Bruce Sussex MBBS MSc

JD Roberts, KW Mong, B Sussex. Successful management of left ventricular free wall rupture. *Can J Cardiol* 2007;23(8):672-674.

Cardiac rupture is a complication of myocardial infarction with an exceedingly high mortality rate. Imaging modalities, such as echocardiography, have facilitated premortem diagnosis, thus increasing chances of survival. A review of pertinent literature is accompanied by a case report that details an episode of left ventricular free wall rupture and its successful outcome.

Key Words: *Cardiac tamponade; Complications; Left ventricle; Myocardial infarction*

Cardiac rupture is a complication of myocardial infarction with an exceedingly high mortality rate. We present a case report that details an episode of left ventricular free wall rupture and its successful outcome followed by a review of pertinent literature.

CASE PRESENTATION

A 74-year-old man presented to the emergency department at 23:30 complaining of severe retrosternal chest pain approximately 2 h in duration. The patient also reported three episodes of chest pain earlier that day, each of which lasted approximately 30 min before spontaneous resolution. The pain was described as 'severe' and 'crushing', radiated into his jaw and left arm, and was associated with nausea and diaphoresis. His cardiac risk factors included smoking, a history of hypertension and type 2 diabetes mellitus. Aside from an elevated blood pressure, he denied any prior cardiac problems, including heart failure, angina or previous myocardial infarction. Physical examination revealed an elderly man in obvious distress with a blood pressure of 180/90 mmHg and a regular pulse of 86 beats/min. The electrocardiogram (ECG) demonstrated normal sinus rhythm with 3 mm of ST-segment elevation in the lateral leads, which prompted a diagnosis of acute myocardial infarction. Thrombolytic therapy was given, and symptom resolution and ECG normalization were achieved within 30 min of administration. Approximately 1.5 h later, the patient suffered a recurrence of chest pain that was not relieved with nitroglycerin or morphine. An ECG demonstrated ST-segment elevation of greater than 4 mm in the interolateral leads, with reciprocal changes in V_1 through V_3 . Rescue angioplasty was arranged, but was subsequently cancelled because the symptoms and ECG changes resolved within 1 h.

La prise en charge positive d'une rupture de la paroi libre du ventricule gauche

La rupture cardiaque est une complication de l'infarctus du myocarde qui s'accompagne d'un taux de mortalité extrêmement élevé. Les modalités d'imagerie, comme l'échocardiographie, facilitent le diagnostic *premortem*, accroissant ainsi les possibilités de survie. Une analyse bibliographique est suivie d'un rapport de cas qui détaille un épisode de rupture de la paroi libre du ventricule gauche et son issue positive.

The patient stabilized and spent the following nine days in hospital. He was treated with the normal regimen of medications for myocardial infarction, along with amiodarone for new-onset atrial fibrillation. A transient pericardial rub was noted on the third day following the myocardial infarction. Aside from that previously mentioned, the period following infarction was relatively uneventful. He experienced no subsequent episodes of chest pain and there was no evidence of ischemic changes on ECG during this period.

On the morning of the ninth day, the patient developed severe chest pain, which he stated was distinctly different from that during previous episodes. He then underwent cardiovascular collapse and had an undetectable systolic blood pressure and distended jugular veins. An ECG revealed sinus tachycardia and electrical alternans. The patient was resuscitated with intravenous fluids and inotropes, and following blood pressure restoration, a pulsus paradoxus of 18 mmHg was documented on physical examination. An urgent transthoracic echocardiogram was performed and demonstrated a diffusely hypokinetic left ventricle and a large quantity of circumferential pericardial fluid; there was evidence of a large thrombus within the pericardial space. Colour flow Doppler imaging failed to identify an area of rupture. The patient was subsequently investigated using left heart catheterization, which reinforced the transthoracic echocardiography findings and further demonstrated single-vessel coronary artery disease (ie, a completely occluded circumflex artery with normal left anterior descending and right coronary arteries). The patient was tentatively diagnosed with free wall rupture of the left ventricle with spontaneous seal.

The patient was brought to the operating room, where incision of the pericardial sac resulted in relief of cardiac tamponade

Memorial University of Newfoundland, St John's, Newfoundland and Labrador

Correspondence: Dr Bruce Sussex, Division of Cardiology, Health Sciences Complex, Room 1283, Memorial University, 300 Prince Phillip Drive, St John's, Newfoundland and Labrador A1B 3V6. Telephone 709-777-7337, e-mail bsussex@nl.rogers.com

Received for publication June 24, 2005. Accepted February 7, 2006

and a marked improvement in blood pressure. The patient was placed on full cardiopulmonary bypass and the area of infarction was localized to the lateral wall of the left ventricle. No active bleeding was noted; however, a ventricular tear approximately 1 cm in length was identified immediately juxtaposed to the soft and necrotic area of the infarcted myocardium. A ventriculotomy was made through the ventricular tear and revealed that the area of infarction had extended to the posteromedial papillary muscle. An oversized Dacron patch was fashioned to include the area of infarct along with a surrounding 1 cm border of healthy myocardium. An interrupted, pledgeted, double-arm silk suture was passed circumferentially through the patch in an interrupted horizontal mattress fashion. The suture was then passed from the endocardial to the epicardial surface of the heart through noninfarcted tissue in an effort to provide a stable anchoring site. The Dacron patch was secured on the endocardial surface after tying the sutures on the outside of the heart. The ventriculotomy was subsequently closed with interrupted, double-arm, pledgeted silk sutures. Finally, the patient was weaned off cardiopulmonary bypass through the use of an intra-aortic balloon pump and moderate doses of inotropes.

The patient had an uneventful postoperative course and was discharged 14 days following the operation. Four years later, the patient had not had any subsequent cardiac events, exhibited no clinical evidence of heart failure and had moderate exercise tolerance. At the time of writing, his functional status was comparable with that before his episode of infarction and rupture, while recent multigated cardiac imaging revealed an ejection fraction of 31%.

DISCUSSION

The most feared complication of myocardial infarction is that of cardiac rupture. Although it has an incidence of only 1% to 4% following a heart attack, it accounts for up to 20% of mortalities associated with infarction (1). The phenomenon was first reported by William Harvey in 1649; however, successful surgical repairs did not appear in literature until 1972 (2). Over 30 years later, reports involving patient survival following cardiac rupture still remain quite rare. The catastrophic consequences of rupture are the result of cardiac tamponade causing rapid hemodynamic collapse. Free wall rupture has been divided into two different forms on the basis of presentation, namely acute and subacute (3). The acute form (also referred to as blowout) involves cardiac arrest resulting from severe hypotension and electromechanical dissociation secondary to cardiac tamponade (4). The subacute form of free wall rupture (also referred to as oozing) does not involve cardiac arrest, but describes cases that consist of "moderate to severe pericardial effusion and hypotension of varied severity associated with sinus bradycardia or nodal rhythm" (3). In both acute and subacute forms of rupture, patients exhibit severe jugular venous distension associated with cyanosis in the neck and face. It is important to note that patients suffering from the subacute form of rupture may remain hemodynamically stable (4).

Analysis of the patient population that has suffered cardiac rupture revealed a variety of common characteristics. Patients are more frequently female (5), older than 55 years of age (6) and often have pre-existent hypertension (7). Rupture follows the first episode of myocardial infarction in 90% of cases (8) and has a strong association with single-vessel disease (9).

The latter indexes are suggestive of nascent coronary disease, and their relationship with rupture is thought to reflect a resultant paucity of collateral vessels. Increased infarct size (defined by a peak creatine kinase [muscle-brain] greater than 150 U/L) has also been associated with an increased tendency to rupture (10).

The management of myocardial infarction also influences the risk of cardiac rupture. A retrospective analysis found that rupture was more likely to occur following inadequate reperfusion of the infarcted area (11). This finding is supported by studies that have shown a reduced incidence of rupture with early thrombolytic therapy (12) and successful percutaneous coronary intervention (13). The reduced incidence of rupture associated with early reperfusion is attributed to its ability to halt the wave of necrosis extending through the myocardium, thus leaving an intact portion of epicardium (14). The structurally intact epicardium is thought to confer resistance, as evidenced by transmural infarction being a prerequisite for rupture (15). In contrast to early thrombolytic therapy, defined as less than 6 h following the onset of symptoms, early reports suggested that late thrombolytic therapy may actually increase the risk of rupture. However, results from the Late Assessment of Thrombolytic Efficacy (LATE) trial demonstrated that although late thrombolytic therapy accelerated the onset of rupture, it did not increase its incidence (16).

Following the initial management of myocardial infarction are a variety of sequelae that serve as risk factors for ensuing rupture, including persistent arterial hypertension (a systolic blood pressure greater than 150 mmHg) during the first 24 h (17), postinfarction angina (17) and the development of a pericardial rub (3). The use of anti-inflammatory agents postinfarction, such as steroids and nonsteroidal anti-inflammatory drugs, has been suggested to increase the risk of rupture (18). The mechanism is thought to be related to an inhibition of fibroblast proliferation and connective tissue regeneration, resulting in impaired wound healing and the potential for infarct expansion. In contrast, the early use of angiotensin-converting enzyme (ACE) inhibitors has been suggested to be protective (19) – a concept supported by the ability of ACE inhibitors to lower the rate of postinfarction shock (20). The mechanism for this possible reduction likely relates to the ability of ACE inhibitors to reduce the collagenolytic activity of matrix metalloproteinases (MMPs) (21). MMPs can cause excessive degradation of the myocardial scaffolding in pathological states – a phenomenon supported by animal studies in which MMP-9-deficient mice exhibited a decreased incidence of rupture (22).

Although successful salvage of cardiac rupture remains relatively rare, the increased use of imaging modalities, such as echocardiography, has led to earlier diagnosis, thus increasing the possibility of survival. This is especially true for the subacute form of rupture (23). However, even following prompt diagnosis, subsequent surgical management remains quite challenging for a variety of reasons. First, patients generally have marked hemodynamic instability and are therefore automatically considered to be within an extremely high-risk surgical population. Results from available case series in literature regarding patients who made it to surgery suggest that the degree of hemodynamic instability may be the most important predictor of outcome, with survival rates ranging from 39% to 100%, depending on the preoperative state of the patient (24). Furthermore, with regard to the surgery itself, the location of

the rupture site can be very elusive – a phenomenon aggravated by the use of cardiopulmonary bypass. In an effort to ease identification of the culprit region of myocardium, surgical approaches are now frequently being performed off-pump (25). Finally, the major technical issue with the surgical repair of a rupture involves the weakened state of the surrounding infarcted myocardium. The weakened necrotic tissue serves as a very poor anchoring site for sutures, and as a result, infarctectomy frequently has to be used to involve viable areas of myocardium. Infarctectomy, however, generally results in a significantly reduced left ventricular cavity size and is thought to have a detrimental effect on outcome. Contemporary techniques have overcome the necessity for infarctectomy through the use of a Dacron or Teflon patch, which is large enough to cover the area of rupture and surrounding necrotic tissue and also encompass the neighbouring viable myocardium (26).

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