A Subacute Left Ventricular Free Wall Rupture After Thrombolytic and Glycoprotein IIb/IIIa Inhibitor Treatment

An Overlooked Finding of Left Ventriculography

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SUMMARY

Subacute left ventricular free wall rupture is a rare complication in acute myocardial infarction. With the increasing use of thrombolytic agents and glycoprotein IIb/IIIa inhibitors, this complication has been increasing recently. We report a case of subacute cardiac rupture with frank pericardial effusion receiving thrombolytic and glycoprotein IIb/IIIa inhibitor therapies. (Jpn Heart J 2002; 43: 289-293)

Key words: Myocardial rupture, Glycoprotein IIb/IIIa inhibitor therapy

ACUTE left ventricular free wall rupture is known as the second leading cause of mortality in acute myocardial infarction (AMI), especially among patients with a first transmural AMI, and is usually fatal.¹⁾ Subacute rupture has a more insidious presentation, but early diagnosis is possible with echocardiography. With the increasing use of thrombolytic, glycoprotein IIb/IIIa inhibitors and anticoagulant agents, alone or in combination, in patients with acute myocardial infarction, this fatal complication may be seen more frequently. We report a case of subacute cardiac rupture with frank pericardial effusion.

CASE REPORT

A 67-year-old Turkish male smoker with a history of hypertension was treated in our Coronary Care Unit due to an acute infero-postero-lateral wall myocardial infarction. Streptokinase was given to the patient at the 16th hour at a dose of 1.5 million units over 1 hour intravenously, in addition to aspirin, beta blocker, nitrate, and angiotensin converting enzyme inhibitor. Because of persis-

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Received for publication October 29, 2001.

Revised and accepted February 14, 2002.

tent angina pectoris, a glycoprotein IIb/IIIa inhibitor (tirofiban) was added at a dose of 0.4 $\mu\text{g/kg}$ per minute bolus over 30 minutes, followed by continuous infusion of 0.1 $\mu\text{g/kg}$ per minute for 12 hours via an infusion pump. Activated clotting time and platelet count were 350 seconds and 125,000/ μL , respectively. He underwent coronary angiography for recurrent angina on day 4. The proximal right coronary artery was occluded and was responsible for the myocardial infarction. There was also a 75% lesion at the middle of the left anterior descending coronary artery, a 75% lesion at the middle of the circumflex artery and obtuse lesions of various degrees on coronary angiography, and akinesia of the infero-postero-lateral wall of the left ventricle. The results of coronary angiography suggested coronary artery bypass surgery was indicated, however the patient was discharged and treated with a beta inhibitor, aspirin, and an angiotensin converting enzyme inhibitor.

Two weeks after discharge, he was admitted to our Emergency Department with a sharp and penetrating chest pain, fatigue, and palpitations. The blood pressure and heart rate were 100/50 mmHg and 150 beats/min, respectively. There was a mild apical systolic murmur and pericardial rub in his examination. Ventricular tachycardia was observed on electrocardiography. Therefore, 100 mg

Figure 1. Frank pericardial effusion in pericardial space and myocardial hole in the basal segment of the lateral wall of the left ventricle (LV=left ventricle; RV=right ventricle; PE=pericardial effusion)

Figure 2. Myocardial bulging at the base of the lateral wall of the left ventricle in the apical 4-chamber view. Arrow shows the thin myocardial segment and bulging (LV=left ventricle; LA=left atrium, PE=pericardial effusion).

Figure 3. Right anterior oblique projection of left ventriculography (Arrows show a little leakage from the posterior wall into the pericardial space).

lidocaine (bolus) was given to establish cardioversion and 2 mg/min lidocaine was infused. Echocardiography revealed a frank pericardial effusion, 2.3 cm on the lateral, 2.4 cm on the posterior, and 0.8 cm on the anterior wall in the pericardial space. The basal segment of the left ventricular posterolateral wall was thin, and a myocardial hole with a 1 mm diameter was observed in the same segment of the left ventricle approximately 1 cm under the mitral valve (Figure 1). In addition, a bulging of this segment to the left ventricular chamber was observed (Figure 2).

When the coronary angiogram and ventriculogram performed two weeks previously were re-evaluated, in addition to three vessel disease, a thin leakage from the posterior myocardial wall of the left ventricle into the pericardial space, overlooked before, was observed (Figure 3).

DISCUSSION

Acute rupture of the cardiac free wall is, after cardiac failure, currently the second most frequent cause of hospital deaths following myocardial infarction. However, in about 30% of the cases, the rupture may be a subacute haemorrhage into the pericardial space, and may be slow with localised containment.²⁾ The insidious nature of this condition, with patients remaining stable for hours or days, makes a diagnosis difficult. It typically occurs within 4 days after infarction, and results from a subacute process with localized necrosis of the myocardium. Significant predisposing factors for ventricular rupture are elderly age (>70 years) and female sex. Possible risk factors also include the use of steroids, anticoagulation, or late thrombolysis. Diagnostic criteria are an abrupt fall in blood pressure, often preceded by recurrent thoracic pain, associated with venous hypertension in the absence of left ventricular failure, and hemodynamic or angiographic, but mainly echocardiographic, findings. Echocardiography frequently demonstrates a small pericardial effusion. Increasing effusion or increased echogenicity within the pericardial space makes the diagnosis of subacute rupture more likely.³⁾ In our case, ventricular tachycardia along with the subacute rupture, which has not previously been described, was observed. The rupture, with pericardial effusion, was diagnosed by directly observing the myocardial hole.

It has been proved that thrombolytic therapy is useful in the treatment of acute myocardial infarction, but is frequently associated with limited vessel reperfusion and early reocclusion. Early studies combining full-dose fibrinolytic treatment and glycoprotein IIb/IIIa inhibitors have been promising with regard to overcoming these limitations, but concern about bleeding has hindered this strategy.⁴⁾ Haemorrhagic infarction has become a more common occurrence with the

advent of thrombolytic therapy, and it has been suggested that cardiac rupture is the direct result of myocardial haemorrhage extending through planes of necrotic and non-necrotic myocardium.⁵⁾ It is known that thrombolytic therapy accelerates the occurrence of rupture within 24 to 48 hours of treatment.⁶⁾ If myocardial haemorrhage is a prerequisite for cardiac rupture, the concomitant administration of fibrinolytic and glycoprotein IIb/IIIa inhibitors could have a facilitator effect among patients with transmural necrosis. There is no evidence from either experimental or clinical trials that adjunctive thrombolytic and glycoprotein IIb/IIIa inhibitors exert a direct effect on the myocardium, however, this protocol is quite a new therapeutic schema, and new studies which will investigate the relationship between this adjunctive therapy and haemorrhagic complications and myocardial rupture are needed. The only severe complication, a pulmonary artery rupture in a patient receiving thrombolytic, abciximab, and anticoagulant therapy under hemodynamic monitoring with a pulmonary artery catheter, has been reported so far.⁷⁾

The risk of a possible subacute myocardial rupture in patients who had an acute myocardial infarction and were then given glycoprotein IIb/IIIa inhibitors in addition to thrombolytic treatment should be considered, and these patients should be followed by careful echocardiographic monitoring. Attention should be paid, especially to certain subgroups at risk during this era of adjunctive therapy. Furthermore, slight pericardial effusion overlooked in the first examination, as in our case, should be considered as a potential cardiac rupture, and the ventriculography should be evaluated more carefully in these patients.

REFERENCES

1. Figueras J, Curos A, Cortadellas J, Sans M, Soler-Soler J. Relevance of electrocardiographic findings, heart failure, and infarct site in assessing risk and timing of left ventricular free wall rupture during acute myocardial infarction. *Am J Cardiol* 1995; 76: 543-7.
2. Coleman JE, Mansfield RJR, Simpson IA. Myocardial infarction and subacute ventricular rupture: successful outcome in two cases. *Int J Cardiol* 2000; 73: 285-7.
3. Lopez-Sendon J, Gonzalez A, Lopez de Sa E. Diagnosis of subacute ventricular free wall rupture after acute myocardial infarction: sensitivity and specificity of clinical hemodynamic and echocardiographic criteria. *J Am Coll Cardiol* 1990; 16: 359-67.
4. Califf RM. Combination therapy for acute myocardial infarction: fibrinolytic therapy and glycoprotein IIb/IIIa inhibition. *Am Heart J* 2000; 139: S33-7.
5. Mathey DG, Schofer J, Kuck KH, Veil U, Kloppel G. Transmural haemorrhagic myocardial infarction after intracoronary streptokinase. Clinical, angiographic and necropsy findings. *Br Heart J* 1982; 48: 546-51.
6. Becker RC, Hochman JS, Cannon CP, *et al*. Fatal cardiac rupture among patients treated with thrombolytic agents and adjunctive thrombin antagonist. *J Am Coll Cardiol* 1999; 33: 479-87.
7. Chadow HL, Gaba MK, Safi A, Afflu E, Feit A. Rescue PTCA complicated by pulmonary artery rupture after tPA and abciximab. *Catheter Cardiovasc Interv* 1999; 46: 89-91.