Cardiac rupture after thrombolytic therapy: the use of aprotinin to reduce blood loss after surgical repair

Catharina A van Doorn, Christopher M Munsch, J Campbell Cowan

Abstract
Emergency cardiac surgery after recent thrombolytic therapy is associated with increased blood loss. A patient underwent emergency repair of a ruptured left ventricle after intravenous streptokinase treatment for acute coronary occlusion. High dose aprotinin was given during the operation to reduce the expected blood loss. Surgical repair was successful without bleeding complications. Total postoperative blood loss was 365 ml.

Myocardial rupture is a well recognised and frequently fatal complication of acute myocardial infarction. The effect of thrombolytic therapy on the incidence of myocardial rupture is not yet known. Although emergency surgery offers the only realistic hope of survival, it is often difficult to control bleeding from the friable, recently infarcted myocardium. Furthermore, recent administration of thrombolytic therapy has been shown to increase substantially perioperative haemorrhage during emergency cardiac surgery. The protease inhibitor aprotinin (Trasylol: Bayer, Germany) has been shown to reduce the blood loss and use of blood products in patients undergoing cardiopulmonary bypass. We describe a patient who underwent emergency surgery for myocardial rupture after thrombolysis and to whom aprotinin was given with a view to reducing the expected excessive blood loss.

Case report
A 67 year old previously fit woman was admitted to the Leeds General Infirmary with a three hour history of chest pain and electrocardiographic evidence of acute anterior myocardial infarction. Treatment with intravenous streptokinase (1.5 x 10⁶ IU over 30 minutes), heparin, nitrates, and aspirin initially led to relief of her symptoms and resolution of the electrocardiographic changes. One hour later, however, she developed recurrent chest pain associated with new ST changes on the electrocardiogram and developed clinical evidence of cardiogenic shock. Her haemodynamic condition was at this stage improved by inotropic support with dobutamine and counterpulsation with an intra-aortic balloon pump (IABP).

Emergency coronary angiography showed a 90% stenosis in the midportion of the left anterior descending coronary artery (LAD) and although this was successfully dilated by angioplasty, she remained in cardiogenic shock. At this stage pericardial tamponade secondary to myocardial rupture was suspected and was confirmed by transthoracic echocardiography. The patient was transferred directly from the angio suite to theatre for emergency operation. Because of recent thrombolytic therapy, excess perioperative blood loss was expected. The table shows the results of her preoperative coagulation tests. On the basis of these it was decided to administer the protease inhibitor, aprotinin. After induction of anaesthesia, a loading dose of 500 000 kallikrein inactivator units (KIU) of aprotinin was given over 10 minutes, followed by 200 000 KIU per hour until the administration of protamine and the end of the operation.

After sternotomy a large amount of unclotted blood was evacuated from the pericardium with immediate improvement in the haemodynamic state. Inspection of the heart showed an extensive haemorrhagic anteropapical infarct covered with clot, beneath which there was a slit like rupture of the left ventricle (figure). After cardiopulmonary bypass was started necrotic muscle was excised and the ventricular defect repaired. Bypass was stopped with the help of the IABP and dobutamine support, but with otherwise satisfactory haemodynamic recovery. After reversal of heparin with protamine the wound bed, including the raw muscular surface of the ventricular repair, rapidly dried up. Postoperative blood loss from the chest drains was 265 ml for the first 12 hours with a total loss of 365 ml over 24 hours, after which the drains were removed. Over the next few days the patient was gradually weaned from the intraaortic balloon pump and off inotropic support. She made a good recovery, was discharged home 15 days after operation and was symptom free at follow up after two months.

Results of preoperative coagulation studies, after streptokinase administration

<table>
<thead>
<tr>
<th>Prothrombin time</th>
<th>25/15</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activated partial thromboplastin time</td>
<td>75/35</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>0.5 g/l</td>
</tr>
<tr>
<td>Fibrinogen degradation products</td>
<td>(reference &lt; 0.5 mg/l)</td>
</tr>
</tbody>
</table>

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Appearance of the heart at operation, showing extensive haemorrhagic anteropapical infarction.

Discussion
Cardiac rupture is a catastrophic event that occurs in about 2%-4% of patients admitted to hospital with an acute myocardial infarction, although its incidence is probably under reported. It is not yet known what effect thrombolytic therapy has on the occurrence of myocardial rupture. Preliminary data from the ISIS-II trial showed that 30 patients receiving streptokinase died from cardiac rupture compared with 18 in the control group. Although the differences are not statistically different, there is some evidence that thrombolysis increases the risk of rupture on the first day and decreases the risk on subsequent days. Because treatment and monitoring of patients with acute myocardial infarction is becoming more intensive, it is to be expected that an early diagnosis will be made in a higher percentage of patients suffering cardiac rupture, and this will lead to more patients being presented for emergency surgery. It is likely that, with the widespread use of thrombolytic therapy for acute infarction, many of these patients will come to surgery with a high risk of bleeding. These points are all well illustrated in the present case report.

The beneficial effects of the protease inhibitor aprotinin in reducing blood loss in open heart surgery are now widely known. Although its mechanism of action is not yet completely understood two potential mechanisms of action have been postulated: firstly that it has a protective effect on platelet function, which is normally reduced during cardio-pulmonary bypass, or secondly that it has an antifibrinolytic effect through its anti-plasmin activity. The reduction in bleeding induced by thrombolytic therapy in the present case lends support to the second of these theories.

Research into the exact mechanism of action of aprotinin is continuing and its clinical applications in both cardiac and non-cardiac surgery are continually being extended. This case illustrates a further indication for the use of aprotinin in reducing the potential complications associated with emergency cardiac surgery after thrombolysis.

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