CASE REPORTS

Recanalization of an Acutely Occluded Aortocoronary Bypass by Intragraft Fibrinolysis

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SUMMARY Acute thrombotic occlusion of an aortocoronary bypass graft to the left anterior descending coronary artery (LAD) was successfully dissolved by selective infusion of streptokinase into the graft at 2000 U/min for 1 hour via catheter. There was partial recanalization of the graft and complete filling of the LAD within 15 minutes. After 1 hour of lysis, the graft was completely patent, although high-degree narrowing at the site of the proximal anastomosis was still present. Follow-up angiography 16 days later revealed persistent patency of the graft and disappearance of narrowing at the site of proximal anastomosis. The relatively low total dose of streptokinase (140,000 U) did not result in a systemic hyperlytic state. There were no complications. The technique may prove useful in acute graft occlusion but needs further evaluation.

THROMBOSIS is usually the cause of graft occlusion during the first month after aortocoronary bypass surgery. Efficient thrombolysis by local application of low doses of streptokinase that did not result in a systemic hyperlytic state has been described in both peripheral and coronary arteries. In this report, we describe the successful application of selective thrombolysis in a patient with acute aortocoronary bypass occlusion.

Case Report

A 49-year-old man suffered a transmural anteroseptal infarction in 1971. Coronary angiography using the Sones technique was performed on September 23, 1979 because of retrosternal pressure brought on by mild exertion during the preceding 4 months. The left anterior descending artery (LAD) was found to be proximally occluded and showed partial retrograde filling via collaterals from the dominant right coronary artery (RCA). There was a proximal lesion of about 50% in the left circumflex branch. Biplane left ventriculography showed anterolateral, apical and septal hypokinesia. Ejection fraction (EF) was 35% and left ventricular pressure was 110/10 mm Hg.

On October 5, 1979, saphenous vein grafts were constructed to the first major diagonal branch and to the LAD. A tear in the vein necessitated reconstruction of the proximal anastomosis of the LAD graft. Otherwise, the operation was uneventful. Postoperatively, the patient received heparin, 5000 U subcutaneously three times a day, and β-acetyldigoxin, 1 mg orally twice a day.

On October 10, 1979 at 8:00 a.m., the patient complained of sudden, severe chest pain at rest that was identical to his preoperative angina. After sublingual administration of 1.6 mg of nitroglycerin (NTG), the pain transiently subsided for about 15 minutes. The ECG obtained at 9:00 a.m. showed a loss of R waves in lead V2 and 0.1 mV of ST elevation in leads V1 to V3, which had not been present before. At 9:05 a.m., i.v. NTG was started at a dose of 3 mg/hour; i.v. heparin was administered at a dose of 1000 U/hour. The patient became asymptomatic within 10 minutes. On a follow-up ECG at 11:00 a.m., the ST segments in leads V2 and V3 had returned to baseline.

Acute Angiography

Informed consent for local lysis was obtained after a full explanation of the experimental nature of the procedure and possible hazards. Angiography was performed by the transbrachial approach at 2:00 p.m. The graft to the diagonal branch was patent, but the LAD graft was occluded. Contrast medium injected into the LAD bypass irregularly outlined the proximal 5 cm of the vein (fig. 1A). There was no washout of contrast medium nor was the LAD visualized by the bypass injection. The native coronary artery system was unchanged compared with the preoperative study. The LAD showed retrograde filling via collaterals from the RCA and from the first major diagonal branch, which had received the patent graft. Left ventricular function was unchanged and left ventricular pressure was 100/10 mm Hg.

Intragraft Streptokinase Application

After diagnostic angiography, the following premedication was administered: prednisolone, 500 mg i.v.; acetylsalicylic acid, 1 g i.v.; diazepam, 5 mg i.v.; heparin, 5000 U i.v. A left coronary artery Amplatz catheter was placed into the proximal anastomosis of the LAD graft. Intragraft application of streptokinase...
(Kabi) was begun at 2:40 p.m., with a bolus of 20,000 U in 5 ml of normal saline solution, followed by infusion of 2000 U/min for 1 hour. The first control injection after 15 minutes revealed patency of the graft and complete filling of the LAD (fig. 1B), but there were several filling defects in the distal part of the graft. These filling defects had disappeared at the time of the control injection after 60 minutes of intragraft lysis, although there still was a high-degree narrowing at the site of the proximal graft anastomosis (fig. 1C). Repeat injection of contrast medium into the diagonal graft no longer showed collateral filling of the LAD.

Fibrinogen was 600 mg% before and after lysis (table 1). There were no complications during or after lysis. There was a slight pathologic rise of CPK to a peak of 110 U at 5:00 p.m. the same day. Follow-up ECGs did not show any further loss of R waves. The ST segments had returned to normal and the T waves were symmetrically negative in leads V₁ to V₄ at 11:00 p.m. Immediately after completion of lysis, sodium warfarin (Coumadin) therapy was begun. Full-dose i.v. heparin was continued for 4 days. The patient remained asymptomatic.

Follow-up Angiography

Follow-up angiography performed on October 26, 1979 revealed patency of both grafts (fig. 1D). The proximal anastomosis of the LAD graft was widely patent. The LAD filled completely via its graft. Collaterals to the LAD were not visible. Left ventricular function was unchanged and left ventricular pressure was 100/10 mm Hg.

Discussion

Early after operation, differentiating chest pain and ECG changes due to the operation from those due to recurrent ischemia can be difficult. However, this patient experienced symptoms that were identical to his preoperative angina, were promptly relieved by NTG and were associated with acute ST elevations.
The most likely cause of recurrent ischemic chest pain early after operation is graft closure. Occlusion of a graft within the first month after surgery is usually due to thrombosis, whereas later closure is frequently caused by intimal proliferation.\(^1\) Full-dose heparin therapy was begun as soon as graft thrombosis was suspected, to minimize apposition of thrombotic material.

Contrast medium injected into the occluded graft revealed an irregular staining of material thought to be a fresh clot. Application of streptokinase directly into the occluded graft resulted in recanalization of the vein within 15 minutes. The stenosis of the proximal anastomosis, which was still present after 1 hour of streptokinase infusion, was no longer present at repeat angiography. Presumably, the dissolution of thrombotic material continued after cessation of streptokinase therapy.

Selective infusion of streptokinase at 1000–2000 U/min into coronary arteries in patients with acute

**Table 1. Systemic Coagulation Factors Immediately Before and After Intragraft Infusion of Streptokinase, 2000 U/minute for 1 Hour and the Next Day**

<table>
<thead>
<tr>
<th></th>
<th>Fibrinogen (mg%)</th>
<th>Plasma thrombin time (sec)</th>
<th>Partial thromboplastin time (sec)</th>
<th>Thromboplastin time according to Quick(^4) (%)</th>
<th>Fibrin-monomer complexes (protamine sulfate) (mg%)(^9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before infusion</td>
<td>600</td>
<td>18.5</td>
<td>42.4</td>
<td>100</td>
<td>1.0</td>
</tr>
<tr>
<td>After infusion</td>
<td>600</td>
<td>300</td>
<td>300</td>
<td>52</td>
<td>0</td>
</tr>
<tr>
<td>Next day</td>
<td>570</td>
<td>126.6</td>
<td>86</td>
<td>76</td>
<td>0</td>
</tr>
</tbody>
</table>
myocardial infarction dissolved coronary artery thrombosis within 15–19 minutes. Recanalization of thrombotic occlusions in peripheral arteries required much longer periods of local lysis in the series reported by Dotter et al., who locally infused streptokinase at 1000–10,000 U/hour. The time required for selective lysis depends on the age of the clot, the amount of thrombotic material and the rate of streptokinase infusion.

The total streptokinase dose of 120,000 U, infused over 1 hour, was much lower than doses needed for systemic lysis. A systemic hyperlytic state is not induced by this dose, which is an important factor early after operation. Prolongation of partial thromboplastin time, plasma thrombin time and thromboplastin time according to Quick* was due to heparin therapy. There were no side effects or complications.

The value and risks of local fibrinolysis in recanalization of aortocoronary bypass graft occlusion remain to be established in a larger series. Rapid lysis probably can be achieved only within a few hours after thrombus formation.

References

Recanalization of an acutely occluded aortocoronary bypass by intragraft fibrinolysis.

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Circulation. 1980;62:1123-1126
doi: 10.1161/01.CIR.62.5.1123

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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