Negative results - Coronary

Acute thrombosis after endarterectomy of stented left anterior descending artery

Tomoya Uchimuro*, Toshihiro Fukui*, Wahei Mihara*, Shuichiro Takanashi*

*Department of Cardiovascular Surgery, Chiba Central Medical Center, 1835-1 Kasoricho, Wakaba-ku, Chiba 264-0017, Japan

*Cardiovascular Surgery, Sakakibara Heart Institute, Tokyo, Japan

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Abstract

Acute thrombosis of the endarterectomized coronary artery is a serious complication after coronary endarterectomy. Herein, we describe a case of a 65-year-old man who had undergone percutaneous stent implantation in the left anterior descending artery, then after he received an endarterectomy with the removal of stents severe in-stent restenosis occurred. Three days after the operation, sick sinus syndrome developed with acute myocardial infarction. Coronary angiography revealed thrombosis at the reconstructed site of the left anterior descending artery. Pacemaker implantation, intra-aortic balloon pumping, and aggressive anticoagulation produced recanalization of the left anterior descending artery.

Keywords: CABG; Endarterectomy; Stents

1. Introduction

Coronary endarterectomy is an established surgical method for revascularization in diffusely diseased coronary arteries. A long diffuse lesion may occur at the stent-implanted site as in-stent restenosis can occur in the severely diseased vessel. Recently, some case studies have demonstrated successful stent removal with endarterectomy in patients with in-stent restenosis [1–3]. However, surgeons should be careful of postoperative myocardial infarction secondary to acute thrombosis of the endarterectomized artery as this is a life-threatening complication. Herein, we report a treatment option for postoperative myocardial infarction after endarterectomy with the removal of stents in the left anterior descending artery (LAD).

2. Case

A 65-year-old man with unstable angina was transferred to our hospital. He had received percutaneous coronary interventions three times since 1988. Six coronary stents had been implanted, with a sirolimus eluting stent and a bare-metal stent in the right coronary artery, two sirolimus eluting stents in the LAD, and two sirolimus eluting stents in the circumflex artery. Coronary angiography showed triple-vessel disease including restenosis in four stents (Fig. 1a,b). Electrocardiogram on admission demonstrated regular sinus rhythm with 40 beats per minute (bpm), although the patient had not taken any medications that may have lowered his heart rate. Echocardiography revealed mild hypokinesis in the anterior myocardium, and the left ventricular ejection fraction was 67%. We planned for an off-pump coronary artery bypass grafting with removal of the stents in the LAD.

After median sternotomy, the bilateral internal thoracic arteries were dissected as in-situ grafts, and a left radial artery graft and a right saphenous vein graft were harvested. All arterial grafts were harvested in a skeletonized fashion. A commercially available heart positioner and stabilizer were used for the off-pump procedures. A bloodless field was obtained using a proximal silastic snare suture and a CO2 blower. The radial artery graft was anastomosed to the obtuse marginal branch of the circumflex artery. The saphenous vein graft was sequentially anastomosed to the posterior descending artery and the atrioventricular node artery of the right coronary artery. The right internal thoracic artery was anastomosed to the diagonal artery. The saphenous vein graft was sequentially anastomosed to the posterior descending artery and the atroventricular node artery of the right coronary artery. The right internal thoracic artery was anastomosed to the diagonal artery. The atheromatous core with two stents and the intima of all side branches were carefully dissected and removed through a 9-cm long incision in the LAD. The proximal and distal ends of the atheromatous core were sharply divided, and the divided intima of the distal end was tacked with 8-0 polypropylene sutures. The left internal thoracic artery (LITA) was longitudinally incised adjusting to the length of incised LAD, and was anastomosed with several 7-0 and 8-0 polypropylene running sutures in order to avoid a purse-string effect.
The patient was hemodynamically stable during and after the operation. Intravenous adrenergic drugs and vasodilator drugs were injected continuously. Electrocardiogram demonstrated regular sinus rhythm with 50 bpm and no ST-segment abnormality. We started temporary atrial pacemaking at 70 bpm because bradycardia might have lowered the blood flow velocity in the LAD and have caused thrombosis on the rough surface of the LAD. Oral nitroglycerin, nicorandil, aspirin, ticlopidine, and warfarin were started from the day after surgery. Intravenous adrenergic drugs and vasodilator drugs were gradually discontinued, and intravenous administration of low molecular weight heparin was started at 5000 U per day. In the evening, spontaneous heart rate increased to 60 bpm. Temporary pacemaking was stopped, and intravenous administration of isoproterenol was started in order to prevent bradycardia. On the second postoperative day, the spontaneous heart rate was mostly maintained at 60 bpm, but occasionally decreased below 50 bpm. On the third postoperative day, atrial fibrillation and transient cardiac arrest accompanied by loss of consciousness occurred suddenly. The electrocardiogram revealed ST-segment elevation in V1–4 leads. We immediately inserted a temporary pacemaking catheter and started ventricular pacemaking at 70 bpm. Emergent coronary angiography revealed thrombosis of the reconstructed LAD (Fig. 2a). Intra-aortic balloon pumping (IABP) was started to secure coronary blood flow. Intravenous administration of heparin was increased to 10,000 U per day. Adrenergic drugs and vasodilator drugs were resumed. The patient’s hemodynamic condition gradually improved. His maximum creatine phosphokinase isoenzyme level was 180 U/l. Electrical defibrillation was performed on the fifth postoperative day as the patient’s heart rate was approximately 70 bpm with atrial flutter. As his heart rate dropped to 40 bpm with regular sinus rhythm, ventricular pacemaking was continued at 70 bpm. On the seventh postoperative day, IABP was removed. As the patient’s heart rate did not increase from 40 bpm, a permanent pacemaker was implanted on the tenth postoperative day. Coronary angiography showed patency of the reconstructed LAD on the twenty-third postoperative day (Fig. 2b). The patient was discharged from hospital the following day.

3. Discussion

Coronary endarterectomy is a surgical option for revascularization of diffusely diseased coronary arteries, and has acceptable clinical and angiographic results after operation [4–7]. Since the development of stent devices, in-stent restenosis has been a major problem. Stent removal with endarterectomy is necessary for the treatment of repeat recurrent in-stent restenosis in the LAD. The long endarterectomy with off-pump technique may be a controversial method, but we believe that a severe diffusely diseased LAD can stand longer ischemic times because significant collaterals have developed and offered protection against myocardial infarction during off-pump surgery [8]. However, it has been performed with cardiopulmonary bypass in patients with unstable hemodynamics. Importantly, after the operation close attention should be paid to the patient due to the potential for thrombosis in the reconstructed LAD. In the present case, the possible causes of acute thrombosis included the following. First, the postoperative anticoagulant should have been insufficient. The actual activated whole blood clotting time (ACT) in the patient was 150–175 s during the postoperative three days. Intravenous heparin administration should have been increased immediately after the operation to keep the ACT over 200 s. Secondly, the intermittent bradycardia on the second postoperative day might have lowered the blood flow velocity in the LAD, which in turn, might have allowed the thrombosis to form on the rough surface of endarterectomized LAD. Thus we should have continued the temporary pacemaking. The use of IABP was effective for the treatment of the acute thrombosis in the reconstructed LAD as IABP increased the diastolic flows in the LAD and the LITA.

In conclusion, this is the first report describing the treatment of a thrombosis in an endarterectomized and stent-removed LAD. Aggressive anticoagulation, sufficient blood flow in the LAD, and IABP were effective in this case.
References


eComment: Islets technique to reduce endarterectomized area included into graft during left anterior descending coronary artery endarterectomy

Authors: Dusko Nezic, Dedijne Cardiovascular Institute, Department of Cardiac Surgery, M. Tepica 1, 11000 Belgrade, Serbia; Aleksandar Knezevic, Slobodan Micovic, Milan Cirkovic
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The rapidly growing use of percutaneous coronary intervention for myocardial revascularization has led to a fundamental change in the patient subset referred to coronary artery bypass grafting surgery. Therefore, surgeons are facing an increasing number of patients with advanced and diffuse coronary artery disease, as well as an increasing number of patients (whose coronary artery lesions have been stented) with instent restenosis. Thus, we read with great interest the report by Uchimuro and colleagues [1] presenting acute thrombosis following endarterectomy of stented left anterior descending (LAD) coronary artery. We would like to congratulate the authors for the successful treatment of this life-threatening complication. However, it would be of interest to mention the patient’s predischARGE left ventricular ejection fraction, as preoperative value was 67%.

We have recently presented a patient in whom the endarterectomy of the LAD coronary artery ended up as a technically unsatisfactory procedure [2]. Finally, along 9 cm of endarterectomized LAD area, we were left with only three segments (‘islets’) of properly endarterectomized coronary bed areas, the third one extending as a very distal part of LAD. These ‘islets’ were separated with totally disintegrated coronary bed portions. These three segments were incorporated into a venous graft, with the inflow obtained from the left internal thoracic artery (LITA). On the 8th postoperative day a 64-slice CT confirmed that the grafted segments of the LAD were patent. Postoperative transthoracic echocardiography confirmed no change in the ejection fraction.

The major concern following coronary artery endarterectomy (CAE) is that the endarterectomized arterial wall acts as a trigger for the new thrombus formation, as an increase of platelet-activating factor was observed in damaged LADs in a canine model, as well as in endarterectomy samples that were taken from the severely diseased coronary arteries of patients with diffuse coronary artery disease [3]. Another major concern regarding CAE is the development of myofibrointimal proliferation [4], which impairs early and long-term clinical and angiographic results.

In our patient [2] all successfully endarterectomized LAD segments were incorporated into a venous graft, with the inflow obtained from the LITA. In such a manner, the endarterectomized area is reduced (due to the exclusion of totally disintegrated LAD areas) and endothelial covering might be achieved rapidly, decreasing the risk of thrombus formation in the early stage and myofibrointimal proliferation later on. Having been encouraged with the outcome in this patient, in the next two patients we have reduced the surface of the endarterectomized LAD coronary bed included into the LITA graft. The in situ LITA conduit connected distal (end-to-end) and proximal (side-to-end) parts of the endarterectomized LAD. Only the parts containing the origin of large septal and diagonal branches were included in the conduit. Although postoperative transthoracic echocardiography confirmed no changes in the patients’ ejection fractions, we do believe that with a reduction of the endarterectomized coronary bed area included in the arterial (venous) conduit, we can decrease the risk of thrombus formation in the early stage and myofibrointimal proliferation later on.

References


