

Emergency Surgical Treatment for Acute Occlusion of the Left Main Coronary Artery—Report of a Case—

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We report a case of acute occlusion of the left main coronary artery (LMCA) successfully treated with percutaneous transluminal coronary angioplasty (PTCA) under the use of percutaneous cardiopulmonary bypass support (PCPS) and subsequent coronary artery bypass grafting (CABG). CABG was started only two hours after admission, and subsequent reperfusion of left anterior descending artery (LAD) after completing distal and proximal anastomosis was achieved 60 minutes later. Although postoperative CK levels were elevated to 10,900 IU/l, akinesis of the left ventricular (LV) wall was limited to segment #1 and #2, and hypokinesis in segment #3 and #6 documented by postoperative left ventriculogram (LVG). The patient was discharged from the hospital on foot without neurologic sequelae and is doing well and in New York Heart Association (NYHA) functional class I in 20 months of follow-up.

Simultaneous efforts to maintain systemic circulation and to achieve reperfusion of the occluded LMCA as soon as possible are essential for survival. Prompt introduction of mechanical circulatory support and early revascularization to minimize the infarct area are both necessary. (Ann Thorac Cardiovasc Surg 2003; 9: 73–8)

Key words: acute myocardial infarction (AMI), acute left main coronary artery obstruction (acute LMCA obstruction), percutaneous cardiopulmonary bypass support (PCPS), coronary artery bypass grafting (CABG), left main shock syndrome

Introduction

Acute occlusion of the left main coronary artery (LMCA) is a rare but serious condition, which carries a very high mortality rate due to massive acute myocardial infarction (AMI).¹⁾ Most of these patients with this clinical setting may suffer from sudden death or profound cardiogenic shock due to malignant arrhythmia or pump failure.^{2,3)} The reported results of emergency coronary artery bypass grafting (CABG) for these patients who have shown profound hypotension are quite unsatisfactory for sur-

vival.^{4,5)} We report a case of acute occlusion of LMCA successfully treated with percutaneous transluminal coronary angioplasty (PTCA) under the use of percutaneous cardiopulmonary bypass support (PCPS) and subsequent CABG.

Case Report

A 51-year-old man weighing 63 kg was referred to our hospital with severe precordial pain that lasted for two hours. He had a history of ileocecal resection for perityphlitis two years ago, but had no atherosclerotic risk factors such as diabetes mellitus (DM), hypertension (HTN), or hyperlipidemia (HL). He had neither smoking nor drinking habits.

On admission, there was no cyanosis, anemia, or jaundice. His consciousness was clear and body temperature was 36.2°C. Physical examination revealed a moderate hypotension with systolic blood pressure of 82 mmHg, regular pulse at a rate of 105 beats/min. Pulmonary aus-

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a1	a2
b	

Fig. 1. a. Coronary angiography (CAG) before percutaneous transluminal coronary angioplasty (PTCA). CAG exhibited a total occlusion of the LMCA and a dominant right coronary artery (RCA) with 50% stenosis in both #1 and #2. b. CAG after reperfusion of the LMCA by PTCA. PTCA successfully resulted in recanalization of the LMCA where 75% stenosis was left. CABG was conducted subsequently.

cultation disclosed no abnormalities. No heart murmur was audible. There were no hepatomegaly, edema in his legs, or neurologic deficits. Laboratory data showed no anemia, white blood cell (WBC) count of 7,700/ μ l, platelet count of $16.3 \times 10^4/\mu$ l, creatine kinase (CK) of 142 IU/l, and creatine kinase-MB isoenzyme (CK-MB) of 2.4 ng/ml. Serum levels of protein, transaminase, blood urea nitrogen, and creatinine were within normal limits. The electrocardiogram (ECG) showed a regular sinus rhythm with depression of the ST segment in the II, III, and aVF leads and with elevation of the ST segment in the V2 to V4 leads, leading to a strong suspicion of extensive anteroseptal AMI.

Immediately after admission, the patient was taken to the catheter laboratory room and underwent an insertion of intraaortic balloon for counterpulsation. Intraaortic balloon pumping (IABP) was then commenced followed by emergency coronary angiography (CAG), which exhibited a total occlusion of the LMCA and a dominant right coronary artery (RCA) with 50% stenosis in both

#1 and #2 (Fig. 1a). Ventricular tachycardia (VT) was provoked soon after CAG, which caused profound shock. Percutaneous cardiopulmonary support (PCPS) was started to maintain systemic perfusion. Since early reperfusion of the LMCA was thought to be essential for reducing the infarct area, direct PTCA was performed on the occluded LMCA. PTCA successfully resulted in recanalization of the LMCA where 75% stenosis was left (Fig. 1b), but we decided to carry out emergency CABG due to recurrent VTs which caused a marked deterioration of hemodynamic conditions.

Surgery was started two hours after admission. CABG was performed through a median sternotomy on the beating heart under PCPS at a flow rate of 3.5 to 3.8 L/min. We distally anastomosed a saphenous vein graft (SVG) onto the left anterior descending artery (LAD) #7 and the dominant RCA #4 AV branch. The time required from the start of operation to reperfusion of the LAD after completing distal and proximal anastomosis was 60 minutes, and that to reperfusion of the RCA was 90 minutes. We

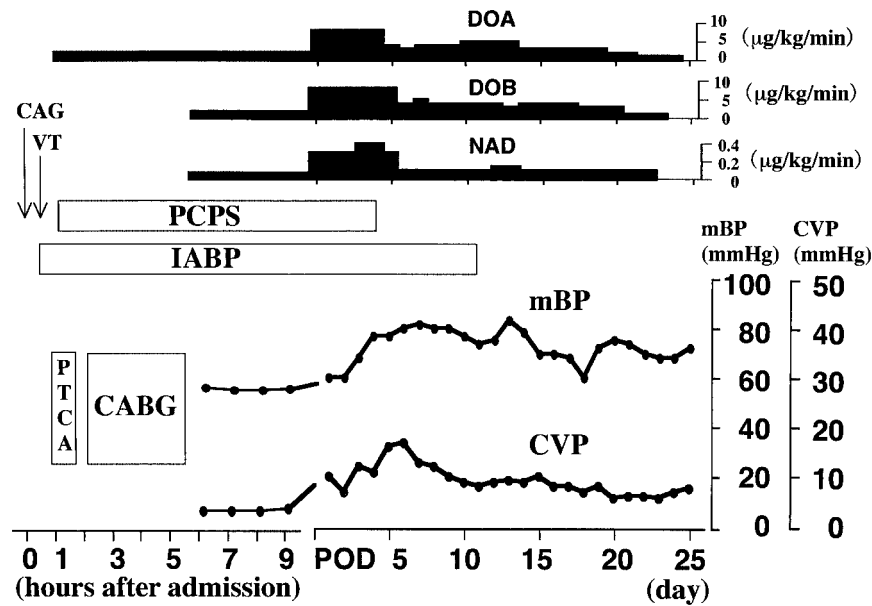


Fig. 2. Summary of pre- and postoperative course from admission to discharge from the intensive care unit. CAG: coronary angiography, VT: ventricular tachycardia, PTCA: percutaneous transluminal coronary angioplasty, CABG: coronary artery bypass grafting, IABP: intraaortic balloon pumping, PCPS: percutaneous cardiopulmonary bypass support, POD: postoperative day, mBP: mean blood pressure, CVP: central venous pressure, DOA: dopamine, DOB: dobutamine, NAD: norepinephrine

attempted to wean the patient from PCPS after reperfusion of the LAD and RCA, but relinquished it because he developed hypotension and VT again. Operation time was 150 minutes and the patient was taken to the intensive care unit (ICU) under the continuing use of IABP of 1:1 and PCPS at a flow rate of 2.5 L/min. Other inotropic supports at the end of operation included continuous infusion of dopamine of 3 $\mu\text{g}/\text{kg}/\text{min}$, dobutamine of 3 $\mu\text{g}/\text{kg}/\text{min}$, and norepinephrine of 0.1 $\mu\text{g}/\text{kg}/\text{min}$.

Figure 2 summarizes the pre- and postoperative course from admission to discharge from the ICU. The patient was weaned from PCPS after its use for approximately 96 hours at a flow rate of 1.5 to 2.0 L/min. The peak levels of CK and CK-MB were 10,900 IU/l and 480 ng/ml, respectively. The patient was also weaned from IABP on the 10th postoperative day, but required a long-term use of catecholamines due to prolonged low output syndrome (LOS). The postoperative left ventriculogram (LVG) demonstrated akinesis in segment #1 and #2, and hypokinesis in #3 and #6 (Fig. 3a). Postoperative CAG showed a good patency of the SVG grafts to LAD and RCA (Fig. 3b). Thallium 201 scintigraphy revealed local hypoperfusion in the anteroseptal and anterolateral segments and perfusion defects in part of the anteroapical

segment. Catecholamines were discontinued on the 26th postoperative day, and the patient was discharged from the hospital on foot without neurologic sequelae on the 69th postoperative day. In 20 months of follow-up, the patient is doing well and in New York Heart Association (NYHA) functional class I.

Discussion

Treatment of total LMCA occlusion has been rarely reported, most likely due to the high mortality rate from massive AMI.¹⁾ Quigley et al. reported that 15 (94%) of the 16 patients with acute occlusion or severe stenosis of the LMCA in cardiogenic shock at presentation died in hospital.²⁾ They also proposed to categorize those patients presenting with anterolateral AMI, severe stenosis of the LMCA, and cardiogenic shock into left main shock syndrome because of its high mortality.²⁾ We had treated three patients with acute occlusion of the LMCA before the present case, but all three patients died in hospital. Since most of the patients with acute occlusion of the LMCA would develop a sudden, profound deterioration of hemodynamics, simultaneous efforts to maintain systemic circulation and to achieve reperfusion of the occluded

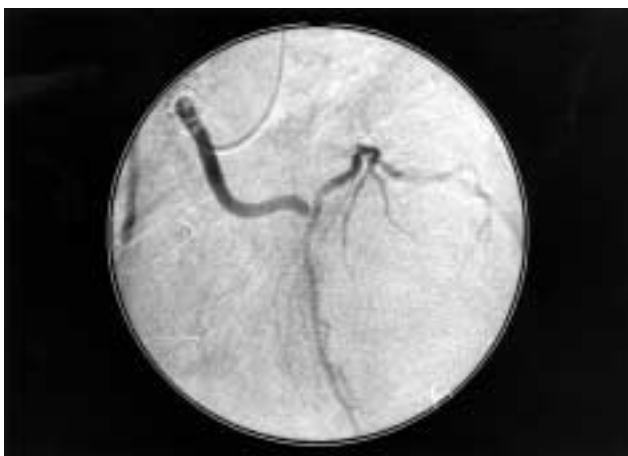
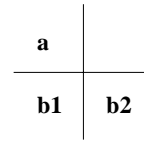
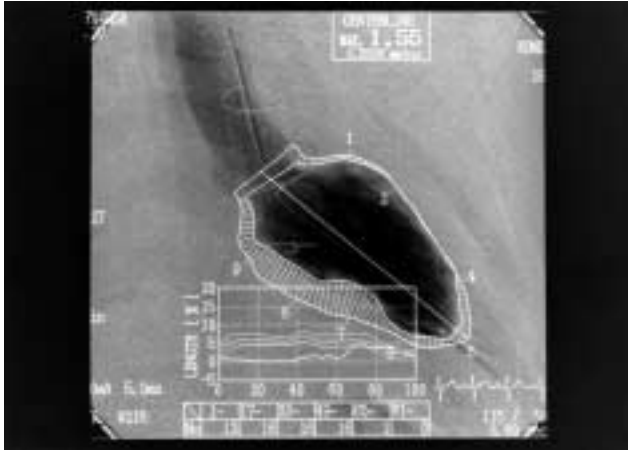


Fig. 3. a. Postoperative left ventriculogram (LVG). LVG revealed akinesis in segment #1 and #2, and hypokinesis in #3 and #6. b. Postoperative CAG. CAG demonstrated a good patency of the SVG grafts to LAD a (b1) and RCA (b2).

LMCA as soon as possible are essential for survival.^{6,7)} For the former purpose, PCPS has been employed and proved to be beneficial for keeping hemodynamics stable for a few days.^{5,8-12)} The present patient was strongly suspected to have an extensive anteroseptal AMI based on the ECG findings and immediately underwent an insertion of a balloon catheter for IABP. Although hemodynamic conditions suddenly deteriorated soon after CAG for a definitive diagnosis, systemic circulation could be soon established by the emergency use of PCPS. Since serum levels of CK and CK-MB had not been elevated by the time of admission, mechanical circulatory support in this case was thought best to be commenced shortly after the onset of LMCA occlusion.

According to the classification of coronary arterial preponderance, 48% of cases have a dominant RCA (Type I), 34% have a balance between the right and left coronary artery (LCA) (Type II), and 18% have a dominant

LCA (Type III).¹³⁾ The angiographic findings of the present case showed a dominant RCA (Type I), which may have been one of reasons that he did not suffer from sudden death out of hospital. Based on the previous literature, most patients with acute occlusion of the LMCA who did not suffer from sudden death and reached hospital would have either a dominant RCA or an extensive collateral circulation from the RCA to the LCA, or both.^{3,11,14)} Therapeutic strategy must include both keeping systemic circulation against profound shock to prevent neurologic sequelae, and conducting early reperfusion of the LMCA to minimize the infarct area.¹⁵⁾ Lack of either strategy would result in failure to salvage the patient. Although PCPS is beneficial for keeping systemic perfusion of the important organs, it does not have therapeutic effects on the heart damaged by extensive infarction. Delayed reperfusion would cause diffuse myocardial necrosis, which may result in the patient unable to be weaned from

mechanical circulatory support. When early reperfusion by PTCA or CABG is conducted under prolonged hypotension or profound shock without stabilizing systemic circulation, most of these patients will develop neurologic sequelae or severe organ failure due to an inadequately low perfusion. To conduct both therapeutic strategies is mandatory in the treatment of acute occlusion of the LMCA.⁸⁾

Although our procedure was successful for survival, there are some problems in the course of treatment with regard to myocardial protection. An autoperfusion balloon catheter should have been placed to ensure perfusion of the LCA during the period between direct PTCA and CABG, then left ventricular function may have been more preserved.⁵⁾ Since the time from the onset of LMCA occlusion to the reperfusion and that from the start of operation to reperfusion of the LCA were ideally short, the use of autoperfusion balloon catheter would have reduced the period of inotropic support and hospital stay. However, the present patient had neither atherosclerotic risk factors nor history of angina, and had poor intercoronary collateral from the RCA to the LCA. Spiecker et al. also emphasized that the clinical outcome of patients with acute LMCA occlusion was strongly dependent on the presence or absence of intercoronary collaterals, in addition to the presence of dominant RCA.¹⁴⁾ A certain degree of myocardial necrosis, which is unable to be salvaged by ideal treatment, is unavoidable once the patient developed acute occlusion of the LMCA in the absence of intercoronary collaterals.

In addition to the coronary arterial preponderance, the interval between the onset of AMI and surgical revascularization is an important determinant for the mortality rate in patients who suffered acute LMCA occlusion. There is no accepted cut-off value of the interval significantly correlated with patient's survival, but the reported interval between the onset of AMI and surgical revascularization in survivors ranged from 2 to 20 hours.^{3,11,16)} In the present case, CABG was started only two hours after admission, and subsequent reperfusion of LAD after completing distal and proximal anastomosis was achieved 60 minutes after the start of operation. Although postoperative CK levels were elevated to 10,900 IU/l, postoperative LVG showed that akinesis of the left ventricular (LV) wall was limited to segment #1 and #2, and hypokinesis in segment #3 and #6. The short interval between the onset of AMI and surgical revascularization in this case may greatly contribute to the salvage of significant amounts of LV myocardium. We employed SVG

grafts for CABG to reduce intervals between the onset of AMI and surgical revascularization as described above, although arterial grafts are superior to SVG grafts with regard to a long patency. The patient has no atherosclerotic or coronary risk factors such as DM or smoking habits, but careful follow-up is required with a special attention to the patency of SVG grafts.

In summary, we report a case of acute occlusion of the LMCA successfully treated with PTCA under the use of PCPS and subsequent emergency CABG. CABG was started only two hours after admission, and subsequent reperfusion of LAD after completing distal and proximal anastomosis was achieved 60 minutes later. The patient was discharged from the hospital without neurologic sequelae and has been doing well in NYHA functional class I for 20 months of follow-up. Simultaneous efforts to maintain systemic circulation and to achieve reperfusion of the occluded LMCA as soon as possible are essential for survival. Prompt introduction of mechanical circulatory support and early revascularization to minimize the infarct area are both necessary.

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