Urgent Surgical Reperfusion for Intraoperative Myocardial Infarction Following Coronary Artery Bypass Grafting

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The primary goal in treating "acute evolving myocardial infarction (AEMI)" is decrease in early myocardial damage and improvement of the clinical course[1]. Emergency coronary artery bypass grafting (CABG) has been performed successfully in patients considered to have AEMI in recent years. The present case was an adult patient with intraoperative myocardial infarction following CABG, who required urgent surgical reperfusion to wean cardiopulmonary bypass.

Case Report

A 42-year-old man developed acute antero-septal myocardial infarction one month after the first angina and then post-infarction angina. Risk factors included a long history of cigarette smoking (60 cigarettes daily), elevated uric acid and hypertension. He had neither history of diabetes nor family history of ischemic heart disease. Elective cardiac catheterization demonstrated two-vessel coronary artery disease, including the left anterior descending (95%), diagonal branch (90%) and proximal right coronary artery (75%) with preserved left ventricular function. Left ventricular end-diastolic pressure was 9 mmHg, ejection fraction 0.58, and cardiac output 6.0 $l \cdot min^{-1}$. He was scheduled for coronary artery bypass grafting. On examination his blood pressure was 160/80 mmHg, pulse 80 min^{-1} and regular, and there was no clinical cardiomegaly. An electrocardiogram showed sinus rhythm and neither significant Q waves nor ST-T changes. The preoperative medications were nifedipine 30 mg and isosorbide dinitrate 15 mg, three times a day. These medications were continued up to the morning of surgery.

The patient was premedicated with diazepam 5 mg orally, and morphine 10 mg intramuscularly. Anesthesia was induced with fentanyl 25 $\mu g \cdot kg^{-1}$, and diazepam 20 mg iv and tracheal intubation was conducted using an iv pancuronium 0.1 $mg \cdot kg^{-1}$. Anesthesia was maintained with enflurane, nitrous oxide and oxygen supplemented by intermittent administrations of fentanyl.

The patient had a double coronary bypass: saphenous vein connected from the aorta to the right coronary artery and the left internal mammary artery to the left anterior descending artery. The bypass time was 133 min and cross-clamp time 86 min.

The patient was weaned from the cardiopulmonary bypass uneventfully. However, 10 min later, he showed marked ST-T wave elevation in lead II and V_5. The graft flows were 33 $ml \cdot min^{-1}$ to the LAD and 20 $ml \cdot min^{-1}$ to the RCA. Intravenous nitroglycerin 1 mg was injected into each

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graft with immediate resolution of the ST segment elevation. Although intravenous injections of isosorbide dinitrate and diltiazem were begun, acute ST segment elevation occured again 20 min later. This episode responded to another injection of nitroglycerin 1 mg into each coronary graft. Although no hemodynamic changes could be found during these episodes, there was a progressive fall of arterial pressure 60 min later followed by sudden ventricular fibrillation when the systolic arterial pressure fell to 80 mmHg.

Cardiopulmonary bypass was reinstituted immediately after a successful cardioversion. Although an intra-aortic baloon pump and peripheral infusion of 1.5 $\mu g \cdot min^{-1}$ of norepinephrine and 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of dobutamine were started, weaning from the bypass was not possible because of recurrent ventricular fibrillation and a transient complete heart block with visually apparent deterioration of bi-ventricular wall motion. Finally, 410 min after the bypass had been reinstituted, surgical reperfusion to LAD with saphenous vein graft was performed again.

The patient remained stable after surgical reperfusion and was transfered to the intensive care unit with his chest opened.

The chest was closed on the 3rd postoperative day, and the patient was extubated by the 4th postoperative day. He was taken off IABP and weaned from all inotropic supports by the 4th postoperative day.

An electrocardiogram of the following day showed Q waves in II, III, aVF, V_5 and V_6, marked ST elevation in V_3–V_6 and an incomplete right bundle branch block. Glutamic oxaloacetic transaminase (GOT) rose to 290 $IU \cdot l^{-1}$ (normal range 5 to 35 $IU \cdot l^{-1}$), total creatin phosphokinase (CPK) to 5, 868 $IU \cdot l^{-1}$ (normal range 5 to 50 $IU \cdot l^{-1}$) and the level of the myocardial specific isoenzyme of CPK (MB-CPK) was 216 $IU \cdot l^{-1}$.

The echocardiogram showed akinesis of the anteroseptal wall motion and severe hypokinesis of the posteroinferior wall motion, consistent with intraoperative myocardial infarction.

The patient has remained free of angina for ordinary physical activity with medication of isosorbide dinitrate, diltiazem, verapamil and digoxin and has undergone a single Master's two step test with negative results. He was discharged on the 67th postoperative day without postoperative angiography.

Discussion

Acute surgical reperfusion, by itself or in conjunction with thrombolysis and/or percutaneous transluminal coronary angioplasty (PTCA), is now an accepted treatment for AEMI. Cohn[2] has presented indications for surgical treatment of acute myocardial infarction, in which perioperative myocardial infarction (PMI) following CABG is not included.

PMI remains a major and the most frequent complication following CABG, but the cause of PMI has not been fully determined. The present case developed intraoperative myocardial infarction after diagnosis of coronary artery spasm. Coronary artery spasm during CABG is known to cause sudden, severe hemodynamic collapse or ventricular arrythmia, and to cause acute myocardial infarction[3]. Criteria for the diagnosis of coronary artery spasm are those shown by Buxton and coworkers[4] as follows: 1) reversible ST segment elevation, 2) evidence of myocardial ischemia such as hypotension or ventricular arrhythmias, and 3) absence of severe atherosclerotic obstruction of the involved vessel. However, in general, coronary artery spasm is an angiographic diagnosis and no acceptable reason has been given as to why these criteria alone are applicable. The spasm has been reported to be superimposed on a fixed atherosclerotic lesion[3]. Thus, an understanding of the causes and definition of the spasm is presently incomplete, although the term coronary artery spasm is often used. Catheterization is not possible in most instances and it seems difficult to rule our other causes of myocardial ischemia. In this case, coronary artery spasm may have been responsible for myocardial ischemia because PMI also developed in the area supplied by legion free circumflex coronary artery. However, there may have been

limited blood flow due to technical problems, and ST elevation in this setting may also be improved transiently by reducing left ventricular end-diastolic pressure following intra-coronary nitroglycerin.

Although a diagnosis of AEMI may not be so easy in the operating room, the time limit for successful reperfusion is considered to be 6 hr[2]. In our case, the viable area of peri-infarction zone may have been quite small because of delayed reperfusion following the series of treatments for the spasm. However, for this patient in baloon-dependent cardiogenic shock, survival without surgical reperfusion would likely have been difficult. Emergent and immediate surgical reperfusion should be indicated in patients with AEMI or severe hemodynamic collapse suggesting AEMI, regardless of the primary cause, even when an elective CABG has just been performed.

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