ACUTE LEFT MAIN CORONARY ARTERY OCCLUSION  
— A Case Report and Review of the Literature —

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Acute left main coronary artery (LMCA) occlusion is rare and typically fatal. According to the recent literature, only three cases have been reported surviving emergent coronary artery bypass grafting (CABG) for acute myocardial infarction (AMI) caused by total LMCA occlusion.

We report the case of a 52-year-old man presenting with total occlusion of the LMCA associated with acute anterolateral myocardial infarction and cardiogenic shock. The patient’s dominant right coronary artery did not supply the collaterals to the left coronary system. After ineffective thrombolysis the patient underwent successful emergent CABG for the management of uncontrollable cardiogenic shock.

Emergent CABG, though controversial, appears to be a feasible approach for patients with AMI in life-threatening situations, when other attempts at reperfusion have been unsuccessful.

Sudden obstruction of the left main coronary artery (LMCA) is usually fatal. Coronary artery recanalization by thrombolysis and/or angioplasty have become common treatments for acute coronary artery disease. These interventions have recently been applied to LMCA occlusion in a few cases.1—10 This report describes a patient with complete LMCA occlusion complicated by refractory cardiogenic shock who was successfully treated by emergent coronary artery bypass grafting (CABG) after thrombolytic therapy had failed.

CASE REPORT

A 52-year-old man, previously physically active and in good health, presented to the local emergency room complaining of prolonged severe chest pain radiating to the left arm. He had had several episodes of intermittent chest pain one week prior to presentation. The patient’s risk factors for coronary disease included mild diabetes mellitus and cigarette smoking. He was transferred to our emergency center, with the diagnosis of acute myocardial infarction (AMI), for intensive coronary care.

On arrival the patient’s supine blood pressure was 90/60 mmHg, and his heart rate was 94 beats per minute and regular. There was remarkable supine jugular venous distension and mild bibasilar rales. Initial auscultation revealed no cardiac murmurs or gallops. The electrocardiogram on admission was indicative of acute anterolateral ischemia (Fig. 1). The patient was immediately treated with intravenous urokinase

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Fig. 1. Electrocardiogram on admission was indicative of acute anterolateral ischemia and of progression to a ventricular tachycardia just before surgery. Postoperative electrocardiogram revealed QS complexes in the precordial leads initially, but R waves appeared by Day 10 accompanied by slight ST elevation in V2 - V4.

(480,000 U). Nevertheless, over the first hour after presentation he developed clinical and roentgenographic evidence of worsening congestive heart failure.

Within 2 1/2 hours of the onset of chest pain, the patient underwent selective coronary angiography which demonstrated total flush occlusion of the LMCA, and the dominant right coronary artery with stenosis at the midportion with no right-to-left coronary collaterals (Fig. 2). Immediately, consultations to discuss the option of surgery were instituted, while intracoronary thrombolysis was attempted. This failed despite intracoronary urokinase infusion of a total 720,000 U. over a 30 min period. Progressive hemodynamic deterioration (systolic blood pressure 60–80 mmHg) demanded support with inotropic agents and intraaortic balloon pump insertion during catheterization. The electrocardiogram demonstrated frequent premature ventricular contractions and bigeminy, followed by ventricular tachycardia for which lidocaine was used. Clinical impressions of the patient’s condition suggested a high risk of mortality without further intervention, and the patient was taken for emergent CABG. The preoperative creatinine phosphokinase peaked at a remarkably high value of 13,100 U/L with an MB fraction of 373 U/L, and the roentgenogram revealed severe congestive heart failure (Killip III). Arterial blood gas analysis showed PH 7.310, PO2 45.0 Torr and PCO2 44.6 Torr (5 L oxygen per minute by mask).

Less than 6 h after the onset of chest pain (two and a half hours after thrombolysis), the patient underwent CABG to the left anterior descending, diagonal and right coronary arteries using saphenous vein grafts. Intraoperatively, a 1.0 mm probe could be passed in a retrograde fashion through the obstructed LMCA. No evidence of intracoronary thrombus was observed. Severe diffuse calcification was encountered along the LMCA and left anterior descending artery on palpation. The left ventricular wall was not hemorrhagic, but was remarkably edematous. The pump was stopped after half an hour’s circulatory assistance. The aortic cross-clamp time was 64 min. The total cardiopulmonary bypass time was
165 min. The patient tolerated the procedure very well and left the operating room in a satisfactory condition with the support of inotropic agents 5 \( \mu \text{g/kg/min} \), noradrenaline 0.15 \( \mu \text{g/kg/min} \) and an intracoronary balloon pump. The electrocardiogram showed tight QRS and persistent ST elevation in the precordial leads.

The postoperative course was unremarkable. The patient was weaned from the intracoronary balloon pump within 3 days and from inotropic agents on Day 7. Postoperative complications included congestive heart failure (pulmonary artery systolic pressure > 60 mmHg) and ventricular tachycardia, the latter requiring cardioversion several times. One month later an assessment of left ventricular function revealed akinesis of the anterolateral wall with an ejection fraction of 61.1\% (Fig. 3), and end diastolic pressure of 20 mmHg and a cardiac index of 3.28 L/min/m\(^2\).

Postoperative selective coronary angiography showed the following (Fig. 4): (1) the LMCA was recanalized with a significant stenosis of 1.0 mm in diameter, probably due to the intraoperative insertion of the 1.0 probe or preoperative effects of urokinase; (2) the left coronary artery was small and rather spastic; (3) the three bypass grafts were all patent; (4) the right coronary artery was dominant and stenotic providing the left coronary system with no retrograde collaterals. Postoperative electrocardiography revealed all QS complexes in the precordial leads initially (Fig. 1), but the R wave
appeared by Day 10 accompanied by slight ST elevation in leads V₂—V₄. The extreme pulmonary congestion seen by chest roentgenogram just after surgery was improved after several days. Radionuclide ventriculogram revealed irreversible ischemia of the anterolateral wall.

Four months later the patient was found to be well (NYHA Class II) and free from anginal symptoms whilst on medical therapy.

**DISCUSSION**

In cases of AMI complicated by cardiogenic shock emergent CABG remains controversial. In reviewing the English literature, we found only 5 reported cases of AMI following acute LMCA occlusion as confirmed by coronary angiogram where patients underwent emergent CABG²⁻⁹⁻¹¹ Of these cases, 3 patients (60%) survived. Certainly, there may be a tendency to report only successful cases of attempted interven-
tions, and possibly many patients with acute LMCA occlusion die before reaching medical care, thereby contributing to a sample bias. Emergent CABG for AMI is associated with an increased operative mortality; specifically, 13% in the period between 1977 and 1979 and 4.9% from 1980 to 1982, a 7-fold greater risk than that associated with elective surgery.

Though extremely rare, AMI due to LMCA occlusion is nevertheless most often lethal because of the large amount of myocardium affected.

Though not the sole factor involved, right-to-left collateral blood flow seems to play an important role in patient survival following LMCA total occlusion. In this case, there was no visible collateral circulation demonstrated by angiogram either emergently or at two months follow-up. O'Shaughnessy et al have reported survival without collaterals in patients with profound shock and treated with vasopressor agents.

While reports exist of survival in cases of AMI complicated by cardiogenic shock using noninvasive techniques or intraaortic balloon pump in the recent use of angioplasty and/or thrombolysis has improved survival in high risk patients. When successful reperfusion is ultimately achieved, however, prolonged periods of ischemia often result in severe left ventricular dysfunction and lethal complications! Therefore, efforts need to focus on achieving reperfusion within the shortest time possible. This should be within 12 hours for survival, and within 6 hours for restoration of normal myocardium. Sigwart et al suggests that despite occasional reports of success, thrombolysis is inappropriate for reestablishing perfusion. They advocate angioplasty as the only means whereby recanalization is guaranteed, even given the risk of downstream embolization.

This case emphasizes the need for early surgical standby when attempting coronary reperfusion in cardiogenic shock. Emergent CABG, if performed early, can be an effective approach in minimizing myocardial damage when other measures are unsuccessful.

REFERENCES


