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# The treatment of acute myocardial infarction due to the occlusion of the left main coronary disease

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# Abstract

Acute myocardial infarction (AMI) due to the occlusion of the left main coronary artery (LMCA) is a rare but serious condition in the era of percutaneus coronary intervention (PCI). Even more rare is AMI involved with both LMCA and its branches like trifurcartion or bifurcation: this is challenging for interventional cardiologists, because it involves the extension of the myocardium complicated by cardiogenic shock and its technical difficulties.

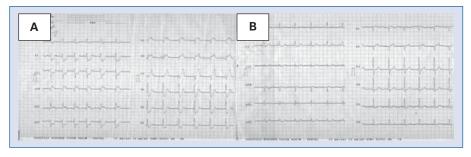
Trifurcating coronary artery disease is a complex atherosclerotic process involving the origin of one or more of three side branches arising from a left main coronary vessel or trunk, with or without the involvement of LMCA itself. There is no classification or standardized methodology to treat LMCA disease in elective percutaneous intervention procedures. Furthermore, acute myocardial infarction presenting with left main coronary artery trifurcation lesion seems to be more troublesome, especially in young patients. Few series of PCI on significant lesions of the left main trifurcations have been described. Herein, we describe a patient who successfully underwent PCI and was supported by post intravascular ultrasound sonography and multislice computed angiography (MSCA), and after an uneventful follow-up with MSCA is now on the ninth month. (Cardiol J 2011; 18, 1: 77–82)

Key words: myocardial infarction, left main coronary artery disease, trifurcation

### Introduction

Acute myocardial infarction due to occlusion of left main coronary artery (LMCA) and its side branches is a rare clinical entity. It carries a very high morbidity and mortality rate due to the involvement of a large myocardial area. In the modern interventional era, its incidence, clinical features, outcome, and prognostic determinants differ according to clinical conditions such asthe pre-existing pump failure or refractory ventricular dysrhythmias, presenting with cardiogenic shock, acute pulmonary edema and involvement of coronary arteries such as coexisting intercollaterals, a dominant right coronary artery (RCA), and an incompletely occluded LMCA and experience of cardiologists such as rapid establishment of complete reperfusion [1]. As evaluation of safe and effective management of patients with LMCA acute myocardial infarction (AMI) and development of strategies in modern interventional cardiology have been carried out. Outcomes of percutaneous coronary intervention (PCI) for LMCA lesions in patient with AMI have been impressive and promising.

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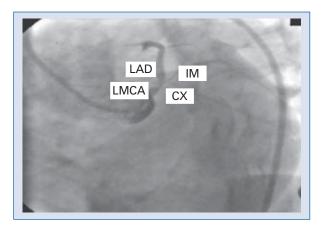


**Figure 1.A.** Pre percutaneous coronary intervention (PCI) electrocardiography showed ST segment elevation in leads I, aVL, aVR, V1 through V3, the anterior leads, ST segment depression in lead DII–III, aVF, and V4 through V6; **B.** Post PCI electrocardiography showed resolution of ST segment changes in previously recorded leads.

#### **Case report**

A 29 year-old male was admitted to our hospital with severe chest pain of one hour duration. He had been smoking for seven years and had no prior history of cardiovascular disease. On physical examination, the patient was clammy, and hypotensive (85/60 mm Hg), with normal heart sounds and bilateral pulmonary rales on the lower zone of lungs. His electrocardiogram demonstrated ST segment elevation in leads I, aVL, aVR, V1 through V3, the anterior leads, ST segment depression in lead DII-III, aVF, and V4 through V6 (Fig. 1A). Acute anterior myocardial infarction Killip Class II was diagnosed and the patient was immediately transferred to the catheterization laboratory from the emergency unit. 300 mg aspirin, 600 mg loading dose of clopidogrel, and 5000 U of intravenous heparin were administered. Coronary angiography was performed and disclosed a left main with a significant stenosis in the distal portion of its body. Distally to this lesion, the left main had presented a trifurcation to left anterior descending (LAD), circumflex (CX), and intermediate branch (IM), presenting a significiant lesion on the proximal portion of these branches (Fig. 2). The RCA was normal.

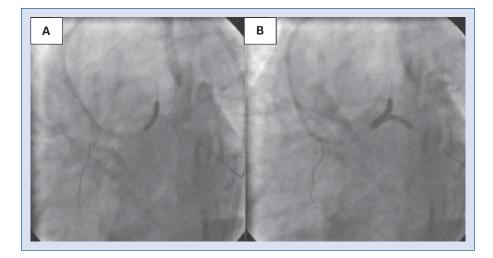
We decided to perform PCI on LMCA and trifurcation lesions. After two coronary soft guide wires (0.014 inch Forté; Boston Scientific, Natick, NJ, USA) had been passed into LAD and CX coronary arteries, the distal left main portion and LAD was predilatated with  $2.5 \times 20$  mm balloon inflated at 14 atm (Fig. 3A). Subsequently, LAD and CX was dilatated with  $2.5 \times 20$  mm balloon inflated at 16 atm using the kissing balloon technique (Fig. 3B). A bare metal  $3.0 \times 12$  stent was implanted into LMCA at 14 atm in 10 s across the lesion and across the ostium of LAD. After deployment of stent into LMCA and LAD, another bare metal stent ( $2.5 \times$  $\times 12$  mm) was implanted into the osteal CX. Finally,



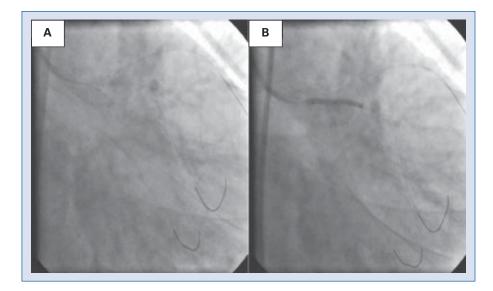
**Figure 2.** Pre percutaneous coronary intervention. Coronary angiography (left anterior oblique caudal view) shows a left main coronary artery (LMCA) with a significant stenosis in the distal portion of its body. Distally to this lesion, the left main had presented a trifurcation to left anterior descending (LAD), left circumflex (CX), and intermediate branch (IM), presenting a significant lesion on the proximal portion of these branches.

the kissing balloon technique at 16 atm in 16 s was performed for LAD and CX. After deflation LMCA, LAD and CX remained patent. Subsequently, the significiant lesion on the proximal intermediate branch was seen and the third guide wire had been passed into this branch. Another  $2.5 \times 12$  mm bare metal stent was deployed (Fig. 4), finally achieving a satisfactory angiographic result (Fig. 5).

The electrocardiogram, taken after 60 minutes of PCI, showed a resolution of ST segment elevation in previously recorded leads (Fig. 1B). After one week, the intravascular ultrasound sonography examination had been performed and showed the correct stent position and the absence of any abnormal patterns. For evaluating optimal platelet inhi-



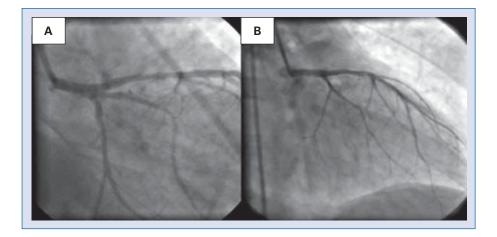
**Figure 3. A.** Two 0.014 soft guidewires were passed into left anterior descending (LAD) and circumflex (CX). Distal left main coronary artery and LAD was predilatated with  $2.5 \times 20$  mm balloon inflated at 14 atm; **B.** Dilatation of LAD and CX with  $2.5 \times 20$  mm balloon inflated at 16 atm with kissing balloon technique.



**Figure 4. A.** Third 0.014 soft guidewire was passed into intermediate branch (IM); **B.** Deployment of  $2.5 \times 12$  mm bare metal stent into IM.

bition, we performed a platelet function test with multiplate electrical impedance aggregometry and effective inhibition was assessed with 150 mg clopidogrel and 300 mg aspirin. Because of the highrisk coronary condition, clopidogrel was prescribed 150 mg for at least 12 months and the patient was discharged home symptom free. After one month, echocardiography showed full recovery of hypokinetic segments and no other wall motion abnormalities. Because of his high risk condition due to early stenosis (even though he was asympotomatic) multislice computed angiography (MSCA) was performed and confirmed the patency of stents (Fig. 6).

On his last follow-up after nine months, we performed patient exercise electrocardiography and MSCA again. No abnormalities were seen on exercise electrocardiography and the MSCA showed only minimally neointimal hyperplasia involved stent which was implanted into distal LMCA across the ostium of LAD, other stents were patent. The patient was still asymptomatic. We planned to fallow up with MSCA exercise test, optimal platelet inhibition (assessed



**Figure 5**. Final angiographic result following stent implantation. Successful stent deployment at the desired position; grade 3 flow in infarct related artery and < 30% residual stenosis according to thrombolysis in myocardial infarction study.

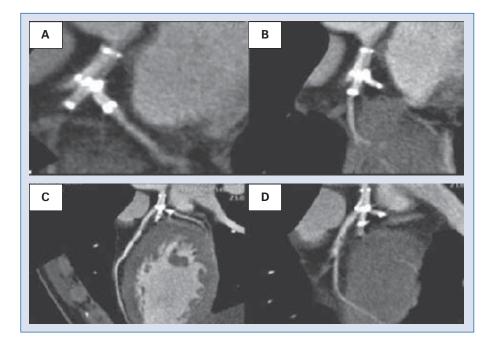


Figure 6. One month visit multislice computed tomography showed patency of all stents.

with 150 mg clopidogrel and 300 mg ASA) under quidance of platelet function test.

## Discussion

Acute LMCA occlusion is a rare clinical entity in patients with AMI who undergo primary PCI. Although previous studies have reported that the incidence of LMCA AMI was 0.37 to 2.96%, this assessment remains uncertain because catastrophic presentations (including malignant arrhythmia, pulmonary edema and sudden death) may underesimate the true incidence of acute LMCA occlusion before admission to the emergency unit [2–4].

Acute LMCA occlusion usually results in severe left ventricular dysfunction; clinical worsening results in a few minutes. A presence of collaterals, a dominant RCA, and a not totally occluded LMCA that is higher in the survival group compared to the mortality group has been noted in previous reports. Morover, AMI involving both LMCA and its branches (like trifucartion or bifurcation) is a rare disease and a challenging condition for interventional cardiologists, because it involves the extension of the myocardium complicating with cardiogenic shock and its technicial difficulties. Trifurcating coronary artery disease is a complex atherosclerotic process involving the origin of one or more of three side branches arising from a left main coronary vessel or trunk, with or without the involvement of LMCA itself [5, 6]. The patency of infarct-related artery is one of the strongest predictors of in-hospital and long-term survival in patients with AMI. LMCA AMI was usually presented with such conditions as cardiogenic shock, arrhytmia and pulmonary edema. Therefore reperfusion must have to be done quickly to prevent serious complication.

Thrombolytic therapy, emergency coronary artery bypass grafting, and primary PCI are regarded as the treatment approach for acute LMCA AMI [7--10]. Which reperfusion therapy was most suitable was not clearly known due to the absence of large randomized trials. In published series there has been controversy about the reperfusion strategy. In early series, Quigley et al. [1] and Chauhan et al. [11] proposed that patients presenting with AMI, severe left main stenosis, and cardiogenic shock (left main shock syndrome) have such a grave prognosis regardless of management, that conservative measures may be indicated. They do not support intervention in the presence of acute myocardial infarction/cardiogenic shock. As the techniques and experience have progressed, favorable data has been published. These recent articles showed that primary LMCA angioplasty was a feasible and effective procedure and it may save lives in this clinical setting. The ULTIMA registry and the study by Neri et al. [13] showed that better results can be attributed to use of intracoronary stents, newer antiplatelet therapy, and mechanical support. The results from the ULTIMA registry and Neri et al. [13] suggest that emergency percutaneous revascularization in patients with left main disease and AMI is technically feasible. The benefit of percutaneous coronary interventions on mortality is likely [12–15].

However, even when the coronary artery has been reperfused succesfully, follow up with LMCA remains more difficult than PCI procedures perfomed on other coronary arteries. Restenosis and the potential risk of sudden cardiac death induced by stent thrombosis of the left main coronary artery risks are problematic. For these reasons, LMCA AMI patients who were successfully revascularized, must be followed up closely. All patients are advised to visit the outpatient clinic every one or two months in the first months after stenting, every 2–3 months between 6–12 months and every 3–6 months after one year, if they are not displaying symptoms. If chest pain or any relevant symptoms develop after stenting, the patient should come to see the doctor immediately.Angiographic follow-up should be performed after six months, or earlier, if clinically indicated by symptoms or myocardial ischemia [16].

Antiplatelet regimen is another important medical treatment option in these patients due to the catastrophic results of stent thrombosis. As indicated in ACC/AHA/SCAI guidelines, for patients in whom sub-acute thrombosis may be catastrophic or lethal (unprotected left main, bifurcating left main, or last patent vessel) platelet aggregation studies may be considered and the dose of clopidogrel increased to 150 mg per day if less than 50% inhibition of platelet aggregation is demonstrated [17]. To evaluate platelet inhibition, multiplate electrical impedance aggregometry platelet function test was used and optimal inhibition was assessed with 150 mg clopidogrel and 300 mg aspirin.

In our patient, who presented with significant lesions in distal LMCA, osteal LAD, CX and IM arteries, our treatment approach was emergent PCI to stabilize the patient's condition. Given the risk of the treatment of the trifurcation lesions with LMCA, we first revascularisized LMCA and LAD. In the case of plaque displacement and occlusion of CX and IM, having a guide wire support in each element of the trifurcation, 'kissing' balloon or stenting technique would have been attempted to each coronary. Intravascular ultrasound sonography examination was performed after the stent implantation, excluding abnormalities such as wrong positioning of the stents and or shifting of any plaque from left main to any element of the trifurcation and distal portion. In routine clinical follow-up, the patient is still asymptomatic and there is no documented myocardial ischemia.

In our patient we have used multi-slice computed tomography (MSCT) for follow-up. Although there have been no randomized studies conducted to demonstrate the significance of MSCT on follow up of patients with LMCA stenting, MSCT is a non--invasive and practical way to characterize coronary arteries and stents around the heart [18, 19].

In our case, deployment of stent in the trifucation lesion contains LMCA in AMI phase seems feasible and not problematic.

To the best of our knowledge, this is the first case reported where a patient has been successfully revascularized and closely followed up with clinical and laboratory methods with MSCT and platelet function analyzer.

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