Successful treatment of left main shock syndrome induced by thrombosed coronary artery dissection A case report

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Abstract

Rationale: Left main shock syndrome (LMSS) induced by thrombosed coronary artery dissection is very rare and has a fatal prognosis. Optimal treatment strategy includes early reperfusion and hemodynamic support to prevent cardiogenic shock. However, it involves the extension of technical difficulties under different conditions.

Patient concerns: A 49-year-old woman developed symptoms of left main shock syndrome. The main clinical manifestations were retrosternal pain radiating to his back and left shoulder, heavy sweating, palpitation and brachypnea.

Diagnoses: Acute anterioseptal myocardial infarction (Killip Class IV) with cardiogenic shock and arrhythmia including ventricular tachycardia and idionodal rhythm, and coronary artery dissection.

Interventions: A thrombus aspiration procedure was performed for the establishment of coronary flow under intra-aortic balloon pumping (IABP) support. Her coronary angiographic finding demonstrated a dissection in the mid-distal segment of the left main coronary artery where a sirolimus-eluting stent was deployed. Then, the veno-arterial extra-corporal membrane oxygenation (ECMO) was placed to improve severe cardiac dysfunction and end-organ failure.

Outcomes: The patient had a good outcome without active symptoms.

Lessons: Thrombosis on the basis of coronary dissection is a very rare cause of LMSS. Successful team treatments, including the prompt thrombus aspiration and stent repair of artery dissection, potent IABP and ECMO support are important to improve the clinical outcome.

Abbreviations: ECMO = extra-corporal membrane oxygenation, IABP = intra-aortic balloon pump, LAD = left anterior descending artery, LCx = left circumflex, LMCA = left main coronary artery, LMSS = left main shock syndrome, PCI = percutaneous coronary intervention.

Keywords: case report, coronary artery dissection, left main shock syndrome, thrombosis

1. Introduction

Left main shock syndrome (LMSS) is defined as acute myocardial infarction (AMI) complicated by cardiogenic shock and left main coronary artery disease, and carries a very high morbidity in a coronary care unit.^[1] The embolic occlusion due to artery dissection in the left main coronary artery (LMCA) is an uncommon cause of LMSS. Therefore, no standard therapeutic procedure is recommended. Several studies have shown that early revascularization with hemodynamic support improves survival

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in patients with LMSS.^[2] But in this case, the lifesaving elements were prompt thrombus aspiration and endovascular repair of artery dissection with stenting of the LMCA under intra-aortic balloon pumping (IABP) support. Medical care with extra-corporal membrane oxygenation following revascularization saved the patient with LMSS. Written informed consent was obtained from the patient.

2. Case presentation

A 49-year-old female patient was admitted to our hospital due to 4 hours of acute retrosternal pain in her back and left shoulder, which is associated with sense of dving, heavy sweating, palpitation, and brachypnea. No relevant history of hypertension, diabetes, dyslipidemia, or familial risk factors for cardiovascular diseases was reported. She had a negative history for alcohol, tobacco, or drug use. On physical examination, the patient was a thin, healthy-appearing Chinese woman with a pained expression. Her heart rate was 116 beats per minute (bpm), and blood pressure was 80/50 mm Hg. Electrocardiogram demonstrated ST segment elevation in leads I, aVL, aVR, V4 through V6, and ST segment depression in lead II, III, aVF, V1 through V3 (Fig. 1). Initial laboratory data revealed that her probrain natriuretic peptide (BNP) was elevated to 702 pg/mL (0-40). Complete blood count tests showed an abnormal leucocytes count of 11.96×10^{9} /L (4–10×10⁹) and an increased neutrophile

The authors have no conflicts of interest to disclose.

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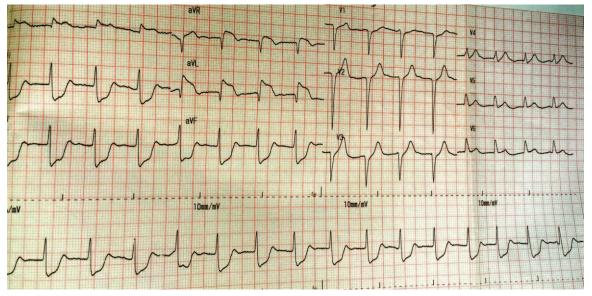


Figure 1. ECG showing ST segment elevation in leads I, aVL, aVR, V4 through V6, and ST segment depression in lead II, III, aVF, V1 through V3.

granulocytes percentage of 91 (50–70). Her initial set of cardiac enzymes displayed elevated levels of both creatine kinase (CK) of 11420U/L (0–40) and CK-MB of 1586U/L (0–40). Acute anterioseptal myocardial infarction (Killip Class IV) with cardiogenic shock and arrhythmia including ventricular tachycardia and idionodal rhythm were diagnosed and emergency cardiac catheterization was performed after 150J electric cardioversion.

A 6 Fr sheath (Medtronic, Mexico) was inserted through the right femoral artery and a 6F JL 4.0 guiding catheter (Medtronic) was used for emergency coronary angiography. Angiography showed total ostial occlusion of LMCA with thrombus material that was also accompanied by poor collateral filling to the left anterior descending artery (LAD) and left circumflex (LCx) (Fig. 2A). Considering the clinical characteristics of the patient, intra-aortic balloon pumping (IABP) was inserted through the left

femoral artery and thrombus aspiration was decided to be the optimal treatment for this case. Coronary soft guidewires (Runthrough, Terumo, Japan) were successfully passed into the LAD (Fig. 2B). Thrombus aspiration was performed with a 6 F aspiration catheter (Export AP, Medtronic) according to standard procedure in thrombus-containing lesions (Fig. 2C). The procedure was completed by multiple dilatations with a balloon of 2.5×15 mm (Sprinter Legend, Medtronic, Mexico) and an optimal distal flow with TIM 3 in the infarct-related artery was observed (Fig. 2D). Due to lack of evidence for atherosclerotic basis, a coronary artery dissection involving the LMCA was highly suspected. Coronary angiographic finding of a thin radiolucent line demonstrated a dissection in the mid-distal segment of the LMCA, extending bifurcation to proximal LAD and LCx arteries (Fig. 3A). In this case, LMCA stenting followed by final kissing-balloon inflation was performed for the treatment

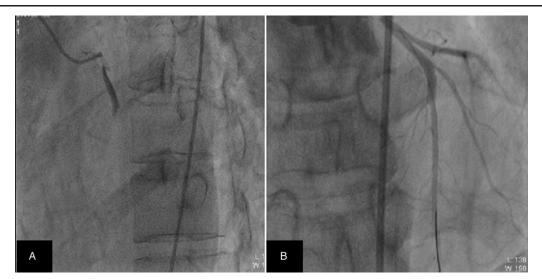


Figure 2. Coronary angiography of the left coronary artery. (A) Complete occlusion of LMCA. (B) Suboptimal angiographic result after thrombus aspiration (TIMI 3) in LMCA. LMCA=left main coronary artery.

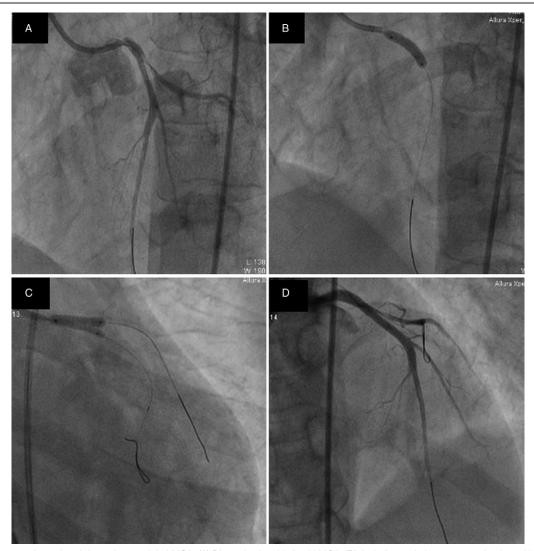


Figure 3. Coronary angiography of dissection repair in LMCA. (A) Dissection in mid-distal LMCA. (B) A sirolimus-eluting stent was implanted in LMCA. (C) Final kissing-balloon inflation in the bifurcation of LMCA. (D) Final angiogram showed good result. LMCA=left main coronary artery.

of bifurcation lesions. Guidewire (BMW ELITE, Abbott) was introduced rapidly through the true lumen of left coronary artery with confirmation by angiogram. A sirolimus-eluting stent (Microport, China) was then deployed from the middle of LMCA to the proximal segment of LAD using standard techniques (Fig. 3B). After stenting, guidewires were easily reinserted into the LCx. A Sprinter $3.5 \times 15 \text{ mm}$ balloon (NC) was inflated at the proximal LAD for 20 seconds at 16 atm, and subsequently a Sprinter $2.5 \times 15 \text{ mm}$ balloon (NC) was inflated at the proximal LCx for 20 seconds at 16 atm. The procedure finally showed positive results after simultaneous balloon inflation was performed at the LMCA bifurcation with both balloons at 8 atm (Fig. 3C and D). During operation, the patient experienced 3 times 150J electric cardioversion and cardiopulmonary resuscitation for 4 minutes due to cardiac arrest and repeated ventricular tachycardia.

The patient was monitored in the intensive care unit while experiencing unconsciousness, breathing shallow, hypotension, and hypoperfusion of end organs (cool extremities, urine output of 25 mL/h and heart rate of 90 beats/min) despite rapid and successful recanalization of the LMCA and IABP support.

Cardiac shock progressed very rapidly leading to inotropic support with dopamine and adrenaline. Then, the extra-corporal membrane oxygenation (ECMO) was performed to improve shock within 24 hours after admission. In the following days of ECMO therapy, left ventricular ejection improved to LVEF of 38%, and urine volume increased from 650 mL of the second day to 2840 mL of the fourth day. ECMO therapy was successfully removed after 4 days, but IABP support continued for another 6 days. The patient was extubated on her 12th hospital day followed by noninvasive positive pressure ventilation. The patient was discharged her 20th day in the hospital with no active symptoms.

3. Discussion

LMSS is less frequent in angiographic finding following acute myocardial infarction. But the mortality in patients with LMSS is about 55% to 80% despite aggressive medical therapy and mechanical support.^[3] The shock state in patients with myocardial infarction appears to be the result of a vicious cycle of reduced cardiac output, reduction in contractility and low

blood pressure due to coronary insufficiency.^[4] PCI and CABG are recommended as the treatment of choice in suitable patients with LMSS in order to immediate reperfusion of the causal lesion.^[5] In the present case, total occlusion of thrombus material found in the left main artery of the angiography was unexpected. Sudden total occlusion of LMCA caused disruption of coronary blood flow and cardiogenic shock. Emergency thrombus aspiration is more reasonable than CABG in order to achieve revascularization of left main artery without any signs of atherosclerosis. This method could be effective in restoring blood flow for myocardial infarction. But in almost every case thrombus aspiration is followed by coronary stent implantation on atherosclerotic basis.^[6] For better coronary reperfusion, IABP support also was considered the first option to improve coronary insufficiency and prevent heart function failure in the course of cardiogenic shock. Flugelman et al^[7] reported the first surviving patient who was treated with only an IABP following LMCA occlusion. Coronary embolism with no atherosclerotic basis is a very rare in acute myocardial infarction. But the known aetiologies of embolic material in coronary artery are quite wide.^[8] Coronary angiography showed LMCA dissection involving the LAD and LCx arteries after thrombus aspiration even though the cause of dissection was unexplained. The false lumen of coronary artery dissection can causes highly disturbed flow patterns and creates local hemodynamic conditions likely to promote the formation of thrombus in the false lumen.^[9] In fact, the possible complications induced by thrombus in coronary artery are influenced by the level of thrombosis in the false lumen. Thus, coronary artery dissection was the most plausible cause that induces thrombosis of left main artery in this case.

Due to unstable hemodynamic status in patient, the important way to manage this condition was to reduce blood flow disturbance and to improve hemodynamic status before arterial dissection repair. It was more feasible and safer implantation of stent other than CABG in the catheterization laboratory. Revascularization of stenting only the main branch and performing balloon angioplasty of the side branch was selected as optimal therapy for arterial dissection lesion. Although there were relative high restenosis rates and significant incidences of sudden cardiac death,^[10] angioplasty by intracoronary stenting in LMCA could limit the extension of dissection and result positively. This outcome would allow the true lumen to reexpand while relieving that thrombus burden of the false lumen. The successful deployment of single stent to LMCA was followed by the kissing balloon dilation of the LAD artery and the LCx artery for treatment of dissection involving bifurcation lesions. The simultaneous kissing balloon inflation is considered preferable for bifurcated lesions of coronary arteries in terms of long-term clinical outcome through expanding large caliber proximal vessels without overdilating smaller vessels distal to the bifurcation site.^[11] Eventually, the interventional procedure in this patient was satisfactory. In this case, after timely coronary revascularization the ischemia/reperfusion injury of cardiomyocytes further worsened and resulted in cardiogenic shock with severe haemodynamic collapse. Rapid recovery from post-acute myocardial infarction complicated by this cardiogenic shock using veno-arterial ECMO has been previously shown.^[12] Venoarterial ECMO is suitable to patients with severe cardiac failure with haemodynamic compromise, when the cardiac dysfunction is thought to be reversible. As shown here, veno-arterial ECMO therapy could improve the progressive cardiac failure and endorgan failure after coronary stenting bridging haemodynamic compromise.

In conclusion, we report the survival case of LMSS induced by LMCA occlusion. Thrombotic occlusion induced by arterial dissection of LMCA is an extremely rare cause of acute myocardial infarction resulting in cardiogenic shock. There is no standard treatment procedure in these cases to recommend. Therefore, treatment strategy should be individualized. After the prompt and suitable interventional therapy for thrombus aspiration and dissection repair of LMCA, potent IABP and ECMO support should be performed for restitution of cardiac function in patients with LMSS.

Author contributions

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