The Heart Surgery Forum #2014-442
18 (3), 2015 [Epub June 2015]
doi: 10.1532/hsf.1321

Online address: http://journal.hsforum.com

Coronary Embolism Causing Acute Inferior Wall and Ventricular Myocardial Infarction in a Patient with Rheumatic Valvular Heart Disease: Treatment with Thrombus Aspiration

Xiao-Yu Du, Peng Hui, Yang Zheng

The Cardiovascular Center, The First Hospital of Jilin University, Changchun, China

ABSTRACT

An elderly man with rheumatic valvular heart disease and atrial fibrillation presented with acute myocardial infarction. A coronary angiogram revealed complete occlusion of the right coronary artery (RCA), and we therefore considered that a thrombus might have obstructed the ostium of the RCA. We used a guiding catheter and the syringe of an aspiration device to remove two large dark red thrombi. A subsequent angiogram revealed that blood flow through the RCA had recovered, and the endomembrane of the RCA was smooth, with no evidence of stenosis or residual thrombus. In this case, thrombus aspiration via a guiding catheter was efficacious for treating this type of coronary embolism.

INTRODUCTION

Acute myocardial infarction (AMI) is most often caused by rupture of atherosclerotic plaque in the coronary artery, resulting in formation of a secondary thrombus. However, it is rare for a coronary embolism to cause an AMI. In this report, we describe a case in which a patient with rheumatic heart disease developed a secondary coronary embolism. We used a guiding catheter and the syringe of an aspiration device to remove the thrombus. The patient was successfully treated without the use of balloon dilation or stent implantation.

CASE REPORT

A 65-year-old man complaining of sudden onset right lower limb pain for 2 hours and chest pain for 1 hour was transferred to our emergency department (ED). He had a history of rheumatic valvular heart disease and chronic atrial fibrillation (AF), but no history of taking oral anticoagulation medications. A physical examination showed a normal level of consciousness, a respiration rate of 22 breaths/minute, a heart rate of 63 beats/minute, and a blood pressure reading of 76/52 mmHg. Following initial treatment with a vasopressor (dopamine 5 ug/min/kg) to maintain blood pressure

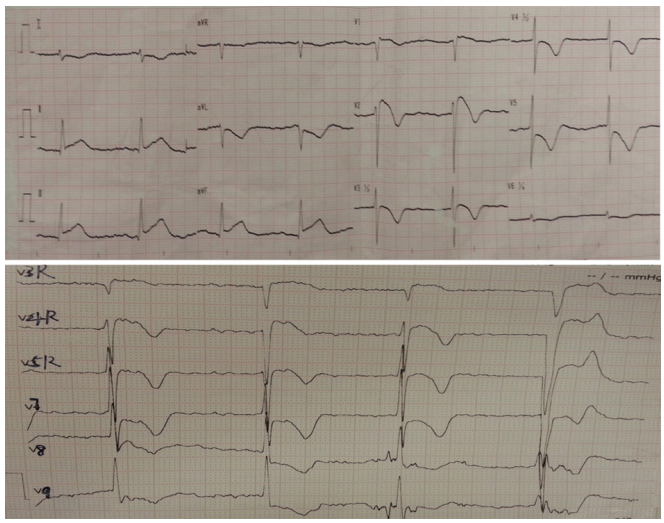

Figure 1. 18-lead electrocardiogram when patient entered the hospital.

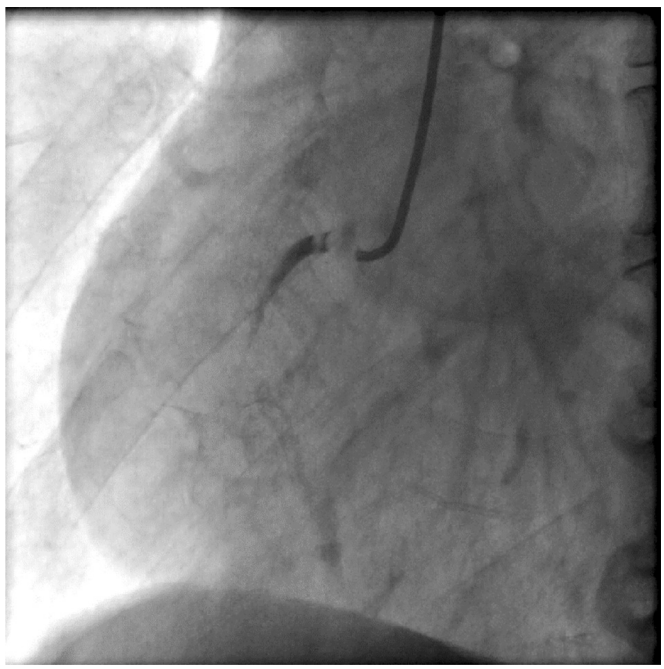

Figure 2. Ostium of RCA showing thrombus, thrombosis, and contrast media detention.

Received October 25, 2014; accepted November 16, 2014.

Correspondence: Yang Zheng, The Cardiovascular Center, The First Hospital of Jilin University, No.71 Xinmin Street, Changchun 130021, China; +86-431-88783787; fax: +86-431-85664811 (e-mail: yangzheng@163.com).

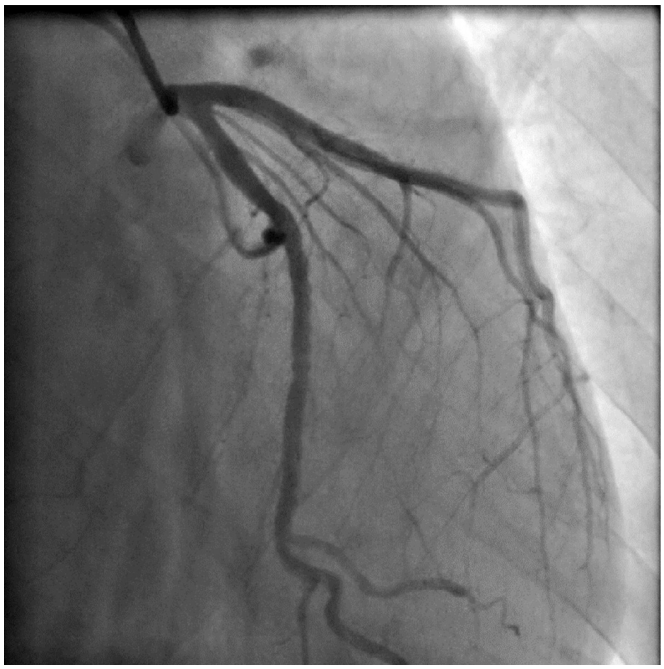

Figure 3. LAD and LCX showing no obvious stenosis.

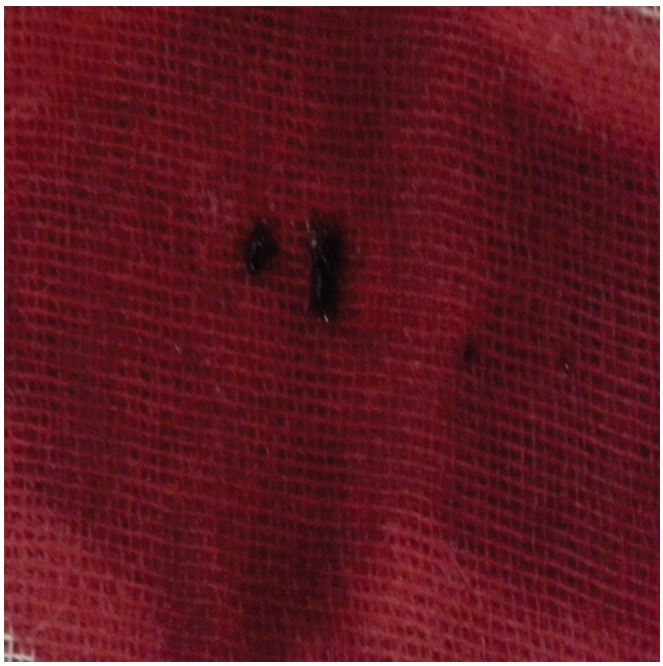

Figure 4. Dark red thrombus obtained by aspiration.

in a range of 110-120/60-70 mmHg, the patient's pulse was 72 beats/minute. Rude respiration, some moist rales, and rhonchus were auscultated in the bottom portions of the lungs. During arrhythmia, the S1 at the apex was louder or weaker, and a mid-diastolic murmur grade III/VI was noted at the apex. The skin temperature of both lower limbs was low, and especially on the right side. The pulse in the left pedis

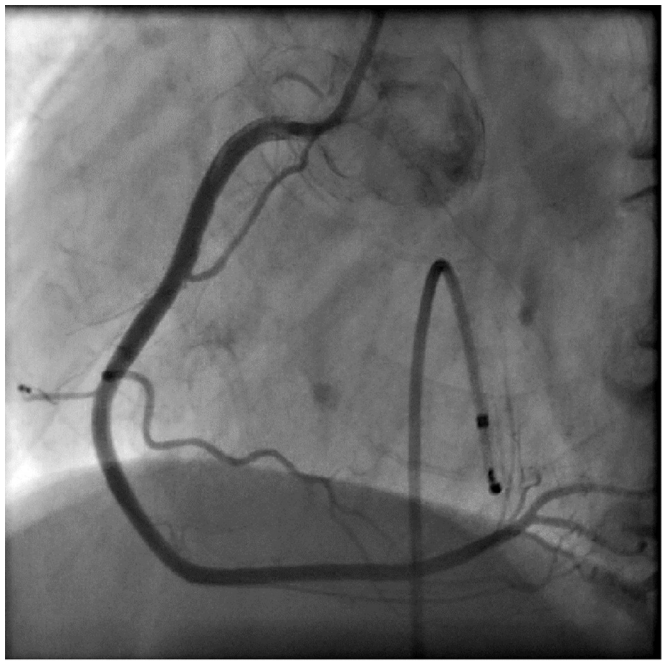

Figure 5. Image of RCA after aspiration showing no stenosis or thrombosis.

artery was abated, while the pulse on the right side was unaffected. An 18-lead electrocardiogram (ECG) showed atrial fibrillation, ST elevation in leads II, III, aVF of ~ 0.2 mV, and a 0.1 mV elevation in leads V3R-V4R (Figure 1). Laboratory tests were significant for myoglobin (384.7 ng/mL), troponin (0.11 ng/mL), CK-MB (3.5 ng/mL), international normalized ratio (INR) (1.13), and prothrombin time (PT) (13.6 s). The levels of BNP and D-dipolymer were 2590 pg/mL and 692 ug/L, respectively. The patient was primarily diagnosed with coronary heart disease, with acute inferior wall right ventricular ST-segment elevation myocardial infarction, Killip II. Treatment was initiated by having the patient chew aspirin-containing (300 mg) gum and taking oral clopidogrel (600 mg). The patient was then evaluated by coronary angiography (through the right femoral artery) in the catheter lab. Angiography demonstrated a complete occlusion at the opening of the RCA, which showed only a small amount of internalized contrast agent, with TIMI flow 0 (Figure 2). The left anterior descending (LAD) and left circumflex (LCX) arteries did not show significant stenosis or thrombosis (Figure 3). Angiographic features strongly suggested an embolic event in the RCA. After an intravenous injection of heparin, we placed the JR4.0 GC in the opening of the RCA. We then connected a negative pressure syringe (DIVER C.E MAX kit) to three consecutive plates, and successfully used negative pressure suction to remove two large pieces of dark red thrombus, sized 4.0 mm × 2.5 mm and 2.5 mm × 2.5 mm (Figure 4). When clots could no longer be detected inside the syringe, tirofiban (10 mL) was injected by a guiding catheter. Finally, radiography showed the thrombus had disappeared, and contrast agent reaching the terminal of the RCA in two cardiac cycles. The endomembrane of the RCA was smooth, showed no stenosis or thrombus, and a TIMI

flow 3 (Figure 5); additionally, the patient's chest pain symptoms disappeared. ECG showed elevated-ST in leads II, III, and aVF had returned to normal. Cardiac color ultrasound (UCG) results showed a 60% ejection fraction (EF), a left atrial diameter of 46 × 53 × 67 mm, and slow blood streaming in the left atrium. Additionally, a parenchyma echo was seen in the left atrium near the left auricular appendage, sized 25 mm × 15 mm. The mitral valve echo was enhanced, indicating a thicken wall change. The diagnosis by ultrasound was rheumatic valvular heart disease, with thrombosis in the left atrium. Examination by double lower limb artery color Doppler ultrasound showed bilateral lower extremity atherosclerosis, the upper right pretibial artery embolized, and occlusion of the right side of the middle period of the peroneal artery. A diagnosis of right lower limb artery embolization was made by vascular surgery. The patient was treated with a vasodilator, an anticoagulant, and a pain reliever. Based on the patient's symptoms, his ECG results, cardiac enzyme test results, a RCA angiogram conducted after thrombus aspiration, and UCG, we concluded that rheumatic mitral stenosis and atrial fibrillation had led to left atrial thrombosis. We further hypothesized that the thrombus subsequently detached and fell onto the ostium of the right coronary artery, leading to myocardial infarction. It may have then entered the right lower limb artery, resulting in lower limb artery embolization. The patient was treated with anti-platelet medication combined with low molecular heparin, and warfarin given post-operatively. The antiplatelet medication was discontinued after one week. At 7 days after admission to the hospital, the patient reported no symptoms of pain. His ECG and elevated ST segment returned to baseline, his T wave inversion and pathologic Q wave had resolved, and at the same time, his right lower limb pain was significantly reduced. Additionally, the temperature in both lower limbs returned to normal, and a pulse was detected when touching the right foot. The patient's condition improved, and he was released from the hospital. His warfarin dose was adjusted based on the results of regular INR tests conducted in the outpatient clinic.

DISCUSSION

Acute myocardial infarction resulting from a coronary artery embolism is a rare event. Melendez reported that only 5-13% of AMIs are derived from an embolic source, and most literature on this topic has been presented as a case report [Marta 2012; Byramji 2011; Kohl 2010; Sial 2009; Charles 1982]. The underlying causes of the event may include an infective endocarditis neoplasm, rheumatic mitral stenosis complicated with left atrial thrombus, atrial fibrillation, heart valve replacement, atrial myxomas, and in very unusual situations, patent foramen ovale. Patients with a rheumatic mitral value are prone to thrombus formation caused by atrial dilation, disturbed blood flow, and atrial fibrillation. The causes of AMI in patients with a rheumatic mitral valve include left atrial dilation and atrial fibrillation, which may cause a thrombus or mural thrombus to dislodge, leading to a coronary embolism. Manzano [Manzano 2007] reported that AMI resulting from a coronary embolism is likely to occur in the LAD artery. The reason for this may be related to the anatomy of the coronary blood flow system, in which the left coronary sinus has a larger diameter and greater blood flow than the right coronary sinus. Because the LAD artery is an extension of the left coronary artery, an embolism may enter the LAD artery and cause an anterior myocardial infarction. When examining an angiogram, an AMI derived from a coronary artery embolism presents as a coronary artery occlusion or embolism. Treatment of AMI by a preferred interventional therapy [Yazici 2007; Sakai 2007; Levis 2011] such as coronary thrombus aspiration, balloon dilation, or stent implantation can recover prograde flow, and improve the prognosis for patients. In most cases, coronary thrombus aspiration cannot entirely aspirate the thrombus, and a portion may migrate further into the blood vessel; however, blood flow can still be partially restored. Dilation and extrusion of the blocked site with a balloon can also restore blood flow in the targeted vessel; however, stent implantation is not a preferred treatment, due to a low risk of producing stenosis in the target vessel.

This case reported here is rare, because the patient's left atrium thrombus detached, resulting in simultaneous embolisms in the RCA and right leg. After completing the coronary angiogram, we placed a JR4.0 GC in the opening of the RCA. To prevent a bubble from entering the coronary artery, we attempted to retract blood through the guiding catheter using a 20 mL syringe; however, the blood could not be retracted. We therefore considered that the embolus might be large enough to have blocked the opening of the guiding catheter. Additionally, if the guiding catheter had been removed from the opening of the RCA at that time, the embolus might have dislodged and fallen into the aorta, becoming available to block blood flow in other areas of the body. Therefore, we chose not to send the guide wire to the terminus of the RCA, and also not to employ balloon dilation. Instead, we used the negative pressure syringe in the DIVER C.E MAX kit connected to the tee-plate to repeatedly aspirate the thrombus through the guiding catheter, which had a diameter wide enough to permit complete aspiration. Using this method, we successfully aspirated two large dark red thrombi that had occluded the opening of the RCA. This procedure restored RCA blood flow in a very short time, reduced reperfusion time, and obviously improved the patient's prognosis. We believe this procedure should be considered for patients who present with a large thrombus that obstructs the opening of the coronary artery.

This case report has some limitations that should be mentioned. First, we did not apply intravenous ultrasound (IVUS) following aspiration of the thrombus. Although the coronary angiogram did not reveal obvious stenosis, IVUS could have detected the presence of endovascular atherosclerosis. Li [Li 2012] utilized thrombus aspiration to treat a patient with RCA occlusion. In that case, a subsequent IVUS examination revealed that the entire endomembrane of the RCA was smooth, and the presence of a small amount of thrombus residue. However, there was no evidence of atherosclerotic plaque; confirmation that the RCA occlusion was a thromboembolism. Second, we did not conduct a pathological examination of the aspirated thrombus. Kotooka [Kotooka

2004] conducted pathological examinations of the aspirated thrombi obtained from 3 patients with coronary embolisms due to atrial fibrillation. The results showed an abundance of red blood cells, but only small numbers of white cells and platelets, and a small amount of fibrous protein. These contents differed from those of a typical coronary embolism, which is enriched in blood platelets, fibrous protein, and cholesterol crystals. He therefore reasoned that the AMI were caused by coronary embolisms. Third, we did not conduct a thrombectomy for treatment of the lower leg embolism. It would have been useful to conduct simultaneous pathologic examinations of both the coronary thrombus and the lower leg embolism. Such results could have been used to confirm that the contents of the embolus and thrombus were basically the same, and that dislodgement of the thrombus may have led to the formation of embolisms at multiple sites.

Finally, during periods of atrial fibrillation, embolisms can form in the coronary artery and leg due to greater than normal clotting activity. Cervera [Cervera 2007] proposed that anticoagulation therapy may be effective for preventing cardiac valve disease, and especially in patients who have experienced an embolic event due to atrial fibrillation. Antiplatelet and anticoagulant drugs can prevent a remote-end embolism from worsening, and reduce the risk of thrombus formation following treatment for a coronary embolism. Therefore, we recommend using an antiplatelet drug at the beginning of treatment, and later prescribing warfarin accompanied by INR monitoring for anticoagulation treatment when the patient is in stable condition.

REFERENCES

Byramji A, Gilbert JD, Byard RW. 2011. Sudden death as a complication of bacterial endocarditis. Am J Forensic Med Pathol 32:140-2.

Cervera A, Amaro S, Obach V, Chamorro A. 2007. Prevention of ischemic stroke: antithrombotic therapy in cardiac embolism. Curr Drug Targets 8:824-31.

Charles RG, Epstein EJ, Holt S, Coulshed N. 1982. Coronary embolism in valvular heart disease. Q J Med 51:147-61.

Kohl S, Bartel T, Mueller S, Pachinger O, Metzler B. 2010. Acute myocardial infarction involving two coronary arteries due to a patent foramen ovale. Wien Klin Wochenschr 122:465.

Kotooka N, Otsuka Y, Yasuda S, Morii I, Kawamura A, Miyazaki S. 2004. Three cases of acute myocardial infarction due to coronary embolism: treatment using a thrombus aspiration device. Jpn Heart J 45:861-6.

Levis JT, Schultz G, Lee PC. 2011. Acute myocardial infarction due to coronary artery embolism in a patient with a tissue aortic valve replacement. Perm J 15:82-6.

Li Zhong-you LJ, Chen Hong. 2012. Coronary embolism causing acute myocardial infarction in a patient with paroxysmal atrial fibrillation. Chin J Cardiovasc Med 17:424.

Manzano MC, Vilacosta I, San Roman JA, et al. 2007. Acute coronary syndrome in infective endocarditis. Rev Esp Cardiol 60:24-31.

Marta L, Peres M, Alves M, Ferreira da Silva G. 2012. Giant left atrial myxoma presenting as acute myocardial infarction. Rev Port Cardiol 31:815-19.

Sakai K, Inoue K, Nobuyoshi M. 2007. Aspiration thrombectomy of a massive thrombotic embolus in acute myocardial infarction caused by coronary embolism. Int Heart J 48:387-92.

Sial JA, Ferman MT, Saghir T, Rasool SI. 2009. Coronary embolism causing acute myocardial infarction in a patient with mitral valve prosthesis: successful management with angioplasty. J Pak Med Assoc 59:409-11.

Yazici M, Kayrak M, Turan Y, Koc F, Ulgen MS. 2007. Acute coronary embolism without valve thrombosis in a patient with a prosthetic mitral valve--successful percutaneous coronary intervention: a case report. Heart Surg Forum 10:E228-30.