Aspiration Thrombectomy of a Massive Thrombotic Embolus in Acute Myocardial Infarction Caused by Coronary Embolism

Koyu SAKAI,1 MD, Katsumi INOUE,2 MD, and Masakiyo NOBUYOSHI,1 MD

SUMMARY

Coronary embolism is one of the less common causes of acute myocardial infarction (AMI). We describe a 72-year-old man with atrial fibrillation having an AMI, in whom a massive intracoronary thrombus of the right coronary artery was successfully removed by aspiration via a thrombectomy catheter, achieving successful reperfusion with a complete resolution of ST-segment elevation. The appearance of the aspirated material suggested coronary embolism was the cause of the AMI. It is concluded aspiration thrombectomy is a feasible and safe approach for treating coronary embolism. (Int Heart J 2007; 48: 387-392)

Key words: Acute myocardial infarction, Coronary embolism, Aspiration thrombectomy, Atrial fibrillation

ALTHOUGH disruption of atherosclerotic plaques and intracoronary thrombus formation is widely accepted as the main pathophysiological cause for the development of acute myocardial infarction (AMI),1) coronary embolism is one of the less common causes of AMI. In this report, we describe a patient with AMI most likely caused by coronary embolism complicating atrial fibrillation and evidence of a massive thrombus in the infarct-related artery in whom aspiration via a thrombectomy catheter resulted in effective removal of a massive thrombus and successful reperfusion was achieved.

CASE REPORT

A 72-year-old man was admitted to our emergency room with constant severe chest pain lasting about 21 minutes. His diabetes was controlled with diet therapy. He had a history of coronary stent implantation of the left anterior descending artery for unstable angina 2 years before this admission. Past medical
history was notable for long-standing hypertension and permanent nonvalvular atrial fibrillation treated with warfarin and aspirin. Physical examination on admission revealed he had a systolic blood pressure of 102 mmHg, was cold and sweaty, had obtunded consciousness, and was in obvious imminent vascular collapse. An electrocardiogram (ECG) showed ST-segment elevation in leads II, III, aVF, V₁, V₂, and V₃, as well as atrial fibrillation and third-degree atrioventricular block. He was transferred to the catheterization laboratory for coronary angiography. Upon securing blood access to the right femoral artery, heparin (10,000 U) was administered. Coronary angiography revealed a left coronary artery without significant stenosis and identified the infarct-related artery as being the proximal segment of the right coronary artery (RCA). The proximal segment was totally

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**Figure 1.** Angiograms of a 72-year-old male patient with acute myocardial infarction. A: Initial selective coronary angiography showed total occlusion at the proximal segment of RCA (LAO view). B: Aortography revealed a filling defect protruding from the RCA ostium into the ascending aorta suggestive of a massive thrombus (arrow). C: A 1.5-mm balloon was inflated. D: The underlying thrombotic lesion was delineated, and estimated to be about 30-40 mm long. E: The Thrombuster™ aspiration catheter successfully passed through the lesion (arrow). F: Final results after repeated aspiration. LAO, left anterior oblique; and RCA, right coronary artery.
occluded with Thrombolysis in Myocardial Infarction (TIMI) grade 0 flow, in agreement with the ECG changes (Figure 1A). Because of the atypical appearance of the occlusion site, aortography was performed to rule out other causes of myocardial infarction such as aortic dissection. Aortography revealed a filling defect protruding from the RCA ostium into the ascending aorta suggestive of a massive thrombus (Figure 1B). Because the AMI was close to being complicated by cardiogenic shock, primary coronary intervention was undertaken instead of thrombolysis. An 8 French JR 4 guiding catheter was positioned in the coronary ostium, which allowed the lesion to be easily crossed with a 0.014 inch coronary guidewire. In order to visualize the whole image of the occluding lesion, a 1.5-mm Ryujin OTW (TERUMO, Tokyo) balloon was inflated to 15 atm for 1 minute (Figure 1C); the time from onset of symptoms to the first balloon angioplasty was 43 minutes. The underlying thrombotic lesion was delineated, and estimated to be at least 30-40 mm long (Figure 1D). Because of the large amount of angiographically visible thrombus, it was thought that conventional balloon angioplasty or stent implantation could result in distal embolization or no-reflow. Therefore, a Thrombuster™ catheter (KANEKA, Osaka, Japan) was used instead. Platelet glycoprotein IIb/IIIa receptor antagonists, which are not available in Japan, were not used. A Thrombuster™ catheter compatible with an 8 French guiding catheter was slowly advanced while manually aspirating 4 times through the culprit lesion, which removed a massive intracoronary thrombus, resulting in complete disappearance of the filling defect (Figure 1E). The final angiography after repeated aspiration revealed almost no residual stenosis, no dissection, and no distal embolization; TIMI grade 3 flow was established (Figure 1F).

In spite of right ventricular pacing and intra-aortic balloon pumping, hemodynamic instability progressed to cardiogenic shock during the interventional procedure because of third-degree atrioventricular block. The condition of the patient stabilized after successful reperfusion.

The patient was transferred to the coronary care unit where continuous infusion of heparin 10,000 U/day for 4 days was undertaken, and the oral combination of warfarin (international normalized ratio [INR] 2.0 to 3.0) plus aspirin 200 mg/day was re-instituted. The percentage resolution of ST-segment elevation at 60 minutes after reperfusion was 77.5%, which according to the Schroder classification would be categorized as complete (≥ 70%).2,3) Seven hours after the interventional procedure, all of the vascular sheaths, including the intra-aortic balloon, were removed with no complications. Creatine kinase peaked at 2929 IU/L (normal, 30-180 IU/L), and transesophageal echocardiography showed neither thrombus in the cardiac chambers, nor atrial septal defect or patent foramen ovale. The patient was discharged on the 17th day postinfarction. During his hospitalization, there were no recurrent ischemic events or other complications.
The material aspirated by the Thrombuster™ catheter consisted of large red thrombi without yellow atheromatous plaques. The thrombotic specimen was analyzed histopathologically and immunohistochemically (Figure 2), however, no component of vessel wall structures or atherosclerotic material could be detected.

**DISCUSSION**

The present case had the unique angiographical appearance of the proximal occlusion site not being within the infarct-related artery but rather in the ascending aorta. In addition, successful reperfusion was achieved practically only by aspiration thrombectomy. The main constituents of thrombi occurring in veins and cardiac chambers are red blood cells and fibrin, and therefore its macroscopic appearance is typically red, whereas arterial thrombi, especially those formed in atherosclerotic lesions, are rich in platelets giving them a white macroscopic appearance. Given the gross findings and microscopic appearances of the aspirated materials showing massive accumulation of erythrocytes, plenty of fibrin...
deposition, and small clusters of activated platelet aggregates, in this case, we may surmise that the AMI was caused by coronary embolism complicating atrial fibrillation.

Nonvalvular atrial fibrillation is the most common cardiac disease associated with systemic embolism, especially with cerebral embolism. Though little information is available showing the important link between atrial fibrillation and risk factors for AMI, many studies have evaluated the risk of stroke in patients with atrial fibrillation.\(^5,6\) Risk factors that predict stroke in patients with nonvalvular atrial fibrillation include a history of previous stroke or transient ischemic attack (relative risk 2.5), diabetes (relative risk 1.7), history of hypertension (relative risk 1.6), and increasing age (relative risk 1.4 for each decade); also, patients whose only stroke risk factor is congestive heart failure or coronary artery disease have stroke rates approximately 3 times higher than do patients without any risk factors.\(^7\) This case with nonvalvular atrial fibrillation had the following risk factors for stroke: diabetes, hypertension, advanced age, and coronary artery disease, and he was at high risk of thromboembolism. Therefore, these data support our hypothesis.

Intracoronary thrombus in the infarct-related artery remains a challenge for interventional catheter-based techniques in AMI. Distal embolization of thrombus during balloon inflation or stent deployment carries an increased risk of poor clinical outcomes;\(^8-12\) in a recent study, it was associated with an 8-fold increase in 5-year mortality.\(^11\) Thus, distal embolization is considered to be a major cause of insufficient reperfusion, despite a fully patent infarct-related artery. Even to date, there is no consensus as to the best management when treating AMI caused by coronary embolism. In this case, aspiration thrombectomy achieved successful reperfusion with a complete resolution of ST-segment elevation to re-establish cardiac function. Thus, the treatment for AMI should include attempts to correct microvascular perfusion as well as large-vessel perfusion.

We utilized the Thrombuster\textsuperscript{TM} (KANEKA) catheter system to effectively diagnose and treat coronary embolism without complication. The Thrombuster\textsuperscript{TM} is an aspiration catheter for coronary and peripheral use with a central aspiration lumen, and can be used with a 0.014 inch guidewire. There are 4 different sizes, each of which is compatible with a 6, 7, 8, or 9 French guiding catheter; a larger catheter may yield a greater aspirated volume. In this case, a Thrombuster\textsuperscript{TM} catheter that could accommodate an 8 French guiding catheter was used, having an outer diameter of 5.25 French and an inner aspiration lumen area of 1.43 mm\(^2\).

We have reported that 1) aspiration thrombectomy removed a massive thrombus from the native coronary artery of a patient with AMI, achieving successful reperfusion with a complete resolution of ST-segment elevation; and 2) the AMI was most likely caused by coronary embolism complicating atrial fibril-
lation. This case demonstrates that aspiration thrombectomy can be a feasible and safe approach for treating coronary embolism.

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**REFERENCES**