CASE REPORT

A Rare Case of Acute Myocardial Infarction with Multivessel Coronary Artery Ectasia Successfully Treated with Percutaneous Coronary Intervention and Systemic Thrombolysis

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Abstract

Coronary artery ectasia (CAE) is defined as a coronary artery dilatation with a diameter ≥1.5 times greater than that of a normal adjacent artery. All 3 coronary vessels can be affected by CAE, but the incidence of multivessel CAE among patients undergoing coronary angiography is quite low. We herein report an extremely rare case of acute myocardial infarction due to massive thrombi in the giant right coronary artery with multivessel CAE. Thrombus aspiration during percutaneous coronary intervention may be limited in giant coronary artery cases, and systemic thrombolysis may be effective in patients with massive thrombi in the giant coronary artery.

Key words: coronary artery ectasia, myocardial infarction, percutaneous coronary intervention, thrombolysis

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Introduction

Coronary artery ectasia (CAE) is defined as the dilatation of an arterial segment to a diameter at least 1.5 times that of the adjacent artery and is incidentally found in 1-4% of patients undergoing coronary angiography (1, 2). The most common known cause of acute coronary syndrome is plaque rupture; however, it can also be caused by CAE through sluggish or turbulent blood flow in the ectasia which can lead to thrombus formation and distal embolization (3). Because patients with multivessel coronary artery ectasia (MCAE) rarely undergo coronary angiography (4, 5), their management has not yet been well established, and options about the optimum management strategy are currently based on personal experience, single-case reports, and small-series reports (6, 7). In this report, we describe an extremely rare case of acute inferior myocardial infarction (MI) due to massive thrombi in the giant right coronary artery (RCA) in a patient with MCAE who underwent successful systemic tissue-plasminogen activator (t-PA) infusion after percutane-

Case Report

A 49-year-old man presented with prolonged chest pain and nausea for 12 hours. He had a history of hypertension and dyslipidemia but had not been prescribed any medications. He was not a smoker and did not have a previous history of chest pain. On the physical examination, the patient’s heart rate was 72 beats/min and his blood pressure was 160/90 mmHg. His chest roentgenogram, pulmonary breath sounds, and heart sounds were normal. An electrocardiogram showed ST segment elevation in the II, III, and aVF leads (Fig. 1A), and transthoracic echocardiography revealed hypokinesia in the inferior wall and a left ventricular ejection fraction of 40%. The creatine kinase level was 2,942 U/L with a muscle brain fraction of 113 U/L. Other laboratory examinations showed a white blood cell count of 16,200/µL, lactate dehydrogenase of 662 U/L, creatinine level of 0.79 mg/dL, low density lipoprotein of 183 mg/dL, high density lipoprotein of 54 mg/dL, triglyceride of 63 mg/
dL, and hemoglobin A1c of 6.0%. The patient was diagnosed with ST segment elevation MI (Killip class 1), and we began a regimen of aspirin (162 mg) and clopidogrel (300 mg loading dose). We then immediately planned to perform PCI due to the patient’s continuous chest pain.

A 6-Fr, 25 cm sheath was inserted into the right femoral artery. After the sheath insertion, 10,000 units of unfractionated heparin were administered intravenously. Coronary angiography showed MCAE with massive thrombi in the giant RCA and diagonal artery stenosis (Fig. 2). Because the thrombolysis in MI (TIMI) grade flow was 0, we attempted PCI. An 8-Fr, 25 cm sheath was used as back-up support to perform PCI. An 8-Fr percutaneous transluminal coronary angioplasty-guiding catheter (Judkins-Right 4.0 with side hole, Vista Brite tip⡴; Cordis, Johnson and Johnson, New Brunswick USA) was engaged, and a Runthrough® floppy (Terumo, Tokyo, Japan) coronary angioplasty 0.014-inch guide wire with a Mogul® (Goodtec, Seki, Japan) microcatheter was inserted into the giant RCA. However, an intravascular ultrasound (IVUS) (Eagle eye platinum⡴, Vol-

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Figure 1. Electrocardiograms (A) at admission, (B) after percutaneous coronary intervention, and (C) on the 12th day.

Figure 2. (A) Coronary angiography of the giant right coronary artery, LAO-Cranial view. There was 90% stenosis with thrombus of the proximal segment and total occlusion due to thrombus of the distal segment. The proximal vessel diameter of the proximal segment was approximately 10mm. LAO: left anterior oblique. (B) Coronary angiography of the giant left coronary artery, RAO-Caudal view. The vessel diameters of the proximal segment were 7.7mm (left anterior descending artery) and 6.3mm (left circumflex artery). RAO: right anterior oblique. (C) Coronary angiography of the giant left coronary artery, AP-Cranial view. There was a 90% stenotic lesion in the diagonal branch. AP: anteroposterior
cano, San Diego, USA) showed a vessel diameter of approximately 10 mm, making it difficult to perform the stent implantation and obtain a TIMI grade flow 3 (Fig. 3). Because a 7-Fr Thrombust® IIIGR system (Kaneka Medix, Osaka, Japan) could not pass the occluded lesion, we replaced the Runthrough® guide wire with a Grandslam® (Asahi Intecc, Nagoya, Japan) guide wire for extra support. After the exchange, we were able to perform thrombus aspiration with the Thrombust® IIIGR; however, we were unable to remove the giant thrombus from the RCA distal segment. Next, we attempted to perform thrombus aspiration using another device (Dio®, Goodtec) and successfully removed small amounts of thrombi. After completing thrombus aspiration, we performed angioplasty for the RCA distal segment with a Mini Trek© 2.5×15 mm balloon (Abbott vascular, Santa Clara, USA) (Fig. 4). Even after the thrombus aspiration and balloon dilatation, the TIMI grade flow was 1 (Fig. 5) and an intravascular ultrasound showed massive residual thrombi. Therefore, we administered 24 million units of alteplase (t-PA) intravenously. After t-PA infusion, the patient’s symptoms gradually improved and his ST segment elevation partially improved (Fig. 1B).

The first day after PCI, we continued treatment with aspirin (100 mg/day) and began a regimen of irbesartan (100 mg/day), bisoprolol (1.25 mg/day), atorvastatin (20 mg/day), and eplerenone (25 mg/day). The patient’s maximum creatine kinase level reached 4,792 U/L with a muscle brain fraction of 530 U/L. Anticoagulation therapy with continuous infusion of unfractionated heparin was started on the second day after the sheath removal due to puncture-site bleeding after the t-PA infusion. After the patient’s condition stabilized, oral warfarin therapy was started, and the prothrombin time international normalized ratio was maintained at 2.0-3.0. We considered the possibility that the patient had IgG4 associated inflammatory disease; however, his IgG4 level was 80 mg/dL. The clinical course was uneventful. We performed second coronary angiography on the 12th day after the primary PCI, and the patient’s TIMI grade flow increased to 3 (Fig. 6). However, the ST segment remained elevated in the inferior leads (Fig. 1C), and the left ventriculogram showed reduced anterior and inferior wall motion (Fig. 7). The patient was discharged on the 13th day with medical therapy. On the 34th day after discharge, we performed cardiac magnetic resonance imaging (MRI) that showed delayed enhancement of the anterior and
inferior wall areas (Fig. 8). We continued to follow the patient for 6 months during which time he patient experienced no chest pain or heart failure.

**Discussion**

CAE is attributed to atherosclerosis in 50% cases, whereas 20-30% cases are considered congenital in origin. Only 10-20% of CAE cases occur in association with inflammatory or connective tissue diseases such as scleroderma, Ehlers-Danlos syndrome, different types of antineutrophil cytoplasmic antibody-related vasculitis, syphilitic aortitis, or Kawasaki disease (7). Our patient did not have any evidence of these diseases. The reported risk factors of CAE are male gender, smoking, and hyperlipidemia, while the inversely associated risk factors are diabetes mellitus and age (3, 8). Our patient was male and had a history of dyslipidemia; however, he was relatively younger than other previously reported patients with coronary artery diseases.

CAE is caused by the initial endothelial damage that activates a series of inflammatory mediators (e.g., macrophages, metalloproteins, etc.) that cause degeneration of the medial layer of the vessel. These structural alterations, together with the action of nitric oxide and other vasodilators, cause dilation of the coronary artery, an extreme form of positive remodeling. Diabetes mellitus primarily affects the intimal but not the medial layer of the vessel, thus causing negative remodeling (8). For those reasons, nitroglycerin does not yield any therapeutic benefit, but instead induces exercise-induced myocardial ischemia (9).

All 3 coronary vessels can be affected by CAE; however, this is rare in cases of MCAE (5). The proximal and middle segments of the RCA are the most common sites for CAE, followed by the proximal left anterior descending and left circumflex arteries (10). CAE is classified based on the following criteria proposed by Markis et al. (11) and modified by Harikrishnan et al. (12): Type I, diffuse ectasia in 2 or 3 vessels; Type II, diffuse ectasia in 1 vessel and local ectasia in the other; Type III, diffuse ectasia in 1 vessel; and Type IV, local ectasia in 1, 2, or 3 vessels. According to the criteria proposed by Harikrishnan et al. (12), a patient with diffuse dilatation of multiple vessels would be classified as Type I; 5.8% of all CAE patients are classified as Type I. Moreover, Nyamu et al. reported only 1 diffuse MCAE patient with MI among 6,938 coronary angiographies (13).

Sudden death has been reported in patients with coronary ectasia, possibly due to a compromised coronary blood flow, thus leading to thrombosis and distal embolization (14). Some patients with coronary ectasia have shown a reduced left ventricular function due to disturbances in their blood flow, and therefore, vasospasm may occur in these patients (9, 14). Gunduz et al. reported two MI cases with diffuse MCAE who were treated with t-PA infusion (7). However, the final TIMI grade flow was not assessed in those patients.

In general, facilitated PCI with thrombolysis is not recommended for ST segment elevation MI, and primary PCI is considered to be superior to in-hospital initiated fibrinolysis, especially if the patient presents with symptomatology for >3 hours (15-18). In thrombolysis, regimens of intravenous thrombolytic agents were found to be as efficacious as intracoronary delivery (19). According to Mauro et al., intense antithrombotic treatment, including glycoprotein IIb/IIIa in-
hibitors and PCI deferral (not performing PCI immediately), were safe and effective for acute coronary syndrome patients who had a large intracoronary thrombus (20). However, glycoprotein IIb/IIIa inhibitors are not available in Japan, and the intracoronary thrombus volume in our patient was more than 30 times greater than that reported by Mauro et al.; therefore, we initially chose treatment with PCI. Since we could not obtain a TIMI grade flow 3 after PCI, we administered t-PA intravenously. A study reported that in intravenous thrombolysis, only 32.5% of patients obtained a TIMI grade flow 3 (21). Tanabe et al. recommended pulse spray thrombolysis for thrombotic occlusion in CAE cases. They presented two cases of MI with CAE in which pulse spray thrombolysis was effective for obtaining a TIMI grade flow 3 (22). Pulse spray thrombolysis was one of our options; however, this was not available at our hospital. Thrombus aspiration may increase the available surface area of the thrombi for systemic thrombolysis. Systemic thrombolysis with thrombus aspiration would be effective in these high thrombotic burden cases. We also estimated the patient’s bleeding risk via the HAS-BLED score because of the bleeding risk associated with t-PA (23). His score was 1, he had no history of bleeding, and he was relatively younger than other MI patients. Therefore, we decided to use t-PA with extreme caution. Since our patient had a TIMI grade flow of 1 after PCI, an intra-aortic balloon pump (IABP) may have been an option to improve his TIMI grade flow (24). We suspect it took some time to gain TIMI grade flow 3 with thrombolysis, therefore, his maximum creatine kinase level was high and the MRI showed delayed enhancement of the inferior wall. An IABP may therefore have been a better option to salvage the myocardium. We also considered the use of distal protection devices such as PercuSurge® (Medtronic, Minneapolis, USA) and Filtrapt® (Nipro, Osaka, Japan) because a previous study showed these devices were effective to gain TIMI flow 3 and to prevent distal embolization for MI patients (25). However, the responsible lesion in this case was the distal segment of RCA. Furthermore, a diffuse thrombus was detected with IVUS and therefore we could not place such devices. Oral anticoagulation therapy has been suggested to prevent thrombus formation in patients with CAE; however, no current data are available to support this (3). Because our patient had a silent anterior wall infarction and a history of MI with giant thrombi, anticoagulation therapy may be essential for MCAE patients. In conclusion, t-PA infusion may be an effective option for patients with massive thrombi in the giant coronary artery after PCI. The authors state that they have no Conflict of Interest (COI). References


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