Original Article

Clinical analysis of acute myocardial infarction caused by coronary embolism

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Background: This study aims to investigate the clinical and angiographic features in patients with acute myocardial infarction (AMI) induced by coronary artery embolism.

Methods: Clinical data of five patients with AMI induced by coronary artery embolism were analyzed retrospectively.

Results: One patient had left atrial myxoma, one patient had non-valvular atrial fibrillation, and three patients had rheumatic heart disease. Furthermore, one patient had mitral mechanical valve prostheses, two patients had atrial fibrillation. Coronary angiography showed the absence of significant atherosclerostic lesions in the coronary arteries except infarct related artery in five patients. Angiography suggested the presence of IRA occlusion caused by embolism.

Conclusions: The status for AMI due to coronary artery embolism usually expresses embolic material originating from the left heart chambers. Rheumatic heart disease and atrial fibrillation are the common reasons for coronary artery embolism. Coronary artery occlusion is the common performance in the results of primary coronary artery angiography. Sometimes it expresses visible signs of embolism.

Keywords: Coronary artery embolism; myocardial infarction; rheumatic heart disease; coronary artery disease; atrial fibrillation

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Introduction

Acute myocardial infarction (AMI) is very common in clinic, and is often caused by incomplete coronary occlusion or complete occlusion induced by acute plaque rupture (1). Coronary angiography plays an important role in the etiology diagnosis and treatment of AMI (2). In previous studies, 1–12% of patients with AMI who underwent coronary angiography did not develop an irregular lumen, or coronary artery stenosis of <50% (3,4). In this type of AMI, causes of infarct-related artery occlusion vary from person to person, such as increased platelet or coagulation activity (hypercoagulability) (5-7), coronary artery spasm (8), coronary artery embolism (9,10), as well as coronary myocardial bridge, coronary artery ectasia and coronary artery dissection (11). AMI caused by coronary artery embolism is rare in clinical practice. This study conducts an analysis of the clinical characteristics of these patients.

Methods

From 2005 to 2015, five patients with AMI caused by coronary artery embolism were admitted to our hospital, and underwent primary percutaneous coronary intervention

Table 1 The situation of primary disease

Case	Gender	Age (years)	Primary disease	Atrial fibrillation	History of chest pain	Anti-coagulation	Other embolism
1	Female	40	Left atrial myxoma	–	–	–	–
2	Male	65	Non-valvular atrial fibrillation	+	–	–	Brain
3	Male	55	Rheumatic mitral stenosis	–	–	–	–
4	Female	78	Rheumatic mitral stenosis	+	–	Hua Falin, 2 weeks ago INR 1.9, at ER INR 1.53	–
5	Female	73	Rheumatic heart disease, mitral valve replacement	+	–	Hua Falin, 2 months ago INR 2.1, at ER INR 1.23	–

INR, international normalized ratio; ER, emergency room. –, negative; +, positive.

(PPCI). Among them, two patients were male and three patients were female; and the average age of these patients was 62 years old (range, 40–78 years old). All five patients were definitely diagnosed with AMI in the Emergency Department according to clinical symptoms, electrocardiogram (ECG) and myocardial enzymology indexes; and underwent PPCI treatment. In this study, clinical features and imaging characteristics of coronary artery angiography were retrospectively analyzed.

Results

Primary diseases

Among the five patients with coronary artery embolism, one patient had left atrial myxoma, one patient had non-valvular atrial fibrillation and three patients had rheumatic heart disease; and among these three patients with rheumatic heart disease, two patients had mitral valve stenosis, and one patient had post-mitral valve replacement. Furthermore, among the three patients with rheumatic heart disease, two patients were combined with atrial fibrillation. Among the three patients with atrial fibrillation, two patients received anticoagulant drug warfarin, while the other patient did not take any anticoagulant drug. Among the two patients that received warfarin, one patient had an international normalized ratio (INR) of 2.1 in a clinical test 2 months prior, and did not undergo any test during the last 2 months, while the other one patient had an INR of 1.9 two weeks prior, and took antibiotics for upper respiratory tract infection a week ago. In the test taken in the Emergency Department, the INR was 1.23 (primary disease conditions are listed in *Table 1*).

AMI manifestations

In all five patients, initial symptoms were chest pain, which was sustained without remission. ECG performances all matched ST segment elevation myocardial infarction, in which two patients had anterior wall myocardial infarction and three patients had inferior and posterior wall myocardial infarction. Myocardial enzymological indexes were consistent with AMI characteristic changes. Coronary angiography results of all five patients revealed the following: except for the occluded blood vessels, the remaining blood vessels were normal, and there were no signs of atherosclerosis (*Figure 1*). After percutaneous transluminal coronary angioplasty (PTCA) and thrombus suction, infarct-related blood vessels were free of residual stenosis; thus, stent implantations were not required. Five patients were all improved and discharged (myocardial infarction conditions are listed in *Table 2*).

Echocardiogram

Four of five patients developed left atrium enhancement, and abnormal wall motions of the left atrium was found. Among them, one patient had post-mitral valve replacement, two patients had mitral stenosis, two patients had aortic valve insufficiency, and one patient had reduced left ventricular ejection fraction (LVEF) (echocardiogram performances are listed in *Table 3*).

Embolism in other parts of the body

One patient developed cerebral infarction in addition to AMI.

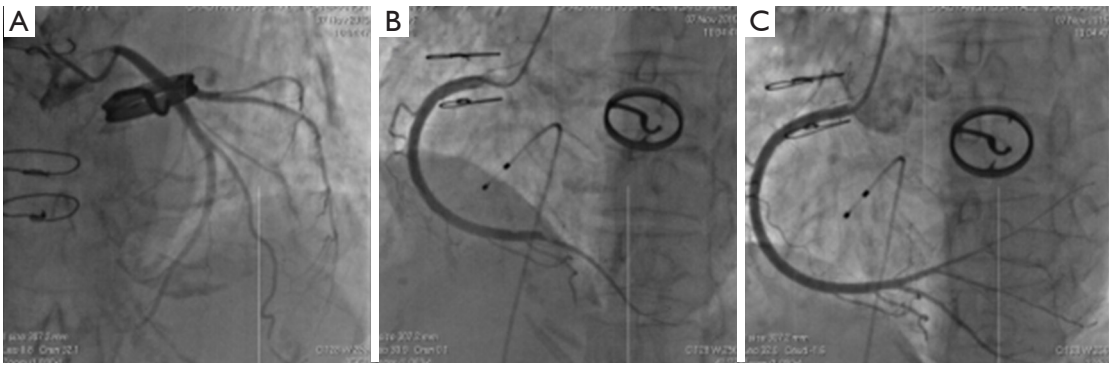

Figure 1 Observation of coronary angiography in coronary angiography. (A) Direct coronary angiography shows: LM, LAD and LCX were not found in atherosclerosis and stenosis; (B) direct coronary angiography shows: the posterior branch of the left ventricle (PLA) at the opening of the 100% block, the posterior descending branch (PDA) at the opening of thrombus; (C) angiography showed thrombus extraction after the PLA vascular wall is smooth, no residual stenosis. LAD, left anterior descending artery; LCX, left circumflex artery; LM, left main coronary artery.

Table 2 The myocardial infarction

Case	The incidence to myocardial infarction time (years)	Infarct position	The degree of segment resolution (mv)	cTnIpeak value	IRA	PCI	TIMI flow	CTFC
1	–	Anterior	0.2	23.06 μg/L	LAD	PTCA	3	23.6
2	4	Inferior and posterior	0.1	>16 ng/mL	LCX	PTCA + aspiration thrombectomy	3	20.8
3	40	Inferior and posterior	0.1	>16 ng/mL	RCA	PTCA + aspiration thrombectomy	3	20.1
4	30	Anterior	0.2	>16 ng/mL	LAD	PTCA + aspiration thrombectomy	3	24.1
5	20	Inferior and posterior	0.1	>16 ng/mL	RCA	PTCA + aspiration thrombectomy	3	22.5

IRA, infarction related artery; PCI, percutaneous transluminal coronary intervention; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction; CTFC, corrected TIMI frame count; PTCA, percutaneous transluminal coronary angioplasty.

Discussion

Coronary artery embolism is not a common cause of myocardial infarction (12). It has been confirmed by autopsy that the incidence of myocardial infarction caused by coronary artery embolism was 10–13% (13). Furthermore, the consequences of embolism are related to the size of the emboli and diameter of the embolized vessels (13,14). Coronary artery embolism events can easily occur in patients after cardiac valve replacement, chronic atrial fibrillation, dilated cardiomyopathy, infective endocarditis, intracardiac shunts, cardiac myxoma, mural thrombus and hypercoagulative state (15-18). Most emboli would fall to the left coronary artery system, due to the shape and flow characteristics of the aortic valve (19). For patients suspected to have emboli inside the coronary artery, esophageal ultrasound should be used to evaluate whether residual intracardiac emboli exist (20). To date, there is still no consensus in the treatment of coronary artery embolism. For early AMI with ST segment elevation, there are currently two available treatments, intravenous thrombolysis and percutaneous intervention. Some literatures have reported that intravenous thrombolysis could be chosen for the treatment of this kind of coronary

Table 3 Echocardiography results

Case	LAD (mm)	LAVI (mL/m^2)	The mitral valve area (cm^2)	SWMA	Other valvular diseases	LVEF (%)
1	33.5	32.2	Normal	+	–	69
2	53.4	52.3	Normal	+	–	61
3	55.0	53.1	0.97	+	Aortic valve insufficiency	59
4	39.5	33.9	1.30	+	Aortic valve insufficiency	64
5	78.5	76.4	Mechanical prosthetic valve	+	–	53

LAD, left atrium diameter; LAVI, left atrium volume index; LVEF, left ventricular ejection fraction. –, negative; +, positive.

artery embolism (21-24). Some studies suggest that a double dose of thrombolysis therapy is superior to a single dose (17), but this view remains controversial. If the emboli falling off are infectious vegetation, thrombolysis therapy is inappropriate (13). Hernández *et al.* reported treatments for three cases of AMI caused by coronary artery embolism; and all cases were successfully treated with PTCA and stent implantation (25). Treatment methods using suction to deal with emboli inside the coronary artery have been reported (5,17,26,27). There are also studies that reported that some surgeons applied a distal protection device to prevent the distal end of the blood vessel from being embolized, which can improve distal blood flow (26,27).

Among the five patients, one patient had left atrial myxoma, one patient had non-valvular atrial fibrillation and three patients had rheumatic heart disease; and among these three patients with rheumatic heart disease, one patient had post-mitral valve replacement and two patients were combined with atrial fibrillation. Patients with non-valvular atrial fibrillation did not receive anticoagulant drugs, while two patients with rheumatic heart disease combined with atrial fibrillation received warfarin anticoagulant therapy. However, one patient did not undergo detection for blood coagulation function in the last two months, and obtained an incompetent INR score during the detection in the Emergency Department; while another patient had an INR of 1.9 two weeks ago, received antibiotics for upper respiratory tract infection one week prior, and INR fell below the norm. All five patients were diagnosed with ST segment elevation myocardial infarction by clinical symptoms, ECG and myocardial enzymologic changes; and received PPCI. For the patient with left atrial myxoma, after balloon dilatation, the emboli fell to the ending of left anterior descending artery (LAD) branch due to the small diameter of the distal vessel; thus, no suction was conducted. The rest of the four patients underwent both PTCA and thrombus suction. After recovery of TIMI-3 blood flow, all infarct-related vessels were free of residual stenosis. All five patients were considered to develop coronary artery embolism.

Among the two patients with rheumatic heart disease, one patient had post-mitral valve replacement with atrial fibrillation, orally received warfarin, but INR was not monitored in accordance with the provisions; and the other rheumatic heart disease patient with mitral stenosis and atrial fibrillation orally took warfarin, and coagulation indicators were monitored. However, for this patient, the last INR test failed to meet the required standard, and antibiotics were administered, which may have affected the anticoagulation effect of warfarin. The cause for the occurrence of coronary embolism in these two patients was considered to be the inferior anticoagulation effect. In addition, another non-valvular atrial fibrillation patient did not take any anticoagulation drug, and this was confirmed to be the culprit of multiple organ embolisms. The rheumatic heart disease patient, though without atrial fibrillation, developed left atrial enlargement; and the enlargement of the left atrium was considered as a risk factor for thrombosis. For asymptomatic left atrial myxoma, it is difficult to prevent the occurrence of embolism events. Five patients with coronary artery embolism were treated with PPCI treatment; and in two patients, the emboli was drawn out by an aspiration catheter. In case of a huge embolus, this was pulled out via a guiding tube (*Figure 2*).

Through the analysis of the above five AMI patients caused by coronary artery embolism, for patients with myocardial infarction caused by coronary artery embolism, emergency percutaneous coronary intervention therapies (including thrombus aspiration, percutaneous coronary artery angioplasty and stent implantation) are very critical. For simple embolization of coronary patients without atherosclerosis, the emboli should be removed as far as

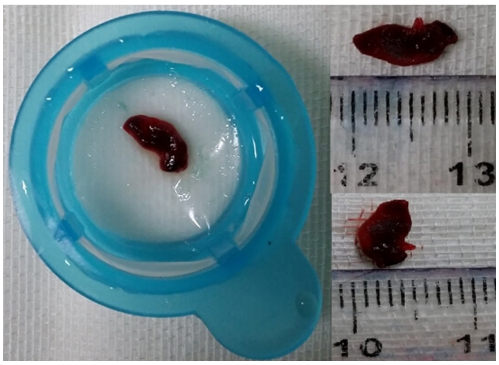

Figure 2 Through the guiding tube removed by coronary artery embolus.

possible through various instruments and means; but it should not try to open the vessel by stent implantation, since this would increase the dose of antiplatelet agents that patients require, increase post-operative bleeding risk, and increase the risk of coronary events. For patients with high risk of embolism, effective anticoagulant therapy and close monitoring of the anticoagulation effect are important means of preventing the recurrence of embolisms.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethical Statement: The study was approved by the Ethics Committee of Beijing Chao-Yang Hospital, Capital Medical University (approval ID: 2015-subject-80).

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