A Case of Acute Myocardial Infarction with Myocardial Damage Caused by Thrombi Originally Located in the Non-culprit Lesion

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Distal thromboembolism can develop in patients with acute myocardial infarction, but is rare in non-culprit lesions. Here, we report a case of acute myocardial infarction in which myocardial damage may have been caused by thrombi originally located in the non-culprit lesion. An 81-year-old woman presented with back pain and was diagnosed with ST-segment elevation acute myocardial infarction. Emergency coronary angiography demonstrated total occlusion in the mid-portion of the left anterior descending coronary artery and severe stenosis in the mid-portion of the right coronary artery. Collateral flow to the right coronary artery from the first septal perforator of the left anterior descending coronary artery, which branched before the total occlusion of the left anterior descending coronary artery, was observed. After recanalization of the left anterior descending coronary artery by thrombus aspiration, thrombi originally located in the right coronary artery migrated to the distal part. Myocardial imaging with thallium-201 and technetium-99m-pyrophosphate showed damaged but viable myocardium in the inferior wall of the mid-left ventricle.

KEY WORDS: collateral artery, myocardial infarction, non-culprit lesion

On examination, she was alert and oriented. Her vital signs were normal except for a blood pressure of 190/80 mmHg. Neither additional heart sounds nor murmurs were heard on auscultation, and the remaining examinations were normal. Electrocardiography demonstrated ST-segment elevations in leads V₁ to V₄. Anteroposterior chest radiography was normal. The complete blood cell counts were normal, as were the renal and liver function tests. Although the creatinine kinase level was 128 U/L, the heart-type fatty acid binding protein was positive and the high-sensitivity cardiac troponin T level was 0.181 ng/dL (reference value, ≤0.100). The level of brain natriuretic peptide was elevated to 303.3 pg/mL (reference value, ≤18.4). In addition, echocardiography demonstrated hypokinesis in the anterior wall and the apex of the left ventricle.

A diagnosis of ST-segment elevation acute myocardial infarction was made, and oral clopidogrel (300 mg), oral aspirin (200 mg), and intravenous heparin (10,000 units) were administered. Emergency coronary angiography demonstrated total occlusion in the mid-portion of the left anterior descending coronary artery and severe stenosis in the mid-portion of the right coronary artery (Fig. 1). Collateral flow to the right coronary artery from the first septal perforator of the left anterior descending coronary artery, which branched before the total occlusion of the left anterior descending coronary artery, was observed. After thrombus aspiration, recanalization of the left anterior descending coronary artery was obtained. Unexpectedly, thrombi migrating to the distal part of the right coronary artery was observed (Fig. 2, videos...
Fig. 1  Initial coronary angiography.
Severe stenosis is seen in the mid-portion of the right coronary artery (A, arrow). Note that the distal part of the right coronary artery is poorly visualized. Total occlusion is observed in the left anterior descending coronary artery (B, arrowhead). Collateral flow is observed from the first septal branch of the left anterior descending coronary to the posterior descending branch of the right coronary artery.

Fig. 2  Coronary angiography after thrombus aspiration.
The posterior descending branch of the right coronary artery is observed (A, white box) after successful recanalization of the left anterior descending coronary artery was obtained with thrombus aspiration therapy. Angiograms show thrombi migrating in the antegrade direction (B, C, and D, arrows). Panels B to D show enlarged images of the white box in Panel A. The images were obtained at an interval of 70 ms.
1 and 2). Soon, hypotension and bradycardia developed without inferior ST elevation, which were treated with intravenous atropine, intracoronary nicorandil, and fluid administration. After recovery from hemodynamic collapse, a stent was implanted because of the suboptimal dilation of the culprit lesion. No thrombus was shown on the final angiography of the left coronary artery, although an angiogram of the right coronary artery was not performed. As the patient’s symptoms subsided, intervention for the severe stenosis in the right coronary artery was deferred. The total procedure time was within 60 minutes.

Myocardial imaging with thallium-201 (111 MBq) and technetium-99m-pyrophosphate (\(^{99m}\text{Tc-PYP}\)) (740 MBq) was performed four days after admission. A total of 36 images over a 180-degree anterior arc were acquired five minutes and three hours after tracer injections using a digital gamma camera equipped with a low-energy, high-resolution parallel-hole collimator. The acquisition lasted 50 beats per projection, was stored in a matrix of 64x64 pixels, and the images were reconstructed using a Hanning filter without attenuation or scatter correction. Single-photon emission computed tomography demonstrated tracer defect of thallium-201 in the mid- to distal anteroseptal wall and interventricular septum as well as the apex, with little overlap with the uptake of \(^{99m}\text{Tc-PYP}\) (Fig. 3). Of note, \(^{99m}\text{Tc-PYP}\) uptake in the inferior wall of the mid-left ventricle overlapped with uptake of thallium-201, a finding consistent with myocardial damage but not infarction of the region.

The clinical course was uneventful and the peak creatine kinase level was 1,630 U/L, which was obtained on the day following admission. Later, stent implantation was successfully performed for the severe stenosis of the right coronary artery; neither plaque rupture nor residual thrombus in the right coronary artery was observed on intravascular ultrasound. The patient was discharged home in a stable condition.

### III. Discussion

Distal thromboembolism during emergency coronary intervention is not uncommon in patients with acute myocardial ischemia, because mechanisms of type-1 myocardial infarction, as in this case, include thrombus formation by definition\(^3\). It is speculated that the culprit lesion in this case was the left anterior descending coronary artery based on the electrocardiographic, echocardiographic, angiographic, and scintigraphic findings. The notable finding was that thrombi in the non-culprit lesion may have caused myocardial damage.

Myocardial scintigraphy with \(^{99m}\text{Tc-PYP}\) is sensitive for the detection of acute myocardial infarcts when performed during
the early phase of acute myocardial infarction\(^4,5\)). Myocardial scintigraphy with \(^{99m}\text{Tc}-\text{PYP}\) can be used to detect necrotic tissue\(^6\); however, it is worth noting that \(^{99m}\text{Tc}-\text{PYP}\) may accumulate in severely damaged but viable myocardium in patients with acute myocardial infarction\(^7\). Given the complete overlap between \(^{99m}\text{Tc}-\text{PYP}\) uptake and thallium-201 uptake in the inferior wall of the mid-left ventricle, it is speculated that the inferior wall in this case was damaged with little necrosis. The damaged area may have been perfused by the posterior descending branch of the right coronary artery, the flow of which originated in the first septal perforator of the left anterior descending coronary artery.

The findings of antegrade blood flow with thrombi in the posterior descending branch of the right coronary artery suggest that the thrombi, possibly along with transient hyperperfusion, caused the inferior myocardial damage. This speculation was supported by the hemodynamic collapse that developed immediately after the antegrade movement of the thrombi despite successful recanalization of the left anterior descending coronary artery. Cardiac receptors with vagal afferents involving the inferior wall of the left ventricle is known to cause bradycardia and hypotension (i.e., the Bezold-Jarisch reflex)\(^8,9\). The origin of the thrombi in the posterior descending branch could be via the anterior descending coronary artery: however, this hypothesis is less likely since no thrombus was detected in the catheter or in the proximal site of the left anterior descending coronary artery on angiograms. We cannot completely rule out the possibility that thrombi in the culprit lesion shifted to the proximal site by thrombus aspiration therapy and migrated to the right coronary artery through the collateral flow of the first septal perforator, but it also seems unlikely when the size of the collateral flow channels is considered.

We propose that the thrombi were originally located in the right coronary artery and migrated in an antegrade direction after successful recanalization. This hypothesis is supported by the severe stenosis in the middle portion of the right coronary artery. The distal part of the right coronary artery was not clearly visualized on the initial angiograms. Flow stagnation may also be associated with the retrograde flow through the collateral channel. It is reasonable to speculate that decreased perfusion pressure via the collateral flow after recanalization of the culprit lesion in the left anterior descending coronary artery caused the thrombi in the posterior descending branch to migrate forward. Angiography after recovery from hemodynamic collapse may have confirmed our theory, but no angiograms of the right coronary artery were obtained except for at the initial assessment.

In conclusion, this case demonstrates that myocardial damage may occur in non-culprit lesions even following successful treatment of culprit lesions of acute myocardial infarction.

Conflicts of interest

The authors state that they have no Conflict of Interest (COI).

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