Three Episodes of Inferior Acute Myocardial Infarction Due to Thrombotic Occlusion of the Right Coronary Artery: A Case Report

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Abstract

A 77-year-old female with two previous inferior myocardial infarctions was transferred to our medical center with a third inferior acute myocardial infarction. Coronary angiography revealed 99% stenosis with rich thrombus in the distal right coronary artery Thrombolysis in Myocardial Infarction (TIMI) grade 2 flow] The angiographic appearance of the right coronary artery was similar to the two previous myocardial infarctions. Coronary aspiration was performed and TIMI grade 3 flow was established. To confirm the presence of thrombus, intravascular ultrasound (IVUS) and coronary angioscopy were performed at pre-discharge. IVUS showed a thrombus-like low-density area at the mid right coronary artery. Red thrombi were observed in the same area using coronary angioscopy. Although warfarin had been prescribed for secondary prevention since the first acute myocardial infarction, both the second and third acute myocardial infarction occurred after cessation of warfarin. Patients with acute myocardial infarction due to thrombotic occlusion, confirmed by IVUS or angioscopy, might be good candidates for permanent warfarin therapy.

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Kev Words

■Myocardial infarction, treatment

■Intravascular ultrasound

■Angioscopy

INTRODUCTION

■Thrombosis

Coronary thrombosis is one of the causes of acute myocardial infarction. However, the diagnosis of coronary thrombosis is difficult to prove using only coronary angiography. The development of intravascular ultrasound (IVUS) and coronary angioscopy has dramatically improved the diagnosis of coronary thrombosis. We treated a patient who suffered three separate episodes of inferior acute myocardial infarction with similar findings on emergency coronary angiography. IVUS or

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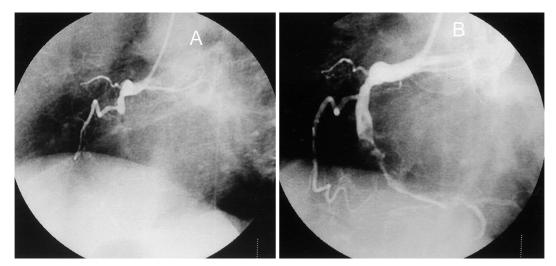


Fig. 1 Coronary angiograms at the first admission

- A: Emergent coronary angiogram demonstrated total occlusion of the mid right coronary artery.
- B: The distal flow reappeared after intracoronary thrombolysis using tissue plasminogen activator.

angioscopy could not be performed at the first two acute myocardial infarctions, but coronary thrombosis at the third acute myocardial infarction was demonstrated using both IVUS and angioscopy.

CASE REPORT

A 77-year-old woman with a history of two previous acute myocardial infarctions was transferred to our medical center for the treatment of inferior acute myocardial infarction on July 23, 2003. Her first episode of inferior acute myocardial infarction occurred in 1995. Emergency coronary angiography revealed total occlusion of the mid right coronary artery that was successfully treated by intracoronary thrombolysis using tissue plasminogen activator (Fig. 1). The right coronary artery was large and ectatic with rich thrombus. In consideration of these angiographic findings, warfarin as well as aspirin was prescribed for secondary prevention. The second episode of inferior acute myocardial infarction occurred in 1999, just after the discontinuation of warfarin on the recommendation of another physician. Emergency angiography was performed and the appearance of right coronary artery was similar to the previous acute myocardial infarction. The occluded vessel was recanalized by coronary balloon angioplasty(Fig. 2). She continued taking both warfarin and aspirin until July 23, 2003, when these drugs were discontinued because she was scheduled to have an operation for canalis carpi syndrome on July 30, 2003. Although the operation was successfully performed, she developed severe chest pain during lunch on July 31, 2003. She was sent to a nearby hospital, and transferred to our medical center.

On admission, her blood pressure was 110/70 mmHg with a regular pulse rate of 65 beats/min. Her height was 145 cm, and body weight was 72kg (body mass index: 34). There were no heart murmurs and lung rales were absent. Complete blood examination revealed an elevated white blood cell count $(10,130/\mu l)$. Levels of cardiac enzymes were normal. However, electrocardiography showed ST elevation in the , and a F levels, and ST depression in the , a L and 6 leads. Chest radiography demonstrated mild cardiomegaly (cardiothoracic ratio of 58%). Echocardiography revealed akinesis of the inferior wall that extended from the base to the apex. Emergency coronary angiography was performed and a 99% stenosis with rich thrombus was found in the distal right coronary artery Thrombolysis in Myocardial Infarction (TIMI) grade 2 flow] We attempted to aspirate the thrombus with the TVACTM system(NIPRO). TIMI grade 3 flow was established after aspiration and her chest pain disappeared despite the 90% residual stenosis (Fig. 3). We completed the acute intervention and she was transferred from the coronary care unit to a general hospital ward.

During rehabilitation, she experienced chest pain with light laborious work. As this chest pain was thought to be due to thrombotic stenosis, anticoagulation therapy using heparin and warfarin was con-

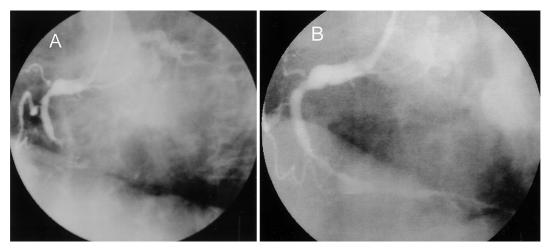


Fig. 2 Coronary angiograms at the second admission

- A: The appearance of the right coronary artery was similar to the previous episode of acute myocardial infarction (Fig. 1 A).
- B: The occluded vessel was recanalized by coronary balloon angioplasty.

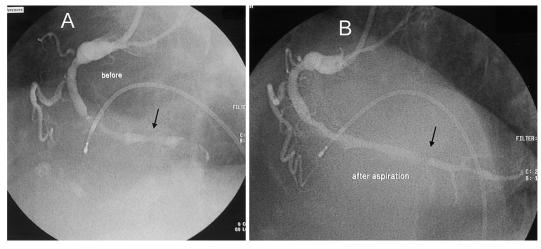


Fig. 3 Coronary angiograms at the third admission

A: A 99% stenosis(arrow)with rich thrombus was found in the distal right coronary artery [Thrombolysis in Myocardial Infarction(TIMI)grade 2 flow]

B: TIMI grade 3 flow was established after aspiration despite the 90% residual stenosis(arrow).

tinued for 1 week. Heparin was administered intravenously to achieve an activated partial thromboplastin time of 45 - 70 sec. Warfarin was administered orally to achieve an international normalized ratio (prothrombin time ratio) of 2 - 3. After 1 week of anticoagulation, her symptoms on exertion disappeared. Pre-discharge coronary angiography was performed on August 12, 2003. The 90% residual stenosis had improved to a 50% stenosis (Fig. 4).

To confirm the presence of thrombus, IVUS and coronary angioscopy were performed. IVUS showed a thrombus-like, low-density area at the

mid right coronary artery(Fig. 5). Red thrombi was observed in the same area using coronary angioscopy(Fig. 6). Although yellow plaques were observed at the mid right coronary artery, ruptured plaque was not detected. Both the IVUS and angioscopic findings confirmed that thrombus played a major role in the development of acute myocardial infarction.

DISCUSSION

The present patient had suffered three episodes of inferior acute myocardial infarction, all thought



Fig. 4 Pre-discharge coronary angiogram

The 90% residual stenosis had improved to a 50% stenosis(arrow).

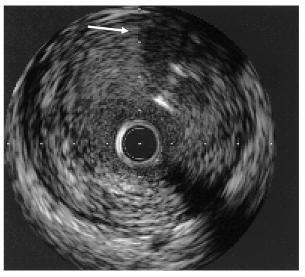


Fig. 5 Intravascular ultrasonogram
A thrombus-like, low-density area(arrow)was seen at the mid right coronary artery.

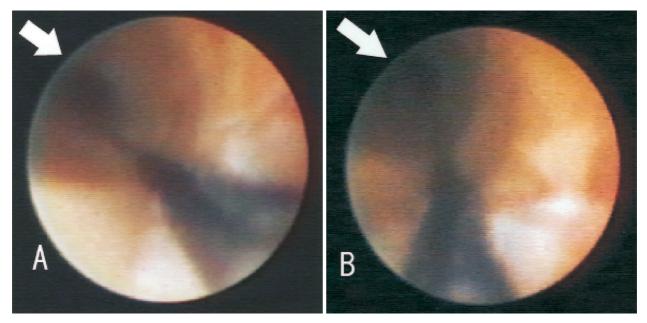


Fig. 6 Coronary angioscopy images

A, B: Red thromb(arrow) were observed in the mid right coronary artery.

to be due to thrombotic occlusion of the right coronary artery as suggested by coronary angiography. Her left coronary artery had been normal since the first acute myocardial infarction in 1995. We could not determine the exact reason why the thrombotic occlusion was confined to only the right coronary artery. Interestingly, thrombi from the right coronary artery contain more erythrocytes than from the left coronary artery¹. Her right coronary artery was

large and entirely ectatic in appearance, and showed no features of plaque rupture. Both the second and third acute myocardial infarctions developed just after discontinuation of warfarin. She had no history of atrial fibrillation. She also had no history of other hematologic disorder, thrombotic disorders such as deep venous thrombosis, or a bleeding tendency. These observations indicate that local thrombogenicity might have been involved in this

type of thrombotic occlusion.

Thrombotic occlusion was thought to be the cause of the previous two acute myocardial infarctions (1995, 1999) judged from the angiographic findings. However, after the third acute myocardial infarction, we demonstrated the presence of thrombus in her right coronary artery using IVUS and coronary angioscopy. IVUS usually shows thrombus as an intraluminal mass, often with a layered, lobulated, or pedunculated appearance²). IVUS is useful to identify coronary thrombus^{3,4}). In this case, we found an intraluminal, hypo-echoic mass indicative of thrombus. Although the diagnosis of thrombus by IVUS should always be considered presumptive, the IVUS findings were important because of the history of three inferior acute myocardial infarctions in this patient.

We were able to obtain a full-color, high-resolution, three-dimensional image of the thrombus using coronary angioscopy⁵. Coronary angioscopy is a superior modality to angiography to detect thrombus^{6,7}. We observed red thrombi at the mid right coronary artery. This finding strongly supports the assumption that her myocardial infarctions were due to thrombotic occlusions.

Warfarin, alone or in combination with aspirin, is superior to only aspirin in reducing the incidence of composite events after acute myocardial infarction, but is associated with a higher risk of bleeding⁸. Therefore, warfarin should be reserved for patients with a predisposing factor for left ventricular or left

atrial thrombus. Patients with acute myocardial infarction complicated by atrial fibrillation or left ventricular dysfunction are thought to be good candidates for warfarin therapy. Although the present patient did not have such predisposing factors, her right coronary occlusion was thought to be due to thrombotic occlusion. The reduction in the degree of stenosis without percutaneous coronary intervention also suggested that the stenotic lesion was a thrombotic stenosis. Furthermore, she suffered the second and third episodes of myocardial infarction after the withdrawal of warfarin. Patients with large ectatic coronary arteries and acute myocardial infarction due to thrombotic occlusion might be candidates for permanent warfarin therapy, especially if the presence of thrombus has been confirmed by IVUS or angioscopy.

Limitation

We were unable to perform a complete evaluation of hypercoagulability in our patient with recurrent acute myocardial infarction. Warfarin must be suspended for several weeks to collect exact data such as protein C, protein S, *etc*. Hereditary protein S deficiency was detected after suspending warfarin for 2 weeks⁹. Although we suggested that she substitute heparin for warfarin for several weeks, she refused because of the history of acute myocardial infarction occurring after the withdrawal of warfarin.

要

約

右冠動脈血栓性閉塞により3回の下壁急性心筋梗塞を起こした1例 坂倉 久保 典史 高木 洋介 片山 卓志 菅原 養厚 船山 大 池田奈保子 石田 岳史 安 隆則 鶴谷 善夫

過去に2度下壁心筋梗塞の既往のある77歳の女性が3回目の下壁心筋梗塞を発症して我々のセンターへ搬送された.冠動脈造影では右冠動脈遠位部に血栓を伴う99%狭窄が認められ,その造影所見は過去2回の心筋梗塞時と酷似していた.急性期は血栓吸引を施行し,Thrombolysis in Myocardial Infarction分類3度を得て終了した.血栓の存在を確かめるために慢性期に血管内エコー法と血管内視鏡を施行した.血管内エコー法では中部冠動脈に血栓様の低エコー領域が認められ,血管内視鏡では同部位に赤色血栓が認められた.この症例は最初の下壁心筋梗塞以後,二次予防のためにワルファリンによる治療が行われていたが,2回目および3回目の梗塞発症はワルファリン中止時に起こっている.この症例のような血栓閉塞型,とりわけ血管内エコー法や血管内視鏡で血栓の存在を確認された症例は,永続的なワルファリン治療のよい適応であることが示唆された.

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