Acute Myocardial Infarction Caused by Acute Ascending Aortic Dissection

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Acute myocardial infarction (AMI) secondary to acute ascending aortic dissection is a rare condition. The clinical presentations are similar but treatment strategies are different between AMI due to thrombotic occlusion of coronary arteries and that secondary to aortic dissection. In the latter, emergency surgery is the first choice and thrombolytic therapy is absolutely contraindicated. We report a 44-year-old man, who suddenly developed acute anterior chest pain. The diagnosis at emergency room was inferior wall AMI and the patient was treated with thrombolysis followed by coronary intervention. However, aortic dissection was suspected during cardiac catheterization and then comfirmed by echocardiography. The patient underwent emergent cardiac surgery to repair the aortic wall and bypass the proximal portion of right coronary artery (RCA). The follow-up coronary angiogram 3 months later showed patent RCA. From this case, we learn that in patients with an AMI, the possibility of aortic dissection should be kept in mind. If there is any suspicion, echocardiography can serve as a safe and quick tool to detect the possibility.

Key Words: Acute aortic dissection ● Acute myocardial infarction ● Echocardiography

INTRODUCTION

Acute myocardial infarction (AMI) is mainly caused by thrombotic occlusion of atherosclerotic coronary arteries. In such cases with ST-segment elevation in the electrocardiogram, thrombolytic therapy within 12 hours of the onset of chest pain is indicated. However, the manifestations of some other cardiovascular disorders, such as pericarditis, myocarditis, and aortic dissection mimic AMI. Administration of thrombolytic agents in such mimicking disorders is absolutely contraindicated. AMI secondary to aortic dissection is very rare, and, owing to the manifestations of myocardial in-

farction, diagnosis of the underlying aortic dissection may be delayed, which could potentially lead to a poor outcome. Surgical repair of the aortic wall with relief of compression to the coronary arteries is the treatment of choice for these patients, assuming it is undertaken quickly, and with adequate perioperative management to maintain stable hemodynamics. We report a case of AMI initially treated with a thrombolytic agent and later found to have aortic dissection.

CASE REPORT

A 44-year-old male without any known systemic disease in the past suddenly developed acute anterior chest pain, diaphoresis, and shortness of breath at 5:00 AM. He was taken to a local clinic, where inferior wall AMI with complete AV block was diagnosed. His blood pressure (BP) was 105/51 mmHg and the pulse rate was 46 beats/min. He was given dopamine infusion at 5 ug/kg/min for correction of bradycardia, and was transferred to our emergency room at 8:30 AM.

On arrival to emergency room, his BP was 110/48 mmHg, pulse rate 53 beats/min, respiratory rate 18/min and temperature 35 °C. No abnormalities were found on examination of the heart, lungs, or abdomen. The electrocardiogram (ECG) showed ST-segment elevation in leads II, III, and AVF, and complete AV block with a junctional escape rhythm at a rate of 50 beats/min (Figure 1). In addition, ST-segment elevation was found in lead V4R. The supine chest x-ray showed mild cardiomegaly with clear lung fields and a normal-looking mediastinum. Blood tests showed BUN 17 mg/dL, white blood cell count

$17600/mm^3$, hemoglobin 15.2 mg/dL, creatine kinase (CK) 134 u/L with MB fraction 4.5 u/L, and troponin-I < 0.10 ng/dL.

The patient suddenly developed ventricular fibrillation, which, after defibrillation at 200 joules, returned to the junctional rhythm within 30 seconds. Since at that time the onset of chest pain was less than 6 hours and the catheterization room was not available, a half-dose (50 mg) of recombinant tissue-type plasminogen activator (rt-PA) was given and facilitated percutaneous coronary intervention (PCI) was scheduled. The patient was sent
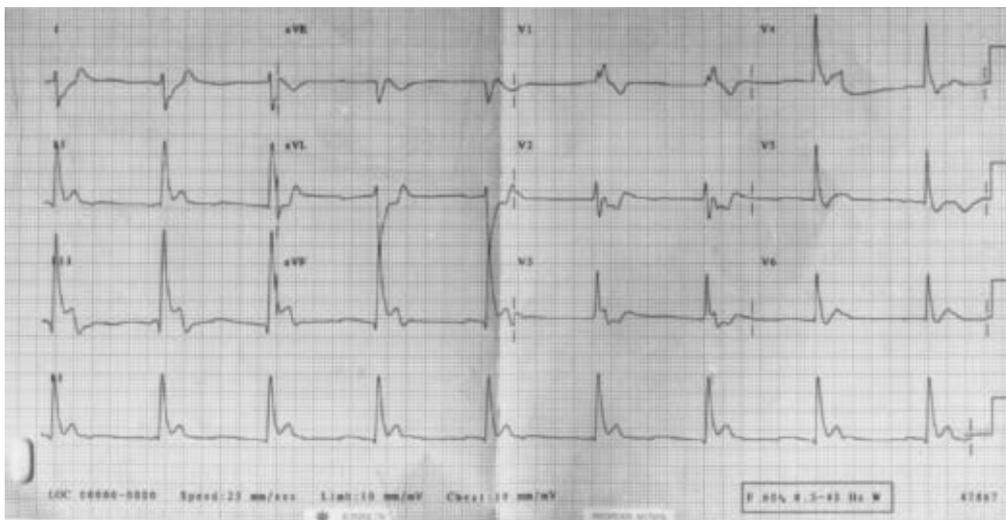
Figure 1. *ECG demonstrates 1- to 3-mm ST-segment elevations in leads II, III, and AVF and complete AV block with a junctional escape rhythm at 50 beats/min.*
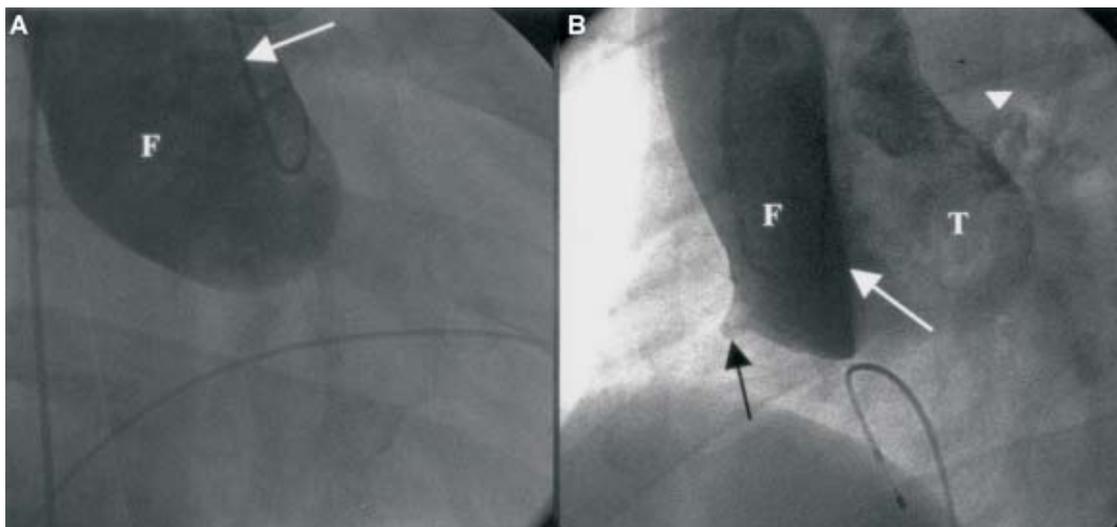
Figure 2. *(A) Aortogram in the right anterior oblique projection. The pigtail cathether (white arrow) was in the false lumen (F) with slow flow of the contrast medium. (B) Aortogram in the left anterior oblique projection. The true lumen (T) and false lumen (F) are separated by an intimal flap (white arrow). The left coronary artery (white arrow head) is seen, and the right coronary artery orifice (black arrow) is occluded by the false lumen.*

to catheterization room 120 minutes later. Prior to coronary intervention, the patient was hemodynamically unstable and required intravascular fluid, inotropic agents, temporary pacing and mechanical ventilation support. During catheterization, though the pressure waveform was normal at the ascending aorta, we were unable to engage the right and left coronary arteries in spite of various attempts, thus, aortography was done in order to delineate the coronary artery orifices. Before injection of the contrast medium, pressure tracing from the pigtail catheter showed a normal pulse wave, however, it turned out that the catheter was in the false lumen of a dissected aorta and the right coronary artery (RCA) was occluded at the orifice (Figure 2). An immediate transthoracic echocardiography (TTE) showed a hematoma inside the aortic wall and a linear flap involving the non-coronary and right coronary cusps of the aorta (Figure 3). In addition, aortic root dilatation with mild aortic regurgitation, left ventricular hypertrophy with inferior and posterior wall hypokinesis, and an enlarged hypokinetic right ventricle were found. Protamine was infused right away to neutralize the effect of heparin.

The patient was prepared for emergency surgery. Before he was moved to the operating room, he once developed cardiac arrest with ventricular fibrillation and cardiopulmonary resuscitation with advanced cardiac life support was given. During operation, the aortic root was transversely incised to identify the intimal flap and entry site. The entry site was about 2 cm above aortic si-

nuses, and a dissecting hematoma involving RCA orifice was found. The surgeon resected the damaged segment of ascending aorta and interposed a 30 mm Hemashield graft to reestablish aortic continuity. Also, the detached aortic valve commisure was suspended and a saphenous vein graft was placed from the aorta to the distal RCA after removal of a large amount of pericardial effusion.

The patient required inotropic agents postoperatively and was discharged uneventfully with nearly no neurological sequelae. Three months after the surgery, a repeat
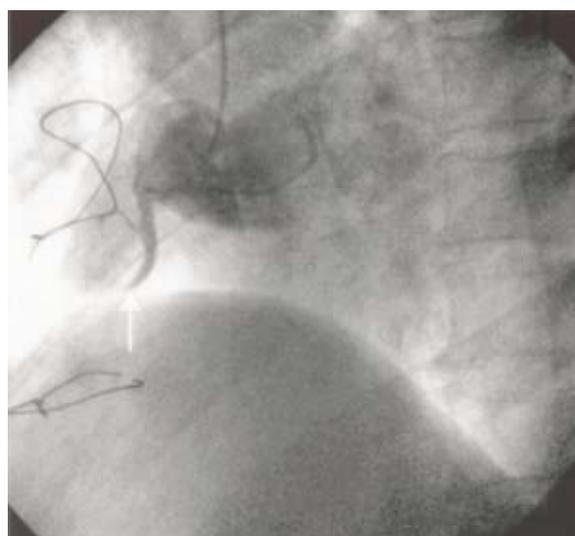
Figure 4. *Postoperative coronary angiogram in the left anterior oblique projection. Note the patent ostium and proximal portion of the RCA (white arrow), compared to (Figure 2B). Due to competing flow from the saphenous vein graft, the entire RCA cannot be fully occupied by contrast medium.*
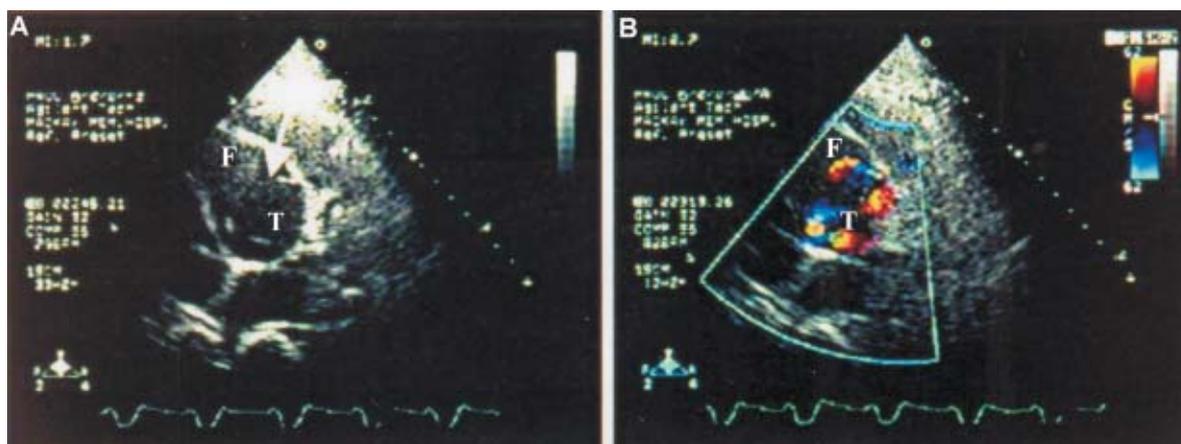
Figure 3. *(A) Transthoracic echocardiogram (TTE) of the proximal aorta showing the false lumen (F), true lumen (T), and intimal flap (White arrow). The false lumen has spontaneous echo contrast (smoke), suggesting a dissecting hematoma. (B) Color flow Doppler TTE of the dissecting ascending aorta. There is color flow in the true lumen but not in the false lumen.*

coronary angiogram revealed a normal left coronary artery (LCA), patent saphenous vein graft, and patent RCA (Figure 4). The left ventriculogram showed hypokinesis of the inferobasal and posteroinferior wall. The patient has remained free of symptoms.

DISCUSSION

AMI is diagnosed based on at least two of the following criteria: typical symptoms, serial EKG of ST-T change, and elevated cardiac enzymes. The patient fulfilled the criteria. Due to the absence of any known risk factor of aortic dissection and no sharp tearing chest pain along the course of aorta, it was reasonable to assume that the infarction was caused by thrombotic occlusion of an atherosclerotic coronary artery. We therefore decided to give a half-dose rt-PA infusion, followed immediately by facilitated PCI,[1] consistent with the guidelines (ACC/AHA 2004 STEMI guidelines).[2]

Aortic dissection occurs when blood flow separates the layers of the aortic media, usually through a tear in the intima.[3] A previous study reported that, regarding the anatomic location of the dissection, the ascending aorta at the level 1 or 2 cm above the aortic sinuses is involved in 60% of cases, the aortic arch in 10%, and the descending aorta in 30%.[4] Acute ascending aortic dissection associated with AMI is rare, with a reported incidence of 1 to 2%.[5,6] The mechanism by which dissection leads to ischemia is obstruction of the orifice of coronary arteries by a dissecting hematoma or intussusception of the circumferentially disrupted inner layers of the aortic wall,[3] and the RCA is more often affected than the left.[7] When an AMI complicates aortic dissection, the symptoms of the primary aortic dissection may be obscured by those of myocardial ischemia.

Either thrombolysis or PCI is currently recommended for an AMI caused by thrombotic occlusion of the coronary arteries when the patient presents within 12 hours of the onset of symptoms. It is clear, however, that thrombolysis is absolutely contraindicated in the presence of aortic dissection. Not only might it complicate surgical repair of the defect, it might also interfere with clotting of the blood within the false lumen, thus increasing the risk of further dissection.

In this case, we did not suspect aortic dissection un-

til the aortogram showed that the catheter was located in the false lumen, as has been previously reported.[5] One may question whether the AMI was primary and the dissection resulted from catheter injury or AMI was secondary to aortic dissection. It was reported that iatrogenic aortic dissections of which about 3/4 occur following cardiac surgery and 1/4 as a complication of cardiac catheterization,[8] accounted for about 5% of all cases of aortic dissection. Considering the location of dissection, those following cardiac surgery prefer the proximal portion of aorta, while those due to cardiac catheterization favor the distal portion.[8] In this patient, in addition to the involvement of proximal portion of aorta, the coronary angiogram obtained 3 months postoperatively revealed a patent RCA, indicating that aortic dissection was the primary event.

Acute ascending aortic dissection requires a prompt and accurate diagnosis, and surgical treatment is the first choice to save the patient. However, it is difficult to differentiate an AMI secondary to acute ascending aortic dissection from that by atherosclerotic thrombotic occlusion. Even after thrombolysis, the regression of ST-segment elevation in an AMI caused by aortic dissection has been reported.[9] Aortic dissection should of course be considered in patients with known risk factors for aortic dissection, such as hypertension, Marfan's syndrome, Ehlers-Danlos syndrome, arteritis, blunt chest trauma, cystic medial degeneration, aortic coarctation, pregnancy, bicuspid aortic valve, previous operations on the thoracic aorta, or cardiac surgery.[4,10] Signs and symptoms that are independent predictors of aortic dissection include: i) the very sudden onset of chest pain, particularly when described as tearing in character, or both; ii) a discrepancy in the pulse or BP in the two arms or upper and lower extremities; and iii) mediastinal or aortic widening on chest x-ray. It is reported that if all three are present, the probability of acute aortic dissection is 96%; if none are present, the probability is only 7%.[11]

When patients fulfill the criteria for AMI yet are suspected to have coexistent aortic dissection, prompt diagnostic studies such as echocardiography should be done before administration of thrombolytic therapy. A plain chest x-ray is inadequate to ruled out aortic dissection because up to 12% of patients with aortic dissection have normal chest radiography.[12] TTE, transesophageal echocardiography (TEE), computed tomography (CT),

magnetic resonance image (MRI), and aortography can be served as differential diagnostic tools.[13] CT, TTE, and TEE are readily available and can be performed quickly in emergency situations. MRI shows greater detail and is more sensitive and specific. It is best suited for serial examination of patients with chronic dissections.[12] Aortography is rarely available in an emergency unit and carries the risk of being invasive.[12] The diagnosis of aortic dissection by TEE is considered definitive when an intimal flap is identified associated with at least one of other features, such as the site of entry, slow-flowing blood or thrombus in the false lumen, or aortic root dilatation.[13]

In conclusion, AMI secondary to aortic dissection is a rare condition. Delay of the diagnosis may be disastrous. Therefore, the possibility of aortic dissection should be considered in patients with an AMI, particularly those with inferior and/or RV infarction, which may be caused by a dissecting hematoma. Careful attention should be paid to the risk factors of aortic dissection in the history, and the physical examination should include assessment of pulses and the BP in all four limbs. If aortic dissection is suspected, appropriate imaging studies should be arranged emergently. For this purpose, bedside TTE can serve as an easy, safe, and rapid procedure to diagnose aortic dissection with little delay to treatment.

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急性升主動脈剝離引發急性心肌梗塞

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急性升主動脈剝離造成急性心肌梗塞是非常少見的情況。急性心肌梗塞因冠狀動脈的血栓阻塞和次發於主動脈剝離二者的臨床表徵很相似但治療方式卻是不同。後者緊急手術是治療首選，而血栓溶解治療是絕對禁忌。我們報告一個 44 歲的男性突然發生急性前胸痛。經急診診斷為急性下壁心肌梗塞並接受血栓溶解治療及隨後冠狀動脈介入性治療。然而在心導管過程中懷疑有主動脈剝離而後被心臟超音波所確定。這病人緊急接受心臟手術來修復主動脈壁並對右冠狀動脈近端做繞道。在 3 個月後做冠狀動脈攝影追蹤，顯示通暢的右冠狀動脈。這個病例使我們學習到對急性心肌梗塞的病人，必須將主動脈剝離可能性謹記在心。假使有任何懷疑，心臟超音波可當做一個安全、迅速的工具來偵測這種可能性。

關鍵詞：急性主動脈剝離、急性心肌梗塞、心臟超音波。