INTERNAL MEDICINE

□ CASE REPORT □

# Aortic Dissection Associated with Acute Myocardial Infarction and Stroke Found at Autopsy

Hiroaki Kawano, Yoichi Tomichi, Satoki Fukae, Yuji Koide, Genji Toda and Katsusuke Yano

## Abstract

It is sometimes very difficult to diagnose dissecting aortic aneurysms in the early stage. We report an autopsy case in which an acute myocardial infarction and cerebral infarction simultaneously occurred and the symptoms were transiently ameliorated in a patient with an acute aortic dissection.

**Key words:** aortic dissection, myocardial infarction, cerebral infarction, reperfusion injury, sudden cardiac death

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## Introduction

The extension of aortic dissection into the brachiocephalic and common carotid arteries and the coronary arteries may lead to ischemic stroke and acute myocardial infarction. In some cases with aortic dissection, the false lumen is closed by thrombus just after the aortic dissection. However, there has so far been no report about acute myocardial infarction and cerebral infarction simultaneously occurring and transient amelioration of the symptoms in a patient with acute aortic dissection.

We herein present an autopsy case report of a patient with acute myocardial infarction and stroke and transient amelioration of the symptoms caused by aortic dissection.

## Case Report

A 49-year-old man presented with an acute onset of chest pain and left hemiparesis at 5 am in December 2003. He was transported to a local hospital. A chest X-ray showed only mild cardiomegaly, and an electrocardiogram (ECG) showed first degree atrioventricular (AV) block, wide QRS complex, and ST-segment elevation in the I, aVL, and V1 to V6 leads (Fig. 1). Chest computed tomography (CT) showed normal findings, and brain CT showed no abnormal findings such as hemorrhage, hematoma, and mass. Thus, the patient was diagnosed as acute myocardial infarction and cerebral infarction, and he was transferred to our hospital for further evaluation and treatment at 8 am. His past medical history included diabetes mellitus. His father had died of aortic aneurysm.

On admission, the physical examination revealed height of 172 cm, weight of 91 kg, a consciousness level of Glasgow coma scale (GCS) 10 [Japan coma scale (JCS) 20], blood pressure of 70/44 mmHg without difference between right and left upper extremities, regular pulse rate of 88 beats/min, and mild left hemiparesis. No significant murmur was heard, and lung was clear.

There was only mild cardiomegaly of chest X-ray on admission, which was same in the previous hospital. His ECG on admission showed left axis deviation, right bundle branch block, and poor R wave progression in V3-6, and ST-segment depression in the II, III, and aVF leads. The AV block and ST-segment elevation in the I, aVL, and V1 to V6 leads had disappeared (Fig. 2). An echocardiogram (UCG) demonstrated left ventricular ejection fraction (LVEF) of 62% with mild hypokinesis of the anteroseptal wall of the left ventricle (LV) and relative hyperkinesis of the posterior wall of LV. Neither valvular diseased nor pericardial effusion was seen.

The white blood cell count (WBC) was 18300 /mm$^3$. The blood urea nitrogen was 18 mg/dl, creatinine 1.6 mg/dl, aspartate aminotransferase (AST) 354 IU/l, alanine

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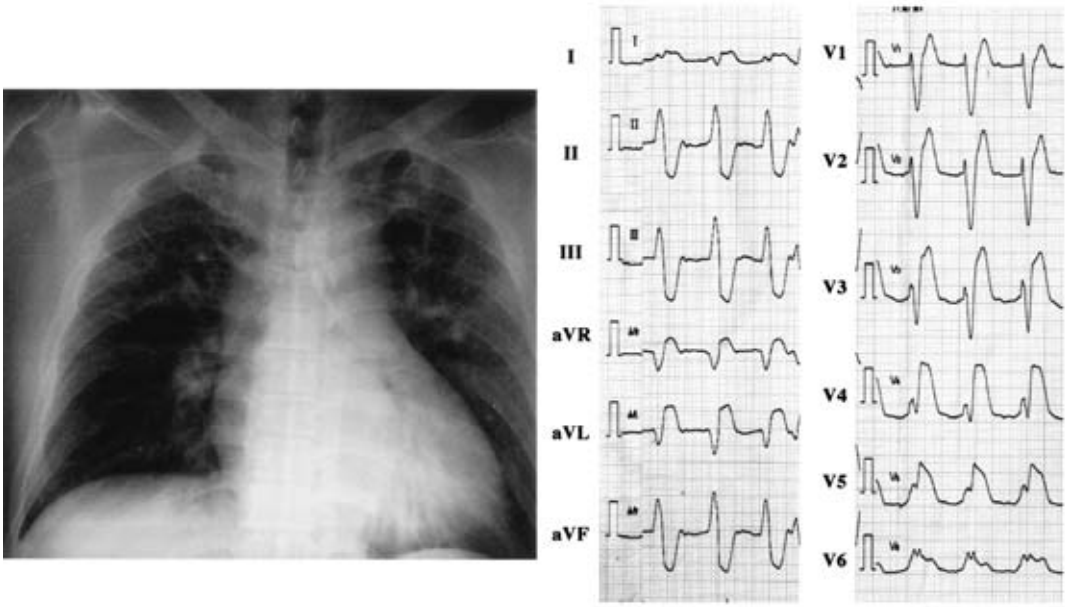


**Figure 1.** **A chest X-ray showed only mild cardiomegaly, and an electrocardiogram showed ST-segment elevation in I, aVL, and V1 to V6 leads in the local hospital.**

aminotransferase (ALT) 111 IU/l, lactate dehydrogenase (LDH) 748 IU/l, creatine kinase (CK) 3705 IU/l, CK-MB 306 IU/l, and C-reactive protein 0.03 mg/dl (Table 1).

An emergency coronary angiogram (CAG) was performed, and it showed no significant stenosis of the coronary arteries (Fig. 2C, D). Although the diameter of left main trunk was smaller than that of the left anterior descending or circumflex coronary artery, there was no finding that indicates aortic or coronary dissection, i.e., compression of coronary artery lumen at the systolic phase or false lumen of ascending aorta detected by contrast medium during CAG. Thus, we thought that the left coronary artery spontaneously recanalized after it was occluded by either spasm or thrombus. These cardiac examinations were quickly followed by brain magnetic resonance imaging (MRI) and brain angiography to identify the cause of left hemiparesis because an abnormal consciousness level and left hemiparesis were his main symptoms at that time. Brain MRI showed several scattered small infarct lesions in the left frontal and parietal lobes. Brain angiography demonstrated an occlusion of the small branches of the right middle cerebral artery although this lesion did not account for his symptoms. In addition, the carotid arteries had no stenotic lesions. We therefore thought that the right middle cerebral artery spontaneously recanalized after occlusion by the thrombus from hypokinetic LV due to a myocardial infarction. He was admitted to the neurosurgery ward, and edaravone, a free radical scavenger, was used for the treatment of cerebral infarction, while the intravenous infusion of heparin and nitroglycerin was done to prevent myocardial infarction. His vital sings including blood pressure and pulse rate were stable, and he gradually recovered and he was able to talk and the GCS improved to 15 (JCS 1). However, the patient suddenly developed dyspnea at 5 pm. An ECG showed Q waves in V1 to V5. An UCG demonstrated 17% of LVEF with akinesis in the anteroseptal wall and apex of LV without valvular disease nor pericardial effusion. A laboratory analysis was performed and showed elevations in WBC (17600 /mm$^3$), serum AST (1820 IU/l), LDH (4015 IU/l), CK (17915 IU/l), and CK-MB(1405 IU/l) (Table 1). The patient blood pressure had gradually decreased, and his cardiac index was 1.8 l/min, and the mean pulmonary artery pressure was 30 mmHg (Forrester IV). Although the intravenous infusion of catecholamine was started, his blood pressure did not increase. Therefore, percutaneous cardiopulmonary support (PCPS) was initiated to correct the systemic hypoperfusion. His hemodynamic state stabilized after PCPS was used. However, at 11 am on the next day, he suddenly suffered cardiac arrest. A temporary pacemaker did not enable the patient to recover from cardiac arrest. As a result, the patient died two days after the admission.

An autopsy was performed, and it revealed a Stanford type A aortic dissection. The point of entry was seen at the ascending aorta about 5 cm above the aortic valve (Fig. 3A) and the dissection extended from the aortic root to the abdominal aorta where the superior mesenteric artery branched. The bilateral carotid arteries were also involved by the arterial dissection. Both the right and left coronary arteries were also involved by the dissection, and their lumens were compressed by the hematoma formed in the false lumen (Fig. 3B). The dissection of the left coronary artery reached the bifurcation of the left anterior descending branch and circumflex branch (Fig. 3C). The dissection of the right coronary artery reached 2 cm from the ostium (Fig. 3D).

A microscopic analysis showed: 1) a hemorrhagic infarc-

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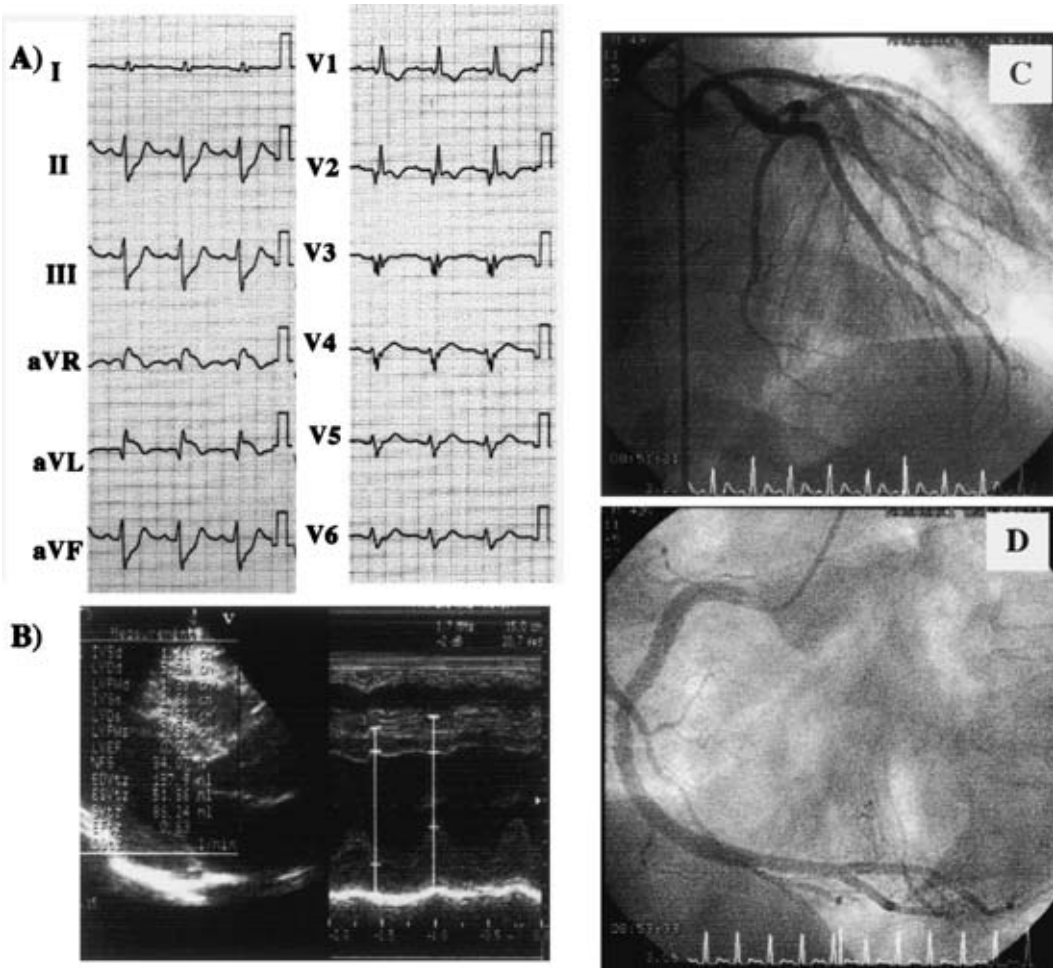


**Figure 2.** **An electrocardiography showed a left axis deviation, a right bundle branch block, poor R wave progression in V3-6, and an ST-segment depression in the II, III, and aVF leads (A). An echocardiogram demonstrated a left ventricular ejection fraction of 62% with mild hypokinesis of the anteroseptal wall of the left ventricle and relative hyperkinesis of the posterior wall of the left ventricle (B). An emergency coronary angiogram was performed, but the patient's coronary arteries were normal, and there was no ostial stenosis of the left (C) or right (D) coronary arteries.**

**Table 1.** **Laboratory Data**

| | On admission | | | One day later | |
|---|---|---|---|---|---|
| | 7:50 am | 12:00pm | 5:40pm | 0:00am | 6:30am |
| WBC (/mm$^3$) | 18300 | - | 19900 | 17600 | 11000 |
| Hb (g/dl) | 14.2 | - | 14.6 | 15.7 | 12.0 |
| T Bil (mg/dl) | 0.8 | - | - | - | 1.0 |
| AST (IU/l) | 354 | 847 | 1820 | 1234 | 1175 |
| ALT (IU/l) | 111 | 184 | 352 | 258 | 265 |
| LDH (IU/l) | 748 | 1540 | 4015 | 3080 | 3456 |
| CK (IU/l) | 3075 | 10090 | 17915 | 10920 | 9895 |
| CK-MB (IU/l) | 306 | - | 1405 | 740 | 595 |
| γ-GTP (IU/l) | 27 | - | - | - | - |
| CRP (mg/dl) | 0.03 | - | 2.54 | 4.56 | 9.43 |
| TP (g/dl) | 6.8 | - | - | - | 6.2 |
| Glu (mg/dl) | 287 | 223 | - | - | - |
| TC (mg/dl) | 111 | - | - | - | - |

WBC, white blood cell count; Hb, hemoglubin; T bil, total bilirubin, AST, aspartate aminotransferase; ALT, alanine aminotransferase; LDH, lactate dehydrogenase; CK, creatine kinase; γ-GTP, γ-glutamyltransferase; CRP, c-reactive protein , TP, total protein; Glu, glucose; TC, total cholesterol;BUN, blood urea nitrogen; Cr, creatinine

tion and contraction band necrosis were seen in the myocardium of the anterolateral wall of LV which indicated the occurrence of reperfusion injury (Fig. 4A); 2) cystic medial necrosis was seen in the media of aortic wall with mild atherosclerotic change (Fig. 4B). [This may suggest that he had Marfan's syndrome] and 3) in the false lumen of the aortic dissection, a thrombus had partially organized (Fig. 4C). However, only a fresh thrombus was seen in the false lumen of the coronary artery (Fig. 4D) and aortic root.

## Discussion

This is the first autopsied case report in which an acute myocardial infarction and cerebral infarction simultaneously occurred and the symptoms were transiently ameliorated in a patient with an acute aortic dissection. Hagan et al (1) reported the incidences of myocardial infarction and cerebrovascular accident to be 3.2% and 4.7% in acute aortic dissection, respectively. Therefore, the occurrence of aortic dissection simultaneously associated with both myocardial

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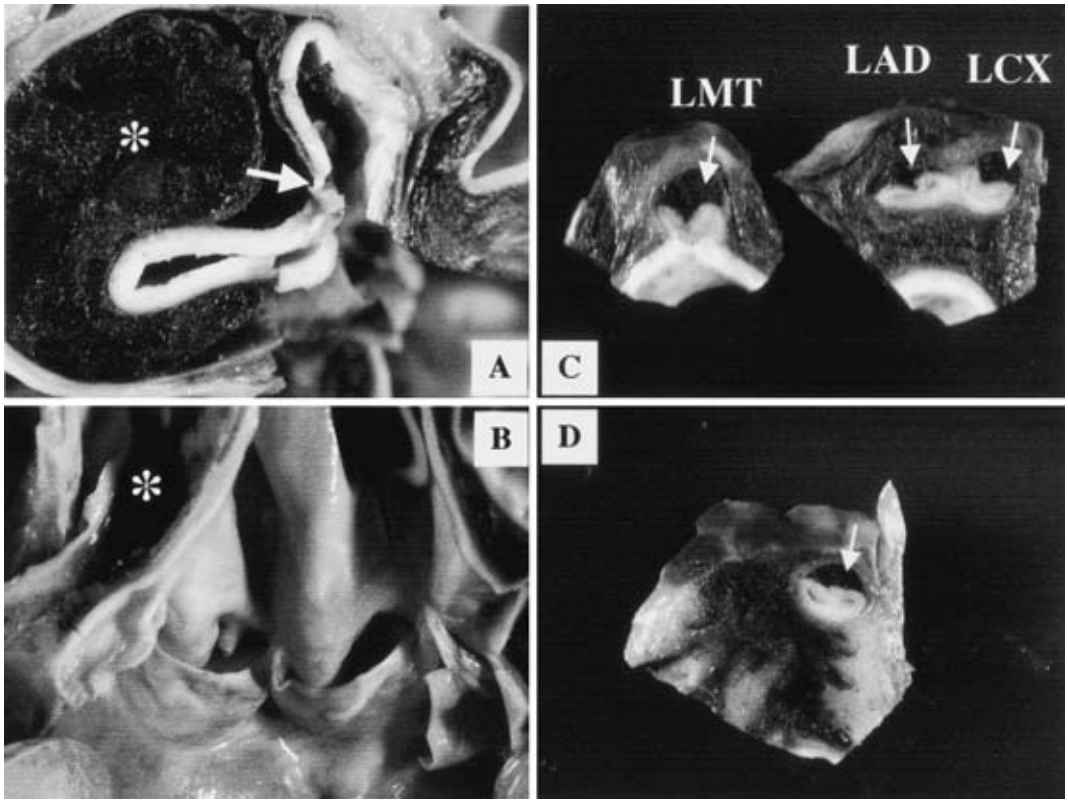


**Figure 3.** **Photographs of autopsy. (A) Autopsy revealed Stanford type A aortic dissection. The entry was seen at ascending aorta about 5 cm above aortic valve (arrow). The thrombus was seen in the false lumen (asterisk). (B) Aortic dissection reached the aortic root, and the thrombus and hematoma were seen in the false lumen of aortic root (asterisk). (C) The dissection of left coronary artery reached the bifurcation of left anterior descending branch and circumflex branch, and their lumens were compressed by the hematoma in the pseudolumen (arrows). (LMT; left main trunk, LAD; left anterior descending branch, LCX; left circumflex branch). (D) The dissection of right coronary artery reached 2 cm from the ostium.**

infarction and stroke seems rare.

Less common symptoms at the initial evaluation for aortic dissection, occurring with or without associated chest pain include congestive heart failure, syncope, cerebrovascular accident, cardiac arrest, sudden death, and so on (1). As a result, it is sometimes very difficult to diagnose dissecting aortic aneurysms (DAA), particularly in the early stages, due to the manifold signs and symptoms (2). The present case presented with chest pain and left hemiparesis, and we were not able to clinically diagnose the aortic dissection even after the examinations including coronary and brain angiographies. In the present case, a transient ST-segment elevation and a spontaneous amelioration of these symptoms, including chest pain and neurological abnormalities, also made it difficult to make an accurate diagnosis. We thought that the myocardial infarction had resulted from a nonatherosclerotic coronary disease such as a coronary embolism and spasm and that the coronary artery had then spontaneously recanalized because coronary angiography showed normal coronary arteries on admission. We also speculated that the cerebral infarction had been due to an occlusion of the cerebral artery by a thrombus from the akinetic LV wall, and that the cerebral artery had then spontaneously recanalized.

Although in most patients, neurological deficits from an aortic dissection are permanent (3-6), only transient symptoms of an aortic dissection have also been reported (7-9). In addition, only one case report of aortic dissection with a transient ST-segment elevation due to a left main coronary artery obstruction has been previously published (10).

The mechanisms of aortic branch obstruction have been discussed as follows: 1) a bulging of the dissected false lumen, which produces an occlusion at the branch orifice, 2) subsequent distal thrombosis, 3) eventual intimal detachment at the branch orifice with perfusion largely via the false channel, and 4) a dissection extending into the branch orifice (11). However, the precise mechanism of such transient ischemia has yet not been fully elucidated. Syed and Fiad (8) speculated that rather than thrombosis or shearing of the artery, a transient occlusive valve phenomenon attributable to the progression of the dissection may lead to transient ischemia. Ashida et al (10) mentioned that a decreased blood pressure and the presence of aortic regurgitation accelerated the collapse of the true lumen during diastole in the ascending aorta by the intimal flap, thus resulting in a functional obstruction of the left main coronary artery, which may have been related to transient ST-segment

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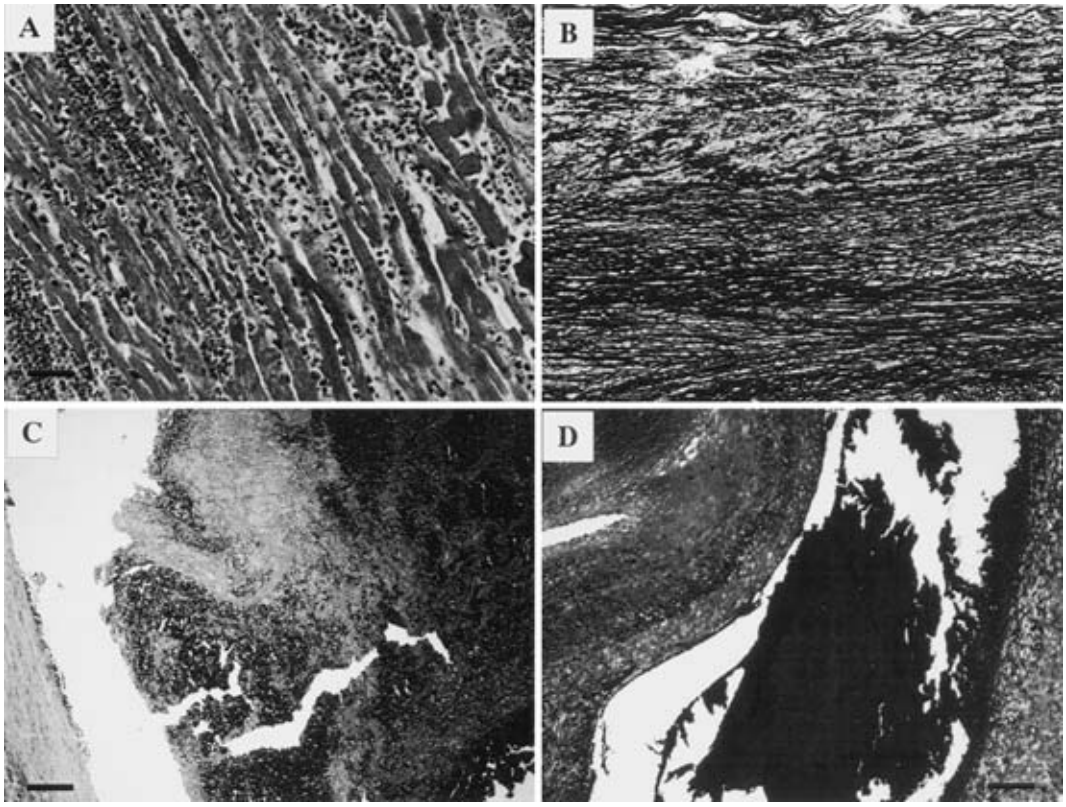


**Figure 4.** **Microphotographs of dissecting aorta. (A) A hemorrhagic infarction and contraction band necrosis were seen in the myocardium of the anterolateral wall of left ventricle (HE stain. Scale bar indicates 50 μm). (B) Mild cystic medial necrosis was seen in the aortic wall (Elastica Van Gieson stain. Scale bar indicates 125 μm). (C) In the false lumen of the aortic dissection, thrombus was partially organized (Azan stain. Scale bar indicates 250 μm). (D) Only fresh thrombus was seen in the false lumen of the coronary artery dissection (Azan stain. Scale bar indicates 250 μm).**

changes.

The mechanism of acute myocardial infarction in patients with acute aortic dissection is probably due to a compression of the extramural portion of the coronary artery by the false channel of the dissecting hematoma (12). An autopsy demonstrated a total obstruction of the bilateral coronary arteries due to compression by the false channel of the dissecting hematoma, which had caused the final sudden cardiac arrest in the present case. However, coronary angiography did not demonstrate coronary ostial stenosis either during systole or diastole when the ST-segment elevation had disappeared at the precordial leads in the ECG on admission. The autopsy also demonstrated that an organized thrombus had been seen in the false lumen of the ascending aorta although only fresh hematoma was seen in the false lumen of the coronary arteries and aortic root near the entry of aortic dissection. These findings suggested that, at first, the aortic dissection extending into the branch orifice had caused the left coronary artery obstruction, and then the thrombosed closing of the false lumen had released the obstruction of the left coronary artery, which may be related to the transient ECG change. And redissection of the ascending aorta near the coronary arteries caused the final sudden cardiac arrest in the present case.

The false lumen aortic arch and the brachiocephalic arteries also demonstrated an organized thrombus. Furthermore, brain angiography demonstrated the occlusion of the small branches of the right middle cerebral artery. Thus, cerebral infarction seems to be caused by both the occlusion of the brachiocephalic artery due to aortic dissection and distal thrombosis. As a result, the transient ischemia of the brain also seems to be related to the occlusion of the false lumen after the dissection.

An acute Stanford type A aortic dissection with coronary involvement is associated with a high mortality rate. Therefore aggressive coronary revascularization and early aortic repair are necessary to salvage these critically ill patients (13). When coronary angiography shows stenosis of the coronary arteries compressed by the false lumen of an acute aortic dissection, either stenting or surgery is performed to save the patient's life (14, 15). However, the left ventricular function of patients tends to remain depressed postoperatively, which limits their quality of life even when successful surgical treatments were performed in patients with a type A acute aortic dissection combined with myocardial infarction caused by a retrograde dissection into the left main trunk of the coronary artery (16, 17). The present case demonstrated the severe myocardal damage after the recanalization. Therefore, reperfusion injury, as well as ostial stenosis may be one of the most important factors to explain why

the left ventricular function of patients with acute myocardial infarction associated with aortic dissection remains depressed after successful coronary revascularization.

If aortic dissection is not considered in the differential diagnosis of patients with myocardial infarction and stroke, aortography, enhanced CT, or transesophageal echocardiography may not be performed, and angiography alone is unable to detect an aortic dissection. We therefore strongly recommended that an aortic dissection be included in the differential diagnosis in patients presenting with acute myocardial infarction and stroke even when the ECG changes and symptoms are transient.

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