

# Acute Myocardial Infarction and Concomitant Stroke as the Manifestations in a Patient with Type A Aortic Dissection: A Case Report with Three Years of Follow-Up

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A 55-year-old male patient presented with repeated acute retrosternal chest pain. Twelve-lead electrocardiogram and cardiac enzymes revealed non-ST elevation myocardial infarction. He was treated as non-ST elevation myocardial infarction at first. The symptoms of left-sided hemiparesis and aphasia occurred later on after admission. The results of emergent brain computed tomography and magnetic resonance imaging demonstrated acute stroke. The unusual presentation warned us of the possibility of aortic dissection. Besides the reports of heart and vessels computed tomography indicated aortic dissection as the underlying cause. Emergent surgical repair with preservation of the aortic valve led to a good recovery of heart and cerebral function. To the best of our knowledge, there were only three cases in the review of literature presenting with acute myocardial infarction and concurrent stroke resulting from acute aortic dissection.

**Key Words:** Acute myocardial infarction • Aortic dissection • Stroke

## INTRODUCTION

Compared to acute myocardial infarction (AMI), acute aortic dissection (AAD) is a relatively rare cause of acute chest pain.<sup>1</sup> It is also one of the most challenging medical emergencies. Especially when it presents with AMI and subsequent stroke, early accurate diagnosis and appropriate interventions are necessary for the survival. Differentiating AAD from AMI is a common and challenging dilemma because they can have the similar clinic presentation but contradictory management. In this report, we describe an AAD patient with a very unusual presen-

tation including both AMI and stroke. To the best of our knowledge, there has so far been only three cases reported about AAD complicated with AMI and stroke simultaneously.<sup>2-4</sup> Only one survived the devastating illness because of timely diagnosis and surgical intervention.

## CASE REPORT

A 55-year-old man had initially presented to the emergency room with sudden onset of retrosternal compressive chest pain relieved by sublingual nitroglycerin. He had a 20-year history of hypertension under good control and he had even received coronary angioplasty with two stents each deployed in the proximal left anterior descending and proximal left circumflex separately 6 years ago. The patient did not have any other previous medical history.

In the emergency room, his temperature was 37.3 °C, regular pulse rate 53 per minute, respiratory rate 22

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per minute, and blood pressure 124/76 mmHg. There was no significant blood pressure difference mentioned between the two arms, nor obvious heart murmur heard on cardiac auscultation. Twelve-lead electrocardiogram (ECG) showed normal sinus rhythm without any ischemic change. Chest radiography demonstrated mild cardiomegaly (Figure 1A). Serial cardiac enzymes surveys did not find any abnormalities. He was then discharged after a 12-hour observation.

Unfortunately he was sent to our emergency room again with recurrent chest tightness associated with diaphoresis one day later. Twelve-lead ECG revealed sinus tachycardia with anterior leads ST segment depression and premature ventricular beats (Figure 1B). The troponin I level was 38.77 (ng/ml). Dual anti-platelet agents and anticoagulant were prescribed immediately and then he was admitted to the intensive care unit under the impression of non-ST segment elevation AMI.

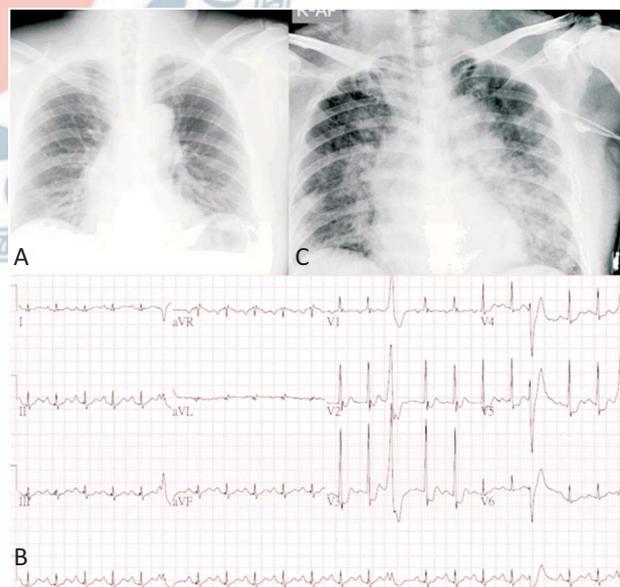
Soon after admission, left-sided hemiparesis and aphasia developed and got worse and worse. Emergent brain computed tomography (CT) and magnetic resonance imaging demonstrated acute infarction in the right frontal, parietal and occipital lobes. This unusual presentation reminded us of the possibility of underlying type A AAD. Transthoracic echocardiography (TTE) documented flap in the ascending aorta with eccentric moderate aortic regurgitation (AR). Mild hypokinesia over anterior and anteroseptal segments were also detected. Emergent heart and vessels CT results proved the diagnosis of type A AAD with involvement of the ascending and descending aorta. The dissection extended to the left main coronary artery, bilateral common carotid arteries and left subclavian artery (Figure 2A-C). Repeated chest radiography showed new appearance of the widening of the superior mediastinum (Figure 1C).

The patient was referred for cardiothoracic surgery immediately and underwent emergent surgical repair with preservation of the aortic valve. But the dissected left main coronary artery did not need bypass grafting. The patient survived the acute stage and did well with good recovery of the heart and cerebral functions. Three years later, follow-up heart and vessels CT demonstrated the results of repaired ascending aorta with preserved left main coronary and right proximal coronary flow (Figure 2D-F) but dilatation of the descending aorta with the maximum diameter of 4.6 cm.

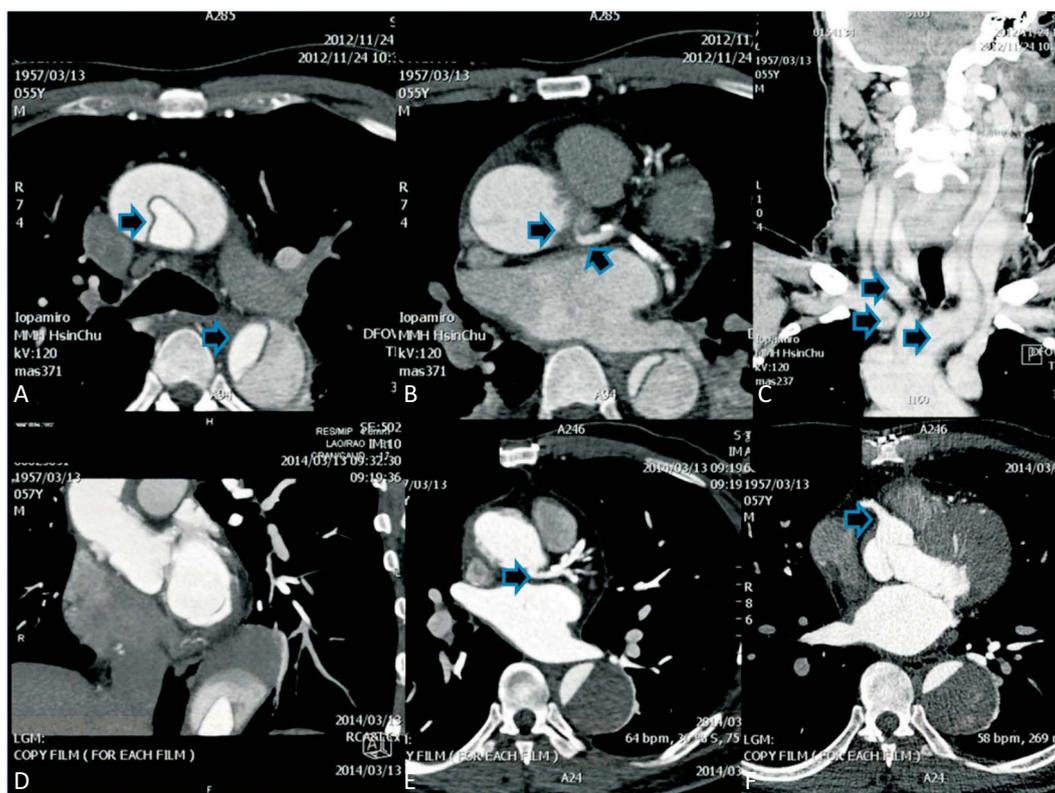
## DISCUSSION

AAD is still the most devastating aortic illness with high mortality and morbidity. If left untreated, the mortality can reach 33% and 50% within the first 24 hours and 48 hours respectively.<sup>5</sup> Since dissection can occur anywhere along the aorta, the clinical spectrum of presentation is variable. It depends on which organ is involved and the presentations can mimic other more common disorders such as AMI or stroke. Hypertension is the most important underlying risk factor and about 70% patients had it; less common risk factors include cystic media necrosis, connective tissue disease, bicuspid aortic valve and several kinds of arteritis.<sup>5-7</sup>

In the national registry of acute aortic dissection (IRAD), the type of pain and the location of pain in Stanford type A dissection are more often described as “sharp” sensation more than tearing or ripping, and their location is more likely at the anterior than posterior.<sup>1,6</sup> Other classic presentations, symptoms and signs of AAD were often less reported or absent. Take migratory pain, AR and pulse deficit for example, there were only 16%, 31.6% and 15.1% respectively.<sup>6</sup> Another study reviewing 239 patients with Stanford type A aortic dis-



**Figure 1.** (A) Plain chest film revealing mild cardiomegaly. (B) Twelve-lead electrocardiogram showing sinus tachycardia with anterior leads ST segment depression and premature ventricular beats. (C) Plain chest film 1 day later demonstrating widening of superior mediastinum and pulmonary edema.



**Figure 2.** (A) Heart and vessels computed tomography revealing dissection (arrows) involved the ascending and descending aorta. (B) Heart and vessels computed tomography demonstrating thrombi and dissection flap (arrows) impaired the left main coronary blood flow. (C) Heart and vessels computed tomography showing dissection (arrows) extended superior to bilateral common carotid arteries. (D) Three years later, follow-up heart and vessels computed tomography revealing well-repaired ascending aorta. (E) Three years later, follow-up heart and vessels computed tomography showing preserved the left main coronary artery (arrow) blood flow. (F) Three years later, follow-up heart and vessels computed tomography demonstrating preserved the right coronary artery (arrow) blood flow.

section also reported that 5% patients had initial presentation mimicking AMI and all were treated as AMI at first.<sup>1</sup> These evidences all showed how difficult it was to differentiate AMI from AAD when a patient came to clinical setting presenting with acute anterior chest pain.<sup>1,5,6,8</sup>

A review found that the ostium of a coronary artery may be involved with the consequence of possible coronary malperfusion or AMI in less than 7% of AAD cases.<sup>5</sup> In IRAD, stroke and AMI resulting from Stanford type A AAD were 6% and 4.7% respectively.<sup>6,9</sup> There was no mention about concurrence of stroke and AMI in patients with AAD.<sup>5,6</sup>

The mechanisms of AMI resulting from AAD can be explained as follows. Firstly, the flap interrupts blood flow to the coronary arteries.<sup>3</sup> Secondly the bulged thrombi in the false lumen resulting from dissection extending into coronary artery, which compressed the true coronary lumen.<sup>2</sup> The mechanisms for stroke include

brain tissue ischemia from hypotension, direct compromise of cerebral circulation and distal embolism.<sup>9</sup> Stroke itself is a predictive factor for more in-hospital complications such as hypotension, coma and malperfusion syndromes and higher in-hospital mortality as well.<sup>9</sup>

In the era of early reperfusion, the emphasis was put not only on AMI but also on stroke in order to achieve better outcome. Improper reperfusion therapy, including thrombolysis, for patient with AAD may produce adverse outcome.

Proximal aortic dissection is mostly treated with emergent surgical interventions and distal dissection is preferably offered with medical control of blood pressure.<sup>5-7</sup> There is no role for medical stabilization prior to surgery when treating proximal aortic dissection. Routine coronary angiography should be avoid because it delays surgery and increases the risk of aortic rupture.<sup>7</sup> There are still some “pioneers” who regarded percu-

taneous coronary intervention in some critical cases as a bridge before definite surgery.<sup>8</sup> In this case, left main coronary lesion is more suitable than the right coronary artery (RCA) lesion even though type A AAD usually involves the RCA.<sup>8</sup> The reason is the involvement of the left main trunk usually leads to unstable hemodynamic status.

Differentiating AAD from AMI is a common and challenging dilemma because they can have identical symptoms but the therapeutic strategies for each one are quite different. A rapid diagnosis, followed by appropriate treatment, is essential for better outcome. There was much effort done in establishing some biomarkers to help us distinguish these similar emergencies quickly.<sup>10</sup> Recently some studies had suggested D-dimer as a useful mark for discriminating between AAD and AMI.<sup>7,10</sup> The D-dimer concentrations in AAD patients were found to be significantly higher than those in AMI patients. A cutoff value of 5.0 µg/ml was effective in distinguishing AAD from AMI with a sensitivity of 68% and specificity of 90%.<sup>10</sup> It was thought that the burden of thrombi in AAD patients is much higher than it in AMI patients. Even though chest radiography showed an absence of mediastinal widening in 37.4% of patients with type A aortic dissection.<sup>6</sup> It is still wise and cost-effective to detect AAD by repeated chest radiography.<sup>3</sup> CT is considered the most widely used imaging modality in diagnosing AAD with near 100% sensitivity and specificity due to easy completion and less operator-dependence.<sup>1,6-8</sup> According to a review study, all patients with AAD complicated with AMI had ever undergone TTE examination; the appearance of AR was 100% but the presence of AR did not always caution the physicians about the possible presence of AAD.<sup>1</sup> TTE may be the most economic and feasible modality when a physician always keeps AAD on the list of differential diagnoses of acute chest pain. Transesophageal echocardiography had a better sensitivity and specificity than TTE but it is more operator-dependent.

Among patients surviving acute stage of aortic dissection, 15~30% required new surgeries for their progressive aortic dilatation and rupture, progressive AR and organ malperfusion.<sup>5,7</sup> The management of these surviving patients includes surveillance of the aortic di-

ameter and, when it reaches a critical point of 5.5~6 cm, a referral for prevention surgery.<sup>7</sup> Regardless of the aortic dimensions, symptomatic patients should be referred for a cardiac surgery.<sup>7</sup>

We strongly suggest that AAD should be taken more seriously into considerations of the differential diagnosis in patients presenting with acute chest pain and complicated with other organ malperfusion symptoms. A high-level suspicion is the most important key in identifying these patients. When a rare condition like this is suspected, the combination of chest X ray film, TTE report and D-dimer level would all be strongly suggested to the clinical practitioner for early detections and there's also CT results for the definite diagnosis as references.

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