

## Inferior myocardial infarction secondary to aortic dissection associated with bicuspid aortic valve

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### ABSTRACT

Aortic dissection (AD) is a life-threatening condition and may present with symptoms which mimic myocardial infarction, leading to misdiagnosis and inappropriate use of anticoagulant and thrombolytic therapy. A 40-year-old woman with no prior history presented in our emergency department with sudden chest pain. Electrocardiography (ECG) showed a ST-segment elevation in leads II, III and avF, suggesting an acute inferior myocardial infarction. The patient was given anticoagulation and antiplatelet treatment. Coronary angiography, transthoracic echocardiography and computed tomography were performed. The patient was diagnosed with DeBakey I aortic dissection extending from ascending aorta to iliac artery, and associated with bicuspid aortic valve. Surgical treatments with a replacement of the ascending aorta, aortic valve replacement and coronary artery bypass grafting were successfully performed. Early imaging examination, if possible, might assist the diagnosis and guide the management of this disease. The condition of myocardial infarction secondary to aortic dissection is discussed.

**Key words:** Aortic dissection, bicuspid aortic valve, inferior myocardial infarction, right coronary artery

### INTRODUCTION

Acute aortic dissection (AD) is one of the most catastrophic illnesses. Patients may also present with complications related to malperfusion such as stroke, myocardial infarction and limb ischemia,<sup>[1]</sup> leading to a misdiagnosis. Acute AD complicated with myocardial infarction is rare but a fatal condition. If such patients are improperly treated with anticoagulation therapy, the mortality rate has been reported to reach as high as

69-100%.<sup>[2]</sup> We here reported a rare case of acute AD due to bicuspid aortic valve (BAV) complicated myocardial infarction. The patient was initially diagnosed as inferior myocardial infarction in the emergency room based on symptoms and electrocardiography (ECG).

### CASE REPORT

A 40-year-old woman with no history of hypertension, diabetes mellitus, coronary heart disease, Kawasaki disease, smoking or addiction of alcohol was admitted to our emergency department due to an acute onset of severe retrosternal chest pain for 9 hours. Her blood pressure was 84/54 mmHg (right arm) and 78/48 mmHg (left arm), heart rate was 50 beats/min and physical examination revealed good consciousness, dyspnea with sweating, a mid-systolic ejection murmur over the right second intercostal space and pan-diastolic murmur over the left third intercostal

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space. electrocardiogram (EKG) showed sinus rhythm with ST segment elevation in leads II, III, and aVF and ST depression in leads I, aVL and V2-V6. ST segment has no change in the right precordial and posterior leads.

Her white blood cell count was  $23.79 \times 10^9/L$  ( $4-10 \times 10^9$ ), granulocyte proportion was 82.2% (50-70%), red blood cell count was 98 g/L, aspartate amino transferase was 121 u/L (0-37 u/L), alanine amino transferase was 30 u/L (0-40 u/L), urea was 8.44 mmol/L (1.84-7.14 u/L), serum creatinine was 65  $\mu\text{mol/L}$  (35-96  $\mu\text{mol/L}$ ), troponin-I was 8.4 ng/ml (0.0-0.15 ng/ml), D2 dimer was 220 ng/ml (0-400 ng/ml), creatine kinase was 1273 u/L (24-195 u/L), creatine kinase MB-isoform was 106 u/L (0-24u/L). She was diagnosed as acute inferior ST-segment elevation myocardial infarction [Figures 1a-c] and cardiogenic shock. Aspirin 300 mg and clopidogrel 300 mg were given orally for antiplatelet therapy. Alteplase 100 mg was administrated intravenously for thrombolysis. Dopamine and 500 mL of normal saline were promptly given for anti-shock treatment. Her chest pain partially subsided, and ST segment in interior lead return to baseline level 3 hours after treatment [Figure 1e]. However, the patient suffered with an intermittent complete right bundle branch block (CRBBB) [Figure 1d], and acute gastrointestinal hemorrhage. She was then treated with a proton pump inhibitor, octreotide, gastric mucous protection agents and adequate volume of intravenous solutions.

Coronary angiography (CAG) and percutaneous coronary intervention (PCI) were scheduled on the seventh day after acute myocardial infarction and gastrointestinal hemorrhage. CAG was performed via right radial artery.



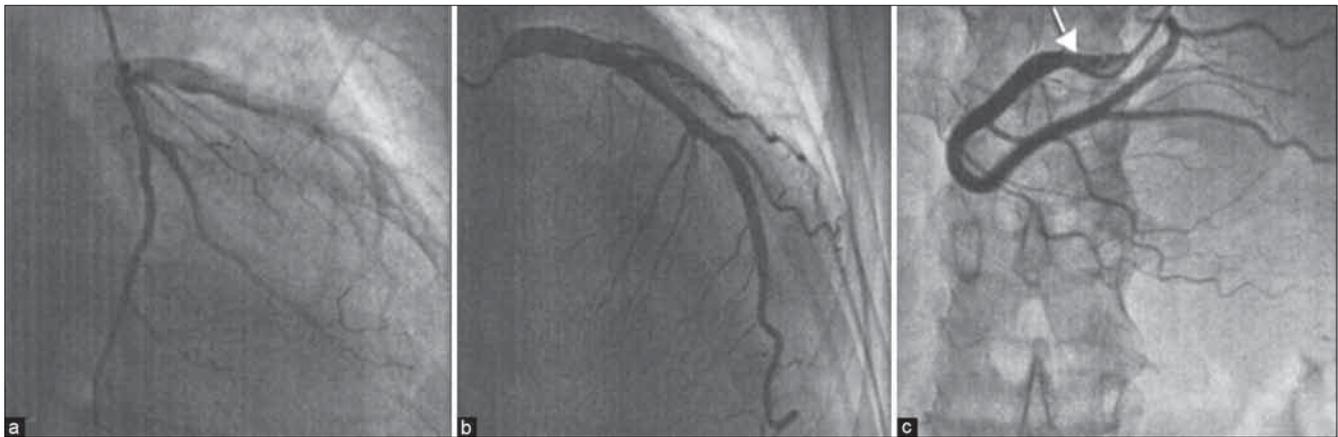
**Figure 1:** Representative electrocardiogram (ECG) of the patients. Panels a-c: A 18-lead ECG at initial admission shows a ST-segment elevation in leads II, III, and aVF. Panels d and e: ECG at 3 hours after admission shows the recovery of ST segment to baselines (d), and a complete right bundle branch block (e)

The proximal segment of the left coronary artery (LCA) was dilated while the mid and distal LCA were smooth [Figures 2a and b]. The catheter was engaged into true lumen of the right coronary artery (RCA), showing a mild narrowostium section [Figure 2c]. Additionally, aortogram showed an enlarged root of ascending aorta. However, a dissection flap was not remarkable. Transthoracic echocardiography (TTE) was performed to evaluate ventricular function after myocardial infarction. The TTE demonstrated a bicuspid aortic valve malformation with sever regurgitation (effective regurgitant orifice, ERO, area 12.36  $\text{cm}^2$ ), an enlargement of left ventricular (53 mm) and left atrium (37 mm), an increase of ascending aorta diameter (50 mm) and pulmonary artery (27 mm), and a dyskinesia of inferior wall. However, DeBakey I aortic dissection was not observed.

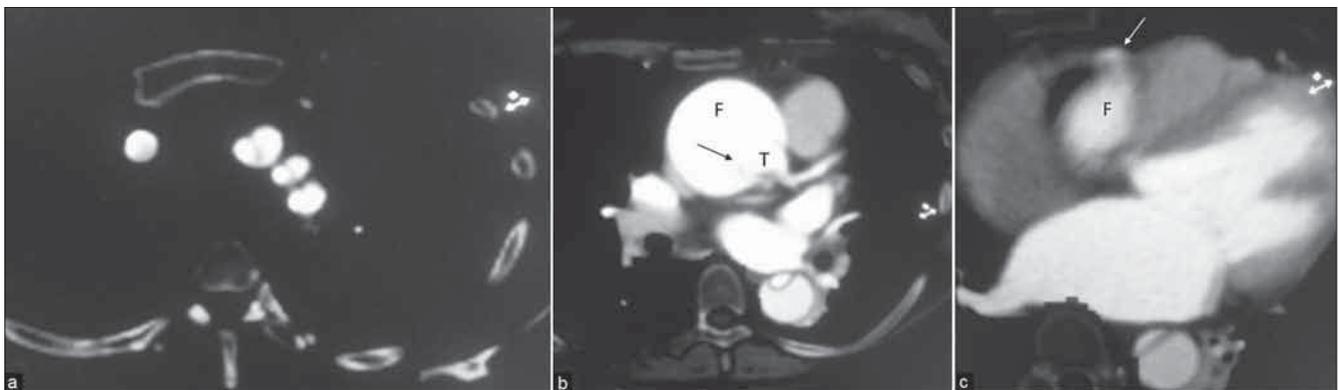
A 64-slice computed tomography throughout ascending aorta to iliac artery was performed, which showed a DeBakey I aortic dissection extending from ascending aorta to iliac artery [Figures 3a, 4a and b]. The dissection was crossed beyond the ostium of right coronary artery and the orifice was affected by the false lumen [Figures 3b and c]. The patient underwent an artificial vascular-valve graft to replace the damaged aortic arch, and a substitute mechanical aortic valve for the bicuspid aortic valve was placed. An endovascular stent was applied to cover the junction of remnant false lumen and artificial aortic arch. Bypass surgery was carried out with a fragment of saphenous vein sewn on the mid segment of right coronary artery to the subclavian artery. DeBakey I aortic dissection from ascending aorta involving the ostium of right coronary artery to iliac artery and a bicuspid aortic valve, were confirmed during thoracic surgery. The patient recovered well post-operation and had no major complaint during a three-month follow up.

## DISCUSSION

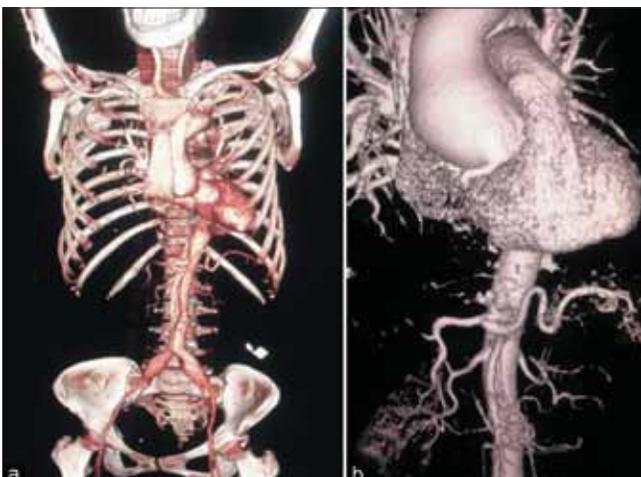
Aortic dissection complicated with myocardial infarction or functional coronary obstruction is rare and it is often misdiagnosed. The incidence of myocardial ischemia in aortic dissection ranges from 1% to 5%.<sup>[3-5]</sup> A study by Asouhidou *et al.*<sup>[6]</sup> has revealed that the initial misdiagnosis of aortic dissection as myocardial infarction may lead to inadequate use of thrombolytic, antiplatelet and anticoagulant agents. Although the use of thrombolytic



**Figure 2:** CAG demonstrates the dilated proximal segment of the left coronary artery (LCA) with a smooth mid and distal LCA (a and b). Panel c shows the mild narrow lumen at the ostium of the right coronary artery as indicated by an arrow



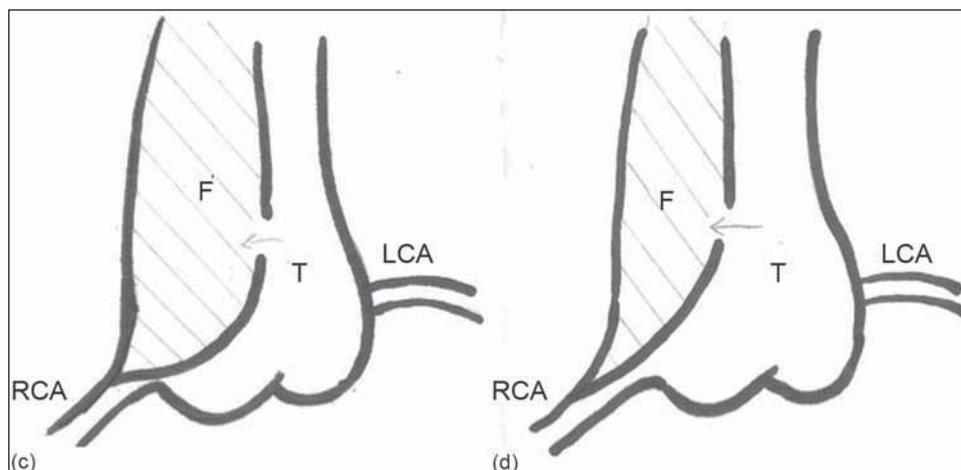
**Figure 3:** At the level of the brachiocephalic trunk, carotid and subclavian (a). At the level of the ostia of left coronary artery - LCA (b). The false lumen in the proximal segments of right coronary artery - RCA is shown(c). The black arrow indicates the intimal flap between the true lumen and false lumen. The white arrow indicates the false lumen involved in RCA; F, false lumen; T, true lumen; I, intimal flap



**Figure 4(a-b):** Panel a shows a reconstructed 3 dimensional imaging of the DeBakey I aortic dissection and aneurysmal dilatation extended from ascending aorta to iliac artery. Panel b shows the involvement of the false lumen in the ostia of right coronary artery

and anticoagulant agents might temporary improve the signs of myocardial infarction, the mortality rate climbs to over 70% from approximately 24% as administration

of thrombolytic drugs, mostly cause hemorrhage into the pericardial sac causing pericardial tamponade.<sup>[4,7]</sup> DeBakey I and II aortic dissection (type A) arise above the right sinus, and retrograde extension into the aortic root more likely affects the right coronary artery than the left. In the current case, the patient presented with a typical retrosternal chest pain, ST segment elevation in EKG and an increased serum troponin I level, all common signs of myocardial infarction, and was initially misdiagnosed as myocardial infarction and inappropriately administrated with thrombolytic and anticoagulant agents. Moreover, clinical manifestations of myocardial infarction such as chest pain and S'T segment elevation improved, and a complete right bundle branch block (CRBBB) occurred, most likely due to reperfusion following thrombolytic and anticoagulant treatment, which further complicated the diagnosis of AD. Fortunately, the thrombolytic and anticoagulant treatment was stopped due to gastrointestinal hemorrhage. The patient was examined with a series of imaging technology and treated successfully with surgery, which also confirmed the diagnosis of DeBakey I and II aortic dissection and the secondary



**Figure 4 (c-d):** A schematic drawing of aorta root (Panels c and d) shows the obstruction of right coronary artery (RCA) during the augmentation of the false lumen (c), and the recovery of blood flow from the orifice of RCA when the false lumen is collapsed (d)

myocardial infarction due to the bulging of the dissected lumen at the ostium of right coronary and causing occlusion [Figures 4 c and d].

As previously reported, the potential mechanisms of coronary arteries obstruction include: (1) bulging of the dissected false lumen, which often produces occlusion at the coronary orifice;<sup>[8]</sup> (2) subsequent distal thrombosis;<sup>[8]</sup> (3) eventual intimal detachment at the coronary orifice, with perfusion largely via the false channel;<sup>[8]</sup> (4) dissection extending into the coronary artery so that obstruction occurs beyond the coronary orifice;<sup>[9]</sup> (5) the coronary artery was not involved in dissection, but the flailing intimal flap obstructs the orifice during diastole;<sup>[10]</sup> and (6) extension of a proximal aortic dissection flap into the right coronary artery.<sup>[11]</sup> Referring to the data from the International Registry of Acute Aortic Dissection [IRAD], the most frequent risk factors of acute aortic dissection were hypertension (72%), and atherosclerosis (30%). Marfan syndrome, Loeys-Dietz aneurysm syndrome, vascular Ehlers-Danlos syndrome, bicuspid aortic valve, Turner syndrome, and familial TAA/D syndrome, are considered as congenital diseases associated with aortic aneurysm and/or dissection. Aortic dissection associated with atherosclerosis, trauma, cocaine abuse, arthritis and pregnancy has also been documented.<sup>[12]</sup> Following imaging examinations and surgery, the current case was diagnosed with congenital defect of bicuspid aortic valve (BAV).

BAV is the most common congenital heart abnormality, affecting up to 2% of the population. It is a risk factor for aortic dilation, aneurysm formation, aortic dissection, and rupture.<sup>[13]</sup> A hemodynamic flow disturbance in the aorta due to BAV is a major cause of aortic dilation and/or dissection [According to “the American Heart Association

Practice Guidelines on Bicuspid Aortic Valve with Dilated Ascending Aorta]. Patients with stenosis and regurgitation associated with tricuspid aortic valves are also at risk for aortic dissections.<sup>[14-16]</sup> In addition, weakness in connective media may also account for aortic dissection in patients with BAV, as histological analysis has demonstrated the presence of cystic medial necrosis in patients with aortic dissection due to BAV. Genetic defects in *Notch1* gene may be associated with BAV.<sup>[17,18]</sup> However, a direct linkage between *Notch1* gene mutation and congenital defect of BAV and connective tissue weakness in aorta has not been established.<sup>[14,19,20]</sup>

Both AD and acute myocardial infarction may have an onset with acute chest pain, and it is sometimes difficult to diagnose them by symptoms, EKG changes and/or cardiac biomarkers. In 2010, the American Heart Association (AHA) and American College of Cardiology (ACC) provided a guideline for the diagnosis of patients with thoracic aortic disease, in which an aortic dissection detection (ADD) risk score is included as an initial clinical tool for the detection of AD. Based on historical and examination features, patients could be divided to 3 groups: Low ADD risk if the score is 0, intermediate risk if the score is 1, and high-risk if the score is 2 and 3. This score system has a 95.7% of sensitivity in detecting AD, while its specificity has not been documented.<sup>[16]</sup> Immediate surgical consultation and an arrangement of Colour duplex ultrasound and aortic computed tomography angiography should be applied in patients with a high ADD risk score. The use of ADD risk score system for the initial bedside screening of patients could improve recognition and diagnosis of AD.<sup>[1]</sup> Furthermore, a recent study suggested that elevation of plasma D-dimer is helpful in the diagnosis

of AD.<sup>[21]</sup> The combination of ADD risk score and plasma D-dimer level should further facilitate the diagnosis of AD and improve the final outcome of AD patients.

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