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Clinical case report based study

A case report of acute myocardial infarction concomitant with Standford type B aortic dissection

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ABSTRACT

Background: Acute myocardial infarction (AMI) concomitant with aortic dissection (AD) is rare but a devastating situation if misdiagnosed as simply AMI, followed by anticoagulant or thrombolytic therapy. In such cases, Standford type B AD was extremely infrequent.
Objectives: To present a case with apparent concordance with the patient's history, symptoms, cardiac enzymes that lead to diagnostic error.
Case report: An 85-year-old man with chronic hypertension and coronary atherosclerotic heart disease presented in our emergency department with squeezing retrosternal chest pain and dyspnea. Elevated cardiac enzymes and electrocardiography result suggested acute non-ST-segment elevation myocardial infarction. Emergency coronary angiography demonstrated a 50–90% diffuse stenosis of the proximal and mid right coronary artery also confirmed the diagnosis. Stents were deployed thereafter. However, the patient was found to be concomitant with Standford type B AD by computed tomography angiography due to unrelieved chest pain and new onset of abdominal pain after the operation. The patient refused to have endovascular operation and died of hemorrhagic shock one week later.
Conclusions: AD may cause AMI due to some indirect mechanisms, and it is of utmost importance to search for the existence of AD before reperfusion therapy in AMI patients. Aortic dissection detection risk score, transthoracic echocardiography and D-dimer help early identification of AD.

1. Introduction

Acute myocardial infarction (AMI) and aortic dissection (AD) are both life-threatening illnesses that require early recognition and treatment in the emergency department. In particular, AMI complicated with acute AD is rare but a fatal condition. Once existed AD is neglected and thrombolysis or anticoagulant agent is given, it would be catastrophic for such patients and the mortality rate has been reported to reach as high as 69–100% due to further expansion, rupture and uncontrolled bleeding.[1,2] The Standford classification distinguishes AD between type A and type B which depends on whether the dissection involves the ascending aorta or not.[3] There are many reports about AMI concomitant with AD, but most of them are Standford type A AD. We here reported a rare case of AMI complicated with type B AD. The patient was initially diagnosed as AMI in the emergency room based on symptoms and cardiac enzymes.

2. Case report

An 85-year-old man was admitted to our emergency department with retrosternal squeezing chest pain and dyspnea that started 3 h before admission. He had chronic hypertension for 30 years and coronary atherosclerotic heart disease (CAHD) for 10 years. His medications included metoprolol 47.5 mg daily, clopidogrel 75 mg daily, and atorvastatin calcium 40 mg daily, but he did not take them regularly. On admission, blood pressure was 175/95 mmHg (right arm) and 180/97 mmHg (left arm), pulse rate was 125 beats/min but irregular. On physical examination, he had fine crackles at the basal portion of both lungs, auscultation of the heart revealed variable intensity of S1 and weakened S2 without murmurs at every auscultatory valve area or pericardial friction. A 12-lead electrocardiogram (ECG) was obtained and showed atrial fibrillation with rapid ventricular response and aberrant ventricular conduction (Fig. 1). His white blood cell count was 11.26×10^9/L (4–10×10^9/L), granulocyte proportion was 79.7% (46–75%), red blood cell count was 4.07×10^{12}/L (4.0–5.5×10^{12}/L), blood platelet count was 167×10^9/L (100–300×10^9/L), creatine kinase-MB was 12.53 ng/ml (0.10–4.94 ng/ml), myoglobin was 74.67 ng/ml (25.00–75.00 ng/ml), troponin T was 0.890 ng/ml (0.000–0.100 ng/

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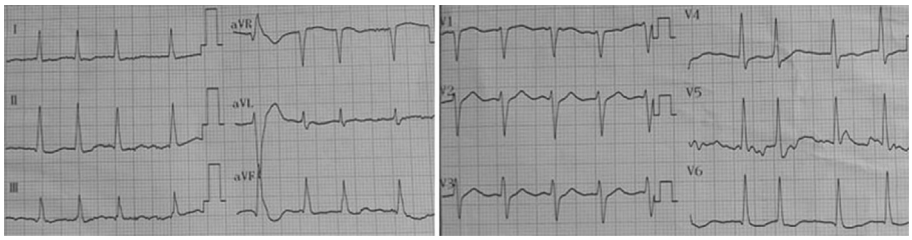

Fig. 1. A 12-lead ECG at initial admission shows atrial fibrillation with rapid ventricular response and aberrant ventricular conduction.

ml), Pro brain natriuretic peptide was 1585.0 pg/ml (0.0–84.0 pg/ml), potassium was 3.54 mmol/L (3.5–5.3 mmol/L), sodium was 137 mmol/L (135–145 mmol/L), urea was 6.6 mmol/L (2.9–8.6 mmol/L), creatine kinase was 112 umol/L (53–115 umol/L). He was diagnosed as acute non-ST-segment elevation myocardial infarction (NSTEMI) with atrial fibrillation. Aspirin 300 mg and clopidogrel 300 mg were given orally for antiplatelet therapy. An emergency coronary angiography (CAG) and percutaneous coronary intervention (PCI) were offered immediately. CAG was performed via the right radial artery. It demonstrated no significant stenosis of the left coronary artery (LCA), a 50–90% diffuse stenosis of the proximal and mid right coronary artery (RCA), as well as 50% local stenosis of the distal RCA (Fig. 2a and b). Two stents were deployed in the proximal and mid RCA respectively to remove the stenosis (Fig. 2c). However, chest pain did not improve and, it diffused to the abdomen two days after the operation. Then a computed tomography angiography (CTA) throughout ascending aorta to iliac artery was performed, which showed the presence of a Standford type B aortic dissection which extended from the descending aorta to the right internal iliac artery (Fig. 3). Both the patient and her relatives were informed of the necessity for endovascular operation, but they refused to give consent and asked for conservative therapy only. The patient died 7 days after the operation because of hemorrhagic shock.

3. Discussion

AMI concomitant with acute AD is an infrequent but devastating situation with a reported incidence of 1%–7%.[3–5] And it is often misdiagnosed as simply AMI without awareness of the existence of AD. Choi et al[6] retrospectively reviewed 78 patients with AD and found that the initial misdiagnosis as AMI was about 8%. The consequences of misdiagnosis may lead to improper antithrombotic or thrombolytic treatments, which bring the mortality rate to over 70% due to hemorrhagic complications.[7–9] Thus, early identification of their coexistence and avoidance of inappropriate treatment is of great importance. Migrating and interscapular pain, blood pressure and pulse difference in the two upper extremities or between upper and lower extremities, murmur of aortic regurgitation help early recognition of AD,[1,10] but in most cases, such classic symptoms and signs are often absent,[11] leading to misdiagnosis. In the current case, the patient had previous history of hypertension and CAHD, he was presented with a typical retrosternal squeezing pain and increased serum troponin T level, all common signs of myocardial infarction, and was initially misdiagnosed as NSTEMI and inappropriately administered with anticoagulant and antiplatelet agents. Severe stenosis of right coronary artery (RCA) also complicated the diagnosis of AD. Fortunately, type B AD was confirmed by CTA as diffuse abdominal pain after the operation.

Many case reports of a Standford type A AD in combination with AMI have been published.[1,12–16] It is undoubtedly that AD may cause AMI as the dissecting membrane may extend into a coronary ostium or the expanding false lumen may compress the proximal coronary.[17,18] Other mechanisms that include coronary spasm,[15] avulsion and ostium obstruction by intimal flap.[19] Our case was a NSTEMI concomitant with type B AD that is rarely seen in previous reports. And it can't be explained by the above direct mechanisms. Some indirect mechanisms may be operative that AD causes a significant stress response which makes a sudden activation of the sympathetic system and an excess release of catecholamines leading to arrhythmia, tachycardia and coronary spasm, and the patient who had been suffering from CAHD was more inclined to develop AMI under such conditions.

AD may mimic AMI due to similarities in clinical presentations and risk factors.[19] Patients with AMI and features suggestive for AD ought to be checked for the presence of AD, minimizing the risk of reperfusion therapy. However, based on the generally accepted concept that "time is myocardium", a rapid, safe and easy method is required to help rule out AD. In 2010, the American Heart Association (AHA) and American College of Cardiology (ACC) provided a guideline in which an aortic dissection detection (ADD) risk score system was used as an initial clinical tool for the detection of AD, they suggested that patients with a high risk score of 2 and 3 were highly suspicious of AD and should be applied for further diagnostic imaging studies. Among the most imaging tests, e.g. CTA,

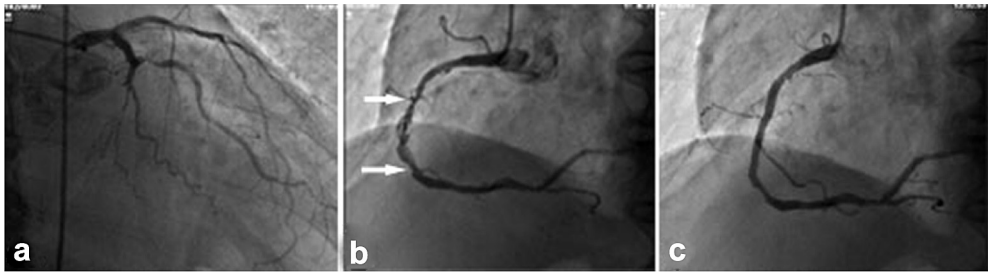

Fig. 2. CAG demonstrates no significant stenosis of the LCA, a 50–90% diffuse stenosis of the proximal and mid RCA, and 50% local stenosis of the distal RCA as indicated by the arrows (a and b). Panel c shows the disappeared stenosis of the proximal and mid RCA after stents deployment.

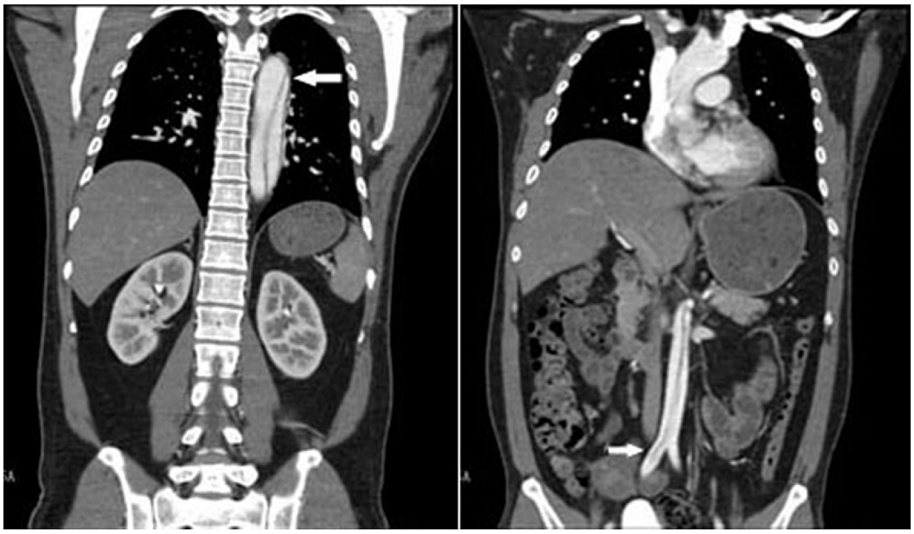

Fig. 3. CTA demonstrates the extended dissection from the descending aorta to the right internal iliac artery as indicated by the white arrows.

echocardiography, magnetic resonance imaging (MRI), and digital subtraction angiography (DSA). Transthoracic echocardiography (TTE) has been proved to be an easy, non-invasive, widely available and minimally time consuming technique to diagnose proximal AD, because it can provide much information about possible aortic dilation and insufficiency of the aortic valve, pericardial or pleural effusion and finally about a dissection flap, which is the hallmark for the diagnosis of AD.[1] Furthermore, a study by Sakamoto et al[20] has revealed that D-dimer is effective for discrimination between AD, pulmonary embolism (PE) and AMI. AMI is of low possibility with a D-dimer higher than 5.0 ug/ml. The combination of ADD risk score, TTE as well as D-dimer is helpful for the initial bedside screening of patients and further help for early recognition of the coexistence of AMI and AD.

4. Conclusion

Doctors should always bear in mind the possibility that AD causes or contributes to AMI in every AMI patients. The combination of ADD risk score, TTE and D-dimer is effective for early identification of the coexistence of AMI and AD.

Conflicts of interest

All authors have none to declare.

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