Aortic dissection presenting as acute myocardial infarction: potential harm of antithrombin and antiplatelet therapy

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In the treatment of acute myocardial infarction, antithrombin and antiplatelet therapy are indicated according to the current guidelines. When a patient presents with symptoms and signs of acute myocardial infarction, an extensive list of diagnoses should be considered. Because of the nonspecific symptoms of aortic dissection, the disease may be easily misdiagnosed. A high clinical suspicion of aortic dissection is therefore required. Once aortic dissection has been diagnosed, surgical intervention provides the only definitive treatment for these patients, regardless of antithrombin and antiplatelet therapy. (*Neth Heart J* 2006;14:147-9.)

Keywords: aorta, dissection, PCI, myocardial infarction (acute)

An initial presentation of aortic dissection with an acute myocardial infarction is not unusual.[1] In the treatment of acute myocardial infarction, antithrombin and antiplatelet therapy are indicated according to the current guidelines.[2,3] Early administration of these therapies in the ambulance or in the regional hospital has also been found to be beneficial for patients with acute ST-elevation myocardial infarction.[4] However, antithrombin and antiplatelet therapy may potentially be harmful in patients who suffer from aortic dissection. We report two acute aortic dissection patients who presented with an ST-elevation myocardial infarction and who were treated with combined antithrombin and antiplatelet therapy in the ambulance and during coronary angiography.

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Case reports

The first patient was a 59-year-old man who was admitted to our hospital after a collapse at home in the morning. After regaining consciousness, he complained of chest pain, loss of sense in his legs and general uneasiness. No symptoms of dyspnoea or palpitations were noticed Apart from hypertension, his past medical history was clinically unremarkable. On his way to the hospital by ambulance, acute myocardial infarction was diagnosed after clinical investigation and electrocardiography. Treatment was started with clopidogrel, aspirin and heparin during transportation to our centre.

On admission, the patient presented with a blood pressure of 97/46 mmHg and 74/49 mmHg on his left and right arm, respectively, with a regular pulse rate of 72 beats/min. He appeared sweaty and cyanotic on examination. Electrolytes, cardiac markers and pH were in the normal range; troponin was slightly elevated (0.4 µg/l; normal <0.16 µg/l). The ECG on admission showed a deep T-wave depression on the chest leads V_3 and V_4 and precordial leads I, II and III, ST-segment elevation on AVR, V_1 and V_2 and a sinus bradycardia of 54 beats/min. During coronary angiography, severe aortic regurgitation was noticed. A hazy appearance of the left anterior descending (LAD) coronary artery suggested an intracoronary thrombus. Transthoracic echocardiography revealed an intimal flap protruding into the ascending aorta, suggesting a proximal aortic dissection; this was confirmed by CT scan (figure 1). Three hours after initial presentation, the patient was successfully treated with an emergency supracoronary ascending aorta replacement, aortic valve resuspension and a venous graft from the aorta to the LAD to revascularise the myocardium.

The second patient was a 61-year-old man with a history of claudication. He complained of chest pain without radiation to the arm and excessive perspiration late in the evening. After 30 minutes, he lost the sense in his right upper leg. On his way to the hospital, the

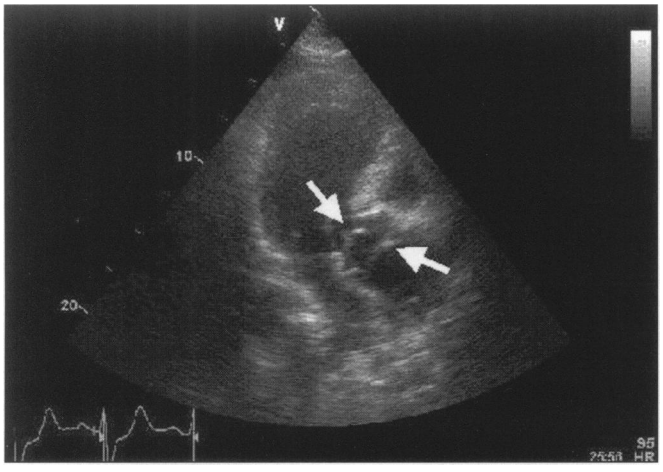

Figure 1. Aortic dissection in aorta ascendens (left arrow) and aorta descendens (right arrow). In aorta descendens three lumina were detected.

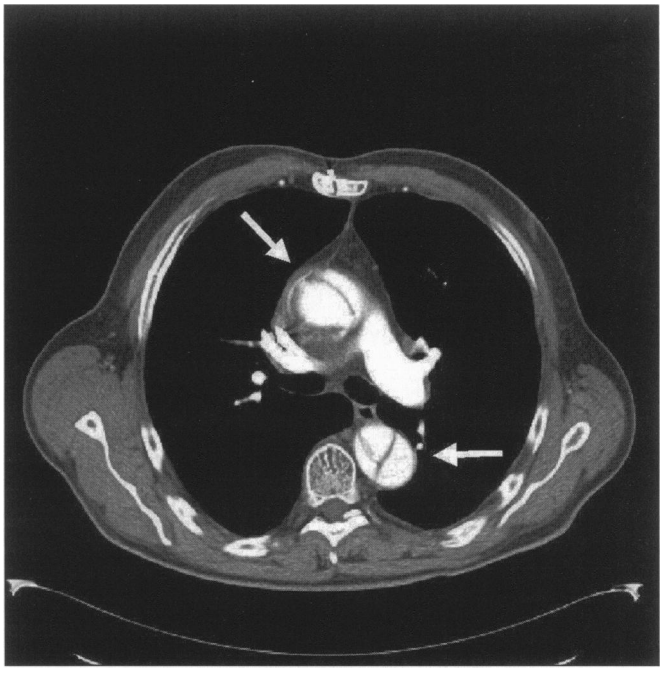

Figure 2. Transthoracic echocardiography revealed an intimal flap protruding into the ascending aorta, suggesting a proximal aortic dissection. Left arrow: aortic valve. Right arrow: intimal flap.

ECG showed a pattern indicating a large anterolateral myocardial infarction. The patient was treated with clopidogrel, aspirin and heparin in the ambulance.

On admission, he appeared grey and sweaty with a blood pressure of 82/30 mmHg and a pulse rate of 87 beats/min. Both legs were cyanotic on inspection and no peripheral pulse could be palpated in the legs. The admission ECG showed progressive anterior ST elevation on the I and AVL leads, confirming the diagnosis of a large anterolateral myocardial infarction. Aortic dissection was suspected, but no signs of dissection could be found during transthoracic echocardiography, which demonstrated a dilated left ventricle with hypokinesia of the anterior wall. During coronary angiography, symptoms and ST-segment elevation resolved and normal flow was found in the left and right coronary artery.

The next morning, the patient complained again of chest pain, dyspnoea, perspiration and pain in his right lower leg, which was white on examination. Increased and persistent ST elevation was again demonstrated by ECG. Repeated transthoracic echocardiography demonstrated backflow through an intimal flap just above the aortic valve, suggesting a type A aortic dissection which intermittently occluded the left coronary artery ostium (figure 2). An emergency supracoronary ascending aorta replacement was performed. However, the procedure was complicated by extensive blood loss. Postoperative left ventricular function was severely depressed due to the large anterior infarction and a stable haemodynamic condition could not be achieved. On the first postoperative day, the patient died on the thoracic intensive care unit.

Discussion

Patients presenting with symptoms and signs of acute myocardial infarction usually suffer from a ruptured coronary plaque and associated thrombosis.[5] Early initiation of antithrombin and antiplatelet therapy is therefore an appropriate strategy,[4,6] recommended by American as well as European guidelines, and facilitates reperfusion therapy by PCI.[2,3] However, in incidental patients the underlying pathophysiology leading to a presentation of myocardial infarction may be fundamentally different.

Firstly, noncardiac conditions such as acute pancreatitis, acute cholecystitis and rupture of the oesophagus or spleen may mimic a myocardial infarction. Secondly, cardiac conditions not affecting the coronary arteries, such as pericarditis and myocarditis, can present in an infarction-like manner. Finally, vascular lesions, such as pulmonary embolism and aortic dissection, can easily be confused with an infarction,[7,8] but can also cause an infarction.[10]

Aortic dissection is, in comparison with myocardial infarction, a rare disease with a frequently fatal outcome. While the pathogenesis of myocardial infarction usually includes a ruptured plaque, atherosclerosis does not appear to be a major risk factor predisposing to aortic dissection.[9] The disease can manifest in a number of different ways including anterior or inferior myocardial infarction.[10] Typically, patients report a sudden onset of sharp or tearing pain, frequently located in the chest or back. However, not all patients present in this manner and diagnosis of acute aortic dissection still remains unsuspected in a substantial number of patients who have indeed had myocardial infarction.[1,10]

Because of the rapid deterioration of the clinical condition in aortic dissection, an accurate diagnosis quickly made is of the utmost importance. Clinical suspicion is the most important step in diagnosing aortic dissection and must be followed by confirmation of the diagnosis. Of the imaging techniques available, echocardiography, CT scanning and MRI are useful diagnostic tests.[9,11] Transthoracic echocardiography (TTE) is a widely available test that can be performed quickly and relatively safely and may enhance the clinical suspicion of dissection, in particular in patients presenting with an acute myocardial infarction. On both transoesophageal and transthoracic echocardiography an intimal flap is a specific finding of a dissection. CT scanning can be regarded as the gold standard in diagnosing aortic dissection because it is a noninvasive test with high diagnostic accuracy allowing rapid diagnosis. MRI is a high-quality imaging technique and enables an accurate detection of aortic dissection. However, it has some limitations including the lack of immediate availability, the delay from bedside to scanner and the difficulties in the use of this test in critically ill patients.

Antithrombin and antiplatelet therapy is known to be beneficial to patients who have been diagnosed with acute myocardial infarction.[4,6] However, if aortic dissection presents as myocardial infarction, antithrombin and antiplatelet therapy will enhance the bleeding problem of aortic dissection. As surgical intervention remains the only definitive treatment option in patients with proximal aortic dissection,[12,13] patients pretreated with clopidogrel or a combination of clopidogrel and aspirin are facing increased risk of postoperative bleeding.[14,15] This contributed to the death of our second patient.

Conclusion

When a patient presents with symptoms and signs of acute myocardial infarction, an extensive list of diagnoses should be considered. Because of the nonspecific symptoms of aortic dissection, the disease may be easily misdiagnosed. A high clinical suspicion of aortic dissection is therefore required. Once aortic dissection has been diagnosed, surgical intervention provides the only definitive treatment for these patients, regardless of antithrombin and antiplatelet therapy. ■

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