



An extensive DeBakey type IIIb aortic dissection with massive right pleural effusion presenting as abdominal pain and acute anemia: particular case report

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Abstract

We describe the case of a 79-year-old male presented with sudden onset of abdominal pain and mild breathlessness, and complicated acute progressive anemia with haemoglobin which declined from 120 g/L to 70 g/L within five days. An urgent computed tomography angiography showed acute thoracic aortic dissection, DeBakey type IIIb, a dissecting aneurysm in the proximal descending thoracic aorta starting immediately after the origin of the left subclavian artery and extending distally below the renal arteries with evidence of rupture into the right pleural cavity for massive pleural effusion. Plasma D-dimer, brain natriuretic peptide and C reactive protein level were elevated. Our case showed that D-dimer can be used as a 'rule-out' test in patients with suspected aortic dissection. A raised BNP may exert a protective role through anti-inflammatory endothelial actions in the systemic circulation.

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1 Introduction

Acute aortic dissection (AoD) is a rare and lethal disease with presenting signs and symptoms that may often simulate other high risk conditions, even though using current modern diagnostic techniques, diagnosis is therefore often delayed or missed. Ruptured aortic aneurysm can be a cause of acute thoracic or abdominal pains, which is present in up to 90% of cases, and is typically severe at onset. We report a particular case of a misdiagnosed ruptured aortic aneurysm.

2 Case report

A 79-year old man with smoking habit who had a history of hypertension, dyslipidemia and thoracic aortic aneurysm presented to a hospital complaining of sudden onset of abdominal pain and breathlessness five days before admission.

His chest radiograph demonstrated significantly dilated descending aorta and clear lung fields (Figure 1) and blood tests showed normal full blood count and haemoglobin (Hb)

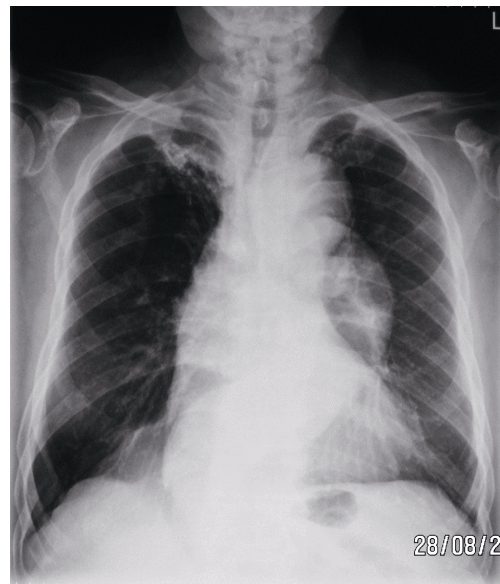


Figure 1. Chest radiograph demonstrated significantly dilated descending aorta and clear lung fields.

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120 g/L. Meperidine was administered, but the symptoms were persistent and worsening, he was transferred to our institution for further management. His blood pressure was 150/80 mmHg, and pulse rate was 106 beats/min. On physical examination, the heart and left lung were normal on examination, but the right hemithorax was dull to percussion with decreased breath sounds and wheezes located in the right mid-to-lower lung zones. The abdomen was soft and not distended, with normal bowel sounds. There was no ascites or pedal edema. Electrocardiography showed sinus tachycardia at 103 beats/min and did not show any ischemic changes. Echocardiography was performed immediately on arrival and revealed an enlarged left atrium, dilated aortic root of 44 mm and pulmonary artery pressure 34 mmHg and left ventricular akinesia, aortic valve calcification as well as regurgitation, mild mitral and tricuspid regurgitation. Blood test showed Hb 70 g/L. Serum levels of troponin I, creatine kinase and MB isoenzyme were: < 0.001 ng/mL (normal < 0.15 ng/mL), 217.8 U/L (normal < 171 U/L), and 18.2 U/L (normal < 25 U/L), respectively. NT-proBNP was 1641.130 pg/mL (normal < 250 pg/mL), D-dimer 840 µg/L (normal < 300 µg/L), and C reactive protein (CRP) was 70.44 mg/L (normal < 10 mg/L). Blood gas analysis showed PaCO₂ 37 mmHg, PaO₂ 78 mmHg.

Rapid and correct diagnosis and treatment are pivotal for patients with AoD. This patient complained of sudden onset of severe abdominal pain, mild dyspnea with a dilated aorta complicated by high NT-proBNP and D-dimer levels, as well as acute progressive anemia indicating that the dissection might involve both the ascending and abdominal aorta, and bleeding into the thoracic or abdominal cavity, which made a progressive anemia.

An urgent computed tomography angiography (CTA) was performed to rule out ruptured aortic dissection. The CTA showed a dissecting aneurysm in the proximal descending thoracic aorta starting immediately after the origin of the left subclavian artery and extending distally below the renal arteries with evidence of rupture into the right pleural cavity (Figure 2). The DeBakey system classified AoD as type IIIb, if dissection originates in the descending aorta and extending below the diaphragm.^[1]

A new European interdisciplinary expert consensus proposed that medical management with close imaging follow-up is the best strategy for uncomplicated type acute, sub-acute and chronic B aortic dissections. Currently, there is no evidence of an advantage with thoracic endovascular repair or open surgery.^[2] The patient declined consideration of the surgery and was being treated with continuous intravenous isosorbide dinitrate infusion (Isoket), orally administered a calcium channel blocker (Amlodipine 5 mg/d), an

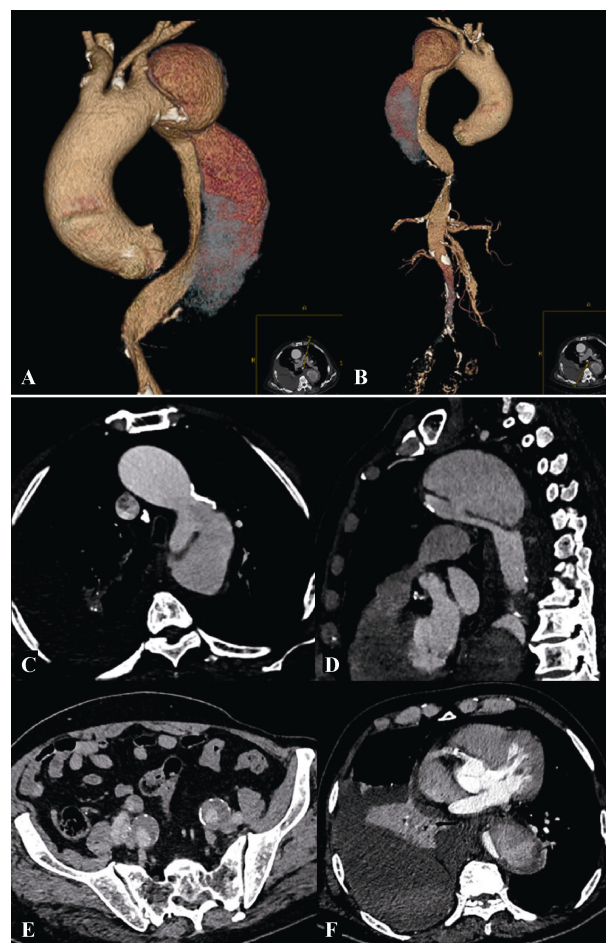


Figure 2. CT angiography of patient with an extensive DeBakey type IIIb aortic dissection extending from the arch to the bilateral internal iliac artery. (A): anteroposterior VR image showed proximal extent of dissection extends from the aortic with false lumen visualized to the left subclavian artery; (B): posteroanterior VR image showed dissection distally to the bilateral iliac artery, the true lumen was snake-like irregular stenosis due to a dilatation of the false lumen; (C&D): MPR images showed the tearing range from the arch to the bilateral internal iliac artery; (E): contrast-enhanced CT image showed that the false lumen has partially thrombosed, but aneurysmal dilatation of the affected internal iliac artery remains; (F): axial CT images showed a large effusion in the right pleural cavity. MPR: multiplaner reconstruction; VR: volume-rendered.

angiotensin receptor blocker/diuretic (Irbesartan/Hydrochlorothiazide 150/12.5 mg), a β -blocker (Metoprolol 50 mg/d), and statins (Atorvastatin 20 mg/d). After medical treatment his symptoms were attenuated and systolic blood pressure controlled to 100 mmHg to 130 mmHg and heart rate 70 to 90 beats/min. The patient remained experiencing only episodic mild chest discomfort over the next week, and he was discharged asymptomatic 10 days later. Five months after hospital discharge, he remains well and to receive routine follow-up care.

3 Discussion

AoD is one form of acute aortic syndromes, which include AoD, intramural hematoma and penetrating aortic ulcer. Population-based studies suggest that the incidence of acute AoD ranges from 2 to 3.5 cases per 100,000 person-years, which correlates with 6,000 to 10,000 cases annually in the United States.^[1] AoD has one of the highest mortality rates of cardiovascular diseases, which occurs commonly in the elderly and its prevalence increases with age.^[3] Despite major advances in the non-invasive diagnosis of AoD in medical and surgical therapies, the complexities of diagnosis and management remain a challenging issue.^[4] Clinical variability can make diagnosis relating to the distribution of aortic involvement difficult,^[5-7] due to symptomatic heterogeneity, such as chest pain, abdominal pain, even neurological deficits and syncope. Because thoracic aortic diseases are usually asymptomatic and not easily detectable until an acute and often catastrophic complication occurs, consequently (therefore) acute AoD is frequently missed on initial presentation. The vast majority of patients with symptoms of pain and a minority of patients with no pain, showed by a respective analysis that 6.4% of AoD patients were painless and up to one-third of patients may have such atypical features.^[8] A study also showed that the elderly AoD patients had less chest and abdominal pain.^[9]

D-dimer fibrin fragments are present in fresh fibrin clot and in fibrin degradation products of cross-linked fibrin. If the dissection starts with a rupture of the vessel intima, blood will enter the media. Inside the aortic wall coagulation is activated, resulting in the presence of thrombogenic material within the arterial wall.^[10] There is evidence that the D-dimer may be a biomarker applicable to diagnosis of AoD.^[11,12] Patients with AoD had significantly higher elevated D-dimer, compared to chronic aneurysms and normal controls. D-dimer levels higher than 700 ng/mL had a sensitivity of 94% and specificity of 59% for diagnosis of AoD.^[11] Recently, Gollidge, *et al.*^[13] reported significantly higher circulating concentrations of D-dimer in patients with abdominal aortic aneurysm.

Cardiac secretion of BNP increases with heart failure (HF) progression, and plasma BNP measurement has emerged recently as a useful biomarker for the diagnosis and prognosis of HF. However, the accumulated evidence demonstrate that elevated BNP levels represents a final common pathway for many cardiovascular pathologic states and can be used as a biomarker for non-HF mechanisms, preclinical disease, and other pathologic states of myocardial disease.^[14] The natriuretic peptide A receptor that binds BNP is present in the vascular wall, and the biological im-

portance of BNP in the control of vascular function has been demonstrated in recent studies.^[14,15] A study demonstrated that in AoD and chronic uncomplicated aneurysms NT-proBNP levels are significantly higher compared to normal controls.^[11] Recently Gutin, *et al.*^[16] reported the NT-proBNP levels were strongly and positively correlated with ascending and descending aortic diameter in women with Turner syndrome against healthy female controls. In the patient, echocardiography revealed the abnormality of left ventricle performance. A previous study showed the high plasma BNP level in a patient with AoD may be related to essential hypertension accompanied by ventricular diastolic dysfunction.^[17]

Since ventricular distension is the classical stimulus, studies have demonstrated that BNP release also may occur with ischemia, inflammation, redox stress and the local effects of catecholamines, angiotensin II and endothelin-1,^[17,18] as well as BNP also exerting a protective role through anti-inflammatory endothelial actions in the systemic circulation.^[18,19] In the patient, elevation of CRP levels might reflect the inflammatory factors involved with AoD. Current evidence indicates that inflammation play an important role in development and progression of AoD.^[20]

In conclusion, AoD has a myriad of manifestations depending on the involvement of aortic branches. In the case of our elderly patient, it was manifested by an extensive dissection from thoracic aorta to distally below the renal arteries with right massive pulmonary effusion, which resulted in progressive anemia, but complaining only of acute severe abdominal pain. The D-dimer is a simple and quick laboratory test that can be used as a 'rule-out' test in patients with suspected AoD, especially in those with a dilated aortic arch. High plasma BNP levels in a patient with AoD may involve cardiovascular comorbidities, as well as also exerting a protective role through anti-inflammation.

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