

CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

Vascular Diseases Quiz – Case 16

A 65-year-old male patient was admitted to the emergency department due to sudden onset of thoracic and back pain, beginning 24 hours prior presentation. His past medical history was unremarkable, except from severe tobacco and alcohol abuse for the past 35 years. At presentation, the patient was afebrile, with markedly elevated blood pressure (BP) (BP: 230/140 mmHg in both arms), easily and symmetrically palpable peripheral pulses and no abdominal tenderness. Cardiological examination and ECG finding revealed atrial fibrillation, with no signs of acute myocardial ischemia. Chest examination was unremarkable, while chest x-ray revealed a mediastinal enlargement. After initial administration of intravenous clonidine to control the arterial hypertension, a CTA of the chest and abdomen was performed, due to continuous pain (figures 1, 2).

Quiz #1: What is the diagnosis?

Quiz #2: What is the optimal treatment for this patient's condition?

Comment

Patients with acute aortic dissection typically present with a tearing sensation in the back which is different in quality and location than the pain of angina. Nonetheless, the pain of acute

aortic dissection may mimic other disorders including angina, heartburn, or pneumothorax. The patient may present with the signs and symptoms of cardiac tamponade if rupture has occurred into the pericardium. Etiologies of acute aortic dissection include Marfan's syndrome, cystic medial necrosis, and hypertension. The presence of a bicuspid aortic valve has a high coincidence with aortic dissection. Acute aortic dissection arises from separation of the aorta within the media. The media and adventitia of the aorta literally dissect away from the intima and the inner portion of the media. Controlling the patient's hypertension is essential in slowing or halting the progression of the aortic dissection. There is always an intimal tear associated with the aortic dissection which permits blood to dissect into the media, and the location of this intimal tear is critical for planning surgical management. The tear is propagated simply by the pulsatile effect of cardiac contraction. Although the progression of the aortic dissection varies, usual locations are the right anterior portion of the ascending aorta, greater curvature of the transverse arch, and left anterolateral wall of the descending aorta.

ARCHIVES OF HELLENIC MEDICINE 2011, 28(1):130-131
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2011, 28(1):130-131

S. Lioudaki,
E. Psathas,
A. Katsargyris,
C. Klonaris

First Department of Surgery, Vascular
Division, Medical School, University of
Athens, "Laiko" General Hospital, Athens,
Greece



Figure 1. Aortic dissection in the thoracic aorta. Note the intimal flap separating the aorta into two lumens (arrow).



Figure 2. Aortic dissection in the abdominal aorta. Note the intimal flap separating the aorta into the two lumens (arrow).

Two main classifications of aortic dissection exist – DeBakey and Stanford – based on the location of the intimal tear. A dissection involving the ascending, transverse, and descending aorta is a DeBakey type I dissection; that involving only the ascending aorta is a DeBakey type II, whereas a dissection involving solely the descending thoracic aorta is a DeBakey type III dissection. This has been simplified in the Stanford classification, where one simply asks the question whether or not there is a component of an ascending aortic dissection. If the dissection does involve the ascending aorta, i.e. a DeBakey type I or II, then this is a Stanford A dissection. If, however, only the descending thoracic aorta is involved (DeBakey type III), then this is a Stanford B. This is a simpler and clinically more useful classification because the management is exactly related to whether it is a type A or type B dissection.

Type A dissections are managed with surgery whereas type B dissections are generally managed only with antihypertensive agents and no surgery. The reasons that type A dissections require surgery is because of the high risk of complications arising from the ascending aortic dissection. This includes dissection into the coronary ostia with infarction, rupture into the pericardium resulting in tamponade, acute aortic insufficiency from dissection into the aortic valve, or free rupture and exsanguination. One year survival rates for patients with acute type A dissections are in the range of 60% for surgical management versus 5% for medical management; surgical management is clearly preferred. Conversely, medical management is superior for type B dissections, in the range of 70%, and is therefore the preferred treatment. Ascending aortic dissection is a dangerous condition that requires treatment. It is important to remember that on occasion there may be a type B dissection which progresses retrogradely to dissect up into the transverse and then ascending aorta even though the actual tear is in the aorta distal to the take-off of the left subclavian artery. This condition still requires surgery because it may further dissect down to the coronaries or

aortic valve or rupture into the pericardium.

Blood pressure control is mandatory in these patients, and includes not only vasodilator treatment with Nipride but also beta blockers to decrease the force of contraction, thus decreasing dP/dT and shear force exerted on the dissection. Hypertensive therapy with beta blockers and vasodilators is the mainstay of management for type B dissections. Generally, these patients do not require surgery unless their pain is not controlled after an appropriate course of antihypertensive management or if they have visceral sequelae of their dissection. This includes decreased blood flow to the renal arteries, celiac axis, superior mesenteric artery, or shearing off of the artery of Adamkiewicz with spinal ischemia or limb threat from shearing off of the iliac vessels.

The patient discussed herein had a type B aortic dissection caused by malignant hypertension and severe aortic calcification, extending from the origin of the left subclavian artery all the way down to the left iliac artery, with no signs of end organ ischemia or limb threat. He was managed with intravenous administration of beta blockers and nitroglycerine, along with close monitoring and medical surveillance for possible complications. After control of the hypertension and size of pain, the patient was treated with oral antihypertensive medication and was discharged after 10 days of hospitalization. Long-term follow-up with CTA of the thorax and abdomen is mandatory in this group of patients, in order to detect early and treat late complications of acute aortic dissection.

Corresponding author:

C. Klonaris, First Department of Surgery, Vascular Division, Medical School, University of Athens, "Laiko" General Hospital, Athens, Greece
e-mail: chris_klonaris@yahoo.com