

Chest pain and paraplegia as a presentation of aortic dissection: apropos of a case

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ABSTRACT

Acute aortic dissection is the most common catastrophic event affecting the aorta. Its early mortality is very high, with a rate of 1-2% per hour, in the early hours, after the dissection takes place. The key symptom is the chest pain presentation, but there are others that may be unnoticed, and they even mask the disease. In this work is described a case with atypical presentation of this disease: a man of 72 years old with a history of hypertension and diabetes mellitus type 2, which went to the emergency service with oppressive chest pain, vomiting and inability to move the lower limbs. When carried out the physical examination, a decrease of peripheral pulses was found in the right lower limb, spastic paraplegia and absent osteotendinous reflexes in both lower limbs. A computed tomographic angiography (CTA) was performed, where an aortic dissection type IIIb was diagnosed. The patient died 24 hours after the diagnosis.

Key words: Acute aortic dissection, Paraplegia, Chest Pain

Dolor torácico y paraplejía como forma de presentación de disección aórtica: a propósito de un caso

RESUMEN

La disección aórtica aguda es el episodio catastrófico más frecuente que afecta a la aorta; su mortalidad precoz es muy alta, con una tasa de hasta 1-2% por hora, en las primeras horas, después de producirse la disección. El síntoma de presentación fundamental es el dolor torácico, pero existen otros que pueden pasar inadvertidos, e incluso enmascarar la enfermedad. En este trabajo se describe un caso con presentación atípica de esta enfermedad: un hombre de 72 años, con antecedentes de hipertensión arterial y diabetes mellitus tipo 2, que acudió al servicio de urgencias con dolor precordial opresivo, vómitos e imposibilidad de mover los miembros inferiores. Al examen físico se constató disminución de los pulsos periféricos en miembro inferior derecho, paraplejía espástica y reflejos osteotendinosos ausentes en ambos miembros inferiores. Se realizó angiografía donde se diagnosticó disección aórtica tipo IIIb. El paciente falleció a las 24 horas del diagnóstico.

Palabras clave: Disección aórtica aguda, Paraplejía, Dolor en el pecho

INTRODUCTION

Aortic dissection (AD) is defined as the aortic artery's rupture of the intima, sometimes caused by an intramural bleeding, resulting in separating the layers of the aortic wall and the subsequent formation of a true and false lumens, with or without communication between them.

The epidemiological studies of AD are scarce, and in some it is estimated approximately 6 in 100 thousand people a year¹. The incidence is higher in men than in women, and it increases with age², being hypertension the most frequent risk factor associated, which is generally poorly controlled and may be present in 65-75% of affected individuals³⁻⁷. Other risk factors include previous illnesses of the aorta or aortic valve, family history of aortic disease, smoking, thoracic trauma and intravenous drug use (cocaine and amphetamines).

The most common symptom is chest pain, which is present in 96% of cases. It is usually intense, of sudden onset, with maximum intensity at the beginning; in 17% of cases it becomes migratory and follows the path of the dissection along the aorta⁸. However, there are other symptoms and signs that are less common, and which may mask the true arterial injury. Here is described the case of a patient that is received in the Emergency Department with chest pain and neurological symptoms, which is diagnosed an AD through a computed tomography (CT) scan with contrast.

CASE REPORT

A 72-year-old man with history of hypertension, treated with enalapril and hydrochlorothiazide, type 2 diabetes mellitus without drug treatment, and a benign prostatic hyperplasia surgery a year ago came to the polyclinic with oppressive chest pain that radiated to the back and the upper abdomen, with no apparent relief; he was given three tablets of sublingual nitroglycerin, and

morphine; and with the absence of clinical improvement he was transferred to the *Hospital Militar Central Dr. Carlos J. Finlay* from Marianao, Havana, Cuba. Here he had vomiting with traces of foods, relaxation of the sphincter and began to complain that he could not move the lower limbs.

Physical examination data

Respiratory rate of 21 breaths per minute. Rhythmic cardiac sounds, no murmurs; heart rate of 86 beats per minute, and blood pressure of 170/90 mmHg.

Both lower limbs with decreased peripheral pulses; the right cold and the left with normal temperature.

The patient was conscious and oriented in time, space and person. There were no meningeal signs. The muscular strength of the upper limb was preserved. There was confirmed a spastic paraplegia, the osteotendinous reflexes were decreased in the upper limbs and absent in the lower. Absence of bilateral cutaneous-plantar response. Sensitive level for the superficial and deep sensitivity in D₉-D₁₀.

Table 1. Complementary blood tests.

Test	Result	Reference values
Hematocrit	0,48	0,42 – 0,52
Leukogram	11,4 x 10 ⁹ /L	4,5 – 10,5 x 10 ⁹ /L
LDH	1166 U/L	200 – 400 U/L
Alkaline phosphatase	226 U/L	100 – 290 U/L
TGP	45 U/L	0 – 49 U/L
GGT	30 U/L	5 – 45 U/L
Cholesterol	3,86 mmol/l	3,87 – 6,30 mmol/L
Triglycerides	1,46 mmol/L	0,46 – 1,88 mmol/L
Creatinine	491,2 µmol/L	47,6 – 113,4 µmol/L
Uric acid	629 µmol/L	155 – 428 µmol/L
Urea	12,8 mmol/L	1,80 – 6,10 mmol/L
Total proteins	68,1 g/L	60,0 – 80,0 g/L
Albumin	46 g/L	38 – 54 g/L
Total bilirubin	6,99 µmol/L	0 – 21 µmol/L
INR	1,63	1,0 – 2,0
Platelet count	195 x 10 ⁹ /L	150 – 400 x 10 ⁹ /L

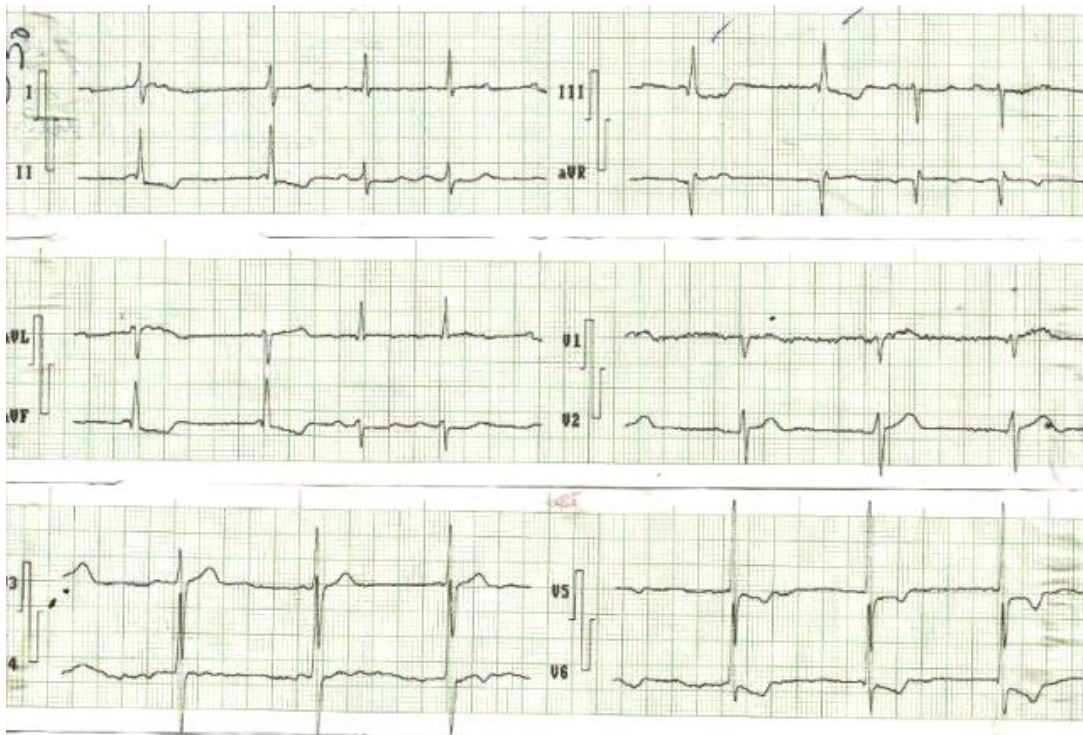


Figure 1. Electrocardiogram ruling out the presence of an acute coronary syndrome as the cause of chest pain.

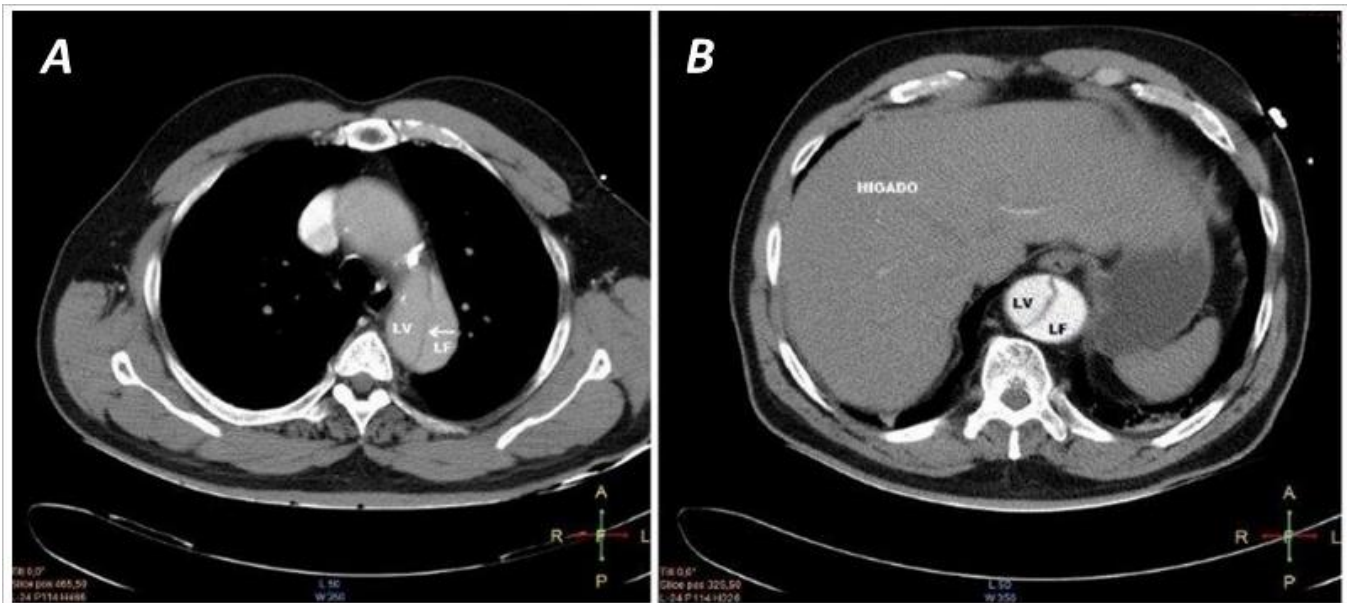


Figure 2. Computed tomography with contrast. **A.** Aortic arch where the dissection begins, and the intimal flap (arrow) **B.** There is observed a cut below the diaphragm where the extent of the dissection is displayed. LV & LF: true and false lumen, by their acronyms in Spanish.

Examinations

- Blood tests: Described in **table 1**.
- Electrocardiogram: Electric axis to the left (Δ QRS: -60°), coupling premature ventricular contractions, left bundle branch block and left ventricular systolic overload (**Figure 1**).
- CT scan (**Figure 2**): There was observed double lumen from the aortic arch to its bifurcation, without affecting the common iliac arteries. Renal arteries in the true lumen. Bilateral pleural thickening, no pericardial effusion or periaortic collection. No retroperitoneal free liquid. The dissection is observed extensively in the three-dimensional reconstruction (**Figure 3**).

With the patient's clinical picture and the findings from the questioning and physical examination, there arises the presence of aortic dissection type IIIb of DeBakey, with involvement of the lumbar spine's spinal arteries. With this diagnosis, the patient was admitted in the Intensive Care Unit, where at 24 hours he suffered a cardiac arrest with asystole and died despite resuscitation manoeuvres. The necropsy confirmed the clinical diagnosis, and it is found, as a direct cause of death, a saddle pulmonary embolism.

COMMENT

AD is mainly presented with chest pain, but there are other less frequent symptoms and signs, which may confuse and delay the diagnosis and treatment of this medical emergency. Neurological symptoms can be dramatic, dominate the clinical picture and mask the real cause of the problem. They generally appear due to cerebral perfusion disorders, hypotension, distal embolism or peripheral nerve compression. The frequency of neurological symptoms in the AD is of 15-40%, and in half of the patients may be transitory⁹.

Acute paraplegia due to spinal ischemia caused by occlusion or loss of perfusion of spinal arteries is uncommon and it can be confused with the Leriche syndrome¹⁰. The most recent International Record of Aortic Dissection (IRAD) described, in AD type A, an incidence of major brain damage (coma and stroke) of less than 10%, and ischemic damage to the spinal cord of 1%⁷.

In the case presented, high clinical suspicion against a patient with chest pain without apparent electrical changes, vomiting and neurological damage given by spastic paraplegia and absence of tendon reflexes in the lower limbs led to the diagnosis of AD, supported in the contrasted images. Unfortunately, despite the early diagnosis, the patient survival was not achieved.

REFERENCES

1. Howard DP, Banerjee A, Fairhead JF, Perkins J, Silver LE, Rothwell PM. Population-based study of incidence and outcome of acute aortic dissection and premorbid risk factor control: 10-year results from the Oxford Vascular Study. *Circulation*. 2013;127:2031-7.
2. Olsson C, Thelin S, Ståhle E, Ekbom A, Granath F. Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. *Circulation*. 2006;114:2611-8.



Figure 3. Three-dimensional tomography reconstruction of the aortic where the line dissection is observed (arrows).

3. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, *et al.* The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA*. 2000;283:897-903.
4. Januzzi JL, Eagle KA, Cooper JV, Fang J, Sechtem U, Myrmel T, *et al.* Acute aortic dissection presenting with congestive heart failure: results from the International Registry of Acute Aortic Dissection. *J Am Coll Cardiol*. 2005;46:733-5.
5. Bonnefoy E, Godon P, Kirkorian G, Chabaud S, Touboul P. Significance of serum troponin I elevation in patients with acute aortic dissection of the ascending aorta. *Acta Cardiol*. 2005;60:165-70.
6. Gilon D, Mehta RH, Oh JK, Januzzi JL, Bossone E, Cooper JV, *et al.* Characteristics and in-hospital outcomes of patients with cardiac tamponade complicating type A acute aortic dissection. *Am J Cardiol*. 2009;103:1029-31.
7. Di Eusanio M, Trimarchi S, Patel HJ, Hutchison S, Suzuki T, Peterson MD, *et al.* Clinical presentation, management, and short-term outcome of patients with type A acute dissection complicated by mesenteric malperfusion: observations from the International Registry of Acute Aortic Dissection. *J Thorac Cardiovasc Surg*. 2013;145:385-90.e1.
8. Trimarchi S, Tolenaar JL, Tsai TT, Froehlich J, Pegorer M, Upchurch GR, *et al.* Influence of clinical presentation on the outcome of acute B aortic dissection: evidences from IRAD. *J Cardiovasc Surg (Torino)*. 2012;53:161-8.
9. Bossone E, Corteville DC, Harris KM, Suzuki T, Fattori R, Hutchison S, *et al.* Stroke and outcomes in patients with acute type A aortic dissection. *Circulation*. 2013;128:S175-9.
10. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, *et al.* Diagnosis and management of aortic dissection. *Eur Heart J*. 2001;22:1642-81.