Painless acute aortic dissection presenting as echocardiographically diagnosed left ventricular outflow tract obstruction

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We present an unusual case of painless acute aortic dissection with an atypical echocardiographic presentation that could be confused with an aortic stenosis. Transesophageal echocardiography excluded the presence of aortic stenosis and disclosed a systolic anterior movement of the mitral valve as the cause of left ventricular outflow tract obstruction. It also revealed a type A aortic dissection.

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The most common presenting symptom of acute aortic dissection is severe pain, found in up to 90% of cases[1]. However, the clinical presentation of such a catastrophic event may be variable and thus the diagnosis may be quite challenging[2-9].

We report the case of a patient who presented with painless acute aortic dissection associated with left ventricular outflow tract (LVOT) obstruction.

Case report

An 80-year-old woman was admitted to our Institution because of nausea, vomiting and cold perspiration. Her past medical history was significant for mild hypertension and chronic ischemic heart disease. Medications included diltiazem 180 mg/day. On physical examination, she appeared confused, with a respiratory rate of 25 breaths/min, a blood pressure of 90/60 mmHg and a heart rate of 61 b/min. Cardiac auscultation revealed soft heart sounds without murmurs or gallops, and the presence of a pericardial friction rub. All peripheral pulses were palpable but constantly decreased while at percussion and auscultation the lung fields were clear. The electrocardiogram showed sinus rhythm with a normal PR interval, Q waves with slight ST segment elevation in the inferior leads and slight ST segment depression in the lateral leads. Laboratory values were in the normal range, except for white blood cell count, which was 13 640/mm^3.

A transthoracic echocardiography was performed with a Sonos 2000 Hewlett-Packard machine (Hewlett-Packard, Andover, MA, USA). A two-dimensional long-axis view revealed normal left ventricular chamber size and function, with concentric hypertrophy, thickened aortic leaflets with restricted motion and an enlarged ascending aorta. A systolic anterior movement of the mitral valve obstructing the LVOT was not detectable. A moderate pericardial effusion, without evident signs of right ventricular or atrial collapse and anterior to the right ventricular wall, was noted in the short-axis views (Fig. 1). Continuous-wave Doppler interrogation of the LVOT disclosed a transaortic jet with a 4 m/s peak velocity, consistent with LVOT obstruction (Fig. 2).

In order to better assess the dimensions of the ascending aorta and to elucidate the cause of the LVOT obstruction, we decided to perform an urgent multiplane transesophageal echocardiographic study.

A short-axis upper esophageal view showed a normal aortic root with thickened leaflets and an opening aortic valve orifice in systole (Fig. 3). As the probe was slightly withdrawn and the array steered to 0°, an enlarged ascending aorta with a true and a false lumen separated by a thickened intimal flap was noted (Fig. 4); the intimal flap extended 4 cm distal to the aortic valve up to the midsegment of the aortic arch, while

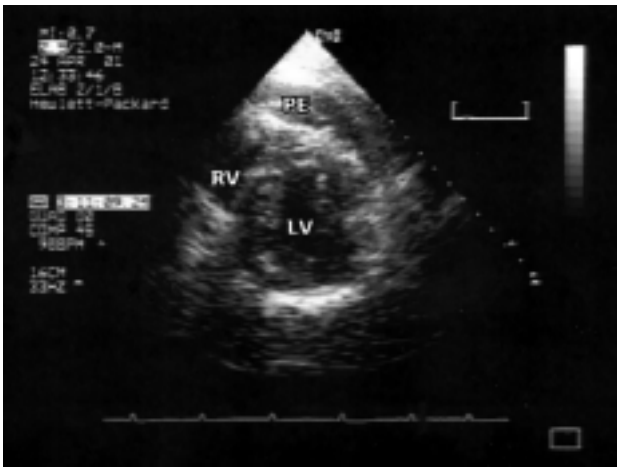

Figure 1. *Moderate pericardial effusion, anterior to the right ventricular wall. LV = left ventricle; PE = pericardium; RV = right ventricle.*

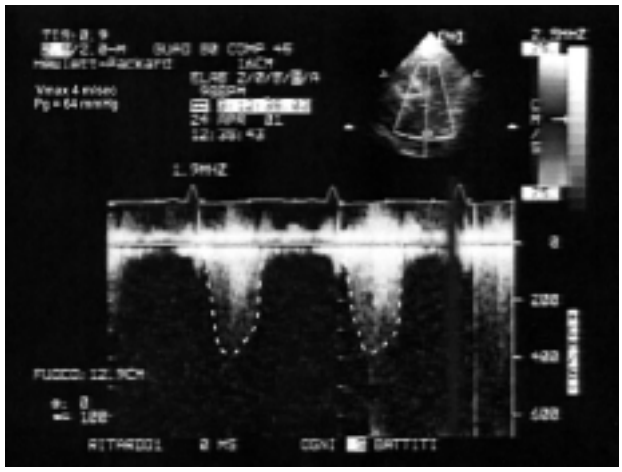

Figure 2. *Transaortic jet with a 4 m/s peak velocity.*

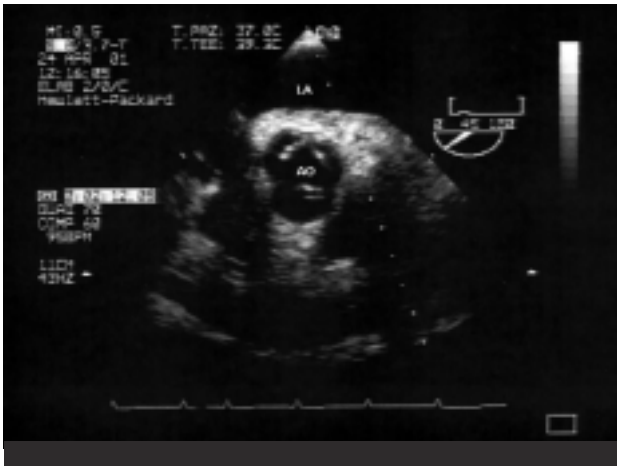

Figure 3. *A short-axis upper esophageal view showing a normal aortic root with thickened leaflets and an opening aortic valve orifice in systole. AO = aorta; LA = left atrium.*

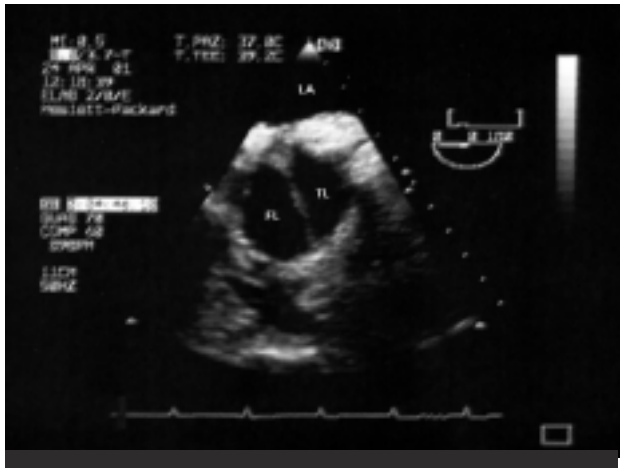

Figure 4. *Ascending aorta with a true (TL) and a false lumen (FL) separated by a thickened intimal flap. LA = left atrium.*

at the level of the descending aorta the false lumen was occupied by a thrombus.

A long-axis midesophageal view revealed a systolic anterior movement of the mitral valve obstructing the LVOT (Fig. 5), and M-mode recording of the intimal flap showed an unusual movement with a midsystolic notch reflecting the effect of the dynamic obstruction (Fig. 6).

On the basis of these data, a diagnosis of acute type A aortic dissection with LVOT obstruction was made and surgery was recommended. Since the patient finally refused the operation, medical therapy, including fluid administration to expand plasma volume, was initiated.

The patient developed atrial fibrillation, which responded to amiodarone infusion. Her conditions slightly improved. On the second day, she became suddenly hypoxic and tachypneic with rapidly declining values of blood pressure. A transthoracic echocardiographic examination was promptly performed and revealed cardiac tamponade with right ventricular collapse. She was immediately intubated and mechanically ventilated, but despite aggressive fluid administration and pericardiocentesis, she expired after 30 min.

Discussion

Acute dissection of the ascending aorta is a life-threatening emergency that, if unrecognized or untreated, has mortality rates at 24 hours and at 1 month of 25 and 90%, respectively[1].

The most common clinical presentation of this catastrophic event is severe, sharp pain. However, other symptoms related to the course of the dissection and to the involvement of other vessels may complicate the clinical picture of this disease, making the diagnosis very difficult[2]. A previous review reported that in 28% of 235 patients with acute aortic dissection the correct diagnosis was not made before *post mortem* examination[3].

The present report describes a case of acute aortic dissection presenting with atypical clinical and

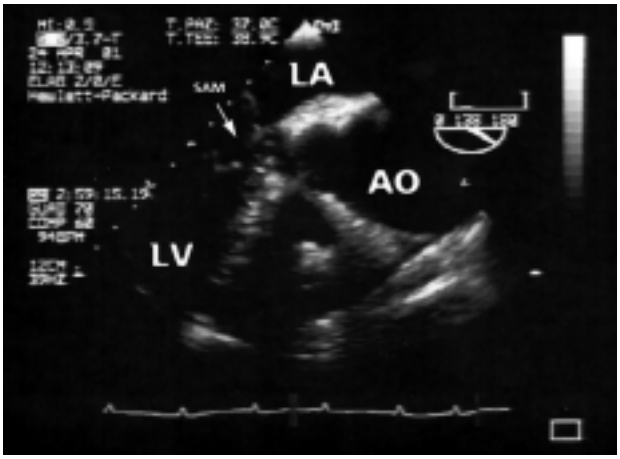

Figure 5. *A long-axis midesophageal view showing a systolic anterior movement of the mitral valve obstructing the left ventricular outflow tract. Abbreviations as in figures 1 and 3.*

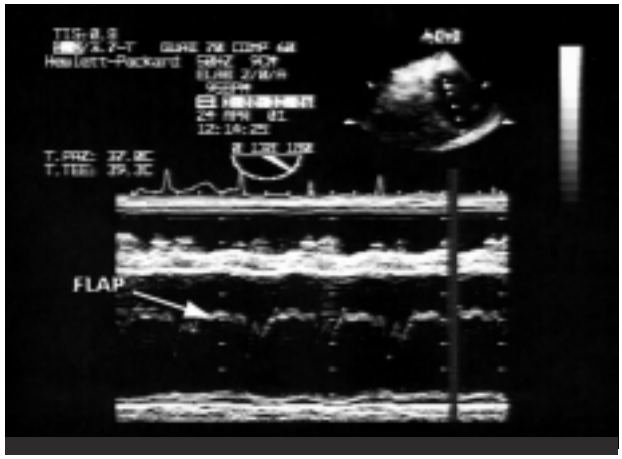

Figure 6. *M-mode recording of the intimal flap showing an unusual movement with a midsystolic notch.*

echocardiographic features. Our patient was hospitalized with a history including vomiting and cold perspiration lasting 6 hours. The electrocardiogram performed in the emergency department showed Q waves in the inferior leads and slight changes of the ST segment, which were not consistent with acute ischemic heart disease. Transthoracic Doppler echocardiography disclosed a transaortic jet of 4 m/s without the classic leftward concave late systole peaking shape that, when associated with thickened aortic leaflets, could suggest the presence of a significant aortic stenosis. Transesophageal echocardiography excluded the presence of aortic stenosis and disclosed a type A aortic dissection with dynamic LVOT obstruction caused by systolic anterior movement of the mitral valve.

Dynamic LVOT obstruction has been well described in hypertrophic cardiomyopathy and in other conditions such as during cardiac tamponade, dobutamine stress echocardiography, and after mitral valve surgery[10-13]. To the best of our knowledge, this is the first case of painless acute aortic ascending dissection with LVOT obstruction. Book et al.[10] have reported a case of proximal aortic dissection with echocardiographic features of LVOT obstruction; however, in the present case, proximal aortic dissection was complicated by cardiac tamponade. This complication, associated with concentric left ventricular hypertrophy resulting from long-standing hypertension, may reduce left ventricular filling and cause LVOT obstruction. In our patient, a moderate pericardial effusion was present but without signs of right ventricular or atrial collapse.

It is our opinion that in the present case report, hypovolemia was the main cause of LVOT obstruction. Hypovolemia reduces the cardiac output and causes an increased release of catecholamines; furthermore, as recently reported in an experimental study on bleeding and volume repletion, left ventricular pseudohypertrophy with concentric left ventricular remodeling is an echocardiographic feature of hypovolemia[14].

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