A 58-year-old female patient, who had reported great abdominal pain in the epigastric and umbilical areas for 3 years followed by an episode of syncope, having been admitted to another health service with probable diagnosis of acute pancreatitis, not confirmed by supplementary exams, was discharged from hospital after 5 days of analgesic use. One year ago, there was abdominal pain relapse, accompanied by faint and sudoreosis, and in the last six months the patient has reported precordial, left thoracic paravertebral and abdominal pain, unrelated to effort and of moderate intensity. She denied any history of high blood pressure, alcoholism, smoking, diabetes mellitus, dyslipidemia, infectious diseases or trauma. She was not in use of medication.

At physical examination, she was in good state, with rosy cheeks, hydrated, without any sign of vestibular disorder, normorhythmic and normophonic cardiac sounds, without cardiac murmurs, blood pressure =130/30 mmHg, heart rate = 88 bpm, symmetrical peripheral pulse, absence of oedema in lower limbs, physiological vesicular murmur, RF = 13 ipm, flaccid abdomen, which was slightly sore to deep palpation, without visceromegaly.

The electrocardiogram displayed sinus rhythm, QRS-axis = 50°, without any sign of myocardial ischaemia or atrial and ventricular load. The chest X-ray revealed normal cardiac area without any sign of myocardial ischaemia or atrial and ventricular hypertrophy. The thickness of the descending aorta wall at T-6 level was 8mm. A spiral computed angiotomography of thorax and abdomen exhibited an extensive intramural haematoma of the descending aorta, starting near the origin of the left subclavian artery and extending to the bifurcation of the abdominal aorta (fig. 1). The thickness of the descending aorta wall at T-6 level was 8mm. Atenolol was then prescribed, 50mg/day, and the protocol of periodic clinical (every month) and angiographic (every three months) reassessment was introduced.

The aortic intramural haematoma was described in 1920 as “dissection without intimal rupture” and regarded as a distinct pathological entity during necropsy\(^1\). With the arise of modern imaging methods, its diagnosis in vivo became possible\(^2\). Nowadays, the aortic intramural haematoma is considered a precursor of the acute aortic dissections, originating from ruptured vasa vasorum in medial wall layers; it occasionally causes a secondary intimal tear and communication with the aortic lumen. Similarly to the classic dissections, it may extend along the aorta, progress, regress, or reabsorb \(^3\).

The initial clinical presentation is not usually different from that of classic aortic dissections. The anterior or posterior chest pain, in a patient with coexisting history of hypertension, is the most common finding. Abnormalities of either the diameter or contour of the thoracic aorta on chest X-rays are present in approximately 90% of the patients \(^4\).

Ruling out intimal rupture or dissection lamina is a prerequisite for the diagnosis of intramural haematoma by an imaging method. The localized thickening of the aortic wall > 7mm, circumferential or crescent shaped, and/or evidence of blood accumulation in the media layer is a diagnosis criterion for the aortic intramural haematoma\(^5,6\). At computed tomography, the recent haematoma is characterized by a higher density area, in comparison to neighbouring layers of the aortic wall; conversely, the partial or total thrombosis is observed as multiple layers of increasing density\(^7\). The diagnostic sensitivity between computed tomography, magnetic resonance imaging and the transesophageal echocardiography does not seem to be much different. The choice of one specific imaging method depends on some variables, including the clinical condition of the patient, the doctor’s preference and the method’s availability\(^8\).

In the reported case, the high density 8mm thickening of the aortic wall suggests recent intramural haematoma. Therefore, we believe that the event started in the last 6 months, coinciding with the clinical presentation of persistent thoracic and abdominal pain.

The intramural haematoma is more frequent in elderly and hypertensive patients, where aortic wall thickening secondary to atherosclerotic disease and aneurysmatic dilatation with mural thrombus are common, which might be a diagnostic challenge. The identification of the intimal layer and the careful observation of the internal surface of the thickened aortic wall are essential for the differential diagnosis\(^8\).

The intramural haematoma is thought to lead to acute dissection in 28% to 47% of the patients. Spontaneous regression is seen in 10% of the patients \(^3\). Nienaber and cols \(^2\) demonstrated that the hospital mortality rate, for the patients who presented ascending aortic involvement, was 80% in the group clinically treated and 0% in the group submitted to surgery. The ones with descending aortic involvement did not show significant difference of mortality rate between the groups treated clinically and surgically.

The Stanford Classification of acute aortic dissections also seems acceptable for intramural haematomas. Patients with type A intramural haematoma (involvement of the ascending aorta), reporting persistent or recurrent chest pain should be treated surgically, with replacement of the afflicted region to prevent rupture,
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tamponade or compression of coronary ostia. Patients with type B intramural haematoma (involvement of the descending aorta) are medically treated and, if necessary, submitted to stent-graft placement. In clinical practice, studies with repeated imaging methods are necessary in order to assess the progression or regression of the aortic intramural haematoma.

References