Rev Port Cardiol. 2014;33(7–8):467.e1–467.e7

Revista Portuguesa de Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

CASE REPORT

Aortic intramural hematoma: An unpredictable evolution☆

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Received 30 September 2013; accepted 29 January 2014
Available online 23 August 2014

KEYWORDS
Intramural hematoma;
Thoracic aorta;
Aortic dissection;
Endovascular stent graft

Abstract Aortic intramural hematoma (IMH) is an acute aortic syndrome characterized by bleeding into the media of the aortic wall without intimal disruption or the classic flap formation. Its natural history is variable and still poorly understood, so strategies for therapeutic management are not fully established. In some cases there is partial or complete regression of the hematoma under medical treatment, but most progress to dissection, aneurysmal dilatation or aortic rupture.

The authors present the case of a 44-year-old hypertensive male patient admitted with a diagnosis of IMH of the descending aorta. Despite initial symptom resolution and optimal medical therapy, the IMH evolved to a pseudoaneurysm, which was successfully treated by an endovascular approach.

PALAVRAS-CHAVE
Hematoma intramural;
Aorta torácica;
Dissecção aórtica;
Prótese endovascular

Hematoma intramural da aorta: evolução (im)previsível?

Resumo O hematoma intramural da aorta (IMH) é uma síndrome aórtica aguda caracterizada pela ocorrência de hemorragia a nível da camada média da parede da aorta, sem evidência de ruptura ou *flap* da íntima. A história natural desta entidade clínica é muito variável e ainda pouco conhecida, pelo que a sua abordagem terapêutica não está completamente estabelecida. Nalguns casos pode ocorrer regressão parcial ou completa do hematoma sob tratamento médico, mas numa proporção significativa existe evolução para dissecção, dilatação aneurismática ou ruptura.

☆ Please cite this article as: Ponte M, Dias Ferreira N, Bettencourt N, et al. Hematoma intramural da aorta: evolução (im)previsível? Rev Port Cardiol. 2014;33:467.

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Os autores apresentam o caso de um homem de 44 anos, hipertenso, admitido com o diagnóstico de IMH da aorta descendente. Apesar de a resolução dos sintomas e do adequado controlo da tensão arterial com a terapêutica médica, o IMH evoluiu a curto prazo para a formação de um pseudoaneurisma, que foi tratado eficazmente por via endovascular.

Introduction

Aortic intramural hematoma (IMH) is considered a variant of classic aortic dissection (AoD) and is characterized by bleeding into the media of the aortic wall without intimal disruption. It accounts for 10–30% of acute aortic syndromes[1] and has similar clinical presentation, morbidity and mortality to AoD. However, its natural history and therapeutic management are not as well established as for AoD. Much of the uncertainty is due to its dynamic and unpredictable behavior over time; it can regress or evolve to dissection, aneurysmal dilatation or rupture, and thus requires continuous clinical monitoring and imaging follow-up.

Case report

A 44-year-old man, a smoker (48 pack-years), obese (body mass index 31 kg/m^2) and with untreated, uncontrolled hypertension, went to the emergency department for sudden-onset chest pain, which he described as stabbing, radiating to the interscapular region and worsening in dorsal decubitus. At admission he was hypertensive (160/85 mmHg), with no significant difference between the arms, normal heart rate (78 bpm), and strong, symmetrical peripheral pulses, and no other relevant abnormalities on physical examination.

The 12-lead electrocardiogram showed sinus rhythm and voltage criteria for left ventricular hypertrophy (LVH). Laboratory tests indicated mild leukocytosis with neutrophilia and elevated C-reactive protein, but no elevation of myocardial necrosis biomarkers. Transthoracic echocardiography showed moderate left atrial dilatation, left ventricular size at the upper normal limit, moderate concentric LVH, mild dilatation of the aortic root and ascending aorta with no evidence of flap formation or aortic regurgitation, and preserved global systolic function of both ventricles.

Chest computed tomography angiography (CTA) was performed to investigate the aortic disease, which revealed mild dilatation of the ascending aorta (maximum diameter 42 mm) and circumferential thickening of the aortic wall (approximately 10 mm) consistent with IMH, beginning immediately after the emergence of the left subclavian artery and involving the entire descending aorta and the proximal segment of the abdominal aorta, up to the emergence of the renal arteries (Figure 1A). It also showed a type A patent ductus arteriosus and a partially calcified atherosclerotic plaque (Figure 1B), and two ulcers in the wall of the proximal descending aorta (Figure 1C).

A diagnosis of uncomplicated Stanford type B IMH of the descending aorta was assumed; the patient was admitted to the cardiac intensive care unit and therapy was begun with intravenous sodium nitroprusside and labetalol, which resulted in blood pressure (BP) control and complete resolution of symptoms. The case was referred for medical and surgical evaluation and it was decided to maintain medical therapy with clinical and imaging monitoring surveillance, given the patient's stable condition under medical therapy and the absence of complications.

CTA was repeated on the eighth day of hospitalization, and showed a slight increase in hematoma thickness (to around 15 mm), although with no increase in length, and one of the ulcers in the aortic wall appeared deeper and more irregular (Figure 2). The case was again discussed with the cardiothoracic surgical team, and it was decided not to operate and to maintain medical therapy and surveillance. The patient remained clinically stable during hospitalization and was discharged on the 19th day, medicated with four classes of antihypertensive drugs including beta-blockers and referred for outpatient cardiology consultation, and imaging follow-up was scheduled.

CTA one month after hospital discharge showed intimal rupture of one of the aortic ulcers and evolution to localized dissection, with a pseudoaneurysm of the proximal segment of the descending aorta measuring 27 mm $\times$ 51 mm (Figure 3). In the light of these findings, the patient was rehospitalized and after discussion with the interventional cardiologist and cardiothoracic surgeon, it was decided to perform thoracic endovascular aneurysm repair (TEVAR). Two endoprostheses (Valiant® 38 mm $\times$ 150 mm and 34 mm $\times$ 150 mm) were implanted, the proximal prosthesis adjacent to the emergence of the left subclavian artery without obstructing its flow, and the distal prosthesis extending down to the beginning of the abdominal aorta, thus covering the ulcers, the pseudoaneurysm, the patent ductus arteriosus and most of the IMH. Post-procedural CTA confirmed that the TEVAR had been successful but an image consistent with dissection was observed in the abdominal aorta distally to the distal endoprosthesis, from the celiac trunk to the superior mesenteric artery, both of which emerged from the true lumen, as did the renal arteries (Figure 4A–C). It was not possible to confirm whether this dissection had been present prior to the TEVAR procedure, since the previous CTA had limited acquisition of the thoracic region. It