Aortic intramural haematoma and chronic anticoagulation: role of transoesophageal echocardiography

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Introduction
Aortic intramural haematoma (IMH) constitutes 15–20% of acute aortic syndromes, mainly involves descending aorta and has similar morbidity and mortality to aortic dissection.¹ Aortic intramural haemorrhage is produced by vasa vasorum bleeding; thus, anticoagulant treatment may be prejudicial. Transoesophageal echocardiography (TEE) affords correct diagnosis and follow-up of intramural haematoma thickness. To date, there is a paucity of information on the risk of anticoagulation in the evolution of intramural haematoma and the presence of complications such as wall re-bleeding or aortic rupture.

Case report
A 66-year-old woman had a history of venous sinus-type atrial septal defect operated on 6 years previously. She was receiving anticoagulant treatment with warfarin for chronic atrial fibrillation. The reason for the present admission was left lumbar pain of sudden onset irradiated to epigastrium. Physical examination revealed systemic blood pressure of 180/120 mmHg. ECG showed atrial fibrillation 70 bpm and chest X-ray was normal. Laboratory analysis revealed no alterations except an INR of 4.4, with normal myocardial lesion markers. Abdominal echography performed was normal. Transthoracic echocardiogram showed non-dilated left ventricle with normal function and dilated left atrium (55 mm). Transoesophageal echocardiogram disclosed an intramural haematoma in descending aorta (Figure 1A). Maximum thickness of the haematoma was 12 mm. Severe spontaneous contrast was observed at left atrium with absence of thrombi. Left atrial appendage emptying flow velocity was diminished (18 cm/s). The diagnosis of aortic intramural haematoma was confirmed by magnetic resonance imaging (MRI) (Figure 2A). The patient was admitted and it was decided to temporarily suspend the anticoagulation. At 5 days of uncomplicated evolution, with adequate systemic blood pressure control and, after verifying by TEE that haematoma thickness remained stable, anticoagulation was restarted with subcutaneous heparin. TEE performed 4 days later showed a marked increase in maximum thickness of the intramural haematoma (20 mm), in the absence of symptoms (Figure 1B). It was decided to suspend the anticoagulation, and the examination was repeated 5 days later; no changes in the aortic intramural haematoma or left atrium were observed.
Two weeks post-discharge, TEE (Figure 1C) showed a significant decrease in intramural haematoma thickness (7 mm), confirmed by MRI 1 month after discharge (Figure 2B). At 6 months of evolution, the haematoma was completely reabsorbed; thus, warfarin treatment was restarted, with no new complications in the aortic wall observed on the TEE performed at 1 year of follow-up.

Discussion

This case demonstrates that anticoagulation in acute phase on an IMH may lead to an increase in IMH thickness secondary to re-bleeding of the aortic wall vasa vasorum. The haematoma stabilized and later regressed progressively when anticoagulation was stopped for 6 months. IMH reabsorbed completely without complications in 30% of cases, with the remainder evolving to aortic dissection or the formation of aortic aneurysms. The indication for anticoagulant treatment in patients over 60 years of age with chronic atrial fibrillation and a history of arterial hypertension, left atrium dilatation and spontaneous atrial contrast is well established. Given the controversy of avoiding the embolic risk of atrial fibrillation or increasing the risk of aortic intramural haemorrhage, it was decided to initially suspend the anticoagulation. The satisfactory clinical evolution, blood pressure control and stabilization of the haematoma prompted the re-initiation of anticoagulation, which led to an increase in thickness of the haematomas, probably due to asymptomatic re-bleeding of the aorta wall. Owing to this complication, and although the cardioembolic risk of atrial fibrillation was significant, anticoagulation was suspended until total reabsorption of the haematoma was verified at 6 months of evolution.

This case illustrates the difficulty in decision-making in these circumstances. We believe that when the thromboembolic risk is intermediate or low, interruption of anticoagulant treatment is advisable until the haematoma regresses significantly. If the thromboembolic risk is very high, as in patients with valvular prostheses, close monitoring with imaging techniques should be performed, and if evolution is not satisfactory, endovascular or surgical treatment should be considered.

In conclusion, anticoagulant therapy in patients with an acute IMH may facilitate re-bleeding of the aorta wall. Individualization of the risk/benefit of anticoagulation and monitoring of haematoma thickness by imaging techniques may facilitate the choice of the most appropriate therapeutic management.

References