Resolution of an aortic thrombus under anticoagulant therapy

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Abstract

In a 50-year-old woman, admitted because of a renal infarction, a 10-cm long floating, highly mobile thrombus with a diameter of 15–20 mm in the descending aorta was detected by transoesophageal echocardiography and magnetic resonance imaging. She was a poor surgical candidate due to obstructive lung disease and obesity. Under intravenous heparinization with prothrombin time test values between 80 and 100 s, followed by oral anticoagulation with international normalized ratio values between 3.0 and 4.0, the thrombus resolved after 10 weeks and no recurrence occurred over the next 30 months.

Keywords: Aorta; Thrombus; Transesophageal echocardiography; Cardiac magnetic resonance imaging

1. Introduction

Uncertainty exists regarding the treatment of aortic thrombi. Therapeutic options are surgical removal, either by aortotomy with endarterectomy, thrombectomy or balloon embolectomy, thrombolysis or anticoagulation [1–10]. We present a case in whom a large, highly mobile thrombus in the descending aorta resolved during therapy with heparin and phenprocoumon.

2. Case report

A 50-year-old obese woman with chronic obstructive lung disease was hospitalized because of sudden lower back pain with pseudoradicular radiation to both legs and the abdomen. At admission, the pain in the legs had already disappeared and all distal pulses of the lower limbs were palpable.

Eight years before, she had undergone a surgical subclavian-carotis-transposition after an embolic event of the left upper extremity due to a left subclavian artery origin stenosis. Angiography, performed at that time, did not show any atherosclerosis. Since then the patient was on oral anticoagulant therapy with phenprocoumon. She had a low compliance and, whenever tested by her general practitioner, international normalized ratio (INR) values were only between 1.2 to 1.6. Because of chronic obstructive lung disease, she had been treated with oral glucocorticoids over the last 3 years. Except for smoking, she had no vascular risk factors. The actual symptoms were explained with – only clinically – suspected emboli to the lower limbs and a left renal infarction, confirmed by computed tomography. Anticardiolipin antibodies, antithrombin III, protein C, protein S, fibrinogen, platelet count, evaluation for vasculitis and serologic tests for syphilis were normal. Transesophageal echocardiography (TEE) detected in the descending aorta a 10-cm long floating, highly mobile thrombus with a diameter of 15–20 mm. The aortic wall was free of any atheroma. Neither TEE, computed tomography nor magnetic resonance imaging (MRI) detected any calcifications or thickening of the aortic wall.

Cardiac MRI revealed the only insertion site of the thrombus in the left subclavian artery stump (Fig. 1). Because of obesity and lung disease, the risk of surgery was considered too high. Thrombolysis was rejected because of the size of the thrombus and concern that it might re-embolize. Thus, the patient underwent intravenous heparinization over 3 weeks with prothrombin time test (PTT) values between 80 and 100 s (baseline PTT 23.7 s), followed by oral anticoagulation with INR values between 3.0 and 4.0. No further clinically apparent embolic event occurred. Because TEE, performed after 4 weeks, showed regression of the thrombus to a length of 4 cm she was dismissed from the hospital. Cardiac MRI after another 6 weeks showed complete resolution of the aortic thrombus. Only in the subclavian artery stump a small thrombus was visible (Fig. 1). After dismissal, the patient’s compliance improved and it was possible to achieve...
and maintain her INR values between 3.0 and 4.0. During a further follow-up period of 30 months, no events occurred suggestive of embolism, nor did echocardiographic and MRI examinations show any recurrence of thrombus formation.

3. Discussion

Aortic thrombi, originating from atheroma, are an embolic source, particularly in patients older than 70 years, and anticoagulant therapy is recommended [5,6]. Rarely, thrombi may originate from an aortic wall without atheroma, like in the presented case [7–10]. Here, the left subclavian artery stump has most probably served as a nidus for thrombus formation. Chronic medication with glucocorticoids might have promoted hypercoagulability as in other reported cases [8,10]. Additionally, the insufficient anticoagulation might have contributed to the development of the thrombus.

The ideal treatment of mobile aortic thrombi without atheromatosis has not been ascertained. Surgical removal, either by aortotomy with endarterectomy, thrombectomy or balloon embolectomy, and thrombolysis are the proposed therapies [1–3,7–10]. Surgical as well as thrombolytic therapy carries associated risks. Anticoagulant therapy could at least avoid the surgical risk but implies the risk of recurrent, potentially life-threatening embolization [8,9]. Resolution of small mobile aortic thrombi, sized 0.5–3 cm, under anticoagulant therapy has been documented by TEE and computed tomography [2,8,9]. In two further cases with large aortic thrombi, sized 10 cm and 15 cm, anticoagulant therapy has been performed without embolic events [4]. Whether in our case the resolution of the thrombus was due to anticoagulant therapy, to silent re-embolization or spontaneous fibrinolysis could not be definitively decided, but we assume that the anticoagulant therapy permitted the endogenous fibrinolytic system to dissolve the thrombus. Intravenous heparinization in our patient necessitated a prolonged hospitalization of 4 weeks. Only after that period, when TEE showed a significant decrease in the size of the thrombus, a change to oral anticoagulation and dismissal of the patient deemed justified.

Since large, mobile aortic thrombi without aortic atheromatosis resolve during strong and well-controlled anticoagulant therapy, this case may provide impetus to use anticoagulant therapy for similar situations. However, anticoagulation in a patient with mobile aortic thrombi should only be performed in hospitals equipped with vascular surgery. Special care should be taken to maintain the level of anticoagulation in the target range, to observe the patient closely for possible embolic events, and to hospitalize the patient until significant reduction in the size of the thrombus can be documented.

References

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