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Asymptomatic aortic mural thrombus in a minimally atherosclerotic vessel

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Abstract

Aortic mural thrombi in a normal (non-aneurysmal or minimally atherosclerotic) vessel are an uncommon condition. They are usually located in the descending aorta and, less frequently, in the aortic arch or in the abdominal aorta. The typical clinical presentation is the appearance of symptoms/signs of peripheral arterial embolization, such as lower limb or visceral ischaemia, but these can also be accidentally found in asymptomatic patients. We report the case of a 40-year old man with untreated hypertension and dyslipidaemia admitted to hospital for atypical chest pain associated with an elevation in high-sensitivity troponin T with normal creatine kinase isoenzyme MB creatine kinase isoenzyme. Elektrokardiogram (EKG) and transthoracic echocardiography were non-diagnostic; in order to exclude an aortic dissection, a gated chest computed tomography was performed and showed an aortic thrombus on a minimally atherosclerotic wall. Then, a transoesophageal echocardiography confirmed an aortic floating thrombus (7 × 4 mm). Cardiac surgeons advised against surgery and therapy with antiplatelet, low molecular weight heparin, β -blocker, antihypertensive and lipid-lowering drugs was initiated. A complete resolution of the thrombus was observed at the 12-day tomographic control.

Keywords: Aortic thrombus • Transoesophageal echocardiography • Thoracic aorta

INTRODUCTION

Aortic mural thrombi in a normal (non-aneurysmal and non- or minimally atherosclerotic) vessel are an uncommon condition [1–3]. They are usually located in the descending aorta and, less frequently, in the aortic arch or in the abdominal aorta. The typical clinical presentation is with the symptoms/sign of peripheral arterial embolization such as lower limbs or visceral ischaemia or, depending on the affected aortic segment, upper extremity ischaemia or stroke [1].

CASE REPORT

A 40-year old man with atypical chest pain that radiated to the left arm and lasting for 3 days was referred to our Hospital emergency department. He is a non-smoker and his past medical history included untreated hypertension and dyslipidaemia. At admission, his blood pressure was 150/95 mmHg, heart rate 96 bpm and his physical examination was normal; particularly, all peripheral pulses were present and there was no carotid or subclavian bruit. On laboratory evaluation, an elevation in high-sensitivity troponin T (334 ng/l) with normal creatine kinase isoenzyme MB creatine kinase isoenzyme was founded. Elektrokardiogram (EKG) and transthoracic echocardiography were non-diagnostic, while a gated

chest computed tomography showed an aortic thrombus on a minimally atherosclerotic wall (Fig. 1C and D). A transoesophageal echocardiography confirmed an aortic floating thrombus of 7 × 4 mm (Fig. 1A and B; Videos 1 and 2). Cardiovascular surgery was not indicated; so, therapy with antiaggregant, low molecular weight heparin, β -blocker, antihypertensive and lipid-lowering drugs was initiated. A tomographic control 12 days later showed the complete resolution of the thrombus (Fig. 1E and F).

Associated known conditions were excluded: a coronary computed tomography angiography showed the absence of plaque occlusion; hypercoagulable, inflammatory and infectious causes have been evaluated and ruled out on biochemical assessment (see Table 1 for coagulation details). Patient had no history of recent or past trauma as well as no familiar history of aortopathies or vascular diseases. Finally, he had no history of skeletal or lens problems that could be associated with congenital syndromes and aortic alterations.

DISCUSSION

Aortic floating thrombus in a normal or minimally atherosclerotic vessel is an uncommon condition in the absence of hypercoagulable, inflammatory, infective or familial aortic diseases [1–3]. The typical clinical presentation is with the symptoms/sign of

Figure 1: Aortic thrombus on a minimally atherosclerotic wall on transoesophageal echocardiography; longitudinal view (A) and transversal view (B). The same finding on chest computer tomography: coronal view (C) and sagittal view (D). Panels E and F show the resolution of thrombus on chest computed tomography on coronal and sagittal views, respectively.

Video 1: Aortic thrombus on a minimally atherosclerotic wall on transoesophageal echocardiography; transversal view.

Video 2: Aortic thrombus on a minimally atherosclerotic wall on transoesophageal echocardiography; longitudinal view.

peripheral arterial embolization, but it can also be found in asymptomatic patients. In this case, it has been found during the imaging work-up carried out in order to exclude an aortic dissection. It has been shown both on chest computer tomography and transoesophageal echocardiography, which are useful and complementary tools, based on the affected aortic site.

Associated conditions that promote the thrombus formation are the presence of malignancies, hypercoagulable disorders, iatrogenic causes (such as aortic catheterization), infective and genetic disorders of the aortic wall [1]. All these conditions have to be excluded before the diagnosis of spontaneous thrombus, such as in the presented case. It should be noted, however, that the

Table 1: Coagulation and fibrinolysis laboratory data

Variables	Value	Reference range
PT (s)	10.4	10–13.5
PT (INR)	1.00	0.90–1.14
aPTT (s)	23.5	20–35
APTT (ratio)	0.86	0.83–1.18
Platelet ($\times 10^9/l$)	260	140–440
Fibrinogen (mg/dl)	306	175–400
Plasminogen (%)	85	70–130
Antitrombin III (%)	101	80–130
C protein (%)	99	65–132
S protein (%)	110	60–150
Antiphospholipid antibody	Negative	Negative
Lupus anticoagulant	Negative	Negative
Factor V Leiden	Negative	Negative

aPTT: activated partial thromboplastin time; PT: prothrombin time; INR: International Normalized Ratio.

thrombus was close to the ductus arteriosus. In that area, the arterial wall can show quite often an uneven surface or local calcifications and these could be, at least in part, the origin for thrombi formation. A limitation of this case report is that, as the vascular surgery was not necessary, we cannot surely exclude a local cause as the origin of the thrombi formation.

Owing to its rarity, actually no definitive consensus on treatment exists. In patients with embolic complications anticoagulant therapy is indicated, with or without a subsequent surgical approach [1]. When feasible, endovascular coverage of the aortic thrombus with stents appears to be an effective and safe procedure but, in case of atypical localization or a very large thrombus, it could be better managed with vascular thromboembolectomy, although it has been associated with significant morbidity and mortality. In our asymptomatic patient, the thrombus was rather small, thus an endovascular or a surgical approach would not have been indicated. Two previous reports of aortic thrombus in the absence of peripheral embolism have shown a good outcome with anticoagulation therapy without surgical procedures [4, 5]. We can speculate on the possibility that such a small thrombus may resolve even independently of anticoagulant therapy.

CONCLUSIONS

In conclusion, the spread of prompt imaging in aortic disease has led to the diagnosis of an asymptomatic aortic thrombus for which management is more difficult due to its rarity. Conservative treatment led to a rapid resolution of the thrombus and an uneventful hospital discharge.

Conflict of interest: none declared.

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eComment. An unusual cause of aortic mural thrombus in non-atherosclerotic vessel

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We have read the case report of Maloberti *et al.* with great interest [1]. The authors mentioned that aortic mural thrombus in a normal (non-aneurysmal or minimally atherosclerotic) vessel was an uncommon condition. From this point of view, etiologies of an aortic mural thrombus in a non-aneurysmal vessel have been particularly evaluated in their patient. However, no specific etiologic factor has been detected. As a conservative treatment, antiplatelet therapy, low molecular weight heparin and beta-blocker, antihypertensive and lipid-lowering drugs have been initiated. A complete resolution of the thrombus has been observed on control tomography angiography at the 12th day. Related with this case, it would be better to discuss the arterial findings of Behcet's disease, which is highly prevalent among countries around the Mediterranean Sea, e.g. Turkey, Algeria, etc. Behcet's disease is a systemic inflammatory vasculitis of which the etiology is still unidentified and clinical presentation is heterogeneous with multisystemic involvement. Its clinical characteristics are oral and genital ulcers and may be accompanied with reduced bone mineral density, arthritis, cardiovascular, and neurological, gastrointestinal and vascular findings [2]. Prevalence of vascular findings may range between 1 and 38% according to series. Vasculitic lesions can be detected in all vessels ranging from arterioles to great arteries or from venules to great veins. Frequency of venous involvement is higher than arterial involvement. However neutrophilic or monocytic inflammation which may involve the great, middle and small arteries may clinically be more significant. Because vasculitis mediated by cellular infiltration or immune reaction promotes thrombosis by producing endothelial dysfunction. This endovascular and perivascular inflammation may have consequences with thrombosis and aneurysm in arterial system. Involvement of the carotid artery, pulmonary and aorta, iliac, femoral, and popliteal arteries is more frequent [3].

In conclusion, Behcet's disease should be reminded when the etiology of mural thrombus in non-atherosclerotic arteries has been evaluated in cases from countries around the Mediterranean Sea.

Conflict of interest: none declared.

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eComment. Mural thrombus in normal appearing aorta: Unfinished saga in uncharted waters

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We read with great interest the recent publication of Maloberti and colleagues [1] who presented a rare case of thoracic aorta mural thrombosis (TAMT) in a patient without predisposing factors who was treated conservatively with excellent short-term results (no long-term follow-up presented). By taking advantage of this case, we aim to answer some scientific queries born on the subject.

TAMT in non-atherosclerotic background is indeed rare (0.8–9%) with potential catastrophic consequences due to the recognized likelihood of visceral and peripheral embolization [2]. Due to its rarity - underdiagnosis or true low prevalence - and heterogeneity of causes, there is still controversy about the appropriate treatment algorithm, since it does not represent a primary disease, but epiphenomenon of other underlying disorders expressed with the same identification mark. So individual and careful evaluation of each case tips the scales against invasive or conservative treatment.

It seems that the patient in this case was not asymptomatic since she developed angina-like, though unexplained, symptoms that could raise the suspicion of a potential unusual trigger. Thus, in cases of embolic episodes (central or peripheral) in young patients without risk factors (atherosclerosis, smoking, coagulopathy, vasculitis, connective tissue disease, trauma and inflammatory bowel disease) [3], TAMT should gain ground in the differential diagnosis. By taking advantage of the imaging improvements (transoesophageal echography, magnetic resonance), the diagnosis could be easily reached without the need of invasive diagnostic procedures.

At the present time, there are many treatments available to our armamentarium, and treatment by anticoagulants is the cornerstone of primary approach. Surgery (open or endovascular) and thrombolysis are used in cases when medical treatment has failed or was contraindicated. The efficacy of antiplatelets on disease recurrence is debatable since there are equivocal results in the literature [2, 4]. Of interest would be the evaluation of NOACs in this setting.

The main disadvantage of conservative treatment seems to be its high recurrence rate that fluctuates from 26.4%–50% [2, 3, 5] and a trend towards higher incidence of limb loss and complications [3] whereas in the surgical group, recurrence is much lower (5.7%) [3]. High risk for recurrence are these cases of TAMT that present with persistent symptoms, are located in aortic arch or ascending aorta and have concomitant atherosclerotic background [3]. Mortality rate of surgery fluctuates from 2.6%–5.7% [2, 3] whereas patients in anticoagulants reach a mortality rate of 6.2% [3].

The decision which treatment modality should be chosen is based on the location, the mobility and the morphology of the thrombus as well as the persistence of symptoms under anticoagulants and the high risk of recurrence. As in the presented case, pedunculated fibrinocruoric thrombus floating in the aortic lumen is the most common morphology [2] and is correlated with increased embolic episodes [4]. The size of the thrombus should not be evaluated as the main criterion for the choice of treatment modality [3].

All in all, treatment of TAMT is a dynamic thought debatable scientific query. It seems that life-long anticoagulation is compulsory with surgery as primary approach being indicated in cases of young, symptomatic patients with high risk of recurrence. New meta-analyses and the evaluation of the role of NOACs and endografts in treatment, will shed some light to the uncharted waters.

Conflict of interest: none declared.

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