Massive thrombus in the aortic arch: a 59-year-old lady with an unknown familial predisposition to vascular thrombosis

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The following is an account of a 58-year-old lady who presented to our institution with two episodes of acute limb ischemia: one upper, which required embolectomy; and one lower, which required a below knee amputation. We subsequently performed transesophageal echocardiography (TOE), which revealed very large mobile echogenic masses adherent to the wall of her aortic arch, described below.

KEYWORDS
Aortic arch;
Thrombosis;
Embolisation;
Amputation

This 58-year-old lady was referred from a nearby Regional Hospital to the vascular surgeons in our institution with an acutely ischemic left upper limb. She subsequently underwent emergent removal of a brachial and axillary artery embolus, confirmed by histology. An intra-operative angiogram confirmed her native vessel was healthy. Three months previously, she had presented to the referral hospital with an acutely ischemic left lower limb. Despite popliteal embolectomy and bypass procedure she eventually required an above knee amputation. Considering that she presented with an acute ischemic limb on two separate occasions, and her native brachial and axillary vessels were healthy, it was reasonably suspected that the origin of the emboli was her heart. She was referred to the cardiology team for advice.

A trans-thoracic echocardiogram (TTE) was technically difficult due to body habitus, but revealed mild LVH with good systolic function, mildly enlarged left atrium, with normal Doppler velocities through the aortic and mitral valves. No intramural thrombi were seen, but the study was sub-optimal. A 48 h holter rhythm monitor revealed sinus rhythm throughout. We consulted the hematology service and they suggested a full thrombophilia screen.

The complete thrombophilia screen including factor II prothrombin gene, factor V leiden gene, lupus anticoagulant (dilute Russell viper venom test [DRVVT]), antiphospholipid antibodies [including cardiolipin IgG antibodies and β2-glycoprotein antibodies]), antinuclear antibodies, anti-mitochondrial antibodies, smooth muscle cell antibodies, reticulin antibodies, anti-LKM antibodies, microsomal agglutination antibodies, Actin FSL, activated protein C resistance (APCR), was negative. Platelet aggregation assay and platelet function testing were normal. Homocysteine and Methionine levels were normal. Renal, liver and thyroid function were normal. Hemoglobin and white cell count were normal, platelets were mildly elevated. A screen for occult malignancy was negative: tumor markers were negative; bilateral mammograms revealed no evidence of malignancy, or any suspicious lesions; CT thorax/abdomen/pelvis revealed no evidence of malignancy.

The patient had 14 siblings in all: eight brothers, one of whom asphyxiated as a child, one of whom was a stillbirth, and one who died aged 69. She had six sisters, all alive. The patient’s family history revealed a significant history of deep venous thrombosis (DVT): one brother in his 60s who died at 69, two sisters at ages 56 and 62 had a DVT, as did her daughter at age 34. Three other brothers also had a cerebrovascular accident (CVA) aged 51, 52, and 65. The brother aged 52 also had an MI in his late forties. One other sister aged 64 had two CVAs at 60 years of age. Thus, the inheritance pattern if any would seem to be autosomal dominant, perhaps with incomplete penetrance.

We proceeded to transesophageal echocardiogram (TOE) to exclude potential sources of embolism: we out-rulled patent foramen ovale (PFO) with an agitated saline (‘bubble’) study, and we imaged the proximal aorta. This revealed mobile echogenic masses in the descending aorta beginning at 30 cm from the incisors and extending proximally to the arch. These masses were homogenous in

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Supplementary material

Supplementary material associated with this article can be found in the online version.

References


