Endovascular treatment for mobile thrombus of the thoracic aorta

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Abstract
Detection levels of mobile thrombus of the thoracic aorta have greatly increased after any embolic event. Although the indication for treatment remains controversial, there is a growing interest about the etiopathogenesis of this rare entity and to define proper diagnostic and therapeutic approaches. We present a case of mobile thrombus of the thoracic aorta causing recurrent peripheral emboli managed with endovascular stent graft.

Keywords: Mobile thrombus; Thoracic aorta; Stent graft

1. Introduction
Thoracic aortic mobile mural thrombus (TAMT) is a rare pathology and a potential source of cerebral, visceral, and peripheral emboli [1]. At the present time, management of TAMT remains unclear [2,3]. We present a case of TAMT, causing peripheral embolic occlusion, successfully managed with endovascular stent graft (SG) as the primary therapeutic approach.

2. Case report
A 56-year-old man was referred to our department after having been initially surgically treated at another surgical unit for an urgent embolectomy following acute left upper limb ischemia. He was obese (BMI > 31) and had a silent cardiologic medical history. Six months before the initial ischemic event he was diagnosed to have thymic hyperplasia associated to myasthenia gravis. Medical therapy included diuretic, steroid (methylprednisolone), and pyridostigmine.

Chest X-rays, ECG, and transthoracic 2D echocardiogram were negative. A computed tomography angiography revealed a free-floating thrombus originating from the inner curve of the isthmic aorta extending for 12 cm, that occluded half of the lumen of the descending aorta (Fig. 1). No penetrating ulcer or atherosclerotic plaques were noted along the entire aorta.

Following a new episode of embolization to the left lower limb, we decided to perform an urgent intervention to prevent a new, potentially massive visceral embolization. A femoropopliteal embolectomy was performed to remove the thromboemboli occluding the profunda and superficial femoral arteries. During the same intervention, a ‘spy’ guidewire was placed in the aortic arch through a percutaneous left transbrachial approach, with particular care taken to avoid dislodging the thrombus. The thrombus was excluded with a 28 mm × 10 cm thoracic aortic SG (TAG®-W. L. Gore & Ass., Flagstaff, AZ, USA). The final angiogram confirmed the exclusion of the thrombus and the patency of the left subclavian artery (Fig. 2A–B). The postoperative course was uneventful and he was discharged 5 days later on warfarin therapy. Histologic examination of the intraoperative femoral thrombus revealed a fibrin-rich red thrombus (Fig. 2C); thrombophilia was confirmed as several screening tests were positive; we recorded alterations of the C-protein, hyperhomocysteinemia, MTHFR homozygous, C-reactive protein, positive anticardiolipin IgM, increased anti-β2-glycoprotein-1 IgG, and IgM. He was last seen 6 months later, and recurrent peripheral embolization or mobile thrombi at another site were never observed again (Fig. 2D–E).

3. Discussion
TAMT has rarely been mentioned in the literature, and described as primary, spontaneous, non-occlusive aortic thrombus [3]; Williams et al. [4] in 1981 first described a mobile thrombus in the aorta [7]. More recently, Bowdish et al. [1] suggested using the term TAMT, which defined aortic thrombi that develop in the absence of pre-existing aortic
disease [1]. Our experience confirmed the previous published data that reported the descending aorta (44%), with a predilection for the aortic isthmus, as the most common location of TAMT [1—5].

Pathogenesis of the TAMT has not been defined yet and considered multifactorial [3,5,6]. Bowdish et al. reported 32 patients who underwent an evaluation for an existing coagulation disorder, noting that nearly 40% had hypercoagulable conditions identified by screening tests [1]. In the present case, serology revealed an alteration of several coagulative tests, similar to those disorders reported in the literature. In addition, steroids might cause direct endothelial damage, as speculated by some authors, to promote the thrombosis of large arterial vessels. Hahn et al. [3] presented an interesting group of steroid-treated patients who presented with distal emboli originated from a TAMT; the present patient started prednisone after being diagnosed to have a myasthenia gravis.

TAMT may be an incidental finding: in the series of Gagliardi et al. [7], the authors found that lower extremities were the most common locations (60%) of distal emboli, most of them (80%) originating from a TAMT in the abdominal aorta, and 20% from the descending thoracic aorta. Also in our experience, recurrent episode of peripheral emboli was the main reason to further investigate an otherwise undetected source of embolization.

Therapy for these patients should focus on preventing the evolution of mobile lesions and provide protection against the embolic potential of these lesions [3,6]. Several approaches have been used: because of the high risk of recurrence, and the mortality/morbidity rates of open repair (thrombectomy with/without graft replacement), medical therapy was suggested to be the first alternative option to treat TAMT, especially for asymptomatic cases [6]. Surgery was considered when anticoagulation therapy failed: this occurred in more than 50% of the cases reported by Choukroun et al. [6]. In addition, complete resolution with anticoagulation has been described in a small number of patients [1,6].

In contrast, Goueffic et al. [8] treated 38 patients by various open surgical techniques with contained mortality of 2.6% but recommended open surgery only in case of recurrent embolic events. Exclusion by an endovascular SG has been recently suggested as an alternative also for TAMT treatment. Similar to open surgery, SG can exclude the thrombus, treats the potential underlying cause of the thrombotic lesion by covering the atherosclerotic aortic wall, and allows the combination of the thoracic endovascular procedure with a peripheral embolectomy through the same remote access. Criado et al. [9] used a SG to exclude a TAMT, after a failed open thrombectomy; previously, Fuglistaler et al. [10] used a SG to treat a TAMT of the distal aortic arch. Our experience is consistent with these previous cases as the patient was last seen 6 months later with no recurrent signs of TAMT or symptoms of embolization.

The intraoperative specimens reported in literature described different types of thrombi; frequently histology revealed the presence of an organized thrombus with multiple fibroblasts [5]. Concerns might arise that an endovascular approach may not allow for histology; however, the thrombus extracted from the femoral arteries in the present case revealed a fibrin-rich red thrombus, and the presence of malignant cells were not detected. In addition, CT angiography failed to detect a potential malignant involvement of the aorta.

4. Conclusion

Mobile thrombus of the thoracic aorta is a rare pathology; available literature shows a lack of agreement about diagnostic and therapeutic approaches. SG exclusion could
be a viable alternative primary treatment for a symptomatic mobile thrombus in the thoracic aorta.

References