Protruding aortic arch thrombus: Treatment with minimally invasive surgical approach

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Background: Protruding aortic arch thrombus is associated clinically with life-threatening emboli. Definitive treatment for aortic arch thrombus removal has demanded complicated vascular surgical procedures, with high morbidity and mortality.

Methods and results: Transesophageal echocardiography (TEE) enabled diagnosis of a protruding thrombus at the aortic arch in 5 patients, and a simultaneous lesion in the descending aorta in 1 patient. Four patients had visceral emboli, coinciding with peripheral emboli in 2 patients, and the fifth patient had peripheral and cerebral emboli. One patient had had ischemic stroke and femoral emboli a few months previously. Mean patient age was 51 years. None had clinical evidence of coronary or peripheral atherosclerotic occlusive disease. Risk factors included hypertension (n = 2), smoking (n = 4), and preexisting thrombophilia (n = 4). Five patients underwent TEE-guided aortic balloon thrombectomy from the arch with a 14F occluding balloon catheter. One patient also underwent balloon thrombectomy from the descending aorta with a 14F Foley catheter. Access into the aorta was obtained through the iliac artery (n = 4) during laparotomy because of visceral ischemia, or through the transfemoral approach (n = 2). Previous procedures included superior mesenteric embolectomy (n = 3), segmental bowel resection (n = 1), splenectomy (n = 1), and peripheral arterial embolectomy (n = 3). Real-time intraoperative TEE enabled visualization of the protruding thrombus and assisted with maneuvering of the balloon catheter. At completion peripheral thrombectomy thrombus material was retrieved in 4 patients. Postoperatively there were no clinically proved new procedure-related visceral emboli, and all patients received anticoagulant therapy thereafter. Follow-up TEE within 2 weeks and up to 7 years revealed no recurrent aortic arch thrombus.

Conclusions: TEE-guided aortic balloon thrombectomy used in 6 procedures was effectively completed without visceral or peripheral ischemic complications. It enabled removal of the life-threatening source of emboli from the proximal aorta, thereby averting the need of major aortic surgery. (J Vasc Surg 2004;40:1083-8.)

Protruding aortic arch thrombus is a mobile lesion that arises from the luminal surface of the proximal aorta. It disintegrates, and sheds arterial emboli, manifesting clinically as cerebral, visceral, and peripheral ischemic events. Aortic arch thrombus is a unique pathologic entity, readily diagnosed with transesophageal echocardiography (TEE). However, the therapeutic approach to this lesion is still controversial. In most cases cited in the literature, major surgical procedures were performed to excise the protruding thrombus. These frequently included segmental resection of the involved aortic tissue by means of thoracotomy. We present a series of 5 patients with aortic arch thrombus treated with TEE-guided aortic balloon thrombectomy through a peripheral approach, averting the complications of major vascular surgery.

METHODS

Five consecutive patients with aortic arch thrombus, all with life-threatening embolic phenomena, received treatment at the Sheba Medical Center, Tel Hashomer, between 1997 and 2003. Demographic data, risk factors, initial symptoms, and method of treatment are presented in the Table.

Medical history in one patient was noteworthy for ischemic stroke and peripheral arterial embolic occlusion necessitating bilateral femoral embolectomy 6 and 4 months previously, respectively. No patients in the series had clinical evidence of coronary or peripheral chronic arterial occlusive disease. At admission, 4 patients had visceral ischemia, including 3 mesenteric emboli, 2 splenic emboli, and 1 renal embolus (Fig 1, A to C). Visceral artery embolic occlusion coincided with femoral emboli in 2 patients. In another patient peripheral embolization to the brachial artery was followed by embolic stroke 2 days later. One patient with splenic infarcts diagnosed at computed tomography angiography had amidodarone-induced thyrotoxicosis. TEE was the method used for diagnosis in all patients, and surgery was initiated within 6 hours of diagnosis. In 4 patients surgical treatment was initiated with laparotomy to address visceral ischemia, enabling splenectomy (n = 1), mesenteric artery embolectomy (n = 3), and segmental small bowel resection (n = 1). In brief, the

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A technical approach to aortic balloon thrombectomy included exposure of the aortic bifurcation and proximal common iliac arteries. After systemic heparinization the left common iliac artery was clamped at its origin, and the right common iliac artery was clamped a few centimeters beyond its origin, followed by a transverse arteriotomy 1 cm from the aortic bifurcation. A 0.035-inch guide wire was initially introduced and advanced under TEE guidance into the aortic arch. The aortic balloon catheter (34-mm venous Stop Flow balloon catheter; Pfm, Koln, Germany) was then introduced over the wire through this arteriotomy, and was used in all 5 patients with aortic arch thrombus. A simultaneous protruding thrombus emerging off the distal descending aorta in one patient was dislodged in a similar fashion, with a 14F silicon-coated Foley catheter, without a guide wire. Bleeding from the aorta was controlled with a vessel loop or with manual compression of the proximal iliac artery, which was performed throughout the process of introduction and withdrawal of the balloon catheter. The entire procedure lasted approximately 1 to 3 minutes, and involved insignificant blood loss. The procedure was effective in all patients. The mobile thrombus, as the source of emboli, was dislodged from its anchorage in the aortic wall, leaving a negligible thrombus remnant. The dislodged mass was instantly delivered for retrieval with peripheral completion Fogarty thrombectomy. It should be noted that although no endovascular precautions were exercised to prevent accidental visceral embolization during aortic thrombus dislodgement, no such event was clinically recorded (data confirmed later at computed tomography angiography).

### RESULTS

In all patients TEE demonstrated a grade II\(^3\) 2 to 3-mm shallow aortic plaque at the site of anchorage for the stalked, mobile 1.5 to 2.3-cm polyp-like lesion. The lesion was located at the distal arch in 4 patients (isthmus), and in the mid-arch, facing the left common carotid artery orifice, in 1 patient. A simultaneous lesion at the distal descending aorta was found in 1 patient. Except for the localized shallow atherosclerotic lesion, no other plaques were visualized with TEE in the proximal aorta in any patients. TEE-guided balloon thrombectomy from the aortic arch was performed in 5 patients, and from the descending aorta in 1 patient (Fig 2). The procedure was effective in all patients. The mobile thrombus, as the source of emboli, was dislodged from its anchorage in the aortic wall, leaving a negligible thrombus remnant. The dislodged mass was instantly delivered for retrieval with peripheral completion Fogarty thrombectomy. It should be noted that although no endovascular precautions were exercised to prevent accidental visceral embolization during aortic thrombus dislodgement, no such event was clinically recorded (data confirmed later at computed tomography angiography).
Thrombus material was collected in 4 procedures. However, in 2 procedures transfemoral completion thrombectomy failed to retrieve any thrombus, suggesting that this material may have relocated within the internal iliac arteries. Histopathologic examination of the retrieved material revealed organized thrombus devoid of any atheromatous material in all analyses (Fig 1, D). All patients underwent full workup of the coagulation system, which revealed preexisting thrombophilia in 4 patients, including activated protein C resistance (heterozygous) in 1 patient, factor II mutation (heterozygous) in 1 patient, and hyperhomocysteinemia (5,10-methylene tetrahydrofolate reductase, homozygous) in 2 patients. The fifth patient, who had a stormy course of amiodarone-induced thyrotoxicosis, exhibited an inflammatory state, another suggested prothrombotic tendency.

There were no further embolic events in the immediate postoperative period or throughout follow-up. Periodic TEE assessments for up to 7 years revealed irregularity of the luminal surface at the site of the treated protruding thrombus. However, there was no recurrence of protruding thrombosis at the aortic arch or at any other site in the proximal aorta. During follow-up 2 patients died, of sepsis related to total thyroidectomy 6 weeks later and metastatic breast carcinoma 2 years postoperatively.

DISCUSSION

The pathogenesis of protruding aortic arch thrombus is obscure. Assumed analogous arterial conditions such as acute coronary syndrome or cerebrovascular ischemia as related to symptomatic carotid plaque, demonstrate the association between atherosclerosis and local thrombosis. Therefore, it is suggested that aortic arch thrombus is another manifestation of the same pathologic entity, and acute rupture or fissuring of a shallow aortic (lipid-rich) vulnerable plaque may initiate the process, with subsequent instant local thrombosis. Among risk factors for acute arterial syndromes at the coronary, carotid, or aortic arteries, systemic inflammation, as elicited by smoking, and prothrombotic states, such as preexisting thrombophilia, seem to have a key role. The presence of these systemic factors may further support the current concept of the “vulnerable patient” prone to plaque rupture. In our series, most patients (4 of 5) were heavy cigarette smokers, and 1 patient sustained amiodarone-induced thyrotoxicosis. In addition, preexisting thrombophilia was diagnosed in 4 patients. It is noteworthy that thrombophilias such as activated protein C resistance (heterozygous) and factor II mutation (heterozygous) have been considered in the past as risk factors for venous thrombogenesis. However, combined with the assumed plaque rupture in our patients, these thrombophilias may have contributed to arterial thrombosis.

Within 7 years of follow-up (1997-2003) the prevalence of arterial emboli directly related to aortic arch protruding thrombus in our institution has been approximately 3% of the total number of spontaneous visceral and peripheral arterial emboli. This is an unexpectedly high percentage, and suggests that aortic arch protruding thrombus may be a significant source of emboli, in light of gradually decreasing rates of emboli of cardiac origin.

All patients in our series had life-threatening emboli, mainly affecting the splanchnic arteries. These critical circumstances warrant early diagnosis and treatment, including reversal of ischemia and removal of the source of emboli. However, diagnosis of aortic arch thrombus was delayed in 2 patients, because TEE was not part of the diagnostic protocol. Furthermore, once performed, TEE enabled accurate diagnosis and directly assisted in definitive treatment. Our data advocate expanded use of TEE to all cases of arterial emboli of unclear
source, and also in relatively young patients with peripheral or visceral emboli.\textsuperscript{14}

From case reports and our own experience, it is clear that the source of emboli should be approached surgically and without undue delay. Among the methods attempted for treatment of aortic arch thrombus, systemic heparinization was partially effective in preventing further emboli in a large series of 23 patients.\textsuperscript{2} In another series of 9 patients intravenously administered heparin may have contributed to resolution of aortic arch thrombus in 4 patients.\textsuperscript{15} However, in both series surgery was necessary in most patients to remove the source of emboli. Fibrinolysis with rt-PA was utilized successfully in one reported case.\textsuperscript{16} However, in our view, this modality may dissolve the thin stalk of anchorage, and bring about uncontrolled dislodgement of the thrombus mass. Endovascular stent grafting has recently been used to contain a recurrent aortic protruding thrombus.\textsuperscript{17} The treatment of choice in most cited clinical series involved surgical excision of the affected aortic wall segment via aortotomy, with major cardiovascular surgical procedures\textsuperscript{15,18} associated with significant morbidity and mortality. In contrast, our surgical approach is minimally invasive, with limited surgical trauma. It involves visually controlled dislodgement of the aortic thrombus from the arch, followed by peripheral completion thrombectomy. Moreover, the aortic balloon thrombectomy was performed in most instances sequentially with surgical procedures to treat embolic occlusion of splanchnic and peripheral arteries. It should be noted that no further visceral emboli attributable to dislodgement of the protruding thrombus were recorded. This may be explained in part by the observation that the mobile thrombus is disconnected from its stalk as a single sizeable mass (as visualized at TEE), thereby reducing the likelihood of new emboli in smaller visceral arteries.

Our limited experience in treating 6 cases of aortic arch protruding thrombus suggests that the abdominal approach may be preferable to the transfemoral approach for the following reasons. It allows initial assessment of the visceral vasculature, and subsequently its reevaluation immediately after aortic thrombectomy, thereby addressing the critical issue of potential embolization into visceral

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Fig 1. Clinical findings in patient 1. A, Proximal aortogram demonstrates normal looking, regular, smooth luminal surface of the aorta. B, Abdominal computed tomography scan demonstrates total infarction of the spleen (arrowhead). C, Selective superior mesenteric arteriogram demonstrates embolic occlusion of the vessel a few centimeters from its origin (arrowhead). D, Histologic analysis of retrieved protruding thrombus revealed a typical appearance of organized thrombus with multiple fibroblasts (arrowheads); Hematoxylin and eosin; original magnification \( \times400 \). Note that thrombus is devoid of any atheromatous material.
vessels. It prevents embolization of the relocated thrombus into the internal iliac arteries. It also provides better control and maneuverability of the guide wire and balloon catheter when introduced directly through the aortic bifurcation.

In summary, our limited experience in 5 patients, presenting 6 cases of symptomatic aortic protruding thrombus suggests that TEE-guided aortic balloon thrombectomy combined with completion peripheral thrombectomy may be effective in removing the source of life-threatening emboli. The safety of this procedure warrants thorough investigation in a larger series of patients before it can be generally adopted by the vascular community as a first-line therapy for aortic arch protruding thrombi.

Furthermore, prevention of aortic arch thrombus recurrence after successful treatment with balloon thrombectomy warrants life-long anticoagulation therapy to attenuate prothrombotic tendencies. In our view, although speculative, extrapolating from coronary and carotid atherosclerotic disease, it seems intuitively appropriate to administer statin drugs postoperatively in patients with aortic arch thrombus. This therapy may contribute to pacification of the aortic arch thrombus plaque of origin, as well as other potentially vulnerable aortic plaques.

REFERENCES


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