Contemporary management of symptomatic primary aortic mural thrombus

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Objective: **Primary aortic mural thrombus (PAMT) is an uncommon condition but an important source of noncardiogenic emboli with a difficult diagnosis and a high rate of complications, including high mortality. We report our experience of thromboembolic disease from PAMT and review its contemporary management.**

Methods: **Retrospective analysis of prospectively collected data of all patients who presented with acute occlusion of a limb or visceral vessels between January 2011 and September 2013 was performed.**

Results: **A total of 88 patients presented with acute occlusion of the extremities or visceral arteries. All underwent extensive evaluation for the possible source of the embolism. Of these 88 patients, 19 patients (mean age, 41.2 years; male:female ratio, 1:2.1) were found to have aortic mural thrombus as the source of distal embolism. Thrombus was located in the thoracic aorta in 10 patients, in the perivisceral aorta in three patients, and in the infrarenal aorta in six patients. Thrombus in the thoracic aorta was treated with stent grafts in four patients, bare metal stents in three patients, and anticoagulation alone in two patients. In the suprarenal abdominal aorta, all three patients underwent trapdoor aortic thrombectomy. Infrarenal aortic thrombus was managed by aortobifemoral embolectomy in two patients, aortic stenting in two patients, surgical thrombectomy in one patient, and anticoagulation alone in one patient. Successful treatment, defined as freedom from further embolic events or recurrence of thrombus, was achieved in 14 of 19 patients (76.4%) with a mean follow-up period of 16.2 months (range, 2-28 months). There were four (21%) thrombus-related deaths, all due to primary thromboembolic insults. One patient needed a below-knee amputation because of a recurrent thrombotic episode.**

Conclusions: **Symptomatic PAMT is an uncommon but important source of noncardiogenic embolus. It appears to occur more frequently in young women. Endovascular coverage of the aortic thrombus, when feasible, appears to be an effective and safe procedure with either stent grafts or closed-cell metal stents. When thrombus is located adjacent to visceral vessels, it should be managed with an open trapdoor thromboembolectomy. (J Vasc Surg 2014;60:1524-34.)**

Aortic mural thrombus in the absence of atherosclerotic occlusive or aneurysmal disease is a relatively uncommon entity. Although the cause of aortic thrombus is idiopathic in many patients, prothrombotic conditions have been described causing aortic mural thrombus (ie, malignant neoplasms, thrombocytosis, polycythemia, hypercoagulable states, primary tumors of the aorta[1-3]). Primary thrombosis of the aorta was first described in the late 1940s,[4] but the possibility of missing a cardiac embolic source was always a major issue because of inadequate imaging techniques at that time.[5] With the advent of two-dimensional transthoracic echocardiography and transesophageal echocardiography, the sensitivity of diagnosis for a cardiac source of embolism has increased to >95%.[6-8]

Because most published reports of this entity are limited to case reports or case series, there is no consensus on the ideal way of managing this problem. We present our experience of managing 19 patients with aortic mural thrombus in an otherwise normal aorta and review published literature and contemporary management strategies of aortic mural thrombus based on morphologic features of the thrombus as well as its anatomic location.

METHODS

As part of our institutional protocol, all patients who present with acute thrombosis of any arterial bed undergo thorough evaluation to determine the possible source of embolism (Fig 1).

We performed a retrospective analysis of hospital records for all patients who presented with symptomatic acute thrombosis of the upper limb, the lower limb, or visceral arterial beds between January 2011 and September 2013. Informed consent was obtained from all patients at the time of the procedure, and this retrospective study was approved by the Institutional Review Board. Eighty-eight patients with acute symptomatic thrombosis of various arterial beds were identified. Clinical presentation, imaging findings, prothrombotic workup, details of operative procedures, and follow-up data were reviewed.

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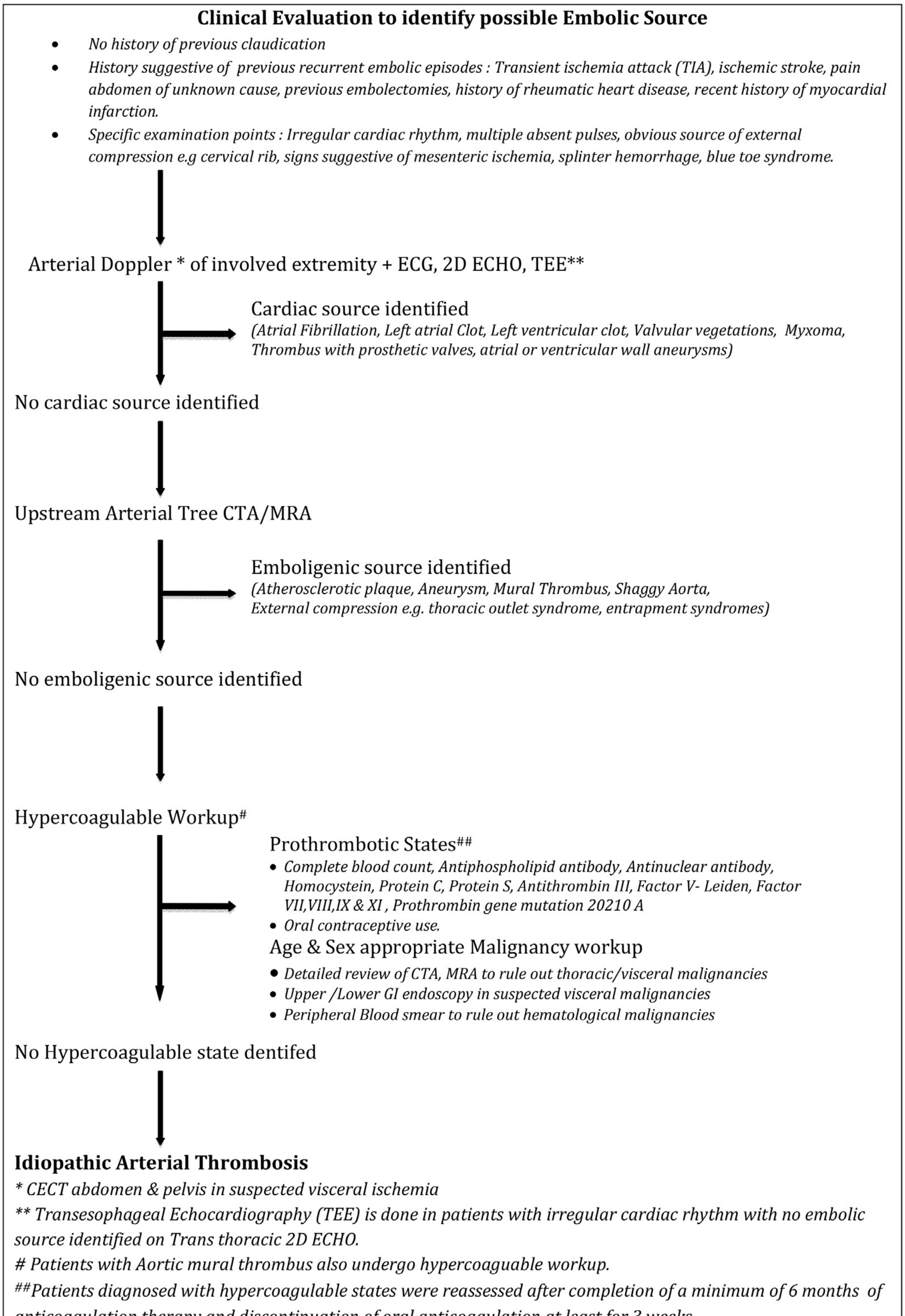


Fig 1. Institutional protocol for investigating acute arterial thrombosis. *CTA*, Computed tomography angiography; *ECG*, electrocardiography; *MRA*, magnetic resonance angiography.

Type I: Mural thrombus in ascending and arch of aorta (up to origin of left SCA)

Type Ia: Thrombus limited to ascending aorta

Type Ib: Ascending aortic thrombus extending into arch or aortic arch thrombus.

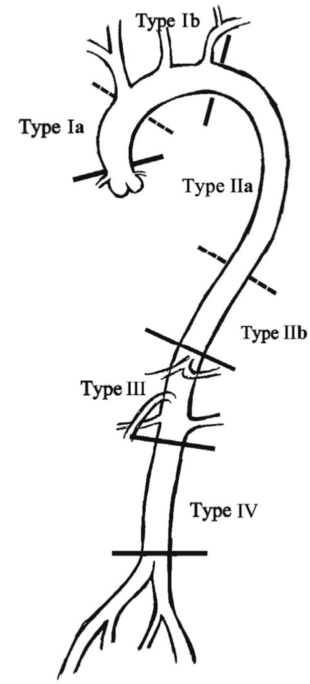
Type II: Mural thrombus descending thoracic aorta (distal to left subclavian artery up to coeliac artery.

Type IIa: DTA thrombus above T8

Type IIb: DTA & supracoeliac aorta thrombus (T8-L1)

Type III: Mural thrombus in aortic segment between coeliac artery to lowest renal artery

Type IV: Thrombus between lowest renal artery to aortic bifurcation.



****Based on morphology of thrombus, each type of thrombus is further classified as 'S','P' or 'O'***

S (Sessile): Eccentric or concentric thrombus with no free floating component

P (Pedunculated): Pedunculated thrombus (Mural thrombus attached to aorta proximally with a distal free floating segment of variable length.)

O (Occlusion): Complete thrombotic occlusion of aorta.

Fig 2. Classification of primary aortic mural thrombus (PAMT). *DTA*, Descending thoracic aorta; *SCA*, subclavian artery.

Primary aortic mural thrombus (PAMT) was defined as thrombus attached to the aortic wall in the absence of any atherosclerotic or aneurysmal disease in the aorta and a cardiac source of embolus. It could be either sessile or pedunculated (with a free-floating intraluminal segment).

Because management strategies have largely been dependent on anatomic location as well as on morphologic features of the thrombus, we classified PAMT into types I to IV (Fig 2).

All patients underwent follow-up with computed tomography (CT) aortography at 6 months. Any residual or new thrombus at a previous PAMT site as well as any new embolic event in visceral branches was looked for. Any new area of thrombus in the aorta or new embolic event on CT aortography was labeled recurrence.

RESULTS

A total of 88 patients with acute thrombosis of a limb or visceral vessels were evaluated between January 2011 and September 2013. All patients underwent evaluation for an embolic source per institutional protocol (Fig 1).

Nineteen (21.6%) patients (mean age, 41.2 years; male:female ratio, 1:2.1) had aortic mural thrombus within an otherwise normal aorta and a normal heart after thorough evaluation of the heart (echocardiography) and great vessels on CT or magnetic resonance angiography evaluation (Fig 1). None of these patients had any signs of either atherosclerotic change in the entire aorta or any aneurysmal dilation. Ten patients (52%) had arch and descending thoracic aortic thrombus; the remaining nine (48%) had thrombus in the abdominal aorta. Two patients (10.5%) had thrombus in the arch of the aorta opposite the left subclavian artery origin, eight patients (42%) had thrombus in the thoracic aorta distal to the left subclavian artery origin, three patients (15%) had thrombus in the perivisceral abdominal aorta, and six patients (31%) had thrombus in the infrarenal aorta.

Interestingly, only one patient (5.3%) was a smoker. Usual risk factors for atherosclerosis (smoking, hypertension, diabetes, coronary heart disease, dyslipidemia) were conspicuously absent in all patients.

Table I. Clinical presentations, imaging findings, management, and outcomes of patients with primary aortic mural thrombus (*PAMT*) type I and type II

No.	*Clinical presentation (SVS grade acute limb ischemia)*	*Aortic thrombus CTA/MRA*	*Distal vessel embolism*	*Management of aortic thrombus*	*Follow-up, months*	*6-month CT/ recurrent embolism/ any complication*
Type I PAMT (thrombus in ascending aorta/arch of aorta)						
1	Acute limb ischemia	Between left CCA and SCA origin, inner aspect of aorta; sessile	Left brachial artery	Anticoagulation alone	10	Thrombus decreased in size No recurrence
2	Acute limb Ischemia	At left SCA origin extending 1 cm in distal arch, outer aspect of aorta; sessile	Left brachial artery	Anticoagulation alone	2	Developed severe psoriasis Switched to LMWH
Type II PAMT (thrombus in DTA)						
1	Acute limb ischemia (IIa)	DTA, T4-T6; sessile	CFA, SFA	Covered stent TAG (W. L. Gore, Flagstaff, Ariz) 26 × 100 mm	20	Complete exclusion No recurrence
2	Acute limb ischemia (III) and ischemic pancreatitis	DTA, T4-T5; pedunculated	Celiac and SFA	Planned for stent grafting	Expired while awaiting aortic stenting	
3	Paraplegia with bilateral LL ischemia (III)	DTA, T8-T10; pedunculated and infrarenal aorta	Bilateral popliteal	Closed-cell bare metal stent[a] (24 × 70 mm) for DTA, aortoiliac embolectomy for infrarenal aortic thrombus	Expired in immediate postoperative period	
4	Acute limb ischemia (III)	DTA, T4-T6; pedunculated	Rt SFA and popliteal	Covered stent Endurant (Medtronic Cardiovascular, Santa Rosa, Calif) 24 × 80 mm	22	Complete exclusion No recurrence
5	Acute limb ischemia (IIb)	DTA, T9-T10; pedunculated	Rt SFA and popliteal	Closed-cell bare metal stent,[a] 24 × 70 mm	11	Complete exclusion No recurrence
6	Acute limb ischemia (IIa) and pain in abdomen	DTA, T6-T8; pedunculated	Splenic and liver infarct Bilateral SFA	Covered stent Endurant (Medtronic Cardiovascular, Santa Rosa, Calif), 20 × 80 mm	28	Complete exclusion No recurrence
7	Pain in abdomen	DTA, T8-T11; pedunculated	Splenic and liver infarct	Covered stent Zenith TX2 (Cook Medical, Bloomington, Ind), 22 × 80 mm	26	Complete exclusion No recurrence
8	Splenic infarction	Abdominal aorta, T12,L1; sessile	Celiac trunk, splenic artery	Closed-cell Bare metal stent,[a] 22 × 70 mm	11	Complete exclusion No recurrence

CCA, Common carotid artery; *CFA,* common femoral artery; *CT,* computed tomography; *CTA,* computed tomography angiography; *DTA,* descending thoracic aorta; *LMWH,* low-molecular-weight heparin; *MRA,* magnetic resonance angiography; *SCA,* subclavian artery; *SFA,* superficial femoral artery; *SVS,* Society for Vascular Surgery.
[a]Closed-cell bare metal stent used: Wallstent (Boston Scientific, Watertown, Mass).

According to the classification of PAMT, clinical details, characteristics, and management of aortic thrombus and follow-up details have been grouped for each type of PAMT in Tables I and II.

All patients diagnosed with hypercoagulable states were discharged with prescription of lifelong anticoagulation. Patients without hypercoagulable states and normal findings on follow-up CT aortography were switched to aspirin 75 mg/day after 6 months.

DISCUSSION

Our series of 19 patients of PAMT is the largest reported so far in the published literature. Our patients presented with visceral and distal embolization without

Table II. Clinical presentations, imaging findings, management, and outcomes of patients with primary aortic mural thrombus (*PAMT*) type III and type IV

No.	Presentation (SVS grade acute limb ischemia)	Aortic thrombus CTA/MRA	Distal vessel embolism	Management of aortic thrombus	Follow-up, months	6-month CT/recurrent embolism/any complication
Type III PAMT (thrombus in visceral segment of abdominal aorta)						
1	Paraplegia with acute renal failure	Abdominal aorta, L1-L4 Circumferential with pedunculated component	Right renal	Trapdoor aortic thrombectomy (supraceliac clamp time 25 minutes)	22	Minimal residual sessile thrombus in CT No recurrence Complete recovery from paraplegia and renal failure
2	Mesenteric ischemia	Abdominal aorta, T12-L1, sessile	SMA, hepatic and splenic artery (liver, gallbladder infarction with ileal gangrene)	Trapdoor aortic thrombectomy (supraceliac clamp time 19 minutes)	Multiple re-look laparotomies Expired on postoperative day 18	
3	Mesenteric ischemia	Abdominal aorta, L1-L3, sessile	SMA with ileal gangrene, right renal artery	Trapdoor aortic thrombectomy (supraceliac clamp time 22 minutes)	High-output jejunostomy, acute respiratory distress syndrome, acute renal failure Expired on postoperative day 7	
Type IV PAMT (thrombus in infrarenal aorta)						
1	Acute limb ischemia (IIb)	Infrarenal, sessile	Right popliteal	Aortobiiliac embolectomy for residual thrombus Closed-cell bare metal stent,[a] 18 × 90 mm	22	No recurrence
2	Acute limb ischemia R (III)	Infrarenal, sessile	Right popliteal	Anticoagulation	16	No recurrence
3	Acute limb ischemia R (II)	Infrarenal at bifurcation, sessile	Left popliteal	Aortic bifurcation thrombectomy and patch plasty	21	Recurrent thrombosis in popliteal segment; underwent BKA
4	Acute limb ischemia R (II)	Infrarenal aorta, pedunculated	Right SFA	Aortobiiliac embolectomy	12	No recurrence
5	Acute limb ischemia (IIb)	Infra renal aorta, pedunculated	Right SFA	Excluder limb (W. L. Gore, Flagstaff, Ariz), 16 × 12 × 70 mm	15	No recurrence
6	Acute limb ischemia B/L (IIa)	Aortic bifurcation, complete occlusion		Aortobiiliac embolectomy	6	Died of malignant disease

BKA, Below-knee amputation; *CT,* computed tomography; *CTA,* computed tomography angiography; *MRA,* magnetic resonance angiography; *SFA,* superficial femoral artery; *SMA,* superior mesenteric artery; *SVS,* Society for Vascular Surgery.
[a]Closed-cell bare metal stent used: Wallstent (Boston Scientific, Watertown, Mass).

any risk factors for cardiac disease, atherosclerosis, or aneurysm formation and did not demonstrate any atheromatous or aneurysmal change in the involved aorta. Management ranged from anticoagulation for sessile, small thrombus to endovascular stenting or stent grafting for large or nonvisceral aortic thrombus and open surgical aortic thrombectomy for visceral thoracoabdominal thrombus.

Review of published data on aortic mural thrombus until 2014[1,2,9-15] revealed fewer than 250 cases, the majority of which were single case reports of mural aortic thrombus.[1] In 10,671 consecutive autopsies, Machleder et al reported an incidence of thoracic aortic mural thrombus at 0.45% (48 cases), of which 17% (eight cases) had autopsy evidence of distal embolization.[16] Because the disease is mostly asymptomatic, its true incidence remains unknown. In our experience, the incidence of aortic thrombus as a source of embolism has been higher (20%) in comparison to previously published series (5%).[17]

Our patients are younger (mean age, 41.2 years) in comparison to contemporary series (>50 years).[1,16] Smoking, hypertension, diabetes, and dyslipidemia[18] are commonly associated comorbidities in previous described series.1,2 In contrast, only one of 19 patients (5.3%) was a smoker, and none of the other patients had any other comorbidity. Similar to other studies, lower limb ischemia was the most common presentation, followed by visceral ischemia. Unlike in our series (60%), yield of hypercoagulable workup has been low in previous reports (25%)[1] (Fig 3). Association of various malignant neoplasms has been previously described,[2] and two patients in our study had malignant disease.

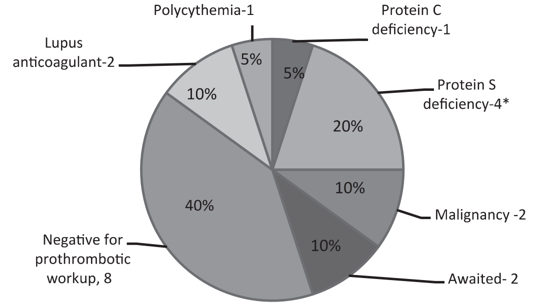


Fig 3. Yield of hypercoagulable workup in patients with primary aortic mural thrombus (PAMT). *One patient had combined protein C and protein S deficiency.

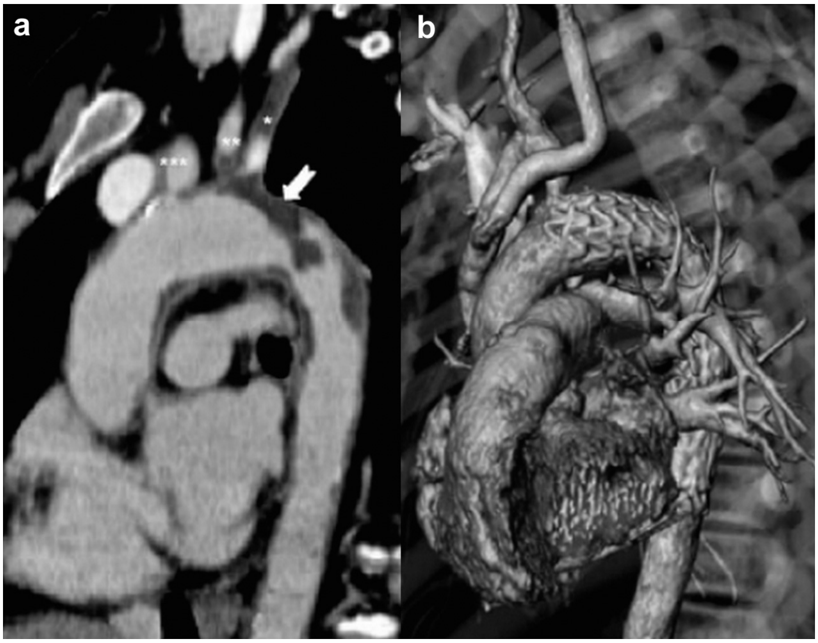


Fig 4. Floating aortic arch thrombus involving supra-aortic vessels (type Ib primary aortic mural thrombus [PAMT]) **(a)** that was managed by supra-aortic debranching and antegrade endograft implantation **(b)**. (Reproduced from Rancic Z, Pfammatter T, Lachat M, Frauenfelder T, Veith FJ, Mayer D. Floating aortic arch thrombus involving the supraaortic trunks: successful treatment with supra-aortic debranching and antegrade endograft implantation. J Vasc Surg 2009;50:1177-80. © Elsevier 2009.)

Previous studies have found early atherosclerotic plaque at mural attachment sites. Therefore, atherosclerosis as an underlying cause could not be denied, especially in patients who have risk factors for atherosclerosis.[16,18]

The morphologic features of the thrombus (sessile vs pedunculated) and the length of the aortic segment involved could be predictors of the embolic potential of such a lesion. Karalis et al reported 73% incidence of embolic events among patients with pedunculated and highly mobile aortic thrombi, as opposed to only 12% when thrombus was layered and immobile.[19] A transesophageal echocardiography-based study of protruding aortic plaques showed that the presence of mobile lesions was linked to 30 times increased risk of distal embolization.[20] In our experience, the source of emboli was from pedunculated and sessile aortic thrombus (nine pedunculated, 10 sessile). In all cases, imaging was obtained soon after the embolic event happened; it was difficult to interpret whether original embolizing lesions were sessile, were pedunculated, or had dislodged, leaving behind the residual sessile component.

Consistently reported in almost all case series, the distal arch and the descending thoracic aorta have been the most common sites of thrombus (74%), followed by the abdominal aorta (14%) and the ascending aorta (12%).[1] In our series, we had two, eight, three, and six patients with type I, type II, type III, and type IV aortic thrombus, respectively, and we discuss each group separately.

Type I PAMT. This is the least common site for aortic thrombus and could be manifested with stroke or upper limb, visceral, or lower limb ischemia.[18,21-23] The majority of cases described in the literature are type Ib (varying degree of involvement of the arch of the aorta) and were managed in an individualized manner including anticoagulation, systemic thrombolysis, and surgical removal of thrombus under cardiopulmonary bypass through median sternotomy.[18,21-23] For large pedunculated thrombi that carry a high risk of recurrent embolism, surgical thrombectomy with cardiopulmonary bypass should be considered. Alternatively, successful hybrid supra-aortic debranching and antegrade stent graft implantation have also been described (Fig 4).[24]

Small sessile thrombus possesses a lower risk of embolization and can be managed with anticoagulation and close follow-up imaging.[19,20] In our study, two patients with type Ib thrombus had small sessile thrombus on the inner aortic and outer aortic curves in the region of the left subclavian artery origin. One patient was diagnosed as lupus anticoagulant antigen positive and managed with lifelong oral anticoagulation alone, with no recurrent embolic episodes.

Type II PAMT. Type IIa has been the most common type of PAMT described in the past.[1-5,9-26]

The junction of the distal aortic arch and descending thoracic aorta has been shown to have a predilection for aortic mural thrombus, although its exact cause is not

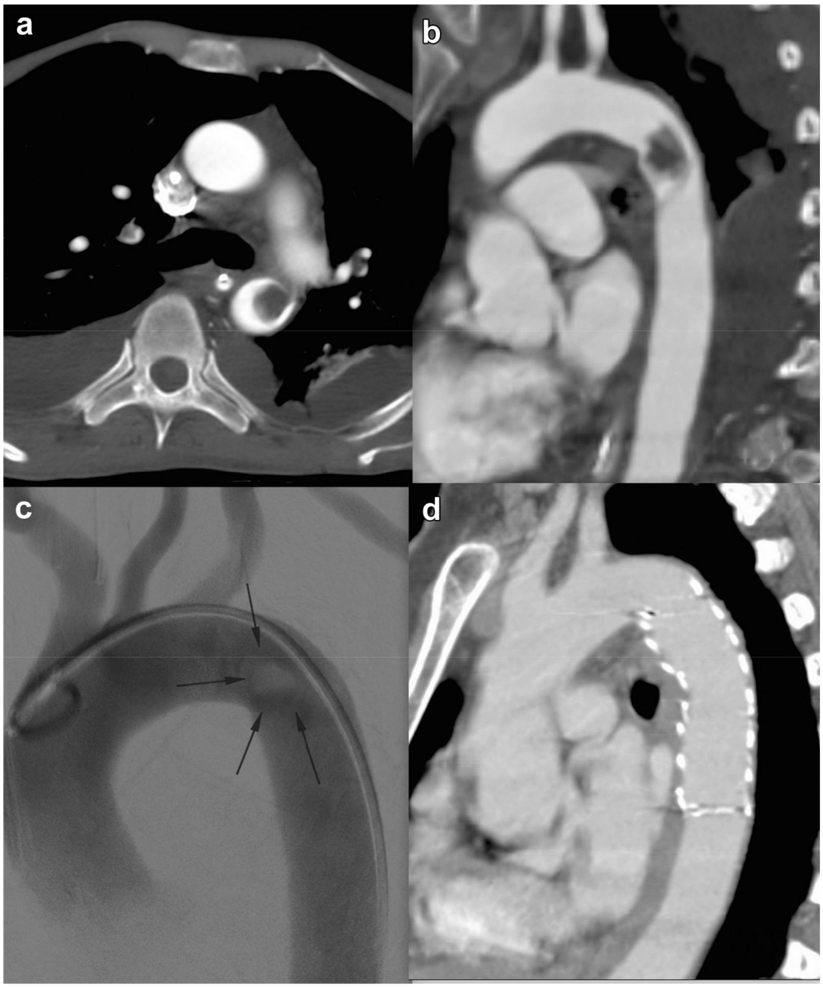


Fig 5. Type IIa primary aortic mural thrombus (PAMT). **a** and **b,** Preoperative computed tomography (CT) scan showing pedunculated thrombus distal to left subclavian artery. **c,** Intraoperative angiogram showing shadow of thrombus (*arrows*). **d,** Six-month follow-up CT scan showing complete exclusion of thrombus.

known. The possibility of an embryologic defect at the aortic isthmus and shear, bending, and torsion stresses[26] attributed to aortic trauma during blunt chest injuries have been described.[10,25] Unlike in previous reports, patients in our series had aortic thrombus not only in the aortic isthmus but extending into the lower descending thoracic aorta: type IIb PAMT (Table I).

Although anticoagulation has been the primary modality of treatment and isolated case reports have shown resolution of thrombus with anticoagulation alone,[2,27] persistence of thrombus load or recurrent embolism risk has been reported to be high (>25%) compared with open surgical removal of thrombus (9%).[1,25] Recurrent embolism significantly increases the risk of major amputation (9% for anticoagulation alone vs 2.3% for surgical group) and life-threatening visceral ischemia.[1] Another problem with anticoagulation alone is that neither the duration of anticoagulation nor the optimal target international normalized ratio range is known.

Minimally invasive options like catheter aspiration and systemic or catheter-directed thrombolysis, although described with varying success rates,[11,28] carry high risk of distal embolization during the procedure itself and do not promise complete removal or exclusion of thrombus.

Surgical thrombectomy has the advantage of yielding an aortic wall histopathologic diagnosis, which may be of diagnostic utility in this relatively unknown entity. However, surgical options are not complication free, with a reported mortality of 2.6%[29] and perioperative complications ranging from 28.9% to 71%.[15,29] This prompted some investigators to consider nonoperative management of aortic thrombus as first-line management.[15]

Endovascular stenting to exclude PAMT has the potential to decrease the size of residual thrombus and recurrent embolization in comparison to anticoagulation alone. It also has fewer perioperative complications in comparison to open surgical thrombectomy.[3,14]

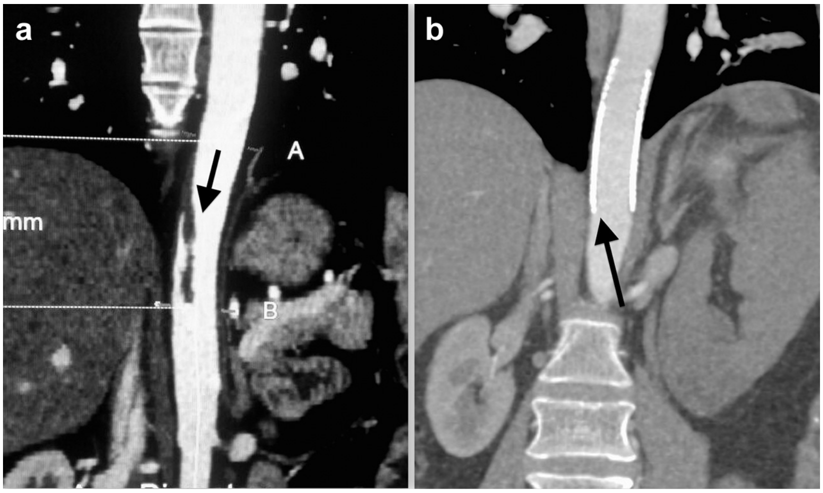


Fig 6. Type IIb primary aortic mural thrombus (PAMT). **a,** Preoperative computed tomography (CT) scan showing type IIb pedunculated thrombus. **b,** Six-month follow-up CT scan shows complete exclusion of thrombus by 22- × 70-mm Wallstent (Boston Scientific, Watertown, Mass).

Covered stent graft exclusion of aortic mural thrombus has been described as an effective option in contemporary series.[1,2,12,13,30]

In our experience, eight patients with type II PAMT presented with either severe limb ischemia or bowel ischemia, or both (Table I). Given the risks involved with open surgical thrombectomy, all patients were considered for stent grafting either during or after addressing of the acute event (eg, peripheral thromboembolectomy). Being partially occlusive, visibility of thrombus on aortic angiography was anticipated to be poor; hence, landing zones of stent grafts were planned preoperatively with CT scan, by use of fixed points (vertebral bodies, origins of left subclavian artery and celiac artery). In addition, obtaining aortograms through a left subclavian artery catheter was useful and revealed "thrombus shadow" in the majority of cases (Fig 5). Stent grafts were oversized not more than 5%, and no balloon molding was attempted. Coverage of thrombus by at least 2 cm was ensured on both ends to avoid thrombus from being squeezed out, resulting in recurrent distal embolization.

In situations in which thrombus shadow is absent, intravascular ultrasound may be potentially useful in guiding endografts and assessing post-stenting thrombus exclusion. Unavailability of intravascular ultrasound in our center did not allow us to assess its utility.

Although stent grafts were our preferred option, because of financial unaffordability by our patients or unavailability of appropriate sizes, we placed closed-cell, bare metal, self-expandable Wallstent (Boston Scientific, Watertown, Mass) in three patients with an intention to plaster the thrombus against the wall to prevent recurrent distal embolism (Fig 6).

Of eight patients in this group, one patient died of ischemic pancreatitis in the preoperative period while awaiting stent graft placement; another patient who underwent bare metal stenting developed acute renal failure and respiratory distress and died in the immediate postoperative period. Of the six surviving patients who underwent endovascular treatment, the implicated thrombus was excluded on CT scans at 6 months. There was no difference in the outcomes between stent graft and bare metal stent implantation (Fig 5, *d* and Fig 6, *b*).

Therefore, stent grafts and bare metal stents both appear to be minimally invasive, low risk, and definitive therapy for thoracic aortic mural thrombus. The efficacy of bare metal stents to exclude embolizing carotid plaque has been well tested,[31] and its role in excluding aortic mural thrombi has been described in the literature with successful outcomes.[14] To be effective for PAMT lesions, bare metal stents should have low radial force and a closed-cell design and be available in large diameters of 14 to 34 mm. Although a disease-specific stent has yet to be developed for PAMT, Wallstent (Boston Scientific) may be the most appropriate currently available stent. It has a small cell area of 1.08 mm^2 and a low radial force of 0.39 ± 0.03 N/cm.[32,33] In addition, where long-segment coverage is required, closed-cell stents have a theoretical advantage of preserving flow to the arteries supplying the anterior spinal cord and lowering the risk of spinal cord ischemia. In our study, bare metal stents excluded aortic thrombus adequately with preservation of the visceral vessel on 6-month follow-up CT scans. The available maximum diameter (24 mm) of closed-cell bare metal stents (Wallstent) is a limiting factor for use in the proximal aorta.

Type III PAMT. Type III PAMT poses a significant challenge because unprotected transfemoral embolectomy has an increased risk of embolism in visceral vessels. Balloon embolectomy after temporary occlusion of visceral vessels has been described as an approach for removal of

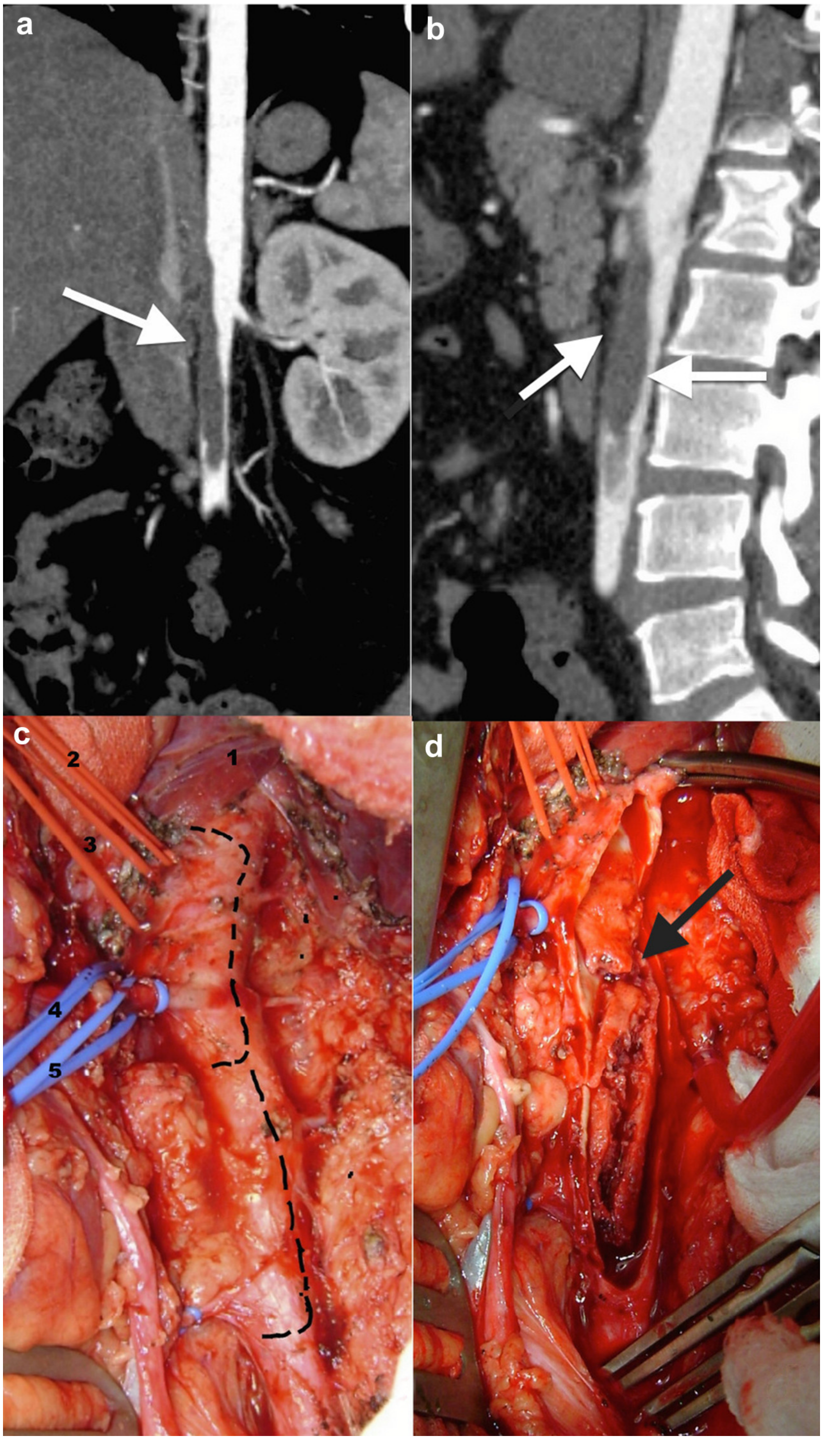


Fig 7. Trapdoor thrombectomy for type III primary aortic mural thrombus (PAMT). **a** and **b,** Preoperative computed tomography (CT) scan showing type III PAMT (*arrow*) in coronal and saggital sections. **c,** Medial visceral rotation and supraceliac aortic dissection until diaphragmatic crus (*1*). Silastic loop control of celiac trunk (*2*), superior mesenteric artery (*3*), and right (*4*) and left (*5*) renal arteries. Incision line for trapdoor aortotomy (*dotted line*) could extend inferiorly if thrombus extends below in infrarenal aorta. **d,** Intraluminal thrombus is seen after trapdoor aortotomy (*arrow*). Aortic thrombectomy and embolectomy from individual visceral vessel could be performed.

such thrombus.[2] However, the possibility of leaving residual friable thrombus is high. In our experience, symptomatic type III PAMT involves more than one visceral artery (celiac, superior mesenteric artery, or renal arteries), causing significant visceral ischemia, and therefore aortic embolectomy alone will not be sufficient. Acute mesenteric ischemia, acute renal failure, or acute aortic occlusion leading to lower limb ischemia mandates urgent exploration and aortic thrombectomy as well as thrombectomy of individual visceral vessels through a retroperitoneal "trapdoor aortic approach."[34] For infrarenal extension of type III PAMT, trapdoor incision could be extended farther inferiorly (Fig 7).

We had three patients of type III PAMT in our series (Table II). All three underwent trapdoor aortic thrombectomy as an emergency procedure. One patient recovered, whereas two patients who had extensive gangrenous changes of viscera died in the postoperative period because of sepsis. Type III PAMT is devastating as recognition of this problem is often delayed and the surgical course is challenging. Delayed presentation of visceral ischemia may result in multiple organ dysfunction and severe sepsis. Therefore, despite adequate removal of thrombus, perioperative mortality could be high and should be adequately consented. In our experience, we did not consider fenestrated or branched endograft therapies as our patients were young and long-term effects of endografting are poorly described. In addition, most cases presented as surgical emergencies, making case planning and graft ordering very challenging. Also, in the setting of visceral ischemia and sepsis, use of endografts might have increased risk of graft infection.

Type IV PAMT. Apart from sessile and pedunculated eccentric thrombus, complete thrombotic occlusion of the aortic bifurcation has been described. In the West, acute thrombotic occlusion of the aortic bifurcation has been typically attributed to saddle embolus originating from the heart, but there are many reports in which no cardiac or proximal embolic source was identified.[1] Similar to other zones, hypercoagulable states have been identified as the cause of PAMT in this zone as well.[35-38]

We had six patients with infrarenal aortic thrombus (3 sessile, 2 pedunculated, and 1 with complete occlusion). All six patients presented with acute lower limb ischemia (Table II). Of the six patients, thrombus removal was considered in five patients, whereas one small sessile thrombus was managed with anticoagulation alone. Of the five, aortobiiliac embolectomy was performed in three patients. Postoperative CT angiography showed significant residual thrombus in two of these patients, which was further excluded by bare metal stents. A sessile but embolizing thrombus at the aortic bifurcation in another patient could not be removed by embolectomy and required aortic thrombectomy and patch angioplasty. Another type IV PAMT that was within 2 cm of the lower renal artery was primarily excluded by stent graft.

In the authors' opinion, unless thrombus is juxtaposed to the renal artery, aortobiiliac embolectomy with Fogarty catheter is the least invasive and a readily available method to remove type IV PAMT. However, when thrombus is juxtaposed to the renal arteries, blind embolectomy may increase risk of embolization to the renal arteries. Therefore, in such cases, we consider either open aortic thrombectomy or exclusion of thrombus with stent graft. In addition, embolectomy alone may not be sufficient to remove adherent aortic thrombus completely.

As residual thrombus can be a potential source of recurrent emboli, a CT scan should be performed to rule out any residual thrombus, which should then be excluded by endovascular stent graft or bare stent.

CONCLUSIONS

PAMT is a significant problem in young patients and has poor outcomes despite aggressive treatment. Advanced organ ischemia is a poor prognostic sign.

We suggest that all large embolic descending thoracic and abdominal aortic lesions be covered. Availability of appropriately sized stent grafts remains a problem in developing countries. Complete exclusion of type II and type IV thrombus can be effectively achieved by large-diameter, closed-cell bare metal stents when stent grafts are not available or there are concerns with coverage of visceral or spinal cord feeding vessels. In patients with type III thrombus, we favor trapdoor aortotomy and thromboembolectomy.

AUTHOR CONTRIBUTIONS

Conception and design: HV, RT
Analysis and interpretation: HV, RT
Data collection: HV, NM, SV
Writing the article: HV, RT
Critical revision of the article: RG, RT
Final approval of the article: RT
Statistical analysis: RT, HV
Obtained funding: Not applicable
Overall responsibility: RT

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