Management of floating thrombus in the aortic arch

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Abstract

Objective: Floating aortic thrombus is an under-recognised source of systemic emboli and carries a life-threatening risk of stroke when located in the aortic arch. Optimal treatment is not established in available guidelines. We report our experience in managing floating thrombi in the aortic arch.

Methods: Consecutive patients diagnosed with a floating aortic arch thrombus at a tertiary referral centre between January 2008 and December 2014 were reviewed. Perioperative and mid-term outcomes were assessed.

Results: Ten patients (eight female) with a median age of 56 years (range 47-82 years) were identified. Eight patients presented with a symptomatic embolic event while two patients were asymptomatic. One patient presenting with stroke due to embolic occlusion of all supra-aortic vessels died two days following admission. Three patients (two asymptomatic and one unfit for surgery) were treated conservatively by anticoagulation, leading to thrombus resolution in two patients. In the third patient, the thrombus persisted despite anticoagulation, resulting in recurrent embolic events. The remaining six patients underwent open thrombectomy of the aortic arch during deep hypothermic circulatory arrest. All patients treated by surgery had an uneventful postoperative course with no recurrent thrombus or embolic event during follow-up. Median follow-up of all patients was 17 months (range 11 - 89 months).

Conclusions: Floating aortic arch thrombus is a dangerous source of systemic emboli. Surgical removal of the thrombus is easy to perform and followed by very good clinical results. Conservative treatment with anticoagulation may be considered in asymptomatic, inoperable or very high-risk patients.

Abstract word count: 246

Central Message

Floating aortic arch thrombus is a dangerous source of emboli. Surgical removal of the thrombus is easy to perform and followed by very good clinical results.

Perspective Statement

Optimal treatment of floating aortic arch thrombus is not well established. Evidence concerning aortic arch thrombectomy for floating thrombi is scarce. With this study, we add a series of patients to the literature and demonstrate that aortic arch thrombectomy can be performed easily and with very good clinical results in terms of prevention of further embolic events in these patients.

Abbreviations and Acronyms

AC = Anticoagulation

ASA = Acetylsalicylic acid

CT = Computed tomography

DHCA = Deep hypothermic circulatory arrest

DL = Dyslipidaemia

DM = Diabetes mellitus

DVT = Deep venous thrombosis

HT = Hypertension

LMWH = Low molecular weight heparin

MRI = Magnetic resonance imaging

NIHSS = National Institutes of Health Stroke Scale

PAD = Peripheral artery disease

TEE = Transesophageal echocardiography

TIA = Transient ischaemic attack

Introduction

Floating aortic thrombus is rare, but with the more frequent use of imaging modalities over the past decades, it has increasingly been identified as a source of systemic emboli (1). In a study of 10'671 consecutive autopsies, the incidence of aortic mural thrombus was 0.45 % (2). The most common location reported in clinical studies (3) is the descending thoracic aorta and the aortic arch. Detailed pathophysiological mechanisms are not yet fully understood. Some authors reported mobile thrombi on aortic arch atheroma in predominantly elderly patients with atherosclerotic disease (4). Floating aortic thrombus however, is often seen in relatively young patients without severe atherosclerosis and many authors agree, that it is a distinct clinical entity that has to be distinguished from atheromatous debris (1, 3, 5), although atherosclerotic processes may contribute to its pathogenesis (1). A high prevalence of haematological disorders and other hypercoagulable conditions, like malignancy, has been reported in other series, suggesting these may also be causative factors for thrombus formation (3). Treatment options include anticoagulation (6, 7), surgical thrombectomy (8-10) and in some cases, endovascular treatment (11, 12). However, comparative data is scarce and available guidelines (13) lack treatment recommendations.

Thrombus localisation in the aortic arch is particularly challenging, as cerebral embolisation is an impending risk with substantial morbidity and mortality. Surgical treatment of aortic arch thrombus requires extracorporeal circulation and circulatory arrest. It is unclear, whether the benefits of open thrombus removal outweigh the perioperative risks of aortic arch surgery. For aortic arch atheroma (with or without mobile components), current stroke guidelines do not recommend surgical treatment to prevent cerebral embolisation (14). This is based on a study by Stern et al. who analysed stroke risk during cardiac surgery in patients with arch atheroma and reported an

unproportionally high incidence of intraoperative stroke (34.9 %) in patients who underwent arch endarterectomy in addition to another cardiac procedure (15).

The aim of this study was to assess detailed narrative data including risk factors, clinical presentation, treatment modality and mid-term outcome of patients with floating aortic arch thrombus. Our hypothesis was that surgical management has a favourable outcome and effectively prevents further embolic events in patients with floating aortic arch thrombus.

Materials and Methods

Consecutive patients treated for floating aortic arch thrombus at a Swiss tertiary referral centre (University Hospital Bern) between January 2008 and December 2014, were identified. Individual patient consent was obtained and the study was performed according to the requirements of the local ethics committee.

Floating aortic arch thrombus was defined as a homogenous mass on computed tomography (CT) or transesophageal echocardiography (TEE) images, attached to the aortic wall and protruding into the lumen of the aortic arch with a mobile aspect (Figure 1). Information on size, exact localisation and quality of the attachment site of the thrombus was retrieved from contrast-enhanced CT scans (1 mm slice thickness). Data including patient demographics, cardiovascular and thrombotic risk factors, embolisation site, treatment method and postoperative complications, were collected from hospital records. Patient follow-up included regular visits in our outpatient clinic and was completed by a telephone interview of all patients or their general practitioner at the end of June 2015 to assess for death, recurrent embolism, continuation of anticoagulation, and subsequently diagnosed malignant disease.

Results

Patient characteristics and clinical presentation

Over a period of seven years, a total of 10 patients were identified. Eight patients were female and median age was 56 years (range 47 – 82 years). All patients had two or more cardiovascular risk factors, mainly hypertension (n = 8), smoking (n = 7) or a body mass index ≥ 30 kg/m^2 (n = 7). Other previously described predisposing factors for aortic thrombus formation (7) were: steroids (n = 2), hormone replacement therapy (n = 1) and malignancy (n = 1, high-grade undifferentiated pleomorphic sarcoma of the pelvic bone). Two patients had either a personal or a family history of venous thromboembolism. Thrombophilia testing was performed in six patients and revealed procoagulant abnormalities in four (Table 1). One patient had a patent foramen ovale. None had atrial fibrillation or any other identifiable embolic source. The diameter of the ascending aorta and aortic arch was normal in all patients. Thrombus developed under combined acetylsalicylic acid (ASA) and statin treatment in two patients (# 2 and 7), under ASA in two patients (# 9 and 10) and under statin therapy in one patient (# 6). No patients were on anticoagulation therapy at presentation.

Eight patients were diagnosed with floating aortic arch thrombus after a symptomatic embolic event including upper or lower limb ischaemia (n = 4), distal aortic occlusion (n = 1), visceral ischaemia (n = 1), and ischaemic stroke (n = 2) (Table 2). Of these patients, six were diagnosed by CT angiography while two were initially diagnosed by TEE. One patient (# 5) had two TEE examinations that did not demonstrate the thrombus, before it was diagnosed by CT angiography.

Two patients had no symptomatic embolic event at presentation (# 4 and 9). The thrombus was incidentally diagnosed by CT, performed for acute chest pain or cancer staging.

Thrombus localisation and morphology

Three patients (# 2, 8 and 9) had two thrombi localised in the aortic arch, resulting in a total of 13 thrombi in 10 patients. Median thrombus length measured on CT scans was 2.8 cm (range 1.3 – 4.3 cm) and median width was 0.9 cm (range 0.5 – 1.8 cm). No correlation between thrombus size and symptoms was observed.

The localisation of the attachment sites is displayed in Figure 2. On CT scans, the attachment site appeared to be normal aortic wall in six patients, whereas in three patients, there were minor calcified atherosclerotic changes. Only patient # 6 showed evidence of a heavily calcified plaque at the attachment site.

Treatment

In six patients (# 1, 2, 3, 6, 7, 8) surgical embolectomy was required to treat the initial embolic event. Histological examination of the embolus was performed in five patients and confirmed thrombus. Endovascular cerebral mechanical thrombectomy was performed in two patients with ischaemic stroke (# 5 and 10). Patient # 10, presenting with stroke due to embolic occlusion of all supra-aortic vessels, died two days following unsuccessful mechanical thrombectomy. For these two patients, no analysis of the removed embolic material was available.

In three patients, the floating aortic arch thrombus was treated conservatively using anticoagulation only, including one symptomatic patient aged 77 years (# 6) who was considered unfit for aortic arch surgery and two asymptomatic patients (# 4 and 9). The remaining six patients were scheduled for open aortic arch thrombectomy.

Aortic arch thrombectomy

Aortic arch thrombectomy was performed via median sternotomy using cardiopulmonary bypass and deep hypothermic circulatory arrest (DHCA). Arterial cannulation site for cardiopulmonary bypass was the right subclavian artery (n = 4) or the ascending aorta (n = 2), depending on thrombus localisation. Median operating time was 180 minutes (range 120 – 277 minutes) and median DHCA duration was 17 minutes (range 12 – 42 minutes) with antegrade cerebral perfusion (median 16 minutes, range 11-42 minutes). In one patient with very short DHCA (12 minutes), antegrade cerebral perfusion was not performed. During opening, preparation for cardiopulmonary bypass and cannulation, the thrombus was monitored by TEE. After incision of the aortic arch, the thrombus was completely removed in all patients (Figure 3). The aortic wall at the attachment site was resected in four patients (full thickness wall resection), whereas the aortic wall seemed macroscopically normal in two patients. Aortotomy as well as the resection site in case of attachment site resection was directly closed by double layer 4-0 polypropylene running sutures in all patients. No prosthetic material was used, neither as a vascular graft nor as a patch.

Histological examination confirmed that the removed material was a thrombus in all patients. Microscopically, the attachment site was unremarkable in one patient while showing a cholesterol-rich plaque in three patients.

Follow-up

Median follow-up was 17 months (range 11 – 89 months). At the end of follow-up, eight out of ten patients were alive. Patient # 10 died in the context of the initial embolic event and patient # 9 died 11 months after diagnosis of the aortic arch thrombus due to

197 the underlying malignant disease. In all other patients, no malignant disease as a
198 potential causative factor for thrombus formation was diagnosed during follow-up.
199
200 *Conservative treatment:*
201 In patient # 4, asymptomatic at presentation, follow-up CT confirmed complete
202 resolution of the thrombus, leading to discontinuation of oral anticoagulation after three
203 months. Seven months later, the patient presented with embolic occlusion of the
204 forearm arteries, requiring embolectomy. Histological examination of the removed
205 material confirmed thrombus, but no recurrent thrombus in the aortic arch or other
206 embolic source could be identified. Under resumed anticoagulation, the patient
207 experienced no further embolic events.
208 The elderly, symptomatic patient (# 6), considered unfit for open aortic arch surgery,
209 had complete resolution of the thrombus after three months (follow-up CT) and no
210 recurrent embolism under continued anticoagulation at the end of follow-up.
211 The patient with malignant sarcoma (# 9), asymptomatic at diagnosis of the aortic arch
212 thrombus, suffered from ischaemic stroke two days following initiation of
213 anticoagulation and underwent intravenous thrombolytic therapy. Thrombus formation
214 in the aortic arch remained unchanged on follow-up CT scans. Due to progressive
215 malignant disease, this patient was not considered a candidate for surgical
216 thrombectomy. Despite continued anticoagulation (low molecular weight heparin), the
217 patient suffered from multiple transient ischaemic attacks and died 11 months later as a
218 consequence of his malignancy.

Surgical treatment:

All six patients treated by open aortic arch thrombectomy had an uneventful postoperative course. No ischaemic stroke, myocardial infarction, significant deterioration of renal function, postoperative haemorrhage or sternal infection was documented. Postoperatively, five patients received oral anticoagulation treatment with coumarin. Patient # 5, initially presenting with stroke, received no anticoagulation due to haemorrhagic transformation of the cerebral infarction. Patient # 3 was prescribed dual antiplatelet therapy (ASA and clopidogrel) at discharge because therapeutic anticoagulation doses could not be established with coumarin. Patient # 2 was switched from coumarin to clopidogrel 28 months after surgery. Three patients were still on coumarin at the end of follow-up. There were no recurrent embolic events or recurrent aortic thrombi in these surgically treated patients.

Discussion

In this study, we present a consecutive series of ten patients with floating thrombus in the aortic arch, six of whom were treated by open aortic arch thrombectomy. Median age of our patients was slightly higher than reported in other series (1, 3, 16). Female predominance has been reported before (5, 17). Mild procoagulant abnormalities were present in 40 % of patients and in one patient, the aetiology of the thrombus was most likely paraneoplastic, but overall, there was a very high prevalence of cardiovascular risk factors. Nevertheless, only one patient had a relevant atherosclerotic lesion at the thrombus attachment site confirmed by CT, whereas in all other patients, the aortic wall appeared normal or with minimal calcifications on CT scans. Histological examination of the attachment sites resected along with the thrombus, showed a cholesterol-rich plaque in three out of four patients. These findings suggest that atherosclerosis does

contribute to the pathogenesis of floating aortic thrombi, but may not be apparent as calcified plaque. The degree of atherosclerotic contribution may differ between patients and other factors, like haematological abnormalities and steroid treatment may additionally facilitate thrombus formation.

TEE is considered the technique of choice to detect and characterise thoracic aortic lesions like intramural haemorrhage, dissection and atherosclerosis (18, 19). However, visualisation of a short segment of the most cranial ascending aorta proximally to the origin of the innominate artery is limited in TEE (18). In our series, one patient underwent two TEE examinations with no pathological findings before aortic arch thrombus was diagnosed by CT. Therefore, especially if no other embolic source is found, diagnostic workup of patients with cerebral, visceral or peripheral emboli should be completed by CT angiography of the whole aorta. Even if a cardiac source of embolism is found, CT angiography should be used liberally to exclude a concomitant aortic embolic source with possible therapeutic consequences.

Emboli from floating aortic thrombi may cause relevant morbidity and mortality. In our series, one patient suffered from acute Leriche syndrome with complete paraplegia, one had intestinal ischaemia and two suffered from extensive ischaemic strokes with one patient dying from the immediate sequelae. Only two patients (# 4 and 9) were initially asymptomatic. As diagnosis of floating aortic thrombus is usually made after an embolic event, there are very few published reports including asymptomatic patients (12, 20, 21). Therefore, little is known about the risk of these patients to suffer from a first-time embolic event, with or without anticoagulation. In a previous autopsy study, 17% of patients with a thrombus in the thoracic or abdominal aorta had evidence of distal embolisation while 6 % had evidence of a major embolic event that was considered the cause of death (2).

In primarily symptomatic patients, a previous study reported recurrent embolism in four out of twenty-three patients with floating arch thrombus despite intravenous heparin therapy (1). In a systematic review including 200 patients with aortic mural thrombus in all locations, three important predictors of recurrent arterial embolisation were identified: thrombus location in the ascending aorta or arch, mild atherosclerosis of the aortic wall and stroke as a presenting symptom (3). In our series, six out of eight patients who presented with an embolic event had radiological evidence or a history of previous, possibly embolic events and one patient had a another clinically evident embolism (limb ischaemia) before aortic arch thrombus was removed (Table 2). We considered these findings as indicators of a high risk of further recurrence and thus, these patients were treated surgically if no relevant contraindications were present.

In our series, no postoperative complications after aortic arch thrombectomy were documented. DHCA time was short and intraoperative TEE monitoring provided additional assurance that thrombotic material did not dislocate during manipulations on the aortic arch or cannulation. Thrombus dislocation would have immediately been detected and would have prompted CT angiography for localisation and subsequent treatment of the embolus with no delay. Although the risk of aortic arch surgery, especially cerebral embolisation, cannot be denied, we believe that with necessary precautions and adequate patient selection, aortic arch thrombectomy can be performed with a high degree of safety as well as efficiency regarding prevention of further embolic events. Floating aortic arch thrombus should therefore be distinguished from aortic arch atheroma or debris, the latter carrying a much higher perioperative risk if surgically removed (15).

Patient # 9, treated by anticoagulation, suffered from ischaemic stroke two days after anticoagulation was initiated. It remains unclear, if this was a coincidence. It has been

postulated before that anticoagulation could possibly trigger further embolic events by lysing the thrombus at a thin attachment site before lysing the thrombus itself. (9). However, it has to be considered that this patient had underlying malignant disease and therefore comparison to patients without malignancy is difficult.

Patient # 4, in whom anticoagulation was stopped after the thrombus resolved, later suffered from an embolic event while no recurrent aortic thrombus or other embolic source was found. The cause of this embolism remains unclear. It may be hypothesised that a new thrombus had formed at the old attachment site and embolised entirely. Local recurrence of a thrombus at the same site has been described in another series before (1). In surgically treated patients, resection of the attachment site along with the thrombus should be considered. Resection of the attachment site was not associated with any complications in our series.

Procoagulant abnormalities seem to be prevalent in patients with floating aortic thrombi, which emphasises the importance of haematologic workup. However, there are no available recommendations on anticoagulation and antiplatelet therapy in patients with floating aortic thrombus. As atherosclerotic processes may contribute to the pathogenesis of floating aortic thrombi, secondary cardiovascular prevention including lifelong ASA as well as a statin is probably indicated in all patients, but there is no evidence.

The main limitation of this study is its retrospective character. As floating aortic arch thrombus is rare, there was no standard protocol in our clinic for such patients and testing for procoagulant abnormalities was not performed routinely. Asymptomatic patients with floating aortic arch thrombus may be underrepresented in this study, as they are less likely referred to our service.

In conclusion, floating aortic arch thrombus is an under-recognised but dangerous source of cerebral, visceral and peripheral emboli and may cause significant morbidity and mortality. Especially if no other source of emboli is found, diagnostic workup of patients with systemic emboli by CT angiography is mandatory. Symptomatic patients with floating aortic arch thrombus should be considered at high risk for recurrent embolism and we therefore advocate open thrombus removal with resection of the attachment site. Conservative treatment with anticoagulation only may be considered in selected cases, e.g. high-risk and older patients with contraindications for surgery as well as in asymptomatic patients.

Figure legends

Central Picture: Floating aortic arch thrombus.

Figure 1: Computed tomography images of floating aortic arch thrombus (patient # 7).

Figure 2: Attachment sites of 13 thrombi in 10 patients (two concurrent thrombi in three patients).

Figure 3: Intraoperative images (patient # 5): View into the proximal aortic arch. The thrombus is removed by means of a dissector (left). The aortic wall at the attachment site is fixed with a thread and completely cut out (right).

Video: Removal of floating thrombus in the proximal aortic arch.

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