

1 **Management of floating thrombus in the aortic arch**

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25 **Abstract**

26

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

31

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

35

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

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

51

52 Abstract word count: 246

53 **Central Message**

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

56 **Perspective Statement**

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

62 **Abbreviations and Acronyms**

63 AC = Anticoagulation

64 ASA = Acetylsalicylic acid

65 CT = Computed tomography

66 DHCA = Deep hypothermic circulatory arrest

67 DL = Dyslipidaemia

68 DM = Diabetes mellitus

69 DVT = Deep venous thrombosis

70 HT = Hypertension

71 LMWH = Low molecular weight heparin

72 MRI = Magnetic resonance imaging

73 NIHSS = National Institutes of Health Stroke Scale

74 PAD = Peripheral artery disease

75 TEE = Transesophageal echocardiography

76 TIA = Transient ischaemic attack

77 **Introduction**

78 Floating aortic thrombus is rare, but with the more frequent use of imaging modalities
79 over the past decades, it has increasingly been identified as a source of systemic emboli
80 (1). In a study of 10'671 consecutive autopsies, the incidence of aortic mural thrombus
81 was 0.45 % (2). The most common location reported in clinical studies (3) is the
82 descending thoracic aorta and the aortic arch. Detailed pathophysiological mechanisms
83 are not yet fully understood. Some authors reported mobile thrombi on aortic arch
84 atheroma in predominantly elderly patients with atherosclerotic disease (4). Floating
85 aortic thrombus however, is often seen in relatively young patients without severe
86 atherosclerosis and many authors agree, that it is a distinct clinical entity that has to be
87 distinguished from atheromatous debris (1, 3, 5), although atherosclerotic processes
88 may contribute to its pathogenesis (1). A high prevalence of haematological disorders
89 and other hypercoagulable conditions, like malignancy, has been reported in other
90 series, suggesting these may also be causative factors for thrombus formation (3).
91 Treatment options include anticoagulation (6, 7), surgical thrombectomy (8-10) and in
92 some cases, endovascular treatment (11, 12). However, comparative data is scarce and
93 available guidelines (13) lack treatment recommendations.
94 Thrombus localisation in the aortic arch is particularly challenging, as cerebral
95 embolisation is an impending risk with substantial morbidity and mortality. Surgical
96 treatment of aortic arch thrombus requires extracorporeal circulation and circulatory
97 arrest. It is unclear, whether the benefits of open thrombus removal outweigh the
98 perioperative risks of aortic arch surgery. For aortic arch atheroma (with or without
99 mobile components), current stroke guidelines do not recommend surgical treatment to
100 prevent cerebral embolisation (14). This is based on a study by Stern et al. who analysed
101 stroke risk during cardiac surgery in patients with arch atheroma and reported an

102 unproportionally high incidence of intraoperative stroke (34.9 %) in patients who
103 underwent arch endarterectomy in addition to another cardiac procedure (15).
104 The aim of this study was to assess detailed narrative data including risk factors, clinical
105 presentation, treatment modality and mid-term outcome of patients with floating aortic
106 arch thrombus. Our hypothesis was that surgical management has a favourable outcome
107 and effectively prevents further embolic events in patients with floating aortic arch
108 thrombus.

109

110 **Materials and Methods**

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

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

126 **Results**

127 ***Patient characteristics and clinical presentation***

128 Over a period of seven years, a total of 10 patients were identified. Eight patients were
129 female and median age was 56 years (range 47 – 82 years). All patients had two or more
130 cardiovascular risk factors, mainly hypertension (n = 8), smoking (n = 7) or a body mass
131 index ≥ 30 kg/m² (n = 7). Other previously described predisposing factors for aortic
132 thrombus formation (7) were: steroids (n = 2), hormone replacement therapy (n = 1)
133 and malignancy (n = 1, high-grade undifferentiated pleomorphic sarcoma of the pelvic
134 bone). Two patients had either a personal or a family history of venous
135 thromboembolism. Thrombophilia testing was performed in six patients and revealed
136 procoagulant abnormalities in four (Table 1). One patient had a patent foramen ovale.
137 None had atrial fibrillation or any other identifiable embolic source. The diameter of the
138 ascending aorta and aortic arch was normal in all patients. Thrombus developed under
139 combined acetylsalicylic acid (ASA) and statin treatment in two patients (# 2 and 7),
140 under ASA in two patients (# 9 and 10) and under statin therapy in one patient (# 6). No
141 patients were on anticoagulation therapy at presentation.

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

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

151 ***Thrombus localisation and morphology***

152 Three patients (# 2, 8 and 9) had two thrombi localised in the aortic arch, resulting in a
153 total of 13 thrombi in 10 patients. Median thrombus length measured on CT scans was
154 2.8 cm (range 1.3 – 4.3 cm) and median width was 0.9 cm (range 0.5 – 1.8 cm). No
155 correlation between thrombus size and symptoms was observed.

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

160

161 ***Treatment***

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

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

173 ***Aortic arch thrombectomy***

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

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

192

193 ***Follow-up***

194 Median follow-up was 17 months (range 11 – 89 months). At the end of follow-up, eight
195 out of ten patients were alive. Patient # 10 died in the context of the initial embolic
196 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.

219 *Surgical treatment:*

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

231

232 **Discussion**

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

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

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

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

269 In primarily symptomatic patients, a previous study reported recurrent embolism in
270 four out of twenty-three patients with floating arch thrombus despite intravenous
271 heparin therapy (1). In a systematic review including 200 patients with aortic mural
272 thrombus in all locations, three important predictors of recurrent arterial embolisation
273 were identified: thrombus location in the ascending aorta or arch, mild atherosclerosis
274 of the aortic wall and stroke as a presenting symptom (3). In our series, six out of eight
275 patients who presented with an embolic event had radiological evidence or a history of
276 previous, possibly embolic events and one patient had a another clinically evident
277 embolism (limb ischaemia) before aortic arch thrombus was removed (Table 2). We
278 considered these findings as indicators of a high risk of further recurrence and thus,
279 these patients were treated surgically if no relevant contraindications were present.
280 In our series, no postoperative complications after aortic arch thrombectomy were
281 documented. DHCA time was short and intraoperative TEE monitoring provided
282 additional assurance that thrombotic material did not dislocate during manipulations on
283 the aortic arch or cannulation. Thrombus dislocation would have immediately been
284 detected and would have prompted CT angiography for localisation and subsequent
285 treatment of the embolus with no delay. Although the risk of aortic arch surgery,
286 especially cerebral embolisation, cannot be denied, we believe that with necessary
287 precautions and adequate patient selection, aortic arch thrombectomy can be performed
288 with a high degree of safety as well as efficiency regarding prevention of further embolic
289 events. Floating aortic arch thrombus should therefore be distinguished from aortic arch
290 atheroma or debris, the latter carrying a much higher perioperative risk if surgically
291 removed (15).

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

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

298 Patient # 4, in whom anticoagulation was stopped after the thrombus resolved, later
299 suffered from an embolic event while no recurrent aortic thrombus or other embolic
300 source was found. The cause of this embolism remains unclear. It may be hypothesised
301 that a new thrombus had formed at the old attachment site and embolised entirely.

302 Local recurrence of a thrombus at the same site has been described in another series
303 before (1). In surgically treated patients, resection of the attachment site along with the
304 thrombus should be considered. Resection of the attachment site was not associated
305 with any complications in our series.

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

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

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

327 **Figure legends**

328

329 **Central Picture:** Floating aortic arch thrombus.

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

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

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

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

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410 **Key words:** Thrombus, aortic arch, embolism