

FLOATING AORTIC ARCH THROMBUS AND MYOCARDIAL INFARCTION WITH NON-ST-SEGMENT ELEVATION IN A 47-YEAR-OLD WOMAN

V. Byalkova¹, B. Kunev², M. Ivanov¹, D. Kyuchukov¹

¹Cardiac Surgery Department, National Cardiology Hospital – Sofia

²Cardiology Clinic, National Cardiology Hospital – Sofia

ПЛАВАЩ ТРОМБ В АОРТНАТА ДЪГА И МИОКАРДЕН ИНФАРКТ БЕЗ ST-ЕЛЕВАЦИЯ ПРИ 47-ГОДИШНА ЖЕНА

В. Бялкова¹, Б. Кунев², М. Иванов¹, Д. Кючуков¹

¹Клиника по кардиохирургия, МБАЛ Национална кардиологична болница – София

²Клиника по кардиология, МБАЛ Национална кардиологична болница – София

Abstract.

Floating thrombus of the aortic arch in the absence of aneurysm or atherosclerotic disease is an exceptionally rare condition. We report the case of a 47-year-old woman, an active smoker with iron-deficiency anemia, who presented with acute retrosternal chest pain, dyspnea, diaphoresis, and vomiting. Initial evaluation showed arterial hypertension, sinus rhythm with poor R-wave progression in the precordial leads on electrocardiography (ECG), and markedly elevated high-sensitivity troponin levels, consistent with acute coronary syndrome. Transthoracic echocardiography (TTE) revealed a large, highly mobile mass in the aortic arch prolapsing into the supra-aortic branches, reduced left ventricular systolic function and apical dyskinesia. Computed tomography angiography (CTA) confirmed a floating thrombus attached to the lesser curvature of the aortic arch, mild non-obstructive coronary artery disease, and an apical left ventricular aneurysm. The patient was initially managed conservatively with unfractionated heparin and aspirin due to high procedural risk. However, despite therapeutic anticoagulation, she developed a transient cerebrovascular event, prompting urgent surgical intervention. Intraoperatively, a loosely attached mixed thrombus was excised from the aortic arch without evidence of atherosclerosis or endothelial injury. The postoperative course was uneventful. This case emphasizes the high embolic potential of floating aortic arch thrombi and supports early surgical consideration when embolic complications occur despite optimal anticoagulation.

Key words:

aortic arch, thrombus, myocardial infarction, apical aneurysm, thrombectomy

Address

Viktoriya Byalkova, MD, Department of Cardiac Surgery, National Cardiology Hospital, 65 Konyovitsa St., BG – 1309

for correspondence:

Sofia, Tel. +359 2 9211 553; e-mail: vbyalkova.md@gmail.com

Резюме.

Плаващ тромб в аортната дъга при липса на аневризма или атеросклероза е изключително рядко състояние. Представяме случай на 47-годишна жена, активен пушач, с желязодефицитна анемия, с внезапно настъпила ретростернална гръдна болка, придружена от задух, изпотяване и повръщане. От първоначалния преглед се установяват артериална хипертония, ЕКГ данни за синусов ритъм със забавена прогресия на R-зъбеца в прекордиалните отвеждания и значително повишени нива на високочувствителен тропонин, съответстващи на остър коронарен синдром. При трансторакалната ехокардиография (ТТЕ) се обективизира голяма, силно подвижна маса в аортната дъга, пролабираща в супрааортните клонове и потисната систолна функция на лявата камера при апикална дискинезия. Компютърнотомографската ангиография (КТА) потвърди плаващ тромб, прикрепен към малката кривина на аортната дъга, наличие на необструктивна коронарна болест и апикална аневризма на лявата камера. Първоначално се предприе консервативен подход с нефракциониран хепарин и аспирин, поради висок процедурен риск. На фона на терапевтична антикоагулация, пациентката разви транзиторна исхемична атака, което наложи хирургично лечение по спешност. Интраоперативно беше отстранен смесен тромб, слабо прикрепен към аортната дъга, при липса на подлежаща атеросклероза или ендотелно увреждане. Следоперативният период протече без усложнения. Този случай подчертава високия емболичен потенциал на плаващите тромби в аортната дъга и необходимостта от своевременно хирургично лечение при изява на емболичен инцидент на фона на оптимална антикоагулантна терапия.

Ключови думи:

аортна дъга, тромб, миокарден инфаркт, апикална аневризма, тромбектомия

Адрес

Д-р Виктория Бялкова, Клиника по Кардиохирургия, МБАЛ Национална Кардиологична Болница, ул. „Коньовица“

за кореспонденция:

№ 65, 1309 София, Тел. +359 -2-9211-553; e-mail: vbyalkova.md@gmail.com

INTRODUCTION

Floating aortic arch thrombus, without aneurysm or dissection, is an extremely rare condition, with reported incidence rate 0,45% [1] and unclear pathophysiology. Acute or chronic inflammation, rupture of an atherosclerotic plaque or atheroma, penetrating atherosclerotic ulcers, aneurysmal dilatation, hypercoagulability, medication use, autoimmune diseases, malignancies and tobacco use could promote clotting, thus leading to aortic thrombus formation. Mural thrombus on a normal or minimally atherosclerotic aorta has also been regarded as an unusual but possible cause of arterial thromboembolism [1, 2, 7]. In 1967 Oliver et al. published the first described case of thromboembolism from the thoracic aorta [3]. In most of the published case reports and small series of patients with thrombus in a non-aneurysmal thoracic aorta, peripheral embolism was the initial clinical manifestation. Diagnosis is often coincidental using chest imaging modalities [4]. There are no evidence-based guidelines for optimal treatment. Multiple management options include anti-coagulation, thrombolysis, surgical thrombectomy, and endovascular stenting.

lism was the initial clinical manifestation. Diagnosis is often coincidental using chest imaging modalities [4]. There are no evidence-based guidelines for optimal treatment. Multiple management options include anti-coagulation, thrombolysis, surgical thrombectomy, and endovascular stenting.

CASE PRESENTATION

A 47-year-old woman, current smoker with a 20-pack-year history, without any family history of cardiovascular disease, with iron deficiency anemia due to dietary causes, cholelithiasis, history of JJ implantation due to hydronephrosis and pyelonephritis in the past. Presented to the Emergency Department (ER) complaining of retrosternal chest pain, shortness of breath, sweating, and a single episode of vomiting.

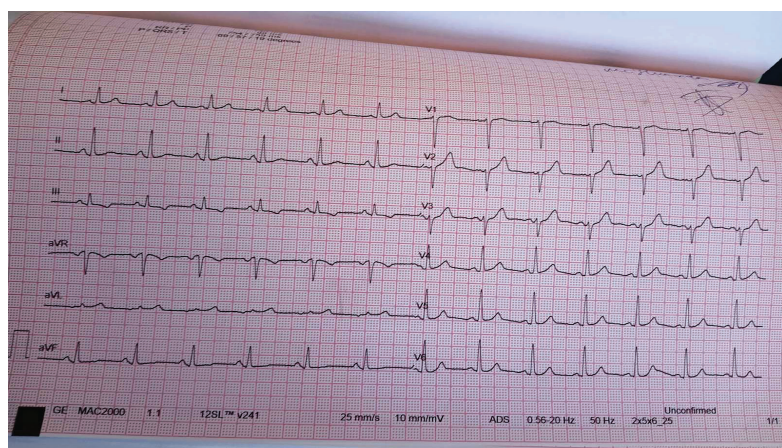


Fig. 1. ECG on the day of admission

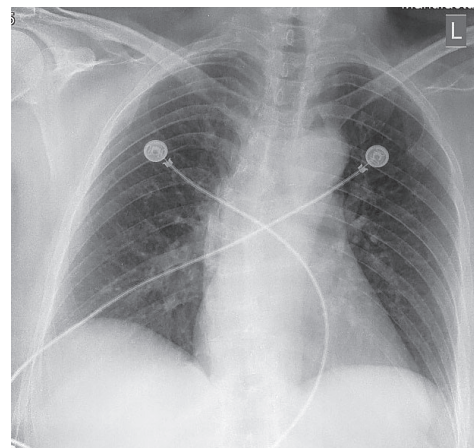


Fig. 2. Chest X-Ray on the day of admission

Table 1. Laboratory values on the first and second day of admission

Age	47		
Gender	Female		
BMI	22,6 kg/m ²		
	1st day at admission	2nd day	Reference Values
Hemoglobin	93 g/L	97 g/L	115-160
Red blood cells	4.6*10 ¹² /L	4.8*10 ¹² /L	4.0-5.2
White blood cells	13.1*10 ⁹ /L	9.9*10 ⁹ /L	4.0-10.0
Proportion of neutrophils	59%	61%	50-74
Platelets	562*10⁹/L	541*10⁹/L	140-400
ASAT/ALAT	14/11 U/L	63/29 U/L	0-31/0-55
CPK/CKMB	44/12,3 U/L	324/41 U/L	0-145/0-24
Total cholesterol	4,86 umol/l	5.01 umol/l	< 5.0
HDL/LDL	1,58/2,76 mmol/l	1.62/2.59 mmol/l	> 1.2/ < 3.0
Iron/Total iron binding capacity	2,1/71.6 umol/l	3.8/68.5 umol/l	6.6-26.7/44.8-71.6
hs Troponin-I 0h/1h	34 ng/L / 49,6 ng/L	10 772.5 ng/L	< 15.6
D-dimer	0.63 mg/L		< 0.5
CRP	0.4 mg/l	1.2 mg/l	< 5
TSH	2.363 uIU/ml	2.014 uIU/ml	0.35-4.94
NT pro BNP		400.3 pg/ml	< 125.0
PT THS INR		1.04	0.9-1.1

The pain was sudden in onset, increasing with inspiration and movement of the upper extremities. The pain was mildly relieved after medication with nonsteroidal anti-inflammatory drugs. The patient denied any palpitations, syncope episodes or bleeding. Vitals on arrival were unremarkable except for blood pressure at 160/110 mmHg symmetrical on both arms. The patient's chest X-ray was normal. Electrocardiogram revealed sinus rhythm with poor R-wave progression in the precordial leads.

Echocardiography showed a large, highly mobile soft tissue mass in the aortic arch that prolapses into the left carotid artery and left subclavian artery. Left ventricular EF about 35% with apical dyskinesia and severely thinned myocardium. Non-dilated ascending aorta, aortic arch and descending aorta, there was no visible flap, non-dilated right heart chambers and pulmonary artery. There was a mild mitral regurgitation. No pericardial effusion.

CT coronary angiography and aortography with contrast showed low-grade coronary stenosis in mid segments of LAD and RCA, a floating thrombus (29/17

mm) in the aortic arch, attached along the lesser curvature of the aortic arch over a length of 12 mm, oriented toward the left subclavian artery and the descending aorta; common origin of the brachiocephalic trunk and the common carotid artery, homogeneously contrasted supra-aortic branches, without stenoses or plaques, an apical aneurysm of the left ventricle 26/19 mm with myocardial thinning 2 mm at the apex.

The case was discussed with an interventional cardiologist, and the patient was deemed not suitable for coronary angiography due to the very high procedural risk. A cardiac surgeon also evaluated the case and considered the patient unsuitable for emergency surgical intervention in the setting of an acute coronary syndrome. A decision was made to re-evaluate in 7-10 days following cardiac MRI. In the meantime, the care team elected to continue with a conservative management strategy utilizing therapeutic anticoagulation with unfractionated heparin combined with single antiplatelet therapy (aspirin) in the acute phase of NSTEMI. Due to the presence of an anemic syndrome, iron replacement therapy was initiated. Serial testing

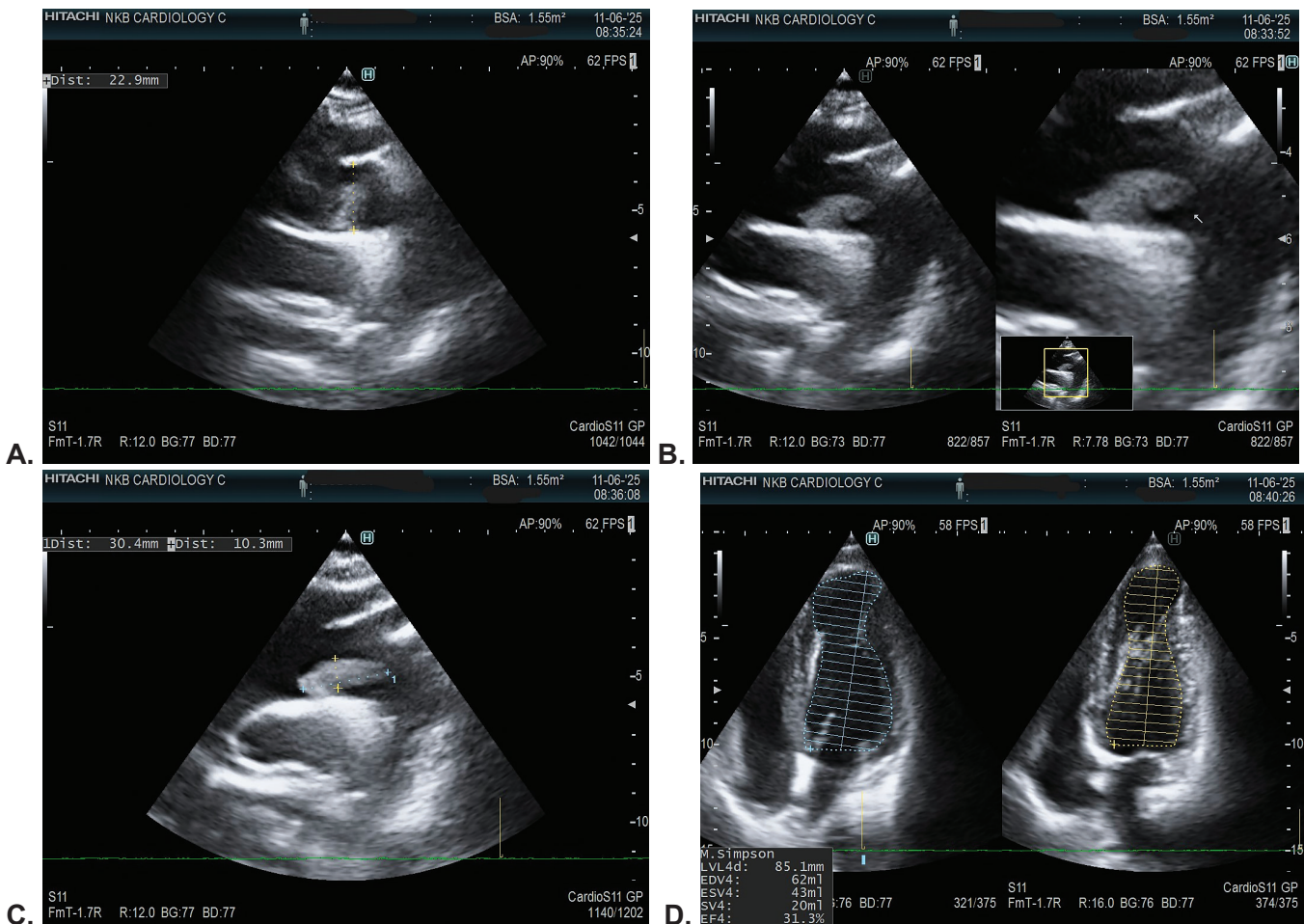


Fig. 3. TTE – suprasternal view. A. Aortic arch is non-dilated 22.9 mm. **B.** Mobile soft tissue mass in the aortic arch that prolapses into the left carotid artery and left subclavian artery. **C.** Mobile tissue mass was measured 30.4/10.3 mm. **D.** Left ventricular dysfunction with apical dyskinesia and severely thinned myocardium

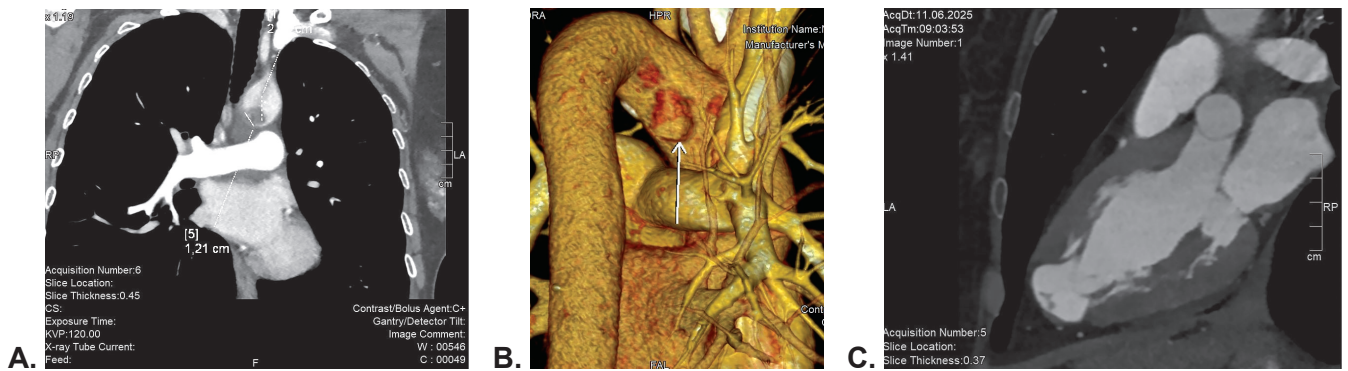


Fig. 4. CT coronary angiography and aortography. A. A floating thrombus (2,9/1,7 cm) in the aortic arch, attached along the lesser curvature of the aortic arch over a length of 1,21 cm. B. 3D reconstruction of the mass, located in the aortic arch C. Apical aneurysm of the left ventricle 2,6/1,9 cm with myocardial thinning 2 mm at the apex

showed a decreasing trend in high-sensitivity troponin levels. During hospitalization, despite optimal anticoagulation with heparin, on the third day of admission, in the evening hours, the patient experienced a transient cerebrovascular event – aphasia. Follow-up echocardiography showed no change in the size of the thrombus in the aortic arch. An urgent neurological consultation was conducted – no neurological deficits were found. A CT scan of the head showed no signs of ischemia or hemorrhage. In light of these findings, the patient was discussed at a multidisciplinary team meeting and on the third day of admission an urgent surgery was performed via median sternotomy. The cardioplegic solution was delivered through the coronary ostia and the heart was arrested successfully. The ascending aorta was clamped subsequently. Intraoperative inspection of the aortic arch revealed a mass consistent with a mixed thrombus, loosely attached to the posterior wall of the aortic arch at the level of the brachiocephalic trunk, measuring approximately 2 × 4 cm. Neither atherosclerotic plaque nor endothelial injury was seen in the thrombus attachment zone. The mass was excised

and sent for histopathological examination, which revealed a layered mixed thrombus with a predominant white agglutinative component.

The early postoperative period was uneventful, with stable hemodynamics. No elevation of enzymatic markers indicative of myocardial necrosis was observed, nor were there any significant ECG changes or rhythm or conduction disturbances. There were no significant pleural effusions. Transthoracic echocardiography at discharge demonstrated apical hypokinesia, no visualized mass in the aortic arch, an intact valvular apparatus, a preserved biventricular systolic function and a minimal pericardial effusion anterior to the right heart chambers. With long-term VKA and aspirin administered, the patient was discharged 9 days after surgery. We recommended MRI, a coronary angiogram and an extensive workup for coagulopathies. MRI on the second month after admission showed an apical aneurysm with transmural enhancement involving the apex and the apical portion of the inferior wall – consistent with infarction in the territories of the distal segments of the right coronary artery and the LAD, with non-viable myo-

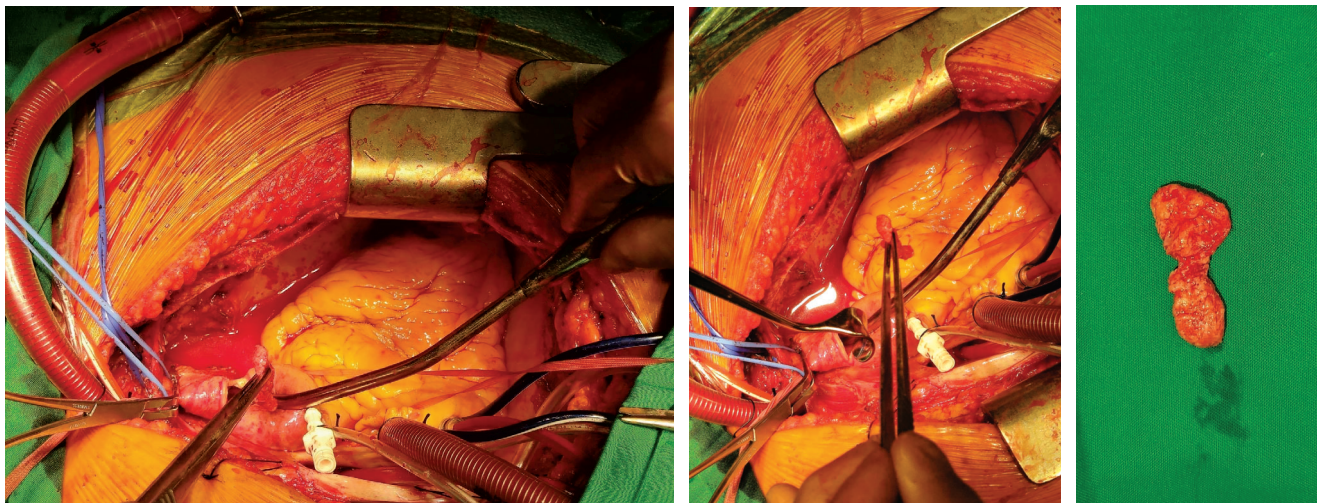


Fig. 5. Operative view. Excised mass from the aortic arch, consistent with a mixed thrombus measuring 2 x 4 cm

cardium. No thrombi were detected within the cardiac chambers or along the major thoracic vessels. Findings on coronary angiography revealed LM and LAD without stenosis. The LCx, represented by a large obtuse marginal branch, had no stenotic disease and a dominant RCA showed mild mid-segment luminal irregularities without significant stenosis.

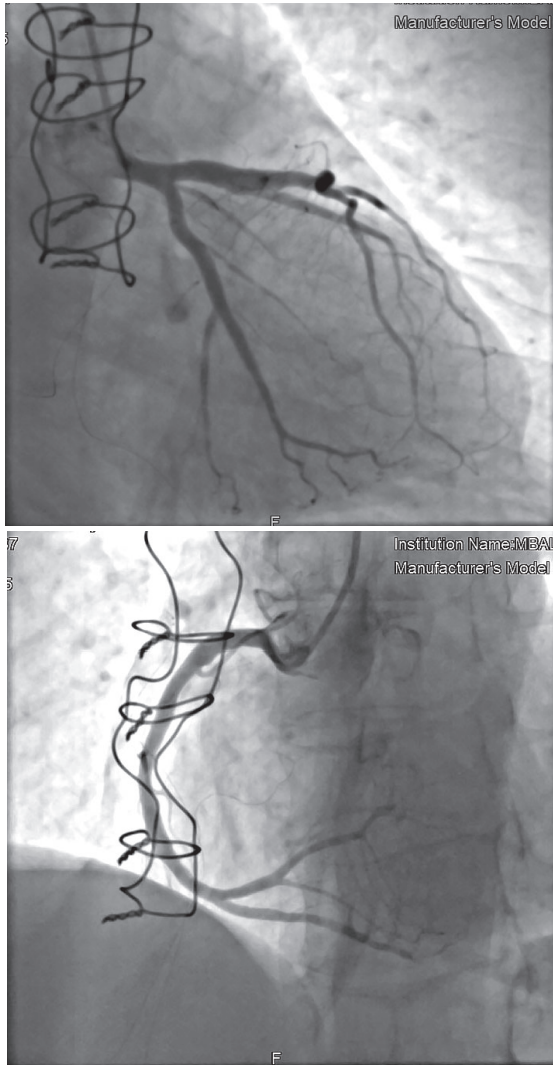


Fig. 6. Coronary angiography imaging

Genetic and hematological investigations revealed multifactorial thrombophilia. A prothrombotic predisposition was identified, including heterozygosity for PAI-1 (4G/5G), heterozygous MTHFR A1298C and C677T mutations, a free protein C level of 86.9%, and a reduced free protein S level of 51.8%.

DISCUSSION

Floating thrombi of the aortic arch represent an exceptionally rare clinical entity and are associated with a substantial risk of systemic embolization owing to their intrinsic mobility. The pathogenesis of non-ath-

erosclerotic aortic thrombus formation remains incompletely elucidated. Proposed mechanisms encompass hypercoagulable states, endothelial dysfunction, tobacco exposure, iron-deficiency anemia with reactive thrombocytosis, and systemic inflammatory activation. The diagnosis of thoracic aortic thrombus poses a significant clinical challenge [5]. In a series of 78 patients, Bowdish et al. demonstrated that acute mural thrombi predominantly localize within the abdominal aorta (64%), followed by the descending thoracic aorta (28%) and, far less frequently, the ascending aorta and aortic arch (8%) [6]. Consequently, floating thrombi of the aortic arch, particularly in the absence of underlying aneurysmal or atherosclerotic disease, remain exceedingly uncommon, and evidence-based management strategies are lacking.

The present case underscores both the diagnostic complexity and therapeutic ambiguity inherent to this condition, particularly when coexisting with an acute coronary syndrome. In this context, the clinical presentation is consistent with myocardial infarction with non-obstructive coronary arteries (MINOCA), a heterogeneous syndrome accounting for approximately 6% of acute myocardial infarctions and associated with significant morbidity and mortality. MINOCA encompasses a broad spectrum of pathophysiological mechanisms, including plaque disruption, coronary vasospasm, microvascular dysfunction, and coronary embolism. Among these, coronary embolism – either direct or paradoxical – assumes particular relevance in the presence of a proximal embolic source such as an aortic arch thrombus.

In the current patient, the absence of aortic dilation, dissection, or atherosclerotic plaque at the thrombus attachment site strongly favors a primary thrombotic process rather than secondary thrombosis superimposed on pre-existing vascular pathology. Iron-deficiency anemia accompanied by marked thrombocytosis likely contributed to a prothrombotic milieu, while active smoking may have further potentiated endothelial dysfunction. Notably, both iron deficiency and iron overload have been implicated in heightened thromboembolic risk [8]. Prior reports have documented severe iron deficiency with extreme thrombocytosis culminating in retinal vein occlusion, as well as cerebral venous sinus thrombosis occurring independently of thrombocytosis [8].

Beyond these acquired factors, the possibility of an underlying thrombophilic diathesis warrants careful consideration. Deficiencies of protein C and protein S – vitamin K – dependent glycoproteins integral to endogenous anticoagulant pathways – result in impaired inactivation of factors Va and VIIIa, thereby facilitating unchecked thrombin generation and a prothrombotic

state. Similarly, resistance to activated protein C, most commonly attributable to the factor V Leiden mutation (G1691A), further augments coagulation by conferring resistance to proteolytic inactivation. Perturbations in fibrinolysis may also contribute; elevated levels of plasminogen activator inhibitor-1 (PAI-1) attenuate fibrinolytic capacity through inhibition of tissue plasminogen activator. The 4G/5G polymorphism within the PAI-1 promoter region modulates transcriptional activity and has been associated with increased circulating inhibitor levels and reduced fibrinolysis.

Alterations in homocysteine metabolism represent an additional, albeit controversial, prothrombotic mechanism. Homocysteine, an intermediary metabolite of methionine, is ordinarily remethylated or trans-sulfurated via pathways dependent on vitamins B12, B6, folate, and the enzyme methylenetetrahydrofolate reductase (MTHFR). Genetic polymorphisms affecting MTHFR function may lead to mild-to-moderate hyperhomocysteinemia, a condition frequently encountered in the general population. While severe elevations, as observed in homocystinuria, are unequivocally associated with premature vascular disease and thrombosis, the independent contribution of mild hyperhomocysteinemia to cardiovascular risk remains uncertain, as reflected in the American Heart Association statement of 2010.

A particularly compelling aspect of this case is the development of a thin-walled apical aneurysm characterized by dyskinesia, myocardial thinning, and transmural late gadolinium enhancement on cardiac magnetic resonance imaging, occurring in the absence of obstructive coronary artery disease. Several pathophysiological mechanisms merit consideration. Coronary microembolization originating from the aortic thrombus may have resulted in transient coronary occlusion followed by spontaneous reperfusion, culminating in irreversible myocardial injury. Alternatively, paradoxical embolism – potentially facilitated by an undetected intracardiac shunt such as a patent foramen ovale – cannot be excluded. Catecholamine-mediated myocardial stunning or a Takotsubo-like cardiomyopathy triggered by acute hemodynamic stress and hypertensive surge represents another plausible mechanism; however, the presence of transmural fibrosis on magnetic resonance imaging argues against classical stress-induced cardiomyopathy. Collectively, these findings reinforce the classification of this presentation within the MINOCA spectrum and highlight the intricate interplay between embolic and non-ischemic myocardial injury mechanisms.

Multimodality imaging was pivotal in establishing the diagnosis. Transthoracic echocardiography enabled the initial identification of a highly mobile aortic arch mass, whereas contrast-enhanced computed to-

mography provided precise anatomical delineation, exclusion of structural aortic pathology, and concurrent evaluation of the coronary vasculature. Cardiac magnetic resonance imaging further facilitated detailed myocardial tissue characterization, thereby elucidating the underlying mechanism of myocardial injury.

Therapeutic strategies for floating aortic arch thrombi remain contentious due to the absence of randomized evidence or formalized guidelines [9]. Systemic anticoagulation is frequently adopted as first-line therapy in hemodynamically stable patients, particularly when surgical risk is prohibitive; however, it may paradoxically predispose to thrombus destabilization and embolization prior to complete resolution. Less invasive approaches, including percutaneous thromb aspiration and thoracic endovascular aortic repair (TEVAR), have emerged as potential alternatives, particularly for thrombi located within the descending thoracic aorta. Indeed, TEVAR has demonstrated high rates of immediate thrombus exclusion with low recurrence in selected cohorts. Nevertheless, manipulation of a highly mobile thrombus carries a non-negligible risk of distal or proximal embolization, and its application is limited in cases involving the ascending aorta or aortic arch, where anatomical considerations and embolic risk often preclude its use.

In the present case, the occurrence of a transient cerebrovascular event despite therapeutic anticoagulation underscored the limitations of conservative management and prompted a decisive shift toward surgical intervention. Surgical thrombectomy affords definitive eradication of the embolic source and enables direct inspection of the aortic intima [10]. Intraoperative findings in this patient confirmed the absence of atherosclerotic or endothelial pathology, and complete resolution was achieved without postoperative complications.

This favorable outcome lends support to early surgical consideration in patients harboring large, highly mobile thrombi or those experiencing embolic complications, even in the setting of concomitant acute coronary syndrome. Early recognition, comprehensive evaluation for underlying prothrombotic conditions, meticulous multimodality imaging, and timely escalation to definitive surgical management are paramount in mitigating the risk of catastrophic embolic complications.

CONCLUSION

Floating thrombus of the aortic arch in the absence of underlying aortic disease is a rare but high-risk condition, particularly when associated with systemic embolization or acute coronary syndrome. This case highlights the diagnostic value of multimodality imaging and the limitations of anticoagulation alone in the setting of large, mobile thrombi. The occurrence of embol-

ic events despite optimal medical therapy emphasizes the potential need for early surgical intervention. An individualized, multidisciplinary approach is essential to balance embolic risk against procedural morbidity. Prompt recognition and timely escalation of therapy may be decisive in achieving favorable clinical outcomes and preventing life-threatening complications.

No conflict of interest was declared

References

1. Machleder HI, Takiff H, Lois JF, et al. Aortic mural thrombus: an occult source of arterial thromboembolism. *J Vasc Surg.* Nov 1986;4(5):473-8.
2. Tunick PA, Kronzon I. Atheromas of the thoracic aorta: clinical and therapeutic update. *J Am Coll Cardiol.* Mar 01 2000;35(3):545-54. doi:10.1016/s0735-1097(99)00604-x
3. Oliver DO. Embolism from mural thrombus in the thoracic aorta. *Br Med J.* Sep 09 1967;3(5566):655-6. doi:10.1136/bmj.3.5566.655
4. Ghoweba M, Gnasigamany J, Chiluveri M, McClish J. A Ticking Time Bomb: A Case of Floating Distal Aortic Arch Intraluminal Thrombus. *Cureus.* Dec 2022;14(12):e32212. doi:10.7759/cureus.32212
5. Pagni S, Trivedi J, Ganzel BL, et al. Thoracic aortic mobile thrombus: is there a role for early surgical intervention *Ann Thorac Surg.* Jun 2011;91(6):1875-81. doi:10.1016/j.athoracsur.2011.02.011
6. Bowdish ME, Weaver FA, Liebman HA, Rowe VL, Hood DB. Anticoagulation is an effective treatment for aortic mural thrombi. *J Vasc Surg.* Oct 2002;36(4):713-9
7. Majdi Gueldich, Mariantonietta Piscitelli, Haytham Derbel, Khaoula Boughanmi, Eric Bergoend, Nora Chanai, Thierry Folliguet, Antonio Fiore, Floating thrombus in the ascending aorta revealed by peripheral arterial embolism, *Interactive CardioVascular and Thoracic Surgery*, Volume 30, Issue 5, May 2020, Pages 762–764, <https://doi.org/10.1093/icvts/ivaa017>
8. Franchini M, Targher G, Montagnana M, Lippi G. Iron and thrombosis. *Ann Hematol.* 2008 Mar;87(3):167-73. doi: 10.1007/s00277-007-0416-1. Epub 2007 Dec 8.
9. Fayad ZY, Semaan E, Fahoum B, Briggs M, Tortolani A, D'Ayala M. Aortic mural thrombus in the normal or minimally atherosclerotic aorta. *Ann Vasc Surg.* 2013 Apr;27(3):282-90. doi: 10.1016/j.avsg.2012.03.011. Epub 2012 Aug 25.
10. Geha AS, El-Zein C, Massad MG, Bagai J, Khoury F, Evans A, Kpodonu J. Surgery for aortic arch thrombosis. *Thorac Cardiovasc Surg.* 2004 Jun;52(3):187-90. doi: 10.1055/s-2004-817812.