Radiology Case Reports 16 (2021) 348–352

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

A floating thrombus detected by CT in the descending aorta in an obese patient: A possible genetic-environmental interaction ☆,☆☆,★

Barbara Brogna, MD[a,*], *Maria Amitrano, MD*[b], *Sara Mangiacapra, MD*[b], *Enzo Gravino, MD* [a], *Pietro Maurizio Fabio Capasso, MD* [a], *Giuliano Gagliardi, MD* [a], *Lanfranco Aquilino Musto, MD* [a]

[a] *Department of Radiology "San Giuseppe Moscati Hospital" Avellino, Amoretta Street, 83100 Avellino, Italy*
[b] *Department of General Medicine "San Giuseppe Moscati Hospital" Avellino, Amoretta Street, 83100 Avellino, Italy*

ARTICLE INFO

Article history:
Received 4 November 2020
Revised 21 November 2020
Accepted 22 November 2020

Keywords:
Floating aortic thrombus
Computed tomography angiography
Obesity

ABSTRACT

A floating thrombus in a nonaneurysmal, nonatherosclerotic aorta is a rare finding and may represent an unusual source of systemic embolism. Less than 130 cases have been reported in the literature. We describe a rare case of aortic floating thrombus in the descending aorta and the proximal portion of the suprarenal abdominal aorta detected by computed tomography angiography in a 50-year-old woman who was admitted to our emergency room with epigastric abdominal pain. The computed tomography angiography also showed some defects in the subsegmentary pulmonary artery branches along with a splenic infarction with splenic artery and vein thrombi, and a left renal thrombus. On genetic testing the patient resulted heterozygous for the polymorphism for 5,10-methylentetrahydrofolate reductase C677T polymorphism and also with homozygous deletion alleles of the angiotensin-converting enzyme gene. The aortic floating thrombus resolved during anticoagulant therapy after 4 weeks.

☆ Ethical Approval: All procedures performed in the studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Informed consent was obtained from the patient.

☆☆ Funding: No funding was received.

★ Competing Interests: The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

* Corresponding author at: Department of Radiology "San Giuseppe Moscati Hospital" Avellino, Amoretta Street, 83100 Avellino, Italy.
E-mail address: brognabarbara1@gmail.com (B. Brogna).

https://doi.org/10.1016/j.radcr.2020.11.041

Introduction

Thrombosis and embolism are key causes of morbidity and mortality. A floating thrombus in a nonaneurysmal normal aorta represents an unusual source of systemic embolism and is rarely found in clinical practice [1–4]. Aortic floating thrombus (AFT) has been recognized in upwards of 9% of patients with arterial thromboembolism [4,5]. Embolization of the thrombus in the aorta can also be associated with atherosclerosis, dissection, trauma, malignancy, and coagulopathies [6–8]. It appears to be more predominant in female patients [1,8]. The most common locations reported are the descending thoracic aorta and the aortic arch [8–10]. Due to its low incidence, there is currently no consensus on the management of this condition. Therapeutic options are surgical removal, either by aortotomy with endarterectomy, thrombectomy or balloon embolectomy, thrombolysis or anticoagulation [8,11].

We report a particularly interesting case of a pedunculated free-floating thrombus in the descending aorta discovered on computed tomography angiography (CTA) scan in a patient who came to the emergency room due to acute epigastric pain.

Case presentation

A 50-year-old woman with a history of hypertension and obesity (body mass index 37 Kg/m^2) presented to the emergency room at our hospital for acute epigastric abdominal pain and mild dyspnea. She reported a previous episode of pancreatitis. She also reported episodes of metrorrhagia by uterine leiomyomas. The results of laboratory tests revealed severe iron deficiency anemia with a low hemoglobin value (Hb) of 7.8 g/dL (normal range values are 12.1-15.1 g/dL), low mean cell volume of 55.9 FL (normal range values are 80-100FL), low serum iron (<50 μg/dL) (normal range values are 50-170 μg/dL) with a low serum ferritin (storage form of iron) (<30 ng/mL) (normal range values are 30-300 ng/mL). She showed a low level of partial thromboplastin time of 15.3s (normal range values are 26.0-36.0 s), as well as slight increases in the D-Dimer value of 3.11 mg/dL (normal value up to 0.5 mg/dL), lactate dehydrogenase with a value of 396 IU/L (normal range values are 125-220 IU/L) and protein C reactive (CRP) of 1.50 mg/dL (normal range values are 0-5 mg/dL). The other laboratory values were in the normal range.

An emergency chest-abdominal CTA showed a pedunculated mass of 7 × 36mm attached to the distal part of the descending aorta and the proximal portion of the suprarenal abdominal aorta and floating in the lumen (Fig. 1 A,B). The chest CTA revealed some defects in the subsegmentary pulmonary artery branches in terms of the lung inferior lobes with consolidation and pleural effusion on the left side (Fig. 1C,D). On the abdominal scans were also visible a low uptake, characteristic of infarcts, partially involving the spleen, accompanied by luminal defects in both the splenic artery and vein (Fig. 2 A,B), as well as in the left renal vein (Fig. 2C), consistent with thrombi. Ultrasonography of the lower limb showed distal deep vein thrombosis in the right limb.

A further laboratory work-up was carried out for autoimmune and coagulation disorders, such as antiphospholipid antibodies, homocystinemia, protein C, protein S and antithrombin III deficiency, and all these values were within the norm. Due to the patient's anemic condition, emergency hemotransfusion therapy was also started. Given that the patient was obese, along with the risk of surgery and the presence of both arterial and venous thrombi, Fondaparinux at a dose of 10 mg/0.8mL with subcutaneous injection once daily was administered as a bridge to dose-adjusted warfarin, which was continued at 5.0 mg daily.

A genetic test for thrombophilia-related genes was carried out and the patient was found to be heterozygous for the polymorphism for 5,10-methylentetrahydrofolate reductase (MTHFR) C677T polymorphism and also with homozygous deletion (DD) alleles of the angiotensin-converting enzyme (ACE) gene. A gastroscopy and colonoscopy as well as a pelvic magnetic resonance imaging were also performed to rule out occult tumors but the results were normal.

After four weeks a chest abdominal CT scan was repeated, and which showed the resolution of the AFT (Fig. 3A,B) with a reduction in the splenic and vein thrombosis (Fig. 3C) and also of the left renal thrombosis (Fig. 3D). The patient is still under oral anticoagulant therapy and under a strict follow-up.

Discussion

Floating thrombus in a nonaneurysmal, nonatherosclerotic aorta is a diagnostic challenge due to its rarity and due to the risk of distal embolization that requires urgent treatment. Less than 130 cases have been reported [4]. This condition is usually associated with hyperocoagulable disorder, trauma, malignant neoplasm, previous surgery, or turbulent blood flow. However, the exact etiopathogenesis is still unknown. CTA scanning is strongly recommended as a first-choice diagnosing tool for AFT due to its rapid acquisition and high sensitivity [12]. Radiologists should pay attention to some imaging features of an AFT such as location, morphology, size, the aortic segment involved, the presence of concomitant embolism and stent, and dynamic changes during the follow-up [12]. Depending on the morphology, AFT can be sessile and pedunculated [12–14]. The pedunculated thrombus has a greater risk of breaking off to produce a severe peripheral embolism in contrast with the sessile thrombus [12]. Embolization of a floating thrombus is reported in 75% of patients [15].

Also the location is important. An AFT in the ascending aorta and in the aortic arch can be associated with an increased risk of cerebral embolization [3,12]. MTHFR gene polymorphisms have been associated with body mass index-defined obesity, with cardiovascular disease risk, cancer, diabetes, stroke, and venous thrombosis [16–18]. The MTHFR enzyme is important for the folate metabolism, which is an integral process for cell metabolism in the DNA, RNA, and protein methylation. The mutation of the MTHFR gene that causes the C677T polymorphism reduces the activity of this enzyme [17].

Homozygous mutated subjects have higher homocysteine levels, while heterozygous mutated subjects have slightly

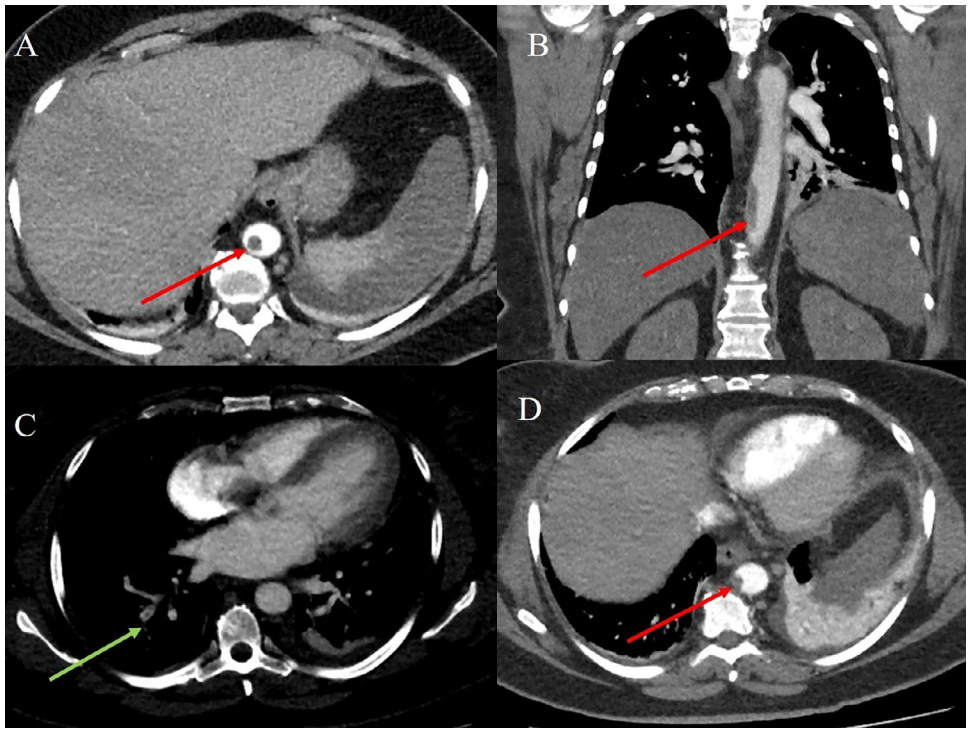

Fig. 1 – A and B show the pedunculated thrombus attached to the distal part of the descending aorta and the proximal portion of the suprarenal abdominal aorta and floating in the lumen (red arrow). There is a luminal defect in a right subsegmentary pulmonary artery branch of the lung inferior lobe (C, green arrow) with consolidation and pleural effusion on the left side and initial consolidation in the right side (D). A small pericardial effusion was also seen (D).

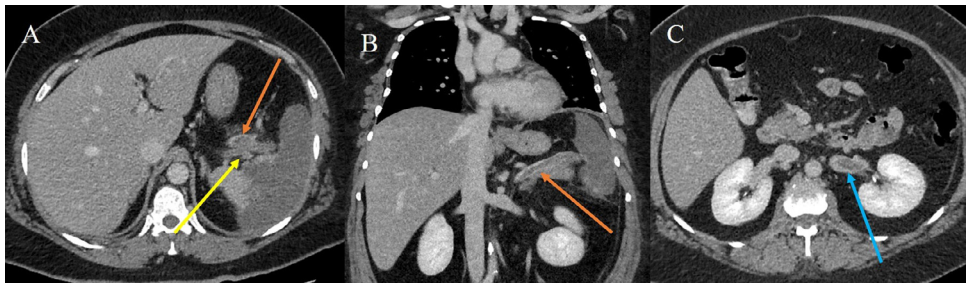

Fig. 2 – A shows the splenic infarction, accompanied by luminal defects in both the splenic artery and vein (yellow and orange arrows, respectively) on axial plane; B shows the extended thrombus in the splenic vein on coronal plane (orange arrow); C shows the left renal vein thrombosis (blue arrow).

higher homocysteine levels than normal, nonmutated controls [17]. However, in our case the homocysteine level was normal. Colak et al [19] reported a case of a floating thrombus of the carotid artery in a patient who was homozygous mutated for the MTHFR C677T genetic polymorphisms and with normal total plasma homocysteine concentration. Alameddine et al [20] described the case of a giant aortic thrombus in a patient with a heterozygous mutation in a gene encoding for 5, 10-methylenetetrahydrofolate reductase (MTHFR) A1298C without hyperhomocysteinemia. Although heterozygous mutation in the gene encoding MTHFR was identified in our patient, it is not clear whether this genetic polymorphism without hyperhomocysteinemia can cause thromboembolic events in the arterial system. The carboxypeptidase ACE catalyzes the conversion of angiotensin I to angiotensin II, a potent vasoconstrictor of the vascular smooth muscle. The ACE gene has an insertion/deletion (I/D) polymorphism in intron 16. Various studies have identified an association or linkage of the D allele of the ACE gene with myocardial infarction, essential hypertension, left ventricular hypertrophy, renal insufficiency and high fasting blood sugar levels [21–23]. In our case, we speculated that given the patient's obesity, also linked to the association of the Methylenetetrahydrofolate reductase (MTHFR) gene polymorphisms and the homozygous deletion (DD) alleles of the ACE gene, may have predisposed our patient to the thrombi formations. Although other cases of AFTs in obese patients have been described, the treatment remains controversial [11,12]. One option is surgical removal, either

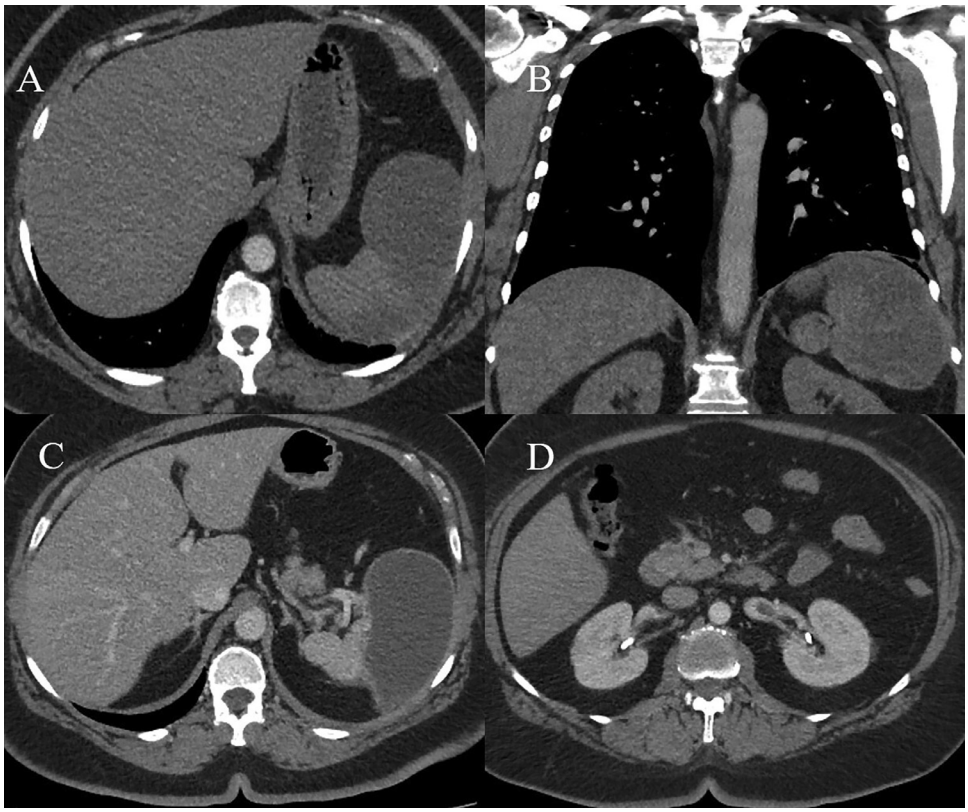

Fig. 3 – The resolution of the AFT (A, B) along with the reduction of the splenic and vein thrombosis (C) and also of the thrombus in left renal vein (D) after 4 weeks of anticoagulant therapy.

by aortotomy with endarterectomy, thrombectomy or balloon embolectomy, and thrombolysis [1,3,4,6,11]. Another option, which avoids the risks of surgery, is medical management with intravenous heparinization followed by oral anticoagulant therapy [4,11].

However anticoagulant therapy can increase the risk of recurrent embolization and should only be performed in hospitals equipped with vascular surgery. Patients who undergo only anticoagulant treatment usually require prolonged hospitalization until transesophageal echocardiography or CTA shows that there has been a significant reduction in the size of the thrombus [11]. The resolution of small mobile aortic thrombi sized 0.5-3 cm, in the descending aorta, under anticoagulant therapy has been reported by Stolberg et al [11] and by Martens et al [4].

In our case the resolution of the AFT was due to anticoagulant therapy and the patient required hospitalization for four weeks. The patient's management was discussed in a multidisciplinary team, and anticoagulant therapy was chosen due to the coexistence of the FAT and venous thrombi.

Conclusion

An AFT may be found incidentally on imaging. Clinicians should be careful to rule out any causes of an AFT including tumors. Our case study suggests that the mechanism underlying aortic thrombus formation is complex and likely multifactorial. It is probably sustained by gene-environment interactions associated with dietary habits and multiple lifestyle factors.

REFERENCES

[1] Weiss S, Bühlmann R, von Allmen RS, Makaloski V, Carrel TP, Wyss TR, et al. Management of floating thrombus in the aortic arch. J Thorac Cardiovas Surg 2016;152(3):810–17. doi:10.1016/j.jtcvs.2016.03.078.

[2] Laperche T, Laurian C, Roudaut R, Steg PG. Mobile thromboses of the aortic arch without aortic debris. A transesophageal echocardiographic finding associated with unexplained arterial embolism. The Filiale Echocardiographie de la Societe Francaise de Cardiologie. Circulation 1997;96:288–94. doi:10.1161/01.cir.96.1.288.

[3] De Maat GE, Vigano G, Mariani MA, Natour E. Catching a floating thrombus; a case report on the treatment of a large thrombus in the ascending aorta. J Cardiothorac Surg 2017;12(1):1–3. doi:10.1186/s13019-017-0600-x.

[4] Martens T, Van Herzeele I, Jacobs B, Vermassen F, et al. Treatment of symptomatic mobile aortic thrombus. Acta Chir Belg 2010;110(3):361–4. doi:10.1080/00015458.2010.11680635.

[5] Girardi Leonard N. Are errors of commission better than errors of omission? J Thorac Cardiovas Surg 2016 2016;152(3):818. doi:10.1016/j.jtcvs.2016.04.061.

[6] Choi JB, Choi SH, Kim NH, Jeong JW. Floating thrombus in the proximal aortic arch. Tex Heart Inst J 2004;31(4):432.

[7] Pousios D, Velissaris T, Duggan S, Tsang G. Floating intra-aortic thrombus presenting as distal arterial embolism. Interact CardioVasc Thorac Surg 2009;9:532–4. doi:10.1510/icvts.2009.206268.
[8] Gueldich M, Piscitelli M, Derbel H, Boughanmi K, Bergoend E, Fiore A, et al. Floating thrombus in the ascending aorta revealed by peripheral arterial embolism. Interact Cardiovasc Thorac Surg 2020;30(5):762–4. doi:10.1093/icvts/ivaa017.
[9] Piffaretti G, Tozzi M, Mariscalco G, Bacuzzi A, Lomazzi C, Rivolta N. et al Mobile thrombus of the thoracic aorta: management and treatment review. Vasc Endovascular Surg 2008;42:405–11. doi:10.1177/1538574408324737.
[10] 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;27:282–90. doi:10.1016/j.avsg.2012.03.011.
[11] Stöllberger C, Kopsa W, Finsterer J. Resolution of an aortic thrombus under anticoagulant therapy. Eur J Cardio-Thorac Surg 2001;20(4):880–2. doi:10.1016/s1010-7940(01)00902-2.
[12] Yang S, Yu J, Zeng W, Yang L, Teng L, Shi H, et al. Aortic floating thrombus detected by computed tomography angiography incidentally: five cases and a literature review. J Thorac Cardiovasc Surg 2017;153(4):791–803. doi:10.1016/j.jtcvs.2016.12.015.
[13] Tsilimparis N, Hanack U, Pisimisis G, Yousefi S, Wintzer C, Rückert RI. Thrombus in the non-aneurysmal, non-atherosclerotic descending thoracic aorta—an unusual source of arterial embolism. Eur J Vasc Endovasc Surg 2011;41:450–7.
[14] Karalis DG, Chandrasekaran K, Victor MF, Ross JJ Jr, Mintz GS. Recognition and embolic potential of intraaortic atherosclerotic debris. J Am Coll Cardiol 1991;17:73–8. doi:10.1016/0735-1097(91)90706-f.
[15] 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(5):1177–80. doi:10.1016/j.jvs.2009.05.052.
[16] Di Renzo L, Marsella LT, Sarlo F, Soldat L, Gratteri S, De Lorenzo A, et al. C677T gene polymorphism of MTHFR and metabolic syndrome: response to dietary intervention. J Transl Med 2014;12(1):329. doi:10.1186/s12967-014-0329-4.
[17] Liew SC, Gupta ED. Methylenetetrahydrofolate reductase (MTHFR) C677T polymorphism: epidemiology, metabolism and the associated diseases. Eur J Med Genet 2015;58(1):1–10. doi:10.1016/j.ejmg.2014.10.004.
[18] Antonaros F, Olivucci G, Cicchini E, Ramacieri G, Pelleri MC, Caracausi M, et al. MTHFR C677T polymorphism analysis: A simple, effective restriction enzyme-based method improving previous protocols. Mol Genetic Genomic Med 2019;7(5):e628. doi:10.1002/mgg3.628.
[19] Colak N, Nazli Y, Kosehan D, Alpay MF, Cakir O. Free-floating thrombus of the carotid artery with a homozygous methylenetetrahydrofolate reductase gene mutation: a case report. Vascular 2013;21(1):35–8. doi:10.1258/vasc.2011.cr0284.
[20] Alameddine AK, Freeman J, Alimov VK, Alamaddine Y, Flack JE, Alimov VK. Giant aortic arch thrombus, methylenetetrahydrofolate reductase (MTHFR) A1298C heterozygous gene mutation, smoking and hormonal replacement therapy. Intern J Med Med Sci 2013;5(2):48–54.
[21] Higaki J, Baba S, Katsuya T, Sato N, Ishikawa K, Ogihara T, et al. Deletion allele of angiotensin-converting enzyme gene increases risk of essential hypertension in Japanese men: the Suita Study. Circulation 2000;101(17):2060–5. doi:10.1161/01.cir.101.17.2060.
[22] Ryan AS, Nicklas BJ, Berman DM, Ferrell RE. The insertion/deletion polymorphism of the ACE gene is related to insulin sensitivity in overweight women. Diabetes Care 2001;24(9):1646–52. doi:10.2337/diacare.24.9.1646.
[23] Shaikh R, Shahid SM, Mansoor Q, Ismail M, Azhar A. Genetic variants of ACE (Insertion/Deletion) and AGT (M268T) genes in patients with diabetes and nephropathy. J Renin Angiotensin Aldosterone Syst 2014;15(2):124–30. doi:10.1177/1470320313512390.