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RESEARCH ARTICLE

WHO'S THE REAL CULPRIT, HYPEREOSINOPHILIA OR CORONARY ARTERY DISEASE? A CASE REPORT OF THROMBOEMBOLIC EVENTS IN A LEFT VENTRICULAR THROMBUS.

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Abstract

Left ventricular thrombosis (LVT) is a significant issue resulting from ischemia and non-ischemic cardiomyopathy. Despite a decrease in prevalence due to reperfusion therapy, it remains a significant concern. Diagnosis is often based on transthoracic echocardiography, but it may yield inconclusive results. Advanced imaging, like cardiac magnetic resonance, is needed to accurately characterize intraventricular masses. The formation of LV thrombus is associated with a 5.5-fold increased risk of thromboembolic events compared with no thrombus (1). Without anticoagulation, the annual stroke or systemic embolization rate is approximately 10% to 15% per year. The clinical outcomes vary based on the locations of embolization. Since these thromboembolic events are generally sudden and unpredictable.

Vitamin K antagonists are traditionally used for treating LVT, but recent guidelines classify direct oral anticoagulants (DOACs) with the same level of evidence as vitamin K antagonists (VKAs) and recommend an individualized approach for both prevention strategies and treatment duration with regard to the therapeutic strategy for LVT.

Attention should be paid to the risk of bleeding. The choice of anticoagulation regimen should be tailored to the individual patient.

Introduction:-

Left ventricular thrombosis (LVT) is a significant issue resulting from ischemia and non-ischemic cardiomyopathy. Despite a decrease in prevalence due to reperfusion therapy, it remains a significant concern. Diagnosis is often based on transthoracic echocardiography, but it may yield inconclusive results. Advanced imaging, like cardiac magnetic resonance, is needed to accurately characterize intraventricular masses. The formation of LV thrombus is associated with a 5.5-fold increased risk of thromboembolic events compared with no thrombus (1). Without anticoagulation, the annual stroke or systemic embolization rate is approximately 10% to 15% per year. The clinical

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Case Presentation.

We present an intriguing case of a 60-year-old Portuguese-born patient with asthma under bronchodilators who has recently stopped smoking. He was admitted two months before for myopericarditis; the myocardial function was normal then without any wall motion abnormalities. Despite our request, he did not perform a cardiac MRI.

His story had begun in Portugal with the onset of left hemiparesis, related to an ischemic stroke diagnosed on a brain MRI upon admission due to a left intraventricular thrombus found on a cardiac TTE.

The patient was initiated on intravenous heparin, which was further complicated by a large retroperitoneal hematoma of the left psoas, measuring 12cm x 9cm x 8.5cm on the abdominal-pelvic CT scan, resulting in the onset of hemorrhagic shock on day 2 of hospitalization. He underwent many transfusions, reaching 5 units of packed red blood cells and 2 units of fresh frozen plasma (FFP), in addition to anticoagulation cessation. He responded well to the conservative line of treatment with stable vitals, and the hematoma was shown to be radiologically stable in subsequent scans. Additionally, renal and splenic emboli were identified. The initial labs showed hypereosinophilia (12 G/L), normal renal and hepatic function, and a normal coagulation profile.

Echocardiogram showed wall motion abnormalities in the apex and inferolateral wall; FE was estimated to be 35%. Coronary angiography was not conducted then.

The patient arrived at our center following a month of hospitalization in Portugal for additional medical care. He was undergoing the following cardiac treatments : low-dose heparin, an ACE inhibitor, statins, and beta-blockers with aldosterone antagonists. We repeated the abdominal CT scan (figure 1) and observed no expansion of the hematoma. We restarted the anticoagulation on 110 mg of low-dose dabigatran twice daily for two reasons: first, to evaluate the hematoma after reintroducing curative anticoagulation, and second, since an antidote, idarucizumab, was available.

Based on all this information, we performed and cardiac MRI in the context of suspected hyper-eosinophilic endomyocardial fibrosis and a viability search in case of proven ischemic cardiomyopathy, which returned in favor of an ischemic pattern with an apical thrombus in the LV without viability (figure 2). FE is estimated to be 35%.

Consequently, we administered aspirin; however, the patient had an episode of melena that necessitated a gastroscopy, which disclosed no lesions. Although he underwent a colonoscopy fewer than six months prior without abnormalities. Additional assessment and monitoring are recommended to determine the appropriate course of treatment.

In the context of hypereosinophilia (HE), the patient received a course of injectable corticosteroids followed by oral steroids at a dosage of 1 mg/kg during his hospital stay in Portugal. We performed a complete screening, which included an evaluation of the potential causes of HE and an infectious workup that involved parasitic serology, fungal studies, HIV, and syphilis, and revealed no significant findings. However, the patient received a course of empiric antiparasitic treatment. Furthermore, the evaluation returned negative for antinuclear antibodies(ANA) , antineutrophil cytoplasmic antibodies (ANCA), double-stranded deoxyribonucleic acid (dsDNA) antibody screen, myeloperoxidase (MPO) antibodies (Ab), and proteinase 3 (PR3) antibodies. It is worth mentioning that extensive imaging studies for malignancies were negative, ruling out another potential cause of secondary HE. HE syndrome

was considered to be the most likely underlying cause of HE. The patient was currently taking 10 mg of prednisolone, and the eosinophil count was 0.52 G/l.

The patient is discharged after the optimization of heart failure treatment with antiepileptic medications. We requested neurosurgical advice for the meningioma seen in the brain MRI (figure 3).

Follow Up.

The patient was assessed three months later, with a stable cardiac situation, improved left ventricular systolic function, no apical thrombus on echocardiogram, and a reduction in the hematoma of the left iliac psoas muscle on CT scan. Aspirin was withdrawn by his physician because of recurring epistaxis.

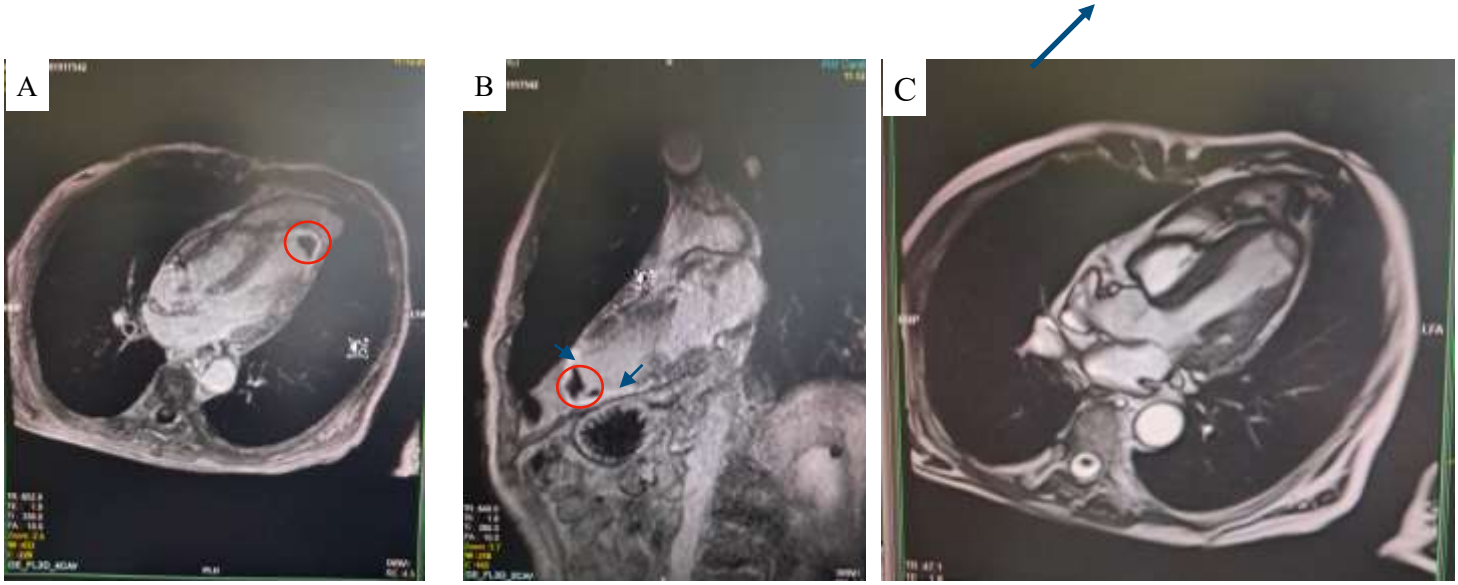
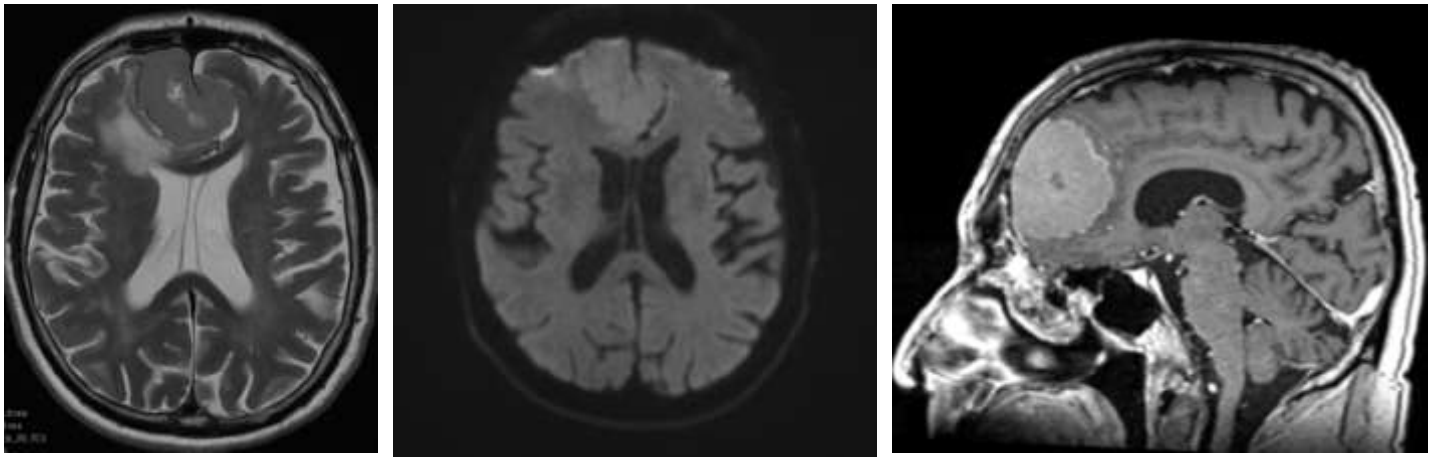


Figure 1 : Cardiac MRI : SSFP four-chamber axial view (A) showing wall thickness in the apex of the left ventricle. Late gadolinium enhancement sequences (B,C) showing left ventricular apical thrombus (red circles) and transmural enhancement (blue arrows) in the same area.

Figure 2: Abdominal and pelvic CT scan revealed a left iliopsoas hematoma measuring approximately 9×10×18cm (arrows)

Figure 3 : Brain MRI showing right frontal meningioma of 4cm and right parenchymal reaction. Probable right occipital ischemic sequelae



Discussion.

Left ventricular thrombus is a recognized consequence of myocardial infarction, especially in ST-elevation myocardial infarction affecting the anteroapical wall with concomitant wall motion abnormalities, and serves as a potential precursor to embolic events [1]. The integration of rapid reperfusion strategies, initially by thrombolysis and later via percutaneous coronary intervention (PCI), into standard treatment protocols for STEMI seems to have reduced, but not eliminated, the risk of left ventricular thrombus development. A 2018 meta-analysis by Bulluck et al. [2] showed a predominant association between LVT and anterior STEMI, with 96% of the thrombi occurring in this infarct location.

In some autoimmune diseases, such as Behçet‘s disease , uncommon cardiac involvement can lead to LVT formation in 1.5% of cases [3], as well as in the hypereosinophilic syndrome, in which—after an initial phase of eosinophil infiltration—the denuded myocardium reacts with a thrombotic-necrotic process in both ventricles, particularly in the ventricular apices [4]. Cardiac involvement manifests in three stages: an initial necrotic stage characterized by eosinophilic infiltration resulting in endocardial microabscesses, a subsequent thrombotic-necrotic stage marked by thrombus formation on the exposed myocardium of both ventricles, and a final fibrotic stage that includes endomyocardial fibrosis, scarring of the chordae tendinae, and symptoms indicative of congestive heart failure [4,5].

At present, cardiac magnetic resonance (CMR) is recognized as the standard technique for LVT detection with the highest diagnostic accuracy for LVT, followed by echocardiography[8].

The superiority of CMR with gadolinium compared to other imaging modalities for the detection of LVT is derived from the immediate strong enhancement of the LV cavity, allowing the detection of abnormal intraventricular structures, as well as the delayed-enhancement technique [6]. Detection of LV thrombus on long TI LGE CMR images was based on its location within the LV cavity and a homogeneous black appearance due to the absence of vascularity, surrounded by structures with contrast uptake (LV cavity and myocardium)[7].It also provides quantitative data regarding cardiac function, perfusion, and vitality .But, on the other hand, CMR has a higher cost and is not widely available, even in developed countries.

In case of endomyocardial fibrosis , sequences after contrast administration confirm the diagnosis by showing typical subendocardial enhancement that extends from the subvalvular regions to the apices of both ventricles. Thrombi are often observed at the apex of the left ventricle and/or right ventricle [4]. In our case, CMR showed an apical thrombus associated with a transmural enhancement supporting the ischemic origin and lack of myocardial viability.

The reported risk of embolic events from an LVT post-MI ranges from 6.1% to 86% and seems to be greatest in the first 3 months after MI [9]. Multiple studies have demonstrated that thrombus characteristics associated with systemic embolism include protrusion into the LV cavity, mobility independent of myocardium, patient age >68, thrombus area, length of the thrombi in the lumen, and LVT recurrence [10]. Embolic events appear to occur even after resolution of LVT, suggesting that anticoagulant therapy needs to be considered for a longer period in some cases.

VKAs, predominantly warfarin, have traditionally been used and recommended for the prevention and treatment of LV thrombus . Anticoagulation with warfarin, however, requires dietary consistency, frequent INR monitoring, and vigilance with regard to drug-food and drug-drug interactions that can be challenging for many patients. Failure to maintain a therapeutic INR (TTR < 50%) appears to increase the risk of stroke in patients with LV thrombus [11, 12]. These challenges have led to increased adoption of DOAC for oral anticoagulation treatment in atrial fibrillation and venous thromboembolism. In current practice, some practitioners have extrapolated the results of studies of DOAC for atrial fibrillation and venous thromboembolism to LV thrombosis and are using it for such treatment. The ideal duration of anticoagulation for LVT treatment in non-ischemic cardiopathy remains contentious; however, an initial treatment period of 3–6 months is judicious in cases of ischemic cardiopathy, with potential extension based on follow-up results [12]. Robust randomized studies are essential to assess the potential function of preventive

anticoagulation in high-risk patients and to furnish definitive evidence for the application of DOACs in LVT treatment.

The anticoagulation complications are challenging, especially when interruption of anticoagulation may threaten vital and functional outcomes. Therefore, a careful evaluation is essential, since no clinical guidelines are available. The occurrence of a spontaneous iliac or psoas muscle hematoma is an infrequent complication of anticoagulation. Until further evidence is available, the treatment approach should be chosen according to the clinical and hemodynamic stability of the patient, the severity of the potential neurological sequelae, and the risks associated with the suspension of anticoagulation [13].

The therapy is dependent on the timing of diagnosis and the hemodynamic condition. In cases of early diagnosis, treatment may be conservative, involving the cessation of anticoagulants, provision of an antidote for overdose, and transfusion therapy. Embolization procedures may be beneficial in cases of active hemorrhage. Surgical evacuation of the hematoma may be necessary in severe cases of uncontrollable hemodynamic collapse or significant neuropathic issues resulting from femoral nerve compression [14]. In the current case, interventional treatment was unnecessary since the patient achieved hemodynamic stability with conservative management.

Conclusion

LVT is more prevalent in patients with anterior STEMI (involving the apex) and reduced EF. The management approach must be adapted to the clinical context and the patient's risk of bleeding. This case highlights the importance of close monitoring and the efficacy of DOACs in treating LV thrombosis following three months of therapy. In the future, data from large randomized controlled trials are needed for the most effective methods .

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Declaration of conflicting interests

None.

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References.

1. Vaitkus PT, Barnathan ES. Embolic potential, prevention, and management of mural thrombus complicating anterior myocardial infarction: a meta-analysis. J Am Coll Cardiol. 1993;22:1004–1009. doi: 10.1016/0735-1097(93)90409-t[Google Scholar] [CrossRef] [PubMed]

2. Bulluck, H.; Chan, M.H.H.; Paradies, V.; Yellon, R.L.; Ho, H.H.; Chan, M.Y.; Chin, C.W.L.; Tan, J.W.; Hausenloy, D.J. Incidence and predictors of left ventricular thrombus by cardiovascular magnetic resonance in acute ST-segment elevation myocardial infarction treated by primary percutaneous coronary intervention: A meta-analysis. J. Cardiovasc. Magn. Reson. 2018, 20, 72. [Google Scholar] [CrossRef]

3. Ben Ghorbel, I.; Belfeki, N.; Houman, M.H. Intracardiac thrombus in Behçet‘s disease. Reumatismo 2016, 68, 148–153. [Google Scholar] [CrossRef]

4. Caudron J., Arous Y., Fares J., Lefebvre V., Dacher J.N. Endomyocardial fibrosis in the context of hypereosinophilic syndrome: the contribution of cardiac MRI. Diagn Interv Imag. 2012;93(10):790–792. doi: 10.1016/j.diii.2012.04.019. [DOI] [PubMed] [Google Scholar]

5. Salanitri, G.C. Endomyocardial Fibrosis and Intracardiac Thrombus Occurring in Idiopathic Hypereosinophilic Syndrome. Am. J. Roentgenol. 2005, 184, 1432–1433. [Google Scholar] [CrossRef]

6 .Weinsaft JW, Kim HW, Shah DJ, Klem I, Crowley AL, Brosnan R, James OG, Patel MR, Heitner J, Parker M, et al. Detection of left ventricular thrombus by delayed-enhancement cardiovascular magnetic resonance prevalence and markers in patients with systolic dysfunction. J Am Coll Cardiol. 2008;52:148–157. doi: 10.1016/j.jacc.2008.03.041[Google Scholar] [CrossRef] [PubMed]

7. Sürder, D.; Gisler, V.; Corti, R.; Moccetti, T.; Klersy, C.; Zuber, M.; Windecker, S.; Moschovitis, A.; Kozerke, S.; Lüscher, T.F.; et al. Thrombus formation in the left ventricle after large myocardial infarction—Assessment with cardiac magnetic resonance imaging. Swiss Med. Wkly. 2015, 145, w14122. [Google Scholar] [CrossRef]
8. P.U. Ogbogu, D.R. Rosing, M.K. Horne 3rd Cardiovascular manifestations of hypereosinophilic syndromes Immunol Allergy Clin North Am, 27 (2007), pp. 457-475
9. Visser CA, Kan G, Meltzer RS, Dunning AJ, Roelandt J. Embolic potential of left ventricular thrombus after myocardial infarction: a two-dimensional echocardiographic study of 119 patients. J Am Coll Cardiol. 1985;5:1276–1280. doi: 10.1016/s0735-1097(85)80336-3 [Google Scholar] [CrossRef] [PubMed
10 .Oh, J.K.; Park, J.-H.; Lee, J.-H.; Kim, J.; Seong, I.-W. Shape and Mobility of a Left Ventricular Thrombus Are Predictors of Thrombus Resolution. Korean Circ. J. 2019, 49, 829–837. [Google Scholar] [CrossRef]
11. Kouvaras G, Chronopoulos G, Soufras G, Sofronas G, Solomos D, Bakirtzis A, Pissimissis E, Tzonou A, Cokkinos D. The effects of long-term antithrombotic treatment on left ventricular thrombi in patients after an acute myocardial infarction. Am Heart J. 1990;119:73–78. doi: 10.1016/s0002-8703(05)80084-5[Google Scholar] [CrossRef] [PubMed]
12. Holbrook A, Schulman S, Witt D, et al. Evidence-based management of anticoagulant therapy. Chest 2012;141 (Suppl):e152–84S. 10.1378/chest.11-2295 [DOI] [PMC free article] [PubMed] [Google Scholar]
13. Sunga K, Bellolio F, Gilmore R, et al. Spontaneous retroperitoneal hematoma: etiology, characteristics, management, and outcome. J Emerg Med 2012;43:e157–61. 10.1016/j.jemermed.2011.06.006 [DOI] [PubMed] [Google Scholar]
14. Chan Y, Morales J, Reidy J et al. Management of spontaneous and iatrogenic retroperitoneal haemorrhage: conservative management, endovascular intervention or open surgery? Int J Clin Pract 2008;62:1604–13. 10.1111/j.1742-1241.2007.01494.x [DOI] [PubMed] [Google Scholar]
15- Levine GN, McEvoy JW, Fang JC, et al. Management of Patients at Risk for and With Left Ventricular Thrombus: A Scientific Statement From the American Heart Association. Circulation 2022;146:e205-23. [Crossref] [PubMed]