Left ventricular mass: Myxoma or thrombus?

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ABSTRACT

Patient with embolic episode should always be evaluated for cardiac mass. Mass in left ventricle can be a myxoma or thrombus even in a normal functioning heart. In either case, mobile mass with embolic potential should be surgically resected.

Key words: Left ventricle; Myxoma; Thrombus

INTRODUCTION

Patient with embolic episode should always be evaluated for cardiac masses. Mass in the left ventricle (LV) can be a myxoma or thrombus even in a normal functioning heart. In either case, mobile mass with embolic potential should be surgically resected.[1-3]

CASE REPORT

A 29-year-old male patient presented with left hemiparesis and dysarthria. Computed tomography scan revealed the embolic infarct in right middle cerebral artery territory. He was managed conservatively with an anticoagulant warfarin keeping international normalized ratio value between 2 and 3. Patient was evaluated and he was found to have pedunculated mass in LV on transthoracic echocardiography. He recovered gradually and was referred to our center for further management after 15 days of the diagnosis. Echocardiographic examination showed 0.9 × 1.7 cm homogenous mass present in LV toward apex attached to the interventricular septum (IVS). No LV regional wall motion abnormality was detected with LV ejection fraction of 65%. Patient had normal sinus rhythm without any arrhythmia or ischemia changes in electrocardiogram. The mass was presumed to be myxoma and considering the potential for embolization, patient was scheduled for open heart surgery next day to remove the mass. After smooth induction and intubation, intraoperative transesophageal echocardiography was done. Mobile LV mass (size 1.1 × 1.8 cm) attached to IVS was seen [Figures 1 and 2, Video Clips 1 and 2]. Another LV mass attached to left ventricular outflow tract (LVOT) was noticed, but there was no LVOT obstruction as evident on insignificant LVOT gradient [Figure 3 and Video Clip 3]. After going on cardiopulmonary bypass, left ventriculotomy on arrested heart was performed on the anterior wall distally parallel to left anterior descending artery. Intraoperatively a pedunculated mass of size 2 × 1.5 cm attached to the IVS was seen. Another calcific fibrotic sessile mass of size 2 × 2 cm was observed on LV wall in outflow tract. Endocardium appeared to be thickened and fibrotic. After the surgery, patient was weaned off bypass smoothly. Resected mass was sent for histopathological examination. Microscopic examination revealed organized and calcified thrombus. No classical or diagnostic features of cardiac myxoma were observed. Patient was started on oral anticoagulants; however thrombophilia profile could not be evaluated due to noncompliance of the patient.
Primary cardiac tumors are uncommon with incidence of 0.02%. Benign cardiac myxomas constitute 88% of cardiac tumor cases. LV myxomas account for only 2.5% of cases. The clinical features of LV myxoma are mostly caused by embolization and obstruction to LVOT. Arrhythmias, conduction disturbances, and LV dysfunction can also be seen. Embolic phenomena in LV myxoma are more common than LA myxomas, occurring in 64% of patients with LV myxoma. Considering the risk for embolization, myxomas should be surgically resected as early as possible.

Thrombus formation in LV is well-known complication in systolic heart failure (incidence 10–30%) and after acute myocardial infarction (incidence 5–15%). Hypercoagulable state or undetectable disorder of the endocardium can lead to ventricular thrombus formation even in the normal heart. Main causes of Inherited thrombophilia are G1691A mutation of factor V gene, G20210A mutation of the prothrombin gene, antithrombin deficiency, protein C and protein S deficiency. LV thrombus formation is also associated with antiphospholipid antibody syndrome and hypereosinophilic syndrome. Autoimmune disorders like Adamantiadis-Behcet’s disease and lupus erythematosus, have been suggested to cause left ventricular thrombus formation. Large doses of alpha epoetin administration causing spontaneous LV thrombus formation has been reported. The rapid increase of hematocrit due to alpha epoetin has been suggested as the reason. Author has cautioned about the use of erythropoietin and hematocrit should not rise >30% above baseline within 10 days. Studies have reported that iron deficiency anemia has been implicated as a cause of systemic thromboembolism. Akins et al. suggested iron deficiency anemia induces turbulence by decreasing viscosity and increasing velocity and thrombosis may be caused by endothelial injury due to turbulent blood flow.

A pedunculated thrombus moving throughout the cardiac cycle has a high tendency to embolize despite adequate anticoagulation. Treatment for such thrombi has included thrombectomy, anticoagulation, or thrombolysis. Surgical removal by ventriculotomy may cause deterioration of LV function and potentially induce ventricular arrhythmia. Transaortic video-assisted removal of an LV thrombus, and also the trans-left atrial appendage and mitral valve approach, can be alternatives and can provide good LV visualization. However, video-assisted cardioscopy can be associated with serious potential complications.
Anticoagulation (high-dose intravenous heparin or low-molecular-weight heparin) has variable resolution rates 13–59%,[25] Rester et al.[26] have successfully used recombinant tissue plasminogen activator for lysis of a mobile, pedunculated LV thrombus in a patient with peripartum cardiomyopathy and evidence of systemic embolization. However, thrombolysis carries high risk of hemorrhagic or embolic complications.

In the present case, LV mass that was supposed to be myxoma preoperatively was found out to be a thrombus on histopathological examination. Outcome of patients with recurrent emboli from mobile, pedunculated thrombi who are treated conservatively is generally very poor compared with that of a limited number who undergo surgery.[27,28] Pedunculated globular thrombi connected to the endocardium by a very narrow stalk and moving freely within the LV lumen carries 60–80% risk for embolization.[29] Hence, such patients with a history of embolization should be treated surgically as early as possible. Causes for spontaneous LV thrombus formation in a normal heart should be evaluated thoroughly. Smooth anesthesia induction in patients with pedunculated cardiac masses will lessen the possibility of embolization. Real-time transesophageal echocardiography monitoring would help the surgeon in deciding the approach.

REFERENCES

1. Sarjeant JM, Butany J, Cusimano RJ. Cancer of the heart: Epidemiology and management of primary tumors and metastases. Am J Cardiovasc Drugs 2003;3:407-21.
2. Korkmaz AA, Tamtekin B, Onan B, Demir AS, Guden M, Uckurt Y. Combination of right atrial and left ventricular myxoma. Ann Thorac Surg 2010;89:e33-5.
3. Meller J, Teichholz LE, Pichard AD, Matta R, Litwak R, Herman MV, et al. Left ventricular myxoma: Echocardiographic diagnosis and review of the literature. Am J Med 1977;63:816-23.
4. Colucci W, Braunwald E. Primary tumors of the heart. In: Braunwald E, editor. Heart Disease: A Textbook of Cardiovascular Medicine. 4th ed., Vol. 2. Philadelphia: WB Saunders; 1992. p. 1451.
5. Buke A, Virmani R. Cardiac myxoma: A clinicopathologic study. Am J Clin Pathol 1993;100:671-80.
6. Mobeirek AF, Al-Nozha M. Multiple left ventricular myxoma: Case report and review of the literature. J Saudi Hear Assoc 1996;8:122-6.
7. Samdarshi TE, Mahan EF 3rd, Nanda NC, Guthrie FW Jr, Bernstein JJ, Kirklin JW. Transesophageal echocardiographic diagnosis of multicentric left ventricular myxomas mimicking a left atrial tumor. J Thorac Cardiovasc Surg 1992;103:471-4.
8. Seethala S. Left ventricular myxoma: Missed vs metastatic. World J Cardiol 2013;5:387-90.
9. Kalra A, Jang IK. Prevalence of early left ventricular thrombus after primary coronary intervention for acute myocardial infarction. J Thromb Thrombolysis 2000;10:133-6.
10. Gotttiener JS, Gay JA, Van Voorhees L, DiBianco R, Fletcher RD. Frequency and embolic potential of left ventricular thrombus in dilated cardiomyopathy: Assessment by 2-dimensional echocardiography. Am J Cardiol 1983;52:1281-5.
11. Verma AK, Alam M, Rosman HS, Brymer J, Keith F. Systemic embolization from thrombus in normal left ventricle. Chest 1988;93:441-2.
12. Lane DA, Mannucci PM, Bauer KA, Bertina RM, Bochek NP, Boulyjenkov V, et al. Inherited thrombophilia: Part I. Thromb Haemost 1996;76:651-62.
13. Aguilar JA, Summerson C. Intracardiac thrombus in antiphospholipid antibody syndrome. J Am Soc Echocardiogr 2000;13:873-5.
14. Ejima J, Ohmura I, Kaji Y, Tsuda Y, Kanaya S, Fujino T. Diffuse endocardial thrombus in left ventricle associated with a case of hypeereosinophilic syndrome. Jpn Heart J 1991;32:267-72.
15. Vanhalewey G, el-Ramahi KM, Hazmi M, Sieko J, Zaman L, Fawzy M. Right atrial, right ventricular and left ventricular thrombi in (incomplete) Behcet’s disease. Eur Heart J 1990;11:957-9.
16. Barjatya MK, Shah NK, Kothari SS, Shah PP, Trivedi HL. Spontaneous left ventricle cavity thrombus in a patient of systemic lupus erythematosus. J Assoc Physicians India 1992;40:195-6.
17. Karabinos IK, Koulouris S, Kranidis A, El Ali M, Marsonis A, Kokkinou V, et al. Spontaneous thrombus formation in a normal left ventricle following administration of large doses of epiopetin. Hellenic J Cardiol 2007;48:44-6.
18. Dubyk MD, Card RT, Whiting SJ, Boyle CA, Zlotkin SH, Paterson PG. Iron deficiency anemia prevalence at first stroke or transient ischemic attack. Can J Neurol Sci 2012;39:189-95.
19. Chang YL, Hung SH, Ling W, Lin HC, Li HC, Chung SD. Association between ischemic stroke and iron-deficiency anemia: A population-based study. PLoS One 2013;8:e82952.
20. Nakamizo T, Ishikawa K, Amari K. Simultaneous thrombosis in a normal left ventricle and normal carotid artery in a patient with a stroke secondary to Iron deficiency anemia. J Med Cases 2014;5:351-4.
21. Akins PT, Glenn S, Nemeth PM, Derdeyn CP. Carotid artery thrombus associated with severe iron-deficiency anemia and thrombocytosis. Stroke 1996;27:1002-5.
22. Nili M, Deviri J, Jortner R, Strasberg B, Levy MJ. Surgical removal of a mobile, pedunculated left ventricular thrombus: Report of 4 cases. Ann Thorac Surg 1988;46:396-400.
23. Chamsi-Pasha MA, Anwar AM, Nosir YF, Chamsi-Pasha H. “Hanging by a thread” left ventricular thrombus in an asymptomatic soldier. Saudi Med J 2009;30:436-8.
24. Kuh JH, Seo Y. Transatrial resection of a left ventricular thrombus after acute myocarditis. Heart Vessels 2005;20:230-2.
25. Heik SC, Kupper W, Hamm C, Bleifeld W, Koschyk DH, Waters D, et al. Efficacy of high dose intravenous heparin for treatment of left ventricular thrombi with high embolic risk. J Am Coll Cardiol 1994;24:1305-9.
26. Rester BT, Warnaux JL, Patel PB, McMullan MR, Skelton TN, Collop NA. Lysis of a left ventricular thrombus with recombinant tissue plasminogen activator. Chest 2001;120:681-3.
27. Rehan A, Kanwar M, Rosman H, Ahmed S, Ali A, Gardin J, et al. Incidence of post myocardial infarction left ventricular thrombus formation in the era of primary percutaneous intervention and glycoprotein IIb/IIIa inhibitors. A prospective observational study. Cardiovasc Ultrasound 2006;4:20.
28. Bakhtiari RE, Khaledifar A, Kabiri M, Danesh Z. Mobile pedunculated left ventricular masses in a man with recurrent emboli. Heart Views 2012;13:146-8.
29. Glikson M, Agranat O, Ziskind Z, Kaplinski E, Vered Z. From swirling to a mobile, pedunculated mass – The evolution of left ventricular thrombus despite full anticoagulation. Echocardiographic demonstration. Chest 1993;103:281-3.