University Heart Journal
Vol. 16, No. 1, January 2020

REVIEW ARTICLES

Echocardiographic Imaging of Intracardiac Thrombus and Evaluated its Underlying cause along Therapeutic Implications: A Review

MOSTASHIRUL HAQUE, MOHAMMAD FAISAL IBN KABIR, MD. RASUL AMIN, MD.FAKHRUL ISLAM KHALED, RAYHAN MASUM MANDAL, S M MUSTAFA ZAMAN, HARISUL HOQUE, FAZLUR RAHMAN,CHOUDHURY MESHKAT AHMED

Dept. of Cardiology, BSMMU, Dhaka

Address of Correspondence: Dr. Mostashirul Haque, Assistant Professor, Dept. of Cardiology, BSMMU, Dhaka

E-mail: mostashirul@gmail.com

University Heart Journal 2020; 16(1): 40-42

Introduction:

Transesophageal echocardiography(TEE) is considered to be superior to the transthoracic echocardiography(TTE) for detecting cardiovascular sources of embolism because of lack of intervening lungs and bone, and the use of higher-frequency imaging transducers provide enhanced spatial resolution and improved detection of intra-cardiac thrombi and spontaneous left atrial echo contrast, a marker of blood stasis.[1] Clinical detection of ventricular thrombi is generally performed by TTE and evaluation of atrial thrombi is generally performed by TEE.[2]

Predisposing factors for left atrial (LA) thrombus are mitral valve pathology, prosthetic mitral valve, poor LV function and abnormal LA contractile function, such as atrial fibrillation(AF).Enlarged and bifid left atrial appendage (LAA) is among the structural or anatomical risk factors.

The LA can be visualized on two-dimentional TTE from all of standard imaging windows. Image of the thrombi in the LA resembles intracavitary masses. The thrombi usually have smooth contours and move synchronously with adjacent heart wall during the heart cycle. Scanning at least two or more different views is warranted for accurate diagnosis of thrombus. Sometimes, thrombi are visualized as an echo-lucent structure and difficult to be described. In such cases, the diagnosis may be confirmed by the use of intravenous echo-contrast agent to improve the discrimination between blood and intra-cavitary masses.[3] Unless thrombi are very large and spreading to the body of the LA, they are hardly identified by means of TTE because the LA lies in the far field of the interrogating ultrasound beam.

The ability of TTE to identify or to exclude LA or LAA thrombi is limited, with a reported sensitivity of 40-60%, due largely to poor visualization of the body of LAA. In contrast, TEE provides detailed visualization of the LA and LAA from multiple imaging planes, so offers superior assessment. TEE is the current gold standard diagnostic method with a reported sensitivity and specificiity for LA thrombi are 93-100% and 99-100%, respectively.[4]

The spontaneous echo contrast (SEC) or smoke like echo which indicates the predisposing stasis, almost always accompanies thrombus, especially among those with AF or left atrial enlargement and may be helpful in the differentiation of thrombi from tumor or normal anatomy. SEC is detected by using a high frequency ultrasound-transducer(>5 MHz, as used in TEE) and high gain settings. Although most widely studied in the LA and LAA, SEC may also occur in RA.

LAA is sometimes seen in the parasternal short axis through the cardiac base and the apical two chamber view. However, LAA is best evaluated by TEE at mid-esophageal view with transducer between 0 and 150 degree along the LAA axis.[5] The ability to estimate blood flow velocity in the left atrium and atrial appendages offers a more quantifiable measure of stasis. LAA mechanical function can be evaluated with TEE utilizing PWD measurement of LAA emtying and filling velocites. The velocity of blood flow at the orifice of the LAA can be sampled by using PWD, with a low Nyquist limit and low wall- filter settings. The PWD sample volume should be placed 1 cm inside LAA at transducer angles between 45 and 120. It

Received: 22 October, 2019 **Accepted:** 01 December, 2019

reveals a characteristic pattern that is dependent upon the patients underlying rhythm and atrial function. In patient with sinus rhythm, there are well-defined filling and emptying waves (peak emptying velocity>55 cm/s). A low appendage blood flow velocity (<25 cm/s) is associated with the presence of appendage thrombus and denser SEC.[6] Thrombogenic risk raises with decreasing LAA velocity. The risk risk of stroke increases sharply with reductions in blood flow velocity(<15cm/s), particularly in the left appendage or posterior LA.[7] Above a thresold LAA velocity >55 cm/s, thrombus was ruled out because this velocity has a negative predictive value of 100%.

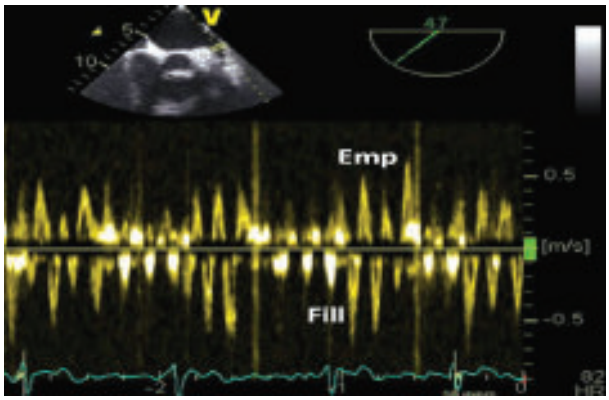

Fig.-1: *Measuring LAA emptying velocity by PWD sample volume within 1 cm inside LAA*

Left ventricular thrombus is most often seen among patients with extensive anterior ST elevation myocardial infarction complicated with anteroapical aneurysm formation. Patients with poor LV function are prone to stasis and thrombus formation. LV thrombus is seen more commonly in patients with dilated ventricle. Echocardiographic indices that correlate with LV thrombosis are low ejection fraction (LVEF <40%), high LV wall motion scores indices, and high E/Em ratio.[3]

The TTE appearance of LV thrombus is variable, and depends upon its age. Recent or actively forming thrombus may appear echo-lucent , and is highly mobile and disposed to protrude into the center of the ventricular cavity. Older thrombus generally has smooth cavitary surface and texture resembling the ultrasound appearance of hepatic tissue. Patients with subacute, protruding, echo-lucent, and mobile thrombi are at higher risk of embolic events, compared with those presented with sessile, laminated, and organized thrombi. Thrombus has higher echogenicity than the adjacent myocardium.[8]

The sensitivity of TTE for detection of LV thrombus may be suboptimal due to poor image quality and the difficulty in differentiation of thrombus from normal trabeculation. In patients with suboptimal acoustic windows or prominent LV apical muscle bands and trabeculations which confound the recognition of thrombus, many experts recommend the use of intravenous contrast agents to enhance the visibility of LV apex to improve the sensitivity and specificity of thrombus detection by TTE.[9]

Imaging of LV apex is difficult by TEE. Because thrombus is often located in the LV apex, it should be suspected when an akinetic cardiac apex is thickened and rounded. A foreshortened image of the LV apex can be minimized by retro flexing the tip of the TEE probe. A deep transgastric view(0-20) may provide better resolution because it is within the near field of the transducer. To distinguish thrombi from artifacts, it is necessary to acquire optimal images in at least two different views, throughout the cardiac cycle. Laminated thrombi may be difficult to visualize clearly, and endocardial border delineation may be improved by a higher transducer frequency(>5MHz) and contrast echocardiography. [3]Thrombi are common at the LV apex, in aneurysms, pseudo aneurysm and deep recesses, or between the trabeculations in which intertrabecular flow can be demonstrated with color-flow doppler and contrast-enhanced echocardiography.

Right atrial thrombi: Thrombus formation is less common in the right atrium compared with the left atrium. Right heart thrombi are most often originated by embolization from a peripheral venous source. Right atrial wall damage by intracardiac devices may be responsible for RA thrombi.[10] Predisposing factors in situ thrombosis in the right atrium are indwelling vascular catheters, pacemaker leads and a prosthetic tricuspid valve. [11]RA thrombi may be diagnosed by TTE but TEE is the best diagnostic tool available. RA thrombi are usually diagnosed in the setting of pulmonary embolism, and are describe in 7-18% of patients with pulmonary embolism. RA thrombi are related to venous thromboembolic disease, as the RA represents a transit zone(thrombi in transit or venous cast) on the pathway between the legs and the pulmonary arteries. [12-13]When a systemic thromboembolic event occurs, a paradoxical embolism should be suspected. Another condition more often linked to systemic thromboembolism is the thrombus straddling the patent foramen ovale(PFO).It is best visualized on short axis of the heart. The diagnosis of a thrombus straddling the PFO is rarely made by TTE. However , TTE may document the consequences of pulmonary embolism(dilated right cavities, paradoxical septum and arterial pulmonary hypertension).

Right ventricular thrombi occur rarely and are often diagnosed in the setting of clinical signs of pulmonary embolism and circulatory collapse. RV dilatation and systolic dysfunction, such as that seen in dilated cardiomyopathy or RV infarct may also predispose to RV thrombosis.

Therapeutic strategy:

2014 AHA/ACC guidelines recommend use of the anticoagulation with vitamin K antagonist(eg.warfarin) for mitral stenosis patients with atrial fibrilation, those with prior embolic events, and those with a LA/LAA thrombus. The use of anticoagulation in patients in sinus rhythm with large LA(diameter >55 mm) recommended by American College of Chest Physicians (ACCP) evidence - based clinical practice guidelines 2012.

Spontaneous contrast echo(SEC) , per se, is not suppressed by anti platelet or anticoagulant therapy and SEC may also occur in absence of AF, in cases of severely enlarged and dysfunctional LA. Should smoke preclude DC cardioversion of AF? Not per se, but it should raise the suspicion of an associated thrombus. The AHA 2018 recommend conservative therapy with 4 weeks of warfarin before cardioversion if thrombus cannot be excluded on TEE. The efficacy of non vitamin K antagonist oral anticoagulant (NOAC) was comparable to warfarin in preventing SEC and thrombus in patients with atrial fibrillation or atrial flutter undergoing DC cardioversion.[12]

The appearance of right heart thrombi(RHT) and the underlying heart on echocardiography will determine the optimal treatment strategy. Once type A of RHT (high risk RHT are mobile,vessel-shape masses that extend from the RA across the tricuspid valve into the RV or PA) has been identified, the patient should be managed in an ICU setting, an immediate individualized treatment options need to be considered because the natural course of RHT is to migrate to lungs, potentially causing a catastrophic cardiovascular collapse. In the largest reported clinical series of type A RHT, more than one in five patients died within 24 hours (or nearly 1% per hour) regardless of the approach to treatment using either thrombolysis or surgical embolectomy.[13]

Warfarin anticoagulation almost abolishes the embolization risk.[14] In addition, warfarin allows intrinsic lysis of the thrombus or at least, organization and endothelialization. In fact, 50% of LV thrombi resolve within 6-12 months of anticoagulation while the rest organize and become laminated. Thus 2013 ACC guidelines recommend the use of warfarin for at least 3 months in a patient with LV thrombus (Class IIa for patient with STEMI and asymptomatic LV thrombus and Class IIb for patients with STEMI and anterior apical akinesis or dyskinesis).

References:

1. DeBruijn SF, Agema WR, Lammers GJ, et al. Transesophageal echocardiography is superior to transthoracic echocardiography in management of patients of any age with transient ischemic attack or stroke. *Stroke* 2006;37:2531-2534.
2. Min JK, Spencer KT, Furlong KT et al. Clinical features of complications from transesophageal echocardiography: a single center case series of 10,000 consecutive examinations.*J Am Soc Ech* 2005;18:925-929.
3. Tan CN, Fraser AG. Transesophageal echocardiography and cardiovascular sources of embolism: implications for perioperative management.*Anaesthesiology* 2007;107:333-346
4. Manning WJ, Weintraub RM, Waksmonski CA, et al. Accuracy of transesophageal echocardiography for identifying LA thrombi, A prospective, intraoperative study. *Ann Int Med* 1995;123:817-822
5. Tan CN, Fraser AG. Transesophageal echocardiography and cardiovascular sources of embolism:implications for perioperative mangement.*Anesthesiology* 2007;107:333-346
6. Fatkin D, Kelly RP, Fenely MP. Relations between left atrial appendage blood flow velocity, spontaneous echocardiographic contrast and thromboembolic risk in vivo. *JACC* 1994;23: 961-969
7. Shively BK, Gelgand EA, Crawford MH. Regional left atrial stasis during atrial fibrillation and flutter: determinats and relation to stroke. *JACC* 1996;27:1722-1729
8. Barkhausen J, Hunold P, Eggebrecht H et al. Detection and characterization of intracardiac thrombi on MRI. *Am J Roentgenology* 2002;179:1539-1544
9. Mansencal N, Nasr IA, Pilliere R et al. Use fullness of contrast echocardiography for assessment of LV thrombus after acute myocardial infarction. *Am J Cardiol* 2007;99:1670-1670
10. Ogren M, Bregqvist D, Eriksson H et al. Prevalence and risk of pulmonary embolism in patients with intracardiac thrombosis: a population- based study of 23769 coseqcutive autopsies. *Eur Heart J* 2005;26:1108-1114
11. Shaprio MA, Johnson M, Feintein SB. A retrospective experience of right atrium and superior vena cava thrombi diagnosed by transesophageal echocardiography. *J Am Soc echo* 2002;15:76-79
12. Kim YG, Choi EK, Park CS et al. Non-vitamin K antagonist oral anticoagulants versus warfarin for the prevention of spontaneous echo-contrast and thrombus in patients with atrial fibrillation or flutter undergoing cardioversion: A transesophageal echocardiography study. *PLoS ONE* 2018;13(1):e0191648
13. Chartier L, Bera J, Delomez, et al. Free-floating thrombi in the right heart: diagnosis, management, and prognostic indexes in 38 consecutive patients. *Circulation* 1999;99(21): 2779-2783.
14. Delewi R, Zijlstra F, Piek JJ. LV thrombus formation after acute myocardial infarction. *Heart* 2012;98:1743-1749