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Editorial

Thrombus in Left Ventricle, Is This a Game Changer or a Marker of Disease?

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▸ See the article "Shape and Mobility of a Left Ventricular Thrombus Are Predictors of Thrombus Resolution" in volume 49 on page 829.

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Prevalence of heart failure (HF) is still increasing and its clinical courses are frequently affected by multiple co-morbid conditions such as left ventricular (LV) thrombus.[1] LV thrombus and its embolization would seriously complicate the clinical course of patients with HF if it involves vital organs such as brain, kidney or intestine. Virchow's triad are well-known factors related with increased thrombogenicity in the patients with HF; abnormal regional wall motion, local myocardial injury and increased hypercoagulability associated with stasis of blood flow.[2] Actually, HF is one of the most important risk factors for thromboembolism and the first initial of 'C' in the acronym of CHA_2DS_2-VASc score stands for 'Congestive HF'.[3]

Although not so frequently found, LV thrombus are noted in patients with HF especially when regional wall motion abnormalities are evident. There have been several studies about the clinical characteristics for the development of LV thrombus. Large infarct size, severe apical wall motion abnormality with aneurysmal formation after anterior myocardial infarction were risk factors for LV thrombus.[2][4] However, only few cases with large anterior myocardial infarction with apical aneurysm were associated with LV thrombus in daily clinical practices and a paucity of data are available about the characteristics of patients who are more prone to this complication. Although studies using vortex images using contrast echocardiography are promising, we need further studies for prospective selection of patients who may benefit form preemptive anticoagulation for prevention of thromboembolic events.[5]

In the current issue of *Korean Circulation Journal*, Oh et al.[6] suggested that the mobility of an LV apical thrombus was the most important predictor of early thrombus resolution and that late resolution was associated with poor long-term clinical outcomes. Interesting finding was that highly mobile thrombus were resolved more quickly, and non-mobile thrombus was associated with worse outcome. As they suggested, persistent thrombi despite the anticoagulation indicate the favorable milieu for thrombus formation.

There are paucity of data about the characteristics of LV thrombus and predictor of its resolution. Actually, protrusion and mobility of thrombus was noted as major echocardiographic risk factors for embolization of LV thrombus.[7] Considering that embolization may occurs before resolution of LV thrombus, both resolution and/or clinically insignificant embolization are more likely to occur in those with highly mobile. As the

current study was retrospective chart reviews, reported rate of systemic embolization or stroke might be underestimated.

Previously a report from our institute suggested that in patients with ischemic LV aneurysms, oral anticoagulation therapy with warfarin might not be effective enough to reduce cardiac and cerebrovascular events including systemic embolism.[8] We have to be very careful in interpretation of the results that there were no significant effect of warfarin on the clinical effect of LV thrombus, since it was retrospective data and anticoagulation was performed based on the clinicians' discretion. This might have resulted in selection bias. In other words, exclusion of patients with protruded or mobile thrombus in whom anticoagulation would be most effective from control group and put them into the warfarin treatment group. The exact opposite situation might be also likely. Mural thrombus are not easy to resolve as they are usually not mobile or protruding and anticoagulation might not be effective. In this situation, thrombus cannot be a game changer but can be a marker of chronicity of the disease in this situation.

Recently it was reported that thromboembolism are associated with patients with HF as frequently as those with atrial fibrillation if they have higher CHA_2DS_2-VASc score greater than or equal to 4.[9] Although COMMANDER-HF resulted in a negative trial either, post hoc analysis showed that low dose rivaroxaban in addition to antiplatelet therapies might reduce the thromboembolic events in ischemic HF with sinus rhythm.[10] However, these events were not the major cause of morbidity and mortality in patients with recent worsening of HF and rivaroxaban had no effect on these hard outcomes.

Currently, there are no controversies about anticoagulation for the management of LV thrombus. If it resolves and regional wall motion abnormality improves, it is reasonable to stop anticoagulation. Then, if thrombus resolves but the apical akinesia persist and HF aggravated? We do not know either about whether prolonged anticoagulation in the patients with persistent LV mural thrombus would be beneficial especially in the current era when we can use using direct oral anticoagulants (DOAC) instead of warfarin. I think this kind of patients would be one of best candidate of DOAC and further studies are strongly recommend to know thrombus is a real game changer in the patients with HF and prone to LV thrombus.

REFERENCES

1. Kim MS, Lee JH, Kim EJ, et al. Korean guidelines for diagnosis and management of chronic heart failure. *Korean Circ J* 2017;47:555-643.
PUBMED | CROSSREF
2. Habash F, Vallurupalli S. Challenges in management of left ventricular thrombus. *Ther Adv Cardiovasc Dis* 2017;11:203-13.
PUBMED | CROSSREF
3. Friberg L, Rosenqvist M, Lip GY. Evaluation of risk stratification schemes for ischaemic stroke and bleeding in 182 678 patients with atrial fibrillation: the Swedish Atrial Fibrillation cohort study. *Eur Heart J* 2012;33:1500-10.
PUBMED | CROSSREF
4. Chiarella F, Santoro E, Domenicucci S, Maggioni A, Vecchio C. Predischarge two-dimensional echocardiographic evaluation of left ventricular thrombosis after acute myocardial infarction in the GISSI-3 study. *Am J Cardiol* 1998;81:822-7.
PUBMED | CROSSREF

https://doi.org/10.4070/kcj.2019.0236

5. Son JW, Park WJ, Choi JH, et al. Abnormal left ventricular vortex flow patterns in association with left ventricular apical thrombus formation in patients with anterior myocardial infarction: a quantitative analysis by contrast echocardiography. *Circ J* 2012;76:2640-6.
PUBMED | CROSSREF
6. Oh JK, Park JH, Lee JH, Kim J, Seong IW. Shape and mobility of a left ventricular thrombus are predictors of thrombus resolution. *Korean Circ J* 2019;49:829-37.
PUBMED | CROSSREF
7. Stratton JR, Resnick AD. Increased embolic risk in patients with left ventricular thrombi. *Circulation* 1987;75:1004-11.
PUBMED | CROSSREF
8. Lee GY, Song YB, Hahn JY, et al. Anticoagulation in ischemic left ventricular aneurysm. *Mayo Clin Proc* 2015;90:441-9.
PUBMED | CROSSREF
9. Melgaard L, Gorst-Rasmussen A, Lane DA, Rasmussen LH, Larsen TB, Lip GY. Assessment of the CHA_2DS_2-VASc score in predicting ischemic stroke, thromboembolism, and death in patients with heart failure with and without atrial fibrillation. *JAMA* 2015;314:1030-8.
PUBMED | CROSSREF
10. Greenberg B, Neaton JD, Anker SD, et al. Association of rivaroxaban with thromboembolic events in patients with heart failure, coronary disease, and sinus rhythm: a post hoc analysis of the COMMANDER HF trial. *JAMA Cardiol* 2019;4:515-23.
PUBMED | CROSSREF

https://doi.org/10.4070/kcj.2019.0236