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Imaging

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 –

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Background: The current study was designed to investigate the correlation between the left ventricular (LV) vortex flow pattern and LV apical thrombus formation in patients with acute anterior wall myocardial infarction (MI).

Methods and Results: Fifty-seven patients with acute anterior wall MI were enrolled in this study. Eighteen patients with apical thrombus (thrombus group) and 39 patients without apical thrombus (non-thrombus group) underwent 2-dimensional contrast echocardiography (CE). Morphology and pulsatility parameters of the LV vortex were measured using Omega flow® and compared between the 2 groups. In the thrombus group, the vortex was located more centrally and did not extend to the apex. In the thrombus group, quantitative vortex parameters of vortex depth (0.409±0.101 vs. 0.505±0.092, respectively; P=0.002) and relative strength (1.574±0.310 vs. 1.808±0.376, respectively, P=0.034) were significantly lower than the non-thrombus group. Following multivariate analysis, the vortex depth below 0.45 remained a significant independent parameter for formation of the LV apical thrombus (odds ratio 9.714, 95% confidence interval 1.674–56.381, P=0.011).

Conclusions: These findings suggest that the location and pulsatility power of the LV vortex are strongly associated with the LV thrombus formation in patients with anterior MI. Therefore, LV vortex flow analysis using CE can be clinically useful for characterizing and quantifying the risk of LV apical thrombus in patients with anterior MI. (*Circ J* 2012; **76:** 2640–2646)

Key Words: Contrast echocardiography; Left ventricle; Thrombus; Vortex

Left ventricular (LV) apical thrombus formation is a major complication in patients with LV dysfunction following an anterior myocardial infarction (MI).[1–10] Although the mechanisms of potential thrombus formation are diverse, abnormalities in apical contraction and the resulting change in the dynamics of flow leading to stagnant flow of the LV apex is one of the most important factors for thrombus formation in these patients.[3–7] Because the LV apical thrombus can be the major source of systemic embolization,[4,8] the characterization and elucidation of the mechanistic dynamics of flow during LV apical thrombus formation is crucial for risk stratification and decisions regarding treatment strategies. However, conventional echo-Doppler parameters are not sufficient to explain flow dynamics and the hemostatic mechanism of LV apical thrombus formation.

In the past several years, several published reports have described the study of LV vortex flow and the evaluation of the LV flow dynamics.[11–13] Recently, we have demonstrated that characterization and quantification of the LV vortex flow using contrast echocardiography (CE) is feasible and useful for evaluating LV flow dynamics.[14] However, the characteristics of LV vortex flow using CE in patients with apical LV thrombus have not been demonstrated. The objective of this study was to investigate the correlation between the LV vortex flow pattern and the LV apical thrombus formation in patients with acute anterior wall MI.

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Table 1. Clinical Characteristics of Patients

	Thrombus (n=18)	No thrombus (n=39)	P-value
Age (years)	66.8±11.9	62.4±15.3	0.286
Male gender	14 (77.8%)	25 (64.1%)	0.370
Hypertension	4 (22.2%)	14 (35.9%)	0.064
Diabetes	3 (16.7%)	9 (23.1%)	0.146
Smoking	5 (27.8%)	14 (35.9%)	0.763
Dyslipidemia	6 (33.3%)	17 (43.6%)	0.567
Post reperfusion TIMI	2.8±0.5	2.7±0.4	0.596
Symptom to balloon (min)	352.4±167.5	361.5±128.3	0.321
Peak CK-MB (μg/L)	187.2±167.3	176.6±163.2	0.482
Q-wave	10 (55.6%)	24 (61.5%)	0.774
Multivessel disease	13 (72.2%)	28 (71.7%)	0.823

Data are presented as the number (%) or mean±standard deviation.
TIMI, Thrombolysis In Myocardial Infarction; CK-MB, creatinine kinase-myocardial band isoenzyme.

Table 2. Comparison of Conventional Echocardiographic Parameters

	Thrombus (n=18)	No thrombus (n=39)	P-value
LVEF (%)	33.2±9.5	36.4±11.9	0.313
LVEDd (mm)	50.1±9.2	49.3±7.0	0.417
LVESd (mm)	39.8±9.6	38.7±8.7	0.413
LAVI (ml/m^2)	34.5±20.4	27.8±11.6	0.131
E velocity (m/s)	0.71±0.21	0.66±0.17	0.536
E/A ratio	1.5±0.9	1.3±0.8	0.151
E' velocity (m/s)	0.058±0.017	0.056±0.018	0.780
E/E' ratio	14.20±6.64	12.75±5.14	0.398
WMSI	1.81±0.36	1.73±0.34	0.419

Data are presented as the mean±standard deviation.
LVEF, left ventricular ejection fraction; LVEDd, left ventricular end-diastolic dimension; LVESd, left ventricular end-systolic dimension; LAVI, left atrium volume index; WMSI, wall motion score index.

Methods

Study Population

Fifty-seven patients with acute anterior wall MI showing akinetic or dyskinetic apical wall motion abnormalities (WMA) on an initial transthoracic echocardiogram were retrospectively included in this study. The 57 patients comprised 18 patients with LV apical thrombus (thrombus group; age 66.8±11.9 years) and age-sex matched 39 patients without thrombus (non-thrombus group; age 62.4±15.3 years). Exclusion criteria were past medication history with warfarin for chronic oral anticoagulation, confirmed coagulopathy, thrombocytosis (defined as a platelet count >450,000/μl), moderate-to-severe valvular heart disease, significant arrhythmia including atrial fibrillation or ventricular tachycardia, and inadequate Doppler recordings. All patients underwent percutaneous coronary intervention (PCI) and were treated appropriately with dual antiplatelets (aspirin plus clopidogrel) and heparinization was routinely given for 72 h after the onset of MI. The CE was performed within 1 week after MI. There were no patients who received the glycoprotein IIbIIIa inhibitor. Informed consent was obtained from all study participants, and the study protocol was approved by the Ethics Committee of Yeungnam University Hospital.

PCI

All patients underwent PCI. Thrombolysis In Myocardial Infarction (TIMI) flow after reperfusion, time from onset of symptom to balloon time, peak creatinine kinase-myocardial band isoenzyme (CK-MB) level, percentage of Q-wave (defined by the Selvester criteria[15]) MI, and the percentage of multivessel disease were evaluated and compared between the 2 groups.

2-Dimensional (D) and CE

All patients underwent 2-D and Doppler echocardiography according to the standards of the American Society of Echocardiography[16] on an Acuson Sequoia C-512 ultrasound platform (Siemens Medical Solutions, Mountain View, CA, USA) with 3V2C phased-array transducer at a frequency of 3.75 MHz. Through the use of the 2-D CE, the apical thrombus was defined as a mass distinct from the endocardium in the apex, with an echo density different from that of the myocardium and visualized in at least 2 orthogonal planes in an area of myocardial asynergy.[10] The LV end-diastolic dimension (LVDd) and LV end-systolic dimension (LVSd) were measured by M-mode at the parasternal long axis and short axis view. LV ejection fraction (LVEF) was measured using the modified Simpson's method on images of the apical 2-chamber and 4-chamber view and the left atrial volume index (LAVI) was calculated by the biplane area-length method. E velocity, E/A ratio, E' velocity, and E/E' ratio was calculated based on the mitral E and A velocity obtained by the Doppler echocardiography and the septal mitral annular E' velocity obtained by tissue Doppler imaging in the pulsed wave Doppler.[17] The wall motion score index was obtained in a 17 segment model to assess the LV WMA. 2-D CE was performed using an echo-enhancing agent, Definity®

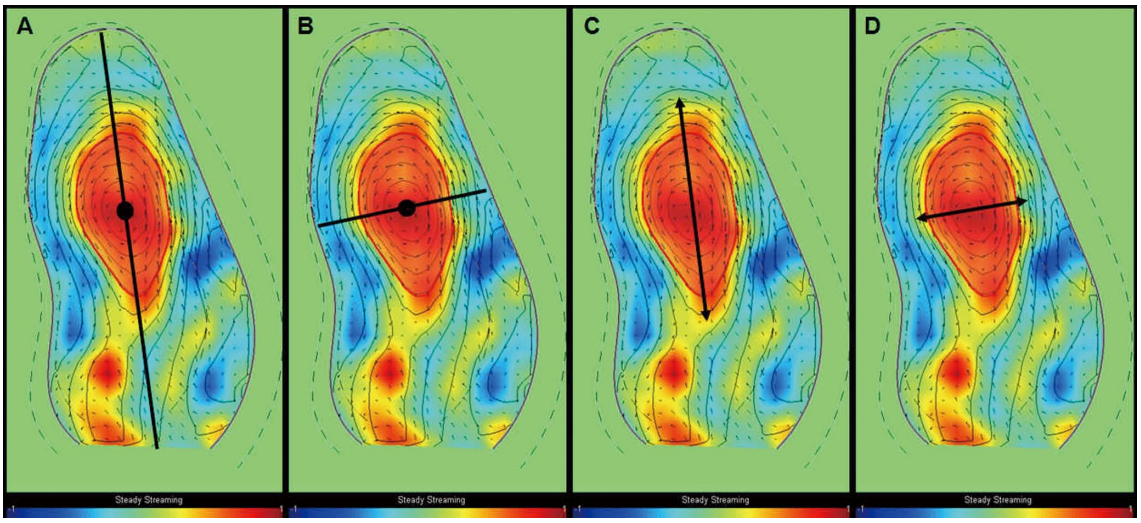

Figure 1. Description of quantitative parameters of the vortex location and shape. Vortex depth represents vertical position of center of vortex relative to left ventricular long axis (**A**, black line), and vortex transverse position represents transverse position relative to posteroseptal axis (**B**, black line). Vortex length was measured by longitudinal length of vortex relative to left ventricular length (**C**, black arrow), and vortex width was measured by horizontal length of vortex relative to left ventricular length (**D**, black arrow).

(Lantheus Medical Imaging, North Billerica, MA, USA) consisting of octaflurorpropane gas-filled, lipid-stabilized micro bubbles. A mixed solution consisting of 0.7 ml of Definity® and 25 ml of normal saline was given as a slow intravenous infusion. The CE imaging was obtained at a mechanical index of 0.4–0.6 and the focal zone was positioned in the middle of the LV. Ultrasound scan width, imaging depth, and spatial temporal settings were optimized to achieve the highest possible frame rate. All cine data from 3 consecutive cycles were acquired with the acoustic capture technique. Four-chamber and apical long axis views were acquired to provide the best visualization of the LV vortex formation. The temporal resolution was 16.4± 3.5 ms with 60 to 80 frames/cardiac cycle. All stored images were analyzed blindly by unbiased independent observers. CE was performed in an average time of 5.7±3.5 days from emergency department visit.

Quantitative Parameters of LV Vortex Flow

Estimation of the velocity vector and quantification of vortex parameters were determined by Particle image velocimetry (PIV) (Omega flow®, version 2.4.5, Siemens Medical Solution, Moutain View, CA, USA). PIV method using in this study was adopted and completed with a Feature Tracking Algorithm to handle the long range correlations as a result of large velocity values. The accuracy of velocity values was bounded from low velocities and high velocities. Velocity was estimated on the basis of displacement of back scatter and results from 3 consecutive beats were averaged to improve the blood flow tracking and quality of results. The angle-independent ventricular velocity field was computed.[14]

The location of the vortex was measured by vortex depth (VD) and vortex transversal position (VT). VD represents the vertical position of the center of the vortex relative to the LV long axis (the distance of its center from the base). VD is expressed as a decimal between 0 and 1, which becomes higher (close to the value 1) when the center of the vortex is located closer to the apex of LV, and becomes lower (close to the value 0) when the center of the vortex is located closer to the base of LV. VT represents the transverse position relative to the postero-septal axis (the distance of its center from the posterior wall). Vortex length (VL; the longitudinal length of the vortex relative to LV length) and the vortex width (VW; the horizontal length of vortex relative to LV length) were measured to obtain the shape of the vortex. A vortex sphericity index was calculated by the VL divided by the VW. We also measured 2 parameters including relative strength (RS) and vortex RS (VRS) to assess the pulsatility of the LV vortex. The RS represents the strength of the pulsatile component of vorticity with respect to the average vorticity within the whole LV. This is the ratio between the total vorticity strength of the first order Fourier harmonic and the vortex strength of the zero order Fourier harmonic. VRS represents the same ratio accounting for the pulsatile vorticity of vortex only, instead of the entire LV.[14]

Two double-blinded examiners repeatedly performed the measurements on 10 randomly selected patients (5 with thrombus and 5 without thrombus) for both intra- and inter-observer analysis. The second observer repeated the measurements 1 week following the initial analysis. The patient case sequence was randomly arranged each time.

Statistical Analysis

Continuous variables are presented as the mean±standard deviation and were compared using the independent Student's t-test. Comparison of categorical variables was made by the chi-square test. A P-value ≤0.05 was considered statistically significant. A multivariate logistic regression analysis was used to assess the correlation among those parameters whose statistical significance was demonstrated through a univariate analysis at a level of P≤0.05 and previously well-known risk factors, which did not demonstrate a significant difference in this study. A receiver operating characteristic (ROC) curve of VD

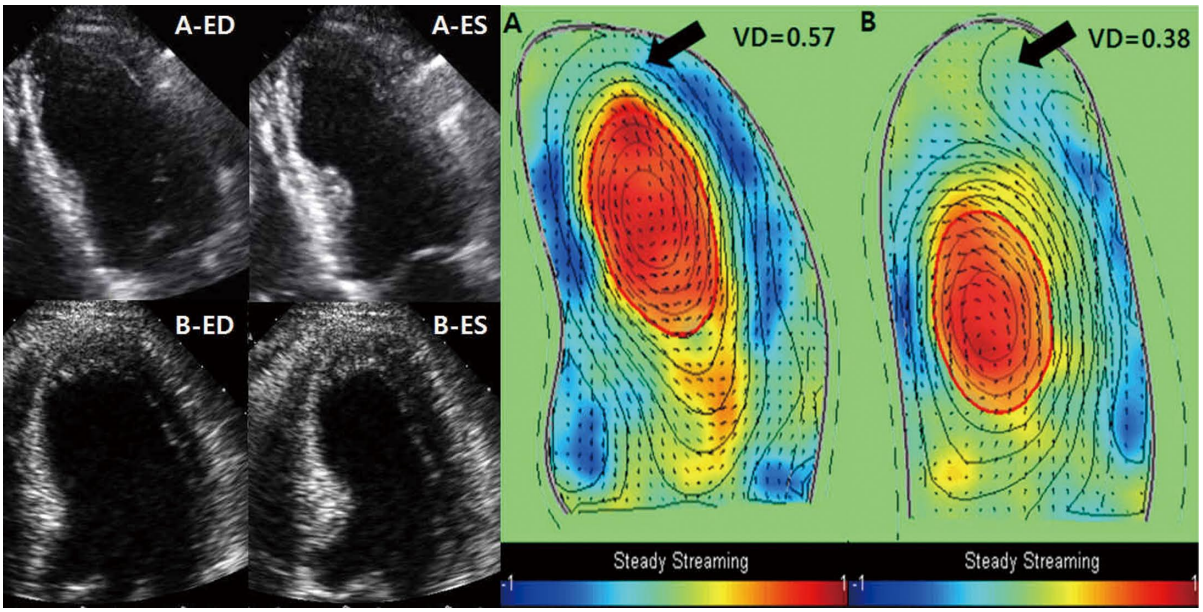

Figure 2. Comparison of the 2-dimensional (D) apical 4-chamber view and location and morphology of the average left ventricular (LV) vortex flow pattern between non-thrombus and thrombus groups. The end-diastole (ED) and end-systole (ES) 2-D image of non-thrombus (**Left panel**, **A**) and thrombus group (**Left panel**, **B**) showing LV ejection fraction (LVEF) is similar in both groups. Parametric representation of the steady streaming field in the non-thrombus (**Right panel**, **A**) and thrombus group (**Right panel**, **B**) are evaluated in apical 4-chamber view. The center of the average vortex flow was located near the apex in the non-thrombus group (**A**). However, in the thrombus group, the vortex was located in the center of the LV, much farther from the apex and did not reach to the LV apex (**B**). A black arrow indicates different vortex flow pattern in the apex between the 2 groups.

was plotted and the area under the curve was calculated. All statistical analyses were performed using the PAWS statistic version 18.0 (SPSS Inc, Chicago, IL, USA).

Results

Clinical Data

Clinical characteristics of the study population are shown in Table 1. The average age and gender distribution of thrombus patients (66.8±11.9 years, 14 males) and non-thrombus patients (62.4±15.3 years, 25 males) was not significantly different. There were no significant differences between thrombus and non-thrombus patients with regards to the history of diabetes (16.7% vs. 23.1%), hypertension (22.2% vs. 35.9%), dyslipidemia (33.3% vs. 43.6%), and smoking (27.8% vs. 35.9%). TIMI flow after reperfusion (2.8±0.5 vs. 2.7±0.4), time from onset of symptom to balloon time (352.36±167.5 min vs. 361.53±128.3 min), peak CK-MB level (187.2±167.3 μg/L vs. 176.6±163.2 μg/L), percentage of Q-wave MI (55.6% vs. 61.5%), and the percentage of multivessel disease (72.2% vs. 71.7%) were not significantly different between the 2 groups.

Conventional Echo-Doppler Parameters

Conventional echo-Doppler parameters of the study population are shown in Table 2. For the thrombus and non-thrombus patients, LVEF (33.2%±9.5 vs. 36.4%±11.9, P=0.313), LVDd (50.1±9.2 vs. 49.3±7.0 P=0.417), LVSd (39.8±9.6 vs. 38.7±8.7, P=0.413), LAVI (34.5±20.4 vs. 27.8±11.6, P=0.131), and wall motion score index (1.81±0.36 vs. 1.73±0.34, P=0.419) were not significantly different between both groups. In addition, parameters representing diastolic function; E velocity

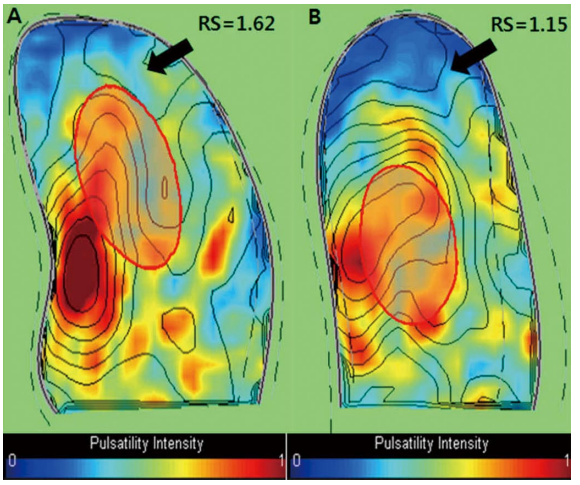

Figure 3. Comparison of the pulsatile strength of the vortex flow between non-thrombus and thrombus group. Parametric representation of the pulsatile strength field in the non-thrombus (**A**) and thrombus group (**B**) are evaluated in apical 4-chamber view. The pulsatility of the vortex was stronger (red color area) in non-thrombus patients (**A**) and weaker (blue color) in patients with thrombus (**B**). The black arrow indicates different vortex pulsatility strength in the apex between the 2 groups.

Table 3. Comparison of Quantitative LV Vortex Flow Parameters Between Thrombus and No Thrombus Group

	Thrombus (n=18)	No thrombus (n=39)	P-value
VD	0.409±0.101	0.505±0.092	0.002
VT	–0.036±0.087	–0.020±0.059	0.428
VL	0.503±0.185	0.514±0.160	0.817
VW	0.245±0.050	0.232±0.046	0.344
SI	2.125±0.856	2.318±1.011	0.485
RS	1.574±0.310	1.808±0.376	0.034
VRS	0.387±0.123	0.410±0.199	0.656

Data are presented as mean±standard deviation.
LV, left ventricular; VD, vortex depth; VT, vortex transverse; VL, vortex length; VW, vortex width; SI, sphericity index; RS, relative strength; VRS, vortex relative strength.

Table 4. Parameters Influencing Formation of the LV Thrombus Based on Multivariate Logistic Regression Analysis

	OR	P-value	CI
LVEF (%)	1.007	0.826	0.826–1.227
LVEDd (mm)	0.942	0.809	0.580–1.530
LVESd (mm)	1.114	0.705	0.638–1.944
WMSI	3.412	0.548	0.062–187.485
E/E' ratio	0.940	0.544	0.771–1.147
VD <0.45	9.714	0.011	1.674–56.381
RS	0.073	0.103	0.003–1.693

OR, odds ratio; CI, confidence interval. Other abbreviations as in Tables 2,3.

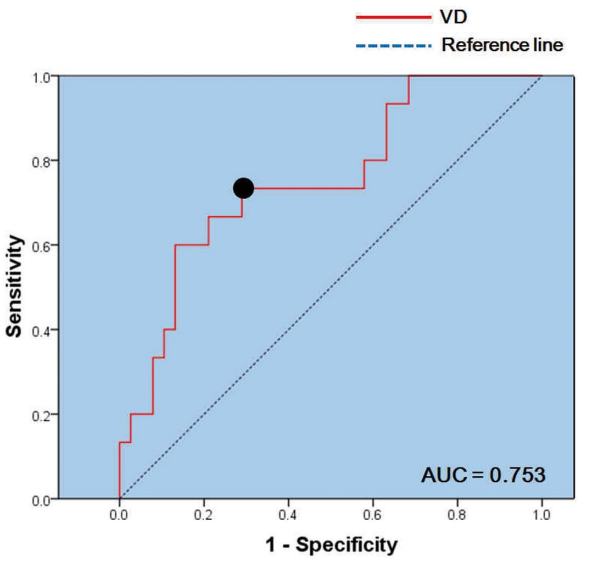

Figure 4. Receiver operating characteristic curves for the prediction of left ventricular (LV) thrombus using vortex depth (VD). The area under the curve for predicting LV thrombus of VD was 0.753. The best cut-off value for VD was determined to be 0.45. At this value, the sensitivity and specificity for the abscence of LV thrombus were 73.3% and 71.1%, respectively. AUC, area under the curve.

(0.71±0.21 m/s vs. 0.66±0.17 m/s, P=0.536), E/A ratio (1.5±0.9 vs. 1.3±0.8, P=0.151), E' velocity (0.058±0.017 m/s vs. 0.056±0.018 m/s, P=0.780), and E/E' (14.20±6.64 vs. 12.75±5.14, P=0.398); were not significantly different between the 2 groups.

Characteristics of LV Vortex Flow in Thrombus and Non-Thrombus Patients

Description of quantitative parameters of the vortex location and shape are shown in **Figure 1**. Characteristics of the LV vortex in the thrombus and non-thrombus group are shown in **Figures 2** and **3**. In the parametric images displayed in the right panel of **Figure 2A**, the center of the average vortex flow was located more apically in the non-thrombus group. However, in the thrombus group, the center of the major vortex was located with increased frequency toward the base when compared to the non-thrombus group. In addition, this vortex did not extend to the LV apex (**Figure 2B**). The pulsatility of the vortex in whole LV was stronger (red color area) in the non-thrombus patients (**Figure 3A**) and weaker (blue color) in patients with thrombus (**Figure 3B**).

Quantitative Analysis of LV Vortex Flow

Quantitative analysis of the LV vortex in the 2 groups is shown in **Table 3**. Among the location and morphologic vortex parameters, the VD (0.409±0.101 vs. 0.505±0.092, P=0.002) was significantly decreased in the thrombus group as compared to the non-thrombus group. There was no significant difference in VT (–0.036±0.087 vs. –0.020±0.059, P=0.428), VL (0.503±0.185 vs. 0.514±0.160, P=0.817), VW (0.245±0.050 vs. 0.232±0.046, P=0.344), and sphericity index (2.125±0.856 vs. 2.318±1.011, P=0.485) between the 2 groups (same respective order). In the pulsatility parameters, RS (1.574±0.310 vs. 1.808±0.376, P=0.034) was significantly lower in the thrombus group compared to the non-thrombus group. However, VRS (0.387±0.123 vs. 0.410±0.199, P=0.656) did not demonstrate a significant difference between the 2 groups.

Upon multivariate logistic regression analysis, which included those parameters whose statistical significance was demonstrated through a univariate analysis (VD and RS) as well as previously well-known risk factors, only the VD <0.45 remained as a significant independent parameter of the LV thrombus formation (odds ratio 9.714, 95% confidence interval 1.674–56.381, P=0.011) (**Table 4**).

The ROC for the VD exhibited a fair level of discrimination between the 2 groups, and correlated significantly with the LV apical thrombus formation (**Figure 4**). The area under the curve was the largest for VD (0.753, P=0.004). Based on the ROC curve, a VD value of 0.45 had a sensitivity of 73.3% and specificity of 71.1% to discriminate the abscence of LV apical thrombus. Of the patients with VD ≥0.45, only 12.9% (4/31) had LV thrombus, while those with a VD <0.45 had a much higher incidence of LV thrombus (53.8%, 14/26; P=0.001).

The intra-observer and inter-observer variability for the quantitative vortex analysis were assessed by determining correlation coefficients. There were no significant differences between the 2 observers (correlation coefficient=0.92, P=0.002) or within the same observer (correlation coefficient=0.93, P=0.042) for the quantitative vortex parameter analysis

Discussion

To our knowledge, the present study is the first to demonstrate that the abnormal LV vortex flow parameters correlate well

with the presentation of LV apical thrombus formation in patients with anterior wall MI.

Thrombus formation in the left ventricle is the major complication after an acute anterior wall MI. Previous studies have reported that the incidence of LV thrombus is 27% to 46% in patients with acute anterior wall MI who have apical akinesis or dyskinesis.[17–19] However, in another recent study, the prevalence of LV thrombus was much lower than previously reported, possibly as a result of improvement in the modern management of acute MI with widespread use of reperfusion therapies and appropriate anti-platelet therapy.[20] In the present study, even all the patients were appropriate treated by PCI and 72 h of anticoagulation with heparin, apical thrombus was observed in 18 (32%) of the 57 patients, which seems to be relatively high. This is maybe because of the retrospective inclusion of the patients who have very high risk of apical thrombus formation.

The accurate prediction of apical thrombus formation is essential as a result of the significantly increased rate of thromboembolism in these patients as compared to those without thrombus.[7] There have been several published reports demonstrating the relationship between LV flow dynamics and LV thrombus formation.[1,3,5,10] Beppu et al, in their first experimental observational study using CE, discovered that contrast echoes did not reach the apex but turned upward to the outflow tract in the middle of the cavity in myocardial infracted dogs with apical akinesia or dyskinesia. Their results suggest that this abnormal pathway of the blood during apical MI might lead to the development of hemostasis in the apex and should be one of the mechanisms of thrombus formation in MI.[3] However, they only demonstrated the change in the LV vortex flow pattern in MI descriptively, and could not suggest quantitative parameters that could be used in clinical practice. Delemarre et al performed a prospective study of 62 patients with acute MI to predict apical thrombus formation based on the LV spatial flow pattern using a pulsed wave Doppler.[1] Van Dantzig et al extended Delemarre's study and suggested that normal spatial distribution of LV flow was the only independent correlate of ventricular thrombus after MI and its predictive accuracy was superior to that of clinical and 2-D echocardiographic variables. These results emphasize the crucial importance of LV flow dynamics for the formation of the LV thrombus.[10] However, the methods used in this study were based on Doppler signals from the LV base and the apex. The Doppler method is limited in several ways. First, the Doppler measures the flow velocity parallel to the scan line, which might not be the principal direction of motion. Second, the color Doppler measures the mean velocities and is angle-dependent thereby underestimating the peak velocities. Third, Doppler signals can be affected by various conditions such as volume status, heart rate, and blood pressure. In addition, they demonstrated a normal and abnormal blood flow pattern based on pulsed wave Doppler, but could not suggest quantitative parameters based on the LV vortex flow, which is important for the prevention of blood stagnation in the LV apex.

The vortex flow of the LV reflects the function and geometry of the ventricle.[3,14] A previous report suggested that there are 3 major roles of the LV vortex: (1) redirecting blood flow effectively, (2) preserving kinetic energy until ejection, and (3) preventing stagnation of blood flow.[14,20,21] Furthermore, the LV vortex in the LV apex is crucial for preventing hemostasis in the apex and thrombus formation in patients with MI.[3,10] In normal individuals, during LV filling, the LV vortex forms and is determined by the shear layer between the high-speed mitral jet and surrounding still fluid. However, in patients with abnormal LV systolic function, organization of shear flow and fluid mechanics in the LV are spoiled, and the vortex is incoherent, and less pulsatile than normal.[11,14] Moreover, in patients with MI, stasis of blood as a result of akinetic wall motion with LV remodeling, aggravation of endothelial dysfunction, and increased viscosity of blood are more prone to cause thrombus formation.[3–6] These complex process of thrombus formation cannot be detected by conventional echocardiographic parameters, and maybe associated with vortex flow in the LV cavity.

We previously reported that it was feasible to quantify LV vorticity and vortex location, morphology, and pulsatility parameters were significantly different between normal and patients with LV dysfunction.[14] Therefore, we wanted to extend this novel technology to ascertain whether LV vortex flow is a critical determinant of LV apical thrombus formation in patients with MI. In this study, using CE with PIV, we could clearly delineate the LV vortex flow and demonstrate obvious differences in LV vortex flow patterns between apical thrombus and non-thrombus patients. In the non-thrombus group, the center of the LV vortex was relatively apical in location and the pulsatility of the LV vortex was well-preserved throughout the whole LV. In the thrombus group, the center of the average vortex flow was located more central, and the pulsatility of LV was relatively weaker in the apical region as compared to the non-thrombus group. These findings are similar to those of a previous experimental study.[3]

Among the quantitative vortex parameters, VD was significantly reduced in patients with LV thrombus. The ROC curve showed that VD value over 0.45 has strong discriminative power and significantly correlated with LV apical thrombus formation. Furthermore, after multivariate analysis, we could confirm VD <0.45 is the independent strongest parameter for the formation of the LV thrombus. We also quantified and compared the pulsatility profile of the vortex. Pulsatility of the LV represented by RS was significantly lower in patients with thrombus when LVEF and LV dimension were comparable. This means that poor pulsatile power in the apical region might correlate with LV thrombus formation. However, on multivariate analysis, RS was not found to significantly associate with the apical thrombus formation. This result might suggest that the vortex location is more important than pulsatility for developing LV apical thrombus in patients with anterior MI.

Clinical Implications

Risk assessment of the formation of LV thrombus by echocardiography is important for optimal management of oral anticoagulant agents, which decreases potential embolic complications.[22] Therefore, if we find a propensity and echocardiographic parameters for thrombus formation, then we can guide antithrombotic therapy in patients with MI. We suggest that VD might represent a useful marker for the occurrence of LV thrombus. In patients with a VD ≥0.45, the incidence of LV thrombus was only 12.9%; in contrast, patients with a VD <0.45 maintain a 53.8% level of incidence, despite comparable conventional echo-Doppler findings. Therefore, a more careful consideration of anticoagulation should be mandatory in patients who are at increased risk for developing LV thrombus based on a decreased VD. It can be clinically applicable to distinguish which patient have increased risk of thrombus formation and can help decisions regarding the use of oral anticoagulation therapy in patients after acute anterior MI.

Study Limitations

There were several limitations within our study. First, a relatively small numbers of patients were included. Second, the roles of coagulation and local factors from the injured endocardium for LV thrombosis were not taken into consideration in this study. Third, this clinical study was a retrospective cross-sectional and observational study for the LV vortex flow in patients with acute anterior MI. Therefore, selection bias might affect our results and we only could compare the LV vortex flow pattern in patients with already-formed thrombus and it could not be determined whether the abnormal flow was the primary event in the genesis of the thrombus or secondary event to thrombus development. However, all of the thrombus was mural type confined to LV apex (mean thrombus size of longitudinal direction to LV cavity was only 1.1±0.7 cm), and none of them were located in the mid LV or protruded to LV cavity which can block or influence the vortex flow of the LV.

Despite these limitations, this study is the first attempt to characterize and quantify the vortex flow of the LV thrombus formation in patients after MI. Based on this study, future long term follow up research and a large-scale prospective randomized controlled study is mandatory to confirm the relationship between the LV thrombus and abnormal vortex flow patterns.

Conclusions

These findings suggest that the location and pulsatility power of LV vortex are strongly associated with LV thrombus formation in patients with anterior MI. Therefore, the LV vortex flow analysis using CE can be clinically useful for characterizing and quantifying the risk of LV apical thrombus in patients with anterior MI.

Disclosures

Conflict of Interest: Geu-Ru Hong: research support from the Siemens Medical Solution. Helene Houle: employee of the Siemens Medical Solution.

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Supplementary Files

Supplementary File 1

Movie S1. The left ventricular (LV) vortex flow images by digital particle image velocimetry from the apical 4 chamber view in a patient without thrombus. The movement of contrast bubbles is mapped by particle image velocimetry. The arrows in the image represent the velocity and vectors of each pixels in the LV and red color and blue color vortex indicate clockwise or counter-clockwise rotated vortex flow.

Supplementary File 2

Movie S2. Left ventricular (LV) vortex flow images of apical 4 chamber view in a patient with apical thrombus. The vortex was located more central and showed lower pulsatility in the apex when compared to non-thrombus patients.

Please find supplementary file(s);
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