

Association between left ventricular global longitudinal strain and acute pulmonary edema caused by increased left ventricular afterload in patients with STEMI

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Background

Acute pulmonary edema (APE) is common complication after ST elevation myocardial infarction (STEMI) and often associated with poor prognosis. It is usually caused by decreases of left ventricular contractility and subsequent increase of left ventricular afterload. In this study we aimed to determine association between left ventricular global longitudinal strain (GLS) parameter and APE caused by increased left ventricular afterload.

Methods

We chose patients with STEMI and who were treated by primary PCI in this study. Two dimensional speckle tracking echocardiography was used to assess left ventricular GLS. Study endpoint was APE caused by increased left ventricular afterload. Univariable and multivariable logistic regression analysis was used to determine association between GLS and APE.

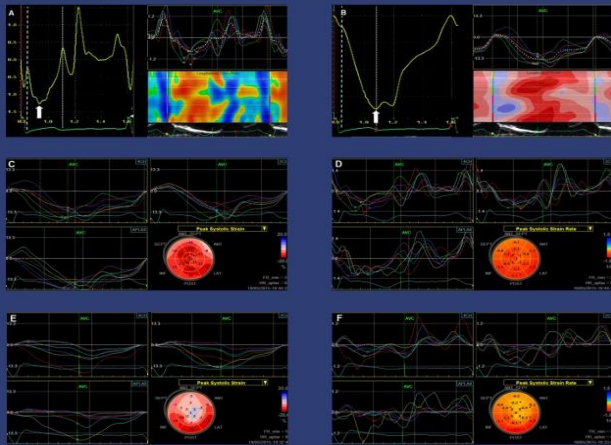


Figure 1. Measurement of strain rate (A, arrow - peak systolic strain rate) and strain (B arrow - peak strain). Examples of longitudinal strain (C and E) and strain rate (D and F) curves from the three apical views of the left ventricle. Baseline strain (C) and strain rate (D) were preserved in patients without events, whereas baseline strain (E) and strain rate (F) were diminished in patients with all-cause mortality during follow-up.

Results

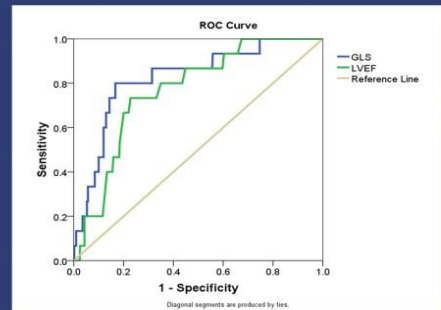
A total of 524 patients were enrolled. Mean age was 60 ± 13 years old and majority of patients were male 444 (84.7%). During admission, APE was occurred in 15 (2.9%) patients. Univariable logistic regression analysis showed GLS is significantly associated with APE and every 1 unit change of GLS is associated with 1.34 times increased probability of having APE (OR 1.34, 95% CI 1.17-1.53, $p < 0.001$). After adjustment of clinical, angiographic and conventional echocardiographic indices, left ventricular GLS was inde-

pendently associated with APE (OR 1.25, 95% CI 1.06-1.46, $p < 0.01$) (Table 1).

Table 1. Univariable and multivariable logistic regression analysis.

	Univariate			Multivariate		
	OR	95% CI	P value	OR	95% CI	P value
Age	1.06	1.02-1.10		1.06	1.01-1.12	<0.05
Gender	0.85	0.19-3.84	0.833			
SBP (mmHg)	1.0	0.99-1.01	0.843			
Heart rate	1.05	1.02-1.07	<0.001			
HTN	3.46	0.96-12.4	0.057			
DM	1.91	0.68-5.35	0.219			
Pre CAD	1.29	0.28-5.89	0.737			
Pre MI	0.75	0.09-5.87	0.788			
Pre CHF	7.68	1.53-38.6	<0.05	10.4	1.20-90.4	<0.05
Peak cTnI	1.00	1.00-1.01	<0.05			
D2B <90	1.26	0.35-4.54	0.723			
MVD	1.04	0.35-3.09	0.945			
LAD culprit	1.25	0.44-3.58	0.669			
Final TIMI 3	0.17	0.05-0.52	<0.01	0.18	0.05-0.68	<0.05
LVEDV (ml)	1.03	1.01-1.04	<0.001	1.03	1.01-1.05	<0.01
LVESV (ml)	1.04	1.02-1.05	<0.001			
LVEF (%)	0.93	0.89-0.97	<0.001			
DT (msec)	0.98	0.97-0.99	<0.001			
EE ratio	1.06	1.02-1.10	<0.01			
GLS (%)	1.34	1.17-1.53	<0.001	1.25	1.06-1.46	<0.01

SBP, systolic blood pressure; HTN, hypertension; DM, diabetes mellitus; CAD, coronary artery disease; MI, myocardial infarction; CHF, congestive heart failure; cTnI, cardiac troponin I; D2B, door-to-balloon time; MVD, multivessel disease; LAD, left anterior descending artery; TIMI, thrombolysis in myocardial infarction; LVEDV, left ventricular end diastolic volume; LVESV, left ventricular end systolic volume; LVEF, left ventricular ejection fraction; DT, deceleration time; EE^{*} mitral inflow peak early velocity (E)/mitral annular peak early velocity (E^{*}).



Predictive capacity of left ventricular GLS was better than LVEF (c-statistic 0.824, 95% CI 0.719-0.929, $p < 0.001$).

Conclusion

Speckle tracking derived GLS is strong and independent predictor of APE caused by increased left ventricular afterload in patients with STEMI after primary PCI. Prognostic capacity of GLS is better than LVEF.

Conflict of interest: No conflict of interest