

Can analysis of myocardial mechanic help me to predict heart failure development in my STEMI patient whose EF is equal or above 50% after pPCI?

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In spite of contemporary STEMI management, heart failure (HF) develops in 4% up to 28% of pPCI-treated patients, with the highest incidence in the first year. Left ventricular ejection fraction (EF) is strong predictor predominately for HFrEF development, but risk stratification in case of preserved post pPCI EF (i.e. EF \geq 50%) is still challenging.

Aim: the current study is a sub-study of PREDICT-VT study (NCT03263949). Its aim is to define clinical and "echocardiographic" profile of STEMI patient at risk to develop HF despite preserved post pPCI EF, including not only conventional echocardiographic data, but data from myocardial mechanic analysis obtained by early speckle tracking echocardiography.

Methods: in 307 consecutive pts enrolled in PREDICT-VT study early echocardiography (5 \pm 2 days after pPCI) was done and included LA and multilayer LV deformation analysis with longitudinal (L), radial (R) and circumferential (C) strain (S; %) and strain rate (SR, 1/sec), calculation of LV index of post systolic shortening for longitudinal (PSS LS) and circumferential (PSS CS) strains and thorough analysis of LV rotation mechanic.

Results: From 242 patients who completed 1-year follow-up, 109 pts (45 %) had post pPCI EF \geq 50%. Of those patients 34 (31%) became NYHA \geq 2 or died during 1-year follow-up (MACE+ group). Patients with MACE were older (63 \pm 8 vs 55 \pm 10, $p < 0.001$), more frequently were female (47% vs 27%; $p = 0.036$) and more frequently had hypertension (40% vs 20%; $p = 0.025$). There were no significant differences in LV EF (56.5 \pm 4.8% vs 56.3 \pm 4.8 %; $p = 0.849$) and from conventional echo parameters only differences in E/A ratio (0.75 \pm 0.24 vs 0.92 \pm 0.32; $p = 0.015$) and MAPSE (1.54 \pm 0.40 vs 1.36 \pm 0.27; $p = 0.015$) reached statistical significance. Surprisingly, there were no significant differences neither in LV longitudinal, nor circumferential deformations. However, LV radial deformation was significantly impaired in MACE+ pts both during systole (global radial strain: 16.4 \pm 7.1 vs 21.1 \pm 10.3; $p = 0.008$; end-systolic radial strain 13.1 \pm 7.3 vs 18.1 \pm 9.9; $p = 0.005$), early (radial SR E: -1.27 \pm 0.66 vs 1.59 \pm 0.79; $p = 0.044$) and late (radial SR A: -0.94 \pm 0.41 vs -1.20 \pm 0.59, $p = 0.011$) diastole. LV rotation was not significantly impaired, but slowed and delayed both during systole (time to peak systolic apical rotation (ms) 168 \pm 86 vs 128 \pm 70; $p = 0.022$) and diastole (rotation rate of LV base during early diastole ($^{\circ}$ /sec): 38.3 \pm 27.4 vs 55.0 \pm 31.5, $p = 0.008$; time to maximal LV untwisting rate (ms) 580 \pm 210 vs 484 \pm 154; $p = 0.044$), despite no differences in HR.

Conclusion: STEMI patients who will develop heart failure despite preserved post pPCI EF might have different clinical profile and different pattern of deviation in LV mechanic (predominately involving radial and rotational mechanic) and can be detected by contemporary echocardiographic techniques.