Soluble ST2 biomarker and reverse remodelling in patients with systolic heart failure

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Left ventricular reverse remodelling (LVRR) has generally been suggested as an important alteration potentially predicting favorable prognosis in patients with systolic heart failure.[1,2] By definition, it is a stepwise and time-dependent improvement in systolic functions characterized by a left ventricular ejection fraction (LVEF) increase of ≥15% or a LVEF increase of ≥10% along with a significant decline in end-systolic diameter or volume index during 1 year follow-up.[1,2] Importantly, certain novel biomarkers including soluble ST2 (sST2) were suggested to have an independent association with LVRR.[2]

In their recently published article,[3] Bahuleyan et al. have demonstrated prognostic value of baseline and serial serum sST2 concentrations in patients with systolic heart failure. However, we would like to make a few comments and have information regarding LVRR and its association with sST2 in their study group:

Firstly; LVRR phenomenon (largely due to the low sST2 levels) might have contributed to the excellent prognosis in patients without adverse outcome on follow-up. Accordingly, were there any patients with LVRR on follow-up? Did patients with and without adverse outcomes differ in terms of LVRR incidence?

Secondly; a variety of independent clinical and laboratory parameters including a baseline sST2 level of <48 ng/mL, beta blocker therapy, non-ischemic etiology and absence of left bundle brunch block (LBBB), etc. were previously suggested to predict LVRR on follow-up.[2] Accordingly, what were the predictors for the evolution of LVRR (if any) in their study?

Lastly; the authors[3] suggested a baseline sST2 value of ≤49 ng/mL to predict a relatively favorable prognosis. More specifically, was there any optimal cut-off sST2 value at baseline to predict LVRR (and hence; to potentially identify patients with the best prognosis)? What about the comparison of baseline and serial sST2 levels between patients with and without LVRR on follow-up?

In summary; as opposed to the unfavorable nature of adverse myocardial remodelling (arising due to neurohormonal activation, cytokines, etc.),[4,5] it seems likely that LVRR phenomenon potentially yields the most remarkable prognostic benefit in patients with systolic heart failure. Importantly, evolution of this auspicious phenomenon appears to be strongly associated with reduced inflammatory, mitogenic as well as pro-fibrotic stimuli as demonstrated with low levels of certain novel biomarkers including sST2.[2] Therefore, future studies should primarily focus on strategies (novel drugs, etc.) aiming to create the most proper milieu for the emergence of LVRR in the setting of systolic heart failure.

Conflicts of interest

None.

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