Correlation and utility of the changes in sST2 level at clinician determined decompensation and recompensation

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Introduction: Soluble Suppressor of Tumorigenicity 2 (sST2) biomarker has been shown to demonstrate prognostic capabilities in patients with heart failure (HF), with serial measurements of sST2 at pre-determined time intervals providing additional predictive value for adverse outcomes. We sought to evaluate what level of sST2 level change, at clinician determined decompensation and recompensation during an acute admission for decompensated HF, could provide prognostic information for adverse outcomes.

Methods: Between January 2020 and February 2022, consecutive patients hospitalized at a secondary care hospital had sST2 measured within 24 hours of decompensated HF admission and following recompensation assessed by qualified clinicians, as part of the INST2ANT-HF study. sST2 samples were batched analysed at the end of recruitment period with a quantitative turbidimetric immunoassay. The primary outcome was the interval change between admission and discharge sST2 in patients admitted with decompensated heart failure. Related-samples Wilcoxon signed rank test was performed to evaluate for difference in sST2 at decompensation and recompensation. Comparison of median sST2 change was made using independent samples median test comparing alive and deceased patients at one year follow up.

Results: In 162 consecutive patients with paired samples, median age was 71 (IQR 61-80). 23.5% were female and 34% had an ischaemic aetiology. 94.4% presented with NYHA class III and IV and median ejection fraction was 26% (IQR 17-32). Median length of stay in hospital was 8 (IQR 5-13) days. Median levels of sST2 at decompensation and recompensation were 60.5 and 39.5ng/ml respectively (p<0.001) (Fig.1a). In patients who died within the first year, median levels of sST2, at decompensation and following recompensation, were significantly higher than in patients who were alive. (decompensation sST2 83 vs 56ng/ml; p = 0.007 and recompensation sST2 58 vs 34ng/ml; p = 0.036) (Fig. 1b). There was however no significant difference in the absolute or percentage change in sST2 level between those who died and those who survived (19.5 vs 16ng/ml, p = 0.437; 27 vs 35%, p = 0.363).

Conclusion: Our initial data demonstrates that in a cohort of patients admitted with acute decompensated HF, there was a significant difference in sST2 levels between clinician determined decompensation and recompensation. Higher levels of sST2 at both decompensation and recompensation were associated with a higher risk of mortality at one year. Utilizing newer biomarkers such as sST2 to predict outcomes may allow better risk stratification and identifying patients needing closer follow up. More research is needed however to determine what values would be useful in clinical practice.

Figure 1
Comparison of sST2 levels in deceased vs alive at 1 year following admission with acute decompensated heart failure

Figure 2