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**Research Article**

# Admission Soluble ST2 and Its Correlation with NT-Probnp and Hscrp in Acute Heart Failure and Acute Myocardial Infarction: A Prospective Observational Study

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**ABSTRACT**
**Background:** Natriuretic peptides such as NT-proBNP are the standard biomarkers for diagnosis and prognosis in heart failure, but their concentrations are influenced by age, renal function, and body-mass index. Soluble suppression of tumorigenicity-2 (sST2), a marker of myocardial fibrosis and remodelling, has emerged as a complementary, load-independent prognostic biomarker in heart failure, but data relating admission sST2 to NT-proBNP and high-sensitivity C-reactive protein (hsCRP), and to clinical outcomes, in Indian patients with acute heart failure (AHF) and acute myocardial infarction (MI) are scarce.
**Aim and Objectives:** To assess the correlation of admission sST2 with NT-proBNP and hsCRP in patients with acute heart failure and acute myocardial infarction. To evaluate the prognostic value of admission sST2 for in-hospital mortality, length of hospital stay, and 3-month mortality and readmission in this population.
**Methodology:**
**Study Design:** Prospective observational study. **Study Setting:** Tertiary cardiac-care centre, Latur, Maharashtra. **Study Population:** Consecutive patients older than 18 years admitted with acute heart failure (AHF, n=75) or acute myocardial infarction (MI, n=75); patients with pregnancy, cirrhosis, chronic pulmonary disease, rheumatic valvular heart disease, collagen vascular disease or rheumatoid arthritis, or septic shock were excluded. **Study period:** 2 April 2025 to 2 April 2026. **Sample Size = 150.** sST2, NT-proBNP, hsCRP, and troponin I were measured within 6 hours of hospitalisation. Correlations were assessed using Spearman's method; group medians were compared using the Mann-Whitney U test or Kruskal-Wallis H test. Patients were followed for in-hospital and 3-month outcomes (mortality and readmission).
**Results:** Of 150 patients (89 [59.3%] men, 61 [40.7%] women; mean age 65.61 ± 12.85 years), admission sST2 correlated positively with NT-proBNP ($r=0.871$) and hsCRP ($r=0.860$; both $p<0.001$) and negatively with left ventricular ejection fraction ($r=-0.878$, $p<0.001$) across the whole cohort, with similar directionally consistent correlations in the AHF and MI groups separately. Median sST2 was higher in patients who died in hospital than in survivors (228.5 vs 107.5 ng/mL, $p<0.001$), in those with a longer hospital stay (215.0 vs 116.0 ng/mL, $p<0.001$), and in those who died (215.5 vs 105.0 ng/mL, $p=0.004$) or were readmitted (204.0 vs 102.0 ng/mL, $p<0.001$) within 3 months.
**Conclusions:** In this Indian cohort, admission sST2 tracked closely with established biomarkers of myocardial wall stress and systemic inflammation and identified patients at higher risk of in-hospital death, prolonged hospital stay, and adverse 3-month outcomes in both AHF and MI. These findings support consideration of sST2 as an adjunct, relatively load-independent prognostic marker alongside NT-proBNP and hsCRP, while highlighting the need for population-specific reference ranges and larger multicentre validation.

**Keywords:** Sst2, NT-Probnp, Hscrp, Acute Heart Failure, Myocardial Infarction, Prognostic Biomarkers.

## INTRODUCTION

As elsewhere in the world, heart failure is a growing clinical problem in India, driven by improved survival after acute coronary syndromes and increasing longevity.[1] Coronary heart disease alone affected an estimated 30 million people in India by 2000 (around 3% prevalence),[2] and the burden of hypertension—

a major precursor of heart failure—was projected to rise from 118 million to 214 million people between 2000 and 2025.[1] As the population at risk of heart failure expands, tools to identify patients most likely to have adverse outcomes, and in whom guideline-directed therapy should be intensified, are increasingly important.

Natriuretic peptides, including B-type natriuretic peptide and its N-terminal prohormone (NT-proBNP), remain the most widely used biomarkers for diagnosis and prognostication in heart failure. However, their concentrations are substantially affected by age, renal function, atrial fibrillation, and body-mass index, which can complicate interpretation. Soluble suppression of tumorigenicity-2 (sST2), also known as interleukin-1 receptor-like 1, is a member of the interleukin-1 receptor family that has emerged as a biomarker of myocardial fibrosis, remodelling, and mechanical stress.[3] The membrane-bound isoform, ST2L, mediates the cardioprotective actions of interleukin-33; the soluble isoform, sST2, acts as a decoy receptor that sequesters interleukin-33 and thereby attenuates this cardioprotective signalling, promoting fibrosis and adverse remodelling when concentrations are high.[4,5]

Several cohort studies and meta-analyses have shown that sST2 is at least as strong a predictor of adverse cardiac outcomes as NT-proBNP in both acute and chronic heart failure, and that combining sST2 with natriuretic peptides improves risk prediction beyond either marker alone.[6–15] Because sST2 is a mechanically induced signal reflecting myocardial stress and fibrosis rather than volume status alone, it is relatively unaffected by renal function, age, or body-mass index, features that make it an attractive complement to NT-proBNP.[3] Despite this extensive evidence base, most data originate from European and North American cohorts, and information on sST2 and its relation to NT-proBNP and hsCRP in Indian patients with acute heart failure and myocardial infarction remains limited.[15]

We did this prospective observational study to assess the correlation between admission sST2, NT-proBNP, and hsCRP in patients with acute heart failure (AHF) and acute myocardial infarction (MI), and to determine whether admission sST2 was associated with in-hospital and 3-month clinical outcomes in this population.

## METHODS

### Study Design and Participants

This single-centre, prospective observational study was conducted in the Department of Cardiology, VDGMC SSH, Latur, Maharashtra, India, between April 2, 2025, and April 2, 2026. We enrolled consecutive adults (aged >18 years) admitted with AHF or acute MI. Patients were assigned to one of two groups: group 1 comprised patients with AHF in whom acute MI had been excluded as the precipitating cause before enrolment; group 2 comprised patients with acute MI. We excluded pregnant women and patients with cirrhosis; chronic obstructive pulmonary disease, bronchitis, asthma, or pulmonary fibrosis; heart failure due to rheumatic valvular disease; collagen vascular disease or rheumatoid arthritis; or septic shock, because these conditions are known to independently alter sST2 concentrations. Heart failure was defined using modified Framingham criteria (concurrent presence of two major criteria, or one major and two minor criteria) and was further classified as heart failure with reduced (≤40%), mildly reduced (40–49%), or preserved (>50%) ejection fraction on two-dimensional echocardiography.[26] Acute MI was defined according to the Fourth Universal Definition of Myocardial Infarction.[28] Written informed consent was obtained from all participants, and the study was approved by the institutional ethics committee of VDGMC SSH, Latur.

The target sample size, calculated for detecting a correlation coefficient with a two-sided α of 0·01 and β of 0·10, was 73 patients per group; we enrolled 75 patients in each group (150 in total).

### Procedures

After enrolment, detailed history, physical examination, and laboratory work-up were recorded for each patient. Venous blood samples for sST2, NT-proBNP, hsCRP, and troponin I were drawn within 6 h of hospitalisation. sST2 was measured by sandwich monoclonal lateral-flow immunoassay (AspectREADER; cut-off 35 ng/mL), hsCRP by nephelometry (BN ProSpec), NT-proBNP by enzyme-linked fluorescent assay (VIDAS), and troponin I by chemiluminescent microparticle immunoassay (ARCHITECT). Killip class was assessed at admission and discharge in the MI group,[29] and New York Heart Association class was assessed at admission, discharge, and 3-month follow-up in both groups. Two-dimensional echocardiography was done to determine left ventricular ejection fraction and

to identify regional wall-motion abnormality. Duration of hospital stay, in-hospital mortality, and status at discharge were recorded for all patients; survivors were followed clinically to 3 months after discharge for mortality and readmission.

**Outcomes**

The primary objective was to assess the correlation of admission sST2 with NT-proBNP and hsCRP in the AHF and MI groups. Secondary objectives were to assess the association between admission sST2 and in-hospital mortality, duration of hospital stay, admission ejection fraction, and 3-month mortality and readmission.

**Statistical Analysis**

Categorical variables are presented as n (%) and compared with the $\chi^2$ test or Fisher's exact test as appropriate. Normally distributed continuous variables are presented as mean (SD) and compared with the independent-samples t test; non-normally distributed variables are presented as median (IQR) and compared with the Mann-Whitney U test (two groups) or Kruskal-Wallis H test (three groups). Correlations between continuous variables were assessed with Spearman's rank correlation. A two-sided p-value of less than 0·05 was considered significant. Analyses were done with SPSS version 22.0 (IBM, Armonk, NY, USA).

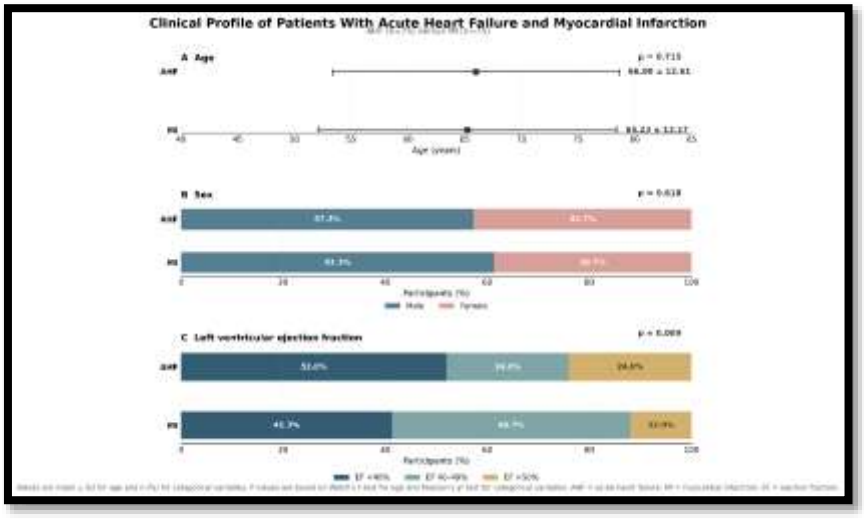
**RESULTS**

Between April 2, 2025, and April 2, 2026, we enrolled 150 patients: 75 with AHF (group 1) and 75 with acute MI (group 2). Overall, 89 (59·3%) of 150 patients were male, and 61 (40·7%) were female (male: female ratio 1·46:1); mean age was 65·61 (SD 12·85) years (range 37–89), with similar age and sex distributions in the two groups (Table 1). In the MI group, 31 (41·3%) patients had ejection fraction below 40%, compared with 39 (52·0%) in the AHF group (Table 1).

Table 1: Baseline Demographic and Echocardiographic Characteristics

| Characteristic | AHF (N=75) | MI (N=75) | Total (N=150) |
|---|---|---|---|
| Age, Years, Mean (SD) | 66·00 (12·61) | 65·23 (13·17) | 65·61 (12·85) |
| Age Range, Years | 37–89 | 37–89 | 37–89 |
| Male Sex, N (%) | 43 (57·3%) | 46 (61·3%) | 89 (59·3%) |
| Female Sex, N (%) | 32 (42·7%) | 29 (38·7%) | 61 (40·7%) |
| Ejection Fraction <40%, N (%) | 39 (52·0%) | 31 (41·3%) | 70 (46·7%) |
| Ejection Fraction 40–49%, N (%) | 18 (24·0%) | 35 (46·7%) | 53 (35·3%) |
| Ejection Fraction >50%, N (%) | 18 (24·0%) | 9 (12·0%) | 27 (18·0%) |

Data are mean (SD), n (%), or as indicated. AHF=acute heart failure. MI=myocardial infarction.




**Sst2 Concentrations and Correlation with Nt-Probnp, Hscrp, and Ejection Fraction**

Median (IQR) admission sST2 was 164·0 (107·0) ng/mL in the AHF group and 107·0 (118·0) ng/mL in the MI group (129·5 [126·5]

ng/mL overall); 134 (89·3%) of 150 patients had sST2 above the 35 ng/mL reference cut-off (table 2). Admission sST2 correlated positively with NT-proBNP in both the AHF group (r=0·830) and the MI group (r=0·946; both p<0·001), and with hsCRP in both groups (AHF r=0·851; MI r=0·867; both p<0·001). Across the whole cohort, sST2 correlated positively with NT-proBNP (r=0·871) and hsCRP (r=0·860, both p<0·001), and negatively with ejection fraction on admission (r=−0·878, p<0·001), with similar inverse correlations in the AHF (r=−0·882) and MI (r=−0·926) groups separately (table 2). Median sST2 fell progressively as ejection-fraction category improved, from 191·0 ng/mL in patients with ejection fraction below 40% to 52·0 ng/mL in those with ejection fraction above 50% (p<0·001 for trend in both groups; Table 3).

Table 2: Distribution of Sst2 and Its Correlation with NT-Probnp, Hscrp, and Ejection Fraction

| Sst2 | AHF (N=75) | MI (N=75) | Total (N=150) |
|---|---|---|---|
| Median (IQR), Ng/Ml | 164·0 (107·0) | 107·0 (118·0) | 129·5 (126·5) |
| Range, Ng/Ml | 14–270 | 12–130 | 12–270 |
| ≤35 Ng/Ml, N (%) | 7 (9·3%) | 9 (12·0%) | 16 (10·7%) |
| >35 Ng/Ml, N (%) | 68 (90·7%) | 66 (88·0%) | 134 (89·3%) |
| Correlation With NT-Probnp, R | 0·830 | 0·946 | 0·871 |
| P Value | <0·001 | <0·001 | <0·001 |
| Correlation With Hscrp, R | 0·851 | 0·867 | 0·860 |
| P Value | <0·001 | <0·001 | <0·001 |
| Correlation With Ejection Fraction, R | −0·882 | −0·926 | −0·878 |
| P Value | <0·001 | <0·001 | <0·001 |

Correlation coefficients are Spearman's r. sST2=soluble suppression of tumorigenicity-2. NT-proBNP=N-terminal pro-B-type natriuretic peptide. hsCRP=high-sensitivity C-reactive protein.

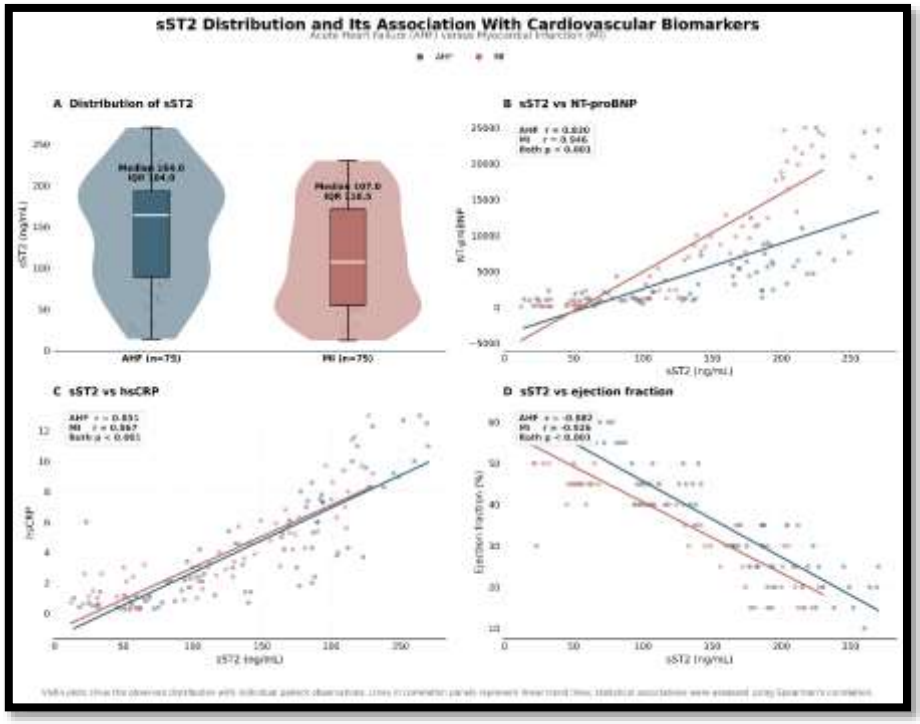



Table 3: Median Sst2 by Admission Ejection-Fraction Category

| Ejection Fraction | AHF Median Sst2 (IQR) | MI Median Sst2 (IQR) | Total Median Sst2 (IQR) |
|---|---|---|---|
| <40% | 193·0 (45·0) | 181·0 (54·0) | 191·0 (45·5) |

| | | | |
|---|---|---|---|
| 40–49% | 112·0 (35·0) | 64·0 (54·0) | 98·0 (58·0) |
| >50% | 60·5 (44·3) | 34·0 (27·5) | 52·0 (39·0) |
| P-Value* | <0·001 | <0·001 | <0·001 |

*Kruskal-Wallis H test. Data are median (IQR) sST2, ng/mL.

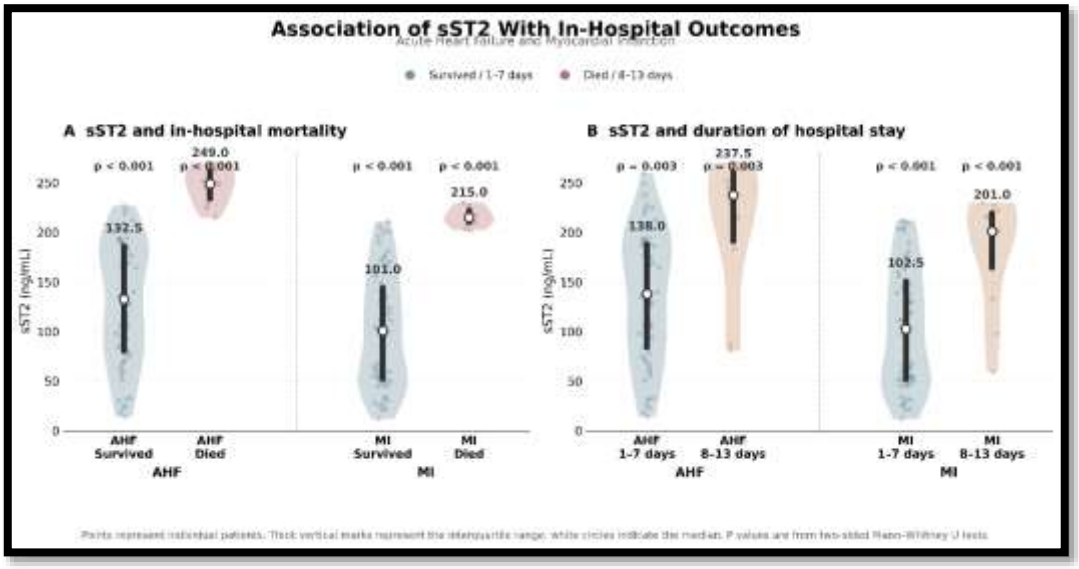
**Sst2 and In-Hospital Outcomes**

At discharge, 20 (13·3%) of 150 patients had died in hospital: 11 (14·7%) of 75 in the AHF group and nine (12·0%) of 75 in the MI group. Median sST2 was higher among patients who died in hospital than among survivors in both the AHF group (249·0 vs 132·5 ng/mL) and the MI group (215·0 vs 101·0 ng/mL; both p<0·001; Table 4). A longer hospital stay (8–13 days vs 1–7 days) was likewise associated with higher median sST2 in both groups (AHF p=0·002; MI p<0·001; Table 4).

Table 4: Sst2 and In-Hospital Mortality and Length of Stay

| In-Hospital Outcome | AHF (N=75) | MI (N=75) |
|---|---|---|
| Died In Hospital, N (%) | 11 (14·7%) | 9 (12·0%) |
| Median Sst2, Survivors (Ng/Ml) | 132·5 | 101·0 |
| Median Sst2, Died (Ng/Ml) | 249·0 | 215·0 |
| P Value† | <0·001 | <0·001 |
| Hospital Stay 8–13 Days, N (%) | 10 (13·3%) | 11 (14·7%) |
| Median Sst2, Stay 1–7 Days (Ng/Ml) | 138·0 | 102·5 |
| Median Sst2, Stay 8–13 Days (Ng/Ml) | 237·5 | 201·0 |
| P Value† | 0·002 | <0·001 |

†Mann-Whitney U test. Data are n (%) or median sST2 (ng/mL).




**Sst2 and 3-Month Outcomes**

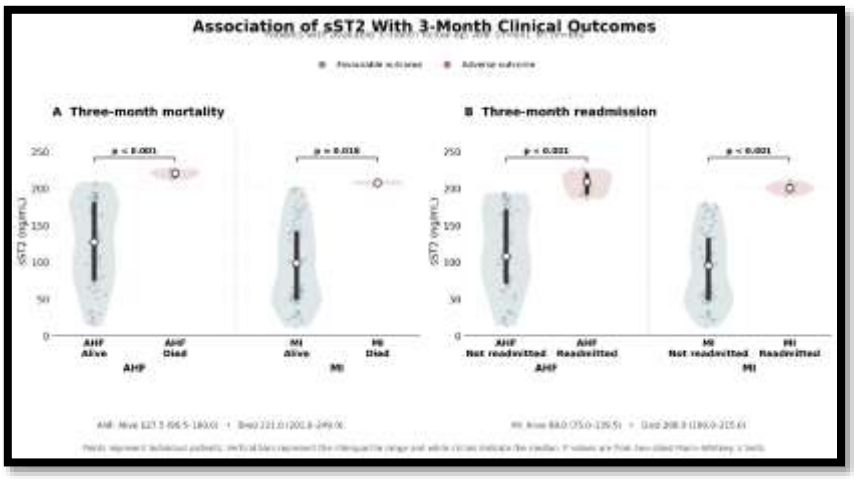
Among the 130 patients who survived to discharge, six (4·6%) died within 3 months (four [6·2%] of 64 in the AHF group; two [3·0%] of 66 in the MI group) and 15 (11·5%) were readmitted (nine [14·1%] of 64 in the AHF group; six [9·1%] of 66 in the MI group). Median sST2 was higher among patients who died within 3 months than among those alive at 3 months in both the AHF group (221·0 vs 127·5 ng/mL, p=0·008) and the MI group (208·0 vs 99·0 ng/mL, p=0·017), and was higher among patients who were readmitted than among those who were not, in both groups (both p<0·001; table 5).

Table 5: Sst2 and 3-Month Mortality and Readmission

| 3-Month Outcome | Ahf (N=64) | Mi (N=66) |
|---|---|---|
| Died Within 3 Months, N (%) | 4 (6·2%) | 2 (3·0%) |
| Median Sst2, Alive At 3 Months (Ng/Ml) | 127·5 | 99·0 |
| Median Sst2, Died Within 3 Months (Ng/Ml) | 221·0 | 208·0 |

| | | |
|---|---|---|
| P Value† | 0·008 | 0·017 |
| Readmitted Within 3 Months, N (%) | 9 (14·1%) | 6 (9·1%) |
| Median Sst2, Not Readmitted (Ng/Ml) | 108·0 | 95·5 |
| Median Sst2, Readmitted (Ng/Ml) | 209·0 | 200·5 |
| P Value† | <0·001 | <0·001 |

†Mann-Whitney U test. Data are n (%) or median sST2 (ng/mL).




**DISCUSSION**

In this prospective study of 150 Indian patients admitted with AHF or acute MI, admission sST2 correlated strongly with NT-proBNP and hsCRP, and inversely with ejection fraction, in both disease groups. Higher admission sST2 identified patients at greater risk of in-hospital death, longer hospital stay, and death or readmission within 3 months. These findings are consistent with sST2's proposed role as a marker of myocardial fibrosis and adverse remodelling that complements, rather than duplicates, the information provided by natriuretic peptides and inflammatory markers.[3–5]

The positive correlation we observed between sST2 and NT-proBNP in AHF is consistent with previous reports.[6,16–19] Direct comparisons of sST2 with hsCRP in AHF are less common in the published literature, but our findings are in keeping with data suggesting that multimarker strategies combining sST2 with inflammatory and neurohormonal markers improve risk discrimination beyond any single biomarker.[20,21] The inverse relation we found between sST2 and ejection fraction is consistent with a previous report linking higher sST2 to worse left ventricular systolic function,[22] and supports the biological premise that sST2 reflects the degree of myocardial mechanical stress and fibrotic remodelling that accompanies systolic impairment.

Median admission sST2 concentrations in our cohort (164·0 ng/mL in AHF, 107·0 ng/mL in MI) were higher than those reported in several Western AHF cohorts, in which median values in the range of 60–97 ng/mL have been described.[8,16,17,19,24] Although the reasons for this difference cannot be established from an observational study of this design, potential explanations include differences in disease severity or stage at presentation, comorbidity profile, assay characteristics, or population-specific reference distributions, and merit dedicated investigation. Notably, our AHF cohort was, on average, older than that reported in the ASIAN-HF registry of Indian patients (mean age 66·0 years vs 59·6 years), with a higher proportion of women (42·7% vs 27·0%), differences that may partly reflect the tertiary, single-centre nature of our cohort.[27]

Consistent with previous studies in both heart failure and MI populations, we found that higher sST2 was associated with increased in-hospital and short-term mortality, longer hospital stay, and higher 3-month readmission.[9,25,30] Because sST2 concentrations can change substantially over time and its interpretation may be influenced by non-cardiac inflammatory or fibroproliferative conditions, it is best used as an adjunct to, rather than a replacement for, established clinical assessment and natriuretic-peptide testing, and its measurement should be interpreted cautiously in patients with

confounding conditions such as asthma, pulmonary fibrosis, rheumatoid arthritis, or active malignancy.[3]

### Limitations

This study has several limitations. It was done at a single tertiary centre with a modest sample size, so findings should be regarded as hypothesis-generating pending confirmation in larger, multicentre south Asian cohorts. There was no separate control group of patients without cardiac disease, and follow-up was limited to 3 months, precluding assessment of longer-term prognostic value. sST2, NT-proBNP, and hsCRP were measured only at admission; serial measurements, which have been shown to add incremental prognostic information in other cohorts,[2,3] were not done because of cost constraints. Finally, as an observational study, it can establish association but not causation, and residual confounding cannot be excluded.

## CONCLUSION

In this prospective cohort of Indian patients with acute heart failure or acute myocardial infarction, admission sST2 correlated positively with NT-proBNP and hsCRP and negatively with ejection fraction, and higher sST2 was associated with greater in-hospital and 3-month mortality, longer hospital stay, and higher readmission. These findings support a role for sST2 as a complementary, relatively load-independent prognostic biomarker in this setting, while underscoring the need for larger multicentre studies to define population-specific reference ranges and to establish whether sST2-guided management improves outcomes.

## REFERENCES


1. Huffman MD, Prabhakaran D. Heart failure: epidemiology and prevention in India. Natl Med J India 2010; 23: 283–88.
2. Gupta R, Joshi P, Mohan V, Reddy KS, Yusuf S. Epidemiology and causation of coronary heart disease and stroke in India. Heart 2008; 94: 16–26.
3. Pascual-Figal DA, Januzzi JL. The biology of ST2: the international ST2 consensus panel. Am J Cardiol 2015; 115 (suppl 7B): 3B–7B.
4. Kakkar R, Lee RT. The IL-33/ST2 pathway: therapeutic target and novel biomarker. Nat Rev Drug Discov 2008; 7: 827–40.
5. Sanada S, Hakuno D, Higgins LJ, Schreiter ER, McKenzie ANJ, Lee RT. IL-33 and ST2 comprise a critical biomechanically induced and cardioprotective signaling system. J Clin Invest 2007; 117: 1538–49.
6. Aimo A, Vergaro G, Ripoli A, et al. Meta-analysis of soluble suppression of tumorigenicity-2 and prognosis in acute heart failure. JACC Heart Fail 2017; 5: 287–96.
7. Aimo A, Vergaro G, Passino C, et al. Prognostic value of soluble suppression of tumorigenicity-2 in chronic heart failure: a meta-analysis. JACC Heart Fail 2017; 5: 280–86.
8. Lassus J, Gayat E, Mueller C, et al. Incremental value of biomarkers to clinical variables for mortality prediction in acutely decompensated heart failure: the Multinational Observational Cohort on Acute Heart Failure (MOCA) study. Int J Cardiol 2013; 168: 2186–94.
9. Manzano-Fernández S, Mueller T, Pascual-Figal D, Truong QA, Januzzi JL. Usefulness of soluble concentrations of interleukin family member ST2 as predictor of mortality in patients with acutely decompensated heart failure relative to left ventricular ejection fraction. Am J Cardiol 2011; 107: 259–67.
10. Januzzi JL, Peacock WF, Maisel AS, et al. Measurement of the interleukin family member ST2 in patients with acute dyspnea: results from the PRIDE study. J Am Coll Cardiol 2007; 50: 607–13.
11. Bayés-Genís A, de Antonio M, Galán A, et al. Combined use of high-sensitivity ST2 and NT-proBNP to improve the prediction of death in heart failure. Eur J Heart Fail 2012; 14: 32–38.
12. Bayes-Genis A, de Antonio M, Vila J, et al. Head-to-head comparison of 2 myocardial fibrosis biomarkers for long-term heart failure risk stratification: ST2 versus galectin-3. J Am Coll Cardiol 2014; 63: 158–66.
13. Gaggin HK, Szymonifka J, Bhardwaj A, et al. Head-to-head comparison of serial soluble ST2, growth differentiation factor-15, and highly-sensitive troponin T measurements in patients with chronic heart failure. JACC Heart Fail 2014; 2: 65–72.
14. Mebazaa A, Di Somma S, Maisel AS, Bayes-Genis A. ST2 and multimarker

testing in acute decompensated heart failure. Am J Cardiol 2015; 115 (suppl 7B): 38B–43B.

15. Bahuleyan CG, Alummoottil GK, Abdullakutty J, et al. Prognostic value of soluble ST2 biomarker in heart failure patients with reduced ejection fraction—a multicenter study. Indian Heart J 2018; 70 (suppl 3): S79–84.
16. Pascual-Figal DA, Manzano-Fernández S, Boronat M, et al. Soluble ST2, high-sensitivity troponin T- and N-terminal pro-B-type natriuretic peptide: complementary role for risk stratification in acutely decompensated heart failure. Eur J Heart Fail 2011; 13: 718–25.
17. Breidthardt T, Balmelli C, Twerenbold R, et al. Heart failure therapy-induced early ST2 changes may offer long-term therapy guidance. J Card Fail 2013; 19: 821–28.
18. Friões F, Lourenço P, Laszczynska O, Almeida PB, Guimarães JT, Januzzi JL, Bettencourt P. Prognostic value of sST2 added to BNP in acute heart failure with preserved or reduced ejection fraction. Clin Res Cardiol 2015; 104: 491–99.
19. Llibre C, Zamora E, Caballero À, et al. The real-life value of ST2 monitoring during heart failure decompensation: impact on long-term readmission and mortality. Biomarkers 2018; 23: 1–8.
20. Pellicori P, Zhang J, Cuthbert J, et al. High-sensitivity C-reactive protein in chronic heart failure: patient characteristics, phenotypes, and mode of death. Cardiovasc Res 2020; 116: 91–100.
21. Wang TJ, Wollert KC, Larson MG, et al. Prognostic utility of novel biomarkers of cardiovascular stress: the Framingham Heart Study. Circulation 2012; 126: 1596–604.
22. Shah RV, Chen-Tournoux AA, Picard MH, van Kimmenade RRJ, Januzzi JL. Serum levels of the interleukin-1 receptor family member ST2, cardiac structure and function, and long-term mortality in patients with acute dyspnea. Circ Heart Fail 2009; 2: 311–19.
23. Manzano-Fernández S, Januzzi JL, Boronat-García M, et al. Serial monitoring of soluble interleukin family member ST2 in patients with acutely decompensated heart failure. Cardiology 2012; 122: 158–66.
24. Gandhi PU, Gaggin HK, Belcher AM, et al. Analysis of BAG3 plasma concentrations in patients with acutely decompensated heart failure. Clin Chim Acta 2015; 445: 73–78.
25. Sabatine MS, Morrow DA, Higgins LJ, et al. Complementary roles for biomarkers of biomechanical strain ST2 and N-terminal prohormone B-type natriuretic peptide in patients with ST-elevation myocardial infarction. Circulation 2008; 117: 1936–44.
26. Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J 2016; 37: 2129–200.
27. Tromp J, Tay WT, Ouwerkerk W, et al. Multimorbidity in patients with heart failure from 11 Asian regions: a prospective cohort study using the ASIAN-HF registry. PLoS Med 2018; 15: e1002541.
28. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). Circulation 2018; 138: e618–51.
29. Killip T 3rd, Kimball JT. Treatment of myocardial infarction in a coronary care unit: a two year experience with 250 patients. Am J Cardiol 1967; 20: 457–64.
30. Dalal JJ, Digrajkar A, Das B, Bansal M, Toomu A, Maisel AS. ST2 elevation in heart failure, predictive of a high early mortality. Indian Heart J 2018; 70: 822–27.