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Original article

Evaluation of Serum Soluble Suppression of Tumorigenicity 2 (sST-2) in Ischemic Heart Disease

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Abstract

Background: Ischemic heart disease (IHD) remains a primary cause of morbidity and mortality worldwide. Identifying effective biomarkers for early diagnosis is crucial for optimal patient outcomes. Serum Soluble Suppression of Tumorigenicity 2 (sST-2), a member of the interleukin-1 receptor family, has gained attention as a novel marker for myocardial stress and injury. **Methods:** This case-control study, conducted at Benha University Hospitals between February and December 2023, involved 60 IHD patients (30 with acute myocardial infarction [AMI] and 30 with angina pectoris) and 20 healthy controls. Serum sST-2 levels and traditional cardiac biomarkers (troponin I, CK, and CK-MB) were measured using ELISA. Statistical analyses were performed to assess correlations between sST-2 and various clinical parameters, including BMI, lipid profiles, blood pressure, and LVEF. **Results:** Serum ST-2 levels were significantly elevated in AMI (78.3 ± 37.3 ng/ml) and angina patients (71.4 ± 39.7 ng/ml) compared to controls (26.1 ± 6.9 ng/ml, $P < 0.001$). Troponin I and CK-MB were also higher in both patient groups versus controls ($P < 0.001$). Positive correlations were identified between sST-2 and BMI ($r = 0.314$, $P = 0.005$), SBP ($r = 0.303$, $P = 0.006$), and troponin I ($r = 0.396$, $P < 0.001$), while significant negative correlations were noted with LVEF ($r = -0.526$, $P < 0.001$). **Conclusion:** Serum sST-2 shows promise as a diagnostic and prognostic biomarker for IHD. Integrating sST-2 into clinical practice could improve the early identification and management of IHD, particularly in acute coronary syndromes.

Keywords: Ischemic heart disease, sST-2, myocardial infarction, biomarkers, angina pectoris.

Introduction

Globally, cardiovascular disease ranks among leading contributors to mortality. Ischemic heart disease (IHD) refers to cardiac dysfunction arising from narrowing of coronary arteries, which restricts perfusion of blood and oxygen to myocardial tissue. primary etiologies underlying IHD include coronary vasospasm, embolic events, inflammatory vasculitis, acute rupture or dissection of atherosclerotic plaques within coronary vessels, and chronic fixed stenosis. Ultimately, IHD develops when oxygen supply becomes inadequate to meet metabolic demands of myocardium (1).

In clinical practice, patients presenting with moderate to high risk for myocardial infarction (MI) typically undergo biomarker evaluation to detect myocardial injury, with cardiac troponin I and Creatine Kinase-MB being most utilized indicators. An optimal biomarker for myocardial damage must exhibit high specificity and sensitivity, provide precise quantification, and demonstrate a prompt elevation in serum concentration, enabling timely and accurate identification of cardiac events (2).

ST-2, a constituent of toll-like/interleukin-1 receptor superfamily, was initially identified in 1989. Its relevance to cardiovascular field gained traction in 2002 when research revealed that myocardial cells could upregulate its expression under conditions of cardiac stress, suggesting a pivotal role in cardiovascular physiology and pathology. ST-2 manifests in two principal isoforms: membrane-bound, cellular variant (ST-2L) and soluble, circulating form (sST-2) (3).

Several studies have identified ST-2 as an early indicator of myocardial remodelling. It is prominently expressed on endothelial cells of both microvasculature and macrovasculature. including those in aorta and coronary arteries, in humans. Additionally, ST-2 is present on cardiomyocytes in mice and rats subjected to biomechanical strain (3). Consequently, sST-2 is considered a promising biomarker for IHD, alongside established biomarkers (3).

As a result, we decided to initiate this research study to investigate sST-2 serum levels in patients with IHD and to explore its potential as a diagnostic biomarker for this condition.

Subjects and Method

Study design and population

This case-control study involved 60 patients diagnosed with IHD, categorized into two groups based on their clinical presentations: 30 patients with acute MI and 30 with angina pectoris. An additional control group of 20 apparently healthy individuals, matched in age and sex with patients, was also included. study was conducted at Departments of Clinical & Chemical Pathology and Cardiology at Benha University Hospitals during period from February 2023 to December 2023. The study protocol was reviewed and approved by the Research Ethics Committee of the Faculty of Medicine, Benha University (MS 1-5-2023). A signed consent form was obtained from each participant.

Inclusion criteria Myocardial infarction cohort included individuals exhibiting typical symptoms of chest pain persisting

for over 20 minutes, accompanied by elevated levels of CK and CK-MB. Additionally, diagnostic criteria involved troponin I levels exceeding 0.1 ng/mL, ST-segment elevation of no less than 0.1 mV across two or more contiguous electrocardiographic leads, or presence of a complete left bundle branch block on ECG (3).

For angina group, inclusion criteria involved patients with typical chest pain, no significant elevation in cardiac enzymes, and evidence of ischemia on ECG without persistent ST-segment elevation (3).

Exclusion Criteria included patients with active infections or inflammatory diseases, organ failure, autoimmune disorders, malignancies, long-term corticosteroid use, as well as those who were pregnant or nursing.

All patients were subjected to following assessments:

Detailed history taking (sex, age, marital status, education, employment, and medical conditions like hypertension and diabetes) was done. Clinical assessments included general examinations (vital signs and temperature) and systemic evaluations of cardiovascular, respiratory, gastrointestinal, and neurological systems. results of routine laboratory investigations, including CBC, blood urea, serum creatinine, sodium (Na), potassium (K), CRP, ALT, and AST, were retrieved and recorded from patients' medical files. Laboratory investigations encompassed cardiac biomarkers (cardiac troponin I, CK-total, CK-MB), a lipid profile (LDL, HDL, triglycerides, total cholesterol), Haemoglobin A1c, and serum sST-2 levels, measured by ELISA.

Assessment of serum sST-2 concentration: A double antibody sandwich-ELISA method was employed to measure serum sST-2 levels using a human sST-2 ELISA kit. Blood samples were collected, allowed to clot, and centrifuged, with serum stored at -80°C. In assay, pre-coated plates containing specific antibodies for human sST-2 captured target protein. detection process employed a biotinylated antibody coupled with an avidin-HRP conjugate, resulting in a colorimetric reaction. Colour generated intensity was assessed spectrophotometrically, with concentration of sST-2 being directly correlated with magnitude of colour development.

Reagent preparation: All reagents were brought to room temperature before use. Wash buffer was prepared by diluting 30 ml of concentrate in 720 ml of water. To create standard working solution, standard was centrifuged and then mixed with reference standard and sample diluent. After allowing mixture to stand, it was serially diluted to achieve concentrations ranging from 20 ng/ml to 0 ng/ml. This dilution involved a stepwise transfer of 500 µl starting from 20 ng/ml to create progressively lower concentrations. Biotinylated detection antibody and HRP conjugate solutions were prepared by diluting concentrated solutions to 1x working solutions, with both solutions centrifuged prior to dilution. Each preparation step ensured that slightly more reagent was available than necessary for experiment.

Assay procedure: Each well was designated for standard, blank, and samples, with 100 µl of each added and incubated at 37°C for 90 minutes. A volume of 100 µl of biotinylated detection antibody working solution was dispensed into each well, followed by a one-hour incubation at 37°C. After completion of incubation, residual solution was discarded. wells were then washed three times with 350 µl of wash buffer, allowing buffer to rest for 1-2 minutes before decanting. Once wells were sufficiently dried, 100 µl of HRP conjugate working solution was added, with incubation maintained at 37°C for 30 minutes. This was followed by five additional wash cycles, performed in the same manner as the initial washing step. Thereafter, 90 µl of substrate reagent was dispensed into each well, followed by a 15-minute incubation at 37°C in complete darkness. reaction was halted by adding 50 µl of Stop Solution to each well. Finally, optical density (OD) at 450 nm was immediately measured for each well using an automated microplate reader.

Statistical analysis

Statistical analysis was conducted using SPSS v28 (IBM, Armonk, New York, United States). Data's normality was evaluated using Shapiro-Wilks test and histograms. Quantitative parametric data were analyzed using ANOVA and Tukey's post hoc test, while non-parametric data were evaluated with Kruskal-Wallis and Mann Whitney tests. Qualitative data were analyzed using Chi-square test. Significance was set at a P value < 0.05. Spearman correlation and multivariate logistic regression were used to assess correlations and relationships between multiple variables, respectively (3).

Results

In present study, baseline characteristics such as age, sex, weight, height, and BMI were not significantly different between two groups. However, regarding risk factors, hypertension (HTN) and diabetes mellitus (DM) showed significant differences between studied groups (P=0.036 and 0.022, respectively), being more prevalent in patients with angina and myocardial infarction. Smoking, dyslipidaemia, and chronic renal failure did not differ significantly among groups. **Table 1**

SBP was significantly higher in both angina and MI groups compared to control group (P<0.001 for both), with no significant difference between angina and MI groups. DBP was significantly elevated only in MI group compared to controls (P=0.005). hazard ratio showed no significant differences across all groups. Serum creatinine levels were higher in MI group than in controls (P=0.013), but similar between angina and other groups.

Other parameters, including hemoglobin, platelet count, white blood cell count, HbA1c, urea, sodium, potassium, and CRP, did not differ significantly among groups. Triglycerides and LDL levels were elevated in both angina and MI groups (P<0.05), with no significant difference between these groups. No significant differences were observed in ALT, AST, total cholesterol, and HDL levels. LVEF was significantly lower in MI group compared to angina and control groups (P=0.033 and P=0.001, respectively), with no significant difference between angina and control groups. **Table 2**

Regarding Cardiac biomarkers, troponin I, CK, CKMB, and ST-2 were significantly higher in angina and MI groups compared to controls (P<0.05), with no differences between angina and MI groups. **Table 3, Fig 1**

A significant positive correlation was observed between ST-2 and BMI, SBP, DBP, HbA1c, serum creatinine, LDL, and troponin I. Conversely, a significant negative correlation was found between ST-2 and HR, hemoglobin, total cholesterol, triglycerides, and LVEF. No significant correlation was noted between ST-2 and other parameters. **Table 4**

Table 1: Baseline Characteristics and Risk Factors of Studied Groups**.**

		Angina group (n=30)	**MI group (n=30)**	**Control group (n=20)**	**P value**
Age (years)	**Mean± SD**	54.2 ± 10.3	55.6 ± 8.29	53.3 ± 10.19	0.693
Sex	**Male**	19 (63.33%)	21 (70%)	9 (45%)	0.197
	Female	11 (36.67%)	9 (30%)	11 (55%)	
Weight (Kg)	**Mean± SD**	76.3 ± 9.56	78.7 ± 11.34	75.6 ± 9.31	0.501
Height (m)	**Mean± SD**	1.68 ± 0.05	1.67 ± 0.04	1.68 ± 0.05	0.973
BMI (Kg/m^2)	**Mean± SD**	27.1 ± 3.61	28.3 ± 4.15	26.9 ± 3.52	0.716
Smoking		12 (40%)	14 (46.67%)	6 (30%)	0.499
HTN		17 (56.67%)	20 (66.67%)	6 (30%)	**0.036***
DM		16 (53.33%)	13 (43.33%)	3 (15%)	**0.022***
Dyslipidaemia		10 (33.33%)	11 (36.67%)	4 (20%)	0.438
Chronic renal failure		5 (16.67%)	3 (10%)	1 (5%)	0.425

BMI: body mass index, HTN: hypertension, DM: diabetes mellitus, *: statistically significant as p value <0.05.

Table 2: Clinical Examinations of Vital Signs, Routine Laboratory Investigations and LVEF of studied groups**.**

		Angina group (n=30)	MI group (n=30)	Control group (n=20)	P value
HR (beat/min)	**Mean± SD**	84.97 ± 6.66	84.6 ± 6.18	86.95 ± 4.88	0.391
SBP (mmHg)	**Mean± SD**	136.7 ± 9.94	133.3 ± 10.93	120.5 ± 7.59	**<0.001***
	Post hoc	P1=0.222, **P2<0.001*, P3<0.001***			
DBP (mmHg)	**Mean± SD**	79.7 ± 7.65	83.0 ± 7.94	75.5 ± 9.99	**0.013***
	Post hoc	P1=0.103, P2=0.102, **P3=0.005***			
Hb (g/dl)	**Mean± SD**	11.78 ± 1.08	11.84 ± 1.12	11.52 ± 1.23	0.600
PLT (*10^9/L)	**Mean± SD**	264.6 ± 48.15	270.5 ± 55.63	257.7 ± 44.38	0.677
WBCs (*10^9/L)	**Mean± SD**	7.47 ± 1.28	6.98 ± 1.61	7.37 ± 1.96	0.471
HBA1C (%)	**Mean± SD**	5.11 ± 1.73	4.98 ± 1.78	4.02 ± 1.16	0.055
Serum creatinine (mg/dl)	**Mean± SD**	1.50 ± 1.08	1.96 ± 1.58	1.05 ± 0.11	**<0.001***
	Post hoc	P1=0.187, P2=0.070, **P3=0.013***			
Urea (mg/dl)	**Mean± SD**	43.6 ± 16.32	38.8 ± 15.56	47 ± 15.64	0.194
Na^+ (mEq/l)	**Mean± SD**	141.7 ± 1.91	141.9 ± 2.16	140.9 ± 1.62	0.177
K^+ (mEq/l)	**Mean± SD**	5.1 ± 0.89	4.7 ± 0.84	5 ± 0.83	0.228
CRP (mg/l)	**Mean± SD**	4.7 ± 1.66	5.4 ± 1.18	5.01 ± 1.51	0.231
ALT (U/l)	**Mean± SD**	30.2 ± 6.58	29.8 ± 5.62	30.9 ± 6.37	0.817
AST (U/l)	**Mean± SD**	35.2 ± 8.13	36.5 ± 8.54	35.7 ± 9.09	0.830
Total cholesterol (mg/dl)	**Mean± SD**	194.9 ± 65.5	168.7 ± 53.81	205.3 ± 58.26	0.080
Triglycerides (mg/dl)	**Mean± SD**	210.0 ± 51.89	216.5 ± 47.95	152.6 ± 51.65	**<0.001***
	Post hoc	P1=0.618, **P2<0.001*, P3<0.001***			
HDL (mg/dl)	**Mean± SD**	50.5 ± 5.2	50.8 ± 5.94	49.7 ± 5.61	0.776
HDL (mg/dl)	**Mean± SD**	168.5 ± 40.16	172.3 ± 36.21	126.6 ± 26.14	**<0.001***
	Post hoc	P1=0.702, **P2<0.001*, P3<0.001***			
LVEF (%)	**Mean± SD**	46.7 ± 5.83	43.1 ± 6.79	49.7 ± 4.77	**0.001***
	Post hoc	**P1=0.033*,** P2=0.066, **P3=0.001***			

*: Statistically significant as P value <0.05, P1: P value between angina and MI groups, P2: P value between angina and control groups, P3: P value between MI and control groups.

Table 3: Cardiac biomarkers & sST-2 concentrations of studied groups.

		Angina group (n=30)	MI group (n=30)	Control group (n=20)	P value
Troponin I (ng/ml)	**Mean± SD**	35.8 ± 20.21	41.8 ± 23.16	0.1 ± 0.01	**<0.001***
	Post hoc	P1=0.289, **P2<0.001*, P3<0.001***			
CKMB (mg/dl)	**Mean± SD**	141.066 ± 47.34	157.33 ± 48.75	19.78 ± 8.73	**<0.001***
	Post hoc	P1=0.164, **P2<0.001*, P3<0.001***			
CK (U/L)	**Mean± SD**	276.65 ±38.70	279.6 ± 39.2	39.12 ± 12.35	**<0.001***
	Post hoc	P1=0.465, **P2<0.001*, P3<0.001***			
ST2 (ng/ml)	**Mean± SD**	71.4 ± 39.72	78.3 ± 37.28	26.1 ± 6.9	**<0.001***
	Post hoc	P1=0.486, **P2<0.001*, P3<0.001***			

CK-MB, creatine kinase myocardial band, *: statistically significant as P value <0.05, P1: p value between angina and MI groups, P2: p value between angina and control groups, P3: p value between MI and control groups.

Table 4: Correlation between ST-2 and different parameters.

	ST-2 (ng/L)	
	r	**P value**
Age (years)	0.048	0.673
BMI (Kg/m^2)	0.314	**0.005***
HR (beat/min)	-0.314	**<0.001***
SBP (mmHg)	0.303	**0.006***
DBP (mmHg)	0.258	**0.021***
Hb (g/dL)	-0.306	**0.006***
PLT (*10^9/L)	0.064	0.573
WBCs (*10^9/L)	-0.104	0.360
HbA1c (%)	0.286	**0.010***
Serum creatinine (mg/dL)	0.609	**<0.001***
Urea (mg/dL)	-0.075	0.508
Na$^+$ (mEq/L)	0.080	0.482
K$^+$ (mEq/L)	-0.122	0.282
CRP (mg/L)	0.057	0.667
ALT (U/L)	-0.193	0.086
AST (U/L)	-0.056	0.625
Total cholesterol (mg/dL)	-0.326	**0.003***
Triglycerides (mg/dL)	-0.272	**0.015***
LDL (mg/dL)	0.327	**0.003***
HDL (mg/dL)	0.188	0.095
Troponin I (ng/ml)	0.396	**<0.001***
LVEF (%)	-0.526	**<0.001***

*: statistically significant as P value <0.05.

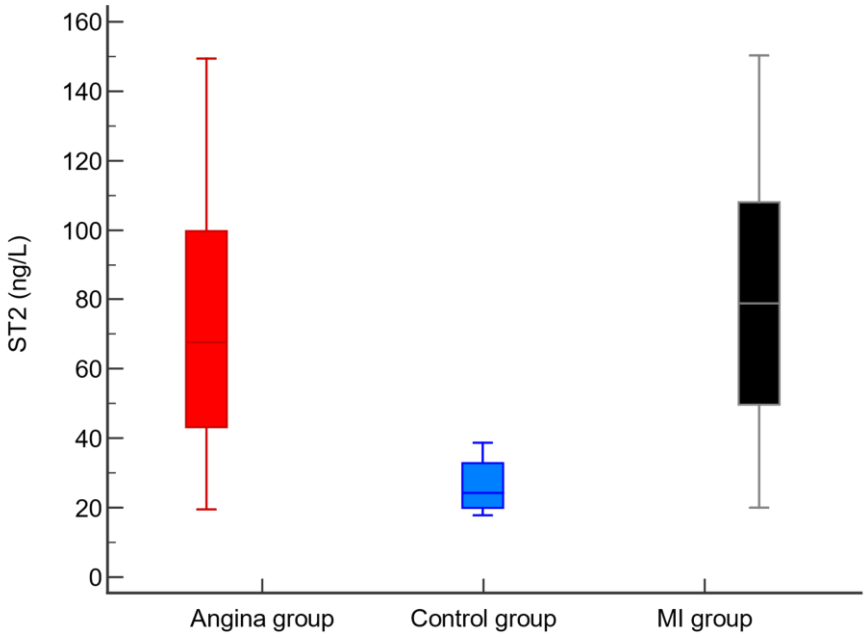

Figure 1: Serum soluble suppression of tumorigenicity 2 (ST2) of studied groups

Discussion

In this study, we explored diagnostic potential of sST-2 in patients with IHD. elevation of sST-2 levels in IHD patients, particularly in those with AMI and angina pectoris, underscores its significance as a marker of myocardial stress.
Regarding baseline characteristics of studied groups, a study reported similar demographic characteristics as reported in current work (4).

Moreover, investigation conducted by Demyanets and colleagues revealed no significant differences across four study groups with respect to age, gender, diabetes status, or renal function. However, control participants exhibited a lower incidence of hyperlipidemia compared to those with CAD, despite presenting with a higher BMI. Notably, prevalence of smoking was markedly higher among individuals with STEMI, whereas occurrence of hypertension was significantly reduced within this group (5).
Our results indicated that ST-2 levels were substantially elevated in both angina and MI groups in comparison to control group ($P < 0.05$). However, no statistically significant variations were detected among remaining groups or between each other.

Our findings are consistent with previous studies that have highlighted elevated levels of serum sST-2 in patients with ischemic heart disease, particularly those experiencing acute MI. Some authors conducted a multibiomarker analysis in patients with acute myocardial infarction and reported that median sST-2 levels were significantly increased in both STEMI (13,210.9 pg/mL) and NSTEMI patients (11,989.1 pg/mL), compared to control group (5,247.7 pg/mL; $P < 0.001$) (5). Parallel results reinforce potential of sST-2 as a robust biomarker for myocardial stress, particularly in acute coronary syndromes, and highlight its diagnostic and prognostic value in ischemic heart disease.

Aligned with our findings, a study also shows significant role of sST-2 in cardiac events, specifically in its capacity as a predictive biomarker for new-onset atrial fibrillation (NOAF) in patients with STEMI undergoing primary PCI. Clinical variables, such as CRP and sST-2, were significantly different between patients with and without NOAF, in addition to creatine kinase ($p < 0.05$) (6).

In concordance with our findings, a study found that sST-2 levels were significantly higher in STEMI patients (median sST-2 levels were 453 pg/mL, IQR 313–688 pg/mL) compared to NSTEMI (269 pg/mL, IQR 157–496 pg/mL) and stable angina patients (169 pg/mL, IQR 79–260 pg/mL), as well as controls (163 pg/mL, IQR 114–260 pg/mL) (5).

A study documented that median sST-2 concentration in control group was 9.38 ng/ml, whereas it reached 29.06 ng/ml in patients with AMI, aligning closely with outcomes observed in our study. Serum ST-2 levels exhibited a statistically significant disparity between AMI group and control group ($P < 0.001$) (4).
In terms of LVEF in current study, it was significantly lower in MI group in comparison to angina and control groups ($P=0.033$, 0.001 respectively), with no

significant difference between angina and control groups.
LVEF serves as a pivotal metric for assessing cardiac function, particularly following myocardial injury such as MI (7). In cases of MI, profound impact on cardiac muscle due to abrupt cessation of blood flow results in significant necrosis of cardiac tissue (8).

Our findings revealed a noteworthy positive correlation between ST-2 levels and various clinical and biochemical parameters, including BMI, SBP, DBP, HbA1c, serum creatinine, troponin I and LDL. There was a significant negative correlation between ST-2 and HR, Hb, total cholesterol, triglycerides and LVEF. There was an insignificant correlation between ST-2 and other parameters.

Compatibly, a study identified a positive correlation between sST-2 and serum creatinine ($r = 0.126$; $p = 0.009$) (5).
Also, our findings are in line a study reported significant correlations between sST-2 levels and key clinical markers in patients with acute myocardial infarction. In their multibiomarker analysis, sST-2 was positively associated with markers of inflammation, including CRP, and negatively correlated with LVEF and triglyceride levels, supporting notion that elevated sST-2 reflects not only myocardial stress but also broader systemic inflammation and metabolic dysregulation (6).

Regarding cardiac biomarkers, troponin I and CKMB, CK were significantly higher in angina and MI groups compared to control group.
Troponin I, CKMB, and CK are reliable indicators of cardiac events, as they are markedly higher in patients with angina and MI than in healthy individuals because they are enzymes and proteins released into bloodstream when heart muscle is damaged. In conditions such as angina and MI, where heart muscle experiences significant stress or injury, these biomarkers are released in large quantities, leading to markedly higher levels in blood compared to healthy individuals (9).

Conclusion

In conclusion, our study demonstrates that ST-2 is a valuable biomarker for diagnosis of IHD. Its integration into clinical practice could improve management and outcomes of patients with IHD.

References

1. Shi HT, Huang ZH, Xu TZ, Sun AJ, Ge JB. New diagnostic and therapeutic strategies for myocardial infarction via nanomaterials. EBioMedicine. 2022;78:103968.
2. Aydin S, Ugur K, Aydin S, Sahin İ, Yardim M. Biomarkers in acute myocardial infarction: current perspectives. Vasc Health Risk Manag. 2019;15:1-10.
3. Sastre C, Bevers MB, Kimberly WT. Role of Interleukin-1 Receptor-Like 1 (ST2) in Cerebrovascular Disease. Neurocrit Care. 2021;35:887-93.
4. Zhang K, Zhang XC, Mi YH, Liu J. Predicting value of serum soluble ST2 and interleukin-33 for risk stratification and prognosis in patients with acute myocardial infarction. Chin Med J (Engl). 2013;126:3628-31.
5. Demyanets S, Speidl WS, Tentzeris I, Jarai R, Katsaros KM, Farhan S, et al. Soluble ST2 and interleukin-33 levels in coronary artery disease: relation to disease activity and adverse outcome. PLoS One. 2014;9:e95055.
6. Zhao TT, Pan TJ, Yang YB, Pei XY, Wang Y. Association of soluble suppression of tumorigenicity 2 protein with new-onset atrial fibrillation in patients with acute ST-segment elevation myocardial infarction undergoing

primary PCI. Front Cardiovasc Med. 2023;10:1207219.
7. Potter E, Marwick TH. Assessment of Left Ventricular Function by Echocardiography: The Case for Routinely Adding Global Longitudinal Strain to Ejection Fraction. JACC: Cardiovascular Imaging. 2018;11:260-74.
8. Miller AL, Dib C, Li L, Chen AY, Amsterdam E, Funk M, et al. Left ventricular ejection fraction assessment among patients with acute myocardial infarction and its association with hospital quality of care and evidence-based therapy use. Circ Cardiovasc Qual Outcomes. 2012;5:662-71.
9. Kim SY, Lee JP, Shin WR, Oh IH, Ahn JY, Kim YH. Cardiac biomarkers and detection methods for myocardial infarction. Mol Cell Toxicol. 2022;18:443-55.

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