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Serum sST2: key biomarkers in COVID-19 patients with implications for coronary artery disease

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Abstract

Background As the coronavirus disease-2019 (COVID-19) pandemic persists, post-COVID-19 syndrome (PS), characterized by symptoms like chest pain, fatigue, and palpitations, is becoming a significant medical and social issue. COVID-19 patients with existing coronary artery disease (CAD) may face higher risks of complications. It is crucial to assess if PS patients also have CAD, though data is limited.

Methods We studied 75 COVID-19 patients and 68 non-COVID-19 patients admitted to our hospital between 2022/12/20 to 2023/01/20. Demographic, laboratory, and clinical data were collected upon admission. The Gensini score (GS) was used to assess coronary atherosclerosis severity. Patients were categorized by GS and clinical traits to identify potential independent risks linked to CAD and COVID-19 severity.

Results COVID-19 patients with existing CAD had higher levels of serum soluble growth stimulation expression of gene 2 protein (sST2), myeloperoxidase, ALT, AST, PT, B-type natriuretic peptide (BNP), and hypersensitive troponin-I (hs-cTnI), along with longer hospital stays, more ICU admissions, and increased heart failure and ACS morbidity compared to those without CAD. Univariate and multivariate analysis identified sST2 as an independent risk factor for COVID-19 patients with coexisting CAD (odds ratio 1.122). sST2 levels were positively correlated with coronary angiography GS ($r = 0.474$, $p < 0.001$) in COVID-19 patients and were significantly higher in cases with GS ≥ 32, regardless of COVID-19 status ($p < 0.001$) and specifically in COVID-19 patients ($p = 0.006$). ROC analysis showed sST2 predicted ICU admission, hospital stay duration, and morbidity of HF and ACS similarly to GS.

Conclusions Admission serum sST2 levels should be considered in COVID-19 patients with CAD-like symptoms for treatment planning and could serve as a prognostic biomarker for COVID-19 with co-existing CAD in clinical practice.

Keywords COVID-19, sST2 level, Gensini score, Coronary artery disease

Introduction

The COVID-19 pandemic has occurred in waves globally, posing severe risks to patients, particularly those with conditions like hypertension, chronic obstructive pulmonary disease, cardiovascular diseases (CVD), and cancer. These patients face heightened risks of lung infections and life-threatening situations [1]. The infection triggers increased inflammatory cytokine release, which can destabilize atherosclerotic plaques [2]. Beyond early complications, long-term symptoms significantly impact patients' quality of life. Long COVID, now widely recognized, includes symptoms such as fatigue, breathlessness, and chest pain lasting over three months post-infection,

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affecting survivors of all ages and severities, especially those with coronary vascular disease [3–5].

Research indicates that COVID-19 patients face a high risk of adverse cardiovascular events, with coronary artery disease (CAD) accounting for about 15% of these cases [6]. Those with cardiovascular conditions are prone to heart injury when infected, leading to worse outcomes and higher mortality, even in patients without prior CAD diagnosis [7, 8]. PS can resemble early symptoms of CAD, making it crucial to quickly identify whether they stem from CAD or long COVID to avoid treatment delays. Timely identification of CAD complications and cardiovascular events in COVID-19 patients is essential for effective secondary prevention.

sST2, a member of the interleukin-11 family and a decoy receptor for IL33, plays a crucial role in cardiac remodeling and inflammation [9]. It is strongly linked to heart failure severity and poor outcomes, with its levels unaffected by age, kidney function, or body mass index [10, 11]. sST2 also correlates with complex coronary lesion morphology and predicts CAD severity [12]. In COVID-19 patients, elevated sST2 levels indicate disease progression and poor prognosis, independent of other conditions [13–16], outperforming C-reactive protein in diagnosing inflammation and the 4C mortality score in predicting ICU admission and ventilator use [13, 17].

Many studies highlight the diagnostic and prognostic importance of serum sST2 levels in COVID-19 patients, but few explore its link to coronary artery stenosis in those with co-existing CAD. The Gensini score (GS) was used to assesses coronary artery narrowing severity [18, 19]. A retrospective study showed a positive correlation between serum sST2 and GS, predicting major cardiovascular events in myocardial infarction and chronic coronary syndrome patients [12, 20]. However, the relationship between GS and sST2 in COVID-19 patients remains unclear. This study aims to investigate their significance and association in COVID-19 patients with or without CAD, to aid early CAD identification in long COVID or PS patients.

Patients and methods

Patients

This retrospective cohort study was conducted at our hospital from 2022/12/20 to 2023/01/20. A total of 75 COVID-19 hospitalized patients diagnosed according to WHO guidance and 68 non-COVID-19 inpatients (35 with CAD and 33 without CAD) were screened. Inclusion criteria were untreated first-wave COVID-19 patients with complete coronary angiography data. Exclusion criteria included incomplete data, treatment for COVID-19 before admission, lack of angiography, severe organ issues, certain diseases, malignancies, pregnancy, and end-stage disease. The research flowchart is shown in Fig. 1. Demographic, laboratory, and clinical data were collected from the Hospital Information System using a standardized form. COVID-19 patients were categorized into CAD (n=50) and non-CAD (n=25) groups based on angiography. Clinical outcomes assessed included ICU admission, hospital stay length, and complications like heart failure (HF) and acute coronary syndrome (ACS). The study was approved by the Shanxi Bethune Hospital Ethics Committee (YXLL-2024-079).

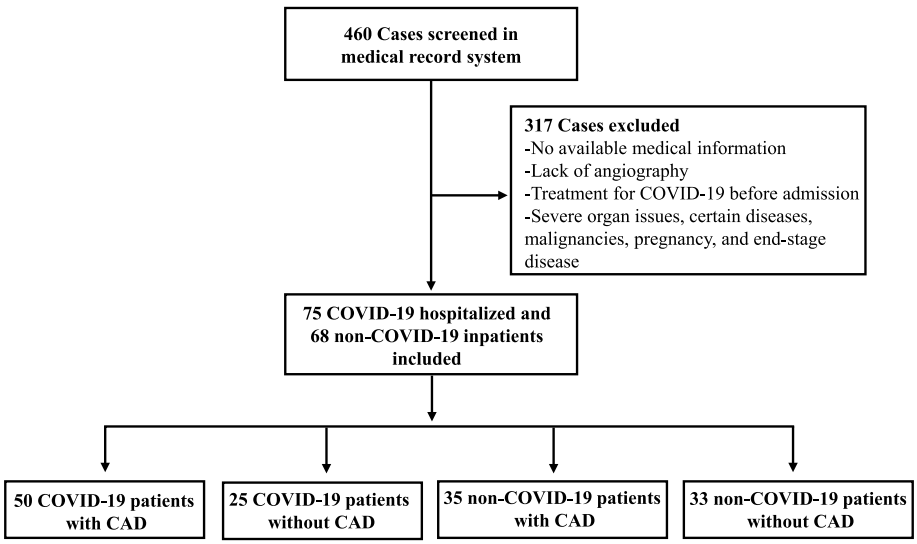

Fig. 1 Flowchart showing the patients included in the study

Laboratory procedures

Upon admission, we collected initial lab data for all patients, including blood tests (white blood cell, neutrophil counts, hemoglobin, platelet counts), coagulation profile (prothrombin time, activated partial prothrombin time, D2-dimer, fibrinogen), and serum biochemical tests (including renal and liver function), lipid levels, homocysteine, myeloperoxidase, and sST2. The severity of coronary stenosis was evaluated using the Gensini score [21], with a score of 32 or higher indicating severe coronary lesions.

Inspection methods and criteria

Coronary angiography

The patient's right arm was positioned at 45 degrees, and a disinfected catheter was inserted into the radial artery, then advanced into the coronary artery. A contrast agent was injected to assess the coronary vessels. Coronary heart disease is identified by a 50% narrowing in any blood vessel.

Statistical analysis

Statistical analyses were performed with SPSS 27.0 (SPSS Inc., Chicago, Illinois, USA). Continuous variables were reported as mean ± SD or median (range), while categorical variables were shown as percentages. Differences between groups were assessed using the Chi-square test for categorical variables, the t-test for normally distributed continuous variables, and the Wilcoxon test for non-normally distributed continuous variables. Univariate and multivariate logistic regression analysis were conducted to identify independent risk factors for COVID-19 patients with coexisting CAD and the correlations between parameters were evaluated by Spearman correlation analysis. The receiver operating characteristic (ROC) curve analysis assessed the effectiveness of these factors for the clinical prognosis of COVID-19 patients. A p-value of less than 0.05 indicated statistical significance.

Results

Comparison of clinical and laboratory characteristics in COVID-19 patients with/without coronary artery disease

Among the 75 eligible COVID-19 patients, 50 presented with concomitant CAD upon admission. The clinical and demographic characteristics of patients with and without CAD are detailed in Table 1. The median age of all recruited patients was 60 years (range 28–84), with a male predominance of 61.30%. Most patients exhibited comorbidities, with hypertension being the most prevalent (42 cases), followed by diabetes (25 cases). Comparative analysis revealed that age, sex, and basic vital signs were comparable between the two groups. However, elevated serum levels of ALT, AST, PT, myeloperoxidase, lymphocyte counts, and the prevalence of diabetes mellitus were associated with CAD complications in COVID-19 patients. Myocardial injury markers in patients with both COVID-19 and CAD indicated impaired cardiac function, with a median BNP of 42 pg/ml, median hs-cTnI of 9.7 pg/ml, and median sST2 of 24.05 ng/ml. Furthermore, the prognosis for patients with both COVID-19 and CAD was relatively poor, as evidenced by longer hospital stays, higher ICU occupancy rates, and increased morbidity from HF and acute coronary syndrome ACS.

Analysis of independent risk factors for co-existing CAD in COVID-19 patients

Multivariate logistic regression analysis was conducted using variables that demonstrated significant results in univariate analysis (refer to Table 2). The model exhibited a good fit, as indicated by the Hosmer–Lemeshow test ($\chi 2 = 5.318$, $P = 0.621$). Serum sST2 was identified as an independent risk factor for co-existing CAD in COVID-19 patients, with an odds ratio (OR) of 1.122 and a 95% confidence interval (CI) of 1.007–1.250 ($P = 0.036$). To further elucidate the pivotal role of sST2 in predicting COVID-19 in conjunction with CAD, we stratified all recruited patients (regardless of COVID-19 RT-PCR results) into four distinct groups based on the presence or absence of COVID-19 infection and CAD. We then conducted a comparative analysis of sST2 levels across these groups (Fig. 2). Our findings indicated that among patients without CAD, serum sST2 levels showed no significant variation regardless of COVID-19 infection status ($p = 0.987$). In contrast, among patients with CAD, serum sST2 levels were significantly elevated irrespective of COVID-19 infection, with a notably higher increase observed in patients with both COVID-19 and CAD compared to those with only COVID-19 ($p = 0.043$).

Correlation of sST2 and Gensini score

We further assessed the correlation between serum sST2 levels and GS in COVID-19 patients, finding a significant positive correlation ($r = 0.474$, $p < 0.001$). This means higher GS is associated with higher sST2 levels (Fig. 3A). In 68 non-COVID-19 patients, a positive correlation still exists but is weaker ($r = 0.283$, $p = 0.020$) (Fig. 3B). All recruited patients (regardless of COVID-19 RT-PCR results) were categorized into high (≥ 32 points) and low (< 32 points) GS groups. The high GS group showed significantly higher median sST2 levels compared to the low GS group (23.40 vs 14.85 ng/ml, $p < 0.001$). When non-COVID-19 patients were excluded, the results remained consistent (24.20 ng/ml vs. 17.80 ng/ml, $p = 0.006$), with sample sizes of 24 and 51, respectively (Fig. 3C). A similar

Table 1 Demographic and clinical characteristics of the patients with/without CAD admitted for COVID-19

Characteristic	Total ($n=75$)	CAD (+) ($n=50$)	CAD (-) ($n=25$)	*p* value
Age (years)	60 (28–84)	62 (35–84)	59 (28–74)	0.251
Male, n (%)	46 (61.30%)	34 (68.00%)	12 (48.0%)	0.094
SBP (mmHg)	126 (90–167)	126 (90–165)	126 (98–167)	0.942
DBP (mmHg)	79 (51–106)	79 (51–106)	82 (61–103)	0.249
Respiratory rate (breaths/min)	20 (15–22)	20 (15–22)	20 (16–20)	0.927
Comorbidity				
Hypertension, n (%)	42 (56.00%)	29 (58.00%)	13 (52.00%)	0.622
Diabetes, n (%)	25 (33.30%)	21 (42.00%)	4 (16.00%)	0.024
CKD, n (%)	5 (6.70%)	4 (8.00%)	1 (4.00%)	0.870
Laboratory findings on admission hospital				
AST (IU/L)	24.4 (10.90–240.40)	31.9 (14.90–240.40)	19.5 (10.90–68.10)	0.001
ALT (IU/L)	27 (6.10–217.00)	29.45 (9.80–217.00)	19.2 (6.10–51.00)	0.020
Albumin (g/L)	39.7 (26.50–312.00)	39.55 (26.50–312.00)	39.9 (35.90–44.60)	0.123
Urea (mmol/L)	5.8 (2.30–40.70)	5.85 (2.70–40.70)	5.3 (2.30–11.90)	0.465
Creatinine (μmol/L/)	88.1 (57.60–758.40)	93.75 (67.80–758.40)	84.8 (57.60–161.90)	0.227
Uric acid (μmol/L/)	320.1 (162.50–562.60)	323.36 (163.50–562.60)	311.4 (162.50–496.60)	0.331
Triglyceride (mmol/L)	1.44 (0.49–12.62)	1.34 (0.49–7.06)	1.54 (0.98–12.62)	0.104
LDL (mmol/L)	2.51 (1.18–4.51)	2.3 (1.21–4.51)	2.6 (1.18–4.51)	0.152
TC (mmol/L)	3.88 (2.06–6.84)	3.77 (2.06–6.84)	4.07 (2.65–6.44)	0.106
Homocysteine (μmol/L/)	17.8 (6.70–52.10)	17.8 (6.70–52.10)	15.4 (9.00–31.20)	0.451
WBC ($\times 10^9$/L)	6.4 (2.70–81.00)	6.5 (3.00–28.30)	5.4 (2.70–81.00)	0.236
Lymphocyte count ($\times 10^9$/L)	1.51 (0.58–3.02)	1.38 (0.58–2.86)	1.59 (1.07–3.02)	0.017
Neutrophil count ($\times 10^9$/L)	4.06 (1.22–27.05)	4.21 (1.43–27.05)	3.23 (1.22–8.12)	0.045
Hemoglobin (g/dl)	132 (14.00–163.00)	132 (53.00–157.00)	135 (14.00–163.00)	0.396
Platelet ($\times 10^{12}$/L)	230 (94.00–502.00)	236.25 (114.00–502.0)	216 (94.00–351.00)	0.169
sST2 (ng/ml)	19.9 (3.40–200.00)	24.05 (6.70–200.00)	13.4 (3.40–37.10)	<0.001
Myeloperoxidase (ng/ml)	94.1 (16.40–1117.50)	107.85 (16.40–1117.50)	68.6 (26.00–238.00)	0.007

Table 1 (continued)

Characteristic	Total ($n=75$)	CAD (+) ($n=50$)	CAD (-) ($n=25$)	*p* value
PT (s)	11.3 (10.20–38.10)	11.5 (10.50–38.10)	11.1 (10.20–15.30)	0.005
Fibrinogen (g/L)	3.26 (1.73–6.74)	3.3 (1.73–6.74)	3.02 (2.45–4.45)	0.282
APTT (s)	31.2 (23.80–400.00)	31.45 (23.80–400)	31.2 (26.70–59.10)	0.657
D2-dimer (ng/ml)	123 (33.00–62895.00)	130 (33.00–62895.00)	114 (40.00–1104.60)	0.496
BNP (pg/ml)	33 (5.00–1018.00)	42 (7.00–1018.00)	30 (5.00–244.00)	0.020
Hypersensitive troponin-I (pg/ml)	7.4 (0.30–15545.90)	9.7 (1.90–15545.90)	4.1 (0.30–32.90)	0.002
Clinical outcome				
ICU, n (%)	23 (30.70%)	22 (44.00%)	1 (4.00%)	< 0.001
HF, n (%)	16 (21.30%)	15 (30.00%)	1 (4.00%)	0.010
Hospital length of stay, days	5 (1–20)	6 (1–20)	4 (1–13)	0.028
ACS, n (%)	21 (28.00%)	21 (42.00%)	0 (0.00%)	< 0.001

$p<0.05$ indicates a statistically significant difference between two groups

Abbreviations: *CAD* coronary artery disease, *SBP* systolic blood pressure, *DBP* diastolic blood pressure; *CKD* chronic kidney disease, *AST* aspartate aminotransferase, *ALT* alanine aminotransferase, *LDL* low-density lipoprotein, *TC* total cholesterol, *WBC* white blood cell, *sST2* soluble growth stimulation expression of gene 2 protein, *PT* prothrombin time, *APTT* activated partial thromboplastin time, *BNP* B-type natriuretic peptide, *ICU* intensive care unit, *HF* heart failure, *ACS* acute coronary syndrome;

disparity in serum sST2 levels was observed among 68 non-COVID-19 patients between two groups (Fig. 3D).

Diagnostic value of sST2 and Gensini score for clinical outcomes in COVID-19 patients

Table 3 presents the sST2 and GS values in relation to clinical outcomes among all COVID-19 patients studied. Significant differences were observed in serum sST2 levels and GS across all clinical outcomes, with both metrics demonstrating a correlation with the length of hospital stay (all $p<0.05$). In the ROC curve analysis, the serum sST2 level was found to predict morbidity in HF and ACS comparably to GS, while it significantly outperformed GS in predicting ICU admission (Fig. 4). The area under the curve (AUC) for sST2 and GS in predicting ICU admission were 0.872 and 0.768, respectively; for HF morbidity, the AUCs were 0.727 and 0.792; and for ACS complications, they were 0.802 and 0.849.

Discussion

In this study, we selected patients experiencing chest tightness, shortness of breath, and other cardiovascular-related symptoms during the COVID-19 pandemic for cardiovascular disease screening and subsequent analysis. Our initial findings indicated that among patients without CAD, serum sST2 levels did not differ significantly, irrespective of COVID-19 infection status. Conversely, in patients with CAD, serum sST2 levels were significantly elevated regardless of COVID-19 infection, with a more pronounced increase observed in those with concurrent COVID-19 and CAD ($p=0.043$). Multivariate logistic regression analysis further identified elevated serum sST2 as an independent risk factor in COVID-19 patients with co-existing CAD.

There are two ST2 subtypes: soluble ST2 (sST2) and trans-membrane ST2 (ST2L). The IL-33/ST2L pathway offers cardiovascular protection by delaying atherosclerosis, preventing myocardial fibrosis, and reducing myocardial cell death. sST2 acts as a "decoy receptor," blocking IL-33/ST2L actions [22–24]. Recent studies link increased sST2 in COVID-19 patients to negative outcomes like ICU admission, ECMO use, HF, organ failure and 30-day death, possibly due to complications with heart and respiratory failure [13]. Patients with both CVD and COVID-19 face severe symptoms and higher mortality risk, likely because COVID-19 damages cardiomyocytes and causes viral myocarditis [25]. Our research indicates that sST2 levels are similar in patients with only COVID-19 and those with neither CAD nor COVID-19, but significantly higher in patients with both conditions. This suggests that the rise in sST2 levels in COVID-19 patients is linked to CAD complications. The underlying

Table 2 Univariate and multivariate logistic regression analysis for independent risk factors of serum level of sST2 in COVID-19 patients with or without CAD

Characteristic	Univariate analysis Odds ratio (95% CI)	*p* value	Multivariate analysis Odds ratio (95% CI)	*p* value
Diabetes, n (%)	3.802 (1.136–12.721)	0.030	0.237 (0.029–1.963)	0.182
AST (IU/L)	1.043 (1.006–1.082)	0.022	0.981 (0.930–1.035)	0.490
ALT (IU/L)	1.043 (1.002–1.085)	0.039	1.059 (0.987–1.135)	0.110
Lymphocyte count ($\times 10^9$/L)	0.389 (0.164–0.924)	0.032	0.489 (0.119–2.013)	0.322
sST2 (ng/ml)	1.132 (1.048–1.223)	0.002	1.122 (1.007–1.250)	0.036
Myeloperoxidase (ng/ml)	1.009 (1.001–1.017)	0.023	1.005 (0.997–1.013)	0.198
PT (s)	1.842 (1.018–3.332)	0.043	1.233 (0.746–2.037)	0.414
BNP (pg/ml)	1.010 (1.001–1.019)	0.029	1.004 (0.993–1.015)	0.493
ICU, n (%)	18.857 (2.364–150.43)	0.006	2.710 (0.132–55.575)	0.518
HF, n (%)	10.286 (1.272–83.147)	0.029	2.399 (0.092–62.262)	0.598
Hospital length of stay, days	1.200 (1.009–1.426)	0.039	1.104 (0.867–1.406)	0.422

P < 0.05 indicates a statistically significant differences between the groups

Abbreviations: *AST* aspartate aminotransferase, *ALT* alanine aminotransferase, *sST2* soluble growth stimulation expression of gene 2 protein, *PT* prothrombin time, *BNP* B-type natriuretic peptide, *ICU* intensive care unit, *HF* heart failure

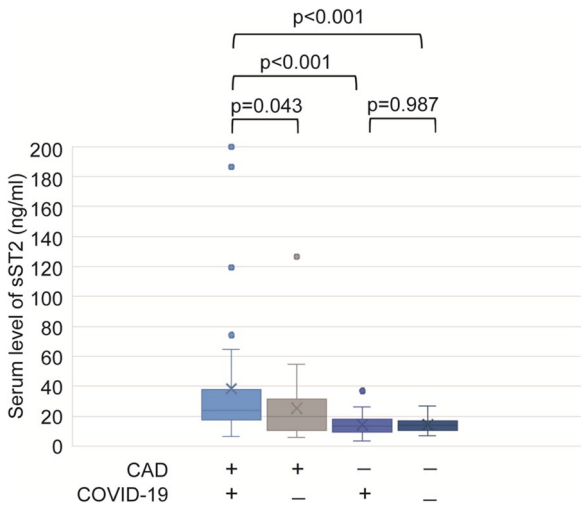

Fig. 2 Comparison of serum sST2 levels in four groups based on CAD and COVID-19 status. $p<0.05$ indicates a statistically significant difference between two groups. Abbreviations: CAD, coronary artery disease; COVID-19, coronavirus disease-2019; sST2, soluble growth stimulation expression of gene 2 protein

mechanism may involve viral invasion of the heart muscle, causing immune inflammation and cardiac pressure changes, ultimately leading to poor outcomes in COVID-19 patients. Additionally, COVID-19 patients with CAD show higher sST2 levels than those without, indicating that COVID-19 may exacerbate heart damage through myocardial ischemia or direct injury, possibly due to hypercoagulation and inflammation during the COVID-19 infection [26]. Early research suggested that elevated serum D2-dimer and cardiac troponin levels are linked to higher in-hospital mortality in COVID-19 patients, recommending anticoagulants for those with high D2-dimer levels [27–29]. Therefore, combining sST2 with troponin, D2-dimer, and other indicators may effectively predict early heart involvement and prognosis in COVID-19 patients.

This study found that serum sST2 levels were positively correlated with CAD severity, as diagnosed by coronary angiography and assessed using GS. Higher sST2 levels were observed in patients with severe coronary artery lesions, aligning with previous research [9, 12, 30, 31]. This indicates that sST2 could serve as an early screening

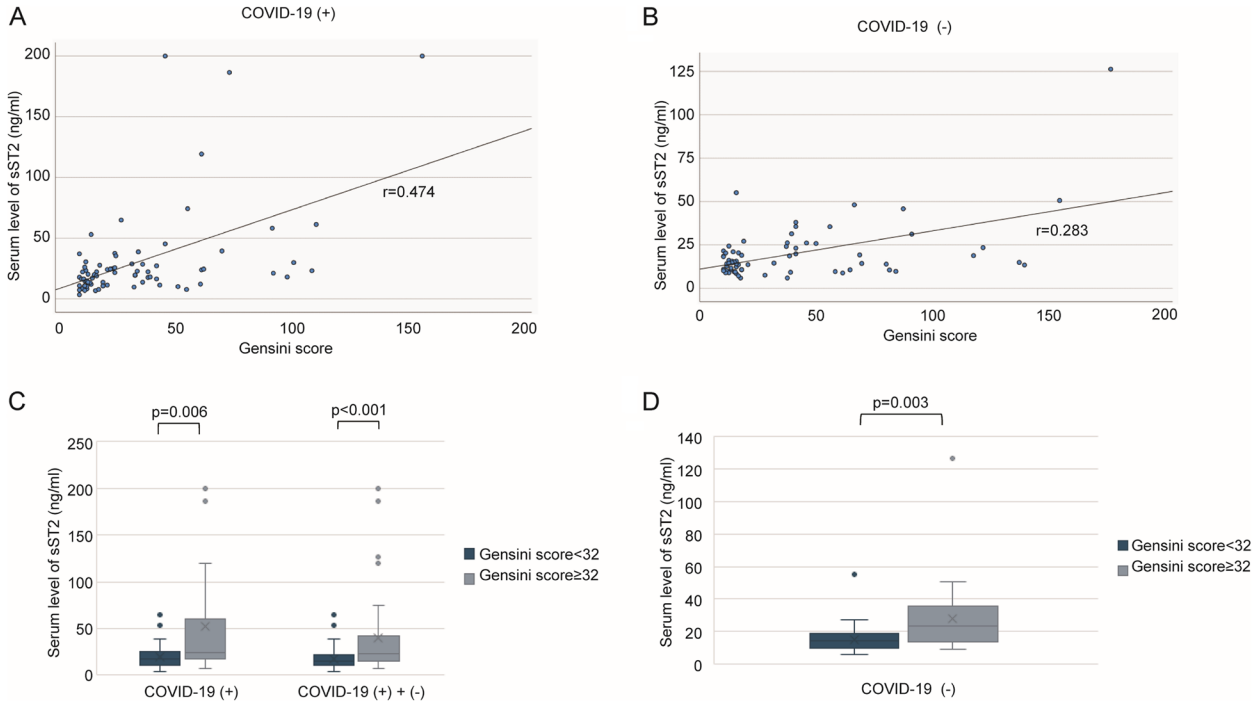

Fig. 3 **A** Spearman correlation analysis illustrated that GS obtained by coronary angiography was significantly positively correlated with serum level of sST2. (Spearman r: 0.474, *P* <0.001). **B** and in non-COVID-19 patients (Spearman r: 0.283, *p*=0.020). **C** Box diagram demonstrates the significant increase in sST2 expression in cases with GS ≥32: In all recruited patients regardless of COVID-19 RT-PCR results (*p*<0.001); In COVID-19 patients (*p*=0.006). **D** and in in non-COVID-19 patients (*p*=0.003). *P* < 0.05 indicates a statistically significant difference between two groups. Abbreviations: GS, gensini score; sST2, soluble growth stimulation expression of gene 2 protein, COVID-19, coronavirus disease-2019

Table 3 Diagnostic value of sST2 and Gensini score in prognosis of clinical outcomes in COVID-19

Variables	ICU Admission			HF			ACS			Hospital length of stay
	Yes	No	p	Yes	No	p	Yes	No	p	p
	n = 23	*n* = 52		*n* = 16	*n* = 59		*n* = 21	*n* = 54		
Gensini score	23 (0–160)	8.5 (0–108.5)	< 0.001	34.5 (2.5–110.5)	9 (0–160)	< 0.001	50 (3.0–160)	8 (0–108.5)	< 0.001	0.015
sST2	35.4 (7.7–200)	16.4 (3.4–64.9)	< 0.001	27.85 (13.7–119.3)	17.8 (3.4–200)	0.006	29.9 (7.7–200)	16.75 (3.4–64.9)	< 0.001	0.028

Abbreviations: *ICU* intensive care unit, *HF* heart failure, *ACS* acute coronary syndrome, *sST2* soluble growth stimulation expression of gene 2 protein

and severity assessment marker for CAD in high-risk individuals. Similar findings were reported in studies on diabetes and hypertension, where elevated sST2 levels were linked to a higher likelihood of CAD and worse prognosis [32, 33].

Using a GS score of 32 as a threshold, we found that sST2 levels were significantly higher in CAD patients with GS ≥ 32, regardless of COVID-19 infection. Zhang et al. [34] reported that sST2 levels were elevated in ACS patients with complex lesions, indicating its potential as a biomarker for coronary plaque stability. Luo et al. [35] showed that higher sST2 levels were linked to plaque vulnerability in non-ST-elevation ACS patients. Takahashi et al. [25] found that increased sST2 contributes to coronary atherosclerosis in hypertensive patients. These studies suggest that sST2 is a crucial early indicator of CAD severity. Thus, in COVID-19 patients with chest discomfort and elevated sST2, the risk of coronary artery lesions is significantly higher compared to those with normal sST2 levels.

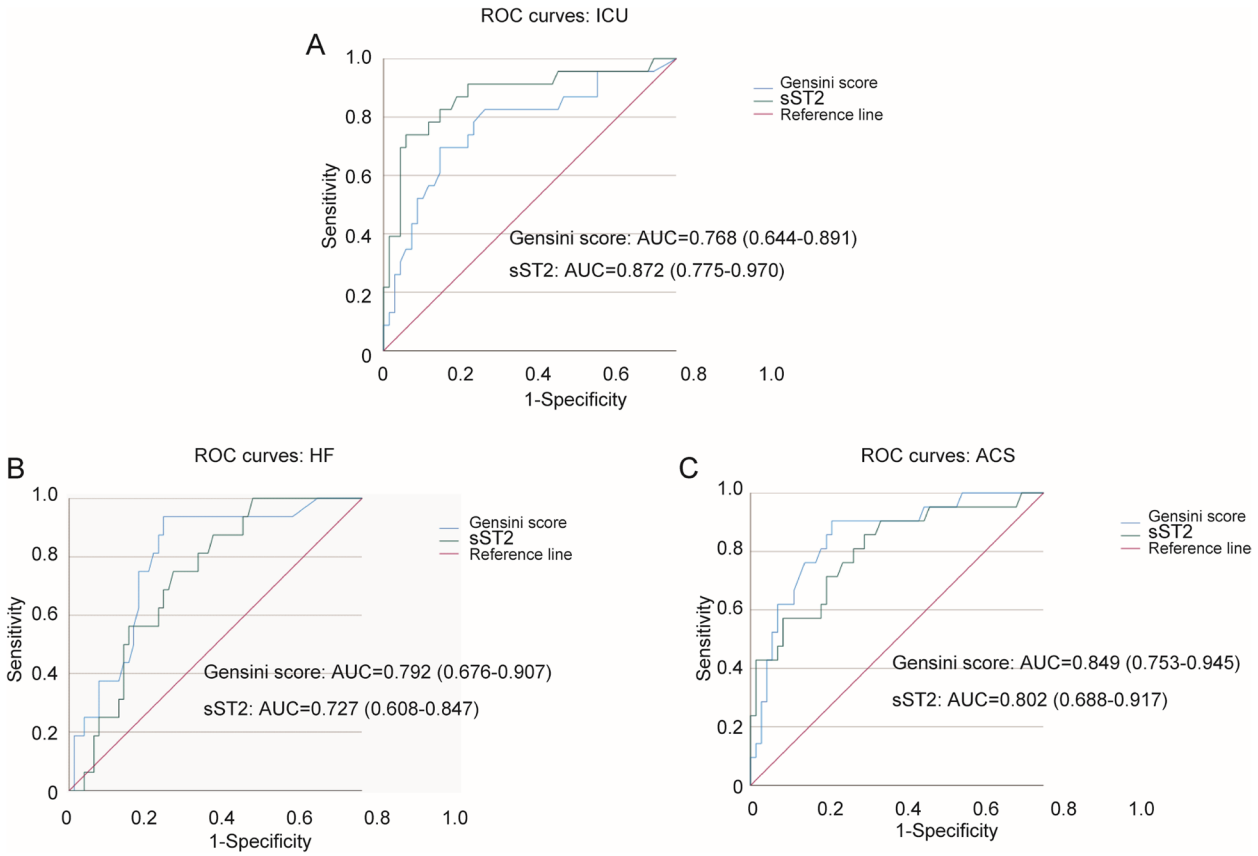

Fig. 4 Receiver operating characteristic curve analysis of sST2 and Gensini score for clinical outcomes. **A** ICU admission. **B** Morbidity of HF. **C** Incidence of ACS. Abbreviations: AUC—area under the curve; HF, heart failure; ACS, acute coronary syndrome; ICU, intensive care unit; sST2, soluble growth stimulation expression of gene 2 protein

This study found that higher GS and sST2 levels in COVID-19 patients were linked to longer ICU stays and increased rates of HF and ACS. Previous research suggests elevated sST2 levels indicate poor prognosis in COVID-19, but the cause is unclear [13]. We categorized patients based on the presence of CAD and observed significantly higher sST2 levels in those with both COVID-19 and CAD, correlating with poorer outcomes. We propose that elevated sST2 in COVID-19 patients may indicate early cardiac involvement, whether due to pre-existing CAD or new viral-induced myocardial damage. Thus, sST2 screening could help identify heart complications or myocarditis in COVID-19 patients with chest discomfort.

This study is subject to several limitations. Firstly, due to the limited number of patients who underwent coronary angiography during hospitalization for COVID-19, caution is warranted when interpreting the associated findings. Secondly, the majority of COVID-19 patients in our study presented with multiple comorbidities, and the average age was 60 years (range 28–84), which may introduce bias and limit the generalizability of our data. Thirdly, the variability in the time between admission and disease onset precluded the collection of laboratory data at consistent intervals for all patients, and the heterogeneous progression of COVID-19 may have influenced our results.

In short, COVID-19, a viral infection, affects the respiratory system and can impact multiple organs, including the heart. It persists in a long-term coexistence with humans, causing symptoms like chest tightness and palpitations that resemble CVD. This study indicates that the serological marker sST2 effectively predicts whether COVID-19 patients have existing or new CAD and correlates with disease severity, offering good prognostic value. Using sST2, BNP, and hs-cTnI together could enhance the accuracy of assessing cardiovascular complications in COVID-19 patients.

Conclusions

In conclusion, it is important to consider serum levels of sST2 in COVID-19 patients presenting with suspected CAD symptoms. Additionally, the peripheral blood levels of this biomarker following discharge and during subsequent follow-up may influence the prognosis of COVID-19. Therefore, further research in this area is warranted.

Abbreviations

CAD	Coronary artery disease
CVD	Cardiovascular diseases
CKD	Chronic kidney disease
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
AST	Aspartate aminotransferase
ALT	Alanine aminotransferase
LDL	Low-density lipoprotein
TC	Total cholesterol
WBC	White blood cell
sST2	Soluble growth stimulation expression of gene 2 protein
PT	Prothrombin time
APTT	Activated partial prothrombin time
BNP	B-type natriuretic peptide
ICU	Intensive care unit
HF	Heart failure
ACS	Acute coronary syndrome
GS	Gensini score
hs-cTnI	Hypersensitive troponin-I
PS	Post-COVID-19 syndrome

Authors' contributions

Project administration and supervision, Jinfang Cheng; patient recruitment and writing original draft preparation, Xueqin Li; data analysis, Yaxin Tian and Hongyan Cao.

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Data availability

All the data supporting our findings are contained within this manuscript. Datasets used in this study may be requested from the first author: Xueqin Li (email: lixueqin93@163.com).

Declarations

Ethics approval and consent to participate

The protocol of this study complied with the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Shanxi Bethune Hospital (YXLL-2024–079). The informed consent to participate was obtained from all of the participants in the study.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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