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**ORIGINAL ARTICLES**

# Clinical significance of elevated serum cardiac troponin T and associated risk factors in patients diagnosed with acute ischemic stroke

Qin Guo *MM*, Xuhui Song *MB*, Zejiang Liu *MB*, Qiyun Long *MB*, Sheng Lin *MB*, Xing Qi *MB*, Huaguo Wang *MB*

*Department of Laboratory Medicine, Ziyang Central Hospital, Ziyang, China*

## *Abstract*

*Objective:* We explored the clinical significance and associated risk factors of increased levels of serum cardiac troponin T (cTnT) in individuals suffering from acute ischemic stroke (AIS). *Methods:* Our study subjects consisted of patients who were admitted with AIS within 48 hours of its onset. These study participants were categorized based on their levels of cTnT into two groups: normal cTnT group and elevated cTnT group. We collected and subjected a range of data to statistical analysis, including general clinical traits, medical history, laboratory test results, electrocardiograms, imaging scans, and medical records like the National Institute of Health Stroke Scale (NIHSS) scores of the patients. *Results:* Out of the 232 patients diagnosed with AIS, 84 individuals (36.21%) exhibited raised levels of cTnT. When comparing this elevated cTnT cohort to the group with regular cTnT levels, those with elevated cTnT were older [with a median age of 76 year (interquartile range: 67 to 83) as compared to 70 year interquartile range: 61 to 79), $P = 0.002$], and they presented with higher NIHSS scores upon admission [8.5 (interquartile range: 4 to 14) compared to 5 (interquartile range: 2 to 9), p = 0.002]. In addition, a larger percentage of patients in the elevated cTnT group had both coronary artery disease (23.81% vs. 7.43%, $P < 0.001$) and cardiac insufficiency (25.00% vs. 3.38%, $P < 0.001$) as comorbidities. Meanwhile, the elevated cTnT group also displayed a higher occurrence of electrocardiogram abnormalities, including bundle branch block (29.76% vs. 9.46%, $P < 0.001$) and atrial fibrillation (32.14% vs. 11.49%, $P < 0.001$), in comparison to the normal cTnT group. The clinical data and related laboratory indicators of patients were collected for risk factor analysis, which showed that bundle branch block [odds ratio (OR) = 4.17, 95% confidence interval (95%CI) = 1.43–12.16), log to base 10 N-terminal pro-brain natriuretic peptide ($Log_{10}$NT-proBNP; OR = 3.41, 95%CI = 1.62-7.16), cystatin C (OR = 6.86, 95%CI = 2.01–23.43), and neutrophil/lymphocyte ratio (OR = 1.13, 95%CI = 1.02–1.25) were independent risk factors for elevated cTnT in patients with AIS. *Conclusions:* Older patients with AIS who had higher levels of cTnT exhibited more severe neurological impairments and a greater number of comorbidities. Furthermore, an elevated cTnT in patients with AIS may be linked to cardiac insufficiency, changes in kidney function, and signs of inflammation.

*Keywords:* Acute ischemic stroke, cardiac troponin T, electrocardiogram abnormalities, N-terminal B-type natriuretic peptide precursor, risk factors

## INTRODUCTION

Cardiac troponin (cTn) serves as a controlling protein that oversees the contraction of cardiomyocytes. It is comprised of three components: cTnT, cardiac troponin I (cTnI), and cardiac troponin C (cTnC).[1] In cases of acute myocardial infarction, the levels of cTn in the body substantially rise. This elevation is considered the preferred biomarker for discerning, gauging risk, managing, and forecasting outcomes related to acute coronary syndromes.[2,3] However, a growing body of evidences indicating that heightened troponin levels are not exclusive to acute myocardial infarction, but are also observable in

*Address correspondence to:* Huaguo Wang, Department of Laboratory Medicine, Ziyang Central Hospital, No. 66 of Rende West Road, Yanjiang District, Ziyang 641300 China. Tel: +86 028-26276519, Email: wagnhuag@outlook.com

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various other heart-related conditions. Increased troponin levels have been noted in noncardiac ailments like renal failure, sepsis, pulmonary embolism, pulmonary arterial hypertension, and stroke.[4,5]

An acute ischemic stroke (AIS) is characterized by the sudden emergence of neurological impairments due to restricted blood supply in a specific area of the brain, along with observable signs of immediate tissue damage.[6] Cardioembolic infarction is a severe subtype of stroke with a high risk of early death.[7] Prior studies demonstrated that 20%-60% of acute stroke patients have elevated cTn levels[8,9], and elevated cTnT within 6 h after the onset of AIS was a predictor of poor neurological prognosis.[10] Though the *Guidelines for the Early Management of Patients With Acute Ischemic Stroke* recommend baseline troponin determination for all AIS patients[11], the cause and clinical significance of elevated cTn in AIS patients are not completely clear.[12] Hence, the utilization of the cTnT test and the evaluation of factors that contribute to increased cTnT levels may aid in comprehending the situation, recognizing potential hazards, and foreseeing the outlook for patients with AIS. Considering this, in the present study, we examined the clinical implications and associated factors contributing to elevated cTn levels in patients with AIS. This may help to understand the mechanism of elevated cTn or guide clinicians to pay necessary attention to specific populations.

## METHODS

We included 232 patients with AIS who were admitted to the Neurology Department of Ziyang Central Hospital City during the period from January 2021 to December 2021. The patients were divided into two groups: one group with elevated cTnT levels (84 cases) and the other with normal cTnT levels (148 cases), based on whether their cTnT levels exceeded 14 ng/L. All these patients met the diagnostic criteria outlined in the *2018 Chinese Guidelines for the Diagnosis and Treatment of Acute Ischemic Stroke*.[13]

### *Collection of medical history*

We collected basic details like age, gender, blood pressure, and heart rate, as well as medical background such as prior instances of stroke, hypertension, diabetes, coronary artery disease, atrial fibrillation, myocardial infarction, and heart failure. Additionally, we conducted supplementary tests post admission, including head magnetic resonance imaging, computed tomography, and electrocardiogram (ECG) for all patients.

### *Measurement of laboratory indicators*

After admission, blood samples were collected from the peripheral veins of patients to assess laboratory markers including blood cell counts (analyzed by the Sysmex XN-1000 hematology analyzer), coagulation function (evaluated by Werfen's ACL700 coagulation analyzer), liver and kidney function (measured with the Siemens ADVIA2400 chemistry analyzer), N-terminal pro-brain natriuretic peptide (NT-proBNP) and cTnT (analyzed by Roche's E601 electrochemiluminescence immunoassay analyzer).

### *Statistical analysis*

The SPSS25.0 and R software were used to statistically analyze the data. Measurement data following normal distribution are expressed as mean ± standard deviation ($\overline{x} \pm s$) and compared between groups with the two independent samples *t*-test. Measurement data without normal distribution are expressed as the median (interquartile range) [M (Q1, Q3)] and compared between groups with the Mann-Whitney U rank sum test. Count data are represented by the number of cases (percentage) and compared between groups with the chi-squared or Fisher's exact test. Risk factors associated with elevated cTnT were analyzed with Lasso regression and multi-factor binary logistic regression. $P < 0.05$ was considered statistically significant.

## RESULTS

### *Clinical analysis*

The study patients comprised 232 patients diagnosed with AIS. In these patients, the average troponin level was 10.49 (with a range of 5.90 to 17.82) ng/L. Within this group, 84 patients (36.21%) showed raised cTnT levels, while 148 patients (63.79%) displayed typical cTnT levels. Significantly divergent age, National Institute of Health Stroke Scale (NIHSS) admission scores, and heart rates and the trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification were observed between the two groups ($P < 0.05$), as indicated in Table 1.

### *Medical background*

Concerning the medical background, there was no

**Table 1: Comparison of demographic data between the normal cTnT and the elevated cTnT groups**

| | Normal cTnT group (n = 148) | Elevated cTnT group (n = 84) | *P* |
|---|---|---|---|
| Age | 70(61,79) | 76(67,83) | **0.002** |
| Gender (male) | 73(49.32%) | 40(47.62%) | 0.580 |
| Systolic blood pressure at admission (mmHg) | 160(146,173) | 155(130,176) | 0.241 |
| Diastolic blood pressure at admission (mmHg) | 90(79,93) | 85(74,92) | 0.081 |
| Heart rate (beats/min) | 75(70,82) | 79(70,92) | **0.014** |
| NIHSS at admission (scores) | 5(2,9) | 8.5(4,14) | **0.002** |
| Infarct location | | | **0.054** |
| Basal ganglia and thalamus | 71(48.00%) | 47(56.00%) | |
| Cerebral lobe | 83(56.10%) | 52(61.90%) | |
| Brainstem | 13(8.80%) | 8(9.50%) | |
| Cerebellum | 8(5.41%) | 8(9.52%) | |
| TOAST classification | | | **<0.001** |
| LAA | 63(42.56%) | 28(33.33%) | |
| CE | 11(7.43%) | 23(27.38%) | |
| SAO | 57(38.51%) | 14(16.67%) | |
| SOE | 3(2.03%) | 5(5.95%) | |
| SUE | 14(9.46%) | 14(16.67%) | |

cTnT: serum cardiac troponin T; NIHSS: National Institute of Health Stroke Scale; TOAST: Trial of ORG 10172 in Acute Stroke Treatment; LAA: large-artery atherosclerosis; CE: cardio embolism; SAO: small-artery occlusion; SOE: stroke of other determined etiology; SUE: stroke of undetermined etiology; Bold numbers indicate statistically significant differences between groups.

significant difference observed in the occurrence of hypertension, diabetes, ischemic stroke, and cerebral hemorrhage between the two groups of patients ($P > 0.05$). However, the percentage of patients with coronary artery disease and cardiac insufficiency was higher in cTnT increased group ($P < 0.05$), as shown in Table 2.

*ECG analysis*

Regarding the ECG, significant differences were noted in the ratio of patients showing ECG abnormalities between the two groups ($P < 0.05$). Among the various types of ECG abnormalities, the atrial premature beat, ratio of patients exhibiting bundle branch block, atrial fibrillation and ST-T change differed significantly between the two groups ($P < 0.05$). (Table 3).

*Laboratory examination*

In laboratory examinations, there were significant difference between the two groups for various parameters. These included myoglobin, N-terminal B-type natriuretic peptide precursor, triglycerides, cholesterol, low-density lipoprotein, homocysteine, prothrombin time, activated partial thromboplastin time, red blood cell count, hemoglobin, neutrophil/lymphocyte ratio (NLR), total protein, albumin, C-reactive protein (CRP), creatinine, blood urea nitrogen, and uric acid, cystatin C (with a significance of $P < 0.05$). On the other hand, no significant differences were observed in the levels of creatine kinase, creatine kinase isoenzymes, blood glucose, hemoglobin A1c, high-density lipoprotein, lipoprotein a, and platelets between the two groups (with a significance of $P > 0.05$) (as shown in Table 4).

**Table 2: Comparison of medical history between the normal cTnT and the elevated cTnT groups**

| | Normal cTnT group (n = 148) | Elevated cTnT group (n = 84) | *P* |
|---|---|---|---|
| History of ischemic stroke | 34(22.97%) | 24(28.57%) | 0.344 |
| History of cerebral hemorrhage | 5(3.38%) | 1(1.19%) | 0.313 |
| Hypertension | 121(81.76%) | 68(80.95%) | 0.880 |
| Diabetes | 36(24.32%) | 26(30.95%) | 0.273 |
| Coronary heart disease | 11(7.43%) | 20(23.81%) | **<0.001** |
| Cardiac insufficiency | 5(3.38%) | 21(25.00%) | **<0.001** |
| Rheumatic heart disease | 5(3.38%) | 7(8.33%) | 0.126 |

cTnT: serum cardiac troponin T

Bold numbers indicate statistically significant differences between groups.

*Lasso analysis of risk factors for elevated cTnT in patients with AIS*

Possible risk factors for elevated cTnT were screened from patients' general condition, medical history, electrocardiogram, imaging examination, laboratory examination and other data by lasso regression analysis. Then a multifactorial binary logistic analysis was conducted. The analysis included age, NIHSS scores upon admission, coronary artery disease, bundle branch block, logarithm to the base 10 of NT-proBNP ($Log_{10}$NT-proBNP), cystatin C and NLR as independent variables. The dependent variable was the presence or absence (yes, 0; no, 1) of elevated cTnT. The findings revealed that bundle branch block in ECG, $Log_{10}$NT-proBNP, cystatin C, and NLR were independent risk factors for elevated cTnT in patients with AIS (refer to Table 5).

## DISCUSSION

In this study, 232 patients diagnosed with AIS were enrolled. We found that 84 patients (36.21%) had increased levels of cTnT. Those with elevated cTnT levels were of higher age and exhibited higher NIHSS scores compared to those with regular cTnT levels, suggesting the presence of more pronounced neurological impairments. Patients with normal and elevated

**Table 3: Comparison of electrocardiogram results between the normal cTnT and the elevated cTnT groups**

| | Normal cTnT group (n = 148) | Elevated cTnT group (n = 84) | *P* |
|---|---|---|---|
| Normal electrocardiogram | 40(27.03%) | 8(9.52%) | **0.003** |
| Sinus arrhythmia | 5(3.38%) | 0(0.00%) | 0.166 |
| Sinus tachycardia | 5(3.38%) | 4(4.76%) | 0.502 |
| Sinus bradycardia | 11(7.43%) | 3(3.57%) | 0.389 |
| Atrial premature beat | 10(6.76%) | 15(17.86%) | **0.005** |
| Ventricular premature beat | 6(4.05%) | 7(8.33%) | 0.145 |
| Atrial flutter | 2(1.35%) | 2(2.38%) | 0.531 |
| Atrial fibrillation | 17(11.49%) | 27(32.14%) | **<0.001** |
| Atrioventricular block | 4(2.70%) | 2(2.38%) | 0.655 |
| Bundle branch block | 14(9.46%) | 25(29.76%) | **<0.001** |
| T-wave change | 42(28.38%) | 18(21.43%) | 0.372 |
| ST-T change | 28(18.92%) | 26(30.95%) | **0.020** |
| ST-segment change | 5(3.38%) | 4(4.76%) | 0.502 |

cTnT: serum cardiac troponin T

Bold numbers indicate statistically significant differences between groups.

**Table 4: Comparison of laboratory indicators between the normal cTnT and the elevated cTnT groups**

| | Normal cTnT group (n = 148) | Elevated cTnT group (n = 84) | *P* |
|---|---|---|---|
| NT-proBNP(pg/ml) | 181.5(56.75,468.3) | 1091(381.1,2797) | **<0.001** |
| $Log_{10}$NT-proBNP | 2.29±0.64 | 3.02±0.62 | **<0.001** |
| Myoglobin (mmol/L) | 31.45(21.62,48.23) | 61.49(33.15,113.78) | **<0.001** |
| Creatine kinase (IU/L) | 81.5(58,117) | 90(65.18,156.5) | 0.097 |
| Creatine kinase isoenzyme (IU/L) | 12(10.75,15.25) | 13(10.75,17) | 0.581 |
| Glucose (mmol/L) | 6.25(5.39,8.41) | 6.87(5.62,8.83) | 0.201 |
| Hemoglobin A1c (%) | 5.8(5.5,6.5) | 5.9(5.5,7.1) | 0.453 |
| Triglyceride (mmol/L) | 1.39(0.9,2.14) | 1.08(0.7,1.65) | **0.007** |
| Total cholesterol (mmol/L) | 4.84±1.07 | 4.28±1.15 | **<0.001** |
| Low-density lipoprotein (mmol/L) | 2.82(2.2,3.6) | 2.51(1.84,3.15) | **0.003** |
| High-density lipoprotein (mmol/L) | 1.28±0.4 | 1.3±0.47 | 0.819 |
| Lipoprotein a (mmol/L) | 127(56.75,322.05) | 171(75,412.5) | 0.306 |
| Homocysteine (mmol/L) | 13.6(11.5,16.88) | 15.1(11.7,22.2) | **0.044** |
| Prothrombin time (s) | 11.25(10.8,11.7) | 11.8(11.05,12.5) | **<0.001** |
| Activated partial thromboplastin time (s) | 31.4(29.1,33.88) | 30.15(27.63,32.95) | **0.015** |
| prothrombin time(s) | 13.8(12.8,14.7) | 14.05(13.1,14.9) | 0.297 |
| Red blood cells ($10^{12}$/L) | 4.48±0.58 | 4.22±0.7 | **0.002** |
| Hemoglobin (g/L) | 135(126,146) | 128(111,141) | **0.001** |
| Platelet ($10^9$/L) | 171(136,214.75) | 164(125.5,197) | 0.086 |
| White blood cells ($10^9$/L) | 7.63±2.42 | 8.61±3.67 | 0.089 |
| Neutrophil ratio (%) | 66.69±12.62 | 72.87±12.54 | **<0.001** |
| Lymphocyte ratio (%) | 25.2±11 | 19.14±10.86 | **<0.001** |
| NLR | 2.71(1.82,4.51) | 4.22(2.45,7.92) | **<0.001** |
| C-reactive protein(mmol/ L) | 1(0.2,5.5) | 2.1(0.8,13.15) | **0.009** |
| Total protein(g/L) | 66.5(63.7,71.1) | 64.95(60.98,68.8) | **0.007** |
| Albumin(g/L) | 39.4(37,42.2) | 36.8(34.18,39.5) | **<0.001** |
| Blood urea nitrogen(mmol/ L) | 5.66(4.46,6.78) | 6.89(5.26,8.82) | **<0.001** |
| Creatinine(μmol/ L) | 62(54,76.88) | 75(60,95) | **<0.001** |
| Uric acid (μmol/ L) | 317.35(263.25,368.75) | 347.1(277,427) | **0.018** |
| Cystatin C(mg/ L) | 1.13(0.94,1.29) | 1.32(1.11,1.83) | **<0.001** |
| eGFR(mL·min-1.(1.73 m2)-1) | 128.67±37.23 | 103.06±37.69 | **<0.001** |

NT-proBNP: N-terminal pro-brain natriuretic peptide; NLR: neutrophil/lymphocyte ratio; eGFR:estimated glomerular filtration rate;

Bold numbers indicate statistically significant differences between groups.

cTnT had different TOAST etiological types (P<0.05). Patients with elevated cTnT had higher percentages of cardiogenic (27.38%VS 7.43%) and unexplained (16.67%VS 9.64%) AIS. An independent risk factor for elevated cTnT in patients with AIS was determined to be $Log_{10}$NT-proBNP (OR = 3.41, 95% CI = 1.62–7.16). This finding is consistent with the study by Král *et al*., who also identified NT-proBNP (OR = 1.05 per 100 μg/L increase; 95%CI = 1.018–1.093) as a significant predictor for the abnormal rise of cTnT levels in patients with AIS.[14]

**Table 5: Analysis of risk factors for elevated cTnT**

| | Univariate | | | Multivariate | | |
|---|---|---|---|---|---|---|
| | **P** | **OR** | **95% CI** | **P** | **OR** | **95% CI** |
| Age | **0.003** | 1.04 | 1.01-1.06 | 0.423 | 0.99 | 0.95-1.02 |
| NIHSS at admission | **0.007** | 1.06 | 1.02-1.11 | 0.683 | 1.01 | 0.95-1.08 |
| Coronary artery disease | **0.001** | 3.89 | 1.76-8.60 | 0.133 | 2.54 | 0.75-8.55 |
| Atrial fibrillation | **0.000** | 3.65 | 1.85-7.22 | 0.080 | 2.47 | 0.90-6.80 |
| Bundle branch block | **0.000** | 4.14 | 2.00-8.53 | **0.009** | 4.17 | 1.43-12.16 |
| $Log_{10}$NT-proBNP | **0.000** | 6.19 | 3.58-10.67 | **0.001** | 3.41 | 1.62-7.16 |
| Cystatin C | **0.000** | 7.47 | 3.22-17.29 | **0.002** | 6.86 | 2.01-23.43 |
| NLR | **0.001** | 1.13 | 1.06-1.22 | **0.016** | 1.13 | 1.02-1.25 |

NT-proBNP: N-terminal pro-brain natriuretic peptide; cTnT: serum cardiac troponin T
Bold numbers indicate statistically significant differences between groups.

According to previous reports, the rise in cTnT levels can be linked to the existing cardiovascular conditions in patients, as well as cardiovascular complications following a stroke and cardiac damage due to neural factors.[15] In our study, patients exhibiting higher cTnT levels had a greater occurrence of both coronary artery disease (7.43% vs. 23.81%) and cardiac insufficiency (3.38% vs. 25.00%) compared to those with normal cTnT levels. Elevated cTn levels in patients with cardiac insufficiency may stem from factors such as inadequate blood supply to the inner lining of the heart, activation of neural hormones, release of inflammatory cytokines, stretching of the heart muscle, heightened wall pressure, oxidative stress, and disruptions in calcium handling within heart muscle cells. These combined processes can ultimately lead to the death, self-destruction, or breakdown of troponin in heart muscle cells, resulting in its release.[16,17] Although patients with elevated cTnT had a higher rate of coronary artery disease, it was found after multi-factor correction that coronary heart disease was not an independent risk factor, which may be related to the common risk factors of stroke and coronary heart disease, including personal lifestyle, diet, related disease history, genetic history and external environmental factors.[18] The association between coronary heart disease and elevated cTnT was weakened after multifactor adjustment, which may also be related to the small number of cases included in this study.

Patients with AIS, especially those with higher levels of cTnT, frequently display abnormal ECG patterns. These deviations primarily encompass ST-segment elevation or depression, alterations in T-wave configuration, and disruptions in bundle branch conduction.[19] The results of our study showed that patients with AIS who have elevated cTnT levels tend to experience a greater prevalence of atrial fibrillation (11.49% vs. 32.14%) and bundle branch conduction abnormalities (8.11% vs. 25.00%). Among these, the presence of bundle branch block emerged as an autonomous factor that heightens the risk of increased cTnT levels in patients with AIS. Atrial fibrillation is the main cause of cardiogenic stroke, atrial fibrillation is associated with higher in-hospital mortality, not only in the ischemic stroke patient population as a whole, but also in cardioembolic stroke subtype patients.[7] However, risk factor analysis showed that atrial fibrillation was not an independent risk factor for elevated cTnT. Bundle branch block signifies modifications in the shape or timing of the QRS complex on the ECG and frequently indicates structural and functional changes within the heart, with a predilection for right bundle branch block. In cases where bundle branch block coexists with elevated cTnT, the underlying cause could be attributed to irregular cardiac hemodynamics and anomalous myocardial metabolism. These factors can impede coronary microvascular function, resulting in diminished local myocardial blood flow and potential myocardial ischemia.[20]

Aside from factors related to heart function, increased levels of cTnT in patients with AIS are also linked to factors not originating from the heart, as mentioned in a previous study.[21] The results of our study revealed that several laboratory markers exhibited significant differences between patients with normal cTnT levels and those with elevated cTnT levels. These markers encompass indicators of inflammation, kidney function, and

blood clotting. Notably distinct inflammatory markers between these two groups comprised CRP, the proportion of neutrophils (NEUT%), and the NLR. Notably, NLR independently emerged as a risk factor for heightened cTnT. This outcome may be due to the stress response of the body during the onset of AIS. The inflammation triggered by stress and the cytokine response pathway could potentially amplify the myocardial cell damage associated with elevated cTn levels in patients with AIS, as also discussed in a previous study.[22] Increased levels of cTnT in patients with AIS also demonstrated a connection with indicators of kidney function. An autonomous risk factor for elevated cTnT among patients with AIS was identified as cystatin C. This factor could be associated with uremic cardiomyopathy, reduced elimination of cTnT by the kidneys, and enlargement of the left ventricle.[23] This association occurs because, as the glomerular filtration rate diminishes, the complete clearance of troponin and its byproducts becomes ineffective. Furthermore, factors like heightened cardiac afterload, excessive fluid volume, myocardial damage unrelated to heart ischemia, and increased tension in the myocardial wall due to changes in calcium/phosphorus metabolism can intensify micro-injuries in cardiomyocytes. These collective effects contribute to the elevation of cTnT levels.

This study also has the following other limitations: First, due to the limitation of retrospective study, the sample size of some groups is insufficient. Second, we are inability to provide brain topography associated with the presence of elevated serum cardiac troponin T. Third, we did not continuously track cTnT levels throughout the study, and there is a lack of information about the long-term progress of patients with elevated cTnT.

In future studies, the sample size of the study should be increased and should focus on cTnT dynamic monitoring. Additionally, the relationship between serum cTnT level and results of cerebral ischemia imaging can be further studied.

In conclusion, older patients with AIS who had higher levels of cTnT also exhibited more severe neurological impairments and a greater number of comorbidities. Furthermore, an elevated cTnT in patients with AIS may link to cardiac insufficiency, changes in kidney function, and signs of inflammation.


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## DISCLOSURES

Ethics: This study was approved by the Ethics Committee of Ziyang Central Hospital (2020-58). Written informed consent was obtained from all participants.

Data availability: All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.


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Conflict of interests: None

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