Original Article



# Association between cardiac troponin testing at scheduled admission and mortality in patients with comorbidities

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**Background:** Cardiac troponin I (cTnI) is a gold-standard biomarker for detecting myocardial infarction. Recently, the prognostic role of cTnI was reported for stable coronary artery disease and other chronic diseases. This study aimed to evaluate the usefulness of cTnI testing at scheduled admission of patients with comorbidities.

**Methods:** We retrospectively enrolled patients with comorbidities who were admitted through the outpatient clinic from April 2010 to December 2018. The enrolled patients were divided into two groups depending on whether cTnI was measured at admission. The primary endpoint was the mortality rate at one year after admission. Secondary endpoints included 30-day and in-hospital mortality rates.

**Results:** A population of 50,119 patients was divided into two groups, with 43,974 (87.8%) patients included in the no cTnI group and 6,145 (12.2%) patients included in the cTnI group. The multivariable analysis showed a reduction of mortality at one year in the cTnI group [5.9% *vs.* 3.8%, hazard ratio (HR) =0.78; 95% confidence interval (CI): 0.68–0.89; P<0.001]. Among 5,882 propensity score-matched pairs, this trend persisted, and the mortality rate was significantly lower in the cTnI group (5.3% *vs.* 3.9%, HR =0.77; 95% CI: 0.65–0.91; P=0.002). Patients with cTnI measurements taken at admission underwent cardiac evaluation and therapy more frequently.

**Conclusions:** The measurement of cTnI at scheduled admission may affect the mortality during one year of follow-up. Further studies are needed to validate our results.

**Keywords:** Acute coronary syndrome; troponin; mortality

Submitted Jul 21, 2022. Accepted for publication Nov 07, 2022. Published online Jan 06, 2023.
doi: 10.21037/atm-22-3681
**View this article at:** https://dx.doi.org/10.21037/atm-22-3681

## Introduction

Cardiac troponin I (cTnI) is highly specific to cardiac myocytes and has become a marker of choice for the evaluation of myocardial injury (1). It has been subsequently included in the definition of acute myocardial infarction and is regarded as the gold-standard biomarker for diagnosis (2,3). Extended from the detection of ischemic injury, cTnI was recently implicated in the diagnosis and prognosis of patients with stable coronary artery disease or chronic conditions (4,5), and some previous studies have reported that cTnI level may also be useful as a prognostic marker even in healthy individuals without cardiac symptoms (6,7).

On the other hand, it is well known that cTnI could also be elevated due to noncardiac condition such as sepsis, embolism, or kidney injury (8). According to previous studies conducted in the emergency room, a high percentage of patients with elevated cTnI levels are not diagnosed with acute coronary syndrome (9,10). Hence, cTnI elevation is not exclusively found in acute myocardial infarction. Indeed, robust evidence supports the link between cTnI level and the prognosis of chronic noncardiac conditions, but data on the use of cTnI testing in noncardiac patients remain scarce. In the outpatient setting, an increasing number of studies suggest the use of cTnI for assessing chronic conditions (11,12). However, controversy persists on the use of cTn as a general prognostic factor, because there also is a report that cTnI was not associated with long-term mortality (13). Considering that cTnI elevations in patients without coronary disease are frequently symptomless (5,8), detection of cTnI elevation is still likely to change the management of patients admitted from outpatient department, but it is not yet clear in which group of patients cTnI measurement would be helpful. In this study, we enrolled comorbid patients who were admitted from the outpatient clinic except for those who were admitted to cardiac or cardiac surgical wards by cardiologists. After dividing these patients according to the availability of cTnI measurements taken at admission, we aimed to investigate whether obtaining data on the cTnI level at planned admission was associated with difference in patient management and mortality in patients with comorbidities. We also aimed to show the serial changes and the incidence of normalization of cTnI level during the hospitalization. Our results may provide valuable information on measuring cTnI level in relatively stable patients in the outpatient clinic, adding evidence to previous studies emphasizing the importance of cTnI measurement in emergency or intensive care settings. We present the following article in accordance with the STROBE reporting checklist (available at https://atm.amegroups.com/article/view/10.21037/atm-22-3681/rc).

## Methods

### *Ethical approval*

This was a retrospective, large single-center study that used data from the electronic medical records at Samsung Medical Center, Seoul, Korea. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the institutional review board of Samsung Medical Center (No. SMC 2021-03-165) and individual consent for this retrospective analysis was waived.

### *Data curation and study population*

The study data were extracted from the "Clinical Data Warehouse Darwin-C" of Samsung Medical Center for investigation, which is an electronic system built for investigators to search and compile anonymized medical records from the institutional electronic archive system. Our archive system contains records of more than 2.2 million surgeries, 900 million laboratory findings, 100 million disease codes, and 200 million prescriptions for nearly four million patients. In addition, using a unique personal identification number, details about deaths outside of our institution are consistently updated and confirmed by the National Population Registry of the Korea National Statistical Office.

For this study, we retrospectively generated a cohort consisting of 289,764 consecutive adult patients who were admitted from the outpatient clinic to any department between April 2010 and December 2018. From this cohort, we selected 50,119 patients with comorbidity based on the Charlson Comorbidity Index (CCI) (14,15). The mortality rates were then compared depending on whether the cTnI level was obtained at admission or not. The group of patients with cTnI measurements was further divided into two groups based on whether cTnI was elevated to predict their impact on mortality. We also collected cTnI values measured during hospitalization to follow-up the serial changes of cTnI.

### *Study variables and endpoints*

Automatically extracted electronic medical records were

used to organize demographic variables, medical history, and clinical course. The presence of a comorbidity was defined by a CCI value of greater than 0, and the diseases relevant for the CCI were calculated by using algorithms designed for International Classification of Diseases' coded administrative datasets (15).

The primary endpoint was the mortality rate at one year after admission. Secondary endpoints included 30-day and in-hospital mortality rates.

### *CTnI measurement*

During the study period, our institution used cTnI, measured by an automated analyzer (Advia Centaur XP; Siemens Healthcare Diagnostics, Erlangen, Germany). According to the manufacturer, the lowest limit of detection was 6 ng/L, with the 99th-percentile upper reference limit being 40 ng/L. For this study, we curated cTnI which was measured with other blood laboratory tests immediately after admission to general wards and during hospitalization. Inclusion of cTnI within the blood laboratory tests was at discretion of the attending clinician based on the patient's recent cardiac symptoms and underlying comorbidities.

### *Statistical analysis*

Baseline characteristics are presented as mean ± standard deviation (SD) values or median with interquartile range (IQR) values for continuous variables or as numbers with percentages for categorical variables, respectively. Differences between the two groups were compared using the chi-squared or Fisher's exact test for categorical variables and the *t*-test or Mann-Whitney U test for continuous variables. Outcomes were compared using a stratified Cox regression model and were reported as hazard ratios (HRs) with 95% confidence intervals (CIs). The following variables were retained in the Cox proportional hazards model: age, sex, hypertension, dyslipidemia, departments of admission, current smoking, cardiac symptoms, and CCI. Kaplan-Meier survival curves were constructed and compared with the log-rank test. To further reduce selection bias between the two groups, we performed a propensity score-matched analysis on all variables. We used caliper widths that were 1.5 of the pooled SD of the logit of the propensity score and generated 1:1 individually matched data without replacement. An appropriate balance between the groups with an absolute standardized mean difference (ASD) of less than 10% suggested successful propensity score matching. The propensity-score matched population was adjusted with departments of admission to compare mortalities. We also performed subgroup analysis using the propensity score-matched population to reveal hidden interactions between the observed association and variables such as sex, departments of admission, and cardiac symptoms. The results of subgroup analysis are presented in the forest plot. All analyses were performed using R 4.0.2 (R Foundation for Statistical Computing, Vienna, Austria; http://www.R-project.org/). All tests were two-tailed, and P<0.05 was considered to be statistically significant.

## Results

### *Patient characteristics*

Among 289,764 consecutive adult patients who were admitted from the outpatient clinic to any department other than cardiology or cardiac, we excluded 239,645 patients without known comorbidities. Thus, a total of 50,119 comorbid patients were finalized for analysis and stratified into two groups according to cTnI measurements at admission as follows: 43,974 (87.7%) patients in the no cTnI group and 6,145 (12.3%) patients in the cTnI group.

The baseline characteristics of the study participants are summarized in *Table 1*. The median durations from admission to discharge were four days in the no cTnI group and eight days in the cTnI group, respectively (P<0.001). Patients who underwent cTnI tests at admission were generally older than those without a history of cTnI testing. The prevalence of cardiac symptoms was also enormously greater in the cTnI group. Although there was no significant difference in CCI values between the two groups, the cTnI group displayed greater prevalence rates of myocardial infarction, heart failure, and cardiovascular disease. More patients in the cTnI group were hospitalized to surgical departments. The departments that the study participants were admitted to are listed according to their group in Table S1.

### *Mortality*

In the entire population, the multivariable analysis showed a significant reduction in one-year mortality in the cTnI group (5.9% *vs.* 3.8%, HR =0.78; 95% CI 0.68–0.89; P<0.001). On the other hand, there were no significant differences in the 30-day and in-hospital mortality rates (0.4% *vs.* 0.5%, HR =1.39; 95% CI: 0.95–2.04; P=0.09 for

**Table 1** Baseline characteristics

| Variables | Entire population | | | | Propensity-score matched population | | |
|---|---|---|---|---|---|---|---|
| | No troponin I (n=43,974) | Troponin I (n=6,145) | P value | ASD (%) | No troponin I (n=5,882) | Troponin I (n=5,882) | ASD (%) |
| Age, years | 58.9 (±13.5) | 64.1 (±12.1) | <0.001 | 40.2 | 64.3 (±12.1) | 63.9 (±12.2) | 3.2 |
| Male | 25,162 (57.2) | 3,378 (55.0) | <0.001 | 4.5 | 3,141 (53.4) | 3,164 (53.8) | 0.8 |
| Current smoking | 9,553 (21.7) | 1,143 (18.6) | <0.001 | 7.8 | 997 (17.0) | 1,053 (17.9) | 2.5 |
| Hypertension | 11,319 (25.7) | 2,043 (33.2) | <0.001 | 16.5 | 1,784 (30.4) | 1,929 (32.8) | 5.3 |
| Dyslipidemia | 7,511 (17.1) | 1,219 (19.8) | <0.001 | 7.1 | 999 (17.0) | 1,104 (18.8) | 4.7 |
| Admission to surgical departments | 18,500 (42.1) | 3,695 (60.1) | <0.001 | 36.7 | 3,386 (57.6) | 3,442 (58.5) | 1.9 |
| Admission to medical departments | 24,692 (56.2) | 2,374 (38.6) | <0.001 | 35.6 | 2,412 (41.0) | 2,364 (40.2) | 1.7 |
| Admission to other departments | 782 (1.8) | 76 (1.2) | 0.003 | 4.4 | 84 (1.4) | 76 (1.3) | 1.2 |
| Cardiac symptoms | 1,167 (2.7) | 786 (12.8) | <0.001 | 38.7 | 526 (8.9) | 534 (9.1) | 0.5 |
| Chest pain | 272 (0.6) | 96 (1.6) | <0.001 | 9.1 | 107 (1.8) | 78 (1.3) | 0.4 |
| Palpitation | 815 (1.9) | 697 (11.3) | <0.001 | 38.9 | 378 (6.4) | 460 (7.8) | 5.4 |
| Dizziness | 694 (1.6) | 639 (10.4) | <0.001 | 37.8 | 317 (5.4) | 418 (7.1) | 7.1 |
| Fatigue | 12 (0.0) | 6 (0.1) | 0.02 | 2.8 | 7 (0.1) | 6 (0.1) | 0.5 |
| Syncope | 11 (0.0) | 13 (0.2) | <0.001 | 5.4 | 7 (0.1) | 9 (0.2) | 0.9 |
| Diaphoresis | 745 (1.7) | 682 (11.1) | <0.001 | 39.2 | 341 (5.8) | 443 (7.5) | 7.0 |
| Arrhythmia | 207 (0.5) | 55 (0.9) | <0.001 | 5.2 | 103 (1.8) | 41 (0.7) | 9.6 |
| CCI | 1.89 (±1.29) | 1.86 (±1.25) | 0.10 | 2.3 | 1.92 (±1.34) | 1.86 (±1.24) | 4.6 |
| Myocardial infarction | 346 (0.8) | 190 (3.1) | <0.001 | 16.8 | 151 (2.6) | 172 (2.9) | 2.2 |
| Heart failure | 449 (1.0) | 215 (3.5) | <0.001 | 16.7 | 165 (2.8) | 190 (3.2) | 2.5 |
| Peripheral vascular disease | 430 (1.0) | 97 (1.6) | <0.001 | 5.3 | 51 (0.9) | 92 (1.6) | 6.4 |
| Cerebrovascular disease | 9,032 (20.5) | 2,761 (44.9) | <0.001 | 53.8 | 2,552 (43.4) | 2,513 (42.7) | 1.3 |
| Dementia | 22 (0.1) | 1 (0.0) | 0.40 | 1.9 | 3 (0.1) | 1 (0.0) | 1.8 |
| Chronic pulmonary disease | 71 (0.2) | 12 (0.2) | 0.66 | 0.8 | 8 (0.1) | 12 (0.2) | 1.7 |
| Rheumatic disease | 1,913 (4.4) | 148 (2.4) | <0.001 | 10.8 | 181 (3.1) | 146 (2.5) | 3.6 |
| Peptic ulcer disease | 43 (0.1) | 6 (0.1) | 1.0 | <0.1 | 1 (0.0) | 6 (0.1) | 3.5 |
| Diabetes without chronic complication | 13,595 (30.9) | 1,849 (30.1) | 0.19 | 1.8 | 1,860 (31.6) | 1,797 (30.6) | 2.3 |
| Diabetes with complication | 3,903 (8.9) | 465 (7.6) | 0.001 | 4.8 | 617 (10.5) | 448 (7.6) | 9.9 |
| Hemiplegia | 693 (1.6) | 85 (1.4) | 0.28 | 1.6 | 90 (1.5) | 83 (1.4) | 1.0 |
| Any malignancy without metastasis | 1,386 (3.2) | 98 (1.6) | <0.001 | 10.2 | 97 (1.6) | 98 (1.7) | 0.1 |
| Moderate or severe liver disease | 420 (1.0) | 23 (0.4) | <0.001 | 7.2 | 21 (0.4) | 23 (0.4) | 0.6 |
| Metastatic solid tumor | 0 (0.0) | 0 (0.0) | – | <0.1 | 0 (0.0) | 0 (0.0) | <0.1 |
| AIDS | 47 (0.1) | 3 (0.0) | 0.26 | 2.1 | 2 (0.0) | 3 (0.1) | 0.8 |

Data are presented as n (%) or mean (± standard deviation). ASD, absolute standardized mean difference; CCI, Charlson comorbidity index; AIDS, acquired immunodeficiency syndrome.

**Table 2** Mortalities according to measurement of troponin I at admission

| Outcomes | No troponin I, n (%) | Troponin I, n (%) | Unadjusted | | Adjusted | |
|---|---|---|---|---|---|---|
| | | | HR (95% CI) | P value | HR (95% CI) | P value |
| Entire population | n=43,974 | n=6,145 | | | | |
| 1-year mortality | 2,595 (5.9) | 233 (3.8) | 0.65 (0.57–0.75) | <0.001 | 0.78 (0.68–0.89) | <0.001 |
| 30-day mortality | 185 (0.4) | 32 (0.5) | 1.24 (0.85–1.80) | 0.27 | 1.39 (0.95–2.04) | 0.09 |
| In-hospital mortality | 129 (0.3) | 23 (0.4) | 1.20 (0.76–1.88) | 0.44 | 1.45 (0.91–2.31) | 0.12 |
| Propensity-score matched population | n=5,882 | n=5,882 | | | | |
| 1-year mortality | 312 (5.3) | 230 (3.9) | | | 0.77 (0.65–0.91) | 0.002 |
| 30-day mortality | 21 (0.4) | 32 (0.5) | | | 1.53 (0.88–2.65) | 0.13 |
| In-hospital mortality | 21 (0.4) | 23 (0.4) | | | 1.78 (0.92–3.42) | 0.08 |

Multivariable adjustment included age, sex, hypertension, dyslipidemia, departments of admission, current smoking, cardiac symptoms and CCI for the entire population, and the propensity-score matched population was adjusted with departments of admission. HR, hazard ratio; CI, confidence interval; CCI, Charlson comorbidity index.

30-day mortality and 0.3% *vs.* 0.4%, HR =1.45; 95% CI: 0.91–2.31; P=0.12 for in-hospital mortality) (*Table 2*).

After propensity score matching, 5,882 well-matched pairs were generated with ASD <10% on all variables. In this population, the results for mortality comparisons yielded similar results to entire population. The mortality was significantly lower in the cTnI group at one year after admission (5.3% *vs.* 3.9%, HR =0.77; 95% CI: 0.65–0.91; P=0.002), but not at 30 days or in the hospital (0.4% *vs.* 0.5%, HR =1.53; 95% CI: 0.88–2.65; P=0.13 for 30-day mortality and 0.4% *vs.* 0.4%, HR =1.78; 95% CI: 0.92–3.42; P=0.08 for in-hospital mortality) (*Figure 1*). In the subgroup analysis, the observed association between cTnI measurement and reduced one-year mortality significantly interacted with the factor of admission to surgical departments. The association appeared to be valid only in those patients who hospitalized to medical departments (HR =0.64; 95% CI: 0.51–0.79; P<0.001 for other than surgical departments and HR =1.06; 95% CI: 0.81–1.40; P=0.674 for planned surgery; P for interaction =0.005) (*Figure 2*).

### *Clinical course*

The major diagnoses and management performed during hospitalization and after discharge are presented in *Table 3*. Patients in the cTnI group tended to undergo cardiac evaluations and therapies more frequently. More patients in the cTnI group were referred for cardiologist consultations (13.7% *vs.* 21.9%, P<0.001 during hospitalization and 17.6% *vs.* 28.0%, P<0.001 for after discharge) as well as cardiac examinations and medical treatments. The prevalence rates of myocardial infarction (0.0% *vs.* 0.4%, P<0.001 for during hospitalization and 0.5% *vs.* 1.1%, P<0.001 for after discharge) and percutaneous coronary intervention (0.1% *vs.* 0.8%, P<0.001 during hospitalization and 1.4% *vs.* 3.0%, P<0.001 for after discharge) were also higher in the cTnI group. In the propensity score-matched population, this trend persisted primarily during hospitalization rather than after discharge.

The change of cTnI level during hospitalization is also presented in *Table 3*. In the cTnI group, the median cTnI level at admission was 6 ng/L (IQR: 6–12 ng/L), and an elevation above the upper reference limit was found in 8.3% of entire patients. In cTnI measurement after admission, more patients in cTnI group showed troponin elevation, but they had lower highest value and higher normalization compared to no cTnI group.

The cTnI group was further divided into 5,633 (91.7%) patients with normal cTnI level and 512 (8.3%) patients with cTnI elevation. The clinical course and mortality of these subgroups are presented in *Table 4*. The mortality was higher in those with cTn elevation despite more actively performed evaluation and treatments during hospitalization and after discharge.

## Discussion

In this study, we demonstrated that cTnI measurement in patients with comorbidities at scheduled admission to a

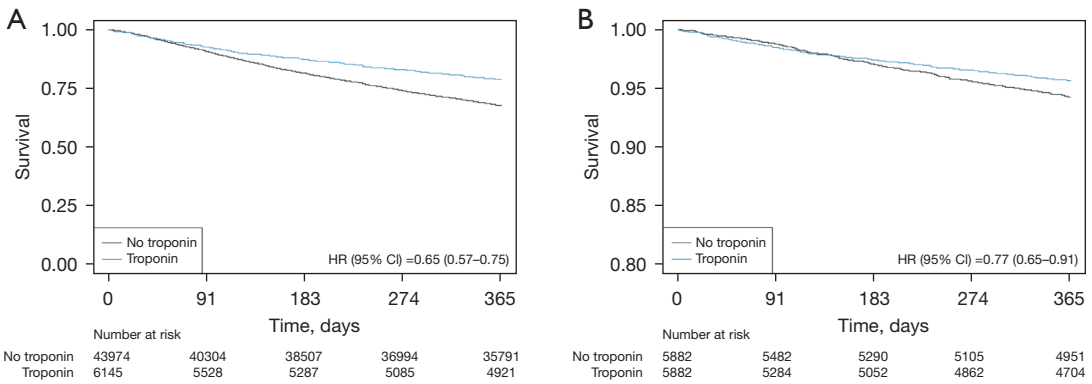


**Figure 1** Kaplan-Meier curves of (A) entire population and (B) propensity score-matched population according to cTnI measurement implementation during one year. HR, hazard ratio; CI, confidence interval; cTn I, cardiac troponin I.

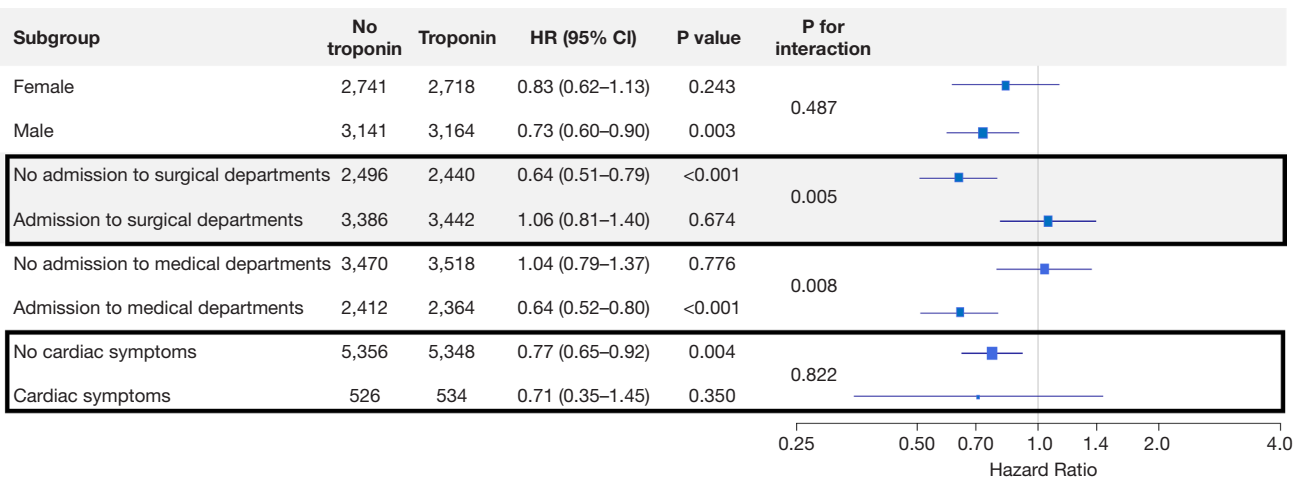


**Figure 2** Subgroup analysis for one year mortality. HR, hazard ratio; CI, confidence interval.

department other than cardiology or cardiac surgery may affect patient management during hospital stay and decrease one-year mortality. Those patients who underwent cTnI level measurement at admission tended to be managed with more intensive cardiac evaluation and therapy. Our study suggests the possibility that a screening of cTnI level may be helpful in improving the mortality of relatively stable patients with comorbidities.

The use of cTnI is firmly established for the early diagnosis of acute coronary syndrome (16). Cardiac-specific troponins are elevated in blood following cardiac injury by the mechanism of necrosis, apoptosis, increased membrane permeability of cardiomyocytes and decreased clearance (1). Both cTnI and cTnT are considered the gold-standard biomarkers for detection of myocardial injury, but cTnI is different from cTnT in its kinetic characteristics and clinical outcomes. Although both have half-lives of 1–2 hours, cTnT has a biphasic release pattern whereas cTnI has a monophasic release pattern (17). Moreover, cTnI appears to be a more specific marker of risk of composite cardiovascular outcomes, whereas cTnT is more strongly associated with risk of non-cardiovascular disease death (18).

In addition to the acute condition, it is well known that cTnI can be elevated in a variety of chronic conditions, including nonischemic and extracardiac conditions such as chronic heart failure, diabetes, pulmonary hypertension, stable coronary artery disease, and chronic kidney disease (8,19,20). The release of cTnI in these conditions is attributed to multiple factors such as oxygen supply-demand mismatch, systemic hypoxia, and inflammatory materials (5). In this study, elevated cTnI levels were found among 8.3% of patients with cTnI measurements

**Table 3** In-hospital and postdischarge management

| Managements | Entire population | | | Propensity-score matched population | | |
|---|---|---|---|---|---|---|
| | No troponin I (n=43,974) | Troponin I (n=6,145) | P value | No troponin I (n=5,882) | Troponin I (n=5,882) | P value |
| Troponin I change during hospitalization | | | | | | |
| Troponin I level at admission, ng/L | – | 6 [6–12] | – | – | 6 [6–12] | – |
| Troponin I elevation at admission | – | 512 (8.3) | – | – | 493 (8.4) | – |
| Lowest troponin I level, ng/L | 6 [6–17] | 6 [6–9] | <0.001 | 6 [6–20] | 6 [6–9] | <0.001 |
| Highest troponin I level, ng/L | 10 [6–34] | 6 [6–19] | <0.001 | 14 [6–43] | 6 [6–18] | <0.001 |
| Troponin I elevation after admission | 442 (1.0) | 354 (5.8) | <0.001 | 117 (2.0) | 322 (5.5) | <0.001 |
| Troponin I normalization | 199 (0.5) | 116 (1.9) | <0.001 | 58 (1.0) | 109 (1.9) | <0.001 |
| Management during hospitalization | | | | | | |
| In-hospital evaluation | | | | | | |
| Echocardiogram | 5,903 (13.4) | 1,897 (30.9) | <0.001 | 887 (15.1) | 1,733 (29.5) | <0.001 |
| Stress echocardiogram | 257 (0.6) | 61 (1.0) | <0.001 | 25 (0.4) | 53 (0.9) | 0.002 |
| Treadmill test | 50 (0.1) | 17 (0.3) | 0.002 | 8 (0.1) | 15 (0.3) | 0.21 |
| Coronary computed tomographic angiography | 222 (0.5) | 73 (1.2) | <0.001 | 65 (1.1) | 60 (1.0) | 0.719 |
| Coronary artery angiogram | 194 (0.4) | 170 (2.8) | <0.001 | 62 (1.1) | 161 (2.7) | <0.001 |
| In-hospital diagnosis | | | | | | |
| Myocardial infarction | 16 (0.0) | 23 (0.4) | <0.001 | 6 (0.1) | 22 (0.4) | 0.005 |
| ST-elevation | 4 (0.0) | 4 (0.1) | 0.007 | 2 (0.0) | 4 (0.1) | 0.683 |
| Non ST-elevation | 12 (0.0) | 19 (0.3) | <0.001 | 4 (0.1) | 18 (0.3) | 0.006 |
| In-hospital cardiovascular drugs | | | | | | |
| Beta-blocker | 4279 (9.7) | 888 (14.5) | <0.001 | 656 (11.28) | 827 (14.1) | <0.001 |
| Calcium channel blocker | 11,115 (25.3) | 2,488 (40.5) | <0.001 | 1,827 (31.3) | 2,337 (39.8) | <0.001 |
| Statin | 10,821 (24.6) | 2,415 (39.3) | <0.001 | 1,844 (31.4) | 2,177 (37.0) | <0.001 |
| Warfarin | 864 (2.0) | 435 (7.1) | <0.001 | 235 (4.0) | 336 (5.7) | <0.001 |
| Antiplatelet | 10,363 (23.6) | 3,133 (51.0) | <0.001 | 1,994 (33.9) | 2,877 (48.9) | <0.001 |
| Renin angiotensin aldosterone system inhibitor | 12,749 (29.0) | 2,536 (41.3) | <0.001 | 2,016 (34.3) | 2,387 (40.6) | <0.001 |
| Direct oral anticoagulant | 703 (1.6) | 208 (3.4) | <0.001 | 101 (1.7) | 203 (3.5) | <0.001 |
| In-hospital care | | | | | | |
| Cardiologist evaluation | 6,011 (13.7) | 1,348 (21.9) | <0.001 | 1,245 (21.2) | 1,286 (21.9) | 0.038 |
| Percutaneous coronary intervention | 62 (0.1) | 48 (0.8) | <0.001 | 21 (0.4) | 48 (0.8) | 0.002 |
| Coronary artery bypass grafting | 3 (0.0) | 8 (0.1) | <0.001 | 1 (0.0) | 8 (0.1) | 0.045 |
| Intensive care unit | 1,618 (3.7) | 2,621 (42.7) | <0.001 | 360 (6.1) | 2,374 (40.4) | <0.001 |

**Table 3** (*continued*)

**Table 3** (*continued*)

| Managements | Entire population | | | Propensity-score matched population | | |
|---|---|---|---|---|---|---|
| | No Troponin I (n=43,974) | Troponin I (n=6,145) | P value | No Troponin I (n=5,882) | Troponin I (n=5,882) | P value |
| ECMO | 31 (0.1) | 32 (0.5) | <0.001 | 12 (0.2) | 30 (0.5) | 0.009 |
| Continuous renal replacement therapy | 28 (0.1) | 32 (0.5) | <0.001 | 6 (0.1) | 31 (0.5) | <0.001 |
| Ventilator | 78 (0.2) | 78 (1.3) | <0.001 | 25 (0.4) | 76 (1.3) | <0.001 |
| Management after discharge | | | | | | |
| Postdischarge evaluation | | | | | | |
| Echocardiogram | 13,303 (30.3) | 2,373 (38.6) | <0.001 | 2,351 (40.0) | 2,257 (38.4) | 0.079 |
| Stress echocardiogram | 1,031 (2.3) | 189 (3.1) | 0.001 | 224 (3.8) | 177 (3.0) | 0.019 |
| Treadmill test | 649 (1.5) | 138 (2.2) | <0.001 | 129 (2.2) | 131 (2.2) | 0.95 |
| Coronary computed tomographic angiography | 942 (2.1) | 240 (3.9) | <0.001 | 231 (3.9) | 220 (3.7) | 0.631 |
| Coronary artery angiogram | 1,494 (3.4) | 395 (6.4) | <0.001 | 341 (5.8) | 366 (6.2) | 0.352 |
| Postdischarge diagnosis | | | | | | |
| Myocardial infarction | 223 (0.5) | 65 (1.1) | <0.001 | 53 (0.9) | 62 (1.1) | 0.453 |
| ST-elevation | 37 (0.1) | 8 (0.1) | 0.367 | 9 (0.2) | 8 (0.1) | 1.0 |
| Non ST-elevation | 186 (0.4) | 57 (0.9) | <0.001 | 44 (0.7) | 54 (0.9) | 0.361 |
| Postdischarge cardiovascular drugs | | | | | | |
| Beta-blocker | 6,925 (15.7) | 1,232 (20.0) | <0.001 | 1,126 (19.2) | 1,158 (19.7) | 0.47 |
| Calcium channel blocker | 15,277 (34.7) | 2,900 (47.2) | <0.001 | 2,559 (43.5) | 2,747 (46.7) | 0.001 |
| Statin | 16,430 (37.4) | 3,159 (51.4) | <0.001 | 2,787 (47.4) | 2,909 (49.5) | 0.026 |
| Warfarin | 1,397 (3.2) | 525 (8.5) | <0.001 | 359 (6.1) | 428 (7.3) | 0.012 |
| Antiplatelet | 15,883 (36.1) | 3,638 (59.2) | <0.001 | 3,063 (52.1) | 3,383 (57.6) | <0.001 |
| Renin angiotensin aldosterone system inhibitor | 18,482 (42.0) | 3,055 (49.7) | <0.001 | 2,890 (49.2) | 2,889 (49.1) | 1.0 |
| Direct oral anticoagulant | 1,749 (4.0) | 419 (6.8) | <0.001 | 299 (5.1) | 399 (6.8) | <0.001 |
| Postdischarge care | | | | | | |
| Cardiologist evaluation | 7,719 (17.6) | 1,718 (28.0) | <0.001 | 1,536 (26.1) | 1,628 (27.7) | 0.058 |
| Percutaneous coronary intervention | 617 (1.4) | 186 (3.0) | <0.001 | 152 (2.6) | 177 (3.0) | 0.18 |
| Coronary artery bypass grafting | 51 (0.1) | 13 (0.2) | 0.076 | 8 (0.1) | 13 (0.2) | 0.382 |
| Intensive care unit | 5,649 (12.8) | 1,001 (16.3) | <0.001 | 847 (14.4) | 942 (16.0) | 0.016 |
| ECMO | 191 (0.4) | 60 (1.0) | <0.001 | 37 (0.6) | 52 (0.9) | 0.136 |
| Continuous renal replacement therapy | 389 (0.9) | 75 (1.2) | 0.012 | 58 (1.0) | 71 (1.2) | 0.288 |
| Ventilator | 411 (0.9) | 61 (1.0) | 0.711 | 78 (1.1) | 58 (1.0) | 0.101 |

Data are presented as n (%) or median [interquartile range]. ECMO, extracorporeal membranous oxygenation.

**Table 4** In-hospital and postdischarge management of the troponin I group according to elevation of troponin I

| Managements | Entire population | | |
|---|---|---|---|
| | No troponin I elevation (n=5,633) | Troponin I elevation (n=512) | P value |
| Troponin I change during hospitalization | | | |
| TroponinI level at admission, ng/L | 6 [6–9] | 84 [54–185] | <0.001 |
| Lowest troponin I level, ng/L | 6 [6–7] | 60 [42–112] | <0.001 |
| Highest troponin I level, ng/L | 6 [6–14] | 112 [61–331] | <0.001 |
| Troponin I elevation after admission | 354 (6.3) | – | – |
| Troponin I normalization | – | 116 (22.7) | – |
| Management during hospitalization | | | |
| In-hospital evaluation | | | |
| Echocardiogram | 1,632 (29.0) | 265 (51.8) | <0.001 |
| Stress echocardiogram | 47 (0.8) | 14 (2.7) | <0.001 |
| Treadmill test | 16 (0.3) | 1 (0.2) | >0.99 |
| Coronary computed tomographic angiography | 68 (1.2) | 5 (1.0) | 0.80 |
| Coronary artery angiogram | 92 (1.6) | 78 (15.2) | <0.001 |
| In-hospital diagnosis | | | |
| Myocardial infarction | 7 (0.1) | 16 (3.1) | <0.001 |
| ST-elevation | 3 (0.1) | 1 (0.2) | 0.76 |
| Non ST-elevation | 4 (0.1) | 15 (2.9) | <0.001 |
| In-hospital cardiovascular drugs | | | |
| Beta-blocker | 781 (13.9) | 107 (20.9) | <0.001 |
| Calcium channel blocker | 2,223 (39.5) | 265 (51.8) | <0.001 |
| Statin | 2,190 (38.9) | 225 (43.9) | 0.03 |
| Warfarin | 377 (6.7) | 58 (11.3) | <0.001 |
| Antiplatelet | 2,801 (49.7) | 332 (64.8) | <0.001 |
| Renin angiotensin aldosterone system inhibitor | 2,257 (40.1) | 279 (54.5) | <0.001 |
| Direct oral anticoagulant | 182 (3.2) | 26 (5.1) | 0.04 |
| In-hospital care | | | |
| Cardiologist evaluation | 1,163 (20.6) | 185 (36.1) | <0.001 |
| Percutaneous coronary intervention | 23 (0.4) | 25 (4.9) | <0.001 |
| Coronary artery bypass grafting | 5 (0.1) | 3 (0.6) | 0.02 |
| Intensive care unit | 2,396 (42.5) | 225 (43.9) | 0.57 |
| ECMO | 11 (0.2) | 21 (4.1) | <0.001 |
| Continuous renal replacement therapy | 17 (0.3) | 15 (2.9) | <0.001 |
| Ventilator | 53 (0.9) | 25 (4.9) | <0.001 |

**Table 4** (*continued*)

**Table 4** (*continued*)

| Managements | Entire population | | |
|---|---|---|---|
| | No troponin I elevation (n=5,633) | Troponin I elevation (n=512) | P value |
| Management after discharge | | | |
| Postdischarge evaluation | | | |
| Echocardiogram | 2,108 (37.4) | 265 (51.8) | <0.001 |
| Stress echocardiogram | 178 (3.2) | 11 (2.1) | 0.26 |
| Treadmill test | 123 (2.2) | 15 (2.9) | 0.35 |
| Coronary computed tomographic angiography | 225 (4.0) | 15 (2.9) | 0.28 |
| Coronary artery angiogram | 324 (5.8) | 71 (13.9) | <0.001 |
| Postdischarge diagnosis | | | |
| Myocardial infarction | 50 (0.9) | 15 (2.9) | <0.001 |
| ST-elevation | 5 (0.1) | 3 (0.6) | 0.02 |
| Non ST-elevation | 45 (0.8) | 12 (2.3) | 0.001 |
| Postdischarge cardiovascular drugs | | | |
| Beta-blocker | 1,096 (19.5) | 136 (26.6) | <0.001 |
| Calcium channel blocker | 2,623 (46.6) | 277 (54.1) | 0.001 |
| Statin | 2,867 (50.9) | 292 (57.0) | 0.01 |
| Warfarin | 451 (8.0) | 74 (14.5) | <0.001 |
| Antiplatelet | 3,304 (58.7) | 334 (65.2) | 0.004 |
| Renin angiotensin aldosterone system inhibitor | 2,739 (48.6) | 316 (61.7) | <0.001 |
| Direct oral anticoagulant | 362 (6.4) | 57 (11.1) | <0.001 |
| Postdischarge care | | | |
| Cardiologist evaluation | 1,504 (26.7) | 214 (41.8) | <0.001 |
| Percutaneous coronary intervention | 149 (2.6) | 37 (7.2) | <0.001 |
| Coronary artery bypass grafting | 11 (0.2) | 2 (0.4) | 0.68 |
| Intensive care unit | 874 (15.5) | 127 (24.8) | <0.001 |
| ECMO | 51 (0.9) | 9 (1.8) | 0.1 |
| Continuous renal replacement therapy | 57 (1.0) | 18 (3.5) | <0.001 |
| Ventilator | 47 (0.8) | 14 (2.7) | <0.001 |
| Mortality | | | |
| 1-year mortality | 182 (3.2) | 51 (10.0) | <0.001 |
| 30-day mortality | 19 (0.3) | 13 (2.5) | <0.001 |
| In-hospital mortality | 12 (0.2) | 11 (2.1) | <0.001 |

Data are presented as n (%) or median [interquartile range]. ECMO, extracorporeal membranous oxygenation.

taken at scheduled admission from the outpatient clinic, and only 0.4% and 1.1% of patients were diagnosed with myocardial infarction afterward during hospitalization and after discharge, respectively. This trend is consistent with the results of past studies conducted in the emergency room (21,22), and so elevated cTnI is mostly used only to rule out myocardial infarction in the emergency room rather than to make a final diagnosis (23). Meanwhile, the use of cTnI level for stable patients in an outpatient setting has gained relatively less attention.

In this study, we aimed to evaluate whether cTnI measurement could be helpful in relatively stable patients by affecting patient management. So, we excluded the patients from emergency department or admitted to cardiology or cardiac surgery part in which cTnI measurement has definitely shown benefit. Instead, we selected patients with comorbidities that had an actual effect on prognosis, by adopting coding algorithms using the International Classification of Diseases to calculate the CCI, which has been validated in numerous large studies (15,24). According to our result, cardiac risk factors such as cardiac symptoms and previous cardiovascular diseases were more frequently found in the cTnI group. This seems to be due to the selective measurement of cTnI by attending clinicians on patients with comorbidities. In addition, myocardial infarction was more frequently diagnosed in cTnI group. According to our results, the cTnI group underwent more intensive cardiac evaluation and therapy which may have resulted in reduced mortality in patients with cTnI measurement. An active cardiac evaluation may have lowered the rate of missed diagnosis of coronary artery disease with cTnI measurement. This explanation could be supported by the fact that more active managements were performed in patients with elevated cTnI elevation and this might have affected reduced mortality. Our result also showed that mortality was higher in patients with elevated cTnI despite an active management, so it is likely that the increase of mortality could become more pronounced when cTnI elevation is undetected. However, selection criteria of patients for cTnI measurement could not be answered from our result. Further prospective studies may be needed to warrant criteria for cTnI testing. In noncardiac surgical patients, routine cTn evaluation is recommended in patients with cardiovascular risk >5%, and it has also shown to be cost-effective (25).

Because the reduced mortality was only observed for the long-term follow-up, the effect of cardiovascular drugs should also be considered. An intensification of cardiovascular drugs may be helpful for patients with cTnI elevation without ischemic injury. A minor elevation of cTnI in the general population was reported to be common and associated with the development of cardiovascular disease and mortality (26,27). Although it is yet unclear how to manage stable patients at high risk for cardiovascular events, the introduction of an intensified cardiovascular therapy regimen may play a protective role in these patients. In fact, cTnI measurement has shown to be useful in identifying patients in whom cardiovascular therapy may be helpful. According to recent studies regarding guidelines for cholesterol management and antihypertensive medication, the incorporation of cTnI level measurements improved risk stratification and identified patients in need of more aggressive preventive therapies (28,29). In conjunction with the consistent evidence for the prognostic value of cTnI level in stable patients with cardiac injury (4,5), the more intensive use of cardiovascular drugs may also be helpful in promoting long-term mortality reduction among these patients.

The prognostic value of cTnI has been demonstrated, usually in the short-term period, in several studies to date (30,31). However, our study did not reveal a significant difference in the 30-day mortality rate despite the receipt of more active management during hospitalization, such as admission to the intensive care unit, in the cTnI group. This finding may be due to the presence of more severely ill patients in the cTnI group. However, on the one hand, it can be assumed that the effect of intensive treatment offered following the cTnI test prevented an increase in short-term mortality; on the other, our results suggest the long-term prognostic value of cTnI evaluation in chronically stable patients, not those in the acute phase. According to previous studies, cTnI is an independent predictor of long-term mortality in patients with unstable angina or severe sepsis (32,33). Similar to these results, cTnI testing may be useful in determining a more aggressive approach to the treatment of patients with clinical symptoms relevant to poor prognosis.

In the subgroup analysis, the observed association significantly interacted with whether patients were admitted for surgery. In patients who were admitted for planned surgery, the cTnI test at admission was not associated with one-year mortality. This may be explained by the large contribution of surgical procedures on prognosis. Postoperative mortality is largely dependent upon the extent and severity of surgical procedures. Additionally, perioperative cTnI has recently gained attention for its association with postoperative mortality (34,35). Thus,

recent guidelines to monitor postoperative cTnI in high-risk patients might have affected clinical practice to offset the effects of cTnI measurement at admission.

There are several limitations to be noted when interpreting our results. First, as this was a single-center, observational study, a degree of residual confounding factor-related bias may have persisted. In particular, the reasons for admission other than the scheduled operation were too broad, so it was difficult to retrospectively identify all of them. In addition, specific departments of admission might have affected the results. Therefore, it is possible that these missed confounding variables may have influenced the outcome despite our rigorous statistical adjustment. Second, we conducted our study only involving stable patients on an outpatient basis, so our results may not be generalizable to other settings and does not provide selection criteria for cTnI measurement. Third, we collected the results of cTnI tests performed only at admission. Considering that cTnI level measurements are obtained serially in real clinical situations, the true prevalence of patients with elevated cTnI levels might have been underestimated. Despite these limitations, we demonstrated that cTnI testing at admission from the outpatient clinic may affect mortality in patients with comorbidities. In addition to previous studies emphasizing the importance of cTnI measurement in emergency or intensive care settings, this study suggests a possibility that cTnI measurement may be helpful in managing relatively stable patients.

## Conclusions

The measurement of cTnI in relatively stable patients at scheduled admission to medical departments was associated with reduced mortality during one year of follow-up. This could be related to more active evaluation and treatment. Further studies are needed to validate our results.

## Acknowledgments

Research support was provided solely by institutional and/or departmental sources.
*Funding*: None.

## Footnote

*Reporting Checklist*: The authors have completed the STROBE reporting checklist. Available at https://atm.amegroups.com/article/view/10.21037/atm-22-3681/rc

*Data Sharing Statement*: Available at https://atm.amegroups.com/article/view/10.21037/atm-22-3681/dss

*Conflicts of Interest*: All authors have completed the ICMJE uniform disclosure form (available at https://atm.amegroups.com/article/view/10.21037/atm-22-3681/coif). The authors declare no conflicts of interest.

*Ethical Statement*: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted according to the Declaration of Helsinki (as revised in 2013), and approved by the Institutional Review Board of Samsung Medical Center (No. SMC 2021-03-165, date of approval: 2019-03-13) and individual consent for this retrospective analysis was waived.



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**Cite this article as:** Oh AR, Lee SH, Park J, Choi DC, Yang K. Association between cardiac troponin testing at scheduled admission and mortality in patients with comorbidities. Ann Transl Med 2023;11(1):7. doi: 10.21037/atm-22-3681